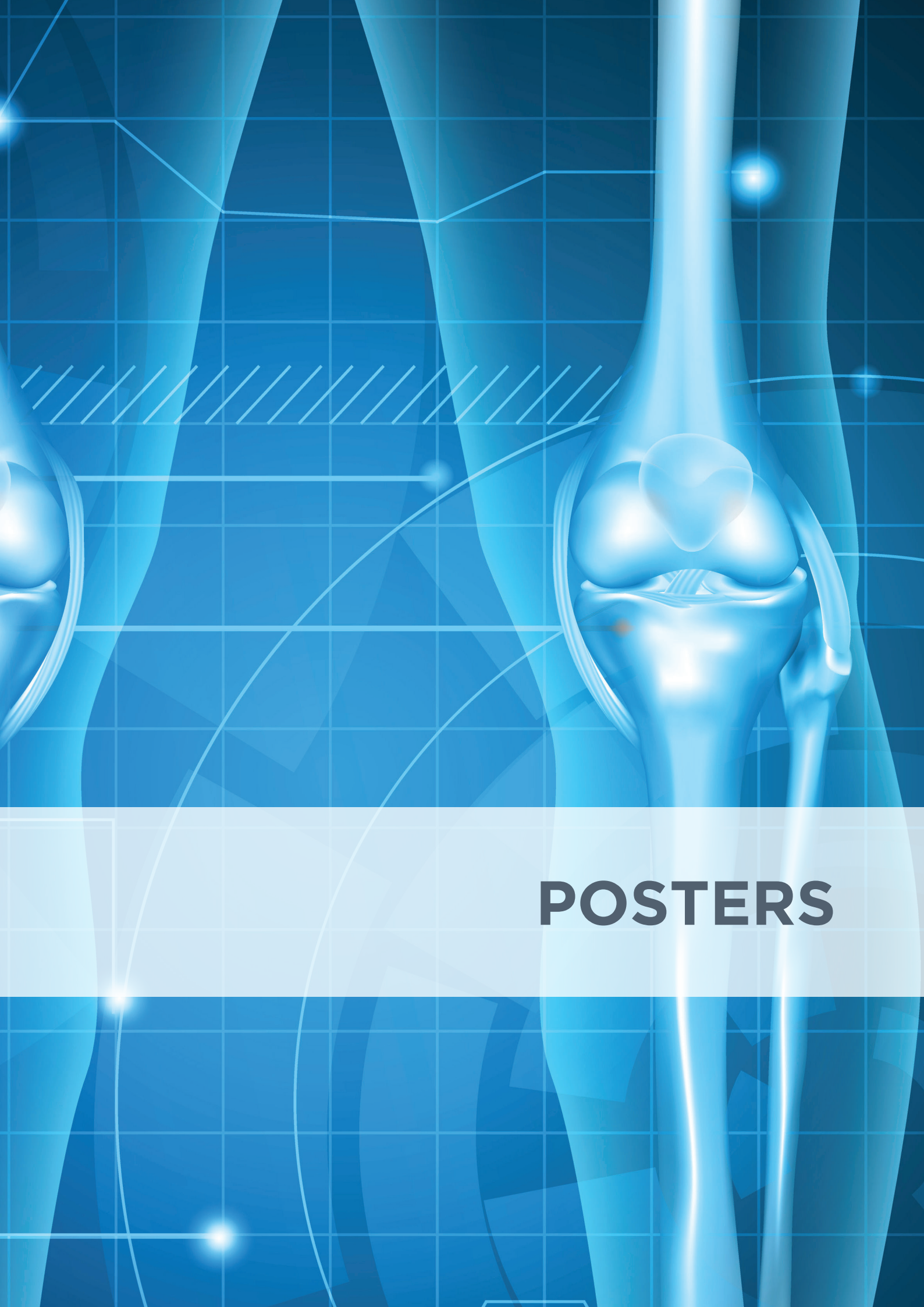

POSTERS

Trabalho Original

006 - ANTIFIBROTICS IN RHEUMATOID ARTHRITIS-ASSOCIATED INTERSTITIAL LUNG DISEASE – REAL-WORLD DATA FROM A PORTUGUESE COHORT

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Introduction: Interstitial lung disease (ILD) is the most common pulmonary manifestation of rheumatoid arthritis (RA) and is associated with an increased mortality. Clinical trials have demonstrated that antifibrotics, including nintedanib and pirfenidone, might slow connective tissue disease-associated ILD progression. The aim of this work is to evaluate the effectiveness and tolerability of antifibrotics in a national, real-world cohort of patients with RA-ILD.

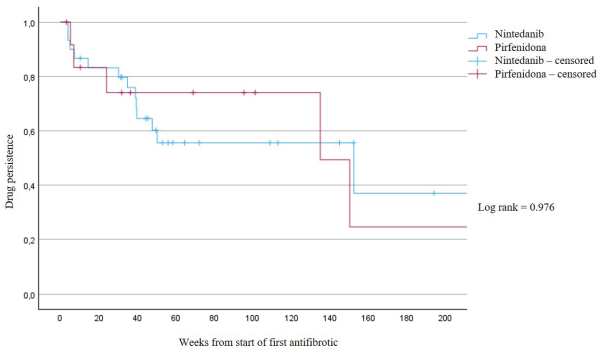
Methods: We conducted an observational, retrospective, multicenter study of RA-ILD patients treated with antifibrotics, prospectively followed in Reuma.pt. Demographic and clinical data, pulmonary function tests (PFTs) results and adverse events (AEs) were collected until March 2024. A linear mixed model with random intercept was used to compare results from PFTs within 12 (±6) months before to 12 (±6) months after antifibrotic initiation. Drug persistence was evaluated using Kaplan-Meier curves.

Results: We included 40 patients with RA-ILD treated with antifibrotics, who had a median RA-ILD duration at last follow-up appointment of 5 [2.3 – 7.5] years. Nintedanib was used as first antifibrotic drug in 27 (67.5%) patients and pirfenidone in 13 (32.5%). From the 13 patients receiving pirfenidone as first-drug, two were switched to nintedanib due to AEs. The characteristics at first antifibrotic initiation are showed in Table 1.

Effectiveness analysis was performed in 20 patients. The use of antifibrotics interrupted the decline of/ slightly improved forced vital capacity (FVC; decline of 300mL in the year before antifibrotic initiation vs. improvement of 200mL in the year following antifibrotic initiation, p=0.336) and total lung capacity (TLC; decline of 800mL in the year before antifibrotic initiation vs. improvement of 600 mL in the year following antifibrotic initiation, p=0.147), but had no effect on diffusion capacity for carbon monoxide decline (decline of 3% in the year before antifibrotic initiation vs. decline of 2.9% in the year following antifibrotic initiation, p=0.75). There were nine deaths, 6.7 ± 3.5 years after RA-ILD diagnosis. Four deaths were related to ILD progression and other four were due to infection (two SARS-CoV2, one Pneumocystis jirovecii, one unknown agent). One patient died of unknown cause.

At the end of follow-up, AEs were reported in 16 patients (40%), thirteen receiving nintedanib and three pirfenidone. Gastrointestinal events were registered in the six patients who had the type of AE specified (four nintedanib, two pirfenidone). In five patients treated with nintedanib, dose reduction led to symptoms resolution in four of them. In the remaining patient the drug was discontinued. From the five patients who reported AEs with pirfenidone, two were switched to nintedanib, which they kept until last appointment.

The initial antifibrotic was discontinued in eighteen



TO 006 – Figure 1. Kaplan Meier curves comparing drug persistence after starting the first antifibrotic

TO 006 – TABLE 1. Characteristics of patients with RA-ILD at initiation of first antifibrotic drug

	Overall (N=40)	Nintedanib (N=27; 67.5%)	Pirfenidone (N=13; 32.5%)
Demographic and lifestyle			
Age (mean ± SD)	70.9 ± 7.1	70.3 ± 7.2	72.2 ± 6.8
Male sex	18 (45%)	9 (33.3%)	9 (69.2%)
Caucasian, missing data = 7	31 (93.9%)	20 (95.2%)	11 (91.7%)
Smoking habits, missing data = 2			
- Current	6 (15.8%)	5 (20%)	1 (7.7%
- Past	15 (39.5%)	6 (24%)	9 (69.2%)
- Never	17 (44.7%)	14 (56%)	3 (23.1%)
RA characteristics			
RA duration (median [IQR]), missing data = 10	12 [5.5-21.75]	15 [5.75-30]	12 [IQR 2.5-20]
Positive RF, missing data = 2	35 (92.1%)	24 (96%)	11 (84.6%)
Positive ACPA, missing data = 2	36 (94.7%)	24 (96%)	12 (92.3%)
Concomitant RA medication			
- Corticosteroids	25 (62.5%)	17 (63%)	8 (61.5%)
- MTX	15 (37.5%)	9 (33.3%)	6 (46.2%)
- Other csDMARDs	15 (37.5%)	10 (37%)	5 (38.5%)
- Rituximab	21 (52.5%)	18 (66.7%)	3 (23.1%)
- Anti-TNFα	3 (7.5%)	1 (3.7%)	2 (15.4%)
- Abatacept	3 (7.5%)	2 (7.4%)	1 (7.7%)
- Anti-IL6	1 (2.5%)	0	1 (7.7%)
ILD characteristics			
ILD duration (median [IQR]), missing data = 5	5 [2.3-7.5]	5.25 [3-7.38]	5 [1-7.5]
HRCT ILD pattern, missing data = 6			
- UIP	29 (85.3%)	18 (81.8%)	10 (83.3%)
- Fibrotic NSIP	4 (11.7%)	3 (13.6%)	2 (16.7%)
- Unclassifiable	1 (3%)	1 (4.5%)	0
Mean FVC % (L), missing data = 13	80.4 (2.5)	79.6 (2.5)	81.2 (2.5)
Mean TLC % (L), missing data = 21	80.3 (4.5)	82 (4.6)	78.5 (4.4)
Mean DLCO %, missing data = 19	58.1	54.9	61.4
Supplemental oxygen therapy	6 (15%)	4 (14.8%)	2 (15.4%)

Legend: RA – rheumatoid arthritis; ILD – interstitial lung disease; RF – rheumatoid factor; ACPA – anti-citrullinated peptide antibodies; csDMARDs – conventional synthetic disease modifying antirheumatic drugs; MTX – methotrexate; HRCT – high resolution computed tomography; UIP – usual interstitial pneumonia; NSIP – nonspecific interstitial pneumonia; FVC – forced vital capacity; TLC – total lung capacity; DLCO – diffusion capacity for carbon monoxide

(45%) patients (thirteen nintedanib, five pirfenidone). Reasons for discontinuation were AEs (n=12), death (n=5) and patient decision (n=1). The median drug persistence was 150.3 weeks (95 %CI 11, 289.6) with no difference between nintedanib and pirfenidone (Figure 1).

Conclusions: This real-world study demonstrated a benefit, albeit modest, of antifibrotics in stabilizing lung function in RA-ILD. However, particular attention must be paid to AEs, as they are quite common in patients receiving antifibrotics, and a major cause of drug discontinuation. We expect this study to reinforce the promising results demonstrated in clinical trials, leading to a widespread use of these drugs.

007 - A TWO-YEAR COMPARISON OF SPINAL AND HIP MOBILITY BETWEEN AXIAL SPONDYLOARTHRITIS AND CHRONIC BACK PAIN PATIENTS IN THE SPONDYLOARTHRITIS CAUGHT EARLY (SPACE) COHORT

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Background: Axial spondyloarthritis (axSpA) frequently leads to reduced spinal and hip mobility, though evidence pertains mostly to patients with longstanding disease and radiographic damage. More recently, impaired mobility has also been reported in early axSpA. However, little is known about how spinal and hip mobility change in the first years of the disease, especially in comparison to patients with non-axSpA chronic back pain (CBP). We aimed to compare spinal and hip mobility at baseline and after 2 years (2y) in early axSpA and non-axSpA CBP patients.

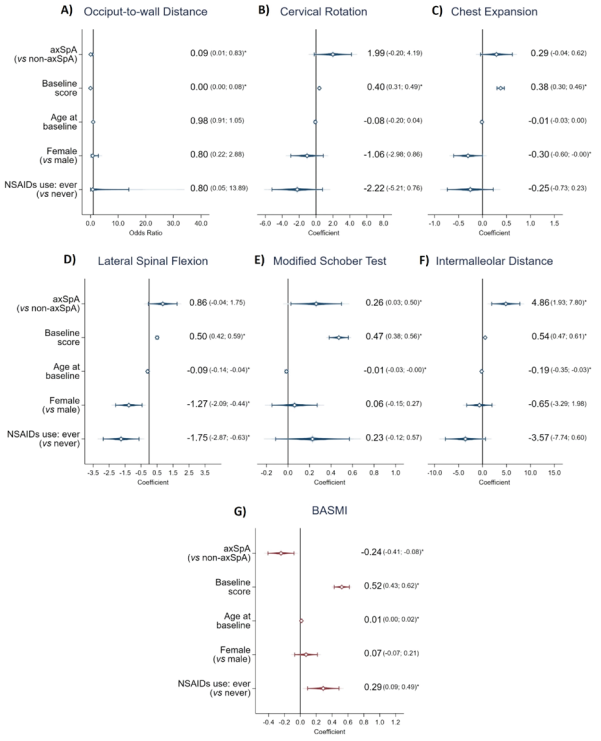
Methods: Baseline and 2y data of the SPondyloArthritis Caught Early (SPACE) cohort (CBP ≥3 months and ≤2y, starting <45 years) were analyzed. Patients had a diagnosis of axSpA or non-axSpA given by the treating rheumatologist at 2y, with a high level of confidence.1 The following mobility measures were assessed: occiput-to-wall distance (OWD), cervical rotation, chest expansion, lateral spinal flexion (LSF), modified Schober test (mSchober) and intermalleolar distance (IMD). BASMI was calculated. The proportion of patients with an impaired measure (<2.5th percentile curves derived from healthy individuals;2 >97.5th for BASMI; >0cm for OWD) was also reported. For the assessment of each outcome, only patients with data available at both baseline and 2y were included. Paired t-test (or Wilcoxon signed rank test, as appropriate) was used to compare baseline and 2y results within groups. Linear or zero-inflated negative binomial regression was conducted to compare 2y outcomes between groups, adjusting for the baseline value (comparable to modelling a 2y change in the outcome), age, sex and NSAIDs use.

Results: Data was available at both timepoints on ≥1 of the assessed outcomes in 286 axSpA and 117 non-axSpA patients. AxSpA (vs non-axSpA) patients were more frequently male (52% vs 25%) and had more SpA features (mean [SD]: 5 [2] vs 3 [1]). Age (mean [SD]: 30 [8] vs 31 [8] years) and symptom duration (mean [SD]:

TO 007 – TABLE 1. Spinal and hip mobility measures at baseline and 2 years in axSpA and non-axSpA chronic back pain patients

	AxSpA			Non-axSpA		
	Baseline	2 years	p-value (within group)	Baseline	2 years	p-value (within group)
Occiput-to-wall distance, mean (SD)	0.5 (1.2)	0.4 (1.1)	p=0.267	0.1 (0.7)	0.1 (0.5)	p=0.474
Impaired, n (%)	65 (23)	61 (22)		6 (5.1)	4 (3.4)	
Cervical rotation, mean (SD)	74.8 (12.0)	77.2 (10.6)	p=0.001*	70.7 (12.0)	73.5 (11.2)	p=0.015*
Impaired, n (%)	32 (11)	16 (5.6)		12 (10)	9 (7.7)	
Chest expansion, mean (SD)	5.9 (1.9)	6.0 (1.7)	p=0.274	5.3 (1.9)	5.4 (1.5)	p=0.365
Impaired, n (%)	21 (7.6)	14 (5.0)		16 (14)	9 (7.7)	
Lateral spinal flexion, mean (SD)	17.9 (4.8)	19.0 (4.6)	p<0.001*	16.4 (4.3)	17.1 (5.0)	p=0.058
Impaired, n (%)	84 (30)	55 (20)		46 (39)	39 (33)	
Modified Schober test, mean (SD)	4.9 (1.2)	5.2 (1.1)	p<0.001*	4.7 (1.1)	4.8 (1.2)	p=0.346
Impaired, n (%)	23 (8.0)	10 (3.5)		12 (10)	12 (10)	
Intermalleolar distance, mean (SD)	117.4 (15.3)	118.2 (14.2)	p=0.337	107.9 (21.5)	108.2 (19.2)	p=0.886
Impaired, n (%)	13 (4.6)	10 (3.5)		27 (23)	19 (16)	
BASMI, mean (SD)	1.8 (0.9)	1.6 (0.7)	p<0.001*	2.3 (0.8)	2.1 (0.9)	p=0.010*
Impaired, n (%)	42 (19)	16 (7.3)		41 (39)	26 (25)	
≥1 impaired spinal mobility measure#, n(%)	146 (51)	117 (41)		62 (53)	55 (47)	

Includes occiput-to-wall distance, cervical rotation, chest expansion, lateral spinal flexion and modified Schober test.
* Statistical significance.



A) Multivariable zero-inflated negative binomial model for occiput-to-wall distance. Logit model represented as odds ratio (95% CI). **B)** to **G)** Multivariable linear regression models for cervical rotation, chest expansion, lateral spinal flexion, modified Schober test, intermalleolar distance and BASMI. Results are presented in β (95% CI).
*p<0.05

TO 007 – Figure 1. Impact of axSpA vs non-axSpA chronic back pain on 2-years spinal and hip mobility measures

13 [7] vs 14 [7] months) were similar between groups.

At baseline, 51% axSpA and 53% non-axSpA patients had ≥1 impaired spinal mobility measure (Table 1). Overall, poorer mobility was observed in non-axSpA (vs axSpA) patients, except for OWD. After 2y, cervical rotation and BASMI significantly improved in both groups (mean [SD] improvement axSpA vs non-axSpA: cervical rotation 2.4 [11.9] vs 2.8 [12.1]; BASMI 0.2 [0.8] vs 0.2 [0.7]), and LSF and mSchober only in axSpA (LSF 1.1 [4.5]; mSchober 0.3 [1.2]).

In adjusted multivariable analysis (Figure 1), at 2y, axSpA (vs non-axSpA) was associated with larger improvements in mSchober (β [95% CI]: 0.26 [0.03; 0.50]), IMD (4.86 [1.93; 7.80]) and BASMI (-0.24 [-0.41; -0.08]), and with higher odds of OWD impairment (OR [95% CI]: 0.09 [0.01; 0.83]). No differences between groups were observed for cervical rotation, chest expansion or LSF.

Conclusion: Impaired spinal and hip mobility are common in early axSpA and, most notably, non-axSpA patients. After 2y, mobility measures remain relatively unchanged. Nevertheless, axSpA is associated with larger improvements in mSchober, IMD and BASMI, and with higher odds of OWD impairment.

REFERENCES:

1. Marques ML, ARD 2024;83:589-98; 2. Ramiro S, ARD 2015;74:1218-24

009 - THE USE OF ASDAS IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS STARTING BDMARDS IN CLINICAL PRACTICE: RESULTS FROM A MULTICENTRE PROSPECTIVE COHORT

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Background: The ASAS-EULAR recommendations for the management of axSpA prescribe the use of the ASDAS to measure disease activity and response to treatment in patients starting bDMARDs.[1] Evidence on whether rheumatologists adhere to this recommen-

TO 009 – TABLE 1. ASDAS EVALUATION AFTER THE START OF THE 1ST BDMARD AT T0 and in ≥1 of the 2 follow-up visits

		ASDAS evaluation at T1 and/or T2		
		Yes	No	TOTAL
ASDAS evaluation at T0	Yes	**493 (74)**	47 (7)	540 (81)
	No	106 (16)	20 (3)	126 (19)
	TOTAL	599 (90)	67 (10)	666

Values are n (%). The denominator is the total number of patients (N=666) in all cells.

dation in clinical practice is still limited. [2]

Objectives: We assessed: i. how many patients with axSpA, starting the first bDMARD, have ASDAS determined at baseline and in ≥1 of two follow-up visits within 6 months; and ii. which alternative outcome measures are used in patients for whom the ASDAS is missing.

Methods: Patients with axSpA from the Reuma.PT registry starting the first bDMARD (2011-2022) were included. Patients were required to have attended the following 3 visits: T0 (baseline visit at the start of the bDMARD), T1 (3 months) and T2 (6 months). The calculation of ASDAS at T0 (yes vs no) was cross-tabulated with the calculation of ASDAS in ≥1 of the two follow-up visits. The use of other outcome measures among patients without an ASDAS evaluation was evaluated.

Results: In total, 666 patients with axSpA [male: 55%; mean age: 43 (SD 12)] were included. Most patients had an ASDAS calculation at baseline (N=540; 81%), and in 493 (74%) of the patients, ASDAS was also assessed at T1 and/or T2 (Table 1). No other outcome measure was predominantly used when ASDAS was absent. For instance, among 126 patients (19%) without ASDAS at baseline, SJC (52%), PGA (44%) and BASDAI (35%) were all similarly used without a clear preference. Of note, CRP was available for most of these 126 patients (87%).

Conclusions: Portuguese rheumatologists adhere to the ASAS-EULAR recommendation of using ASDAS. Failing to use ASDAS does not seem to be explained by missing CRP or a preference for another disease activity score, but rather by the rheumatologist's willingness to use measurement instruments in general.

REFERENCES:

1. S. Ramiro et al. Ann Rheum Dis. 2023; [2] E. Portier et al. Rheumatol Int. 2022

013 - THE RELATIONSHIP BETWEEN SMOKING STATUS AND SJÖGREN'S DISEASE DIAGNOSIS AND DISEASE ACTIVITY

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Background: The relationship between smoking and the risk of Sjögren's disease (SjD) remains unclear. Most studies show that current smoking is negatively correlated with SjD whereas past smoking may be positively associated. The possibility of reverse causation, whereby patients stop smoking due to dryness, has not been excluded.

Objectives: To assess if smoking status is a risk factor for SjD and how it affects the main features of the disease.

Methods: Patients referred with suspected SjD were recruited into the Optimising Assessment in Sjögren's Syndrome (OASIS) cohort established in Birmingham, UK (2014-2023). Subjects were assessed and diagnosed as SjD or non-Sjögren's sicca. Smoking status was regularly obtained within clinic with additional information available through a risk factor questionnaire. Univariate analysis was performed using chi-square, Fisher's exact, Mann- Whitney or t-test, and correlation between continuous variables using Pearson or Spearman, according to their distribution. Predictors of Sjogren's disease diagnosis were identified through binomial logistic regression modelling.

Results: 487 patients were included, 170 non-Sjögren's sicca syndrome and 317 SjD patients, 81.1% of whom (n=257) met 2016 ACR/EULAR classification criteria for SjD. Compared to sicca, SjD patients meeting 2016 criteria are more frequently never smokers and, amongst ever smokers, have a lower smoking exposure measured by pack-year units (Table 1). Time between onset of symptoms and smoking cessation for past smokers was calculated and the majority of patients stopped smoking before symptom onset. In the multivariate analysis, both current smoking (OR 0.37, 95%CI: 0.15-0.92, p=0.032) and white ethnicity (OR 0.26, 95%CI: 0.12-0.56, p=0.001) were protective factors for SjD diagnosis in suspected patients, irrespective of age and sex. Amongst SjD patients meeting 2016 criteria, rheumatoid factor (p=0.038), IgG (p=0.004) and IgM (p=0.006) levels are significantly higher in never smokers with no passive smoking history. Amongst patients with a clinical diagnosis of Sjögren's, those with smoking exposure are less frequently positive for anti-Ro antibodies (p=0.008) and rheumatoid factor (p=0.016) and have lower IgG levels (p=0.002). An inverse correlation trend was observed between smoking exposure (pack-years) and IgG levels (rho=-0.186, p=0.076) in the whole population. Never smoking was confirmed as a predictor of IgG levels (β 1.94, 95%CI: 0.12-3.77, p=0.037) in a linear regression model including age, sex, ethnicity, and disease classification [R2=0.287, F (5,254) = 20.487, p<0.001]. Looking at 2016 criteria SjD patients, ESSPRI is higher in ever smokers (p=0.039), with fatigue (p=0.040) being accountable for this difference. Likewise, amongst sicca patients, symptoms of fatigue, but not dryness, were significantly higher in ever smokers (p=0.022). No differences in the main features of minor salivary gland biopsies attributable to smoking were seen.

Conclusion: Our study shows that smoking is negatively associated with SjD. In the multivariate analysis, current smoking and white ethnicity were protective factors for SjD classification in suspected patients, irrespective of age and sex. Avoidance of factors that may exacerbate dryness symptoms might not explain the association with smoking status. Clinically diagnosed SjD patients exposed to tobacco are more frequently seronegative and have lower rheumatoid factor and IgG levels.

TO 013 – TABLE 1. Clinical and laboratorial characteristics in cases and controls

		Sjögren's disease* (n=257)	Sicca (n=170)	Univariate analysis (p-value)
Age at inclusion, years		54.0±15.2 (256)	54.9±12.0 (168)	0.765
Female sex		242/257 (94.2)	152/170 (89.4)	0.095
Age at symptom onset, years		45.7±15.5 (248)	48.0±13.3 (155)	0.131
Ethnicity	**White**	**134/205 (65.4)**	**104/118 (88.1)**	**<0.001**
	Asian	**50/205 (24.4)**	**10/118 (8.5)**	
	Black	**9/205 (4.4)**	**3/119 (2.5)**	
	Other	**12/205 (5.9)**	**1/119 (0.8)**	
BMI		**27.4±9.0 (249)**	**28.5±6.5 (164)**	**0.027**
Smoking status	**Never smoker**	**155/220 (70.5)**	**76/143 (53.1)**	**0.002**
	Current smoker	**13/220 (5.9)**	**18/143 (12.6)**	
	Past smoker	**52/220 (23.6)**	**49/143 (34.3)**	
Passive smokers (amongst never smokers)		43/92 (46.7)	27/51 (52.9)	0.491
Smoking status	**Non-exposed**	**49/157 (31.2)**	**24/118 (20.3)**	**0.032**
	Passive smoker	**43/157 (27.4)**	**27/118 (22.9)**	
	Ever smoker	**65/157 (41.4)**	**67/118 (56.8)**	
Pack-year units		**10.8±11.0 (37)**	**15.3±13.1 (40)**	**0.048**
Smoking cessation	>5 years before symptom onset	28/39 (71.8)	25/37 (67.6)	0.725
	<5 years before symptom onset	7/39 (17.9)	5/37 (13.5)	
	<5 years after symptom onset	2/39 (5.1)	4/37 (10.8)	
	>5 years after symptom onset	2/39 (5.1)	3/37 (8.1)	

Data presented as mean ± SD (N) or n/N (%), as adequate; Abbreviations: n – number of positive patients, N – number of patients without missing information, SD – standard deviation, BMI – body mass index; *meeting 2016 ACR/EULAR classification criteria

014 - ASDAS RESPONSES IN PATIENTS WITH AXIAL SPONDYLOARTHRITIS STARTING BDMARDS: RESULTS FROM A MULTICENTRE PROSPECTIVE COHORT

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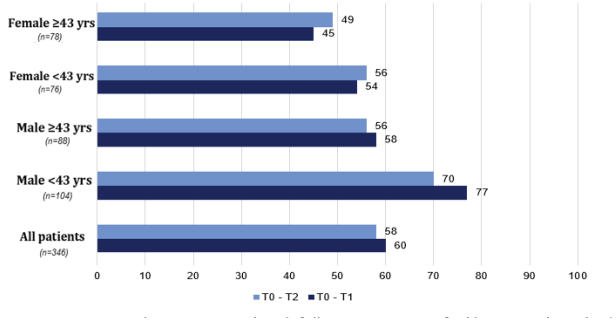
Background: ASAS-EULAR recommends the use of an improvement ≥1.1 in ASDAS at 12 weeks to determine the continuation of a bDMARD.[1] However, it is debated whether improvements can occur and whether patients' characteristics influence (time to) response.
Objectives: To assess the likelihood of fulfilling the ASAS-EULAR criteria for treatment continuation at 3 and 6 months after the start of bDMARDs and whether there are patient characteristics that influence this response.
Methods: Patients with axSpA from the Reuma.PT registry who started the first bDMARD (2011-2022) were included. Complete data on ASDAS at T0 (baseline visit at bDMARD starting), T1 (3 months), and T2 (6 months) were required. ASDAS response criteria (decreased ≥ 1.1 compared with baseline) at T1 and T2 were determined. Patient characteristics (e.g., age, sex) were compared across four groups: no response in both visits, response only at T1 and only at T2, and response in both visits. If relevant differences were found, the response at T1 and T2 was then evaluated in the subgroups stratified by these characteristics.
Results: In total, 346 patients [male: 56%; mean age: 43 (SD 12)] were included. After 3 months, 199 (58%) patients fulfilled the criteria of treatment continuation (Table 1). This number was similar at 6 months (N=207; 60%). The four groups were comparable except for sex and age: compared with those who never responded, patients who responded at both visits were more often male (65% vs 45%) and somewhat younger (mean age: 42 vs 45). Analysis across subgroups showed that young males had the highest likelihood of response, while females had lower response rates regardless of age (Table 2).
Conclusions: The likelihood of delayed response (>3 months) is low, which should prompt questioning whether it is justified to wait for 6 months to decide on the continuation of bDMARDs. Young male patients are more likely to respond than female patients, irrespective of their age, and apparently, there are many patients' characteristics that influence the time to respond.



TO 014 – Figure 1. ASDAS response between T0 and each follow-up visit, stratified by age and gender (%)

REFERENCES:

1. S. Ramiro et al. Ann Rheum Dis. 2023

021 - IMPACT OF A BEHAVIOUR-CHANGE INFORMED HYBRID PHYSICAL EXERCISE PROGRAM FOR INDIVIDUALS WITH FIBROMYALGIA

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TO 014 – TABLE 1. ASDAS response between T0 and each follow-up visit among patients with ASDAS available in all 3 visits

		ASDAS response T0 → T2		
		Yes	No	TOTAL
ASDAS response T0 → T1	Yes	171 (50)	28 (8)	199 (58)
	No	36 (10)	111 (32)	147 (42)
	TOTAL	207 (60)	139 (40)	346 (100)

Values are n (%). The denominator is the total number of patients (N=346) in all cells.

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Background: The recommendations for the intervention in fibromyalgia have reinforced the importance of self-management programs. Physical exercise plays an important role, but adherence tends to be low [1]. The use of behaviour-change techniques, as well remote and/ or digital solutions may promote adherence and overcome barriers to regular physical exercise [2]. This study is part of the Selfie Project "Supported self-management of fibromyalgia, co-designing ehealth interventions", in which a behaviour-change informed hybrid physical exercise program for individuals with fibromyalgia was developed, concurrently with a mobile app (Selfie), developed in collaboration with patients and health professionals.
Objectives: To study the impact of a behaviour-change informed hybrid exercise intervention using a mobile app (Selfie), on pain intensity, functional capacity and impact, perceived global impression of change, self-efficacy to manage the chronic disease and physical fitness in individuals with fibromyalgia, and to contribute for the study of usability of Selfie mobile app.
Methods: A case series study design with descriptive analysis was used. After obtaining ethical approval, participants completed a 12-week behaviour-change informed hybrid exercise program, using a mobile app. Participants were assessed before starting the program and after 6 and 12 weeks, with the Numeric Pain Rating Scale, and the Portuguese versions of the Revised Fibromyalgia Impact Questionnaire, Self-efficacy for Managing Chronic Disease 6-Item, Patient Global Impression of Change and System Usability Scale. At the initial and final assessments, a physical fitness test battery was applied. The program comprised 3 weekly individual and personalized exercise sessions (aerobic, resistance and flexibility), 2 with supervision by the physiotherapist (face-to-face or remote), and the third one autonomously.
Results: Twelve participants enrolled in the program; two dropped out due to time restrictions. Ten participants completed the exercise program. The descriptive analysis revealed clinically important differences in the Numeric Pain Rating Scale (30%; 5participants), Revised Fibromyalgia Impact Questionnaire (14%; 7participants) and Patient Global Impression of Change (≥5; 8participants), at the final assessment. Concerning physical fitness, the analysis revealed minimal detectable changes in the lower limb strength test (2.52 repetitions; 6participants), upper limb strength test (3.16 repetitions; 7participants), balance (1.6 seconds; 7participants) and endurance (65.2 meters; 6participants). Participants rated the Selfie app usability between 62.5 and 87.5, out of 100.
Conclusion: The hybrid physical exercise program informed by behaviour change techniques promoted important improvements in the studied outcomes, suggesting that this approach might be promising for people with fibromyalgia. The usability scores revealed different experiences within participants and contributed for future improvements.
Keywords: fibromyalgia, self-management, behaviour change, hybrid physical exercise program

REFERENCES:

1. Macfarlane,GJ, Kronisch,C, Dean,LE, et al. (2017). EULAR revised recommendations for the management of fibromyalgia. Annals of the Rheumatic Diseases, 76; 318–328.
2. O'Dwyer,T, Maguire,S, Mockler,D, Durcan,L, Wilson,F. (2019). Behaviour change interventions targeting physical activity in adults with fibromyalgia: a systematic review. In Rheumatology International. Springer Verlag, 39; 805-817

022 - BEYOND WHAT IS FREQUENT: PREGNANCY OUTCOMES IN RARE CONNECTIVE TISSUE DISEASES

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Background: Mixed connective tissue disease (MCTD), systemic sclerosis (SS) and inflammatory idiopathic myopathies (IIM) are uncommon conditions that can occur in women of childbearing age. Pregnancies in these patients seem to be associated with an increased risk of adverse pregnancy outcomes (APOs), however, data remain scarce.
Objectives: To describe maternal and perinatal outcomes in women with rare connective tissue diseases (CTD) and to identify potential risk factors for APOs.
Methods: Retrospective observational study including pregnant women with CTD followed at our rheumatology-obstetric clinic from 2009 to 2023. Variables were compared using parametric or non-parametric tests, as applicable.
Results: We identified 31 pregnancies in 23 women: 13 with MCTD, 6 with IIM and 12 with SS. Table 1

TO 022 – TABLE 1. Maternal and Perinatal Outcomes in Women with Rare Connective Tissue Diseases

	Mixed Connective Tissue Disease	Inflammatory Idiopathic Myopathy	Systemic Sclerosis	Total
Pregnancies, n (%)	13/31 (41.9)	6/31 (19.4)	12/31 (38.7)	31 (100)
Miscarriages, n (%)	2/13 (15.4)	2/6 (33.3)	2/12 (16.7)	6/31 (1.4)
Stillbirths, n (%)	1/13 (7.7)	1/6 (16.7)	0/12 (0)	2/31 (6.5)
Gestational age delivery (weeks), median (IQR)	36.9 [2.3]	37.9 [*]	38.9 [2.4]	37.7 [2.1]
Birth weight (grams), median (IQR)	2517.5 [398]	2920 [*]	2870 [740]	2620 [540]
Small for gestational age, n (%)	3/10 (30)	0	0	3/23 (13)
Fetal growth restriction, n (%)	2/8 (25)	0	1/10 (10)	3/20 (15)
Preterm births, n (%)	5/10 (50)	0/3 (0)	1/10 (10)	6/23 (26.1)
Pregnancy flares, n (%)	4/11 (36.4)	1/6 (16.7)	3/12 (25)	8/29 (27.6)
Postpartum flares, n (%)	3/11 (27.3)	1/2 (50)	2/10 (20)	6/23 (26.1)

Legend: IQR: Interquartil Range. *Not applicable.

summarizes clinical data. The mean age at conception was 33.4±5.1 years, with a median disease duration of 8.3 years (IQR 29–38). Most patients n=19/29 (66%) had their disease stable at the time of conception. Major-organ involvement was present in 5 (22%) patients, all of whom exhibited interstitial pulmonary disease.

We documented 23 live births, 6 early miscarriages and 2 stillbirths [one at 37 weeks of gestation (WG) and one at 23 WG]. No congenital abnormalities were found. There was one neonatal infection reported in a baby of a mother with MCTD born at 32 WG. The median gestational age at delivery was 37.7 (2.1) WG. Six (26%) viable pregnancies resulted in preterm births (only one before 34 WG), 5 in women diagnosed with MCTD and 1 with SS. Fetal growth restriction was detected in 3 (15%) pregnancies - 2 in one woman with MCDT with active disease at conception on both occasions; and 1 in a mother with SS. Tree (13%) newborns were small for gestational age (SGA), all from mothers with MCDT. Cesarean was performed in 9 (38%) patients, mostly for obstetric reasons. All patients were ANA positive. SSA/B antibodies were detected in 10 (44%) women. No cases of neonatal lupus were recorded. Antiphospholipid antibodies were negative in all women. Regarding disease activity 8 (28%) patients relapsed during pregnancy (4 MCTD, 1 IIM and 3 SS) - 50% exhibited pulmonary involvement, 38% had muscular involvement and all of them had vasculopathy. In the postpartum period, 6 (26%) women had flares: 3 MCDT, 1 IIM and 2 SS. Concerning treatment, 19 (61%) patients received glucocorticoids (GC) during pregnancy, most of them (63%) at low doses (<7.5mg/day of prednisolone or equivalent). Twenty-two (71%) pregnancies were managed with cDMARDs: hydroxychloroquine (20), azathioprine (5), cyclosporine (2), tacrolimus (1) and immunoglobulin (1). Only 1 patient with IIM received a bDMARD (Rituximab) in the first trimester. Disease activity before conception increased the risk for APOs (p=0.020). No association was found concerning maternal age, use of DMARDs/GC or flares during pregnancy, and the risk of APOs (p>0.05).

Conclusion: Pregnant women with rare CTD, including those with multiorgan involvement, can achieve successful pregnancies. However, they are more likely to experience APOs, particularly if disease activity is present before conception. Multidisciplinary management is of the most importance.

023 - OLDER PATIENTS WITH ANTIPHOSPHOLIPID SYNDROME PRESENTING WITH ARTERIAL THROMBOSIS ARE AT RISK TO DEVELOP SMALL VESSEL INVOLVEMENT

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Introduction: Antiphospholipid syndrome (APS) is a systemic disease characterized by thrombotic manifestations and/or pregnancy morbidity in patients with persistent antiphospholipid antibodies (aPL). Besides macrovascular venous and/or arterial thrombo-

TO023 – TABLE 1. Demographics, clinical and laboratory features of APS patients with and without small vessel involvement

	Overall population	No SV involvement	SV involvement	P value
Number of patients, n (%)	85 (100)	73 (85.9)	12 (14.1)	-
Demographics				
Sex (female), n (%)	69 (81.2)	61 (83.6)	8 (66.7)	0.165
Age of symptom onset, mean (SD)	35.4 (13.9)	33.8 (13.5)	44.0 (12.9)	0.018
Caucasian, n (%)	80 (94.1)	68 (93.2)	12 (100)	0.832
Deaths, n (%)	2 (2.4)	1 (1.4)	1 (8.3)	0.140
Classification				
Primary APS, n (%)	54 (63.5)	44 (60.3)	10 (83.3)	0.145
APS associated with other rheumatic diseases, n (%)	31 (36.5)	29 (39.7)	2 (16.7)	0.145
Systemic lupus erythematosus, n (%)	21 (24.7)	19 (26.0)	2 (16.7)	0.486
Antiphospholipid antibodies, n (%)				
LA	62/81 (76.5)	54 (79.4)	8 (66.7)	0.330
aCL	61/80 (76.3)	53 (79.1)	8 (66.7)	0.344
IgG	47/80 (58.8)	42 (62.7)	5 (41.7)	0.172
IgM	27/80 (33.8)	23 (34.3)	4 (33.3)	0.947
aB2GP	50/80 (62.5)	40 (59.7)	10 (83.3)	0.118
IgG	39/80 (48.8)	34 (50.7)	5 (41.7)	0.562
IgM	22/80 (27.5)	16 (23.9)	6 (50)	0.063
Triple positivity	32/79 (40.5)	26 (38.8)	6 (54.5)	0.822
Cardiovascular risk factors*, n (%)				
Hypertension	31/79 (39.7)	24 (32.8)	7 (63.6)	0.092
Obesity	24/79 (30.4)	21 (28.8)	3 (30.0)	0.947
Diabetes	6/77 (7.9)	4 (6.0)	2 (18.2)	0.159
Dyslipidemia	32/77 (41.6)	25 (34.2)	7 (63.6)	0.113
Smoking	35/74 (47.3)	32 (49.2)	3 (30.0)	0.246
APS presenting manifestation				
Arterial event	30 (35.3)	20 (27.4)	10 (83.3)	<0.001
Venous event	46 (55.4)	44 (61.1)	2 (16.7)	0.004
Obstetric manifestations **	23/62 (37.1)	21/56 (37.5)	2/8 (25)	0.644
APS ever treatment, n (%)				
Anticoagulation	72/81 (88.9)	62 (88.6)	10 (83.3)	0.608
Vitamin K antagonist	59/80 (72.8)	49 (71.0)	10 (83.3)	0.376
DOAC	11/80 (13.9)	11 (16.4)	0 (0)	0.13
Low dose aspirin	53/80 (65.8)	46 (63.0)	7 (63.6)	0.869
Hydroxychloroquine	40/80 (50)	36 (52.2)	4 (33.3)	0.228
Statin	31/80 (38.8)	23 (33.8)	8 (66.7)	0.031

Legend: ab2GPI: APS: Antiphospholipid syndrome; LA: Lupus anticoagulant; anti-β2-glycoprotein I; aCL: anticardiolipin; DOAC: Direct oral anticoagulants; SD: Standard Deviation; SV: small-vessel. *Cardiovascular risk factors were retrieved as stated in the medical notes. ** Percentages according to total number of females

sis, small vessel (SV) involvement may also be present due to the formation of clots in capillaries, arterioles and venules, potentially affecting any organ or system. Globally, SV can be targeted in up to 12% of APS patients (1). We aim to describe the manifestations related to SV involvement in a cohort of patients with APS followed at a Rheumatology tertiary center.

Methods: We included all patients with APS followed at a Rheumatology tertiary center from January 1996 until March 2024. Only patients meeting the Sydney classification criteria were included. Descriptive statistic was used to characterize the population, and the appropriate statistical tests to compare groups. Significance was set as an alpha<0.05.

Results: We included 85 patients with APS, 12 (14.1%) of whom with manifestations related to SV involvement (Table 1). Retinal involvement was found in 5 patients (5.9%): 2 retinal artery thrombosis, 1 retinal vein thrombosis and 2 cases of non-granulomatous ischemic optic neuropathy (ION). Central nervous system (CNS) microvascular lesions, aPL-valvular thrombus and livedoid vasculopathy were found in 5 (5.9%), 3 (3.5%) and 1 (1.2%) patients, respectively. No APS nephropathy or pulmonary microhemorrhages were reported in this cohort. SV involvement represented the presenting (and, to date, only) manifestation of APS in 1 patient - incidental findings of microvascular lesions on brain magnetic resonance imaging (MRI) performed due to suspicion of prolactinoma. One patient with SV disease died from non-APS related causes at 66 years old.

Patients with SV involvement were older at disease onset (44.0±12.9 vs 33.8±13.5 years, p=0.018) and had more often a history of arterial thrombosis as the presenting manifestation (83.3% vs 26.4%, p<0.001). No other clinical or immunological differences were noted, including sex, cardiovascular comorbidity, aPL profile or obstetric morbidity. Most patients with SV involvement (10/12, 83%) were treated with vitamin K antagonists (VKA). The remaining 2 were on low-dose aspirin (LDA): one due to asymptomatic microvascular CNS disease; the other one had ION, but was non-compliant to anticoagulation due to a psychiatric disorder. Patients with SV involvement used statins more frequently than the group without this involvement (66.7% vs 33.8%, p=0.031).

Conclusions: Around one in seven patients in our APS cohort had SV occlusions, which warrant prompt diagnosis and treatment. Our data suggest that particular attention should be given to these manifestations in older patients and those experiencing arterial thrombosis as the presenting manifestation. Larger and prospective studies are needed to confirm these findings.

REFERENCES:

1. Gaspar P, Sciascia S, Tektonidou MG. Epidemiology of antiphospholipid syndrome: macro- and microvascular manifestations. Rheumatology (Oxford). 2024 Feb 6;63(SI):SI24-SI36. doi: 10.1093/rheumatology/kead571. PMID: 38320589; PMCID: PMC10846913.

031 - THE ROLE OF ANTIPHOSPHOLIPID ANTIBODIES IN SPONDYLOARTHRITIS- PROSPECTIVE OBSERVATIONAL STUDY

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Introduction: Spondyloarthritis (SpA) includes a group of inflammatory rheumatic diseases were immune dysregulation plays a central role, although pathogenesis is not fully understood. Recent studies have highlighted the presence and potential role of antiphospholipid antibodies (aPL) in SpA, adding complexity to the disease's immunological management.

Aim: To identify the presence of aPL in patients with SpA and its relationship with different clinical-epidemiological data.

Methods: A prospective observational study between September 2023 and February 2024 involved adult patients diagnosed with axial SpA followed in the rheumatology department at ULS Aveiro. Anticardiolipin and anti-ß2 glycoprotein antibodies and Lupus anticoagulant (LA) were assessed and repeated 12 weeks after, in case of positivity. Clinical data, including thrombotic and obstetric events, were collected. Disease activity was measured based on ASDAS PCR, and BASDAI.

Results: A total of 141 patients were screened, 9 were excluded due to coagulation therapy and active neoplasia. The majority were women (56.1%), with a mean age of 52.2 years (+/-1.22), a mean disease duration 10.8 years (+/-3.5), 62.9% were HLAB27 positive, 69.7% were classified as radiographic SpA and 30.3% as non-radiographic SpA, 26.5% presented peripheral involvement. The most frequent cardiovascular risk factors were dyslipidemia (42.4%), followed by hypertension (33.3%), and obesity (15.9%). 40.2% were under biotechnological therapy, being anti-TNF the most common (94.3%). More than one-third had ASDAS CRP > 2.1 and BASDAI > 4. Positive aPL were found in 16.7% of the sample, of which 40.9% (6.8% of total patients) were confirmed. LA was positive in 59%, followed by IgM anti-ß2 glycoprotein in 40.9% and anticardiolipin IgM in 31.8%. None of the patients presented thromboembolic or obstetric events. Radiographic SpA (p=0.048), obesity (p=0.045), and higher

CPR levels (p=0.000) showed a statistically significant tendency to the presence of aPL.

Conclusions: This study identified the presence of aPL in 16.7% of SpA patients, particularly those with radiographic SpA, obesity, and elevated CRP levels. Despite the detection of aPL, there were no associated thromboembolic or obstetric events. While aPL is present in some SpA patients, their clinical implications with thrombotic risk require further investigation.

033 - COMPARATIVE ANALYSIS OF BIOLOGIC THERAPY SURVIVAL IN SPONDYLOARTHRITIS AND PSORIATIC ARTHRITIS: A KAPLAN-MEIER APPROACH

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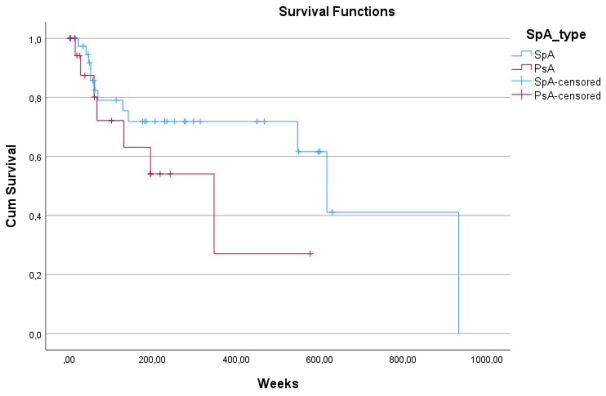
Introduction: Both Spondyloarthritis (SpA) and Psoriatic Arthritis (PsA) are classified under spondyloarthritides, a group of disorders characterized by inflammation of the spine and joints, although they exhibit distinct clinical features. The advent of biologic therapies has significantly impacted the treatment landscape for these diseases. However, not all patients respond equally and some may even experience treatment failure. We aimed to investigate the differences in treatment persistence between patients with SpA and PsA.

Methods: This is a retrospective, observational study including adult patients followed in our department with axial or peripheral SpA and PsA, all of whom were treated with their first biologic or targeted synthetic



TO 033 – Figure 1. Kaplan-Meier survival function.

TO 033 – TABLE 1. Population characteristics

	All (n=62)	SpA (n=42)	PsA (n=20)	p-value
Gender, F (%) / M (%)	34 (54.8) / 28 (45.2)	23 (54.8) / 19 (45.2)	11 (55) / 9 (45)	p = 0.986
Age at diagnosis, years, mean±SD	44.6±12.5	43.36 ± 2.2	47.1 ± 2.0	p = 0.296
Follow up time since bDMARD prescription, weeks, mean±SD	243 ± 246	285 ± 269	154 ± 161	p = 0.050
Involvement type, N (%)				
Axial	49 (79.0)	40 (95.2)	9 (45.0)	p < 0.001
Peripheral	17 (27.4)	2 (4.8)	15 (75.0)	
Manifestations, N (%)				
Arthritis	22 (35.5)	9 (21.4)	13 (65.0)	p < 0.001
Psoriasis	18 (29.0)	0 (0.0)	18 (90.0)	p < 0.001
Enthesitis	11 (17.7)	8 (19.0)	3 (15.0)	p = 0.697
Dactylitis	6 (9.7)	1 (2.4)	5 (25.0)	p = 0.005
Uveitis	3 (4.8)	3 (7.0)	0 (0.0)	p = 0.220
Nail distrophy	1 (1.6)	0 (0.0)	1 (5.0)	p = 0.144
HLA-B27, N (%)	21 (33.9)	20 (62.5)	1 (11.1)	p = 0.006
Sacroileitis on magnetic resonance imaging, N (%)	44 (71.0)	37 (94.9)	7 (46.7)	p < 0.001
First prescribed bDMARD, N (%)				
Adalimumab				
Golimumab	30 (48.4)	20 (47.6)	10 (50.0)	
Secukinumab	10 (16.1)	8 (19.0)	2 (10.0)	
Etanercept	7 (11.3)	4 (9.5)	3 (15.0)	
Infliximab	5 (8.1)	5 (11.9)	0 (0.0)	
Tofacitinib	4 (6.5)	3 (7.1)	1 (5.0)	
Certolizumab	3 (4.8)	1 (2.4)	2 (10.0)	
Risankizumab	2 (3.2)	1 (2.4)	1 (5.0)	
	1 (1.6)	0 (0.0)	1 (5.0)	
Methotrexate at biologic initiation, N (%)	18 (29.0)	7 (16.7)	11 (55.0)	p = 0.002

F – female; M – male; SD – standard deviation; Bdmard – biological/targeted syntethic Disease-Modifying Antirheumatic Drugs;

Disease-modifying antirheumatic drug in our center. Socio-demographic, disease and treatment-related data were collected. Primary endpoint was overall survival (OS), defined as the time from treatment initiation to treatment failure from any cause. Kaplan-Meier survival analysis was performed using SPSS®. Survival curves were compared using the log-rank test. Chi-squared and T-tests were also performed.
Results: 62 patients were analyzed. Table 1 summarizes population characteristics and existing differences between SpA and PsA.
19 patients failed first-line biologic treatment: 6 had primary failure and 13 had secondary failure, with inflammatory back pain being the most common reason for failure in the latter group (11 patients).
Figure 1 shows the results of the Kaplan-Meier survival function.
The mean OS time for biological treatment was 524 weeks (95% CI, 376 to 673), and it was greater in SpA (mean OS of 586 weeks, (95% CI, 417 to 754) than in PsA (mean OS of 292 weeks, (95% CI, 157 to 426).
A log rank test indicated no statistically significant differences between the two groups ($\chi 2(2) = 1.898$, p=0.168).
Conclusions: The survival curve suggests a trend towards better biological persistence for the SpA group, though this difference is not statistically significant. However, patients with PsA had a significantly inferior follow-up time compared to SpA patients, suggesting that PsA patients fail sooner than the SpA patients. they tend Further research with larger sample sizes is needed to better estimate biological persistence outcomes in spondyloarthritides.

045 - ANXIETY DISORDERS IN SPONDYLOARTHRITIS PATIENTS ON BIOLOGIC THERAPY REGISTERED IN REUMA.PT: PREVALENCE, ROLE OF DISEASE-RELATED FACTORS AND INFLUENCE OF BIOLOGIC THERAPY

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Introduction: Anxiety disorder prevalence is higher in chronic conditions like axial and peripheral Spondyloarthritis (SpA), possibly linked to inflammation. We aimed to determine anxiety disorder prevalence in SpA patients at first biologic prescription (bDMARD) and to assess its impact on anxiety disorder.
Methods: We conducted a multicenter, retrospective, observational study including adult patients registered in Reuma.pt with SpA treated with their first bDMARD. Patients that completed the Hospital Anxiety and Depression Scale (HADS) at baseline (T0), after 3 (T1) and/or 6 months (T2) of treatment were included. Socio-demographic, disease and treatment-related data were collected. Anxiety disorder was considered when subscale anxiety (HADS-A) ≥ 11. Pearson and Spearman correlations, ANOVA and T-tests were used.
Results: Of the 141 patients included, 55.3% were female. Mean age at diagnosis was 37.4 ± 10.5 years. Adalimumab was the most frequently bDMARD prescribed (59.6% of initial prescriptions).
At the moment of first biologic prescription, 34.8% of the patients (n=49) had anxiety symptoms.
At T0, more females had anxiety than males (43.6% versus 23.8%) (p = 0.014).
People with anxiety were older at diagnosis (40.5 ± 10.9 versus 36.0 ± 10.1) (p= 0.034).
Clinical indices of disease activity and patient reported outcomes (PROs) reported in table 1 all correlated with the presence of anxiety disorder (p<0.05).
Patients with anxiety disorder had significantly higher Bath Ankylosing Spondylitis Disease Activity Index (p<0.001), Ankylosing Spondylitis Disease Activity Score – C-reactive protein (p=0.004) and Bath Ankylosing Spondylitis Metrology Index (p=0.007) scores at T0 than those who did not have anxiety disorder.
Mean scores of HADS-A significantly differed between the three time points (F(1.862, 158.264) = 15.321, p<0.001). Post hoc analysis with Bonferroni adjustment revealed that HADS-A significantly decreased from T0 to T1 (1.709 (95% CI, 0.750 to 2.669), p<0.001), from T0

TO 045 - TABLE 1. Correlations between disease activity scores or patient reported outcomes with anxiety disorder at baseline.

	Pearson	Spearman	p-value
Disease activity scores			
ASDAS-CRP	0.245		0.004
BASMI		0.228	0.016
BASDAI		0.411	<0.001
Patient reported outcomes			
PGA	0.255		0.002
BASFI	0.464		<0.001
PAP		0.270	0.001
FACIT-F		-0.507	<0.001
SF36 – PF		-0.348	<0.001
SF36 – RP		-0.254	0.003
SF36 – BP		-0.390	<0.001
SF36 – GH		-0.425	<0.001
SF36 – VT		-0.306	<0.001
SF36 – SF		-0.344	<0.001
SF36 - RE		-0.497	<0.001
SF36 - MH	-0.647		<0.001

ASDAS – PCR - Ankylosing Spondylitis Disease Activity Score – C-reactive protein; BASMI - Bath Ankylosing Spondylitis Metrology Index; BASDAI - Bath Ankylosing Spondylitis Disease Activity Index; PGA - Patient Global Assessment; BASFI - Bath Ankylosing Spondylitis Functional Index; PAP - Patient Assessment of Pain; FACIT-F - Functional Assessment of Chronic Illness Therapy – Fatigue Scale; SF36 - 36-Item Short Form Survey; PF – physical functioning; RP – physical role; BP – bodily pain; GH – general health; VT – vitality; SF – social function; RE – emotional role; MH – mental health.

to T2 (1.733 (95% CI, 0.828 to 2.637), p<0.001), but not from T1 to T2 (0.023 (95% CI, -0.730 to 0.777), p=1).
No significant differences were found in HADS-A at the three time points between patients with axial disease and those with peripheral disease (F(2.000, 52.000) = 3.020, p=0.057) and between patients treated with anti-tumor necrosis factor-alpha and those with an anti-interleukin17 (F(1.862, 156.391) = 0.768, p= 0.457).
Conclusions: Anxiety prevalence in SpA aligns with chronic disease estimated rates (between 18-35.1%), meaning that it should be evaluated in all stages of the disease.
Anxiety disorder improvement with bDMARD therapy suggests a relationship with disease activity and physical function. Inflammation hypothesis in anxiety should be considered and further investigated.

048 - BONE VOLUME VERSUS BONE MINERAL DENSITY: THE IMPACT ON FRACTURES IN PREDIALYSIS CHRONIC KIDNEY DISEASE

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Background: Chronic kidney disease (CKD) patients develop mineral bone disorder and are at increased fracture risk. Bone turnover, mineralization and volume (TMV) classification defines different subtypes of renal osteodystrophy (ROD) and is evaluated through bone biopsies and respective histomorphometric analysis. Whether the subtype of ROD and these parameters contribute to fracture risk is not currently known since bone biopsies are not commonly performed.
Objectives: We aim to evaluate the relationship of ROD subtypes and bone volume (BV) with the occurrence of fractures in predialysis CKD patients.
Material and Methods: Retrospective study with 54 patients followed in a predialysis clinic between 2014-2023. Blood tests including bone biomarkers, bone biopsies and histomorphometric analysis were performed at the beginning of follow-up. Data from dual x-ray absorptiometry (DXA) scan and regarding clinical evident fractures was recorded from medical registries. Radiographies of the thoracic and/or lumbar spine were evaluated to detect asymptomatic vertebral fractures. Two groups (with or without fractures) were compared, and p-values were calculated using Mann-Whitney U test for continuous variables and Chi-square with Fisher exact test for categorical variables.
Results: Median follow-up time was 7,5±3 years. Mean age at time of bone biopsy was 65.4±9.8 years old and most patients were male (79.6%). The majority had CKD stage 4 (53.7%), 20 patients progressed to dialysis and 5 died. DXA scan was performed in 19 patients and 5 had osteoporosis. In the histomorphometric analysis, 40.7% (n=22) patients had normal bone histology, 37% (n=20) low bone turnover (BT) with normal mineralization and 22.3% (n= 12) high BT with normal mineralization. Two patients sustained clinical evident fractures and asymptomatic vertebral fractures were identified in radiographies in 3 (5.9%) patients.
Patients who fractured had higher phosphorus levels (4.1 mg/dL vs 3.5 mg/dL, p=0.047, table 1). None of the patients with osteoporosis diagnosed by DXA scan had a prevalent fracture.
Bone mineral density (BMD), histomorphometric subtypes and circulating bone biomarkers did not correlate with the incidence of fractures.
Patients with low bone volume, defined by bone volume fraction (BV/TV) <18% and assessed with histomorphometry, had higher incidence of any fractures,

TO 048 – TABLE 1. Characteristics of the groups with and without fractures during follow-up.

	Fractures during follow-up n=5 (9.3%)	No fractures during follow-up n=49 (90.7%)	p-value
Biochemistry			
Creatinine, mg/dL	2.2 (1.1)	2.2 (0.5)	0.415
GFR, mL/min/1.73m2	26.5 (15.0)	30.5 (12.0)	0.157
Calcium, mg/dL	4.7 (0.0)	4.8 (0.4)	0.685
Phosphorus, mg/dL	4.1 (0.8)	3.5 (0.95)	**0.047**
25(OH)Vitamin-D, ng/mL	13.5 (3.0)	16 (9.8)	0.221
Alkaline Phosphatase, U/L	75.5 (19.0)	71 (43.0)	0.815
PTH, pg/mL	105.7 (40.6)	79.9 (93.7)	0.728
FGF23, pg/mL	30.9 (10.8)	22.1 (21.1)	0.874
Sclerostin, pmol/L	83.9 (105.0)	58.4 (44.7)	0.986
DKK1, pg/mL	1004.5 (283.9)	738.0 (498.9)	0.366
sRANKL, pg/mL	2.7 (0.3)	2,5 (1.4)	0.231
Osteoprotegerin, pg/mL	1554.4 (408.3)	1404.1 (545.3)	0.385
DXA scan, n=19			
Femoral neck T-Score	-1.05 (0.7)	-1.4 (1.8)	0.530
Femoral neck BMD	0.90 (0.103)	0.89 (0.185)	0.530
Osteoporosis, n (%)	0 (0.0)	5 (9.3)	0.665
Bone Histomorphometric Classification, n (%) Normal Bone Histology Low-turnover bone disease High-turnover bone disease	 3 (60.0) 1 (20.0) 1 (20.0)	 19 (38.8) 19 (38.8) 11 (22.4)	0.833
Bone volume, n (%) Low (<16%) Normal (16-23%) High (>23%)	 2 (40.0) 3 (60.0) 0 (0.0)	 12 (24.5) 24 (49.0) 13 (26.5)	0.579
BV/TV	16.1 (11.9-17.2)	19.0 (16.2-23.6)	**0.052**

Footnote: GFR – glomerular filtration rate; PTH- Parathyroid hormone; FGF-23- Fibroblast growth factor-23; DKK1- Dickkopf-1; sRANKL- Soluble receptor activator of nuclear factor-kappaB ligand; DXA - Dual x-ray absorptiometry; BMD- bone mineral density; BV/TV: bone volume fraction; IQR- interquartile range; PTH- Parathyroid hormone; FGF-23- Fibroblast growth factor-23; DKK1- Dickkopf-1; sRANKL- Soluble receptor activator of nuclear factor-kappaB ligand; BMD- bone mineral density; FRAX - Fracture Risk Assessment; CKD – chronic kidney disease.

although not significant (40% vs 24.5%, p=0.579). BV/TV was lower in the group that sustained fractures during follow-up (16.1 (11.9-17.2) vs 19.0 (16.2-23.6)) and this achieved borderline significance (p=0.052). Femoral neck BMD measures in the patients who had an available DXA scan did not show any correlation with BV/TV (r=0.039, p=0.874).

Conclusion: Different ROD subtypes had no association with the incidence of fractures. When considering BV isolated (but not BMD assessed by DXA), a marginally significant association was found between low BV fraction and the occurrence of fractures. High phosphorus levels also associated with fractures and have been previously described as a possible risk factor for fractures not only in CKD patients but also in general population. Further studies with a larger population are needed to validade these results with statistical power.

049 - DIAGNOSTIC ACCURACY OF FRAX® IN PREDIALYSIS CHRONIC KIDNEY DISEASE PATIENTS

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Background: Fracture Risk Assessment (FRAX®) predicts 10-year fracture risk. Although it is used in chronic kidney disease (CKD) patients, it can underestimate fracture risk and some adjustments may be needed.

Objectives: To evaluate the fracture risk and the incidence of fractures in patients with predialysis CKD and estimate the accuracy of FRAX® in this population.

Methods: Retrospective study that enrolled 54 patients (40-89 years old) followed in a predialysis clinic between 2014-2023. Relevant data and information regarding clinical evident fractures was recorded from medical registries. Radiographies of the thoracic and/or lumbar spine were evaluated to detect asymptomatic vertebral fractures. FRAX® without bone mineral

TO 049 – TABLE 1. Comparison of the accuracy of FRAX® (without BMD) with or without CKD as secondary osteoporosis in predicting fracture risk.

	FRAX® (without BMD) including CKD as secondary osteoporosis	FRAX® (without BMD)
Sensitivity	80% (95% CI 0.372-0.987)	20% (95% CI 0.013-0.628)
Specificity	77.3% (95% CI 0.639-0.879)	95 % (95% CI 0.866-0.992)
Positive Predictive Value	28.6% (95% CI 0.099-0.545)	33.3% (95% CI 0.023-0.839)
Negative Predictive Value	97.1% (95% CI 0.880-0.998)	91.3% (95% CI 0.809-0.972)
AUC for major fracture risk	0.882 (95% CI 0.777-0.987)	0.886 (95% CI 0.781-0.992)
AUC for hip fracture risk	0.820 (95% CI 0.685-0.956)	0.814 (95% CI 0.671-0.956)

Footnote: FRAX- Fracture Risk Assessment; BMD- bone mineral density; CKD – chronic kidney disease; AUC- area under the curve; 95% CI- 95% confidence interval.

density (BMD) was calculated with the web-based tool (portuguese version). To determine the ability of the FRAX® tool to discriminate prevalent fractures, we constructed receiver operating characteristic (ROC) curves for each predictor variable: FRAX® tool without BMD and FRAX® without BMD considering CKD as secondary osteoporosis. We evaluated the area under the ROC curves (AUC) for each model of FRAX®.

Results: Median follow-up time was 7.5±3 years. Mean age at the beginning of follow-up was 65.4±9.8 years old and most patients were male (79.6%). Median glomerular filtration rate was 28.5 ml/min/1.73m2 (22-33), 20 patients (37%) progressed to dialysis and 5 patients (9.3%) died.

Median FRAX® was 3.2% (1.9-5.1) for major fracture risk and 0.9% (0.4-1.9) for hip fracture risk. When including CKD as secondary osteoporosis, median FRAX® was 4.6% (2.7-7.6) for major fracture risk and 1.5% (0.6-3.4) for hip fracture. A total of 14 (25.9%) patients achieved the intervention threshold (FRAX® major fracture risk ≥11% and/or hip fracture risk ≥3%) when including CKD as secondary osteoporosis, but only 3 patients were in the high-risk group with FRAX® without adjustments.

Two patients sustained clinical evident fractures. Radiographies of 51 patients were reviewed and asymptomatic vertebral fractures were identified in 3 (5,9%).

High-fracture risk calculated by FRAX® was more prevalent in the group who sustained fractures, but this difference was only statistically significant when considering CKD as secondary osteoporosis (80% vs 22.7%, p=0.019).

Area under the curve (AUC) for FRAX® for major fracture risk without adjustments was 0.886 (CI 95% 0.781-0.992) and 0.882 (CI 95% 0.777-0.987) with CKD as secondary osteoporosis. For hip fracture risk, AUC for FRAX® without adjustments was 0.814 (95% CI 0.671-0.956) and with CKD as secondary osteoporosis 0.820 (95% CI 0.685-0.956). The sensitivity for the prediction of occurrence of fractures by FRAX® was higher when including CKD as secondary osteoporosis (80% vs 20%) and so was the negative predictive value (97.1% vs 91.3%, table 1).

Conclusion: FRAX® (without BMD), independently of the inclusion of CKD as secondary osteoporosis, showed an overall good diagnostic accuracy for predicting fractures in predialysis CKD. However, considering CKD a cause of secondary osteoporosis improved the sensitivity of FRAX® in this population.

051 - LOCAL GLUCOCORTICOID INJECTIONS AND THE RISK OF SECONDARY ADRENAL INSUFFICIENCY: A SINGLE-CENTER PROSPECTIVE STUDY

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Background: Local glucocorticoid injections (LCI) are commonly used in rheumatic patients. Even though their safety profile is better than systemic glucocorticoids (GC), a small proportion of injected GC can reach systemic circulation and have an impact on the hypothalamic-pituitary-adrenal axis (HPAA). Studies have reported an incidence of secondary adrenal insufficiency (SAI) after LCI between 20-50%. Multiple injections, higher doses, triamcinolone acetonide, inflammatory arthropathies and concomitant treatment with strong cytochrome P450 3A4 inhibitors contribute to a higher risk of SAI.

Objectives: To evaluate the incidence and persistence of SAI in patients submitted to LCI and identify possible predictors for SAI.

TO 051 – TABLE 1. Serum matinal cortisol and ACTH levels of patients at baseline and at different timepoints.

Time	Week 0		Week 2		Week 4		Week 8		Week 12	
Patient	Cortisol	ACTH	Cortisol	ACTH	Cortisol	ACTH	Cortisol	ACTH	Cortisol	ACTH
1	14,3	18,7	14,9	16	12,1	9,9	15,9	17,9	N/A	N/A
2	16	10	15,2	5,7	16,3	11	13,9	6,8	N/A	N/A
3	10,6	13,1	**8,5**	16,2	12,1	11,3	10,8	24,9	**8,7**	11,2
4	12,8	13,5	15,6	11	14,7	11,5	15,2	10,6	19,2	12,6
5	15,7	30,9	14,3	30,6	16,8	18,1	**10,7**	25,8	15,9	23
6	14,6	29,1	16,3	26,2	12,1	18,7	17,6	43,2	12,9	22,5
7	28,3	28,2	26,5	18	16,6	22,8	14,4	15,5	34	55
8	14,4	12,5	15,6	12,5	20,6	5,7	18,5	13	13,1	16,3
9	11,3	24,2	**10,7**	22,5	N/A	N/A	12,6	34	11,9	20,1
10	17,3	21,2	17,4	24	12,7	13,4	17,7	30,6	13,9	18,1
11	15,8	61,8	17,5	49,2	15,5	42,3	15,3	54,7	18,4	55,5
12	20,5	28,3	20	28,4	20,2	27,3	17,6	23,2	18,1	21,6
13	13,6	30,5	17,2	26,8	12	19,6	11,7	22,7	11,3	22
14	16,1	19,6	15,1	22,7	17,4	31,4	21,5	25,6	16,7	44

Footnote: ACTH - adrenocorticotropic hormone

Methods: Prospective single-center study. Patients were recruited sequentially from the rheumatology department between october 2023 and february 2024 if they had an indication for an intra-articular or peri-articular GC injection. Exclusion criteria included having had a LCI or systemic, topical, nasal spray, eye drops and inhalation of steroid compounds in the previous 3 months or during the follow-up. After informed consent, patients were submitted to an ultrasound guided LCI and measurements of morning serum adrenocorticotropic hormone (ACTH) and cortisol were obtained prior to the injection (week 0) and at weeks 2, 4, 8 and 12. Cortisol levels of <11.2µg/dL were considered for risk and <5.5µg/dL for high risk of SAI.

Results: Fourteen patients were included, two were lost to follow-up at week 12, and one patient missed the evaluation at week 4. Mean age was 59.43±9.37 years and 71.4% were female. Most patients had axial spondyloarthritis (n=4) and rheumatoid arthritis (n=3). Seven LCI were peri-articular (50%), 5 intra-articular (35.7%) and 2 (14.3%) patients were submitted to shoulder hydrodistension. Methylprednisolone acetate (MPA) in the median dosage of 40mg (20-40) was the GC used in 11 (78.6%) and triamcinolone hexacetonide (20mg) in 3 (21.4%) patients.

All participants had normal serum morning cortisol at baseline. Three patients (21.4%) had a morning cortisol level <11.2µg/dL at some point during follow-up, but none had levels <5.5µg/dL (table 1). A 63-year-old man with radiographic spondyloarthritis submitted to a LCI of the subacromial-subdeltoid bursa with 40mg of MPA had a cortisol of 8.5µg/dL at week 2, with normalization between weeks 4 to 8 and recurrence at week 12 (8.7µg/dL). An additional measurement was performed at week 20, and morning cortisol was 10.8µg/dL. During follow-up he had no symptoms suggestive of SAI. Two other patients had isolated values of cortisol <11.2µg/dL, with subsequent normalization (patient 5: 10.7µg/dL at week 8; patient 9: 10.7µg/dL at week 2). There were no significant differences between cortisol levels at baseline and at weeks 2,4,8 and 12 (p= 0.753, p=0.925, p=0.925, p=0.433, respectively).

Conclusion: To the best of our knowledge, this is the first study to report the incidence and persistence of SAI after LCI evaluated until week 12. Median cortisol levels were similar throughout the defined timepoints. The patient that was at risk of SAI showed low morning cortisol levels at week 2 with recrudescence at week 12, suggesting that there is a continuous and variable absorption of the GC from the injected joint over time. The type and doses of GC used, and the precision provided from the use of ultrasound to guide LCI may have contributed to the low incidence of SAI. Due to the low number of participants, we could not find significant predictors for SAI.

054 - CLINICAL AND IMMUNOLOGICAL FEATURES OF PRIMARY SJÖGREN'S SYNDROME WITH ARTICULAR INVOLVEMENT: RESULTS FROM A PORTUGUESE TERTIARY CENTER

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Background: Primary Sjögren's syndrome (pSS) is a complex and heterogeneous disorder characterized by a wide spectrum of glandular and extraglandular features.(1) Articular involvement (AI) is frequent and usually mild, but such manifestations have been associated with pluri-systemic involvement in pSS.(2) Data regarding AI in pSS patients, including its prevalence and associated features, is scarce.
Objectives: To characterize articular involvement in patients with pSS and identify associated features.
Methods: Cross-sectional study in a tertiary sin-

TO 054 – TABLE 1. Frequency of pSS-associated clinical manifestations, comorbidities, and serological characteristics in patients with and without articular involvement

	Whole Cohort n = 155 (100%)	With Articular Involvement n = 55 (35.5%)	Without Articular Involvement n = 100 (64.5%)	p-value
Age at time of study median (IQR)	61.0 (20.0)	57.0 (22.0)	64.0 (18.0)	0.494
Age at pSS diagnosis median (IQR)	53.3 (22.5)	49.0 (24.0)	55.2 (20.3)	0.084
Sex (n [%])				
- Female	145 (93.5)	54 (98.2)	91 (91.0)	0.098
- Male	10 (6.5)	1 (1.8)	9 (9.0)	
Clinical features [n (%)]				
- Xerophthalmia	152 (98.1)	53 (96.4)	99 (99.0)	0.254
- Xerostomia	145 (93.5)	49 (89.1)	96 (96.0)	0.094
- Parotitis	22 (14.2)	7 (12.7)	15 (15.0)	0.698
- Parotid gland enlargement	16 (10.3)	4 (7.3)	12 (12.0)	0.355
- Lymphadenopathies	10 (6.5)	3 (5.5)	7 (7.0)	0.708
- Raynaud's phenomenon	29 (18.7)	15 (27.3)	14 (14.0)	0.043
- Neurological involvement	5 (3.2)	1 (1.8)	4 (4.0)	0.455
- Renal involvement	6 (3.9)	3 (5.5)	3 (3.0)	0.574
- Cutaneous involvement	28 (18.1)	13 (23.6)	15 (15.0)	0.181
- Pulmonary involvement	15 (9.7)	9 (16.4)	6 (6.0)	0.037
Previous or current treatments [n (%)]				
- NSAIDs	27 (17.4)	15 (27.3)	12 (12.0)	0.018
- Immunosuppression	70 (45.2)	33 (60.0)	37 (37.0)	0.006
- Hydroxychloroquine	58 (37.4)	27 (49.1)	31 (31.0)	0.026
- Methotrexate	3 (1.9)	2 (3.6)	1 (1.0)	0.254
- Leflunomide	1 (0.6)	1 (1.8)	0 (0.0)	-
- Azathioprine	8 (5.2)	3 (5.5)	5 (5.0)	0.903
- Mycophenolate mofetil	3 (1.9)	1 (1.8)	2 (2.0)	0.937
- Prednisolone	20 (12.9)	12 (21.8)	8 (8.0)	0.014
* Prednisolone dose (median (IQR))	0.9 (4.4)	1.2 (2.8)	0.7 (5.1)	0.010
Other common comorbidities and clinical manifestations [n (%)]				
- Lymphoma	9 (5.8)	4 (7.3)	5 (5.0)	0.563
- Fibromyalgia	44 (28.4)	17 (30.9)	27 (27.0)	0.606
- Chronic fatigue	53 (34.2)	20 (36.4)	33 (33.0)	0.704
- Depression	58 (37.4)	18 (32.7)	40 (40.0)	0.371
- Osteoporosis	25 (16.1)	7 (12.7)	18 (18.0)	0.393
Laboratory features [n (%)]				
- Leukopenia (<3.5x10^{9}L)	58 (37.4)	22 (40.0)	36 (36.0)	0.622
- Lymphopenia (<1.5x10^{9}L)	50 (32.3)	18 (32.7)	32 (32.0)	0.926
- Thrombocytopenia (<150x10^{9}L)	19 (12.2)	6 (10.9)	13 (13.0)	0.717
- Hypergammaglobulinemia	95 (61.3)	32 (58.2)	63 (63.0)	0.790
- Hypocomplementemia	37 (23.9)	13 (23.6)	24 (24.0)	0.911
- Antinuclear antibodies	154 (99.4)	54 (98.2)	100 (100.0)	0.176
- Anti-Ro/SSA positive	141 (91.0)	49 (89.1)	92 (92.0)	0.545
- Anti-La/SSB positive	74 (47.7)	27 (49.1)	47 (47.0)	0.803
- Rheumatoid factor	77 (49.7)	33 (60.0)	44 (44.0)	0.064
ESSDAI median (IQR)	0.9 (2.8)	0.8 (2.5)	0.9 (3.0)	0.614
Inpatient admission (≥1)	31 (20.0)	19 (34.5)	12 (12.0)	0.007

IQR: Interquartile range.

gle-center including pSS patients fulfilling the ACR/EULAR 2016 and/or AECG 2002 classification criteria. Demographic features, clinical manifestations, comorbidities, treatment, and immunological characteristics were collected and compared between the groups. AI, defined as inflammatory arthralgias or arthritis attributable to the disease(3), was ascertained by clinical assessment and/or articular ultrasound during the follow-up. Articular activity at the last visit was assessed through Disease Activity Score-28 (DAS28-CRP). Sjögren's syndrome disease activity index (ESSDAI) score was used to evaluate disease activity. The continuous variables were described as means or medians, according to distribution. Categorical variables were expressed in percentages. The Chi-squared test/Fisher test and Mann–Whitney U-test were used to compare categorical and continuous variables, respectively. A p-value <0.05 was considered statistically significant.
Results: 155 pSS patients were included [female: 93.5%, median age 61.0 (IQR 20.0) years]. The median ESSDAI score was 0.9 (IQR 2.8). AI was registered in 35.5% (n=55) of patients, with a median disease manifestation time of 8.0 (IQR 13.0) years. Of these, 85.5% (n=47) presented inflammatory arthralgias, and 40.0% (n=22) had arthritis at any point during the disease. Hands and wrists were the most frequently involved joints, affecting 83.6% (n=46) of the patients with AI. Most patients presented a symmetrical polyarticular pattern (63.6%). No patients presented erosions. The median DAS28-CRP score at the last visit was 0.2 (IQR 0.9). pSS-AI group showed a higher frequency of some clinical features, such as Raynaud's phenomenon (27.3% vs 14.0%, p=0.043) and pulmonary involvement (16.4% vs 6.0%, p=0.037). Significant differences were found regarding treatment, with the pSS-AI group demonstrating a higher use of NSAIDs (27.3 vs 12.0%, p=0.018) and immunosuppressive drugs (60.0% vs 37.0%, p=0.006), including hydroxychloroquine (49.1% vs 31.0%, p=0.026) and prednisolone (21.8% vs 8.0%, p=0.014). Furthermore, these patients also had a higher frequency of hospitalization (34.5% vs 12.0%, p=0.007). No other statistically significant differences were found between the groups concerning demographic, glandular and immunological characteristics (Table 1).
Conclusion: AI was reported in 35.5% of our 155 patients with pSS, with 40.0% of them presenting with arthritis. These patients showed a higher frequency of Raynaud's phenomenon and pulmonary involvement. Additionally, they had increased use of NSAIDs and immunosuppressive agents, as well as higher rates of hospitalization. This subgroup may require more frequent monitoring, early appropriate treatment and pulmonary involvement screening.

056 - O IMPACTO DO BLOQUEIO NERVOSO SUPRAESCAPULAR NA OMALGIA CRÓNICA: EXPERIÊNCIA DE UM CENTRO

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Introdução: A omalgia crónica (≥3 meses de evolução) é uma condição frequente com um impacto funcional, social e económico significativos. Pode originar-se em diferentes estruturas anatómicas do ombro e as opções terapêuticas podem incluir tratamento farmacológico, infiltrações locais ou tratamento fisiátrico. O nervo supraescapular enerva aproximadamente 70% das fibras sensoriais do ombro e o bloqueio nervoso supraescapular (BNS) pode ser um tratamento alternativo na omalgia crónica refratária a tratamentos convencionais.
Objetivos: Implementar o BNS ecoguiado e avaliar o seu benefício clínico e funcional na omalgia crónica ao longo de 12 semanas.
Métodos: Estudo piloto observacional prospetivo entre junho de 2022 e maio de 2024. Doentes adultos com omalgia crónica foram submetidos a BNS ecoguiado com ropivacaína 0.2% (4 mL) e acetato de metilprednisolona (40 mg, 1ml). Foram excluídos doentes que tinham feito tratamento fisiátrico dirigido ao ombro há menos de 6 meses, infiltração dirigida ao ombro nos 3 meses anteriores e com história prévia de cirurgia ao ombro. Foram colhidos dados sociodemográficos e realizada a caracterização da omalgia, exame objetivo (amplitudes de movimento ativas e palm up test) e ecografia musculoesquelética do ombro. Aplicou-se a Escala Numérica de Dor (END) e a versão portuguesa da escala de dor e função do ombro Oxford Shoulder Scale (OSS) no momento inicial e nas semanas 1, 4 e 12 após o BNS. Para avaliar as diferenças na END e OSS entre o início e os diferentes momentos foi utilizado o Wilcoxon Signed-rank test.
Resultados: Um total de 13 doentes foram incluídos, a maioria (53.8%) do sexo masculino. A média de idades foi 66.6±10.8 anos e a maioria dos doentes tinham antecedentes de doença reumática inflamatória (66.7%) incluindo artrite reumatoide (n=3), artrite psoriática (n=2), síndrome de Sjogren (n=2) e lúpus eritematoso sistémico (n=1). No que diz respeito à etiologia da omalgia, 6 doentes (46.2%) apresentavam rotura com-

pleta da coifa dos rotadores. A amplitude global do ombro foi considerada diminuída em 61.5% dos doentes e o teste palm up foi positivo em 9 (69.2%).
Ao longo das 12 semanas após o BNS houve uma redução da intensidade da omalgia em repouso, em movimento e noturna (tabela 1). Esta redução foi estatisticamente significativa às 1 e 4 semanas, no que diz respeito à dor em repouso e dor noturna (p=0.005 e 0.011, respetivamente). Embora se observe este efeito até às 12 semanas, os resultados não foram estatisticamente significativos. Além disso, observou-se uma melhoria da funcionalidade do ombro (avaliada pela escala OSS) significativa na primeira semana (p=0.033), mantendo-se este efeito no primeiro mês, mas com agravamento ulterior até à 12ª semana.
Os doentes foram estratificados relativamente à presença de doença reumática e à presença ou não de rotura tendinosa completa, não existindo diferenças estatisticamente significativas nas escalas END e OSS no momento inicial e no final do seguimento, nestes subgrupos.
Ao longo do período de follow-up não foram reportados eventos adversos.
Conclusão: Este estudo permitiu a implementação do BNS num centro de reumatologia como uma alternativa terapêutica na omalgia crónica e o procedimento demonstrou ser seguro e bem tolerado pelos doentes. O BNS ecoguiado com ropivacaína e metilprednisolona em doentes com omalgia crónica associou-se a uma melhoria na intensidade da dor até às 12 semanas e do impacto funcional no primeiro mês. A pequena amostra incluída limita as conclusões sobre o potencial de benefício deste procedimento.

TO 056 – TABLE 1. Caracterização da intensidade da omalgia (END) e funcionalidade do ombro (OSS) nos diferentes momentos avaliados

Caracterização da omalgia	Momento			
Mediana, (VIQ)	Momento inicial	Semana 1	Semana 4	Semana 12
	N=13	N=13	N=11	N=10
END em repouso	5.0 (2.5-8.5)	1.0 (0-4.0) ***p=0.005***	1.0 (0-5.0) ***p=0.011***	1.0 (0-5.5) *p=0.075*
END em movimento	7.0 (3.5-8.5)	5.0 (3.0-8.0) *p=0.190*	5.0 (3.0-8.0) *p=0.291*	5.5 (1.5-8.3) *p=0.355*
END noturna	8.0 (5.0-9.5)	3.0 (0-7.5) ***p=0.015***	5.0 (3.0-5.0) ***p=0.037***	6.5 (2.3-8.3) *p=0.283*
OSS total	29.0 (20.5-37.0)	34.0 (29.5-42.0) ***p=0.033***	30 .0 (19.0-42.0) *p=0.722*	21.5 (14.0-46.0) *p=0.508*

VIQ: variância interquartil; END: Escala Numérica da Dor; OSS: Oxford Shoulder Scale

058 - RHEUMATIC IMMUNE-RELATED ADVERSE EVENTS ASSOCIATED WITH IMMUNE CHECKPOINT INHIBITORS: A DESCRIPTIVE STUDY REGARDING THE EXPERIENCE OF A RHEUMATOLOGY-ONCOLOGY CONSULTATION

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Introduction: Immune checkpoint inhibitors (ICIs) are antibodies targeting inhibitory molecules on T cells, such as programmed cell death protein 1 (PD-1), programmed cell death ligand 1 (PD-L1) or cytotoxic T-lymphocyte-associated protein (CTLA-4) and in the last decade have improved prognosis and survival in several cancers. As a consequence of the non-specific activation of T-cells, ICIs are also associated with immune-related adverse events (irAEs), including rheumatic irAEs, which can occur in 5-10% patients.
At our institution, since 2021, there is a multidisciplinary referral process between Rheumatology and Oncology to evaluate patients under ICIs with suspected rheumatic irAEs.
Objectives: To describe the experience with rheumatic irAEs secondary to ICI in a Portuguese rheumatology center.
Methods: Descriptive retrospective study. All adult patients with suspected rheumatic irAEs secondary to ICI referred to a specialized appointment of Rheumatology-Oncology between July 2021 and June 2024 were included. Data was collected from the clinical registries.
Results: A total of 12 patients were evaluated, with a median follow-up time of 9 months (3.0-11.8). Mean age was 66.3 (7.8) years, most patients were male (66.7%), and the majority had either lung cancer (n=4) or melanoma (n=3). Pembrolizumab (n=3) was the most used agent, followed by durvalumab (n=2), nivolumab (n=2), avelumab (n=2), a combination regimen of nivolumab and ipilimumab (n=2) and atezolizumab (n=1). Four patients had a previously diagnosed rheumatic disease, namely rheumatoid arthritis (RA), psoriatic arthritis, gout and undifferentiated oligoarthritis. Of the

TO 058 – TABLE 1. Description of the patients with rheumatic irAEs.

Age	Sex	Malignancy	ICI	Previous rheumatic disease	Rheumatic irAEs	Tenosynovitis	Other irAEs	Latency of rheumatic irAEs (days)	US evidence of synovitis	CRP at onset (mg/L)	RF (UI/mL) ACPA (U/mL)	ANAs ENAs	Treatment	Improvement with treatment	ICI held for irAEs
62	M	Lung	Durvalumab	N/A	PMR-like	No	N/A	180	N/A	4.1	Negative	1/1000 Negative	Systemic GC	Yes	No
69	F	Lung	Durvalumab	RA	Symmetric polyarthritis	Yes	N/A	40	Yes	176.1	40.3 82.0	1/1000 Negative	Systemic GC	Yes	Yes
54	M	Melanoma	Nivolumab + Ipilimumab	N/A	Symmetric polyarthritis	Yes	N/A	58	Yes	101.3	Negative	Negative	Systemic GC + Sulfassalazine	Yes	Yes
74	M	Urothelial	Avelumab	N/A	Sicca Syndrome	N/A	Psoriasis	49	N/A	6.2	Negative	Negative	Symptomatic	Yes	Yes (for psoriasis)
74	M	Gastric	Nivolumab	N/A	Symmetric polyarthritis	No	Rash	149	N/A	77.8	Negative	1/640 Anti-CENP B	Systemic GC	Yes	Yes
56	F	Tongue	Nivolumab	N/A	Symmetric polyarthritis	Yes	N/A	119	Yes	6.2	Negative	1/320 Negative	Systemic GC	Yes	No
64	M	Renal	Nivolumab + Ipilimumab	Gout	Monoarthritis	No	N/A	41	N/A	35.6	Negative	1/640 Negative	Intra-articular GC	Yes	No
68	M	Lung	Pembrolizumab	Unspecified oligoarthritis	Asymmetric oligoarthritis	No	Systemic	390	N/A	146.90	Negative	Negative	Systemic GC	Yes	Yes
78	M	Urothelial	Avelumab	N/A	PMR-like	No	Rash	40	N/A	51.5	N/A	N/A	Systemic GC	Yes	No

Footnote: ICI – immune checkpoint inhibitor; irAEs – immune-related adverse events; US – ultrasound; CRP – c-reactive protein; RF – rheumatoid factor; ACPA - anti-citrullinated protein autoantibodies; ANAs – antinuclear antibodies; ENAs - extractable nuclear antigen antibodies; M – male; F – female; N/A: - not applicable; RA – rheumatoid arthritis; GC - glucocorticoids

referred 12 patients, 2 patients had osteoarthritis, 1 patient had a sensitive polyneuropathy secondary to ICIs and 9 (75%) had rheumatic irAEs. Three patients died during follow-up, due to the oncological disease.

When examining the 9 patients with rheumatic irAEs (table 1), 4 patients presented with symmetric polyarthritis, 2 with a polymyalgia rheumatica-like syndrome, 1 with sicca syndrome, 1 with knee monoarthritis and 1 with asymmetric oligoarthritis. Of these, 3 patients had previously diagnosed rheumatic diseases and were considered to have a RA flare, a gout flare, and an exacerbation of a previously diagnosed undifferentiated oligoarthritis, respectively. Alongside the rheumatic irAEs, four patients had additional irAEs: psoriasis, an unspecified rash, pneumonitis and multiple manifestations (anemia, diarrhea, and peripheral edema). The time from start of ICI therapy to onset of rheumatic irAEs was variable, ranging from 40 to 390 days (median 58 days).

ICI was withdrawn due to irAEs in 5 (55.5%) patients. Most patients (n=6, 75%) were treated with systemic glucocorticoids, one patient required additional therapy with sulfasalazine, an intra-articular glucocorticoid injection was performed in another patient and the patient with sicca syndrome was adequately controlled with artificial tears and saliva. Clinical improvement was observed in all patients during follow-up.

Conclusion: Our results are in line with current knowledge and show that rheumatic irAEs that develop in cancer patients under ICIs can occur either as a flare of a previously known rheumatic disease or as a de novo manifestation. Almost all patients showed an adequate response to glucocorticoids, but ICIs had to be held in half. Given the important decisions regarding immunotherapy discontinuation and the starting of immunosuppression, a multidisciplinary approach should be pursued in these patients.

068 - CLINICAL AND IMMUNOLOGICAL FEATURES OF PRIMARY SJÖGREN'S SYNDROME WITH CUTANEOUS MANIFESTATIONS: RESULTS FROM A PORTUGUESE TERTIARY CENTER

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Background: Primary Sjögren syndrome (pSS) is a chronic autoimmune inflammatory disease that mainly affects the exocrine glands and strongly associated with anti-SSA(Ro) and anti-SSB autoantibodies (1). Notably, in patients with lupus, anti-SSA(Ro) is a major risk factor for rashes, namely subacute cutaneous lupus, neonatal lupus and photosensitive rashes. However, in pSS, reported cutaneous manifestations are annular erythema, alopecia, angular cheilitis and eyelid dermatitis (2).

Objectives: To characterize cutaneous manifestations in patients with pSS and identify associated features.

Methods: Cross-sectional study in a tertiary single center including pSS patients fulfilling the ACR/EULAR 2016 and/or AECG 2002 classification criteria. Demographic features, clinical manifestations, comorbidities and immunological characteristics were collected and compared between groups. Cutaneous manifestations were defined based on clinical evaluation by a der-

matologist and/or histological examination. Sjögren's syndrome disease activity index (ESSDAI) score was considered to assess disease activity. The continuous variables were described as means or medians, according to distribution. Categorical variables were expressed in percentages. The Chi-squared test/Fisher test and Mann–Whitney U-test were used to compare categorical and continuous variables. A p-value <0.05 was considered statistically significant.

Results: 152 pSS patients were included [female: 93.4%, median age: 61 (IQR 20.0) years]. The median ESSDAI score was 0.88 (IQR 2.8). Cutaneous manifestations were registered in 17.1% (n =26) of the patients and cutaneous small-vessel vasculitis was the most frequent feature (n =13; 8.6%). Other cutaneous manifestations included annular erythema (n =9; 5.9%), erythema nodosum (n =2; 1.3%), alopecia (n =2; 1.3%), eyelid dermatitis (n =1; 0.7%), angular cheilitis (n =1; 0.7%), and chilblains lupus (n =1; 0.7%). Patients with cutaneous small-vessel vasculitis presented more frequently with Raynaud phenomenon (38.5% vs 17.3%, p=0.041), renal involvement (15.4% vs 2.9%, p=0.020) and cryoglobulinemia (38.5% vs 9.4%, p=0.038) (Table 1). No other statistically significant differences were found between patients with and without cutaneous manifestations concerning demographic, clinical and laboratory features.

Conclusion: In this cohort with 152 patients with pSS, cutaneous manifestations were reported in 17.1%. Cutaneous small-vessel vasculitis was the most frequent (8.6%) and associated with Raynaud's phenomenon, renal involvement, and cryoglobulins.

TO 068 – TABLE 1. Frequency of pSS-associated clinical manifestations, comorbidities, and serological characteristics in patients with and without cutaneous small-vessel vasculitis. IQR: Interquartile range

	Whole Cohort n = 152 (100%)	Cutaneous Vasculitis n = 13 (8.6%)	Without Cutaneous Vasculitis n = 139 (91.4%)	p-value
Age at time of study median (IQR)	61.0 (20.0)	54.5 (23.0)	61.0 (20.3)	0.250
Age at pSS diagnosis median (IQR)	52.3 (22.2)	49.2 (23.2)	52.0 (22.1)	0.319
Sex (n [%])				
- Female	142 (93.4)	13 (100)	129 (92.8)	0.334
- Male	10 (6.6)	0 (0)	10 (7.2)	
Clinical features [n (%)]				
- Xerophthalmia	150 (98.7)	13 (100)	137 (98.6)	1.000
- Xerostomia	142 (93.4)	13 (100)	129 (92.8)	1.000
- Parotitis	22 (14.5)	1 (7.7)	21 (15.1)	0.676
- Parotid gland enlargement	16 (10.5)	1 (7.7)	15 (10.8)	1.000
- Lymphadenopathies	10 (6.6)	1 (7.7)	9 (6.5)	0.184
- Arthritis	22 (14.5)	2 (15.4)	20 (14.4)	0.640
- Raynaud's phenomenon	29 (19.1)	5 (38.5)	24 (17.3)	0.041
- Neurological involvement	5 (3.3)	0 (0)	5 (3.4)	1.000
- Renal involvement	6 (3.9)	2 (15.4)	4 (2.9)	0.020
- Pulmonary involvement	15 (9.9)	1 (7.7)	14 (10.0)	0.470
Other common comorbidities and clinical manifestations [n (%)]				
- Lymphoma	8 (5.3)	0 (0)	8 (5.8)	0.391
- Fibromyalgia	43 (28.3)	4 (30.8)	39 (28.1)	0.770
- Depression	56 (36.8)	8 (61.5)	48 (34.5)	0.104
- Osteoarthritis	43 (28.3)	2 (15.4)	41 (29.5)	0.362
- Osteoporosis	25 (16.4)	1 (7.7)	24 (17.3)	0.419
Laboratory features [n (%)]				
- Leukopenia (<3.5x10^{9}L)	58 (38.2)	6 (46.2)	52 (37.4)	0.538
- Lymphopenia (<1.5x10^{9}L)	51 (33.6)	4 (30.8)	47 (33.8)	0.959
- Thrombocytopenia (<150x10^{9}L)	19 (12.5)	3 (23.1)	16 (11.5)	0.186
- Hypergammaglobulinemia	94 (61.8)	10 (76.9)	84 (60.4)	0.536
- Hypocomplementemia	36 (23.7)	3 (23.1)	33 (23.9)	0.944
- Cryoglobulinemia	18 (11.8)	5 (38.5)	13 (9.4)	0.028
- Antinuclear antibodies	151 (99.3)	13 (100)	138 (99.3)	1.000
- Anti-Ro/SSA positive	138 (90.8)	13 (100)	125 (89.9)	0.605
- Anti-La/SSB positive	73 (48.0)	8 (61.5)	65 (46.8)	0.556
- Rheumatoid factor	77 (50.7)	9 (69.3)	68 (48.9)	0.547
ESSDAI median (IQR)	0.88 (2.8)	0.88 (2.9)	0.58 (1.4)	0.866

REFERENCES:

1. Negrini, S., Emmi, G., Greco, M., Borro, M., Sardanelli, F., Murdaca, G., Indiveri, F., & Puppo, F. (2022). Sjögren's syndrome: a systemic autoimmune disease. Clinical and experimental medicine, 22(1), 9–25. https://doi.org/10.1007/s10238-021-00728-6
2. Jhorar, P., Torre, K., & Lu, J. (2018). Cutaneous features and diagnosis of primary Sjögren syndrome: An update and review. Journal of the American Academy of Dermatology, 79(4), 736–745. https://doi.org/10.1016/j.jaad.2018.05.02

069 - FUNCTIONAL ABILITY, MOBILITY, AND QUALITY OF LIFE OF OLDER ADULTS WITH HIP FRACTURE

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Introduction: Hip fractures are one of the major consequences of osteoporosis in the older population, representing a global public health concern[1]. Although most of these patients were independently living before the fracture, many patients do not regain pre-fracture function. This study aimed to characterize the functional ability, functional mobility, and quality of life of older adults with a hip fracture in a Portuguese Hospital.

Methods: Older adults with stable trochanteric fracture surgically stabilized with cephalomedullary nails (n=8, 79±10y.o./5 females) who were admitted to the ULS S.José CRI-TO and were aged ≥65y.o., without cognitive impairment (Montreal Cognitive Assessment [MoCA]>22) and could walk independently, were recruited. Functional ability was tested at 1 month (pre-fracture function), 3 months (3M), and 6 months (6M) post-surgery (post-op), through the Hip Disability and Osteoarthritis Outcome Score-HOOS. The Timed Up and Go Test (TUG) was performed to measure functional mobility and EQ5D to assess the health-related quality of life (HRQoL), at 3M and 6M post-op. Differences in HOOS dimensions, EQ5D, and TUG were determined between timepoints using t-tests (α=0.05).

Results: The HOOS score at 6M post-op significantly decreased in all its dimensions [pain, symptoms, activities of daily living (ADL), sports/recreational activities (Sport/Rec), and QoL] compared to the pre-fracture period (Fig.1). The biggest difference was found in HOOS-QoL dimension ($p<0.01$). The mean EQ5D at 3M was 0.73 and 0.85 at 6M, increasing significantly from 3M to 6M ($p<0.001$). At 3M post-op, the participants performed a 19s' TUG and a 17s at 6M, meaning no differences between the 2 timepoints post-op ($p=0.58$).

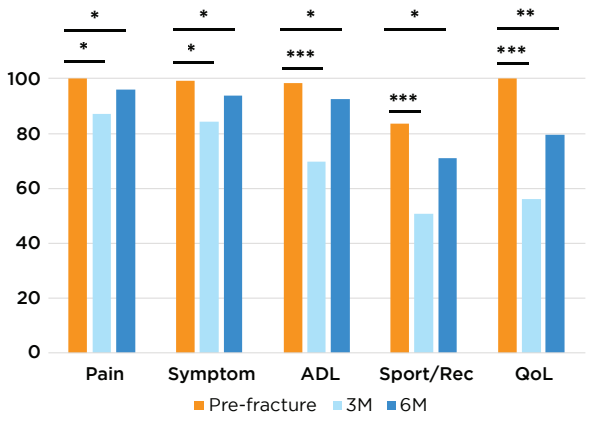
Fig. 1. Functional ability at pre-fracture, 3M, and 6M post-op (*p<0.05;**<0.01;***<0.001).

Discussion: The older hip fracture adults didn't achieve their pre-fracture function level at 6M post-surgery, the period in which much of the recovery has been shown to occur[2]. The HOOS-QoL showed a more significant reduction, but the lowest average score was found in the sports/recreational dimension, representing the most strenuous activities. Although all dimensions had improved during the post-op period, none reached the pre-fracture scores. These results are in line with other studies[3], although the scores were generally higher in our study. Still, it is important to note that we only included patients with stable trochanteric fractures. The HRQoL at 6M post-op also remained below what is considered "perfect health" (=1). While the TUG has not improved significantly from 3M to 6M, it represents better functional mobility when compared to the results in the other study mentioned[3] (TUG=22.5s), although above the value (12s) of which there is considered to be a high risk of falling for older adults.

In conclusion, older hip fracture patients presented lower functional ability than before the fracture and a high risk of falling 6M after surgery, which seems to affect their HRQoL.

REFERENCES:

1. Sing, et al. (2023). Global Epidemiology of Hip Fractures: Secular Trends in Incidence Rate, Post-Fracture Treatment, and All-Cause Mortality. J. Bone Miner. Res. 38.



TO 069 – Figure 1.

2. Fischer et al (2019). Timeline of functional recovery after hip fracture in seniors aged 65 and older . Osteoporos. Int. 30.
3. Makridis et al (2019). Searching for the 'winner' hip fracture patient: the effect of modifiable and non-modifiable factors on clinical outcomes following hip fracture surgery. HIP Int. 31.

070 - UPADACITINIB TREATMENT IN MODERATE TO SEVERE RHEUMATOID ARTHRITIS PATIENTS; REAL WORLD DATA FROM THE PORTUGUESE REGISTRY REUMA.PT – RAPORT STUDY

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Background: Upadacitinib (UPA), an oral JAK inhibitor (JAKi), was evaluated in the SELECT-RA program in patients (pts) with moderate-to-severe Rheumatoid Arthritis (RA), both as monotherapy and in combination with conventional synthetic (cs) Disease Modifying Antirheumatic Drugs (DMARDs). However, real-world (RW) effectiveness and safety of UPA in these patients is still needed.

Objectives: To investigate the RW effectiveness and safety of UPA for the treatment of pts with active RA included in Reuma.pt registry.

Methods: RAPORT is a multicentre, single-country, non-interventional study of RA pts treated with UPA 15mg once a day, included in the Portuguese registry. All procedures followed local routine clinical practice. Patients were followed-up for 6 months and those with available data on disease activity at baseline (UPA initiation) and 6 months were included.

The primary endpoint was the proportion of pts achieving DAS28-CRP (Disease Activity Score-28 C-reactive protein) remission (<2.6) at 6 months. Secondary and exploratory efficacy endpoints included the proportion of pts achieving remission defined as CDAI (Clinical Disease Activity Index) ≤2.8 and SDAI (Simplified Disease Activity Index) ≤3.3; DAS28-CRP Low Disease Activity (LDA) ≤3.2 at 6 months; and change from baseline to 6 months in pt-reported outcomes (PROs). Results are presented using descriptive statistics. Safety was assessed throughout the 6-month observation period by collection of adverse events (AE) data. AEs of special interest (AESIs) were based on the known safety profiles of JAKi, and included serious infections, op-

TO 070 – TABLE 1. Patient demographics and disease characteristics at UPA initiation and after 6 months of follow-up and reasons for discontinuing UPA.

Characteristic	Baseline	6 months
Demographic data		
Age, years, mean (SD)	56.4 (11.49)	
Sex, female, n (%)	116 (86.57)	
≥65 anos n (%)	39 (29.10)	
Comorbidities, n (%)		
Total	80 (59.7)	
Hypertension	19 (14.1)	
Diabetes mellitus	6 (4.5)	
Dyslipidaemia	1 (0.7)	
Osteoporosis	3 (2.2)	
Smoking habits, n (%)		
Unknown	10 (7.5)	
Ex-smoker	10 (7.5)	
Smoker	22 (16.4)	
Non-smoker	59 (44)	
Missing data	40 (29.9)	
RA disease duration, years, mean (SD)	16.91 (10.54)	
RA previous therapy, n (%) patients		
Corticosteroids	86 (64.2)	-
csDMARDs	21 (15.7)	
bDMARDs	103 (76.9)	
tsDMARDs	13 (9.7)	
RA current therapy, n (%) patients		
Corticosteroids	71 (53.0)	66 (49.25)
csDMARDS	89 (66.42)	81 (60.42)
Monotherapy with b/tsDMARDs	35 (33.58)	53 (39.55)
1st bDMARD or tsDMARD treatment, n (%)		-
Anti-TNF	74 (55.22)	
Anti-IL-6	14 (10.48)	
Anti-CD20	4 (2.99)	
tsDMARD	42 (31.34)	
non-UPA tsDMARD	4 (2.99)	
UPA as first line	38 (28.4)	
UPA as second line	41 (30.58)	
UPA as third line	33 (24.63)	
UPA as fourth and more line	18 (13.43)	
Parameters of disease activity		
Disease activity, mean (SD)		
DAS28-CRP	4.19 (1.19)	2.56 (1.22)
CDAI (total n/%)	22.2 (12.72) (103/76.87)	8.82 (7.40) (85/63.43)
SDAI (total n/%)	23.3 (12.99) (100/74.63)	9.14 (7.60) (86/64.18)

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TO 070 – TABLE 1. continuation

Characteristic	Baseline	6 months
Patient Report Outcomes, mean (SD)		
Pain VAS(total n/%)	57.9 (25.5) (109/81.34)	33.01 (23.9) (93/69.4)
PGA (total n/%)	61.96 (25.03) (111/82.84)	34.7 (25.12) (94/70.15)
HAQ- DI (total n/%)	1.17 (0.68) (73/54.48)	0.94 (0.66) (61/45.52)
FACIT (total n/%)	31.37 (10.26) (25/18.66)	34.76 (6.81) (n=25/18.66)
SF-36 – PCS (total n/%)	44.49 (27.20) (30/22.39)	58.87(22.02) (n=33/24.63)
Routine labs and serology		
ESR (mm/hr), mean (SD)	26.37 (23.24)	19.37 (17.73)
CRP (mg/dl), mean (SD)	1.25 (1.87)	0.86 (1.75)
RF, n (%) patients	87 (64.92)	NA
ACPA, n (%) patients	88 (65.67)	NA
Remission and LDA, n (%) patients		
DAS28-CRP≤3.2		59 (44.1)
CDAI<10		52 (38.8)
SDAI<11		52 (38.8)
Discontinuation of UPA and adverse events		
Discontinuation of UPA, n (%) patients		
Total		22 (16.4)
Therapeutic ineffectiveness		6 (4.5)
Adverse events		11 (8.2)
Other / unknown		5 (3.7)
Adverse events (AE), n (%) patients		
Total AE		13 (9.7)
Infections[a)]		4 (2.2)
GI manifestation[b)]		2 (1.5)
Skin manifestation[c)]		2 (1.5)
Malignancy[d)]		1 (0.7)
Hematological AE (cytopenia)[e)]		1 (0.7)
Other/unknown		3 (2.2)
MACE		0
VTE		0
Creatine phosphokinase (CPK) elevation		0

AE. adverse events; cs/b/tsDMARD. conventional synthetic/biologic/targeted synthetic disease-modifying antirheumatic drugs; CDAI. Clinical Disease Activity Index; CRP. C-reactive protein; DAS28-CRP. disease activity score 28 joints – CRP; ESR. erythrocyte sedimentation rate; GI. gastrointestinal; HAQ-DI. Health Assessment Questionnaire - Disability Index; Anti-IL-6. anti-interleukin-6; LDA. low disease activity; MACE. Major cardiovascular event; PGA. patient global assessment; RA. rheumatoid arthritis; RF. rheumatoid factor; SD: standard deviation; SDAI. Simple Disease Activity Index; Anti-TNF. tumour necrosis factor inhibitor; UPA. Upadacitinib; VTE. Venous thromboembolic event. a) Two cases of COVID infections and two urinary tract infections; b) Two cases of raised liver enzymes; c) One case of acne and one of dermatitis; d) One case of testicular neoplasia; e) One case of neutropenia

portunistic infections (excluding herpes zoster and tuberculosis(TB)), herpes zoster, active TB, malignancy, hepatic disorder, major adverse cardiovascular events (MACEs), venous thromboembolism (VTE), and creatine phosphokinase (CPK) elevation. Safety data are reported as numbers of AEs.

Results: Out of 194 pts registered in Reuma.pt with a 6-month follow-up period, 134 pts were included in the analysis (the remaining were excluded due to missing data). Pt demographics and disease characteristics at UPA initiation are summarized in Table 1. 28.4% of patients started UPA as a first-line advanced therapy, while almost 40% have been previously treated with ≥3 DMARDs. 33.6% of patients were on monotherapy with UPA. At 6 months, 44% were on LDA and 39.6% were on remission (DAS28-CRP), regardless of population characteristics, such as disease duration, and failure to previous b/tsDMARDs. Improvements from baseline to 6 months were observed across different PROs, including pain, PGA (Patient Global Assessment), HAQ-DI (Health Assessment Questionnaire Disability Index), FACIT (Functional Assessment of Chronic Illness Therapy) and SF-36-PCS (Short Form 36 physical component summary) (Table 1). A total of 22 pts (16.4%) of the analysed sample discontinued UPA during the follow-up period. The most common reasons for discontinuation were AEs (n=11 [8.2%]) and lack of effectiveness (n=6 [4.5%]). Infections were the most common AE reported (n=4 [2.2%]). No MACEs, VTE events or deaths were reported.

Conclusion: The results of the RAPORT study suggest that UPA 15mg is effective for the treatment of moderate-to-severe RA in RW practice, with more than 40% of pts achieving remission or LDA at 6 months and improvements in PROs such as pain and fatigue. The difference between these results and other RW studies reported from other registries or RW cohorts may be due to a highly experienced population (several prior DMARDs) in this cohort.

071 - BEYOND NEONATAL LUPUS: MATERNAL AND PERINATAL OUTCOMES IN WOMEN WITH ANTI-SSA AND ANTI-SSB ANTIBODIES. EXPERIENCE FROM A MULTIDISCIPLINARY CLINIC

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Background: Anti-SSA and -SSB antibodies are associated with neonatal lupus (NL), a condition that can affect fetuses/newborns of women with these circulating antibodies regardless of the presence of a systemic disease. However, data is inconclusive regarding the association of these antibodies and other obstetric complications.

Objectives: To describe maternal and perinatal outcomes of women with anti-SSA and SSB followed at a high-risk multidisciplinary pregnancy clinic and to address possible risk factors for adverse pregnancy outcomes (APO) in this population.

Methods: Retrospective observational study of patients followed at a tertiary hospital from 01/2009 to 05/2023. Included patients exhibited serum anti-SSA and/or anti-SSB determined by ELISA.

Results: A total of 103 pregnancies in 85 patients were included. There was 1 twin pregnancy. The median age at conception was 35.0 (IQR 31.0-37.0) years. Systemic Lupus Erythematosus (SLE) was the main diagnosis (n=36, 35.0%) followed by primary Sjögren syndrome (SSj) (n=19, 18.4%). Concerning SLE patients, 7 had secondary antiphospholipid syndrome and 7 had secondary SSj. One patient with primary SSj had concomitant primary biliary cholangitis and another one had concomitant mixed cryoglobulinemia. All pregnancies were anti-SSA positive - 69 (67.0%) anti-Ro52 and 86 (83.5%) anti-Ro60; 39 (37.9%) were anti-SSB positive. Table 1 summarizes clinical data. There was 1 stillbirth from a preterm labor, 1 medical termination of pregnancy because of severe malformations, and 11 (10.9%) miscarriages. Preeclampsia (PE) occurred in 4 (4.5%) pregnancies and preterm labor (PTL) in 12 (13.3%), 1 before the 34th week of gestation (WG). Fetal growth restriction (FGR) occurred in 7 (8.0%) cases and 16 (19.8%) newborns were small for gestational age. There were 4 (5.5%) cases of NL, expressed as skin rash (2) thrombocytopenia (1) and first-degree congenital heart block (1). The latter successfully reverted after dexamethasone and IVIG treatment. The occurrence of PTL was associated with hypocomplementemia (p=.042). We found no correlation between

TO 071 – TABLE 1. Maternal and perinatal outcomes in women with anti-Ro/SSA and anti-La/SSB followed at a rheumatology-obstetric clinic.

Main diagnosis	N (%)	Gestational age at delivery (mean± SD; weeks)	BW (mean± SD; grams)	SGA N (%)	Miscarriages N (%)	FGR N (%)	Preterm births N (%)	Preeclampsia N (%)	NL N(%)
SLE	45 (43.7)	35.8 ± 9.0	2954 ± 662	7 (19.4)	6 (13.3)	4 (10.3)	7 (18.4)	2 (4.4)	2 (5.3)
SSj	21 (20.4)	38.7 ± 1.1	3141 ± 357	1 (5.9)	1 (4.8)	0 (0)	0 (0)	0 (0)	0 (0)
UCTD	13 (12.6)	38.0 ± 1.7	2842 ± 630	2 (20.0)	2 (15.4)	1 (10.0)	1 (9.1)	0 (0)	1 (9.1)
MCTD	11 (10.7)	36.3 ± 2.2	2694 ± 596	2 (28.6)	2 (27.3)	2 (33.3)	2 (25)	1 (12.5)	1 (12.5)
RA	5 (4.9)	36.9 ± 3.7	2807 ± 961	1 (25.0)	0 (0)	0 (0)	2 (40)	0 (0)	0 (0)
SSc/IIM	2 (1.9)	37.9 ± 1.2	2578 ± 81	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
No SRD	2 (1.9)	40.0	2950 ± 778	1 (50)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
SSc	1 (1.0)	37.1	2720	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
axSpA	1 (1.0)	40.3	3430	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
AAV	1 (1.0)	40.6	2845	1 (100)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
PAN	1 (1.0)	38.0	2440	1 (100)	0 (0)	0 (0)	0 (0)	1 (100)	0 (0)
Total	103 (100)	37.5 ± 4.6	2913 ± 599	16 (19.8)	11 (10.9)	7 (8.0)	12 (13.3)	4 (4.5)	4 (5.5)

AAV: ANCA associated vasculitis; AxSpA: axial spondyloarthritis; BW: body weight; FGR: fetal growth restriction; IIM: idiopathic inflammatory myopathy; MCTD: mixed connective tissue disease; PAN: polyarteritis nodosa; RA: rheumatoid arthritis; SGA: small for gestational age; SLE: systemic lupus erythematosus; SRD: systemic rheumatic disease; SSc: systemic sclerosis; SSj: Sjogren syndrome; UCTD: undifferentiated connective tissue disease.

anti-SSA/SSB titers and the occurrence of APOs.
Conclusions: Only one case of 1st degree heart block was recorded, reverting after treatment. PTL was associated with hypocomplementemia in our cohort. The majority of pregnant patients managed at our rheumatology-obstetric high-risk clinic had successful gestations. The risk of APO in positive anti-SSA/SSB pregnant women does not seem to be related with antibody titers. Further studies are needed to definitively establish if anti-SSA/SSB positive pregnant women are at higher risk of maternal and perinatal outcomes.

072 - A EULAR/ACR (2019) SLE CLASSIFICATION CRITERIA SCORE ≥ 20 IS A MARKER OF SEVERE DISEASE IN A PREVALENT SLE COHORT FROM THE REUMA.PT REGISTRY: A CROSS-SECTIONAL STUDY OF 709 PATIENTS

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Background: Recently, a EULAR/ACR 2019 systemic lupus erythematosus (SLE) classification criteria (EULAR/ACR 2019) score≥20 was identified in a SLE inception cohort with less than 2 years from diagnosis as a marker for more severe disease, including higher disease activity, more frequent flares, higher use of immunosuppressants, lower probability of achieving

remission, and more damage accrual. However, this analysis has not been performed in SLE patients with longer disease duration.1

Objective: To assess a EULAR/ACR 2019 score ≥20 as a marker for severe disease in a prevalent SLE cohort.

Methods: We performed a cross-sectional multicenter study of patients fulfilling the EULAR/ACR 2019 classification criteria for SLE in the Portuguese registry of rheumatic diseases (Reuma.pt). Disease activity (SLE-DAS) and the EULAR/ACR score were assessed at the last visit (June 2023-March 2024). Groups of patients with EULAR/ACR 2019 score≥20 or <20 were compared for demographic, clinical and treatment features, with parametric and non-parametric tests, as appropriate.Separate models were tested using different definitions of severe SLE (dependent variable), as present/absent: (1) cumulative SLE major organ involvement;(2) moderate/severe disease activity, defined as SLE-DAS>7.64;(3) ongoing immunosuppressants; (4) ongoing systemic prednisolone>7.5 mg/day;(5) organ damage, defined as SLICC/ACR Damage Index (SDI)≥1. Predictors for each of these definitions were assessed in a two-step approach with logistic regression (LR) univariate analysis, followed by multivariate LR models including variables with p<0.10 in the first step, while excluding variables with multicollinearity. Multivariate analysis was used to identify independent predictors and estimate the respective adjusted odds ratios (OR) with 95% confidence intervals.

Results: There were 2459 patients registered in Reuma.pt, from 37 participating centers. A total of 709 patients, from 18 centers, who had data on and fulfilled the EULAR/ACR 2019 classification criteria and had a SLE-DAS scoring were included, 65.6% having an EULAR/ACR 2019 score≥20. These patients were younger at diagnosis (p<0.001), had longer disease duration (p<0.001), higher SLE-DAS score (p=0.001), received less frequently antimalarials (p=0.004) and were more frequently treated with synthetic and/or biologic immunosuppressants (p<0.001) and glucocorticoids (p<0.001). On univariate LR analysis, a EULAR/ACR 2019 score≥20 was associated with all definitions of severe disease (table 1).

On multivariate LR analysis, a EULAR/ACR 2019 score≥20 was an independent predictor of cumulative major organ involvement (OR 7.30, 95%CI 4.86-10.95, p<0.001), moderate/severe disease activity (OR 7.36, 95%CI 2.18-24.82, p=0.001), ongoing immunosuppressants (OR 2.73, 95%CI 1.82-4.09, p<0.001) and ongoing systemic prednisolone>7.5 mg/day (OR 2.71, 95%CI 1.29-5.69, p=0.009), adjusted for significant covariates. In multivariate LR, a EULAR/ACR score≥20 was not associated with organ damage, defined as SDI≥1.

TO 072 – TABLE 1. Univariate and multivariate logistic regression analysis for severe SLE

MODEL 1 (dependent variable: cumulative major organ involvement†)				
	Unadjusted OR (90% CI)	**p**	**Adjusted OR (95% CI)**	**p**
Female	0.46 (0.31-0.70)	**0.002**	0.47 (0.26-0.84)	**0.010**
Age at diagnosis	0.97 (0.96-0.98)	**<0.001**	-	-
Early onset*	1.91 (1.46-2.51)	**<0.001**	1.61 (1.10-2.36)	**0.015**
Disease duration	1.05 (1.03-1.06)	**<0.001**	1.03 (1.01-1.05)	**0.002**
Score EULAR/ACR ≥20	8.80 (6.42-12.07)	**<0.001**	7.30 (4.86-10.95)	**<0.001**
MODEL 2 (dependent variable: SLE-DAS>7.64)				
	Unadjusted OR (90% CI)	**p**	**Adjusted OR (95% CI)**	**p**
Female	0.51 (0.26-1.01)	0.103	-	-
Age at diagnosis	0.97 (0.95-1.00)	**0.040**	0.97 (0.94-1.00)	**0.034**
Early onset*	1.24 (0.72-2.13)	0.509	-	-
Disease duration	0.97 (0.94-1.00)	**0.065**	0.94 (0.91-0.98)	**0.003**
Score EULAR/ACR ≥20	3.89 (1.75-8.64)	**0.005**	7.36 (2.18-24.82)	**0.001**
MODEL 3 (dependent variable: ongoing immunosuppressants)				
	Unadjusted OR (90% CI)	**p**	**Adjusted OR (95% CI)**	**p**
Female	0.71 (0.48-1.05)	0.152	-	-
Age at diagnosis	0.98 (0.97-0.99)	**<0.001**	-	-
Early onset*	1.51 (1.16-1.97)	**0.010**	1.51 (1.04-2.20)	**0.030**
Disease duration	0.98 (0.96-0.99)	**0.001**	0.95 (0.93-0.97)	**<0.001**
Score EULAR/ACR ≥20	2.78 (2.12-3.65)	**<0.001**	2.73 (1.82-4.09)	**<0.001**
Hydroxychloroquine	0.49 (0.33-0.71)	**0.002**	0.53 (0.30-0.93)	**0.027**
Oral corticosteroids	4.98 (3.86-6.41)	**<0.001**	4.47 (3.12-6.40)	**<0.001**
MODEL 4 (dependent variable: ongoing systemic prednisolone >7.5 mg/day)				
	Unadjusted OR (90% CI)	**p**	**Adjusted OR (95% CI)**	**p**
Female	0.68 (0.38-1.21)	0.268	-	-
Age at diagnosis	0.99 (0.98-1.01)	0.412	-	-
Early onset*	0.89 (0.57-1.37)	0.652	-	-
Disease duration	1.00 (0.98-1.02)	0.782	-	-
Score EULAR/ACR ≥20	3.72 (2.03-6.81)	**<0.001**	2.71 (1.29-5.69)	**0.009**
Hydroxychloroquine	0.43 (0.26-0.70)	**0.004**	0.46 (0.23-0.91)	**0.026**
Synthetic immunosuppressants and/or biologics	2.47 (1.62-3.77)	**<0.001**	1.40 (0.78-2.50)	0.255
SLE-DAS score	1.18 (1.12-1.23)	**<0.001**	1.17 (1.10-1.24)	**<0.001**
MODEL 5 (dependent variable: SDI score ≥1)				
	Unadjusted OR (90% CI)	**p**	**Adjusted OR (95% CI)**	**p**
Female	0.47 (0.31-0.69)	**0.001**	0.40 (0.24-0.67)	**<0.001**
Age at diagnosis	1.01 (1.00-1.02)	**0.019**	-	-
Early onset*	0.68 (0.52-0.90)	**0.021**	0.52 (0.36-0.75)	**<0.001**
Disease duration	1.05 (1.04-1.06)	**<0.001**	1.05 (1.03-1.07)	**<0.001**
Score EULAR/ACR ≥20	1.79 (1.35-2.37)	**<0.001**	1.43 (0.98-2.08)	0.063

*Diagnosis at age <26 years
†Presence of one or more of the following items: nephritis, neuropsychiatric lupus, hemolytic anemia, deforming or erosive arthritis and pulmonary items of SDI.

CI, Confidence Interval; EULAR/ACR, European League Against Rheumatism/American College of Rheumatology Systemic Lupus Erythematosus Classification Criteria; OR, Odds Ratio; SDI, Systemic Lupus International Collaborating Clinics/American College of Rheumatology Damage Index; SLE-DAS, Systemic Lupus Erythematosus Disease Activity Score.

Conclusion: A EULAR/ACR 2019 score≥20 is associated with severe disease in a prevalent SLE cohort. Although this is not surprising, given the similarity and close relationship between items included in all instruments, this score may, in association with measures of disease activity, contribute to management in clinical practice, stratification of cases in observational studies and selection of patients for clinical trials.

REFERENCES

1. Whittall Garcia LP et al.New EULAR/ACR 2019 SLE Classification Criteria: defining ominosity in SLE.Ann Rheum Dis.2021;80(6):767-774

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079 - PULMONARY ARTERIAL HYPERTENSION ASSOCIATED WITH CONNECTIVE TISSUE DISEASES - CHARACTERIZATION OF A PORTUGUESE COHORT IN A TERTIARY CENTRE

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Background: Pulmonary arterial hypertension (PAH), group 1 of pulmonary hypertension classification, is a rare disease (with prevalence ranging from 48 to 55 cases/million adults) which can be primary (idiopathic and heritable forms) or secondary to other conditions including connective tissue diseases (CTD-associated PAH). CTD-associated PAH is the second most common subtype of PAH.

Objective: To describe clinical, functional and hemodynamic parameters in a Portuguese cohort of patients with CTD-associated PAH. To assess its evolution during the disease follow-up.

Methods: Single-centre retrospective cohort study in a tertiary hospital. All patients included had the diagnosis of CTD (based on classification criteria) and pulmonary hypertension based on right heart catheterization (RHC; hemodynamically defined as a mean pulmonary artery pressure (mPAP) >20 mmHg). Demographic, clinical, functional and hemodynamic data were collected from 3 moments: T0, time of PAH diagnosis; T1, at 12th month of PAH treatment; T2, last visit at the Rheumatology department – PAH consultation.Descriptive statistics were performed. Data were analyzed using student's t test for continuous variables with normal distribution, non-parametric tests for continuous variables not normally distributed. Pearson's $\chi 2$ or Fisher's exact tests were used for categorical variables and Wilcoxon test was used to assess differences between ordinal variables.

Results: Sixteen patients were included, mostly female (68.8%) with a mean age at PAH diagnosis of 61.9±11.9 years. The median of follow-up duration was 2 years (IQR of 4; maximum of 13 years). Eight patients (50%) had systemic sclerosis (SSc), 4 had systemic lupus erythematous (SLE), 1 (6.3%) patient had primary Sjogren's syndrome (pSS), 1 (6.3%) had SLE/pSS overlap syndrome, 1 (6.3%) had mixed CTD and 1 (6.3%) had undifferentiated CTD. Six (37.6%) patients died and their average follow-up time was 1.67(±0.8) years. Regarding initial treatment, 62.5% of patients received monotherapy with endothelin receptor antagonist (ERA) or phosphodiesterase 5 inhibitor (PDE5i), and only 5 patients started dual therapy (31.3%). Five

TO 079 – TABLE 1.

Patients characteristics	n = 16
Sex - n (%)	
Female	11 (68.8)
Male	5 (31.3)
Age at CTD diagnosis - mean±SD	53.1±14.3
Age at last evaluation - median (IQR)	65.0 (13.0)
Age at PAH symptoms - mean±SD	60.2±12.3
Age at PAH diagnosis - mean±SD	61.9±11.9
Years between CTD and PAH diagnosis - median (IQR)	6.0 (16.0)
Follow-up duration since PAH diagnosis, years - median (IQR)	2.0 (4.0)
Minimum	1
Maximum	13
CTD - n (%)	
SSc	8 (50.0)
SLE	4 (25.0)
pSS	1 (6.3)
MCTD	1 (6.3)
SLE/pSS	1 (6.3)
UCTD	1 (6.3)
PID - n (%)	
No	9 (56.3)
NSIP	3 (18.8)
UIP	2 (12.5)
NSIP/UIP	1 (6.3)
LIP	1 (6.3)
Death/ cause - n (%)	6 (37.5)
Acute heart failure	2 (33.3)
Unknown	2 (33.3)
COPD exacerbation	1 (16.7)
HCAP	1 (16.7)
Follow-up duration since PAH diagnosis in deceased patients - mean±SD	1.67±0.8
T0 WHO functional class - n (%)	
Class I	3 (18.8)
Class II	6 (37.5)
Class III	7 (43.8)
Class IV	0
T1 WHO functional class - n (%)*	
Class I	6 (37.5)
Class II	3 (18.8)
Class III	4 (25.0)
Class IV	1 (6.3)
T2 WHO functional class - n (%)*	
Class I	4 (25.0)
Class II	5 (31.3)
Class III	2 (12.5)
Class IV	0

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TO 079 – TABLE 1. Continuation

Patients characteristics	n = 16
T0 1-year mortality risk - n (%)*^	
Low risk (<5%)	3 (18.8)
Intermediate risk (5-20%)	5 (31.3)
High risk (>20%)	7 (43.8)
T1 mortality risk in living patients - n (%)	
Low risk	6 (37.5)
Intermediate–low risk	3 (18.8)
Intermediate–high risk	2 (12.5)
High risk	4 (25.0)
T2 mortality risk in living patients - n (%)	
Low risk	6 (37.5)
Intermediate–low risk	4 (25.0)
Intermediate–high risk	1 (6.3)
High risk	2 (12.5)
T0 6MWD/m - n (%)*	
>440	1 (6.3)
320-440	4 (25.0)
165-319	3 (18.8)
<165	5 (31.3)
T1 6MWD/m - n (%)*	
>440	0 0
320-440	5 (31.3)
165-319	0 0
<165	4 (25.0)
T2 6MWD/m - n (%)*	
>440	0 0
320-440 3	(18.8)
165-319	2 (12.5)
<165	2 (12.5)
T0 NTpro-BNP (ng/mL)*	
<300	6 (37.5)
300-699	2 (12.5)
700-1100	1 (6.3)
>1100	5 (31.3)
T1 NTpro-BNP (ng/mL)	
<300	6 (37.5)
300-699	4 (25.0)
700-1100	3 (18.8)
>1100	3 (18.8)
T2 NTpro-BNP (ng/mL)	
<300	6 (37.5)
300-699	2 (12.5)
700-1100	2 (12.5)
>1100	2 (12.5)
T0 FVC/DLCO ratio - n (%)*	
<1.8	3 (18.8)
≥1.8)	9 (56.3

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TO 079 – TABLE 1. Continuation

Patients characteristics	n = 16
T1 FVC/DLCO ratio - n (%)*	
<1.8	3 (18.8)
≥1.8	8 (50.0)
T2 FVC/DLCO ratio - n (%)*	
<1.8	3 (18.8)
≥1.8	6 (37.5)
PASP (echocardiogram) - mean±SD*	
T0 PASP	65.8±29.3
T1 PASP	49.4±15.9
T2 PASP	65.5±24.1
RHC (at PAH diagnosis) - n (%)*	
mPAP	
20-25 mmHg	3 (18.8)
>25 mmHg	11 (68.8)
RAP	
<8 mmHg	5 (31.3)
8-14 mmHg	7 (43.8)
>14 mmHg	0
CI	
≥2.5 L/min/m2	2 (12.5)
2-2.4 L/min/m2	5 (31.3)
<2 L/min/m2	4 (25.0)
PVR	
<3 WU	1 (6.0)
3-5 WU	4 (25.0)
≥ 5 WU 7 (43.8)	
T0 treatment - n (%)*	
Monotherapy	10 (62.5)
Dual therapy	5 (31.3)
ERA	
PDE5i	6 (37.5)
ERA+PDE5i	5 (31.3)
Ambrisentan	1 (6.3)
Bosentan	3 (18.8)
Sildenafil	6 (17.5)
Ambrisentan + tadalafil	5 (31.3)
Hypocoagulation	3 (18.8)
T1 treatment - n (%)*	
Monotherapy	10 (62.5)
Dual therapy	5 (31.3)
ERA	5 (31.3)
PDE5i	5 (31.3)
ERA + PDE5i	5 (31.3)
Ambrisentan	3 (18.8)
Bosentan	3 (18.8)
Sildenafil	5 (31.3)
Ambrisentan + tadalafil	4 (25.)

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TO 079 – TABLE 1. Continuation

Patients characteristics	n = 16
T2 treatment - n (%)*	
Monotherapy	3 (18.8)
Dual therapy	8 (50.0)
ERA	2 (12.5)
PDE5i	1 (6.3)
ERA + PDE5i	8 (50.0)
Ambrisentan	1 (6.3)
Bosentan	1 (6.3)
Sildenafil	1 (6.3)
Ambrisentan + tadalafil	5 (31.3)
Macicentan + sildenafil	1 (6.3)
Bosentan + sildenafil	1 (6.3)
Ambrisentan + sildenafil	1 (6.3)
Switch from mono to dual therapy	5 (31.3)

Footnote: CI - cardiac index. COPD - chronic obstructive pulmonary disease. CTD - conective tissue disease. ERA - endothelin receptor antagonist. HCAP - health associated acquired pneumoniae. IQR - Interquartile Range. LIP - . MCTD - mixed conective tissue disease. mPAP - Pulmonary artery pressure. NTpro-BNP - N- terminal pro–B-type natriuretic peptide. NSIP - nonspecific interstitial pneumonia. PDE5i - phosphodiesterase 5 inhibitor. PID - Pulmmonary Intersticial disease. pSS - primary Sjogren's syndrome. PVR - pulmonary vascular resistance. RAP - right artrial pressure, mean. RHC - Right heart catheterization. SD - Standard deviation. SLE - Systemic Lupus Erythemous. SSc - systemic sclerosis. T0 - first eavaluation (strating treatment for PAH). T1 - 12 months after PAH treatment. T2 - last evaluation. UCTD - Undifferentiated connective tissue disease. UIP - usual intersticial pneumonia. WHO - World Health Organization. 6MWT - 6-minute walking distance (m). *2 missing value for T1 WHO funcional class; 5 missing values for T2 WHO functional class; 1 missing value for mortality risk; 3 missing values for T0 6MWD; 7 missing values for T1 6MWD; 9 missing values for T2 6MWD; 2 missing values for T0 NTpro-BNP; 4 missing values for T2 NTpro-BNP; 2 missing values for T0 FVC; 4 missing values for T1 FVC; 7 missing values for T2 FVC; 4 missing values for T0 FVC/DLCO; 5 missing values for T1 FVC/DLCO; 7 missing values for T2 FVC/DLCO; 1 missing value for T0 PASP; 4 missing values for T1 PASP; 8 missing values for T2 PASP; 2 missing values for mPAP; 4 missing values for RAP; 5 missing values for CI; 4 missing values for PVR; 1 missing value for T0 treatment; 1 missing value for T1 treatment; 5 missing values for T2 treatment. ^Mortality risk according to 2022 ESC/ERS Guidelines for the diagnosis and treatment of pulmonary hypertension.

patients switched from mono to dual therapy between T0 and T2. At T2, 50% of patients were on dual scheme (ERA + PDE5i), mostly ambrisentan and tadalafil. Considering the mortality risk (according to ESC recommendations), 7 (43.8%) patients were at high risk of 1-year mortality at baseline, while in T2, 10 (62.5%) patients had a low or intermediate-low risk of mortality. Regarding the functional class (WHO), considering the surviving patients, there was a significant improvement between the beginning of follow-up and the last assessment (p=0.03). RHC and echocardiography parameters and other assessed prognostic predictor factors (including 6 minutes walking distance test – 6MWD; N-terminal pro–B-type natriuretic peptide – NTpro-BNP; FVC/DLCO ratio) are presented in table 1.

Conclusions: In our cohort and according to the literature, the most frequent CTD associated with PAH was SSc. CTD-associated PH remains to be a noticeable cause of morbidity and mortality (almost 40% in our sample). Considering our small sample size, no statistically significant differences regarding 6MWD, NTproBNP and FVC/DLCO ratio were found. Despite this, the current treatment options (with a focus on combination therapy) appear to contribute to improvements in functional class and a reduction in mortality risk, as we can verify from the trend towards decreased functional class and mortality risk between the initial and final evaluations.

080 - IS THERE ANY DIFFERENCE BETWEEN SWITCHING AND CYCLING PATIENTS AFTER TUMOR NECROSIS FACTOR INHIBITOR (TNFI) FAILURE IN PSORIATIC ARTHRITIS? - CHARACTERIZATION OF A PORTUGUESE COHORT WITH ONE YEAR OF TREATMENT WITH TNFI AS THE FIRST BIOLOGICAL DMARD

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Background: Treatment with tumor necrosis factor alfa inhibitors (TNFis) tend to be the primary choice as first targeted therapy in patients with psoriatic arthritis (PsA) who had an inadequate response to conventional synthetic anti-rheumatic drugs (csDMARD) and/or NSAIDs. Although other biological DMARDs (bDMARD) with a different mechanism of action (MoA) may be used as first or second line treatment (IL-12/23- or IL-17-inhibitors or targeted synthetic DMARDs), the management of a first line bDMARD failure may consist in changing to a second bDMARD with a similar (cycling) or a different (switch) MoA, depending on patient and physician factors. Understanding prescribing patterns may help optimize treatment outcomes.

Objectives: To assess the prevalence of switching and

TO 080 – TABLE 1. Differences between cycling and switching patients.

	Cycling patients (n=13)	Switching patients (n=9)	p value
Age at symptoms onset - mean±SD	34.46±11.79	38.48±7.34	0.362
Age at diagnosis - mean±SD	40.65±11.84	40.46±8.17	0.987
Disease duration until first bDMARD, years – median, IQR	5.60 (15.82)	5.32 (3.98)	0.896
Months on first TNFi - n (%)			0.079
6 months	3 (23.0)	6 (66.7)	
12 months	10 (76.9)	3 (33.3)	
Sex - n (%)			0.655
Female	10 (76.9)	6 (66.7)	
Male	3 (23.1)	3 (0.33)	
Smoke habits (current or past) – n (%)	2 (15.4)	4 (44.4)	0.141
Disease pattern - n (%)			0.079
Symmetric polyarthritis	7 (53.8)	4 (44.4)	
Asymmetric oligoarthritis	2 (15.4)	5 (55.6)	
Axial pattern	4 (30.8)	0 (0)	
Skin psoriasis - n (%)	11 (84.6)	8 (88.9)	1.000
Nail psoriasis - n (%)	2 (15.4)	5 (55.6)	0.074
Enthesitis - n (%)	8 (61.5)	6 (66.7)	1.000
Dactylitis - n (%)	3 (23.1)	3 (33.3)	0.655
Treatment at baseline - n (%)			
NSAIDs	2 (15.4)	2 (22.2)	1.000
Glucocorticoid	6 (46.1)	5 (55.5)	1.000
csDMARD	6 (46.2)	9 (100)	**0.017**
bDMARD at baseline (TNFi) - n (%)			1.000
Adalimumab	5 (38.5)	4 (44.4)	
Golimumab	4 (30.8)	3 (33.3)	
Etanercept	3 (23.1)	1 (11.1)	
Certolizumab	1 (7.7)	1 (11.1)	
Disease activity and function at baseline			
Tender joints 68 - median, IQR (n=21)	15.00 (15.00)	5.00 (17.00)	0.464
Swollen joints 68 – median, IQR (n=21)	8.50 (12.00)	3.00 (10.00)	0.917
ESR (mm/1st hour) - mean±SD (n=21)	36.67±17.83	35.44±24.94	0.897
CRP (mg/dL) - median, IQR (n=21)	2.23 (3.88)	1.05 (5.47)	0.602
Patient VAS - mean±SD (n=21)	72.75±15.15	82.22±16.42	0.311
Physician VAS - mean±SD (n=20)	59.36±16.68	66.33±22.73	0.439
Pain VAS - median, IQR (n=20)	80.00 (60.00)	90.00 (50.00)	0.261
DAS28 4V CRP - mean±SD (n=21)	4.42±1.26	4.59±1.19	0.756
CDAI - median, IQR (n=20)	17.00 (22.50)	20.50 (15.90)	0.331
SDAI - mean±SD (n=20)	24.05±11.71	26.84±11.60	0.600
DAPSA - mean±SD (n=17)	38.02±18.80	32.22±11.40	0.461
BASDAI – median, IQR (n=18)	6.50 (1.75)	8.10 (4.15)	0.102
ASDAS CRP - mean±SD (n=18)	4.07±0,92	4,23±0.66	0.728
BASFI - mean±SD (n=17)	6.02±2.88	8.82±0.90	0.094
HAQ - mean±SD (n=17)	1.50±0.63	1.04±0.83	0.208
Disease activity and function before change			
Tender joints 68 - median, IQR (n=21)	6.00 (9.00)	2.00 (21.00)	0.702
Swollen joints 68 - median, IQR (n=21)	0.50 (2.00)	0.00 (11.00)	0.602
ESR (mm/1st hour) - median, IQR (n=21)	28.00 (23.25)	13.00 (19.00)	0.148
CRP (mg/dL) - median, IQR (n=21)	1.17 (5.44)	0.55 (2.95)	0.277
Patient VAS - mean±SD (n=21)	69.54±24.54	66.67±32.40	0.813
Physician VAS - mean±SD (n=21)	28.10±20.35	38.56±28.00	0.309
Pain VAS - median, IQR (n=20)	70.00 (30.00)	80.00 (71.50)	0.882
DAS28 4V CRP - mean±SD (n=21)	30.79±0.87	30.72±1.51	0.917
CDAI - mean±SD (n=21)	13.90±5.62	17.86±12.81	0.406
SDAI - mean±SD (n=21)	15.53±5.62	18.78±13.04	0.498
DAPSA - mean±SD (n=15)	21.49±8.29	22.72±20.42	0.885
BASDAI - mean±SD (n=18)	5.56±1.77	6.89±2.59	0.214
ASDAS CRP - mean±SD (n=18)	3.36±0.99	3.60±0.98	0.628
BASFI - mean±SD (n=18)	5.08±2.69	7.09±2.82	0.148
HAQ - mean±SD (n=18)	1.31±0.75	1.31±0.74	0.574

Footnote: ASDAS-Ankylosing Spondylitis Disease Activity Score; BASDAI- Ankylosing Spondylitis Disease Activity Index; BASFI- Bath Ankylosing Spondylitis Functional Index; bDMARD - biological anti-rheumatic drugs; CDAI - Clinical Disease Activity Index; CRP - C reactive protein; csDMARD - conventional synthetic anti-rheumatic drugs; DAPSA-Disease Activity in PSoriatic Arthritis; DAS-28 - Disease Activity Score 28; ESR - erythrocyte sedimantion rate; HAQ- Health Assessment Questionnaire; IQR - interquartile range; NSAIDs - non-steroidal anti-inflammatory drugs; SDAI-Simplified Disease Activity Index; VAS - visual analogue scale

cycling bDMARD after failure of TNFis therapy in the first year of treatment; to evaluate the differences in sociodemographic and disease characteristics between these 2 groups at the start of treatment with the first TNFi and at the time of bDMARD switching.

Methods: Single-center retrospective cohort study. All included patients met the CASPAR classification criteria for PsA diagnosis, were >18 years old and had failed response to TNFis (as the first bDMARD) within one year of treatment. Patients were divided into 2 groups, cycling or switching, if they change to other TNFis bDMARD or change MoA, respectively. Data were analyzed using student's t test for continuous variables with normal distribution, non-parametric tests for continuous variables not normally distributed and Pearson's $\chi 2$ or Fisher's exact test for categorical variables.

Results: Twenty-two patients were included; mostly female (72.7%) with a mean age at diagnosis of 40.57 years (±10.27). Eleven (50%) patients had symmetric polyarthritis, 7 (31.8%) had asymmetric oligoarthrytis and 4 (18.2%) had an axial pattern. Regarding initial treatment, 68.2% of patients received csDMARD, 50% received glucocorticoids and 18.2% received NSAIDs. Only 4 patients received TNFi as monotherapy.

Thirteen (59.1%) patients underwent cycling, and 9 (40.9%) patients underwent switching (7 patients to IL-17i, 1 patient to IL-12/23i and 1 to tocilizumab). Most of cycling patients (76.9%) changed bDMARD at 12 months; 6 (66.6%) switching patients changed at 6 months ($p>0.05$). All patients had high disease activity at baseline and before bDMARD change. No differences in disease activity/function scales were observed between the 2 groups ($p>0.05$). Regarding treatment at baseline, all patients taking csDMARD underwent switching ($p<0.05$). None of the patients with an axial pattern experienced switching. No other statistically significant differences were observed between the two groups (table 1).

Conclusions: Of the 22 patients who failed TNFi therapy in the first year, 59.1% switched to another TNFi and 40.9% to a different MoA therapy. According to literature, both strategies appear to be effective, with no differences in responses. Our results show no difference in change time (6 or 12 months) between groups, although 66.6% of switching patients changed at 6 months, likely due to primary lack of efficacy. This suggests other reasons involved in the choice of a second targeted therapy, behind treatment efficacy. All switching patients received csDMARDs at baseline, which could indicate some role of disease pattern in the choice of a second bDMARD in patients with PsA. Further studies (and larger cohorts) are necessary to determine prescribing factors (including physician perspective) and to verify its influence in the treatment response.

081 - FRACTURE RISK ASSESSMENT BASED ON FRAX® ALGORITHM IN PORTUGUESE PATIENTS WITH INFLAMMATORY BOWEL DISEASE: ASSOCIATION WITH CLINICAL FEATURES

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Background: Lower total body mineral density (BMD) has been found in patients with inflammatory bowel disease (IBD) which may increase osteoporosis (OP) and pathological bone fractures risk (FR) due to several mechanisms.
Objectives: To assess the prevalence of osteoporosis and proportion of treated patients in an IBD cohort and to evaluate the association of demographic, clinical and analytical features with fracture risk (based on FRAX® model) and BMD.
Methods: Monocentric retrospective study in a tertiary hospital. All patients included had IBD and were referred from Gastroenterology to the Rheumatology Department over 10 years (January 2013 – June 2023). Demographic, clinical and analytical data and BMD by dual-energy X-ray absorptiometry (DXA, total hip, femoral neck and lumbar spine) were collected at the time of the first visit in the Rheumatology outpatient center (V1); for patients over 40 years old, major (MFR) and hip (HFR) fracture risk was assessed with DXA based on FRAX® (validated for Portuguese population) and proportion of patients with indication of therapy for OP was calculated (according to Portuguese recommendations). Correlations between continuous variables were evaluated by Spearman rank test and Pearson's correlation coefficient and Mann-Whitney U test was used in the comparison analysis between groups.
Results: Two hundred forty-four patients patients were included, predominantly female (57.4%), with a mean age at V1 of 46.65 years. 75.4% had Crohn's disease (CD), and 24.6% had ulcerative colitis (UC). Azathioprine (41.0%) and infliximab (28.3%) were the most used IBD treatments. At V1, 4.9% were taking glucocorticoids, with 75.8% having prior use; 14.1% had osteoporosis (DXA T score ≤ -2.5), 4.1% had prior fragility fractures, and 78.7% had low vitamin D levels (<30ng/mL). Concerning to individuals >/= 40 years old (n=148, 60.7%), total proteins and albumin correlated negatively with HFR (r=-0.194, p=0.035) and MFR (r=-0.187, p=0.026), respectively. Erythrocyte sedimentation rate (ESR) correlated positively with MFR (r=0.175; p=0.045), ferritin correlated positively with MFR (r=0.214; p=0.017) and HFR (r=0.206; p=0.022); beta-CTX correlated positively with MFR (r=0.221; p=0.010). Other relevant correlations and differences are presented in table 1. Of interest, levels of 25(HO) VitD correlated negatively with beta-CTX (r=-0.255; p=0.004). Fifteen patients (10.1%) had an indication for osteoporosis treatment (no differences between genders, p=0.848) and only 3 were undergoing treatment with bisphosphonates. Across patients under 40 years old (n=96, 39.3%), hemoglobin levels (Hb) correlated positively with total hip BMD (r=0.294; p=0.05) and with femoral neck BMD (r=0.221; p=0.042), ESR correlated negatively with total hip BMD (r=-0.251; p=0.020), beta-CTX correlated positively with total hip (r=0.217; p=0.043) and femoral neck (r=0.238; p= 0.027) BMD and osteocalcin correlated positively with femoral neck BMD (r=0.221; p=0.041). No other statistically significant correlations were observed.
Conclusions: This study shows a higher prevalence of OP in comparison with the general Portuguese population and an important number of untreated patients with high FR. It also reveals significant associations between clinical/analytical variables and FR (over 40 years old) and BMD (under 40 years old) in IBD patients: inflammatory state (positive correlation of FR with ESR and ferritin; negative correlation of BMD with ESR; positive correlation of BMD with Hb) and nutritional status (albumin correlated negatively with FR).

TO 081 – TABLE 1. Patients ≥ 40 years old (n=148): features related to major and hip fracture risk assessed by FRAX®Port.

	Major fracture risk (%)	Hip fracture risk (%)
Age at V1	**r=0.593; p<0.001**	**r=0.439; p<0.001**
BMI	**r=-0.212; p=0.010**	**r=-0.317; p<0.001**
Age at IBD diagnosis	**r=0.271; p<0.001**	**r=0.180; p=0.030**
Hemoglobin	n.s.	n.s.
Total proteins	n.s	**r=-0.194; p=0.035**
Albumin	**r=-0.187; p=0.026**	n.s.
Beta-CTX	**r=0.221; p=0.010**	n.s.
Osteocalcin	**r=0.236; p=0.006**	n.s.
25(HO)VitD	n.s.	n.s.
CRP	n.s.	n.s.
ESR	**r=0.175; p=0.045**	n.s.
Ferritin	**r=0.214; p=0.017**	**r=0.206; p=0.022**
Calprotectin	n.s.	n.s.
Sex, median (IQR)	n.s.	n.s.
Female (n=90)	2.10 (2.30)	0.30 (0.90)
Male	1.95 (2.10)	0.35 (1.00)
Previous fracture, median (IQR)	**p<0.001**	**p<0.001**
Yes (n=10)	9.90 (10.70)	4.10 (9.10)
No	1.90 (1.90)	0.30 (0.80)
Exposition to GC, median (IQR)	**p=0.005**	n.s.
Yes (n=114)	2.20 (2.30)	0.35 (1.00)
No	1.30 (1.40)	0.20 (0.60)
IBD type	n.s.	n.s.

083 - ANTIPHOSPHOLIPID ANTIBODIES POSITIVITY AMONG PORTUGUESE PATIENTS WITH SYSTEMIC SCLEROSIS: IS THERE ANY ASSOCIATION WITH VASCULOPATHY?

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Introduction: Systemic Sclerosis (SSc) is a connective tissue disease that predisposes to vasculopathy and fibrosis which leads to organ injury (skin, lung, heart, gastrointestinal tract) and symptoms such as Raynaud´s phenomenon (RP), digital ulcers (DU), gangrene or pulmonary arterial hypertension (PAH). Fibroblast activation, endothelial dysregulation, abnormal coagulation and fibrinolysis and immune-mediated chronic inflammation are mechanisms involved in arterial occlusion. An increased positivity of antiphospholipid antibodies (APPa), anti-B2 glycoprotein antibodies (aβ2GPIa), anticardiolipin antibodies (aCLa) and lupus inhibitor (LI) in SSC patients has been reported and some results have suggested a role of APPa in physiopathology of vasculopathy.

TO 083 – TABLE 1. Patient characteristics

Characteristics	Patients (n=137)	
Age at diagnosis - mean±SD	51.5±12.3	
Sex - n (%)		
Female	123	89.8
Male	14	10.2
Classification criteria* - n (%)	137	100
Disease form - n (%)		
Limited cutaneous scleroderma	127	92.7
Diffuse scleroderma	10	7.3
Telangiectasia - n (%)	84	61.3
Raynaud's phenomenon - n (%)	133	97.1
Digital ulcers - n (%)	48	35.0
Gangrene - n (%)	6	4.4
Presence of capillaroscopic alterations* - n (%)	82	59.9
≥ 1 APPa positivity - n (%)	22	16.1
Lupus Inhibitor*	1	0.7
Anticardiolipin antibody*	10	7.3
Anti-β2-glycoprotein*	21	15.3
Triple APPa positivity - n (%)	0	0.0
Pulmonary arterial hypertension	3	2.2
Antinuclear autoantibodies*	132	96.4
SSc-related autoantibodies		
Anticentromere*	85	62.0
Anti-Scl70*	26	19.0

SD - Standard deviation. *ES1989 and/or ACR/EULAR 2013; 41 missing values for capillaroscopy; 87 missing values for lupus inhibitor; 18 missing value for anticardiolipin antibody; 18 missing values for anti-ß2-glycoprotein; 1 missing values for antinuclear antibodies; 2 missing values for anticentromere antibodies; 3 missing values for anti-Scl70 antibodies.

Objectives: To assess the prevalence of APPa positivity in Portuguese patients with SSc and its association with vascular manifestations.
Material and methods: Cross-sectional retrospective single-center study with all included patients (registered on Rheumatic Diseases Portuguese Register, Reuma.pt) meeting the ACR/EULAR 2013 or ACR 1980 classification criteria for SSC diagnosis. Overlap syndromes were excluded. Descriptive analysis were performed. Data were analyzed using student's t test for continuous variables and Pearson's $\chi 2$ or Fisher's exact test for categorical variables.
Results: One hundred and thirty-seven patients were evaluated (table 1), mostly female (89.8%), with a mean age at diagnosis of 51.5 years (±12.3). Ten patients had diffuse scleroderma and 127 patients had limited cutaneous scleroderma. Of all patients, 133 (97.1%) experienced RP, 48 (35%) developed DU and 6 (4.4%) developed gangrene. Regarding APPa, 22 patients (16.1%) tested positive for at least 1, IgM/IgG [21 (15.3%) for aβ2GPIa, 10 (7.3%) for aCLa and 1 patient for LI (0.7%)]. Although the difference was not statistically significant, 81.8% of patients who tested positive for AAPa had telangiectasia, compared to 52.8% of patients who tested negative for AAPa (p=0.09). Mean age at diagnosis was lower among positive APPa patients (47.31±6.93; p=0.02). None of the other clinical parameters showed a statistically significant association with APPa.
Conclusion: The prevalence of APPa seems increased in patients with SSc as described in the literature, however we did not find a statistically significant association of APPa positivity with development of DU or other features (RP, PAH). Some published data fail to establish a strong correlation due to varying study methodologies. Of note, we defined positivity using the manufacturer's specified cut-off value, whereas in some other studies, lower titers were considered. Curiously, a lower average age at diagnosis was observed in APPa positive patients' group (p<0.05). For more consistent conclusions, further studies are necessary to understand the role these antibodies might have in SSc presentation and severity.

086 - REMISSION VERSUS LOW DISEASE ACTIVITY AS TREATMENT TARGETS IN RHEUMATOID ARTHRITIS: HOW TO STRIKE THE RIGHT BALANCE BETWEEN TOO STRICT AND TOO LENIENT TARGETS? A META-ANALYSIS OF INDIVIDUAL PATIENT DATA

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Objectives: To evaluate the impact of using SDAI-LDA (low disease activity) versus different definitions of remission as a treatment target in established rheumatoid arthritis.
Methods: Individual-patient data from 8 randomised controlled trials were meta-analysed. Four definitions of the target were considered at 6 months: i) SDAI-LDA: SDAI ≤11; ii) SDAI-Remission: SDAI ≤3.3; iii) 4V-Remission: Tender and swollen 28-joint counts and CRP (mg/dl) all ≤ 1 and patient global assessment (PGA) ≤2; iv) 3V-Remission: as 4V, excluding PGA. The mean radiographic change in the modified total Sharp-van der Heijde score (mTSS) and the Good Radiographic Outcome (GRO) rates (change ≤0.5 units mTSS) over two years were compared among target definitions. Radiographic progression and the distribution of the individual criteria of the Boolean definition in the only-LDA subgroup (3.3 < SDAI ≤11) were analysed.
Results: In total, 4374 patients (mean disease duration of 5.9 years (95%CI:4.6-7.1)) were included. The pooled rate of SDAI-LDA at 6 months was 48.9%, with 12.9% in SDAI-remission. The 4V- and 3V-Remission were achieved by 16.2% and 23.1%, respectively. Mean radiographic progression was 0.55(0.14-0.96) units for SDAI-LDA and 0.20(-0.09-0.54), 0.28(-0.07-0.62), 0.28(-0.10-0.65) for SDAI-Remission, 4V- and 3V-Remission states, respectively. Patients with SDAI Pure-LDA presented significantly more radiographic progression than patients in SDAI-Remission (mean 0.74 vs 0.22 units,p<0.05). Over 53% of all patients achieving SDAI-LDA were not in 3V-Remission and had more mean radiographic progression over 2 years than those who met both targets (0.70 vs 0.25 units, p=0.014). Among patients with SDAI-LDA but not in SDAI-Remission, 40% scored PGA>2, reflecting relevant disease impact.
Conclusion: SDAI-LDA is associated with more structural damage than any of the definitions of remission, including 3V-Remission. It also allows substantial disease impact to go unchecked and uncontrolled. Remission should be the target in RA.

088 - CLINICAL-IMMUNOLOGICAL PROFILING AND PREDICTORS OF THROMBOSIS IN PATIENTS WITH ANTIPHOSPHOLIPID SYNDROME: DATA FROM A DEDICATED CLINIC AT A TERTIARY CENTRE

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Introduction: Antiphospholipid syndrome (APS) is a systemic disease characterized by thrombotic manifestations and/or pregnancy morbidity in the presence of persistent antiphospholipid antibodies (aPL). We aimed to describe the clinical and immunological features of a cohort of patients with APS followed at a tertiary center and to find potential predictors for thrombosis.
Methods: We included all patients with APS followed at a rheumatology tertiary center from January 1996 to March 2024. Only patients meeting the Sydney classification criteria were included.
Results: A total of 85 patients (81.2% female) were included (Table 1). Almost 2/3 of patients had primary APS (63.5%), whereas most patients with secondary APS had associated systemic lupus erythematosus (SLE) (n= 21/31; 67.8%). Mean age at diagnosis was 37.1±14.5 years, and median disease duration 11 years (IQR 15). Based on the presenting manifestation, 71 patients (83.5%) had thrombotic APS, 46 (54.1%) and 30 (35.3%) of whom with venous or arterial events, respectively. Deep vein thrombosis, pulmonary embolism and stroke were the most frequent presenting thrombotic events. Obstetric morbidity was the first disease expression in 23 patients (27.1%) while one patient (1.1%) was diagnosed due to non-criteria manifestations – Libman–Sacks endocarditis with subsequent stroke. Over a follow-up of 28 years, two patients (2.4%) died, both from non-APS related causes. More than half of patients (64.7%) had at least one cardiovascular (CV) comorbid condition, namely smoking (47.3%) and dyslipidemia (41.6%)). Lupus anticoagu-

TO 088 – TABLE 1. Demographics, clinical and laboratory features of APS patients

Demographics N=85 (100%)	
Sex (female), n (%)	69 (81.2)
Caucasians, n (%)	80 (94.1)
Age at APS diagnosis (years), mean ± SD	37.1 ± 14.5
Age of symptom onset (years), mean ± SD	35.4 ± 13.9
Disease duration (years), median [IQR]	11 [IQR 15]
Deaths, n (%)	2 (2.4)
Classification, n (%)	
Primary APS	54 (63.5)
APS associated with other rheumatic diseases	31 (36.5)
Systemic lupus erythematosus	21 (24.7)
Other diseases	10 (11.7)
Antiphospholipid antibodies, n (%)	
LA	62 (76.5)
aCL	61 (76.3)
IgG	47 (58.8)
IgM	27 (33.8)
IgG and IgM	13 (15.3)
aB2GPI	50 (62.5)
IgG	39 (48.8)
IgM	22 (27.5)
IgG and IgM	10 (11.8)
Triple positivity	32 (40.5)
Cardiovascular risk factors *, n (%)	
Any of the following	55 (64.7)
Hypertension	31 (39.7)
Obesity	24 (30.4)
Diabetes	6 (7.9)
Dyslipidemia	32 (41.6)
Smoking	35 (47.3)
APS presenting manifestations, n (%)	
Thrombotic manifestations	71 (83.5)
Arterial events	30 (35.3)
Venous events	46 (55.4)
Microvascular	1 (1.2)
Obstetric manifestations	23 (27.1)
Non thrombotic manifestations	1 (1.2)
APS ever treatment, n (%)	
Anticoagulation	72 (88.9)
Vitamin K antagonist	59 (73.8)
DOAC	11 (13.9)
LMWH	23 (27.1)
Low dose aspirin	53 (66.3)
Combined antiplatelet and anticoagulant	46 (54.8)
Hydroxychloroquine	40 (50.0)
Thrombotic recurrence**, n (%)	12 (15.4)
Arterial	5 (10.0)
Venous	7 (14.3)
Bleeding events ***, n (%)	11 (13.9)
Major bleeding	8 (10.1)

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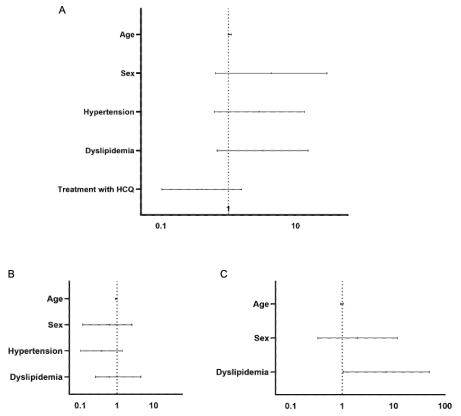
TO 088 – TABLE 1. Continuation

Type of cumulative thrombotic events, n (%)	
Lower limb deep vein thrombosis	32 (38.6)
Upper limb deep vein thrombosis	1 (1.2)
Superficial vein thrombosis (upper and lower limb)	7 (8.2)
Acute lower limb arterial thrombosis	5 (5.9)
Acute upper limb arterial thrombosis	1 (1.2)
Stroke	15 (17.6)
Transient ischemic attack	**5 (5.9)**
Central nervous system microvascular disease	5 (5.9)
Central sinus venous thrombosis	5 (5.9)
Thrombosis of central/branch retinal artery	4 (4.7)
Thrombosis of central/branch retinal vein	1 (1.2)
Pulmonary embolism	16 (18.8)
Myocardial infarct	4 (4.7)
Mesenteric artery thrombosis	1 (1.2)
Mesenteric vein thrombosis	1 (1.2)
Splenic vein thrombosis	1 (1.2)
Cutaneous ulcers	1 (1.2)

Legend: ab2GPI: anti-β2-glycoprotein I; aCL: anticardiolipin; APS: Antiphospholipid syndrome; DOAC: Direct oral anticoagulants; IQR: Interquartile Range; LA: Lupus anticoagulant; LMWH: Low-molecular-weight heparin. SD: Standard Deviation; *Cardiovascular risk factors were retrieved as stated in the medical notes. **We only considered recurrent events occurring during antithrombotic therapy (anticoagulant and/or antiplatelet therapy). ***Bleeding events were classified according to the International Society on Thrombosis and Haemostasias definitions. We only considered bleedings occurring during antithrombotic therapy (anticoagulant and/or antiplatelet therapy).

lant, anticardiolipin and anti-β2-glycoprotein I antibodies were detected in 62 (76.5%), 61 (76.3%), and 50 patients (62.5%) respectively. Triple positivity was observed in 40.5% of patients. Regarding treatment, anticoagulation was ever used in 72 patients (88.9%), most often (n=59, 73.8% of total) with vitamin K antagonists (VKA); low-dose aspirin (LDA) was used in 52 (66.3%), and combined antiplatelet/anticoagulant therapy in 46 patients (54.8%); hydroxychloroquine (HCQ) was prescribed to 40 patients (50.0%), 26 (65%) of whom had other concomitant rheumatic diseases (19 had SLE). Bleeding events occurred in 11 patients (13.9%), most of which (n=8; 72.7%) were major bleedings - all but one in patients under combined antiplatelet/anticoagulant therapy. Thrombotic recurrence was noted in 12 patients (15.4%) - 7 (58.3%) venous and 5 (41.7%) arterial events.

In univariate analysis, we found arterial thrombosis to be more frequent in men (75% vs 26.8%, p<0.001) and in patients with CV comorbidities, such as hypertension (58.1% vs 21.7%, p=0.001) and dyslipidemia (62.5% vs 15.9%, p<0.001), and less frequent in patients treated with HCQ (20% vs 55%, p=0.001). No associations were found with venous thrombosis. Re-



TO 088 – Figure 1. Multivariate model for prediction of arterial and venous thrombosis and thrombotic recurrence. **1-A: Predictors of arterial thrombosis.** Male sex (OR=4.355, 95% CI 0.647-29.336, p=0.131); Age of symptom onset (OR=1.050, 95% CI 0.994-1.109, p=0.082); Hypertension (OR=2.884, 95% CI 0.616-13.512, p=0.179); Dyslipidemia (OR=3.243, 95% CI 0.679-15.483, p=0.140); Treatment with hydroxychloroquine (HCQ) (OR=0.403, 95% CI 1.579-0.103, p=0.192). **1-B: Predictors of venous thrombosis.** Male sex (OR=0.545, 95% CI 0.116-2.549, p=0.441); Age of symptom onset (OR=0.946, 95% CI 0.900-0.994, p=0.029); Hypertension (OR=0.374 95% CI 0.101-1.387, p=0.141); Dyslipidemia (OR=1.086, 95% CI 0.263-4.481, p=0.909) **1-C: Predictors of thrombotic recurrence.** Male sex (OR=1.984, 95% CI 0.336-11.719, p=0.449); Age of symptom onset (OR=0.988, 95% CI 0.926-1.055, p=0.725); Dyslipidemia (OR=7.181, 95% CI 1.045-49.320, p=0.015)

currency of thrombotic events were more frequent in patients with dyslipidemia (23.3% vs 6.8%, p=0.041). In multivariate analysis adjusted for gender and age, younger age at disease onset was predictor of venous thrombosis, while dyslipidemia was a predictor of thrombotic recurrence. We found no predictors for arterial thrombosis. (Figure 1)

Conclusion: In our cohort of APS patients, younger patients seem more prone to venous thrombosis. We also found an association between arterial thrombosis and risk of thrombotic recurrence with CV comorbidities, namely dyslipidemia, highlighting the need to tightly control these conditions. HCQ exhibited a trend to be protective for arterial thrombosis, in line with its antithrombotic effect documented in basic research studies.

089 - MYOSITIS-ASSOCIATED INTERSTITIAL LUNG DISEASE CHARACTERIZATION AND RESPONSE TO TREATMENT

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Introduction: Interstitial lung disease (ILD) is a common extramuscular manifestation of idiopathic inflammatory myopathies (IIM), associated with a worse prognosis.

Objectives: To characterize the lung involvement of an IIM cohort and its response to treatment.

Methods: We retrospectively collected data from all IIM patients followed in our Rheumatology Department from June 2016 to March 2024. For response-to-treatment analysis, we selected ILD patients with at least 1 year of follow-up and 2 different pulmonary function test (PFT) evaluations. Associations between the different variables were tested using Chi-Square, Fisher's Exact, Student's t, paired-samples t, McNemar, or Mann-Whitney tests. Definite and likely associations were defined by p<0.050 and p<0.100, respectively.

Results: 198 IIM patients were included, of whom 144 were females, with a median age at diagnosis of 47.5 (IQR 31.0) years, and a median disease duration of 5.0 (IQR 7.0) years.

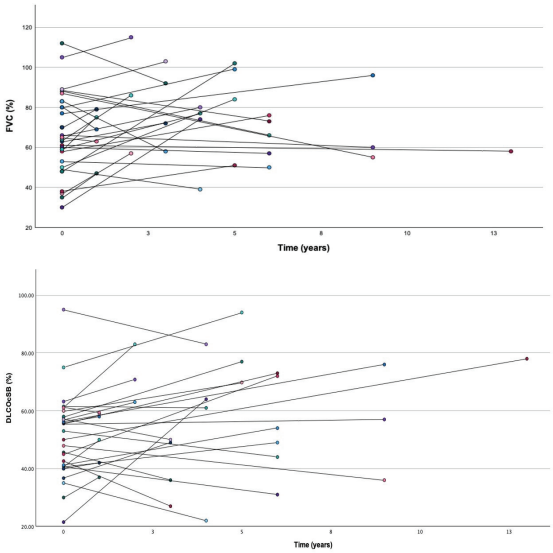
Sixty-nine patients had ILD based on high-resolution computed tomography (HRCT). Of these, 48 patients exhibited a radiological pattern compatible with non-specific interstitial pneumonia (NSIP), of whom 7 had fibrotic NSIP.

The most common diagnoses were antisynthetase syndrome (ASSD) (33/69) and dermatomyositis (12/69). ASSD was significantly more common than in non-ILD patients [7/129, p<0.001].

Patients with ILD more commonly reported exertional dyspnea (39/62), grade 1 on the mMRC scale (21/39) and grade 2 on the NYHA classification (19/39), asthenia (37/61), and cough (34/61), most commonly non-productive (26/61). Smoking status (current, past, or never smoking) had no statistically significant impact on symptoms during the follow-up (p=0.472 at baseline, p=0.192 at the last visit).

The most prevalent autoantibodies were anti-Jo1 (25/64) and anti-MDA-5 (8/64), both significantly more prevalent than in non-ILD patients [vs 4/106, p<0.001; vs 3/106, p=0.021].

IIM-associated ILD patients presented with lower FVC% (82.0±25.1 vs 99.2±23.0, p<0.01), DLCOsb% (61.2±20.0 vs 82.4±16.7, p<0.01), and DLCO-VA%



TO 089 – Figure 1. . Evolution of FVC (%) and DLCOcSB (%) values over time for each patient with ILD (N = 31).

(82.0±16.2 vs 98.0±38.6, p=0.005) than non-ILD patients. From the first to the last visit (4.1±4.0 years), there was a likely mean FVC% improvement (6.0±19.2, p=0.097) and a definite mean DLCOsb% improvement (6.2±14.1, p=0.022) (Figure 1). This was especially true for patients treated with intravenous immunoglobulin (IVIg) (Table 1).

Additionally, patients treated with azathioprine (AZA) showed a significant improvement in DLCOsb% (p=0.029) and DLCO-VA% values (p=0.029), and patients treated with a combination of mycophenolate mofetil (MMF) and IVIg, or rituximab (RTX) and IVIg, also had significant PFT improvements (Table 1).

When comparing HRCT changes between baseline and the most recent evaluation, the majority of ILD patients showed stability or improvement in radiologic patterns. Specifically, in 89.6% (43/48) of patients with at least two HRCT scans for comparison, there was no new-onset fibrosis, ground-glass opacities (42/47), honeycombing (41/46) or bronchiectasis (35/46). There was a likely association between prevention/improvement of bronchiectasis and treatment with RTX (p=0.063).

Conclusions: In our cohort, most patients demonstrated stability in HRCT patterns and PFT parameters over time, with definite improvement in DLCOsb values following immunosuppressive therapy. Therapeutic schemes combining IVIg with MMF, IVIg with RTX, and AZA were particularly effective. Additionally, there was likely a significant impact on the prevention/improvement of bronchiectasis with RTX treatment.

091 - RECENT AND PAST PAIN EXPERIENCES IN JUVENILE IDIOPATHIC ARTHRITIS PATIENTS: PERSPECTIVES OF ADOLESCENTS AND CAREGIVERS

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Introduction: In juvenile idiopathic arthritis (JIA), symptom management is mostly directed to pain that results from chronic joint inflammation. However, centralized pain mechanisms have been documented in many rheumatic disorders which originate pain experiences that are disproportionate to joint lesions. Little is known about the relative burden of non-specific pain when compared to peripheral joint pain in JIA patients, considering both youth and caregivers' perspectives.

Aim: To quantify recent and past pain experiences, including peripheral joint pain and typically non-specific pains, reported by adolescents with JIA and their caregivers.

Methods: As part of the SEPIA – Studying Experiences of Pain In Adolescents study (https://ispup.up.pt/sepia/), 61 eligible families were identified by Reuma.pt centers in 2022/23: patients with a diagnosis of JIA, aged between 14 and 17 years, and whose caregivers consented to be contacted by the study team. Of those eligible, 42 participants from five centers (21 adolescent-caregiver pairs) installed our data collection mobile app. The app was used remotely and included: a) the Lübeck Pain-Screening Questionnaire to assess all pains over the preceding 3 months; b) items on any recalled persistent/recurrent pain at ages 13, 10 and 7 years; and c) visual trajectories questionnaires on the course of back/neck pain, headache and abdominal pain over the previous year. Both caregivers (90.5% were mothers) and adolescents (57.1% were females) reported on the adolescent's pain.

Results: The presence of any pain in the previous 3 months was reported by 57.1% of adolescents with JIA

and caregivers, with the knee being the most frequent site and around 80% of participants with pain reporting chronicity. When asked about recalled experiences, 66.7% of adolescents with JIA and 76.2% of parents reported a history of persistent or recurrent pain at age 13 years, 38.1% and 52.4% reported remembering persistent/recurrent pain at age 10, and 28.6% and 33.3% at age 7. Beyond peripheral joints, 76.2% of adolescents and 61.9% of caregivers reported episodic or persistent back/neck pain trajectories in the previous year (JIA-related or not), whereas 81.0% of adolescents and 66.7% of caregivers reported episodic or persistent headache trajectories and 71.4% of adolescents and 66.7% of caregivers reported episodic or persistent abdominal/pelvic pain trajectories.

Conclusion: In the majority of adolescents with JIA, bodily pain was a frequent experience, both recent and remote, even though it was slightly overestimated by caregivers relative to their children. However, pain trajectories beyond peripheral joint pain, such as back/neck pain, headache, and abdominal/pelvic pain, were even more frequent and seemed underestimated by caregivers.

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096 - TRANSLATION AND CROSS-CULTURAL ADAPTATION OF THE MSQUASH INTO EUROPEAN PORTUGUESE

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Background and aim: Regular physical activity is recommended for all patients with axial spondyloarthritis (axSpA), but it can be challenging to investigate or measure its frequency, duration and intensity. The modified Short Questionnaire to ASsess Health-enhancing physical activity (mSQUASH) is a patient-reported outcome measurement instrument to assess daily physical activity in patients with axSpA and the mSQUASH that has four domains: 1) commute to/from work or school and commute to/from other destinations, 2) work (paid/unpaid) or school/study, 3) household activities, 4) leisure activities and sports and exercise. It was developed and validated in Dutch and since then translated and validated in English. The aim of this study was to translate the mSQUASH into European Portuguese and to evaluate de the face validity,

TO 096 – TABLE 1. Demographic and clinical characteristics of the patients who participated in the field testing of the European Portuguese version of the mSQUASH questionnaire.

ID	Age	Gender	Education	Working status	Disease duration (y)	axSpA subtype	HLA-B27	Treatment	ASDAS
1	59	F	Lower secondary	Full-time	22	r-axSpA	-	NSAIDs	1.4
2	36	M	Bachelor	Full-time	15	r-axSpA	+	NSAIDs	2.4
3	60	M	Illiterate	Work disability due to health	20	r-axSpA	-	ADA	3.1
4	58	M	Associate	Part-time	4	nr-axSpA	+	ADA	1.0
5	46	M	Professional course	Full-time	26	nr-axSpA	+	NSAIDs	0.8
6	49	F	Upper secondary	Part-time	8	nr-axSpA	+	SEC	0.9
7	51	F	Lower secondary	Job seeking	12	r-axSpA	-	ADA	2.8
8	42	M	Upper secondary	Part-time	15	r-axSpA	+	ADA	2.4
9	49	F	Primary	Work disability due to health	10	nr-axSpA	-	ADA	2.0
10	49	F	Associate	Full-time	30	nr-axSpA	+	CZP	2.7

ID, Identification number in study; F, female; M, male; ISCDED, International Standard Classification of Education; y, years; r-axSpA, radiographic axial spondyloarthritis; nr-axSpA, non-radiographic axial spondyloarthritis; NSAIDs, nonsteroidal anti-inflammatory drugs; ADA, Adalimumab; SEC, Secukinumab; CZP, Certolizumab pegol; ASDAS, Axial Spondyloarthritis Disease Activity Score.

equivalence and applicability of the translated version in patients with axSpA in Portugal.
Methods: The translation was done using the English version of the mSQUASH, following the forward-backward procedure proposed by the Beaton method. The scientific committee responsible for the translation comprised six rheumatologists, two rheumatology fellows, three bilingual translators and the developer of the original mSQUASH. Two bilingual translators performed independent forward translations from English into Portuguese, and these versions were harmonized in a consensual version. Another translator back translated the synthesized version into English. Translation discrepancies were discussed within a scientific committee and a pre-final version of the questionnaire was developed. The pre-final version was field tested with cognitive debriefing interviews in 10 patients with axSpA with large variation in gender, age, disease duration, treatment, educational level and working status (Table 1).
Results: The translation process of the Portuguese version of the mSQUASH was completed without major issues and all minor discrepancies were solved in consensus meetings. During field testing of the pre-final version of the questionnaire, all patients reported that it was clear and appropriate. The median time for completion of the Portuguese mSQUASH was 4:15 (standard deviation: 2:44) minutes. The analysis of the comments and suggestions by the patients led to the correction of minor spelling errors and to the addition of examples to item "12. Home maintenance". This question was perceived by patients as conceptually confusing with the items related to household activities. The final version of the questionnaire, incorporating the feedback from the patients, was approved by the scientific committee.

099 - UNDERSTANDING FRAILTY IN RHEUMATOID ARTHRITIS AND PSORIATIC ARTHRITIS: A COMPARATIVE ANALYSIS

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Background: Frailty, characterized by reduced physiological reserve and increased vulnerability to stressors, is common in chronic inflammatory conditions such as rheumatoid arthritis (RA) and psoriatic arthritis (PsA).
Objective: To explore the risk factors associated with higher frailty risk in RA and PsA patients and evaluate their overall health impact.
Methods: This unicentric, cross-sectional study included patients with RA and PsA meeting the American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) 2010 and Classification for Psoriatic Arthritis (CASPAR) criteria, respectively. Frailty was assessed using validated Portuguese versions of FRAIL-S, scoring from 0 to 5, classifying patients as robust (0), pre-frail (1-2) or frail (≥3). Data on demographics, clinical characteristics, comorbidities, and patient-reported outcomes, including patient global assessment [PGA], pain [VAS], function [HAQ], and quality of life [EQ-5D], were collected. Associations with frailty risk were evaluated using parametric and non-parametric tests followed by multivariate analysis.
Results: A total of 112 patients were included: 70 with RA and 42 with PsA. Twenty-eight (25,0%) patients were identified at high risk of frailty (21 RA and 7 PsA), 40 (35,7%) as pre-frail (27 RA and 13 PsA) and 44

TO 099 – TABLE 1. Demographics, clinical, and patient-reported outcomes associated with frailty in patients with rheumatoid arthritis and psoriatic arthritis

	Rheumatoid Arthritis			Psoriatic Arthritis		
	Frail (n=21)	Non-Frail (n=49)	*p*	Frail (n=7)	Non-frail (n=35)	*p*
Mean age (SD) (years)	70,2 (9,5)	56,8 (15,3)	**0,002**	66,3 (10,0)	56,2 (13,4)	**0,039**
Women, n (%)	16 (76,2%)	36 (73,5%)	0,595	4 (57,1%)	14 (35,0%)	0,385
Body mass index (SD) (kg/m²)	23,3 (4,7)	27,2 (4,5)	**0,028**	28.1 (5,5)	28,3 (5,7)	0,976
Disease duration (SD) (years)	16,3 (11,1)	9,9 (9,5)	**0,039**	19,0 (11,8)	9,3 (4,5)	**0,011**
RF positivity, n (%)	17 (80,9%)	32 (65,3%)	0,198	n/a	n/a	
ACPA positivity, n (%)	16 (76,2%)	36 (73,5%)	0,596	n/a	n/a	
TJC28, median (IQR)	1 (0-3)	0 (0-2)	0,387	0 (0-4)	0 (0-2)	0,320
SJC28, median (IQR)	0 (0-2)	0 (0-1)	0,599	0 (0-4)	0 (0-2)	0,246
ESR, median (IQR) (mm/hr)	19 (11-32)	16 (9-26)	0,936	16 (10-27)	13 (8-25)	0,681
CRP, median (IQR) (mg/dL)	0,51 (0,22-1.09)	0,37 (0,15-0,97)	0,684	0,67 (0,15-1,99)	0,59 (0,10-1,47)	0,487
DAS28 3v ESR (SD)	3,15 (0,88)	2,76 (0,93)	0,199	2,67 (1,28)	2,37 (0,80)	0,589
DAS28 3v CRP (SD)	2,65 (0.94)	2,24 (0,93)	0,151	2,46 (1,05)	2.14 (0,78)	0,534
PGA (0-10) (SD)	4,8 (2,3)	2,9 (2,2)	**0,004**	5,7 (2,5)	3,0 (2,1)	**0,049**
Pain VAS (0-10) (SD)	4,9 (2,8)	2,1 (2,0)	**<0,001**	4,7 (4,1)	3,3 (2,6)	0,055
HAQ score	1,669 (0,756)	0,670 (0,559)	**<0,001**	1,458 (0,617)	0,762 (0,606)	0,217
EQ-5D score	0,2063 (0,3470)	0,6194 (0,2159)	**<0,001**	0,2717 (0,1350)	0,6466 (0,3061)	0,052
Radiographic Erosions, n (%)	15 (71,4%)	10 (20,4%)	**<0,001**	5 (71,4%)	6 (17,7%)	**0,002**
Extraarticular disease, n (%)	12 (57,1%)	12 (24,5%)	**0,011**	n/a	n/a	
Multimorbidity*, n (%)	14 (71,4%)	17 (34,7%)	**0,005**	7 (100%)	19 (54,3%)	**0,021**
Treatment						
PDN daily dose ≥ 5mg, n (%)	10 (47,6%)	18 (36,7%)	0,239	2 (28,6%)	6 (17,1%)	0,098
csDMARD, n (%)	18 (85,7%)	43 (87,5%)	0,898	6 (85,7%)	32 (91,4%)	0,690
bDMARD, n (%)	5 (23,8%)	9 (18,3%)	0,461	3 (42,9%)	8 (22,9%)	0,086

*Multimorbidity was defined has ≥ 2 chronic medical conditions
ACPA: anti-citrullinated peptide antibodies; bDMARD: biologic disease-modifying anti-rheumatic drugs; CRP: C-reactive protein; csDMARD: conventional synthetic disease-modifying anti-rheumatic drugs; DAS28: Disease Activity Score 28; ESR: erythrocyte sedimentation rate; HAQ: Health Assessment Questionnaire; IQR: Interquartile range; n: number PDN: Prednisolone; PGA: Patient Global Assessment; RF: rheumatoid factor; SD: standard deviation; SJC28: swollen joint count 28; TJC28: tender joint count 28; VAS: Visual Analogue Scale;

(39,3%) as robust (22 RA and 22 PsA). High frailty risk was linked to older age, longer disease duration, higher PGA score, multimorbidity, and the presence of erosions in both RA and PsA. In RA, there was also an association with lower body mass index (BMI), extra-articular disease, osteoporosis, lower function, and lower quality of life. No significant differences were found regarding tender and swollen joints, disease activity, acute phase reactants, and the use of b/tsDMARDs.
Conclusion: Frailty risk is prevalent in our cohort of RA and PsA patients, particularly among older patients with longer disease duration, higher PGA, multimorbidity, and erosive disease. This risk is also higher in RA patients with lower BMI and extra-articular disease, significantly impacting function and quality of life. Interestingly, traditional disease activity scores did not correlate with frailty risk, suggesting that frailty primarily reflects cumulative disease damage rather than current disease activity levels.

103 - PULMONARY INVOLVEMENT IN PRIMARY SJÖGREN'S SYNDROME

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Introduction: Primary Sjögren's syndrome (pSS) is a chronic, immune-mediated disease mainly characterized by inflammation of the exocrine glands. It can present with several extra-glandular manifestations, among which pulmonary involvement is included.
Objectives: We aim to estimate the frequency and type of pulmonary involvement in pSS, identify differences between patients with and without pulmonary involvement and characterize patients with pSS-interstitial lung disease (ILD).
Methods: We conducted a retrospective study in a cohort of patients with clinical diagnosis of pSS, followed at Hospital Garcia de Orta, and registered in Reuma.pt. Parametric and nonparametric tests were applied, and the results were considered statically significant for a p-value<0.05.
Results: We analyzed 198 patients. From these, 19 (9.6%) had lung involvement. ILD was reported in eleven patients (5.6%) and bronchiectasis in eight patients (4%). One patient with bronchiectasis had concomitant follicular bronchiolitis. Patients with pulmonary involvement were older (63.2 ± 9.3 years vs. 55.2 ± 13.3 years, p-value 0.01), had a longer pSS duration (8.69 [4.75-13] years vs. 5.5 [IQR 2-9] years, p-value 0.023) and were more likely to be smokers (21.1% vs. 11.2%, p-value 0.05).
In 36.3% of ILD patients, the diagnosis of lung disease preceded the diagnosis of pSS. Nonspecific interstitial pneumonia was the most prevalent ILD pattern (63.7%), followed by lymphocytic interstitial pneumonia (36.4%). Immunosuppressive drugs were used in six (54.5%) ILD patients, with antifibrotic being associated in half of them.
Conclusions: Pulmonary involvement was reported in 9.6% of patients in our cohort, with ILD accounting for the most frequent presentation, followed by bronchiectasis. An older age, a longer pSS duration and smoking seem to be associated with the development of lung disease.

104 - PERSISTENCE RATE OF ONCE-WEEKLY ADALIMUMAB IN RHEUMATOID ARTHRITIS, SPONDYLARTHRITIS AND PSORIATIC ARTHRITIS: REAL-WORLD EVIDENCE

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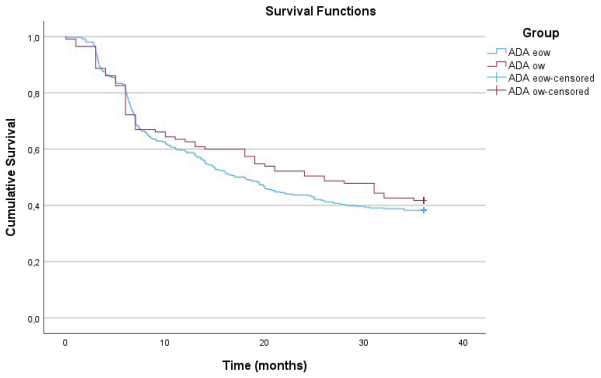
Introduction: Adalimumab (ADA) at the dose of 40mg once-weekly (ow) has on-label indications for the treatment of Rheumatoid Arthritis (RA), Psoriasis, Crohn's disease, Ulcerative Colitis and Suppurative Hidradenitis. The Summary of Product Characteristics mentions that, in the case of decrease in clinical response in monotherapy, RA patients may benefit from dose escalation to 40mg ow, based on a double blind, placebo controlled, phase III clinical trial. The choice between switching or maintaining a bDMARD has clinical and economic implications. Immunogenicity plays a key role in drug survival and lack of efficacy: dose increment or shortening the dosing interval could be a strategy to reduce immunogenicity, by potentially increasing the drug molecules/antidrug antibody ratio. Dose escalation might be a good alternative to bDMARD switching in patients with insufficient response to ADA 40mg every other week (eow).
Objective: Provide real-world evidence on the efficacy and safety of ADA 40mg ow in patients with RA, Spondylarthritis (SpA), and Psoriatic Arthritis (PsoA).
Methods: We performed a retrospective observation-



TO 104 – Figure 1. Drug survival of Adalimumab every other week (eow) and once weekly (ow) at 36 months of follow-up in RA, PsA and PsoA

al study, using data collected prospectively from The Rheumatic Diseases Portuguese Registry (Reuma.pt). We included adult patients followed at the Rheumatology Department of ULS São João, diagnosed with SpA, RA or PsoA, who initiated ADA between January 2008 and June 2021, with a minimum of 3 years of follow-up. To investigate the effectiveness and safety of dose escalation of ADA to 40mg ow, we measured the persistence till discontinuation for any reason (persistence rate, PR) within a period of 3 years of treatment in both the eow and ow groups, using the Kaplan-Meier Estimator. Then we compared the PR of both groups using the Chi-Square test. Reasons for treatment discontinuation were summarized using descriptive statistics.

Results: We included 368 participants. Since most patients that were, at some point, treated with ADA ow, were initially prescribed ADA eow, these were included in both treatment groups, and the corresponding data was attributed according to the date of dose escalation. In this way, the ADA eow group counted 366 patients, and the ADA ow group 115 patients. The PR in the eow group was 38.3%, with a mean time on treatment of 20.24 ± 0.72 months. The PR in the ow group was 41.7%, with a median time on treatment of 21.91 ± 1.31 months (Figure 1). The difference between the groups was not statistically significant (p-value 0.458). Additionally, we analysed the reasons for treatment discontinuation in the ow group: the most frequent were primary and secondary failure, both accounting for 27.3% (18/66), followed by sustained remission with 24.2% (16/66). In the latter case, all participants continued ADA but increased the dosing interval, most often to 40mg eow (81.3% [13/16]). The remaining reasons were adverse events (8/66), surgery (2/66), patient's decision (2/66), pregnancy (1/66) and loss of follow-up (1/66).

Conclusion: The PR was slightly higher in ADA 40mg ow group compared to eow, but the difference was not statistically significant. About half of patients that discontinued treatment within 3 years in the ow group did so due to therapeutic failure (primary or secondary). At the 3-years follow-up, 41.7% were still on ADA ow and about a quarter of patients who discontinued this dosage where still on ADA, but at a different dosage, due to sustained remission. This suggests that shortening the dosing interval is an effective strategy, increasing the retention time of the drug.

108 - HEALTH-RELATED QUALITY OF LIFE - A MULTIDISCIPLINARY APPROACH IN PSORIATIC DISEASE

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Introduction: Psoriatic Disease (PsD) is a chronic disease that affects the musculoskeletal system, skin, and nails with a negative impact on a patient´s quality of life. Our study aimed to analyze the HRQoL in PsD patients and to identify influencing factors.

Methods: Retrospective cross-sectional study including consecutive adult patients with PsD, followed in a combined dermatology-rheumatology Clinic, between September 2018 and April 2024. Demographic and clinical data were collected. A DLQI (Dermatology Life Quality Index) or PsAQoL (Psoriatic Arthritis Quality of Life) score greater than 10 indicates poor quality of life. Patients were grouped based on their quality-of-life scores. Descriptive and statistical analysis were performed, as appropriate, and a p-value ≤ 0.05 was considered statistically significant.

Results: Fifty-eight PsD patients were included (table 1). Of the 47 patients who completed both the DLQI and PsAQoL questionnaires, 21.3% had scores > 10 on both, while 19.1% had only a PsAQoL >10 and 14.9% had only DLQI >10. Patients with both DLQI and PsAQoL >10 had a higher Health Assessment Questionnaire (p = 0.008), Patient Pain Assessment (p = 0.041), and Patient Global Assessment (p = 0.004). An

TO 108 – TABLE 1. Clinical and demographic patients characteristics (n=58)

Age at first appointment - mean ± SD	52.98 ± 10.71
Age at onset of PsO symptoms - mean ± SD	32.21 ± 13.96
Age at onset of joint symptoms - mean ± SD	40.10 ± 12.13
Sex (M/F) - n	(30/28)
Education - n (%)	
First cycle of basic education	7 (21.9)
Second cycle of basic education	8 (25.0)
Third cycle of basic education	2 (6.3)
Secondary education	7 (21.9)
Higher education	8 (25.0)
Employment status - n (%)	
Full-time employed	26 (53.1)
Part-time employed	0 (0)
Housekeeper	6 (12.2)
Unemployed	8 (16.3)
Retired	5 (10.2)
Retired due to disability	3 (6.1)
Medical leave of absence > 1 month	1 (2.0)
Student	0 (0)
Type of clinical pattern - n (%)	
Asymmetric oligoarthritis	16 (36.4)
Symmetric polyarthritis	20 (45.5)
Predominant distal interphalangeal joint	2 (4.5)
Mutilans arthritis	0 (0)
Predominant axial involvement	6 (13.6)
Dactylitis (ever) - n (%)	20 (34.5)
Enthesitis (ever) - n (%)	9 (15.5)
Uveitis (ever) - n (%)	1 (1.7)
IBD - n (%)	0 (0)
Axial involvement - n (%)	21 (36.2)
Comorbidities - n (%)	
Arterial Hypertension	16 (27.6)
Dyslipidemia	19 (32.8)
Diabetes Mellitus	4 (6.9)
Obesity	27 (46.6)
Depression	12 (20.7)
Current smoking - n (%)	4 (6.9)
Current regular alcohol consumption - n (%)	8 (16.0)
DLQI - median (IQR)	6.0 (11.0)
PsAQoL - median (IQR)	9.00 (11.0)
HAQ - mean ± SD	0.81 ± 0.64
Patient pain assessment - mean ± SD	52.37 ± 21.10
Patient global assessment - median (IQR)	45.00 (38.00)
Rheumatologist global assessment - mean ± SD	29.76 ± 21.27
Dermatologist global assessment - mean ± SD	24.36 ± 22.11
PASI - median (IQR)	3.4 (9.7)

continues on the next column

TO 108 – TABLE 1. Continuation

ESR - median (IQR)	13.0 (9.0)
CRP - median (IQR)	2.72 (1.40)
Tender joints, total - median (IQR)	1.0 (2.0)
Tender joints, DAS28- median (IQR)	0.0 (1.0)
Swollen joints, total- median (IQR)	0.0 (2.0)
Swollen joints, DAS28- median (IQR)	0.0 (1.0)
Composite indices	
DAS28-CRP, 4 variables- median (IQR)	2.39 (2.15)
DAS28-CRP, 3 variables - median (IQR)	2.01 (1.86)
DAS28-ESR, 4 variables - mean ± SD	3.19 ± 1.46
DAS28-ESR, 3 variables - median (IQR)	2.54 (1.96)
Disease activity (DAS28-ESR, 3 variables) - n (%)	
Remission	17 (51.5)
Low disease activity	5 (15.2)
Moderate disease activity	10 (30.3)
High disease activity	3 (3.0)
Current treatment - n (%)	
Topical	29 (50.9)
cDMARD	30 (52.6)
bDMARD	17 (29.8)
Oral retinoid	6 (10.3)
PUVA therapy	0 (0)

bDMARD - biologic disease-modifying anti-rheumatic drug; cDMARD- conventional disease-modifying anti-rheumatic drug; CRP- C-reactive protein; DAS28 – Disease activity score 28 joints; DLQI - Dermatology Life Quality Index; ESR- Erythrocyte Sedimentation Rate; HAQ – Health Assessment Questionnaire; IBD – Inflammatory Bowel Disease; PASI – Psoriasis Area and Severity Index; PsAQoL - Psoriatic Arthritis Quality of Life; PsO – Psoriasis; PUVA – Psoralen and ultraviolet A.

association was observed between the absence of bDMARD treatment and both scores elevated (p = 0.042). Patients with only PsAQoL >10 were older (p= 0.011). Fewer patients were in remission, p= 0.012, had more tender and swollen joints and higher DAS28-ESR and DAS28-CRP 3 variables, p < 0.05. Rheumatologist global assessment was also higher (p = 0.002). No associations were found in patients with only DLQI > 10. **Conclusions:** PsAQoL is linked to articular disease activity and global disease assessment by the Rheumatologist. Patients with both high PsAQoL and DLQI reported worse reported outcomes, but no association was found with disease activity or characteristics, underscoring HRQoL's multidimensional nature, requiring a holistic approach.

109 - PERSISTENCE RATE OF ONCE-WEEKLY ADALIMUMAB IN SPONDYLARTHRITIS: REAL-WORLD EVIDENCE

Mariana Rocha Sebastião[1, 2], Inês Almeida[2, 3], Carlos

Marques-Gomes[2, 4], Mariana Diz-Lopes [2, 4], Miguel Correia Natal[2], Bárbara Fernandes Esteves[2], Teresa Martins-Rocha[2, 4], Miguel Bernardes[2, 4], Lúcia Costa[2]
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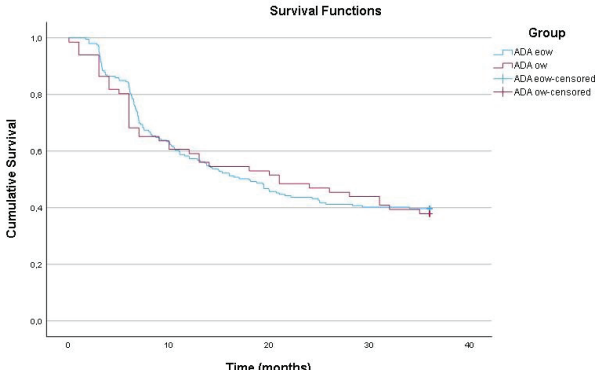
Introduction: Adalimumab (ADA) at the dose of 40mg once-weekly (ow) has on-label indications for the treatment of Rheumatoid Arthritis (RA), Psoriasis, Crohn's disease, Ulcerative Colitis and Suppurative Hidradenitis. The Summary of Product Characteristics mentions that, in the case of decrease in clinical response in monotherapy, RA patients may benefit from dose escalation to 40mg ow, based on a double blind, placebo controlled, phase III clinical trial. The choice between switching or maintaining a bDMARD has clinical and economic implications. Immunogenicity plays a key role in drug survival and lack of efficacy: dose increment or shortening the dosing interval could be a strategy to reduce immunogenicity, by potentially increasing the drug molecules/antidrug antibody ratio. Dose escalation might be a good alternative to bDMARD switching in patients with insufficient response to ADA 40mg every other week (eow).
Objective: Provide real-world evidence on the efficacy and safety of ADA 40mg ow in patients with Spondylarthritis (SpA).
Methods: We performed a retrospective observational study, using data collected prospectively from The Rheumatic Diseases Portuguese Registry (Reuma.pt). We included adult patients followed at the Rheumatology Department of ULS São João, diagnosed with SpA, who initiated ADA between January 2008 and June 2021, with a minimum of 3 years of follow-up. To investigate the effectiveness and safety of dose escalation of ADA to 40mg ow, we measured the persistence till discontinuation for any reason (persistence rate, PR) within a period of 3 years of treatment in both the eow and ow groups, using the Kaplan-Meier Estimator. Then we compared the PR of both groups using the Chi-Square test. Reasons for treatment discontinuation were summarized using descriptive statistics.
Results: We included 201 participants with SpA. Since all patients that were, at some point, treated with ADA ow, were initially prescribed ADA eow, these were included in both treatment groups, and the corresponding data was attributed according to the date of dose escalation. In this way, the ADA eow group counted 199 patients, and the ADA ow group 66 patients. The PR in the eow group was 39.7%, with a mean time on



TO 109 – Figure 1. Drug survival with Adalimumab every other week (eow) and once weekly (ow) at 36 months of follow-up in SpA

treatment of 20.36 ± 0.98 months. The PR in the ow group was 37.9%, with a mean time on treatment of 20.68 ± 1.76 months (Figure 1). The difference between the groups was not statistically significant (p-value 0.811). Additionally, we analysed the reasons for treatment discontinuation in the ow group: the most frequent were primary and secondary failure, accounting for 32,5% (13/40) and 30,0% (12/40) respectively, followed by sustained remission with 20,0% (8/40). In the latter case, all participants continued ADA but increased the dosing interval, most often to 40mg eow (75% [6/8]). The remaining reasons were adverse events (4/40), surgery (1/40), patient's decision (1/40) and pregnancy (1/40).
Conclusion: The PR was similar between the eow and the ow groups. About 60% of the patients that discontinued treatment within 3 years in the ow group did so due to therapeutic failure (primary or secondary). At the 3-years follow-up, 37.9% were still on ADA ow and about a fifth of patients who discontinued this dosage where still on ADA, but at a different dosage, due to sustained remission. This suggests that shortening the dosing interval is an effective strategy, increasing the retention time of the drug.

112 - OFF-LABEL USE OF TOCILIZUMAB IN SYSTEMIC SCLEROSIS: CHARACTERISATION OF A CLINICAL CASE SERIES

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Introduction: Systemic sclerosis (SSc) is a rheumatic

TO 112 – TABELA 1 – Características demográficas, clínicas e analíticas na baseline

Idade, anos	46.6 (18.1)
Sexo	
Masculino	3 (25%)
Feminino	9 (75%)
Raça / Etnia	
Branco de origem europeia	10 (83.3%)
Branco de origem não-europeia	1 (8.3%)
Negro	1 (8.3%)
Hábitos tabágicos	4 (33.3%)
Hábitos alcoólicos	0 (0%)
Comorbilidades	
Hipertensão arterial	5 (41.7%)
Diabetes mellitus	1 (8.3%)
Dislipidémia	5 (41.7%)
Duração da ES, meses	
Mediana (IIQ)	25.1 (18-85.6)
Fenótipo cutâneo de ES	
Difuso	10 (83.3%)
Limitado	2 (16.7%)
Anticorpos positivos	
Anti-topoisomerase I	6 (50%)
Anti-centrómero	2 (16.7%)
Anti-Ku	1 (8.3%)
Anti-U3-RNP	3 (25%)
Anti-nucleares	11 (91.7%)
Manifestações clínicas cumulativas	
Fenómeno de Raynaud	11 (91.7%)
Espessamento cutâneo	12 (100%)
Telangiectasias	5 (41.7%)
Puffy fingers	10 (83.3%)
Esclerodactilia	12 (100%)
Úlceras digitais	7 (58.3%)
Pittings scars	8 (66.7%)
Calcinose	5 (41.7%)
Contraturas	4 (33.3%)
Fricções tendinosas	2 (16.7%)
Artralgias	5 (41.7%)
Miosite	1 (8.3%)
Microstomia	4 (33.3%)
Envolvimento gastrointestinal	10 (83.3%)
Envolvimento cardíaco	5 (41.7%)
Doença intersticial pulmonar	3 (25%)
Provas de função respiratória	
CPT, % prevista, n=9	98.8 (14)
CVF, % prevista, n=11	83.4 (23.6)
DLCO, % prevista, n=10	74 (21.4)
mRSS, n=11	21.9 (12)
Parâmetros analíticos	
Hemoglobina, g/dL	12.5 (1.3)
Leucócitos, x109/L	8.2 (2.5)
Plaquetas, x109/L	300.3 (116)
PCR, mg/L	
Mediana (IIQ)	0.4 (0.1-1.1)
VS, mm/h	53.3 (20.4)
AST, U/L	21.3 (5.3)
ALT, U/L	17 (4.6)
Creatinina, mg/dL, n=11	0.6 (0.2)
Colesterol total, mg/dL, n=11	171.9 (41.3)
HDL, mg/dL, n=11	46.7 (12.6)
LDL, mg/dL, n=11	108.6 (33.9)
Triglicéridos, mg/dL	115.5 (45.9)
CK, U/L	115.8 (56.7)

Os dados são n (%), média (DP), salvo indicação em contrário. Os valores de n especificados surgem em variáveis com valores em falta. ES = esclerose sistémica. CPT = capacidade pulmonar total. CVF = capacidade vital forçada. DLCO = capacidade de difusão do monóxido de carbono. mRSS = score cutâneo de Rodnan modificado. PCR = proteína C reativa. VS = velocidade de sedimentação. AST = aspartato-aminotransferase. ALT = alanina-aminotransferase. HDL = lipoproteína de alta densidade. LDL = lipoproteína de baixa densidade. CK = creatinoquinase. IIQ = intervalo interquartil. DP = desvio-padrão.

disease associated with high morbidity and mortality, with interstitial lung disease being the main cause of death in these patients. The therapeutic options available are limited. Clinical trials have shown the effectiveness of Tocilizumab (TCZ), an interleukin-6 receptor antagonist, on lung function compared to placebo, despite it not being approved for this purpose. Its off-label use has been advocated for stabilizing lung function in diffuse cutaneous SSc.

Objectives: To characterize patients with SSc treated with off-label TCZ, identify the reasons for its prescription, and assess its safety and effectiveness.

Methods: We performed a retrospective observational cohort study, including all SSc patients treated with TCZ at our center. A descriptive analysis of the population was conducted, including the reasons for therapy prescription, discontinuation and the reported adverse events. Effectiveness was assessed through variation in the modified Rodnan skin score (mRSS), pulmonary function tests, hemoglobin levels and erythrocyte sedimentation rate (ESR) from baseline to 12 months of therapy.

Results: Twelve patients were included, 75% female, with a mean age of 47 ± 18 years. Population characterization is shown on table 1. The off-label use of TCZ was reported in 7.7% of all SSc patients. The main reasons for its prescription were cutaneous and pulmonary manifestations, predominantly in patients with diffuse cutaneous SSc (83.3%). We found a statistically significant reduction in mRSS from 21.2 to 17.6 ($p = 0.038$) and in ESR ($p < 0.01$) at 12 months. Reported adverse effects were not considered severe.

Conclusion: The off-label use of TCZ is a possible therapeutic option for severe SSc patients and/or when other immunosuppressive therapies fail, particularly useful for skin involvement, with a significant improvement in mRSS at 12 months. The safety profile was considered acceptable.

113 - PERSISTENCE RATE OF ONCE-WEEKLY ADALIMUMAB IN RHEUMATOID ARTHRITIS: REAL-WORLD EVIDENCE

Inês Almeida[1, 2], Mariana Rocha Sebastião[2, 3], Carlos Marques-Gomes[2, 4], Mariana Diz-Lopes [2, 4], Miguel Correia Natal[2], Bárbara Fernandes Esteves[2], Teresa Martins-Rocha[2, 4], Miguel Bernardes[2, 4], Lúcia Costa[2]

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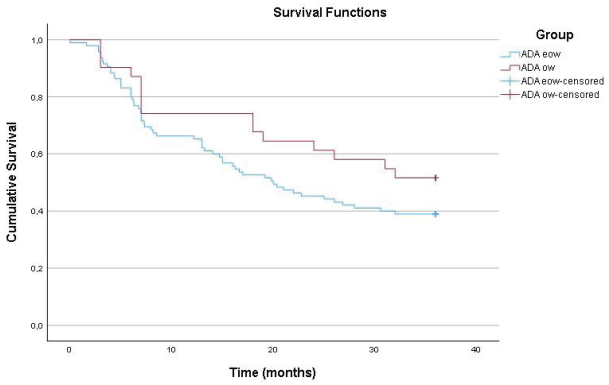
Introduction: Adalimumab (ADA) at the dose of 40mg once-weekly (ow) has on-label indications for the treatment of Rheumatoid Arthritis (RA), Psoriasis, Crohn's disease, Ulcerative Colitis and Suppurative Hidradenitis. The Summary of Product Characteristics mentions that, in the case of decrease in clinical response in monotherapy, RA patients may benefit from dose escalation to 40mg ow, based on a double blind, placebo controlled, phase III clinical trial. The choice between switching or maintaining a bDMARD has clinical and economic implications. Immunogenicity plays a key role in drug survival and lack of efficacy: dose increment or shortening the dosing interval could be a strategy to reduce immunogenicity, by potentially increasing the drug molecules/antidrug antibody ratio. Dose escalation might be a good alternative to bDMARD switching in patients with insufficient response to ADA 40mg every other week (eow).
Objective: Provide real-world evidence on the efficacy and safety of ADA 40mg ow in patients with RA.
Methods: We performed a retrospective observational study, using data collected prospectively from The Rheumatic Diseases Portuguese Registry (Reuma.pt). We included adult patients followed at the Rheumatology Department of ULS São João, diagnosed with RA, who initiated ADA between January 2008 and June 2021, with a minimum of 3 years of follow-up. To investigate the effectiveness and safety of dose escalation of ADA to 40mg ow, we measured the persistence till discontinuation for any reason (persistence rate, PR) within a period of 3 years of treatment in both the eow and ow groups, using the Kaplan-Meier Estimator. Then we compared de PR of both groups using the Chi-Square test. Reasons for treatment discontinuation were summarized using descriptive statistics.
Results: We included 95 participants with RA. Since all patients that were, at some point, treated with ADA ow, were initially prescribed ADA eow, these were included in both treatment groups, and the corresponding data was attributed according to the date of dose escalation. In this way, the ADA eow group counted 95 patients, and the ADA ow group 31 patients. The PR in the eow group was 38.9%, with a mean time on treatment of 21.06 ± 1.39 months. The PR in the ow group was 51,6%, with a mean time on treatment of 25.39 ± 2.34 months (Figure 1). The difference between groups was not statistically significant (p-value 0.174). Additionally, we analysed the reasons for treatment discontinuation in the ow group: the most frequent were secondary failure and sustained remission, both accounting for 26.7% (4/15). In the latter case, all participants continued ADA but increased the dosing interval to 40mg eow. The remaining reasons were adverse events (3/15), primary failure (2/15), surgery (1/15) and patient's decision (1/15).



TO 113 – Figure 1. Drug survival with Adalimumab every other week (eow) and once weekly (ow) at 36 months of follow-up in RA

Conclusion: The PR was higher in ADA 40mg ow group, but the difference was not statistically significant. About 40% of patients that discontinued treatment within 3 years in the ow group did so due to therapeutic failure (primary or secondary). At the 3-years follow-up, 51.6% were still on ow ADA and about a quarter of patients who discontinued this dosage where still on ADA, but at a different dosage, due to sustained remission. This suggests that shortening the dosing interval is an effective strategy, increasing the retention time of the drug.

114 - PERSISTENCE RATE OF ONCE-WEEKLY ADALIMUMAB IN PSORIATIC ARTHRITIS: REAL-WORLD EVIDENCE

Mariana Rocha Sebastião[1, 2], Inês Almeida[2, 3], Mariana Diz-Lopes [2, 4], Carlos Marques-Gomes[2, 4], Miguel Correia Natal[2], Bárbara Fernandes Esteves[2], Teresa Martins-Rocha[2, 4], Miguel Bernardes[2, 4], Lúcia Costa[2]
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Introduction: Adalimumab (ADA) at the dose of 40mg once-weekly (ow) has on-label indications for the treatment of Rheumatoid Arthritis (RA), Psoriasis, Crohn's disease, Ulcerative Colitis and Suppurative Hidradenitis. The Summary of Product Characteristics mentions that, in the case of decrease in clinical response in monotherapy, RA patients may benefit from dose escalation to 40mg ow, based on a double blind, placebo controlled, phase III clinical trial.
The choice between switching or maintaining a bD-

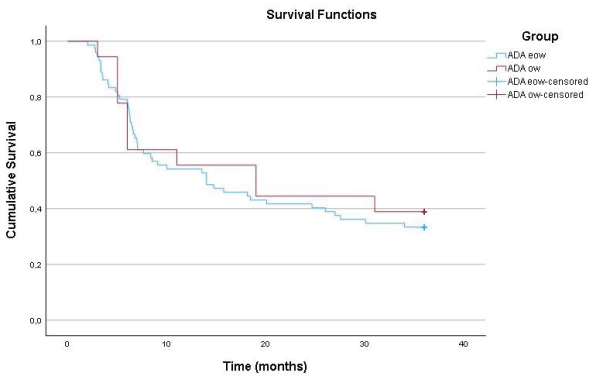
MARD has clinical and economic implications. Immunogenicity plays a key role in drug survival and lack of efficacy: dose increment or shortening the dosing interval could be a strategy to reduce immunogenicity, by potentially increasing the drug molecules/antidrug antibody ratio. Dose escalation might be a good alternative to bDMARD switching in patients with insufficient response to ADA 40mg every other week (eow).

Objective: Provide real-world evidence on the efficacy and safety of ADA 40mg ow in patients with Psoriatic Arthritis (PsoA).

Methods: We performed a retrospective observational study, using data collected prospectively from The Rheumatic Diseases Portuguese Registry (Reuma.pt). We included adult patients followed at the Rheumatology Department of ULS São João, diagnosed with PsoA, who initiated ADA between January 2008 and June 2021, with a minimum of 3 years of follow-up. To investigate the effectiveness and safety of dose escalation of ADA to 40mg ow, we measured the persistence till discontinuation for any reason (persistence rate, PR) within a period of 3 years of treatment in both the eow and ow groups, using the Kaplan-Meier Estimator. Then we compared the PR of both groups using the Chi-Square test. Reasons for treatment discontinuation were summarized using descriptive statistics.

Results: We included a total of 72 participants with PsoA. Since all patients that were, at some point, treated with ADA ow, were initially prescribed ADA eow, these were included in both treatment groups, and the corresponding data was attributed according to the date of dose escalation. In this way, the ADA eow group counted 72 patients, and the ADA ow group 18 patients. The PR in the eow group was 33.3%, with a mean time on treatment of 18.86 ± 1.63 months. The PR in the ow group was 38.9%, with a mean time on treatment of 20.44 ± 3.31 months (Figure 1). The difference between groups was not statistically significant (p-value 0.694). Additionally, we analysed the reasons for treatment discontinuation in the ow group: the most frequent were sustained remission with 36.6% (4/11): all these participants continued ADA but increased the dosing interval, most often to 40mg eow (3/4). The remaining reasons were secondary failure (3/11), primary failure (2/11), adverse events (1/11), and loss of follow-up (1/11).

Conclusion: The PR was slightly higher in ADA 40mg ow group, but the difference was not statistically significant. Almost half of patients that discontinued treatment within 3 years in the ow group did so due to therapeutic failure (primary or secondary). At the 3-years follow-up, 38.9% were still on ADA ow and about a third of patients who discontinued this dosage where still on ADA, but at a different dosage, due to sustained remission. This suggests that shortening the dosing interval is an effective strategy, increasing the retention time of the drug.



TO 114 – Figure 1. Drug survival of Adalimumab every other week (eow) and once weekly (ow) at 36 months of follow-up in PsoA

115 - EFFICACY OF ONCE-WEEKLY ADALIMUMAB IN RHEUMATOID ARTHRITIS: REAL-WORLD EVIDENCE

Inês Almeida[1, 2], Mariana Rocha Sebastião[2, 3], Carlos Marques-Gomes[2, 4], Mariana Diz-Lopes [2, 4], Miguel Correia Natal[2], Bárbara Fernandes Esteves[2], Teresa Martins-Rocha[2, 4], Miguel Bernardes[2, 4], Lúcia Costa[2]

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Introduction: Adalimumab (ADA) 40mg once-weekly (ow) regimen has on-label indications in Rheumatoid Arthritis (RA), Psoriasis, Crohn's disease, Ulcerative Colitis and Suppurative Hidradenitis. The Summary of Product Characteristics mentions that, in case of decreased clinical response in monotherapy, RA patients may benefit from dose escalation to 40mg ow, based on a double blind, placebo controlled, phase III clinical trial.

The choice between switching or maintaining a bDMARD has clinical and economic implications. Immunogenicity plays a key role in drug survival: dose increment or shortening the dosing interval could be a strategy to reduce immunogenicity, by potentially increasing the drug molecules/antidrug antibody ratio. Dose escalation might be in alternative to switching in patients with insufficient response to ADA 40mg every other week (eow).

Objective: Evaluate the efficacy of ADA 40mg ow in achieving remission or low disease activity in patients

TO 115 – TABLE 1. DISEASE ACTIVITY AT BASELINE, 6, 12, 24 AND 36 MONTHS IN THE EOW AND OW REGIME

	BASELINE			6 MONTHS			12 MONTHS			24 MONTHS			36 MONTHS		
	EOW	OW	p-value	EOW	OW	p-value	EOW	OW	p-value	EOW	OW	p-value	EOW	OW	p-value
SJC, median (IQR)	5.5 (7)	4.0 (3)	**<0.001**	2.0 (2)	1.0 (2)	0.146	0.0 (2)	0.0 (3)	0.146	0.5 (2)	0.5 (2)	0.933	0.0 (2)	1.0 (3)	0.721
n available	n=151	n=50		n=126	n=35		n=80	n=28		n=42	n=22		n=23	n=13	
TJC, median (IQR)	11.5 (12)	6.0 (3)	**<0.001**	1.5 (2)	2.0 (6)	0.259	1.5 (2)	2.5 (8)	0.640	2.0 (5)	3.0 (9)	0.313	1.0 (3)	4.0 (5)	0.242
n available	n=151	n=50		n=126	n=35		n=80	n=28		n=42	n=22		n=22	n=13	
DAS28-4V, mean (SD)	4.82 (1.22)	3.83 (1.04)	**<0.001**	3.29 (1.31)	2.83 (0.70)	**0.008**	2.92 (1.20)	2.85 (0.87)	0.747	2.64 (0.96)	2.86 (0.94)	0.390	2.57 (0.93)	3.13 (0.62)	0.070
n available	n=157	n=51		n=124	n=35		n=80	n=28		n=43	n=22		n=23	n=12	
CDAI, mean (SD)	25.45 (10.71)	16.75 (8.16)	**<0.001**	12.36 (10.05)	9.34 (4.99)	**0.023**	9.35 (8.64)	7.75 (6.50)	0.411	7.01 (5.53)	9.41 (6.80)	0.154	6.91 (4.57)	8.97 (3.87)	0.207
n available	n=135	n=44		n=113	n=31		n=69	n=24		n=36	n=21		n=19	n=12	
SDAI, mean (SD)	26.97 (11.08)	18.09 (8.76)	**<0.001**	13.18 (10.57)	10.00 (4.85)	**0.019**	9.90 (8.88)	8.39 (6.40)	0.448	7.40 (5.69)	9.86 (6.80)	0.151	7.39 (4.90)	9.94 (3.72)	0.134
n available	n=135	n=44		n=109	n=31		n=69	n=24		n=35	n=21		n=19	n=12	
HAQ-DI, mean (SD)	1.52 (0.66)	1.31 (0.64)	0.068	1.13 (0.61)	1.17 (0.64)	0.719	1.15 (0.65)	0.94 (0.51)	0.123	1.09 (0.63)	1.02 (0.63)	0.665	1.10 (0.56)	1.07 (0.72)	0.919
n available	n=144	n=41		n=117	n=30		n=59	n=24		n=37	n=19		n=20	n=13	
ΔHAQ-DI, mean (SD)	-	-	-	-0.31 (0.67)	-0.11 (0.35)	**0.035**	-0.38 (0.55)	-0.19 (0.45)	0.200	-0.53 (0.61)	-0.22 (0.54)	0.096	-0.24 (0.65)	-0.13 (0.66)	0.704
n available				n=106	n=27		n=53	n=19		n=33	n=14		n=17	n=9	
DAS28-4V <2.6, % (n/N)	3.8 (6/158)	5.9 (3/51)	0.524	29.0 (24/126)	11.8 (4/34)	0.321	25.6 (20/78)	11.1 (3/27)	0.116	33.3 (14/42)	9.1 (2/22)	**0.033**	34.8 (8/23)	8.3 (1/12)	0.089
CDAI ≤2.8, % (n/N)	0.7 (1/135)	2.3 (1/44)	0.401	17.7 (20/113)	9.7 (3/31)	0.280	23.2 (16/69)	16.7 (4/24)	0.503	30.6 (11/36)	9.5 (2/21)	0.068	21.1 (4/19)	16.7 (2/12)	0.763
SDAI ≤3.3, % (n/N)	0.7 (1/135)	2.3 (1/44)	0.401	15.6 (17/109)	6.5 (2/31)	0.190	23.2 (16/69)	16.7 (4/24)	0.503	31.4 (11/35)	14.3 (3/21)	0.151	26.3 (5/19)	8.3 (1/12)	0.217

CDAI: Clinical Disease Activity Index; DAS28-4V: Four Variable Disease Activity Score based on 28-joint count; HAQ-DI: Health Assessment Questionnaire Disability Index; SDAI: Simple Disease Activity Index; SJC: Swollen joint count; TJC: Tender joint count

with RA.

Methods: We performed a retrospective observational study, using data from Reuma.pt. We included adult patients followed at the Rheumatology Department of ULS São João, diagnosed with RA, who initiated ADA between 2008 and 2023, with a minimum 6 months of follow-up. Descriptive analysis of continuous variables was reported as mean and standard deviation or median and interquartile range based on normality. Descriptive analysis of categorical variables was presented as frequency or proportion with confidence intervals at 95% (95% CI). We considered p-value<0.05 significant.

Results: We included a total of 166 patients with RA. Since all patients that were, at some point, treated with ADA ow, were initially prescribed ADA eow, these were included in both treatment groups, and the corresponding data was attributed according to the date of dose escalation. In this way, the ADA eow group counted 166 patients (median age 54.95, IQR 15,47 years, 15,7% male) and the ADA ow group 56 patients (median age 55.25, IQR 11.24 years, 14,3% male). At baseline, the eow group had higher scores in the number of swollen and tender joints, DAS28-4V, CDAI and SDAI (p<0.001, table 1). At 6 months of follow-up, DAS28-4V, CDAI, and SDAI remained higher in the eow group (p=0.008, 0.023, 0.019). However, at 12, 24, and 36 months, there were no statistically significant differences in these scores between the groups. The HAQ-DI variation was greater in the eow group at 6 months (p=0.035), but this was not observed in subsequent evaluations. The proportion of patients in remission according to DAS28-4V, CDAI, and SDAI did not differ significantly between the groups, except for DAS28-4V at 24 months, which favoured the eow group (33.3% vs. 9.1%).

Discussion: At baseline and after 6 months of follow-up, patients on ADA eow had higher disease activity scores than patients on ADA ow. This might be because patients on ADA ow were initially in the eow regimen and were expected to experience some, albeit insufficient, clinical response. At 12, 24, and 36 months, there were no statistically significant and consistent differences in the disease activity scores or the proportion of patients in clinical remission.

Conclusion: The similar outcomes in disease activity scores between eow and ow regimens suggest that, in RA, shortening the dosing interval of ADA might be an effective alternative strategy to bDMARD switching.

116 - EFFICACY OF ONCE-WEEKLY ADALIMUMAB IN SPONDYLARTHRITIS: REAL-WORLD EVIDENCE

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Introduction: Adalimumab (ADA) 40mg once-weekly (ow) regimen has on-label indications in Rheumatoid Arthritis (RA), Psoriasis, Crohn's disease, Ulcerative Colitis and Suppurative Hidradenitis. The Summary of Product Characteristics mentions that, in case of decreased clinical response in monotherapy, RA patients may benefit from dose escalation to 40mg ow, based on a double blind, placebo controlled, phase III clinical trial. The choice between switching or maintaining a bDMARD has clinical and economic implications. Immunogenicity plays a key role in drug survival: dose increment or shortening the dosing interval could be a strategy to reduce immunogenicity, by potentially increasing the drug molecules/antidrug antibody ratio. Dose escalation might be an alternative to switching in patients with insufficient response to ADA 40mg every other week (eow).
Objective: Evaluate the efficacy of ADA 40mg ow in achieving remission or low disease activity in patients with SpA.
Methods: We performed a retrospective observational study, using data from Reuma.pt. We included adult patients followed at the Rheumatology Department of ULS São João, diagnosed with SpA, who initiated ADA between 2008 and 2023, with a minimum 6 months of follow-up. Descriptive analysis of continuous variables was reported as mean and standard deviation or median and interquartile range based on normality. Descriptive analysis of categorical variables was presented as frequency or proportion with confidence intervals at 95% (95% CI). We considered p-value<0.05 significant.
Results: We included a total of 270 patients. Most patients treated with ADA ow, were initially prescribed ADA eow: as such, we included them in both treatment groups, and the corresponding data was attributed according to the date of dose escalation. Ergo, the ADA eow group counted 268 patients (median age 46.09, IQR 18.66 years, 45.9% male) and the ADA ow group 142 patients (median age 45.99, IQR 13.54 years, 33.9% male). At baseline, the eow group had higher scores of BASDAI (p<0.001), BASFI (p=0.011) and ASDAS-CRP (p<0.001, table 1), but there were no statistically significant differences in the proportion of patients with inactive disease or low disease activity by ASDAS-CRP. The eow group showed higher BASDAI scores at 12, 24 and 36 months (p<0.001; 0.003; <0.001), but this variance of BASFI was only observed at 12 months (p=0.049). At 6, 12 and 24 months, the ADA eow group revealed greater frequency of inactive disease by ASDAS-CRP (p=0.047; <0.001; <0.001), but this was not seen at 24 months, nor in frequency of low disease activity at any time. In contrast with the data from ASAS response and the Clinically Important Improvement by ASDAS-CRP, the eow group revealed Major Improvement more frequently throughout the follow-up period (p=0.002; <0.001; <0.001; <0.001).
Discussion: At baseline, patients on ADA eow had higher disease activity scores than patients on ADA ow. This might be because patients on ADA ow were initially in the eow regimen and were expected to experience some, albeit insufficient, clinical response. Although the results were not verified at all times and for all the scores, globally the ow group did not achieve clinical outcomes equivalent to the eow group.
Conclusion: The date suggests that, despite the inferior treatment response in ow vs eow regimen, ADA dose escalation might be an effective alternative strategy to bDMARD switching in SpA, since all the disease activity scores showed a favourable variation throughout time.

119 - MUSCULOSKELETAL MANIFESTATIONS OF CHECKPOINT INHIBITORS - EXPERIENCE FROM A TERTIARY CENTRE

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Introduction: Checkpoint inhibitors (ICIs) have changed dramatically cancer therapy in the last decade. However, their use is linked with various immune-related adverse events (irAEs), including musculoskeletal manifestations. This monocentric study aims to evaluate the onset, clinical presentation, and management of musculoskeletal symptoms in patients undergoing treatment with ICIs.
Methods: A monocentric retrospective analysis between 2013 and 2023 of patients receiving checkpoint inhibitors who were examined in the rheumatology department. 8 patients were identified (table 1). Data

TO 119 – TABLE 1.

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6	Patient 7	Patient 8
Patient Demographics								
Sex	Female	Male	Male	Female	Male	Male	Male	Male
Age starting ICI	52	61	53	74	83	61	54	87
ICI	Pembrolizumab	Nivolumab	Pembrolizumab	Pembrolizumab	Nivolumab	Pembrolizumab	Pembrolizumab	Pembrolizumab
Time starting symptoms (months)	3	60	24	7	12	6	3	3
Cancer	Colorectal	Renal	Lung	Lung	Lung	Lung	Colorectal	Lung
Clinical History								
Arthralgia	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Myalgia	No	No	No	No	No	No	Yes	No
Tenosynovitis	Yes	No	No	No	No	No	No	No
Arthritis	Yes	Yes	Yes	Yes	Yes	No	No	Yes
Extra-articular Symptoms	No	Yes	Yes	No	No	No	No	Yes
Laboratory findings								
RF	Negative	Negative	Negative	Negative	Negative	Negative	Positive	Negative
Anti-CCP	Negative	Negative	Negative	Negative	Negative	Negative	Negative	Negative
ANA	Positive	Negative	Negative	Negative	Negative	Negative	Negative	Negative
VS	Negative	Negative	Negative	Positive	Negative	Negative	Negative	Positive
CRP	Negative	Positive	Negative	Positive	Negative	Negative	Negative	Positive
Musculoskeletal Ultrasound								
Arthritis	Yes	Yes	Yes	Yes	Yes	No	No	Yes
Tenosynovitis	Yes	No	Yes	No	No	No	No	No
Bursitis	No	No	No	No	Yes	No	No	No
Erosions	No	No	Yes	No	No	No	No	No
Treatment								
NSAIDs	No	No	No	No	No	Yes	Yes	No
Intra-articular corticosteroids	No	Yes	No	No	No	No	No	No
Oral corticosteroids	Yes	Yes	Yes	Yes	Yes	No	No	Yes
C DMARD	No	No	Yes	No	No	No	No	No
B DMARD	No	No	No	No	No	No	No	No
Required stopping ICI	No	No	No	No	No	No	No	No

were collected on patient demographics, clinical history, laboratory findings, musculoskeletal ultrasound and treatment.
Results: The study included 8 patients (6 males, 2 females), with a median age of 67.12 years (52-83). The majority of patients were treated with pembrolizumab (6/8), and the rest with nivolumab. Musculoskeletal symptoms appeared at a median of 16.43 months (3-60). All patients present arthralgia, but 2 didn't had arthritis, being the knee the most frequent location. 2 patients presented with polyarthritis, 2 with oligoarthritis and 2 with monoarthritis. 1 patient presented with myalgia and 1 with tenosynovitis. 3 patients presented extra-articular manifestations (2 with psoriatic plaques, 1 with sclerodactyly). Laboratory results revealed that most patients had negative rheumatoid factor (7/8) and antinuclear antibodies (7/8). Only two patients presented elevated inflammatory markers. Imaging showed that there was no case of subclinical arthritis, with 2 cases of tenosynovitis (both digital flexors), and 1 case with articular erosion, at the second metacarphalangeal joint. Corticosteroids were the mainstay of treatment with 6/8 patients receiving. Only 1 case required c DMARD (sulfasalazine), and no case was treated with b DMARD. No patient needed to discontinue ICIs due to musculoskeletal irAEs.
Conclusion: In conclusion, musculoskeletal irAEs associated with ICIs present predominantly as arthralgia, often with arthritis, with the knee being most commonly affected. Our study highlighted diverse manifestations, including polyarthritis, oligoarthritis, monoarthritis, myalgia, and tenosynovitis. Corticosteroids proved effective in most cases for symptom management. All patients continue ICI therapy. These findings underscore the importance of recognizing and managing musculoskeletal irAEs to optimize cancer treatment outcomes, relevant for the rheumatology field.

125 - CYCLING VERSUS SWITCHING AFTER INEFFECTIVENESS OF FIRST TUMOR NECROSIS FACTOR INHIBITOR DURING FIRST YEAR OF THERAPY - DEMOGRAPHIC AND CLINICAL DIFFERENCES IN RHEUMATOID ARTHRITIS

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Introduction: Tumor necrosis factor inhibitors (TNFi) are usually used following conventional synthetic disease-modifying antirheumatic drug (csDMARD) failure in Rheumatoid Arthritis (RA), preferably combined with the latter. Patients with inadequate response to first TNFi can cycle to another TNFi or switch to therapies with a different mechanism of action (MOA) such as other biologic disease-modifying anti-rheumatic drugs (nonTNFi bDMARDs) or targeted synthetic disease-modifying anti-rheumatic drugs (tsDMARDs).
This study aimed to identify demographic or clinical differences between patients TNFi cycling or MOA switching due to ineffectiveness of their first TNFi during the first year of therapy.
Methods: This is a retrospective single-center study including adult patients with RA registered in Reuma.pt fulfilling ACR/EULAR 2010 classification criteria who changed to a second targeted therapy after failing a first TNFi during the first year of treatment. Demographic and clinical data were collected. Patients were grouped based on the class of their second targeted therapy either as TNFi cyclers or MOA switchers. Disease parameters were compared at baseline and at the last evaluation before cycling or switching therapy.
Results: Out of 745 RA patients, 28 met the study inclusion criteria. Eight patients were TNFi cyclers (28.6%) and 20 switched to a second targeted therapy with a different MOA (71.4%).
Overall, the mean disease duration was 9.8±6.5 years with mean age of 51.4±10.5 years (table 1). Most patients were females (78.66%) and Caucasians of European origin (95.7%). Arterial hypertension was significantly more frequent in MOA switchers (p=0.008), but no significant differences were found regarding tobacco use (p=0.403), alcohol use (p=0.162), obesity (p=1.000), dyslipidemia (p=0.281), diabetes mellitus (p= 1.000) or psoriasis (p=0.286). Rates of positivity for rheumatoid factor and for anti-citrullinated protein antibodies and erosive disease were similar for TNFi cyclers and MOA switchers (p=1.000). No extra-articular manifestations prevailed between groups (p=1.000). Half of TNFi cyclers received adalimumab as first TNFi and 40% of MOA switchers started with etanercept (p=0.399). Overall, 85.7% of patients were prescribed a csDMARD concomitantly with their first bDMARD. Median time on first TNFi did not differ between groups (p=0.589). Most patients cycled to etanercept (87.5%) or switched to tocilizumab (85.0%). There were no switchers to tsDMARDs.
Joint count was similar both at baseline and at the final evaluation before cycling or switching (table 2). Erythrocyte sedimentation rate (ESR) was higher in MOA switchers, reaching statistical significance at baseline

TO 125 – TABLE 1. Demographics and characteristics of patients TNFi cycling or MOA switching after inefficacy of first TNFi during the first year of therapy

	All patients (N=28)	TNFi cyclers (N=8)	MOA switchers (N=20)	p-value
Disease duration at time of first TNFi, years - mean±SD	9.8±6.5	10.0±7.0	9.7±6.4	0.914
Age at time of first TNFi, years - mean±SD	51.4±10.5	50.3±9.1	51.9±11.2	0.715
Female sex - n (%)	22 (78.6)	5 (62.5)	17 (85.0)	0.311
Caucasian of European origin* - n (%)	22 (95.7)	6 (85.7)	16 (100.0)	0.304
Body mass index*, Kg/m^2 - mean±SD	27.1±4.7	26.2 ±3.8	27.3±5.1	0.646
Tobacco use* - n (%)				0.403
Current smoker	7 (26.9)	3 (37.5)	4 (22.2)	
Never smoker	16 (61.5)	5 (62.5)	11 (61.1)	
Quit	3 (11.5)	0 (0.0)	3 (16.7)	
Alcohol use* - n (%)				0.162
Current drinker	3 (11.1)	0 (0.0)	3 (15.8)	
Never or occasional drinker	23 (85.2)	7 (87.5)	16 (84.2)	
Quit	1 (3.7)	1 (12.5)	0 (0.0)	
Comorbidities - n (%)				
Obesity*	6 (28.6)	1 (20.0)	5 (31.3)	1.000
Dyslipidemia	5 (17.9)	0 (0.0)	5 (25.0)	0.281
Diabetes *mellitus*	1 (3.6)	0 (0.0)	1 (5.0)	1.000
Arterial hypertension	12 (42.9)	0 (0.0)	12 (60.0)	**0.008**
Psoriasis	1 (3.6)	1 (12.5)	0 (0.0)	0.286

	All patients (N=28)	TNFi cyclers (N=8)	MOA switchers (N=20)	p-value
Immunological and radiographic characteristics - n (%)				
Positivity for rheumatoid factor	21 (75.0)	6 (75.0)	15 (75.0)	1.000
Positivity for anti-citrullinated protein antibodies	23 (82.1)	7 (87.5)	16 (80.0)	1.000
Erosive disease*	18 (72.0)	5 (71.4)	13 (72.2)	1.000
Extra-articular manifestations - n (%)				
Subcutaneous nodules	8 (28.6)	2 (25.0)	6 (30.0)	1.000
Rheumatoid vasculitis	1 (3.6)	0 (0.0)	1 (5.0)	1.000
TNFi at first use - n (%)				0.399
Adalimumab	9 (32.1)	4 (50.0)	5 (25.0)	
Certolizumab pegol	1 (3.6)	0 (0.0)	1 (5.0)	
Etanercept	9 (32.1)	1 (12.5)	8 (40.0)	
Golimumab	9 (32.1)	3 (37.5)	6 (30.0)	
Concomitant csDMARD with first TNFi - n (%)	24 (85.7)	7 (87.5)	17 (85.0)	1.000
Months on first TNFi at time of therapeutic change decision - median (IQR)	6.0 (5.0)	6.0 (5.0)	6.0 (5.0)	0.589
Targeted therapy at second use - n (%)				
Adalimumab	1 (3.6)	1 (12.5)	0 (0.0)	
Etanercept	7 (25.0)	7 (87.5)	0 (0.0)	
Rituximab	3 (10.7)	0 (0.0)	3 (15.0)	
Tocilizumab	17 (60.7)	0 (0.0)	17 (85.0)	

Footnote: TNFi - Tumor necrosis factor inhibitors. MOA - Mechanism of action. SD - Standard deviation. csDMARD - Conventional synthetic disease-modifying antirheumatic drug. IQR - Interquartile range. *Total of 5 missing values for ethnicity (1+4); total of 7 missing values for body mass index (3+4); total of 2 missing values for tobacco use (0+2); total of 1 missing value for alcohol use (0+1); total of 7 missing values for obesity (3+4); total of 3 missing values for erosive disease (1+2).

TO 125 – TABLE 2. Disease parameters of patients TNFi cycling or MOA switching after inefficacy of first TNFi during the first year of therapy

	Baseline evaluation				Final evaluation (before cycling or switching)			
	All patients (N=28)	TNFi cyclers (N=8)	MOA switchers (N=20)	p-value	All patients (N=28)	TNFi cyclers (N=8)	MOA switchers (N=20)	p-value
Joint count - median (IQR)								
Number of tender joints out of 28*	7.0 (11.0)	5.0 (3.0)	8.5 (14.0)	0.330	5.5 (6.0)	5.5 (3.0)	5.5 (8.0)	0.798
Number of tender joints out of 68*	10.0 (14.0)	7.0 (4.0)	12.0 (20.0)	0.126	9.5 (10.0)	11.0 (9.0)	7.0 (11.0)	0.432
Number of swollen joints out of 28*	6.0 (9.0)	5.0 (5.0)	6.5 (10.0)	0.657	5.0 (6.0)	4.5 (5.0)	5.0 (7.0)	0.608
Number of swollen joints out of 66*	6.0 (9.0)	5.0 (5.0)	7.0 (12.0)	0.899	6.5 (8.0)	5.0 (8.0)	7.0 (7.0)	0.451
ESR* (mm/h) - median (IQR)	35.0 (43.0)	14.0 (29.0)	41.5 (39.0)	**0.029**	29.0 (39.0)	10.0 (35.0)	38.0 (36.0)	0.056
CRP* (mg/dL) - median (IQR)	1.4 (1.5)	0.7 (1.3)	1.8 (1.3)	0.069	1.2 (2.3)	0.7 (2.7)	1.6 (2.1)	0.182
Patient global assessment* (mm) - mean±SD / median (IQR)	77.0±15.1	79.3±14.8	76.1±15.5	0.643	70.0 (30.0)	72.5 (26.0)	65.0 (33.0)	0.838
Evaluator global assessment* (mm) - mean±SD	63.4±17.8	68.7±19.5	61.2±17.1	0.360	49.4±28.5	52.3±25.8	48.1±30.1	0.551
Pain visual analog scale* (mm) - mean±SD	74.3±16.1	75.3±14.2	73.8±17.2	0.845	67.9±18.9	72.1±17.3	66.2±19.7	0.736
DAS28 - mean±SD / median (IQR)								
DAS28-ESR(3v)*	5.3±1.3	4.4±0.7	5.5±1.3	**0.041**	5.0 (1.5)	3.8 (1.3)	5.0 (1.4)	0.075
DAS28-ESR(4v)*	5.7±1.2	5.1±0.8	6.0±1.2	0.072	5.1 (1.6)	4.2 (1.2)	5.4 (1.4)	0.060
DAS28-CRP(3v)*	4.8±1.1	4.2±0.7	5.0±1.2	0.138	4.0 (1.4)	3.8 (0.8)	4.4 (1.4)	0.317
DAS28-CRP(4v)*	5.3±1.0	4.9±0.7	5.5±1.1	0.207	4.5 (1.7)	4.4 (0.9)	4.8 (2.0)	0.912
CDAI* - median (IQR) / mean±SD	24.5 (20.0)	25.0 (6.5)	24.0 (23.8)	0.300	23.7±10.0	21.0±5.2	24.7±11.3	0.284
SDAI* - median (IQR) / mean±SD	25.9 (19.8)	25.2 (14.5)	26.5 (23.6)	0.312	25.7±11.0	22.1±5.4	27.3±12.5	0.172
HAQ* - median (IQR) / mean±SD	2.0 (1.2)	2.0 (1.1)	2.0 (1.3)	0.977	1.8±0.5	1.7±0.5	1.8±0.6	0.543

Footnote: TNFi - Tumor necrosis factor inhibitors. MOA - Mechanism of action. IQR - Interquartile range. ESR – Erythrocyte sedimentation rate. CPR – C-reactive protein. SD - Standard deviation. DAS28 – Disease activity score. CDAI - Clinical disease activity index. SDAI - Simplified disease activity index. HAQ - Health assessment questionnaire. *Total of 1 missing value for number of tender joints out of 28; total of 6 missing values for number of tender joints out of 68; total of 1 missing value for number of swollen joints out of 28; total of 6 missing values for number of tender joints out of 66; total of 1 missing value for ESR; total of 4 missing values for CRP; total of 2 missing values for patient global assessment; total of 7 missing values for evaluator global assessment; total of 7 missing values for pain visual analog scale; total of 1 missing value for DAS28-ESR(3v); total of 2 missing values for DAS28-ESR(4v); total of 4 missing values for DAS28-CPR(3v); total of 5 missing values for DAS28-CPR(4v); total of 7 missing values for CDAI; total of 9 missing values for SDAI; total of 4 missing values for HAQ.

[median 41.5 (39.0) vs. 14.0 (29.0) mm/h, p=0.029]. Disease activity score (DAS28) tended to be higher in the MOA switching group, showing statistically significant difference at baseline evaluation with DAS28-ESR three variables (mean 5.5±1.3 vs. 4.4±0.7, p=0.041). There were no significant differences regarding C-reactive protein, patient or evaluator global assessments, pain visual analog scale, clinical disease activity index, simplified disease activity index or health assessment questionnaire.

Conclusions: Most patients received a first TNFi combined with a csDMARD, in accordance with current guidelines.

MOA switchers showed greater prevalence of arterial hypertension as well as higher titles of ESR and higher disease activity at baseline according to DAS28-ESR three variables. These findings may influence therapeutic decisions, but further investigation is needed.

126 - CHRONIC NONBACTERIAL OSTEOMYELITIS IN PEDIATRIC AGE: A CASE SERIES

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TO 126 – TABLE 1. Demographic, clinical and therapeutic characterization of patients with nonbacterial chronic osteomyelitis

Patient	1	2	3	4
Sex	Female	Female	Female	Female
Age at diagnosis (years)	10	8	1	10
Delay in diagnosis (months)	5	12	1	3
Involved bones				
Vertebra/Rib	-	-	+	+
Humerus/Ulna	-	-	+	-
Ilium/Sacrum	+	+	-	+
Femur/Tibia	-	-	+	-
Tarsus	-	+	-	-
Signs and symptoms				
Bone pain	+	+	+	+
Local swelling	-	+	+	-
Fever	-	-	+	-
Peripheral arthritis	-	-	+	-
Pustulosis	-	-	+	-
Acne	-	-	-	+
Initial treatment	NSAID	NSAID	NSAID, CS, pamidronate	NSAID
1-month response				
Clinical	+	+	-	+
Analytical	+	-	-	+
Radiographic	+	NE	-	NE
Course	Acute	Persistent	Mixed	Acute
Follow-up (months)	1	18	131	103

NSAID=non-steroidal anti-inflammatory drug; CS=corticosteroid; NE=not evaluated.

Introduction: Chronic nonbacterial osteomyelitis (CNO) is an autoinflammatory bone disease most prevalent in children that covers a wide clinical spectrum, with mild and limited single-site involvement at one end and severe, chronically active or recurrent multifocal disease at the other end. (1)

Objectives: Our study aimed to characterize the demographic features, clinical presentation and short-term response to treatment of CNO patients.

Methods: This is a retrospective single-center study including patients under 18 years of age diagnosed with CNO between 2007 and 2023. Demographic and clinical data were collected. The diagnosis of CNO was based on the history of bone pain and/or local swelling, presence of radiologic evidence of compatible lesions and bone biopsies showing sterile chronic inflammation.

Results: A total of 4 patients were included, all of whom were females. Main demographic, clinical and therapeutic characteristics are described in Table 1. The mean age at diagnosis was 7.3±4.3 years and the mean delay in diagnosis was 5.3±4.8 months.

Most patients had multifocal disease (n=3; 75%) and the pelvic girdle was the most commonly involved location (n=3; 75%). Bone pain was universally present with local swelling in 50% (n=2), mild fever in 25% (n=1) and acne in 25% (n=1). Patient 3 presented early-onset peripheral arthritis and pustulosis suggestive of a monogenic form of CNO, but no genetic cause was identified. No patients in this series had axial arthritis, enthesitis, psoriasis, uveitis or inflammatory bowel disease.

A non-steroidal anti-inflammatory drug (NSAID) was used as the first-line therapy in all the patients, with clinical improvement at the first month in 75% (n=3). Exceptionally, in case 3, NSAID was combined ad initium with a corticosteroid and pamidronate; however, no response was observed after 1 month.

The mean follow-up time was 63.3±63.5 months and no complications were reported.

Conclusion: Our case series corroborates the clinical heterogeneity of CNO. In addition to the lack of awareness for this condition, this can justify a significant diagnostic delay, although we report a lower value than other studies. (1, 2)

REFERENCES:

1. McCann L, Beresford M, Hahn G, Hedrich C. Chronic Nonbacterial Osteomyelitis. In: Efthimiou P, ed. Auto-Inflammatory Syndromes: Pathophysiology, Diagnosis, and Management. New York: Springer; 2019:227-247.
2. Girschick H, Finetti M, Orlando F, et al. The multifaceted presentation of chronic recurrent multifocal osteomyelitis: a series of 486 cases from the Eurofever international registry. Rheumatology (Oxford). 2018;57(7):1203-1211.

127 - MICROVASCULAR STATUS IN SYSTEMIC SCLEROSIS PATIENTS COMPARING LIMITED AND DIFFUSE CUTANEOUS INVOLVEMENT: A NAILFOLD VIDEOCAPILLAROSCOPY ANALYSIS

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Background: Systemic sclerosis (SSc) is a rare immune-mediated connective tissue disease characterized by microvascular damage, skin fibrosis and internal organs progressive involvement. The extent of skin fibrosis defines 2 clinical phenotypes - limited (lSSc) and diffuse (dSSc) cutaneous SSc. Nailfold videocapillaroscopy (NVC) is a non-invasive tool able to evaluate microcirculation[1]. To date, the association of NVC findings and the extent of skin involvement, is not yet clearly stablished[2-3].

Objective: The aim of our study was to compare the NVC findings between patients with lSSc and dSSc.

TO 127 – TABLE 1. Descriptive and comparative statistics of the demographic, clinical, laboratorial and capillaroscopic variables in lSSc and dSSc.

	lSSc (N=100)	dSSc (N=27)	p-value
AGE AT NVC (years) M (SD)	58.64±12.85	50.11±13.79	0.002
SEX n (%)			0.010
Women	91 (91.0)	19 (70.4)	
Men	9 (9.0)	8 (29.6)	
DISEASE DURATION (years) M (SD)	9.23±10.45	4,93±7.58	0.024
ANTIBODY PROFILE* n (%)			
Anti-centromere	57 (57.0)	1 (3.7)	<0.001
Anti-Scl70	24 (24.0)	19 (70.4)	<0.001
Only ANA positive	13 (13.0)	4 (14.8)	0.510
Other SSc-associated antibodies**	9 (9.0)	5 (18.5)	0.146
ANA negative	1 (1.0)	0 (0)	0.787
CLINICAL MANIFESTATIONS n (%)			
RP	98 (98.0)	26 (96.3)	0.515
DUs	19 (19.0)	14 (51.9)	<0.001
PAH	6 (6.0)	1 (3.7)	0.539
ILD	26 (26.0)	9 (33.3)	0.449
Esophageal involvement	25 (25.0)	13 (48.1)	0.020
MSK involvement	8 (8.0)	8 (29.6)	0.006
mRSS M (SD) (n=83)	4.69±4.36	17.16±13.08	<0.001
TREATMENT n (%) (n=81)			
Immunosuppressors$	6 (9.7)	8 (42.1)	0.003
Vasodilators#	30 (48.4)	7 (36.8)	0.377
NVC PATTERN n (%)			
Normal	2 (2.0)	0 (0)	0.619
Non-Specific Alterations	13 (13.0)	4 (14.8)	0.510
"Early" Scleroderma	32 (32.0)	2 (7.4)	0.010
"Active" Scleroderma	34 (34.0)	11 (40.7)	0.516
"Late" Scleroderma	7 (7.0)	8 (29.6)	0.004
Scleroderma-Like	12 (12.0)	2 (7.4)	0.391
SCORE M (SD)			
Score A (early NVC changes)	4.04±1.25	4.04±1.58	0.496
Capillary Dilations	1.99±0.41	2.07±0.62	0.397
Microhaemorrhages	0.91±0.57	0.81±0.74	0.296
Giant Capillaries	1.13±0.69	1.15±0.77	0.860
Score B (late NVC changes)	2.38±2.29	4.37±2.45	<0.001
Capillary Density (1 mm linear)	1.03±0.93	1.74±0.86	<0.001
Altered microvascular architecture	0.74±0.81	1.48±0.89	<0.001
Abnormal shapes (angiogenesis)	0.62±0.75	1.15±0.91	0.005

*Positivity for more than one antibody was observed; **Includes the following autoantibodies: anti-RNA polymerase III, anti-U3 RNP, anti-PmScl75, anti-PmScl100, anti-NOR90, anti-Th/To, anti-Ku and anti-SSA.; $Include methotrexate, azathioprine, cyclosporine A, mycophenolate mofetil, cyclophosphamide, and rituximab; #Include pentoxifylline, calcium-channel blockers, phosphodiesterase 5 inhibitors, iloprost, bosentan and aminaftone (Italy); M: mean; SD: standard deviation; ANA: anti-nuclear antibodies; ENA: extractable nuclear antigen antibodies; RP: Raynaud phenomenon; DUs: digital ulcers; PAH: pulmonary arterial hypertension; ILD: interstitial lung disease; MSK: musculoskeletal; mRSS: modified Rodnan Skin Score.

Methods: One hundred and fifty-seven patients were recruited at the Division of Rheumatology of the University of Genova; 127 patients with SSc and 30 patients with primary Raynaud phenomenon (pRP). The assessment focused on NVC parameters, including NVC patterns, the absolute number of capillaries (1 linear mm) and the semiquantitative scores A and B in SSc patients. The scores A (early changes: capillary dilations, microhaemorrhages and giant capillaries) and B (advanced changes: capillary density, altered microvascular architecture and abnormal capillary shapes - ramifications/angiogenesis) are graded between 0-3 in relation to the extent of each NVC change (0: no changes; 1: ≤33% changes; 2: 33-66% changes; 3: ≥66% changes)[4].

Results: The absolute number of capillaries showed a statistically significant difference comparing patients with pRP, lSSc and dSSc ($F(2,154)=50.18$, $p<0.001$). At the post-hoc analysis the capillary numerosity was preserved in pRP patients (8.87±0.78, $p<0.001$), in

comparison to SSc patients. Capillary loss was present in both lSSc and dSSc, but was significantly more pronunced in dSSc (4.89±1.53, p<0.001) compared to lSSc (6.18±1.75, p<0.001). Moreover, a significantly higher B score was reported in dSSc (t(125)=-3.95, p<0.001), with significant lower capillary density (H(1)=12.01, p<0.001), altered capillary architecture (H(1)=13.96, p<0.001) and presence of abnormal shapes (angiogenesis) (H(1)=8.00, p=0.005) (Table1). Furthermore, a statistically significative association between "late" NVC scleroderma pattern and dSSc (χ^2(1,126)=10.45, p=0.004) was established, as well as "early" NVC scleroderma pattern and lSSc (χ^2(1,126)=6.56, p=0.010). Early microvascular damage (score A) did not correlate with the extent of skin fibrosis in SSc.
Conclusions: Our study emphasizes the contribution of NVC in distinguishing microvascular damage between lSSc and dSSc. Both lSSc and dSSc showed capillary loss, however this parameter was significantly higher in dSSc, despite the greater use of immunosuppressants. In addition, higher B scores seem correlated with dSSc, not only concerning capillary density, but also regarding abnormal shapes (angiogenesis). The data also highlighted the association of "late" NVC scleroderma pattern with dSSc. We found no association between the presence of giants and the extent of skin fibrosis. This can be explained by the observation of higher prevalence of "early" NVC pattern in lSSc (presence of few giants) and "late" NVC pattern in dSSc (absence of giants).

REFERENCES:

1. Cutolo M et al. Nat Rev Rheumatol. 2021
2. Ingegnoli F et al. Microvasc Res. 2013
3. Sato LT et al. Acta Reumatol Port. 2009
4. Sulli A et al. Ann Rheum Dis. 2008

131 - RETHINKING PREDICTORS: SJOGREN'S SYNDROME AS A POSITIVE PREDICTOR OF RITUXIMAB EFFECTIVENESS IN RHEUMATOID ARTHRITIS

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Background: Rheumatoid arthritis (RA) is a chronic inflammatory rheumatic condition that often requires targeted therapies like rituximab. Understanding baseline predictors of response is crucial for optimizing treatment outcomes. This retrospective study aims to investigate various patient characteristics, baseline patient-reported outcomes, laboratory levels, and immunological markers as potential predictors of rituximab response in RA.
Objective: The primary objective is to identify baseline factors associated with rituximab response in RA patients in a real-life setting.
Methods: A monocentric retrospective study between 2008 and 2023 was conducted. RA patients receiving rituximab were included. Patients below age 18 were excluded. Patient characteristics, disease-related variables, baseline patient-reported outcomes, and laboratory parameters were collected. The study employed statistical tests, including chi-square tests and t-tests, to compare variables between patients with and without a response to rituximab.
Results: Out of 745 RA patients, 98 met the study inclusion criteria. Among the notable findings (see table I), we observed a significantly higher response rate to rituximab in RA patients with secondary Sjogren`s syndrome (p=0.011). Baseline patient-reported outcomes (HAQ and PG-VAS), DG-VAS, disease activity scores (DAS, CDAI, SDAI), 28 TJC and 28 SJC were significantly higher in the responder group (p<0.001). Baseline serum levels of immunoglobulin A (p=0.036) and 25(OH)vitamin D3 (p=0.028), and erythrocyte sedimentation rate (ESR) (p=0.003) also displayed significant associations with rituximab response. Moreover, baseline serum levels of immunoglobulin G and CRP were numerically higher in the rituximab responder group (p=0.080 and p=0.084, respectively).
Conclusion: This retrospective study provides valuable insights into baseline predictors of rituximab response in RA. Secondary Sjogren`s syndrome, higher baseline disease activity, and specific laboratory parameters such as serum immunoglobulin A and ESR emerge as potential indicators of a favorable response to rituximab treatment. These findings contribute to the ongoing efforts to personalize RA treatment strategies and enhance therapeutic effectiveness with rituximab. Further prospective studies are warranted to validate and expand upon these observations, fostering more precise and individualized management approaches in RA patients undergoing rituximab therapy.

132 - SIX-MONTH PREDICTORS OF LATE CLINICAL RESPONSE TO RITUXIMAB IN RHEUMATOID ARTHRITIS PATIENTS

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TO 131 – TABLE 1.

	No Response to Rituximab	Response to rituximab	Total sample	p value
Patient Characteristics				
Female gender, n (%)	32/40 (80.00)	46/58 (79.31)	78/98 (79.59)	0.919
Ever smokers, n (%)	10/40 (25.00)	17/58 (29.31)	27/98 (37.76)	0.624
Alcohol consumption, n (%)	7/40 (17.50)	8/58 (13.79)	15/98 (15.31)	0.628
BMI, mean (SD, range), kg/m2	26.49(5.50; 18.67-39.68)	26.10 (4.10; 20.34-36.56)	26.28 (4.75; 18.67-39.86)	0.966
Comorbidities, n (SD, range)	1.00 (1.07; 0-4)	1.18 (1.20 ;0-4)	1.07(1.15; 0-4)	0.728
Age disease started, mean (SD, range), years	44.41 (14.45; 25.12-72.35)	44.26 (13.64; 17.32-79.37)	44.32 (13.91; 17.32-79.37)	0.479
Age diagnosis, mean (SD, range), years	46.84 (13.79; 25.44-72.89)	46.76 (12.96; 24.25-80.29)	46.80 (13.25; 24.26-80.29)	0.488
Age started Rituximab, mean (SD, range), years	57.63 (12.69; 35.35-77.63)	57.19 (10.00; 32.59-81.40)	57.37 (11.13; 32.59-81.40)	0.288
Disease duration until rituximab started, mean (SD, range), years	13.09 (9.05; 1.00-34.71)	12.79 (9.36; 0.46-38.00)	12.91 (9.19; 0.46-38.00)	0.837
RA-related lung involvement, n (%)	12/40 (30.00)	10/58 (17.24)	22/98 (22.45)	0.449
Secondary Sjogren`s Syndrome, n (%)	2/40 (5.00)	14/58 (24.14)	16/98 (16.33)	0.011
Late Onset RA, n (%)	6/40 (15.00)	7/58 (12.07)	12/98 (13.27)	0.894
Previous bDMARD prescribed, n (%)	29/40 (72.50)	21/58 (36.21)	50/98 (51.02)	0.507
Rheumatoid factor (positive), n (%)	29/40 (72.50)	46/58 (79.31)	75/98 (76.53)	0.448
Anti-CCP antibodies (positive), n (%)	32/40 (80.00)	52/58 (89.65)	84/98 (85.71)	0.185
Radiographic erosions, n (%)	20/40 (50.00)	50/58 (86.20)	70/98 (71.42)	0.422
Baseline Disease Outcomes				
DAS 4V, mean (SD, range)	4.48 (1.34; 1.96-7.23)	6.06 (1.16; 3.35-8.33)	5.41 (1.46; 1.96-8.33)	<0.001
DAS 3V, mean (SD, range)	4.16 (1.28; 1.60-6.87)	5.63 (1.21; 2.47-7.95)	5.02 (1.43; 1.60-7.95)	<0.001
DAS 3V CRP, mean (SD, range)	3.64 (1.27; 1.42-6.03)	4.95 (1.23; 2.10-7.50)	4.41 (1.40; 1.42-7.50)	<0.001
DAS 4V CRP, mean (SD, range),	4.01 (1.33; 1.86-6.42)	5.41 (1.15; 2.66-7.85)	4.81 (1.41; 1.86-7.85)	<0.001
CDAI, mean (SD, range)	17.8 (11.36; 0.70-42.00)	30.74 (13.25; 9.5-65.5)	25.50 (13.99; 0.7-65.5)	<0.001
SDAI, mean (SD, range)	19.27 (11.80; 1.86-42.76)	33.24 (15.08; 11.55-74.67)	27.50 (15.38; 1.86-74.68)	<0.001
28 TJC, mean (SD, range)	4.88 (5.13; 0-19)	11.36 (8.12; 0-28)	8.65 (7.70; 0-28)	<0.001
28 SJC, mean (SD, range)	4.12 (4.09; 0-16)	9.19 (6.37; 0-25)	7.07 (6.06; 0-25)	<0.001
Patient global VAS, mean (SD, range), mm	56.07 (25.88; 0-100)	71.66 (19.42; 10-100)	53.39 (27.28; 0-95)	0.002
Doctor VAS, mean (SD, range),mm	41.73 (28.77; 3-84)	61.34 (23.36; 0-95)	53.39 (27.28; 0-95)	0.005
HAQ, mean (SD, range)	1.54 (0.73; 0-2.88)	1.86 (0.65; 0.13-2.75)	1.72 (0.70; 0-2.88)	0.023
Baseline Laboratory parameters				
Uric Acid, mean (SD, range), mg/dL	4.24 (1.26; 1.93-7.60)	4.74 (1.40; 2.70-8.00)	4.55 (1.36; 1.93-8.00)	0.091
Albumin, mean (SD, range), g/L	39.89 (2.99; 35.60-49.00)	39.85 (5.65; 28.99-63.20)	39.87 (4.67; 28.99-63.20)	0.853
Creatinine mean (SD, range), mg/dL	0.72 (0.21; 0.38-1.32)	0.77 (0.27; 0.46-1.87)	0.75 (0.24; 0.38-1.84)	0.584
ALT, mean (SD, range), U/L	20.00 (8.70; 5.00-40.00)	20.40 (9.57; 8.00-54.00)	20.00 (9.20; 5.00-54.00)	0.918
AST, mean (SD, range), U/L	22.10 (6.52; 11-41)	21.80 (7.04; 10.00-46.00)	21.90 (6.8; 10.00-46.00)	0.421
ALP, mean (SD, range), U/L	81.00 (27.80; 38.00-154.00)	91.04 (50.62; 42.00-358.00)	87.10 (43.30; 38.00-358.00)	0.425
GG-T, mean (SD, range), U/L	28.17 (15.05; 8.00-75.00)	45.45 (70.19; 10.00-455.00)	38.59 (55.7; 8.00-455.00)	0.284

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TO 131 – TABLE 1. Continuation

	No Response to Rituximab	Response to rituximab	Total sample	p value
Total Bilirrubin, mean (SD, range), mg/dL	0.54 (0.18; 0.22-0.93)	0.52 (0.20; 0.07-1.14)	0.52 (0.19; 0.07-1.14)	0.343
Haemoglobin, mean (SD, range), g/dL	12.74 (1.54; 9.20-16.40)	12.92 (1.72; 8.8-16.00)	12.85 (1.64; 8.80-16.40)	0.318
Leucocytes, mean (SD, range), x109/L	9.17 (3.22; 2.56-16.64)	8.69 (3.42; 4.19-21.07)	8.88 (3.33; 2.56-21.07)	0.303
Platelets, mean (SD, range), x109/L	288.9 (93.66; 176.00-530.00)	280.41 (83.07; 165.00-513.00)	283.72 (86.79; 165.00-513.00)	0.862
Neutrophil-lymphocyte ratio, mean (SD, range)	2.84 (1.27; 1.14-5.63)	4.07 (3.94; 0.11-17.15)	3.47 (2.98; 0.11-17.15)	0.593
Monocyte-Lymphocyte mean (SD, range)	0.41 (0.19; 0.14-0.75)	0.43 (0.22; 0.20-1.00)	0.42 (0.20; 0.13-1.00)	0,888
Platelets-Lymphocyte mean (SD, range)	161.12 (69.93; 44.37-360.54)	163.55(72.33; 62.67-342.85)	162.87 (70.24; 44.37-360.54)	0.884
Rheumatoid factor, mean (SD, range), UI/mL	514.63 (834.05; 14.90-2740.00)	520.96 (762.44; 14.4-3240.00)	519.25 (770.56; 14.40-3240.00)	0.782
Anti-CCP antibodies, mean (SD, range), U/mL	607.18 (671.58; 0.70 –1960.00)	1047.3 (1920.09; 0.8-8960)	904.26 (1624.86; 0.70-8960.00)	0.711
Anti-nuclear antibodies (positive), n (%)	3/40 (7.50%)	12/58 (20.69%)	15/98 (15.30%)	0.141
Anti-dsDNA antibodies (positive), n (%)	3/40 (7.50%)	5/58 (8.60%)	8/98 (8.16%)	0.947
Immunoglobin A, mean (SD, range), mg/dL	275.00 (104.00; 132.00-467.00)	340.94 (121.74; 133.00-538.00)	318.43 (119.27; 132.00-538.00)	0.036
Immunoglobin G, mean (SD, range, mg/dL	1146.00 (282.00; 736.00-1610.00)	1344.81 (516.10; 683.00-3610.00)	1277.23 (457.83; 683.00-3610.00)	0.080
Immunoglobin M, mean (SD, range, mg/dL	155.00 (85.20; 50.00-335.00)	205.9 (210.05; 34.00-1200.00)	188.15 (177.36; 34-1200)	0.413
25(OH)Vitamin D3 mean (SD, range, mg/dL) ng/mL	33.70 (17.30; 12.00-81.00)	22.47 (11.67; 4.00-51.00)	27.44 (15.24; 4.00-81.00)	0.028
ESR, mean (SD, range), mm (first hour)	33.29 (25.42; 3-110)	47.69 (24.94; 2-102)	43.78 (27.09; 2-110)	0.003
CRP, mean (SD, range), mg/dL	1.45 (1.49; 0.07-5.15)	2.45 (4.26; 0.08-30.42)	3.05 (5.19; 0.03-30.42)	0.084

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Background: Rheumatoid arthritis (RA) is a chronic inflammatory rheumatic condition that may require targeted therapies like rituximab. Understanding predictors of response is crucial for optimizing treatment outcomes. This retrospective study aims to investigate various patient characteristics, disease-related parameters, patient-reported outcomes, laboratory levels, and immunological markers, at 6 months of treatment with rituximab, as potential predictors of late response to rituximab in RA.

Objective: The primary objective is to determine predictors of late response to rituximab in RA patients, at the assessment visit at 6 months of treatment.

Methods: A monocentric retrospective study between 2008 and 2023 was conducted. Out of 745 RA patients, 45 under rituximab met the study inclusion criteria. Patients below age 18 and patients with early response to rituximab (at 6 months) were excluded. Patients were divided in two groups based on having EULAR response to rituximab at 12 months. Patient characteristics, disease-related variables, baseline patient-reported outcomes, and laboratory parameters were collected. The study employed statistical tests, including chi-square tests and t-tests, to compare variables between patients with and without a response to rituximab.

Results: Among the notable findings (see table I), a positive association was observed between late response to rituximab and positivity for anti-CCP antibodies (p=0.036). Additionally, the same results were also significantly associated with the presence of higher six-month disease activity outcomes [including DAS4V (p=0.044), DAS3V (p=0.015) and DAS3V CRP (p=0.050), and with the exception of DAS4V CRP (p=0.095)]. Moreover, higher swollen joint counts (p=0.028), at six months of treatment, were also linked to late response to rituximab.

Conclusion: In summary, at six months of treatment with rituximab, positive anti-CCP antibodies, elevated DAS, and swollen joint counts are key predictors of late clinical response among RA patients. These insights underscore the importance of tailoring treatment strategies, integrating serological and clinical factors for enhanced therapeutic outcomes in RA.

136 - ARRITMIAS E ALTERAÇÕES DA CONDUÇÃO NOS DOENTES COM ESCLEROSE SISTÉMICA

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Introdução: A esclerose sistémica (ES) é uma doença autoimune rara, que se carateriza por vasculopatia e por fibrose de órgãos, podendo afetar órgãos major como o coração. Pode afetar todos os componentes estruturais do coração, incluindo o pericárdio, o miocárdio, as válvulas e o sistema de condução, por mecanismos de inflamação, isquemia e fibrose, conferindo um pior prognóstico vital. As alterações de condução são frequentes nos doentes com ES e podem ocorrer logo no início da doença, mesmo antes do desenvolvimento

TO 136 – TABELA 1. Caraterísticas clínicas e demográficas

	Esclerose sistémica (n=146)	Alterações da condução (n=12)	Sem alterações da condução (n=134)	Valor de p
Sexo feminino, n (%)	129 (88.4)	10 (83.3)	119 (88.8)	0.633
Caucasiano, n (%)	136 (93.8)	11 (91.7)	125 (94)	0.551
Fenótipo ES, n (%)				
Forma cutânea limitada	112 (76.7)	5 (41.7)	107 (79.9)	0.007
Forma cutânea difusa	32 (21.9)	7 (58.3)	25 (18.7)	0.005
Sine scleroderma	2 (1.4)	0 2 (1.5)		*
Idade início de sintomas, anos	50.5 ± 15.1	50.4 ± 19.1	50.5 ± 14.7	0.976
Idade ao diagnóstico, anos	56.4 (17.9)	66.8 (22.6)	55.9 (17.2)	0.053
Manifestações imunológicas, n (%)				
Anticorpos anti-nucleares	139 (95.9)	12 (100)	127 (95.5)	*
Anti-centrómero	79 (54.1)	4 (33.3)	75 (56)	0.132
Anti-topoisomerase I	43 (29.5)	7 (58.3)	36 (26.9)	0.041
Anti-RNA polimerase III	5 (3.6)	0	5 (3.9)	*
Anti-Th/to	1 (0.7)	0	1 (0.8)	*
Anti-u3 RNP	7 (5)	1 (9.1)	6 (4.7)	0.443
Anti-pm/scl	1 (0.7)	0	1 (0.8)	*
Anti-ku	2 (1.4)	0	2 (1.6)	*
Anti-U1 RNP	1 (0.7)	0	1 (0.8)	*
Manifestações clínicas, n (%)				
Telangiectasias	73 (49.3)	6 (50)	67 (50.8)	0.960
Fenómeno de Raynaud	141 (97.2)	12 (100)	129 (97)	*
Espessamento cutâneo	137 (93.6)	11 (91.7)	126 (94)	0.548
Úlceras digitais	48 (32.9)	3 (25)	45 (33.6)	0.751
Envolvimento esofágico	80 (54.8)	7 (58.3)	73 (54.5)	0.797
Envolvimento gástrico	22 (15.1)	1 (8.3)	21 (15.7)	0.694
Envolvimento intestinal	26 (17.8)	1 (8.3)	25 (18.7)	0.694
Envolvimento articular	60 (41.1)	4 (33.3)	56 (41.8)	0.762
Miosite	5 (3.4)	0	5 (3.8)	*
Envolvimento pulmonar	46 (31.5)	6 (50)	40 (29.9)	0.195
Envolvimento renal	2 (1.4)	0	2 (1.5)	*
Calcinose	18 (12.4)	1 (8.3)	17 (12.8)	1.000
Contraturas	19 (13.2)	2 (18.2)	17 (12.8)	0640
Comorbilidades, n (%)				
Hipertensão arterial	69 (47.6)	8 (72.7)	61 (45.5)	0.082
Dislipidemia	31 (21.5)	2 (18.2)	29 (21.8)	1.000
Diabetes mellitus	11 (7.6)	1 (9.1)	10 (7.5)	0.594
Doença arterial coronária	6 (4.4)	2 (18.2)	4 (3.2)	0.074
Insuficiência cardíaca	9 (6.6)	3 (27.3)	6 (4.8)	0.025
Fumadores	32 (26.4)	1 (11.1)	31 (27.7)	0.442
Consumo alcoólico	6 (5.5)	1 (11.1)	5 (5)	0.408

de fibrose ou outras manifestações viscerais, sendo por isso importante identificar os doentes de alto risco e realizar um rastreio e deteção precoces.

Objetivos:

1) Determinar a proporção de doentes com esclerose sistémica com arritmias e alterações da condução;

2) Identificar as caraterísticas clínicas e demográficas associadas a arritmias/alterações da condução.

Métodos: Foram incluídos os doentes com o diagnóstico de esclerose sistémica seguidos no Hospital Garcia de Orta que cumpriam os critérios de classificação ACR/EULAR 2013, registados no registo português de doenças reumáticas (Reuma.pt) . Foram excluídas as síndromes de sobreposição.

A identificação de doentes com arritmias e alterações da condução foi feita com base nos registos de eletrocardiogramas e/ou holters de 24/48 horas. Foi realizada uma análise bivariada para identificar as caraterísticas clínicas e demográficas associadas a arritmias/alterações da condução.

Resultados: Foram incluídos um total de 146 doentes, dos quais 12 com arritmias e/ou alterações da condução (8.2%). A arritmia mais prevalente foi a extrassistolia supraventricular (50%, n=6), enquanto que o distúrbio de condução mais frequente foi o bloqueio de ramo direito (33.3%, n=4). Apenas foi documentado um caso de bloqueio de ramo esquerdo e um caso de bloqueio aurículo ventricular de 3º grau. Dos 12 doentes identificados, 10 (83.3%) eram do sexo feminino e 11 (91.7%) eram caucasianos. 7 doentes (58.3%) tinham envolvimento cutâneo difuso e 5 doentes (41.7%) envolvimento cutâneo limitado. Em termos de comorbilidades cardiovasculares, 8 doentes (72.7%) com hipertensão arterial e 2 doentes (18.2%) com doença arterial coronária.

Nos doentes com alteração da condução a mediana da idade ao diagnóstico foi de 66.8 anos (22.6) vs. 55.9 (17.2) nos doentes sem alterações da condução (tabela 1). No grupo com alterações da condução verificou-se uma maior prevalência do anticorpo anti-topoisomerase I (58.3% vs 26.9%, p< 0.041) e de insuficiência cardíaca (27.3% vs 4.8%, p< 0.025).

Conclusão: A prevalência de doentes com arritmias e alterações da condução foi de 8.2%. A arritmia mais comum foi a extrassistolia supraventricular, enquanto que o distúrbio de condução mais frequente foi o bloqueio de ramo direito. Não se pode excluir a possibilidade de subdiagnóstico destas alterações, especialmente em doentes assintomáticos, uma vez que os exames electrocardiográficos, nomeadamente Holter, não foram realizados de forma sistemática. No grupo com alterações da condução verificou-se uma maior prevalência do anticorpo anti-topoisomerase I e de insuficiência cardíaca.

148 - PERSISTENCE IN TREATMENT WITH TNF-ALPHA INHIBITORS IN SPONDYLOARTHRITIS: COMPARISON BETWEEN ORIGINAL AND BIOSIMILAR DRUGS - A PILOT STUDY

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Introduction: Biosimilars have demonstrated comparable efficacy, safety and immunogenicity to original drugs in randomized clinical trials, with subsequent extrapolation of their therapeutic indications to those of the original drug. Some studies with real-world data supporting this biosimilarity have been published. However, long-term data of their use in Spondyloarthritis (SpA) is still scarce.

Objectives: To compare effectiveness and safety of original TNF-alpha inhibitors (iTNF) and biosimilars in bDMARD-naïve patients diagnosed with SpA, measured by persistence rates (PR) over 3 years of follow-up; to compare disease activity and response rates after 6, 12 and 24 months; to investigate the frequency and reasons for discontinuation comparing the rates of adverse events (AEs).

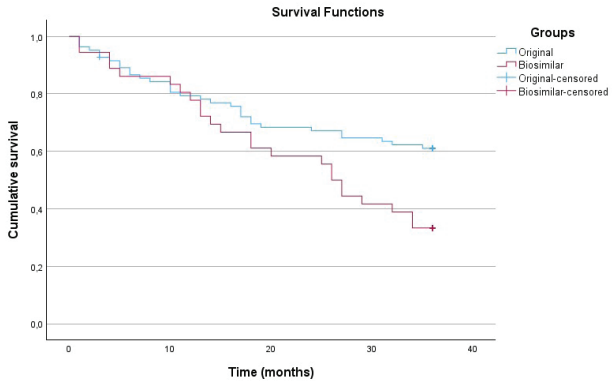
Methods: A retrospective observational study of patients followed at the Rheumatology Unit of ULS Viseu Dão-Lafões, using data collected prospectively from The Rheumatic Diseases Portuguese Registry (Reuma.pt) was performed, including patients with: age ≥18 years old; diagnosis of SpA (axial or peripheral) and bDMARD-naïve, who initiated treatment with Humira®, Enbrel®, Simponi®, Imraldi®, Hyrimoz® or Benepali®, between January 2010 and May 2022. Kaplan-Meyer and Cox regression were used to calculate the PR in treatment. Disease activity mean and standard deviation at 6, 12 and 24 months of treatment were compared. Causes for therapy discontinuation were summarized using descriptive statistics. Statistical significance was assumed for p-values <0.05.

Results: A total of 127 patients were included, 83 under original iTNF and 44 under biosimilar iTNF. The majority had radiographic axial SpA (56.6% under original iTNF, 59.1% under biosimilar iTNF) and were HLA-B27 positive (65.1% under original iTNF, 68.2% under biosimilar iTNF). PR at 3-years was significantly higher under original iTNF (61.4%) (figure 1), with an average drug use time of 27.2 months; compared to 33.3% under biosimilar iTNF, with an average drug use time of 23.7 months (p=0.012). The main cause of discontinuation was secondary failure, with a higher

TO 148 – TABLE 1. Disease activity at 6, 12 and 24 months under original and biosimilar iTNF

	6 months			12 months			24 months		
	Original	Biosimilar	p-value	Original	Biosimilar	p-value	Original	Biosimilar	p-value
BASDAI <4, % (n/N)	68.4 (26/38)	38.1 (8/21)	**0.024**	61.5 (24/39)	48.3 (14/29)	0.276	64.7 (22/34)	33.3 (4/12)	0.059
ΔBASDAI ≥2, %(n/N)	65.2 (15/23)	35.0 (7/20)	**0.048**	68.2 (15/22)	25.0 (11/27)	0.056	62.50 (10/16)	63.6 (7/11)	0.952
BASFI <4, % (n/N)	70.4 (19/27)	20.0 (3/15)	**0.002**	71.4 (20/28)	38.5 (10/26)	**0.015**	65.4 (17/26)	54.5 (6/11)	0.534
ASDAS-PCR, mean (SD)	1.592 (1.167)	2.443 (0.935)	**0.046**	1.622 (0.930)	2.300 (1.436)	0.229	1.660 (0.825)	2.259 (0.865)	0.159
ASDAS-PCR <2.1, % (n/N)	76.9 (10/13)	35.7 (5/14)	**0.031**	77.8 (7/9)	69.2 (9/13)	0.658	80.9 (8/10)	25.0 (2/8)	**0.020**
ΔASDAS-PCR ≥1.1, % (n/N)	-	-		25.0 (¼)	41.7 (5/12)	0.551	25.0 (¼)	60.0 (3/5)	0.294

ASDAS: Ankylosing Spondylitis Disease Activity Score; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BASFI: Bath Ankylosing Spondylitis Functional Index; SD: Standard deviation



TO 148 – Figure 1. Drug survival with original and biosimilar TNF-alpha inhibitors

proportion observed under biosimilar iTNF (52.8% vs 18.1%, p<0.01). No differences were found regarding primary failure or the rate of AEs.

The most commonly reported AEs were infections, skin reactions at the injection site and paradoxical psoriasis. The cumulative risk of AEs was higher under original iTNF (15.7% vs. 8.3%), although not statistically significant (p=0.282).

The proportion of subjects in remission or low disease activity was greater under original iTNF at 6 months, with statistically significant differences in BASDAI (p=0.024), BASFI (p=0.02), ASDAS-PCR (p=0.031) and BASDAI response (p=0.048). However, this difference was not consistently reproduced at 12 and 24 months, except for BASFI at 12 months (p=0.015) and ASDAS-PCR at 24 months (p=0.020) (table 1).

Conclusions: Our results suggest greater efficacy of the original iTNF, with no differences in the safety profile, compared to biosimilars. However, the number of missing data and the sample size were a limitation of the study, which limits the extrapolation of our conclusions. Further studies with a larger sample size are needed to confirm these results, as they could have important implications for clinical practice.

149 - CRISES DE ARTRITE GOTOSA NUM INTERNAMENTO DE MEDICINA INTERNA: PREVALÊNCIA E CARACTERIZAÇÃO

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Introdução: A gota constitui uma das formas mais comuns de artrite , sendo causada por hiperuricemia prolongada com consequente formação de cristais de monourato de sódio. Vários fatores foram associados ao desenvolvimento de crises de gota e alguns estudos demonstram que o internamento hospitalar pode aumentar a probabilidade da sua ocorrência.

Objetivos: Conhecer a prevalência de crises de gota num internamento de Medicina Interna e caracterizar os doentes que desenvolvem esta condição. Avaliar as diferenças entre doentes com valores de uricemia acima do alvo definido pela EULAR (≥6mg/dl), previamente ao internamento, com aqueles com uricemia controlada (<6mg/dl), relativamente a dados demográficos, principais comorbilidades associadas e alguns fármacos utilizados.

Métodos: Estudo retrospetivo, incluindo doentes internados no serviço de Medicina Interna da ULS de Coimbra, desde 1 de janeiro de 2018 até 31 dezembro de 2022, com codificação, segundo o ICD-10, do diagnóstico "crise de gota" à data da alta. Realizou-se análise descritiva dos dados demográficos, comorbilidades, di-

agnósticos que motivaram o internamento e tratamento implementado. O teste Chi-quadrado (para variáveis categóricas) e os teste T e de Mann-Whitney (para variáveis contínuas) foram utilizados para comparar o grupo de doentes com uricemia acima do alvo com o grupo com uricemia controlada. Foram considerados os valores de ácido úrico do último estudo analítico realizado previamente ao internamento. Um valor de p<0.05 foi considerado estatisticamente significativo.

Resultados: De um total de 13 380 doentes, 204 desenvolveram crise de gota, o que corresponde a uma prevalência de 1.5%. Destes, 71.1% (n=145) eram do sexo masculino, com idade média de 81.0 (±10.0) anos, e mais de metade já tinha gota descrita como antecedente patológico (49.5%, n= 101). Apenas 3.9% (n= 8) dos doentes foram submetidos a artrocentese diagnóstica. Mais de metade destes doentes tinha comorbilidades como hipertensão arterial (n= 180, 88.2%), insuficiência cardíaca (n= 153, 75%), dislipidemia (n= 124, 60.8%) e doença renal crónica (n= 115, 56.4%). Os principais diagnósticos à admissão foram "insuficiência cardíaca descompensada" (n= 41, 20.1%), "pneumonia adquirida na comunidade" (n= 28, 13.7%) e "pielonefrite aguda" (n= 16, 7.8%). De acordo com a patologia que motivou o internamento, foi administrada terapêutica diurética a cerca de 77.5% (n= 158) destes doentes e fluidoterapia endovenosa a 58.3% (n=119). O tratamento da crise de gota fez-se maioritariamente com colchicina (n= 138, 67.7%) ou corticóide (n= 115, 56.4%).

Verificou-se, ainda, que os doentes com valores de uricemia acima do alvo eram mais frequentemente do sexo masculino (76.2 vs 57.9%; p=0.010) e apresentaram com maior frequência lesão renal aguda (LRA) durante o internamento (61.2 vs 49.9%; p=0.025). Não foram encontradas diferenças significativas relativamente a outras comorbilidades e fármacos utilizados.

Conclusão: A prevalência de crises de gota durante o internamento de Medicina Interna, ao longo de 5 anos, foi de 1.5%. A maioria dos doentes eram homens, idosos e apresentavam várias comorbilidades frequentemente relacionadas com esta condição. A uricemia acima do alvo previamente ao internamento estava associada ao sexo masculino e desenvolvimento de LRA durante a hospitalização. Estes resultados reforçam a importância do reconhecimento destas comorbilidades, com vista a uma gestão mais adequada destes doentes, tanto em internamento como em ambulatório.

153 - B-ACTIVE: AN INTEGRATIVE INTERACTIVE PLATFORM FOR CHILDREN WITH RHEUMATIC DISEASES

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Introduction: Young people often struggle to self-manage chronic diseases during the transition from childhood to adulthood. Doctors do their best to treat diseases, but also have a central role in promoting healthy lifestyles since childhood. We strongly believe that we must put in practice interventions to make the children "sit less and move more, sleep well and eat better", not only to improve the disease symptoms but also to prevent future damage.

Methods: We have developed a virtual platform, where patients with juvenile rheumatic diseases answered questionnaires about disease activity (by completing the visual analogue scale (VAS) and painful joints in the homunculus), nutritional and physical activity habits and a questionnaire on mental health. Before using the platform, they had a first evaluation performed by a multidisciplinary team (composed by rheumatologist, psychologist, personal trainer and nutritionist). After this evaluation, they had to fill out the platform on a bi-weekly basis and could access an exercise plan according to their disease involvement, a nutritional plan adjusted to individual preferences and needs. Also, mindfulness programs and stress management strategies are given, according to the answers.

Results: We have started recruiting patients in June 2022. Thirty-two patients were included (21 females and 11 males). The diagnosis were oligoarticular juvenile idiopathic arthritis (JIA) (n=8), polyarticular JIA (n=5), psoriatic JIA (n=4), enthesitis-related arthritis (n=3), systemic lupus erythematosus (SLE) (n=4), primary Raynaud's

phenomenon (n=2), juvenile fibromyalgia (n=2), Marfan syndrome (n=1), Takayasu arteritis (n=1), juvenile osteoporosis (n=1) and one case of periodic fever, aphthous stomatitis, pharyngitis, adenitis (PFAPA, n=1).
On the first use of the application, most patients (n=20) had a VAS of 0, 5 patients had a VAS between 20 and 40, 6 patients had a VAS between 40 and 60, and 3 patients had a VAS superior to 60. The 3 patients that had a VAS superior to 60 were diagnosed with juvenile fibromyalgia and Marfan´s syndrome.
The median Body Mass Index (BMI) was 22 (normal BMI). Considering eating habits, 25% of the included patients did not eat fruit or vegetables daily, and half of the patients did not eat fish regularly. We highlight that five patients reported to eat candys daily.
In the psychological evaluation, we found that the majority (94%) of our sample felt happy and fulfilled, but a small percentage of patients (6%) reported hating themselves and feeling sad frequently.
Regarding physical activity, half of the patients don't exercise at all, not even during physical education classes. Patients who reported to do no exercise had a VAS between 0-30.
Conclusions: Education about healthy lifestyles is important right from childhood, since looking at this data (despite the small sample size) we can conclude that healthy eating and regular exercise are not part of people's daily lives.
Multidisciplinary approach in paediatric rheumatic diseases is of utmost importance and the development of these online platforms benefits the professional-patient relationship.

155 - FREQUENCY, CHARACTERISTICS AND PREDICTORS OF CENTRAL AND PERIPHERAL NEUROLOGICAL INVOLVEMENT IN SJÖGREN'S DISEASE: DATA FROM PORTRESS, THE PORTUGUESE REGISTRY OF SJÖGREN'S DISEASE

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Background: Sjogren's disease (SjD) systemic affection is being increasingly recognized. Prevalence of peripheral (PNS) and central (CNS) nervous system involvement in SjD and risk factors for these manifestations

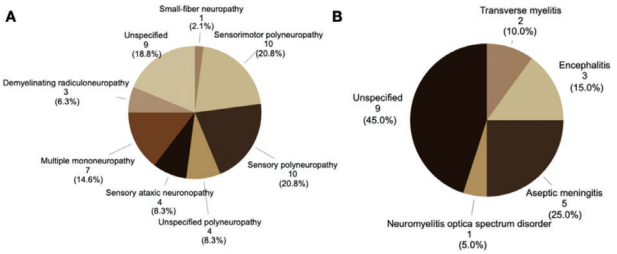
TO 155 – TABLE 1. Demographic and clinical characteristics of SjD patients with and without PNS and/or CNS involvment

	SjD with PNS and/or CNS involvement N=65	SjD without PNS or CNS involvement N=1168	p
Female	57 (87.7)	110 (94.6)	**0.029**
Caucasian / White	52 (92.9)*	915 (92.9)**	1.000
Age at diagnosis	52.7±15.2*	52.8±14.5**	0.960
Age at symptom onset	49.6±14.4*	47.8±14.8**	0.365
Disease duration	11.2 [12.3]*	11.0 [10.7]**	0.361
AECG 2002	37 (59.7)*	663 (64.1)**	0.479
ACR/EULAR 2016	41 (64.1)*	676 (60.0)**	0.517
ANA	57 (90.5)*	957 (90.6)**	0.969
anti-Ro	51 (81.0)*	920 (80.6)**	0.939
anti-La	23 (38.3)*	489 (44.6)**	0.343
Rheumatoid factor	27 (49.1)*	485 (48.2)**	0.899
Cryoglobulin	10 (27.8)*	52 (14.2)**	**0.031**
Hypergammaglobulinemia	30 (48.4)*	489 (48.5)**	0.991
Low C3	12 (20.0)*	177 (17.5)**	0.615
Low C4	10 (16.7)*	88 (8.7)**	**0.037**
Persistent salivary gland swelling	9 (14.8)*	75 (7.2)**	**0.043**
Purpura / cutaneous vasculitis	10 (16.7)*	57 (5.5)**	**0.002**
Lymphopenia	16 (25.4)*	205 (19.9)**	0.288
CD4+/CD8+ ratio ≤0.8	1 (5.0)*	4 (1.0)**	0.216
Monoclonal gammopathy	8 (13.6)*	52 (5.3)**	**0.016**
Ectopic lymphoid structures on salivary glands	4 (11.1)*	41 (6.2)**	0.281
Lymphoma	2 (4.4)*	17 (2.2)**	0.283
Non Hodgkin lymphoma	1 (2.2)*	2 (0.3)**	0.157
Focus score ≥1	25 (52.1)*	405 (48.5)**	0.629
Baseline ESSDAI	4.5 [11.5]*	2.0 [4.0]**	**<0.001**
Baseline ESSDAI without neurological domains	3.0 [7.0]*	2.0 [4.0]**	**0.005**
Baseline ESSPRI	6.0 [5.3]*	5.3 [4.66]**	0.324
Involvements			
- Constitutional	17 (26.6)*	207 (17.8)**	0.077
- Lymphadenopathic	11 (17.2)*	126 (10.8)**	0.114
- Glandular	27 (42.2)*	356 (30.5)**	0.050
- Articular	32 (50.0)*	500 (42.9)**	0.263
- Cutaneous	19 (29.7)*	200 (17.2)**	**0.011**
- Pulmonary	12 (18.8)*	89 (7.6)**	**0.002**
- Renal	4 (6.3)*	29 (2.5)**	0.084
- Muscular	0*	18 (1.5)**	NA
- Haematologic	24 (38.7)*	392 (33.6)**	0.409
- Biologic	41 (65.1)*	592 (50.9)**	**0.028**
- Hepato/Gastrointestinal	2 (3.2)*	33 (2.8)**	0.696
- Other	13 (22.6)*	159 (15.1)**	0.115
Treatment			
- Corticosteroids	35 (62.5)*	299 (37.3)**	**<0.001**
- Hydroxychloroquine	33 (58.9)*	628 (79.6)**	**<0.001**
- Pilocarpine	11 (19.6)*	218 (27.2)**	0.218
- Methotrexate	8 (14.3)*	134 (16.7)**	0.824
- Azathioprine	18 (32.1)*	79 (9.9)**	**<0.001**
- Leflunomide	2 (3.6)*	31 (3.9)**	1.000
- Rituximab	11 (19.6)*	27 (3.4)**	**<0.001**
- Mycophenolate mofetil	3 (5.4)*	24 (3.0)**	0.413
- IVIG	2 (3.6)*	4 (0.5)**	0.053
- Cyclophosphamide	3 (5.4)*	1 (0.1)**	**0.001**
Death	2 (4.0)*	4 (0.6)**	0.064

Results presented as mean±standard deviation or median [interquartile range] or n (%)

* caucasian / white: N=56; age at diagnosis: N=61; age at symptom onset: N=56; disease duration: N=56; AECG 2002: N=62; ACR/EULAR 2016: N=64; ANA: N=63; anti-Ro: N=63; anti-La: N=60; Rheumatoid factor: N=55; Cryoglobulin: N=36; Hypergammaglobulinemia: N=62; Low C3: N=60; Low C4: N=60; Persistent salivary gland swelling: N=61; Purpura / cutaneous vasculitis: N=60; Lymphopenia: N=63; CD4+/CD8+ ratio ≤0.8: N=20; Monoclonal gammopathy: N=59; Ectopic lymphoid structures on salivary glands: N=36; Lymphoma: N=45; Non Hodgkin lymphoma: N=45; Focus score≥1: N=48; Baseline ESSDAI: N=58; Baseline ESSDAI without neurological domains: N=58; Baseline ESSPRI: N=41; Constitutional: N=64; Lymphadenopatic: N=64; Glandular: N=64; Articular: N=64; Cutaneous: N=64; Pulmonary: N=64; Renal: N=63; Muscular: N=63; Haematologic: N=62; Biological: N=63; Hepato/gastrointestinal: N=62; Other: N=62; Hydroxychloroquine: N=56; Corticosteroids: N=56; Pilocarpine: N=56; Methotrexate: N=56; Azathioprine: N=56; Rituximab: N=56; Leflunomide: N=56; Mycophenolate mofetil: N=56; IVIG: N=56; Cyclophosphamide: N=56; Death: N=50

** caucasian / white: N=985; age at diagnosis: N=1060; age at symptom onset: N=893; disease duration: N=893; AECG 2002: N=1034; ACR/EULAR 2016: N=1127; ANA: N=1056; anti-Ro: N=1142; anti-La: N=1097; Rheumatoid factor: N=1006; Cryoglobulin: N=367; Hypergammaglobulinemia: N=1009; Low C3: N=1014; Low C4: N=1014; Persistent salivary gland swelling: N=1040; Purpura / cutaneous vasculitis: N=1034; Lymphopenia: N=1032; CD4+/CD8+ ratio ≤0.8: N=403; Monoclonal gammopathy: N=988; Ectopic lymphoid structures on salivary glands: N=662; Lymphoma: N=770; Non Hodgkin lymphoma: N=770; Focus score≥1: N=835; Baseline ESSDAI: N=927; Baseline ESSDAI without neurological domains: N=927; Baseline ESSPRI: N=541; Constitutional: N=1163; Lymphadenopatic: N=1166; Glandular: N=1166; Articular: N=1166; Cutaneous: N=1164; Pulmonary: N=1164; Renal: N=1165; Muscular: N=1166; Haematologic: N=1166; Biological: N=1164; Hepato/gastrointestinal: N=1162; Other: N=1052; Hydroxychloroquine: N=802; Corticosteroids: N=802; Pilocarpine: N=802; Methotrexate: N=802; Azathioprine: N=802; Rituximab: N=802; Leflunomide: N=802; Mycophenolate mofetil: N=802; IVIG: N=802; Cyclophosphamide: N=802; Death: N=637



TO 155 – Figure 1. Frequency distribution of the types of manifestations of the peripheral (A) and central (B) nervous systems

have not been clearly established.

Objectives: To define the clinical characteristics of SjD patients with CNS and/or PNS involvement and to find predictors of this involvement.

Methods: We included patients from the Portuguese registry of SjD (PORTRESS) who had information regarding presence or absence of PNS and/or CNS involvement. Demographic, clinical and treatment data were collected. Variables were compared according to parametric or non-parametric tests, as applicable. Predictors of neurological (PNS and/or CNS), PNS (vs. no PNS) and CNS (vs. no CNS) involvement were identified through binomial logistic regression modelling.

Results: We included 1233 patients, 94.2% female, mean age 61.8±14.3 years and median disease duration 11.0 [IQR 11.2] years (table 1). 65 patients (5.3%) had a history of neurological involvement, 48 (3.9%) with PNS and 20 (1.6%) with CNS affection (Figure 1). Sensorimotor polyneuropathy (20.8%) and sensory polyneuropathy (20.8%) were the most common subtypes of PNS affection. Aseptic meningitis (25.0%) the most common CNS manifestation. Patients with neurological involvement were more commonly male and had more active disease, translated by higher ESSDAI scores, markers of B cell activity (cryoglobulinemia, low C4, monoclonal gammopathy), and skin/glandular/respiratory involvements. On multivariate analysis, male sex (OR 2.77 [1.22-6.31], p=0.015), purpura (OR 2.72 [1.20-6.19], p=0.017), monoclonal gammopathy (OR 2.49 [1.00-6.17], p=0.049) and a higher non-neurological baseline ESSDAI (OR 1.08 [1.02-1.15], p=0.011) were associated with neurological involvement, irrespective of age at diagnosis, low C4, persistent salivary gland swelling and respiratory involvement.

PNS involvement was associated with older age at symptoms onset (OR 1.03 [1.01-1.06], p=0.008), low C4 (OR 3.08 [1.25-7.59], p=0.015), persistent salivary gland swelling (OR 3.98 [1.63-9.76], p=0.003), purpura/cutaneous vasculitis (OR 2.98 [1.10-8.03], p=0.031) and CNS affection (OR 7.32 [1.78-30.16], p=0.006), irrespective of sex, non-neurological baseline ESSDAI,

constitutional, respiratory and other (according to the registry definition) involvements. In turn, CNS disease was only associated with PNS involvement (OR 5.29 [1.10-25.35], p=0.037), irrespective of sex, ethnicity and age at diagnosis.
Conclusions: In the Portuguese cohort of SjD, 5.3% of patients had confirmed neurological involvement. Predictors of these manifestations included male sex, purpura, monoclonal gammopathy and a higher baseline ESSDAI. Moreover, a bidirectional association was observed between CNS and PNS affection. In addition, PNS disease was specifically associated with purpura, older age at SjD symptoms onset, low C4 and persistent salivary gland swelling.

158 - HANDGRIP STRENGTH IN RHEUMATOID ARTHRITIS VERSUS PSORIATIC ARTHRITIS: CLINICAL ASSOCIATIONS AND PATIENT IMPACT

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Background: Rheumatoid Arthritis (RA) and Psoriatic Arthritis (PsA) are chronic inflammatory conditions that significantly impair hand function, exhibiting distinct clinical patterns. Handgrip strength (HGS) is a simple, non-invasive measure linked to sarcopenia and increased morbimortality. Evaluating HGS and how different clinical factors influence it, may provide relevant insights into the functional impact of these diseases.
Objectives: To compare the impact of RA and PsA on HGS and examine its association with clinical variables, functional status, and quality of life.
Methods: We conducted a unicentric, cross-sectional study involving patients with RA who met the American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) 2010 criteria and patients with PsA who met the Classification for Psoriatic Arthritis (CASPAR) criteria, all with peripheral involvement. Data on demographic characteristics, clinical variables, pain (VAS), function (HAQ), and quality of life (EQ-5D) were collected. HGS was measured in the dominant hand using a hand dynamometer. Univariate and correlation analyses were performed using both parametric and non-parametric tests, followed by multiple linear regression.
Results: A total of 108 patients were included, 70 with RA and 38 with PsA. The mean HGS in patients with RA was 16,9±8,0kg and 23,0±11,8kg in PsA. Lower HGS was associated with higher age, female gender, erosive disease, higher PGA, higher levels of pain, higher disability, and lower quality of life in both RA and PsA. Lower HGS was also significantly correlated with higher disease activity and longer disease duration in RA, but not in PsA. No significant associations were found in either group regarding body mass index, acute phase reactants, or the use of b/tsDMARDs. After adjusting for age and gender, HGS was not statistically significantly different between RA and PsA patients.
Conclusion: Our study suggests that in both PsA and RA, HGS is influenced by age and gender and is associated with pain, functional status, quality of life and presence of erosions. In RA, it was also associated with disease activity and disease duration. These results highlight the potential importance of evaluating HGS in both PsA and RA in a large multicentre study.

159 - 18 MONTHS OF A NEWLY IMPLEMENTED FRACTURE LIAISON SERVICE IN A TERTIARY CARE CENTRE

Catarina Abreu[1], Susana Matias[1], Tomás Stein Novais[1], Vanessa Fraga[1], Jorge Pestana Lopes[1, 2], Raquel Roque[1], Viviana Tavares[1], Maria José Santos[1, 3], Alice Castro[1], Raquel Freitas[1]
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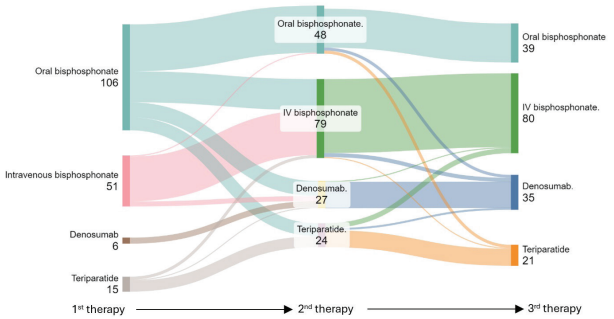
Background: Despite the socioeconomical impact of fragility fractures, they are often overlooked, resulting in a significant number of patients not receiving adequate treatment. Fracture Liaison Services (FLS) are a multidisciplinary care model designed and implemented to address secondary fracture prevention among osteoporotic patients. Implemented at our centre in October 2022, our FLS focuses on identifying patients with recent fractures, with special focus on vertebral, to initiate both non-pharmacological and pharmacological treatment.
Our aim is to describe the sociodemographic and clinical characteristics of patients observed at our FLS and their pharmacological management.
Methods: All adult patients with a diagnosis of osteoporosis observed at our FLS due to a fragility fracture since October 2022 until May 2024 were included. A descriptive analysis of sociodemographic and clinical characteristics and therapeutic options was performed.
Results: Over the first 18-months since its implemen-

tation, 182 patients with a history of fragility fracture were observed at our FLS. Among them, 87.9% (n=160) were female and 98.3% (n=177) were Caucasian. The median age of observed patients was 74.5 (14.8) years,

TO 159 – TABLE 1 - Sociodemographic and clinical characteristics of patients observed at our FLS

	FLS patients N=182
Female, n (%)	160 (87.9)
Caucasian, n (%)	177 (98.3)
Age at first appointment, years	74.5 (14.8)
Educational Level, n (%)	
No schooling	11 (6.3)
Primary education	90 (157)
Lower secondary education	32 (18.4)
Upper secondary education	18 (10.3)
Bachelor's or equivalent	18 (10.3)
Doctoral or equivalent	2 (1.1)
Household, n (%)	
One-person	40 (22.3)
One-person with a caregiver	20 (11.4)
Family household	96 (53.7)
Residential home	4 (2.2)
Gynecological history	
Age at menarche, years	13.0 ± 1.64
Age at menopause, years	49 (6)
Previous hormone replacement therapy, n (%)	14 (8.8)
Comorbidities, n (%)	
Hypertension	99 (54.4)
Cardiovascular disease	25 (13.7)
Solid neoplasia	23 (12.6)
Hematological neoplasia	6 (2.7)
Gastroesophageal reflux	18 (10)
Hepatitis C virus infection	4 (2.2)
Secondary causes, n (%)	
Alcohol consumption (current or previous)	26 (14.3)
Current smokers	21 (11.5)
Previous smokers	33 (18.1)
Low weight (BMI <20 kg/m2), n (%)	23 (12.8)
Parental fragility fracture	34 (18.8)
Rheumatic/inflammatory diseases	11 (5.9)
Human immunodeficiency virus/antiretrovirals	2 (1.1)
Early menopause	45 (24.7)
Diabetes mellitus	22 (12.1)
Chronic kidney disease	7 (3.9)
Anticonvulsants	5 (2.7)
Selective serotonin reuptake inhibitors (SSRI)	36 (19.8)
Aromatase inhibitors/tamoxifen	8 (4.3)
Chemotherapy	8 (4.4)
Loop diuretics	14 (7.7)
Proton pump inhibitors (PPI)	57 (31.3)
Anti-psychotics	11 (6)
Bone mineral density by dual-energy x-ray absorptiometry (DEXA)	
Lumbar vertebra, t-score	-2.52 ± 1.06
Femoral neck, t-score	-2.44 ± 1.46



TO 159 – Figure 1. Therapy sequencing in patients observed at our FLS.

with a median age at first fragility fracture of 68 (16) years and at the beginning of the first anti-osteoporotic treatment of 69 (15) years. The sociodemographic and clinical characteristics are described in table 1.

The most common first fracture was vertebral (59.9%) followed by wrist fractures (14.8%). More than half of the patients (53.3%, n=97) had at least one previous fragility fracture before the one that led to our observation, and half of these (50.5%, n=49) had never received anti-osteoporotic treatment. Nonetheless, 58.8% (n=50) of patients without a previous fragility fracture had already been treated with anti-osteoporotic agents.

Among the patients observed at our FLS, 60.5% had experienced a fracture in the preceding year, most of them (78.2%) being vertebral fractures.

Regarding pharmacological interventions, 92 patients (50.5%) started anti-osteoporotic therapy at our FLS, of whom 21.7% had previously received anti-osteoporotic treatment but were not under any therapy at time of observation. Additionally, 46.7% (n=85) were already undergoing anti-osteoporotic treatment, of whom 60% kept the same therapy and 40% were switched to another medication. Drug holiday was decided in 2.2% (n=4) of patients and other 2.2% (n=4) only started non-pharmacological therapy. Out of patients that started therapy in our FLS, 3 were switched to other therapies due to intolerance or new fractures. At their last appointments, most patients were under intravenous bisphosphonates (45.4%), followed by oral bisphosphonates (22.4%) and denosumab (22.1%), and lastly teriparatide (12.1%). The therapy sequencing is represented in graph 1.

Conclusion: Since October 2022, 182 patients have been observed at our FLS mostly due to recent vertebral fractures. Half were not under pharmacological treatment and started therapy at our FLS and 20% were switched to another medication. These findings underscore the need for FLS implementation, as a cru-

cial tool for secondary fragility fracture prevention. Identifying patients with fragility fractures, particularly vertebral fractures, frequently asymptomatic is an ongoing challenge.

162 - UNDERSTANDING "DIFFICULT-TO-TREAT" PSORIATIC ARTHRITIS: DATA FROM REUMA.PT

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Background: In clinical practice it is common to find psoriatic arthritis (PsA) patients with persistently high disease activity who fail to respond to multiple therapeutic agents. In 2020, the European Alliance of Associations for Rheumatology (EULAR) proposed a definition for difficult-to-treat (D2T) rheumatoid arthritis (RA). Recently, this concept was adapted to PsA, but there is no universally accepted definition of D2T PsA. Our aim is to estimate the prevalence of D2T PsA in clinical practice using two definitions and to identify clinical and demographic characteristics associated with D2T PsA.

Methods: Adult patients with the diagnosis of PsA as determined by the treating physician and registered in the Rheumatic Diseases Portuguese Register (Reuma.pt), with available follow-up information after September 2017 (date when 3 different mechanisms of action (MoA) became available for prescription) were included. Patients with exclusive axial involvement were excluded.

For the first definition of D2T PsA we adapted the EULAR definition of D2T RA, patients had to fulfil two criteria: 1) failure of ≥2 b/tsDMARD (with different MOA), 2) evidence of active disease, defined by the presence of one of the following: at least moderate disease activity according to validated composite measures (DAPSA>14); clinical activity (≥1 swollen and tender joint); persistently high acute phase reactants or inability to taper glucocorticoid treatment <7.5mg/day of prednisone or equivalent.

The second definition of D2T PsA was the failure of ≥3 b/tsDMARD (with different MOA).

Results: A total of 1875 patients were included, of whom 3.8% (n=72) were identified as D2T PsA according to the first definition. D2T PsA patients showed signs suggestive of active disease, with 73.6% having at least one swollen and tender joint, 65.3% having DAPSA>14, 22.2% unable to taper glucocorticoids and 12.5% having persistently high acute phase reactants. Compared to non D2T PsA, those with D2T PsA were younger at symptom onset (37.7±10.9 vs 41.5±12.7 years; p=0.014) and at diagnosis of PsA (40.7±10.5 vs 44.7±12.3 years; p=0.008). They presented more commonly as symmetrical polyarthritis (76.8% vs 53.6%, p<0.001), less commonly as asymmetric olygoarthritis (11.6% vs 31.5%; p<0.001) and were more likely to fulfil CASPAR criteria (97.1% vs 88.1%; p=0.027). Extra-articular manifestations, specifically enthesitis (31.5% vs 19.1%; p=0.025), were associated with D2T PsA.

Regarding comorbidities, there were no statistically significant differences between D2T and non-D2T PsA. However, the prevalence of depression was numerically higher among D2T PsA (10.6% vs 5%; p=0.078).

D2T PsA patients had a higher disease activity at the

TO 162 – TABLE 1. Descriptive analyses of clinical and demographic characteristics of patients with D2T and non-D2T PsA

	PsA (N=1875)	D2T PsA (n=72)	Non-D2T PsA (n=1803)	p-value
Female sex, n (%)	886 (47.3)	42 (58.3)	884 (46.8)	0.55
Caucasian, n (%)	1446 (98.3)	56 (94.9)	1390 (98.4)	0.075
Age, years	57 ± 12.5	55.4 ± 10.6	57.1 ± 12.6	0.205
Age at symptom onset, years	41.2 ± 12.7	37.7 ± 10.9	41.5 ± 12.7	0.014
Age at diagnosis, years	44.6 ± 12.3	40.7 ± 10.5	44.7 ± 12.3	0.008
CASPAR criteria, n (%)	1578 (88.8)	66 (97.1)	1512 (88.1)	0.027
PsA phenotype, n (%)				
Symmetric polyarthritis	973 (54.5)	53 (76.8)	920	<0.001
Asymmetric olygoarthritis	548 (30.7)	8 (11.6)	540 (31.5)	<0.001
Predominant axial disease	167 (9.4)	6 (8.7)	161 (9.4)	0.848
Distal interphalangeal	73 (4.1)	2 (2.9)	71 (4.1)	0.610
Mutilans	24 (1.4)	0	24 (1.3)	0.323
HLA-B27 positivity, n (%)	144 (16.1)	5 (14.3)	139 (16.2)	0.767
Rheumatoid factor, n (%)	69 (4.6)	4 (6.6)	65 (4.5)	0.460
Comorbidities (cumulative), n (%)				
Tobacco exposure	470 (34.5)	18 (35.3)	452 (34.5)	0.904
Alcohol consumption	340 (27.5)	12 (23.5)	328 (27.7)	0.518
Overweight	443 (27.4)	17 (29.3)	426	0.742
Obesity	364 (22.5)	9 (15.5)	355 (22.8)	0.195
Hypertension	394 (26)	16 (24.2)	378 (26)	0.746
Hypercholesterolemia	317 (20.9)	17 (25.8)	300 (20.7)	0.319
Diabetes mellitus	141 (9.3)	58 (12.1)	133 (9.2)	0.418
Cardiovascular disease	71 (4.7)	3 (4.5)	68 (4.7)	1.000
Cerebrovascular disease	19 (1.3)	1 (1.5)	18 (1.2)	0.572
Chronic kidney disease	10 (0.7)	1 (1.5)	9 (0.6)	0.359
Depression	79 (5.2)	7 (10.6)	72 (5)	0.078
Fibromyalgia	48 (3.2)	2 (3)	46 (3.2)	1.000
Cumulative extra-articular manifestations, n (%)	1344 (71.7)	62 (86.1)	1282 (71.1)	0.006
Psoriasis, n (%)	1231 (81)	54 (81.8)	1177 (80.9)	0.860
Dactylitis, n (%)	375 (26)	12 (24)	363 (26.1)	0.738
Enthesitis, n (%)	289 (19.6)	17 (31.5)	272 (19.1)	0.025
Nail dystrophy, n (%)	310 (21.5)	9 (18)	301 (21.7)	0.537
Uveitis, n (%)	55 (3.6)	3 (4.5)	52 (3.6)	0.680
Colitis, n (%)	18 (1.2)	0	18 (1.2)	*
Disease activity at start date of first b/tsDMARD				
Tender joints, 68 count	7 (9)	13 (14.5)	6 (8)	0.035
Swollen joints, 66 count	4 (6)	8.5 (9.5)	4 (6)	<0.001
ESR, mm/1 hour	22 (31)	26 (30)	21.5 (32)	0.375
CRP, mg/dL	0.86 (1.57)	1.1 (2.37)	0.81 (1.57)	0.180
VAS disease activity, 0-10	6.1 ± 2.3	6.7 ± 2.2	6.1 ± 2.4	0.139
VAS pain, 0-10	5.9 ± 2.4	6.6 ± 2.4	5.9 ± 2.4	0.312
DAPSA	25.8 (15.9)	36.9 (32.8)	25.4 (15.6)	0.015
Age beginning first b/tsDMARD, years	48.7 ± 11.6	45.6 ± 9.8	48.8 ± 11.7	0.015
Time until first b/tsDMARD, years	6.8 (9.7)	4.1 (8.8)	6.8 (9.7)	0.138
Duration first b/tsDMARD. months	33.4 (49.5)	20.3 (35.9)	34 (50)	0.022

*not possible to compute. PsA – Psoriatic Arthritis; D2T – Difficult-to-treat; CASPAR – Classification criteria for psoriatic arthritis; VAS – Visual Analogue Scale; ESR – Erythrocyte sedimentation rate; CRP – C-reactive protein; b/tsDMARD - biologic/targeted synthetic disease modifying anti-rheumatic drugs

beginning of first b/tsDMARD, with a higher tender and swollen joint count (13 (14.5) vs 6 (8); p=0.035; 8.5 (9.5) vs 4 (6); p<0.001) and DAPSA (36.9 (32.8) vs 25.4 (15.6); p=0.015).
On multivariate analysis a higher DAPSA at beginning of first b/tsDMARD was a predictor of D2T PsA (OR 1.04, 95%CI 1.01-1.06; p<0.001).
According to the second definition 0.9% (n=17) of patients could be classified as D2T PsA.
Conclusion: The proposed definitions, based mainly on the number of b/tsDMARDs previously used, identified a small proportion of patients with D2T PsA. Younger patients, those with enthesitis and with higher disease activity at first bDMARD onset were more likely difficult to manage. However, the proposed definitions do not accurately distinguish patients who are refractory to b/tsDMARDs from those who are difficult to manage due to other reasons, such as comorbidities.

163 - THE PROTOCOL OF THE TASTY TRIAL: THE TRIAD OF NUTRITION, INTESTINAL MICROBIOTA AND RHEUMATOID ARTHRITIS - AN EXPLORATORY PILOT STUDY

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Introduction: Immunologically active compounds from the gut microbiota are increasingly suggested to play a role in Rheumatoid Arthritis (RA) progression. It has been proposed that gut dysbiosis may impair intestinal barrier function, leading to an alteration in mucosal integrity and immunity, allowing for bacterial translocation, and ultimately contributing to the inflammation process. Since diet is recognized as one of the main environmental factors influencing gut microbiota, nutritional interventions aiming at the management of RA are being explored. The main goal of this pilot study is to investigate whether a dietary intervention based on a Mediterranean Diet enriched with fermented foods (MedDiet+) can impact gut microbiota and RA related outcomes.
Methods: 100 RA patients will be recruited at a tertiary center and randomly assigned to the intervention (MedDiet+) or control groups. Nutritional intervention (12 weeks) includes a personalized dietary plan based on a MedDiet+ style pattern, educational resources, food baskets delivery and clinical culinary workshops, all developed and monitored weekly by registered dietitians. The control group will receive general recommendations on a healthy diet at baseline. The effects of the nutritional intervention will be assessed on saliva and gut microbiota, gut permeability (Lactulose/Mannitol test, zonulin, IFABP), endotoxemia (evaluated by TLR4 reporter cells), inflammation (soluble CD14, faecal calprotectin), serum proteomics, glycolipids, gut derived immune cells, disease activity (Disease Activity Score in 28 joints, DAS28-ESR, and doppler ultrasound scores in 32 joints for grey scale and power doppler), functional status (Health Assessment Questionnaire) and quality of life (36-Item Short Form Survey). All procedures will be covered by national (Fundação para a Ciência e a Tecnologia, I. P. reference: 2022.07462.PTDC) and international (ENDOTARGET Horizon Europe project grant No 101095084) funding.
Results: We anticipate obtaining integrative information on the interplay between diet, gut and RA, while also exploring the mechanisms whereby these changes may occur, by analyzing all the biomarkers described above. The effects of the adherence to the MedDiet+ pattern will be assessed on oral and gut microbiota, gut permeability and inflammatory biomarkers, as well as their correlation to RA disease activity, functional status and quality of life of RA patients.
Conclusion: This multidisciplinary research with nutritionists, rheumatologists, biologists and immunologists aims to contribute to bridge the existing gap between nutrition-related knowledge and RA.

164 - SYNOVIAL SPECIMENS' DIAGNOSIS ACROSS DEPARTMENTS IN A PORTUGUESE TERTIARY CENTER

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Background: Synovial biopsies are conducted for clinical or research purposes. Their main aim in the clinical setting is assisting in arthritis diagnosis, complementing the synovial fluid analysis and other diagnostic tests, in particular if infections, synovial tumors, granulomatous or deposition diseases are suspected. The indication for synovial biopsies and the established diagnosis can be quite diverse across both medical and surgical departments.

Objectives: To compare the histopathological diagnosis of synovial specimens of biopsies with origin in the Rheumatology Department versus other Departments, in a Portuguese tertiary Center; To increase awareness for non-rheumatic synovitis etiologies in the rheumatology community.

Methods: This was a single center, cross sectional study of all synovial specimens received at the Pathology Department in Hospital de Santa Maria (Lisboa, Portugal), between 2010 and 2022. Histological diagnoses were classified as inflammatory or non-inflammatory lesions. Inflammatory lesions were further divided into non-granulomatous (low-grade or high-grade synovitis according to Krenn's Score and infectious arthritis); granulomatous, and crystal or foreign-body induced. Non-inflammatory lesions were subdivided into tumor-like and others. The categories were then matched for comparison according to its origin's Department (Rheumatology versus Orthopedics, Plastic Surgery, General Surgery, Stomatology, Internal Medicine, Infectious Diseases and Pediatrics as a group).

Results: A total of 598 reports pertaining to 539 patients (306 female, 56.8%) with an average age of 50.2 years old (ranging from 0 to 93) were reviewed. 212 specimens (35.4%) were from the Rheumatology Department while 386 (64.5%) from other Departments. Most of the cases from Rheumatology were classified as high-grade synovitis (n=112, 52.8%), whereas the largest group of the cases from other Departments were low-grade synovitis (n=108, 28.0%). The Rheu-

TO 164 – TABLE 1. Histological diagnosis distribution according to the Department of origin (Rheumatology versus other Departments)

Histological Diagnosis			Rheumatology	Other Departments
Inflammatory lesions	Non-granulomatous	Low-grade synovitis	19.3% (n=41)	28.0% (n=108)
		High-grade synovitis	52.8% (n=112)	13.2% (n=51)
		Infectious arthritis	6.6% (n=14)	12.2% (n=47)
	Granulomatous		1.4% (n=3)	4.4% (n=17)
	Crystal/ foreign body-induced	Monosodium urate (gout)	1.4% (n=3)	0.3% (n=1)
		Calcium pyrophosphate dihydrate (pseudogout)	1.9% (n=4)	1.0% (n=4)
		Hydroxyapatite microcrystals	0.9% (n=2)	0% (n=0)
		Metallosis	0% (n=0)	4.1% (n=16)
Non-inflammatory lesions	Tumor-like lesions	Pigmented vilonodular synovitis	0.9% (n=2)	8.0% (n=31)
		Lipoma arborescens	1.4% (n=3)	1.0% (n=4)
		Masson tumor	0% (n=0)	0.3% (n=1)
	Others	Synovial cyst/ ganglion	0% (n=0)	14.8% (n=57)
		Chondromatosis	0.5% (n=1)	2.8% (n=11)
		Hemarthrosis	0% (n=0)	0.5% (n=2)
		Hypertrophic osteoarthropathy	0% (n=0)	0.3% (n=1)
Insufficient for diagnosis			8.0% (n=17)	4.7% (n=18)
No pathological findings			4.7% (n=10)	4.4% (n=17)
Total			100% (n=212)	100% (n=386)

N = number

matology Department also had more cases of crystal induced diseases (4.2% vs 1.3%). Other Departments had a higher percentage of septic arthritis (12.2% vs 6.6%), granulomatous lesions (4.4% vs 1.4%), metallosis (4.1% vs 0%), tumor-like lesions (24.1% vs 2.4%) and chondromatosis (2.8% vs 0.5%). In the Rheumatology cases, after laboratory and histological results, there was a change in diagnosis in 61.8% (n=131) of the cases, mainly due to exclusion/ confirmation of septic arthritis and/or crystal-induced arthritis.

Conclusions: Synovial histopathological evaluation can be determinant for the diagnosis of a broad range of synovitis etiologies. As expected, most of the cases referred from the Rheumatology Department were high grade inflammatory lesions, whereas cases suggestive of other diagnosis such septic arthritis, tumor-like lesions or mechanical causes are mainly referred from other specialties. Taking in consideration that synovitis is a common manifestation of inflammatory and non-inflammatory diseases, the recognition of the relevance of integrating histological data in the diagnosis workup of synovitis is fundamental for a more precise medicine in the Rheumatology practice.

165 - HIGHER EDUCATIONAL LEVEL, FUNCTIONAL CAPACITY, AND DLCO ARE ASSOCIATED WITH HIGHER HEALTH-RELATED QUALITY OF LIFE IN SYSTEMIC SCLEROSIS PATIENTS – RESULTS FROM A CROSS-SECTIONAL COHORT STUDY

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Background: Systemic sclerosis (SSc) is an autoimmune disease characterized by fibrosis and vasculopathy. Although its course is somehow heterogeneous among affected patients, some of its clinical manifestations carry the potential for a significant impact on health-related quality of life (HRQoL) (1). It is possible to infer that several sociodemographic, psychosocial, and clinical factors may influence the impact of SSc on HRQoL. Understanding their relative contribution is key to an effective and holistic treatment approach for affected patients.

Objective: To evaluate HRQoL and identify its demographic and clinical correlates in an SSc cohort.

Methods: In this cross-sectional, unicentric study, consecutive patients with SSc, fulfilling the 2013 American College of Rheumatology / European League Against Rheumatism classification criteria for SSc, were enrolled. Variables were collected from the patients' registries at the last rheumatology appointment. HRQoL was measured using the EuroQol Five-Dimensional descriptive system (EQ-5D; ranging from –0.59 to 1, with 1 representing the best possible health state). Independent t-test, Mann-Whitney-U test, and Spearman's rank correlation coefficient were performed, as appropriate, to evaluate differences between groups. Variables with $p<0.1$ were included in a stepwise multiple linear regression analysis to assess the independent association of variables with the EQ-5D.

Results: We included 43 patients (86.0% female, mean age 63.0 ± 11.3 years). Clinical and sociodemographic variables are described in Table 1. The median EQ-5D score was 0.59 (IQR=0.48). There was a statistically significant difference in EQ-5D scores in patients with and without gastric involvement (p=0.030), lung involvement (p=0.016), and arterial pulmonary hypertension (p=0.011). EQ-5D was positively moderately correlated with the single-breath diffusing capacity of the lung

TO 165 – TABLE 1. Clinical and sociodemographic characteristics of included SSc patients

Female gender, n (%)	37.0 (86.0)
Age (mean, SD)	62.3 (11.5)
Disease duration, years, mean (SD)	8.1 (4.6)
Diffuse cutaneous subset, n (%)	9.0 (20.9)
Anti-centromere positive, n (%)	32.0 (74.4)
Anti-topoisomerase positive, n (%)	9.0 (20.9)
Modified Rodnan Skin score, median (IQR)	9.1 (12.0)
Digital ulcers, n (%)	19.0 (44.2)
Joint pain, n (%)	9.0 (20.9)
Esophageal involvement, n (%)	12.0 (27.9)
Gastric involvement, n (%)	19.0 (44.2)
Pulmonary involvement, n (%)	13.0 (30.2)
Interstitial lung disease, n (%)	12.0 (27.9)
Pulmonary arterial hypertension, n (%)	5.0 (11.6)
FVC, % of predicted, median (IQR)	98.0 (23.0)
DLCOsb, % of predicted, mean (SD)	72.9 (16.8)
EQ-5D, median (IQR)	0.59 (0.48)
SHAQ, median (IQR)	0.90 (1.8)
FACIT, median (IQR)	38.0 (21.0)
C reactive protein, mg/dL, median (IQR)	0.18 (0.44)
Years of schooling, median (IQR)	5.0 (8.0)
Full-time worker, n (%)	17.0 (39.5)
Retirement, n (%)	16.0 (37.2)
Unemployed, n (%)	3.0 (7.0)

N: number of patients. SD: standard deviation. IQR: interquartile range. FVC: forced vital capacity. DLCOsb: single-breath diffusing capacity of the lung for carbon monoxide. EQ-5D: EuroQol Five-Dimensional Descriptive System. SHAQ: Scleroderma Health Assessment Questionnaire. FACIT: Functional Assessment of Chronic Illness Therapy Fatigue Scale.

for carbon monoxide (DLCOsb) (r=0.62; p<0.001), and weakly with the forced vital capacity (FVC) (r=0.48; p=0.002) and years of schooling (r=0.31; p=0.041). EQ-5D showed a negative correlation with the Scleroderma Health Assessment Questionnaire (SHAQ) (r=-0.63; p<0.001), age (r=-0.39; p=0.017), and disease duration (r=-0.37; p=0.014). After multiple linear regression, EQ-5D showed a significant association with SHAQ (β=-0.138 [95% CI -0.232 to -0.043]; p=0.006), DLCOsb (β=0.006 [95% CI 0.002 to 0.011]; p=0.011), and years of schooling (β=0.025 [95% CI 0.011 to 0.040]; p=0.002); R2=0.612; p<0.001.

Discussion: In this cohort of SSc patients, higher HRQoL assessed by the EQ-5D was associated with higher educational level, lower functional impairment assessed by SHAQ, and higher levels of DLCOsb. This reflects the multifaceted nature of contributors to HRQoL, determined by both clinical and sociodemographic aspects.

REFERENCES:

1. van Leeuwen, Nina M., et al. "Health-related quality of life in patients with systemic sclerosis: evolution over time and main determinants." Rheumatology 60.8 (2021): 3646-3655.

167 - PAGET'S DISEASE OF BONE: CLINICAL AND EPIDEMIOLOGICAL CHARACTERIZATION OF THE POPULATION OF A PORTUGUESE TERTIARY CENTER

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Introduction: Paget's disease of bone (PDB) is the second most prevalent metabolic bone disorder, influenced by genetic and environmental factors which remain unclear.

Objectives: Characterize the clinical and demographic profile of a Portuguese PDB cohort. This is the first phase of a two-step project aimed at identifying environmental and genetic etiological factors in PDB.

Methods: Retrospective observational study including patients with PDB. Demographic and clinical data were collected. Occupations were classified according to the International Standard Classification of Occupations-2008. Binomial logistic regression was used to find independent associations with polyostotic involvement and need for retreatment.

Results: Eighty patients (58.8% females) with a diagnosis of PDB between 1974 and 2021 (mean age at diagnosis of 63.0±12.2 years) were included (Table 1). 72.5% of patients were born in rural areas. Their parent's birthplaces showed concentration in Alentejo (35.0/38.9%) and 81.3/86.3% were born in rural areas. Family history of PDB was reported in 13.9% of patients.

PDB commonly affected the pelvis (75.0%), sacrum (25.0%), femur (25.0%), vertebra (21.3%) and skull (13.8%), with 51.2% of patients having polyostotic involvement. Alkaline phosphatase (ALP) levels were elevated in 74.7% of cases at baseline, but this rate decreased to 2.7% 6-12 months after last treatment. Zoledronate was the most used drug (88.8%).

Polyostotic patients had higher exposure to goats (39.0% vs 12.8%, p=0.008), more years working in elementary occupations (16.5±21.7 vs 7.9±15.0, p=0.027) and fewer in professional occupations (2.0±8.9 vs 7.1±14.5, p=0.049). Polyostotic disease was associated with higher baseline levels of ALP (U/L) (200.0 [167.0] vs 149.0 [123.5], p=0.035), P1NP (ng/mL) (196.2 [188.1] vs 115.3 [75.8], p=0.015) and CTx (ng/mL) (0.7±0.3 vs 0.6 [0.3], p=0.031). Polyostotic patients had a higher frequency of involvement of the following bones: skull (22.0% vs 2.5%, p=0.029), pelvis (87.8% vs 61.5%, p=0.007), vertebrae (36.6% vs 5.1%, p<0.001), femur (39.0% vs 10.3%, p=0.003), and sacrum (36.6% vs 12.8%, p=0.014). In multivariate analysis, affection of vertebrae (OR 279.4 [10.2-7670.6], p<0.001), femur (OR 150.6 [7.2-3131.8], p=0.001), pelvis (OR 101.2 [7.1-1431.6.], p<0.001), sacrum (OR 61.6 [5.1-737.6], p=0.001) and skull (OR 47.5 [1.1-2052.5], p=0.044) were associated with polyostotic involvement, irrespective of sex, age at diagnosis, occupation and baseline ALP, CTx or P1NP.

Patients who underwent retreatment (n=18, 23.4%) were younger at diagnosis (57.0±11.0 vs 65.0±12.3, p=0.015), had higher baseline ALP levels (236.0 [254.5] vs 164.0 [113.8]; p=0.005), and more frequently had humerus involvement (5.2% vs 1.3%, p=0.010) than those who were not retreated (n=59). Additionally, retreated patients had received more often first line treatment with pamidronate (38.9% vs 1.7%, p<0.001) and less frequently with zoledronate (33.3% vs 91.5%, p<0.001). On multivariate analysis, zoledronate as first line therapy was negatively associated with retreatment (OR 0.048 [0.012-0.193], p<0.001), irrespective of sex, age at diagnosis, baseline ALP, pamidronate as first line or humeral affection.

TO 167 – TABLE 1. Clinical and epidemiological characterization of the cohort of Paget's disease of bone

Demographic data	N=80
Female, n(%)	47/80 (58.8)
Caucasian / White, n(%)	78/80 (97.8)
Age, years	73.7±11.0
Age at diagnosis, years	63.0±12.2
Disease duration, years [IQR]	8.0 [14.8]
Body Mass Index	
Underweight, n(%)	0/80
Normal weight, n(%)	20/80 (25.0)
Overweight, n(%)	42/80 (52.5)
Obesity class I, n(%)	14/80 (17.5)
Obesity class II, n(%)	3/80 (3.8)
Obesity class III, n(%)	1 (1.3)
Family history and residencies	
Family history of Paget's disease of bone, n(%)	11/79 (13.9)
Father's birthplace [Rural], n(%)	65/80 (81.3)
Mother's birthplace [Rural], n(%)	69/80 (86.3)
Consanguinity, n(%)	6/79 (7.6)*
Patient's birthplace [Rural], n(%)	58/80 (72.5)
Rural area, years living in	29.4±27.2
Urban area, years living in [IQR]	54.0 [55.0]
Occupation (years)	
1. Managers, years	1.6±7.0
2. Professionals, years	4.5±12.2
3. Technicians and associate professionals, years	2.5±8.1
4. Clerical support workers, years	2.8±9.2
5. Services and sales workers, years	8.4±16.3
6. Skilled agricultural, forestry and fishery workers, years	2.2±9.3
7. Craft and related trades workers, years	6.7±14.5
8. Plant and machine operators and assemblers, years	1.2±5.5
9. Elementary occupations, years	12.3±19.3
10. Armed forces occupations, years	1.1±4.9
Animals	
Domestic animals (time of exposure, years)	29.1±22.7
- Cat, n(%)	43/80 (53.8)
- Dog, n(%)	61/80 (76.3)
- Bird, n(%)	25/80 (31.3)
- Rabbit, n(%)	22/80 (27.5)
- Rodent, n(%)	2/80 (2.5)
- Turtle, n(%)	4/80 (5.0)
- Other, n(%)	1/80 (1.3)
Cattle animals (time of exposure, years) [IQR]	12.0 [25.0]
- Birds, n(%)	37/80 (46.3)
- Sheep, n(%)	25/80 (31.3)
- Pig, n(%)	25/80 (31.3)
- Donkey, n(%)	13/80 (16.3)
- Goat, n(%)	21/80 (26.3)
- Horse, n(%)	7/80 (8.8)
- Bovine, n(%)	22/80 (27.5)
Childhood infectious diseases	
Measles, n(%)	49/66 (74.2)
Mumps, n(%)	24/56 (42.9)
Chickenpox, n(%)	21/56 (37.5)
Rubella, n(%)	3/48 (6.3)
Diphteria, n(%)	0/49
Whooping cough, n(%)	7/51 (13.7)
Scarlet fever, n(%)	2/50 (4.0)
Comorbidities	
Arterial hypertension, n(%)	61/80 (76.3)
Dyslipidemia, n(%)	51/80 (63.7)
Ischemic heart disease, n(%)	11/80 (13.8)

TO 167 – TABLE 1. Continuation

Valvulopathies, n(%)	9/80 (11.3)
Stroke, n(%)	4/80 (5.0)
Smoking, n(%)	32/80 (40.0)
Topography	
Skull, n(%)	11/80 (13.8)
Face, n(%)	2/80 (2.5)
Pelvis, n(%)	60/80 (75.0)
Vertebra, n(%)	17/80 (21.3)
Sacrum, n(%)	20/80 (25.0)
Femur, n(%)	20/80 (25.0)
Tibia, n(%)	5/80 (6.3)
Fibula, n(%)	0
Scapula, n(%)	2/80 (2.5)
Humerus, n(%)	5/80 (6.3)
Radius, n(%)	2/80 (2.5)
Ulna, n(%)	0
Other bones, n(%)	7/80 (8.8)
Howarth's table, %	5.2 [5.3]
Polyostotic, n(%)	41/80 (51.2)
Complications	
Osteoarticular pain, n(%)	64/80 (78.8)
Secondary osteoarthritis, n(%)	27/80 (33.8)
Blood work at the time of diagnosis or first determination before treatment	
Elevated alkaline phosphatase, n(%)	56/80 (74.7)
Alkaline phosphatase, U/L [IQR]	180.0 [123.0]
Elevated calcium, n(%)	8/80 (11.0)
Reduced phosphorus, n(%)	6/80 (8.5)
Elevated parathyroid hormone, n(%)	11/80 (19.3)
Reduced 25-hydroxy vitamin D, n(%)	19/80 (37.3)
Elevated P1NP, n(%)	46/80 (94.9)
P1NP, ng/mL	142.3 [142.8]
Elevated CTx, n(%)	28/80 (46.7)
CTx, ng/mL	0.6±0.3
Pharmacological treatment	
Calcitonin, n(%)	3/80 (3.8)
Etidronate, n(%)	1/80 (1.3)
Clodronate, n(%)	1/80 (1.3)
Alendronate, n(%)	6/80 (7.5)
Risedronate, n(%)	1/80 (1.3)
Pamidronate, n(%)	10/80 (12.5)
Zoledronate, n(%)	71/80 (88.8)
Patients retreated, n(%)	18/77 (23.4)
Number of drugs used to treat PDB per patient	
0, n(%)	4/80 (5.0)
1, n(%)	63/80 (78.8)
2, n(%)	10/80 (12.5)
3, n(%)	2/80 (2.5)
4, n(%)	1/80 (1.3)
Bone remodelling markers 6-12 months after treatment	
Elevated alkaline phosphatase, n(%)	2/73 (2.7)
Alkaline phosphatase, U/L [IQR]	58/73 [25.5]
Elevated P1NP, n(%)	10/67 (14.9)
P1NP, ng/mL [IQR]	23.4 [13.9]
Elevated CTx, n(%)	1/67 (1.5)
CTx, ng/mL [IQR]	0.2 [0.1]

Results presented as mean±standard deviation or median [interquartile range] or n (%)

Conclusion: To the best of our knowledge, this study describes the largest Portuguese cohort of PDB patients. A high prevalence of this disease in rural areas and a high biochemical remission rate after treatment

were noted. Zoledronate as first line therapy seems to be protective against retreatment. A genetic characterization of this cohort is currently under work.

169 - GLUCOCORTICOIDS IN SYSTEMIC SCLEROSIS

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Background: Systemic sclerosis (SSc) is a heterogeneous disease regarding clinical and immunological manifestations, progression, and prognosis. It has significant morbimortality, especially when major organ involvement is present, making its management a significant challenge. Unlike other immune-mediated diseases in which glucocorticoids (GC) are widely used with robust clinical evidence, their role in SSc is controversial. Although GCs may be useful to manage some inflammatory manifestations of SSc, their use has been associated with an increased risk of Scleroderma Renal Crisis (SRC). As a result, the use of GC in SSc patients in clinical practice is highly variable among rheumatologists.
Objectives: To characterize the pattern of GC use in patients with SSc, as well as the reasons for GC prescription, its benefits and risks, particularly concerning SRC development.
Methods: Retrospective study of patients with SSc diagnosis according to ACR/EULAR 2013 criteria, followed at Hospital Garcia de Orta rheumatology department and registered in Reuma.pt database. A descriptive analysis was conducted to characterize the treatments performed with GC. Patients with SSc were divided into two groups: those who received at least one GC treatment and those who never received GC treatment. The comparison between the two groups was performed using the chi-square test, Student's T-test, or Mann-Whitney test, as appropriate. The threshold for statistical significance was a p-value inferior to 0.05.
Results: 157 patients with SSc were included, of whom 47.1% were treated with GC. Patients exposed to GC more often had a SSc-overlap syndrome phenotype, digital ulcers, myositis, and diabetes mellitus. Those receiving GC were also more likely treated with other immunosuppressants. Most patients received only one course of systemic GC, however 33.6% were treated twice or more, with a total of 111 treatments performed. The most frequently used GC was oral prednisolone, and musculoskeletal involvement was the most frequent reason. Musculoskeletal and skin involvement were more frequently treated with low (≤7.5mg/day) and intermediate (>7.5mg/day and ≤30mg/day) doses while lung involvement was more frequently treated with high doses (>30mg/day). Serositis was the SSc manifestation with the best clinical response to GC. Only one case of SRC was reported in a patient that was never treated with GC.
Conclusion: Despite safety concerns, GC are frequently used in clinical practice to treat SSc, particularly musculoskeletal, skin and lung manifestations. Our data suggest a therapeutic benefit of GC in the treatment of SSc and its use was not associated with the development of SRC in our cohort.

171 - AXIAL PSORIATIC ARTHRITIS VERSUS AXIAL SPONDYLOARTHRITIS WITH PSORIASIS IN CLINICAL PRACTICE

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Background: Spondyloarthritis (SpA) comprise a group of inflammatory arthropathies that can involve both axial and peripheral joints, often associated with extra-musculoskeletal manifestations, particularly psoriasis. The distinction between Psoriatic Arthritis (PsA) with axial involvement and axial SpA (axSpA) with concomitant psoriasis has been a topic of debate, potentially influencing clinical decisions.
Objectives: To identify clinical and demographic characteristics associated with clinician's decision to diagnose patients with psoriasis and axial disease as axSpA or as PsA with axial involvement in practical clinical settings.
Methods: All adult patients registered in the Rheumatic Portuguese Disease Register (Reuma.pt) with the clinical diagnosis of PsA with axial involvement (axPsA) or axSpA with concomitant psoriasis were included.
Results: Out of 854 patients, 88.3% (n=754) received a diagnosis of axPsA and 11.7% (n=100) were diagnosed with axSpA with psoriasis.
The diagnosis of axPsA included concomitant peripheral involvement in 82.2% of patients (n=620) and exclusive axial involvement in 17.8% (n=134). In axSpA diagnosis, the prevalence of concomitant peripheral involvement was 48% (n=48).
Considering the whole cohort, axSpA diagnosis was associated with less peripheral involvement (48% vs 82.2%; p<0.001), younger age at diagnosis (axSpA 36.7 ± 9.4 vs axPsA 43.8 ± 13.1; p<0.001) and at symptom onset (axSpA 30.1 ± 9.7 vs axPsA 39.6 ± 12.9; p<0.001) and higher positivity for HLA-B27 (75% vs 25.1%; p<0.001) (Table 1). Regarding extra-articular manifestations, uveitis (OR 7.59; 95% CI 4.5-12.81; p<0.001) and inflammatory bowel disease (OR 21.02; 95% CI 8.01-55.18; p<0.001) were associated positively, while dactylitis was associated negatively with axSpA diagnosis (OR 0.13; 95% CI 0.06-0.29; p<0.001). axSpA patients more commonly received bDMARDs (OR 3.82; 2.13-6.84; p<0.001) and had a longer time from symptom onset until start of first bDMARD (axSpA 13.7 ± 9.3 vs axPsA 9.5 ± 9.1; p<0.001). Cardiovascular comorbidities were negatively associated with axSpA (OR 0.53; 95% CI 0.32 - 0.88; p<0.013). Multivariate analysis identified HLA-B27 positivity, younger age at symptom onset, presence of uveitis and inflammatory bowel disease and lower prevalence of dactylitis as independently associated with axSpA diagnosis.
When considering patients with exclusive axial involvement (N=186), axSpA diagnosis was associated

TO 171 – TABLE 1. Demographic and clinical characteristics of patients with axial involvement

	Axial involvement (N=854)	axPsA diagnosis (n=754)	axSpA diagnosis (n=100)	p-value	Odds Ratio (95% Confidence Interval)
Female sex - no (%)	342 (40)	299 (39.7)	43 (43)	0.521	1.14 (0.753 - 1.75)
Caucasian - no (%)	645 (98.6)	559 (98.6)	86 (98.9)	1	0.81 (0.1 – 6.58)
Concomitant Peripheral Involvement	668 (78.2)	620 (82.2)	48 (48)	<0.001	0.20 (0.13 - 0.31)
PsA Phenotype - no (%)	754 (88.3)	754 (100)	0	**	
Predominant axial involvement - no (%)	340 (39.8)	340 (45.1)	0		
Symmetric polyarthritis - no (%)	242 (28.3)	242 (32.1)	0		
Predominant distal interphalangeal - no (%)	18 (2.1)	18 (2.4)	0		
Asymmetric olygoarthritis - no (%)	148 (17.3)	148 (19.6)	0		
Mutilans - no (%)	6 (0.7)	6 (0.8)	0		
Age at diagnosis - years	42.9 ± 12.9	43.8 ± 13.1	36.7 ± 9.36	<0.001	
Age at symptom onset - years	38.5 ± 13.0	39.6 ± 12.9	30.1 ± 9.67	<0.001	
HLA-B27 positivity - no (%)	183 (32.6)	120 (25.1)	63 (75)	<0.001	8.95 (5.24 - 15.3)
Cardiovascular Disease/Risk Factors - no (%)	243 (31.7)	222 (33.3)	21 (21)	0.013	0.53 (0.32 - 0.88)
Extra-articular Manifestations					
Psoriasis - no (%)	829 (98)	729 (97.7)	100 (100)	0.246	1.13 (1.11-1.16)
Uveitis - no (%)	74 (9.6)	41 (6.1)	33 (33)	<0.001	7.59 (4.5 - 12.8)
Enthesitis - no (%)	283 (35.9)	247 (35.9)	36 (36)	0.985	1.0 (0.65 - 1.56)
Dactylitis - no (%)	257 (32.7)	250 (36.4)	7 (7)	<0.001	0.13 (0.06 - 0.29)
Inflammatory Bowel Disease - no (%)	22 (2.9)	6 (0.9)	16 (16)	<0.001	21.02 (8.01 - 55.2)
Treatment with bDMARDs – no (%)	551 (64.5)	465 (61.7)	86 (86)	<0.001	3.82 (2.13 - 6.84)
Time until first bDMARD - years	7 (12)	9.47 ± 9.05	13.7 ± 9.33	<0.001	

TO 171 – TABLE 2. Demographic and clinical characteristics of patients with exclusive axial involvement

	Exclusive Axial Involvement (N=186)	axPsA diagnosis (n=134)	axSpA diagnosis (n=52)	p-value	Odds Ratio (95% Confidence Interval)
Female sex - no (%)	74 (39.8)	56 (41.8)	18 (34.6)	0.370	0.737 (0.38 - 1.43)
Caucasian - no (%)	128 (98.5)	84 (98.8)	44 (97.8)	0.645	1.91 (0.12 - 31.3)
Age at diagnosis – years	41.7 ± 13.3	43.9 ± 13.9	36.4 ± 10.3	**<0.001**	
Age at symptom onset – years	35.7 ± 13.3	38.3 ± 13.6	29.3 ± 10.1	**<0.001**	
HLA-B27 positivity - no (%)	73 (50.7)	35 (35.7)	38 (82.6)	**<0.001**	8.55 (3.59 - 20.4)
Cardiovascular Disease/Risk Factors - no (%)	43 (26.4)	32 (28.8)	11 (21.2)	0.300	0.66 (0.30 -1.45)
Extra-articular manifestations					
Psoriasis - no (%)	186 (100)	134 (100)	52 (100)	**	
Uveitis - no (%)	27 (16.2)	8 (7)	19 (36.5)	**<0.001**	7.7 (3.09 - 19.2)
Enthesitis - no (%)	32 (19.3)	21 (18.4)	11 (21.2)	0.679	1.19 (0.53 - 2.69)
Dactylitis - no (%)	10 (6.1)	9 (8)	1 (1.9)	0.173	0.23 (0.03 - 1.84)
Inflammatory Bowel Disease- no (%)	20 (1.1)	0	8 (15.4)	**	
Treatment with bDMARDs – no (%)	106 (57)	64 (47.8)	42 (80.8)	**<0.001**	4.59 (2.13 - 9.90)
Time until first bDMARD - years	10 (15)	8.47 ± 7.99	14.8 ± 10.2	**<0.001**	

** Not possible to compute.

bDMARD – biologic disease modifying rheumatic drugs.

with a younger age at diagnosis (axSpA 36.4 ± 10.3; vs axPsA 43.9 ± 13.8; p<0.001) and at symptom onset (axSpA 29.3± 10.1 vs axPsA 38.3 ± 13.6; p<0.001), a higher prevalence of HLA-B27 (OR 8.55; 95% CI 3.59-20.4; p<0.001) and uveitis (OR 7.7; 95% CI 3.0-19.2; p<0.001) (Table 2). Regarding treatment, axSpA patients were more frequently treated with bDMARDs (OR 4.59; 95% CI 2.13 - 9.90; p<0.001) and had a longer time from symptom onset until start of first bDMARD (axSpA 14.8 ± 10.2 vs axPsA 8.5 ± 7.9; p<0.001). However, multivariate analysis did not identify any variables independently associated with the diagnosis of axPsA or axSpA.

Conclusions: Patients with axPsA diagnosis exhibit more frequently concomitant peripheral involvement, whereas those with axSpA diagnosis have more often exclusive axial involvement, are younger and more frequently HLA-B27 positive. Considering patients with exclusive axial involvement, no demographic or clinical variables were independently associated with the diagnosis of axPsA or axSpA with psoriasis.

176 - UNDERSTANDING AXIAL PSORIATIC ARTHRITIS IN CLINICAL PRACTICE - DATA FROM REUMA.PT

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Background: Psoriatic Arthritis (PsA) can be categorized as either axial or predominantly peripheral disease. Axial Psoriatic Arthritis (axPsA) may occur exclusively or concomitantly with peripheral involvement. The prevalence of exclusive axPsA is estimated at 5% of patients increasing to 25-70% when associated with peripheral arthritis. However, the absence of a consensus definition for axPsA makes the real prevalence difficult to estimate.

Objectives: 1) determine the proportion of axPsA among PsA patients and understand how it is diagnosed in clinical practice; 2) identify clinical and demographic characteristics associated with axPsA.

Methods: Adult patients with the diagnosis of PsA or

TO 176 – Table. Demographic and clinical characteristics of PsA patients according to the phenotype

	PsA (total) (N=2304)	axPsA (N=854)	Exclusive Peripheral PsA (N=1450)	p-value	Odds Ratio (95% Confidence Interval)
Female sex - no (%)	1078 (53.2)	342 (40.0)	736 (50.8)	<0.001	0.65 (0.55 - 0.77)
Age at diagnosis – years	44.9 ± 12.7	42.9 ± 12.9	46.2 ± 12.50	<0.001	
Age at symptom onset – years	41.3 ± 13.0	38.5 ± 13.0	43.0 ± 12.70	<0.001	
Caucasian - no (%)	1639 (98.4)	645 (98.6)	994 (98.1)	0.600	0.73 (0.33 - 1.62)
PsA Phenotypes- no (%)	2204 (95.7)	754 (88.3)	1450 (100.0)		
Predominant axial involvement - no (%)	440 (19.1)	440 (51.5)	0		
Symmetric polyarthritis - no (%)	1106 (48.0)	242 (28.3)	864 (59.6)		
Predominant distal interphalangeal - no (%)	97 (4.2)	18 (2.1)	79 (5.4)		
Asymmetric olygoarthritis - no (%)	636 (27.6)	148 (17.3)	488 (33.7)		
Mutilans - no (%)	25 (1.1)	6 (0.7)	19 (1.3)		
HLA-B27 positivity - no (%)	272 (21.0)	183 (32.6)	89 (12.1)	<0.001	3.50 (0.64 - 4.65)
Comorbidities					
Obesity - no (%)	407 (19.4)	149 (18.8)	258 (19.8)	0.609	0.94 (0.75 - 1.12)
Cardiovascular disease or risk factors - no (%)	668 (33.3)	243 (31.7)	425 (34.3)	0.234	0.89 (0.73 - 1.08)
Extra-articular manifestations					
Psoriasis - no (%)	2203 (95.6)	829 (98.0)	1374 (96.5)	0.041	1.78 (1.02 - 3.10)
Nail dystrophy - no (%)	831 (36.1)	284 (35.9)	547 (41.8)	0.009	0.78 (0.65 - 0.94)
Uveitis - no (%)	105 (4.6)	74 (9.6)	31 (2.5)	<0.001	4.17 (2.71 - 6.41)
Enthesitis - no (%)	613 (29.6)	283 (35.9)	330 (25.7)	<0.001	1.61 (1.33 - 1.96)
Dactylitis - no (%)	781 (37.4)	257 (32.7)	524 (40.2)	<0.001	0.72 (0.59 - 0.87)
Inflammatory Bowel Disease- no (%)	28 (1.4)	22 (2.9)	6 (0.5)	<0.001	6.07 (2.45 - 15.04)
Tobacco exposure - no (%)	586 (36.5)	262 (41.3)	324 (33.3)	0.001	1.41 (1.15-1.73)
Alcohol consumption - no (%)	387 (18.2)	174 (21.8)	213 (16.0)	<0.001	1.46 (1.17-1.83)
Age at start date of first bDMARD - years	47.9 ± 11.5	46.6 ± 11.8	48.8 ± 11.1	<0.001	
Time until start of first bDMARD – years	7 (10)	7 (12)	6 (9)	0.003	

**not possible to compute. PsA – Psoriatic Arthritis; SpA – Spondyloarthritis; ACPA – anti-citrullinated protein antibody; bDMARD – biologic disease modifying anti-rheumatic drugs.

the diagnosis of axSpA with psoriasis registered in the Rheumatic Diseases Portuguese Registry (Reuma.pt) and fulfilling CASPAR criteria were included. Axial involvement was defined as either physician-reported spondylitis or the presence of imaging findings suggestive of axial involvement (radiographic sacroiliitis (SI) according to modified New York Criteria (mNYC), SI in magnetic resonance imaging (MRI), or the presence of syndesmophytes in axial radiography).

Results: A total of 2304 patients were included. The prevalence of axPsA was 37.1% (N=854), with 21.8% (N=186) having exclusive axPsA and 78.2% (N=668) having concomitant peripheral involvement.

The diagnosis of axPsA was made based on suggestive imaging findings in 30.1% of patients, with radiographic SI being the most common (N=195; 75.9%) followed by the presence of syndesmophytes (N=93; 36.2%) and SI on MRI (N=54; 21%). In the remaining 69.9% of cases, spondylitis was physician-reported (imaging exams inaccessible).

axPsA was associated with male sex (OR=1.54; 95% CI 1.30-1.81; p<0.001), positivity for HLA-B27 (OR=3.5; 95% CI 2.64-4.65; p<0.001), a younger age at diagnosis (42.9 ± 12.9 vs 46.2 ± 12.5; p<0.001) and at symptom onset (38.5 ± 13 vs 43.0 ± 12.7; p<0.001) (Table). Regarding extra-articular manifestations, axPsA was associated with a higher proportion of enthesitis (OR=1.61; 95% CI 1.33-1.96; p<0.001), uveitis (OR=4.17; 95% CI 2.71-6.41; p<0.001), psoriasis (OR=1.78; 95% CI 1.02-3.1; p=0.041) and inflammatory bowel disease (OR= 6.07; 95% CI 2.45-15.04; p<0.001) and lower proportion of dactylitis (OR=0.72; 95% CI 0.59-0.87; p<0.001) and nail dystrophy (OR=0.78; 95% CI 0.65-0.94; p=0.008). Current or previous tobacco exposure and

alcohol consumption were also associated with axPsA (OR=1.41; 95% CI 1.15-1.73; p=0.001 and OR=1.46; 95% CI 1.17-1.83; p<0.001, respectively). Patients with axPsA started therapy with first bDMARD at younger age (46.6 ± 11.8 vs 48.8 ± 11.1; p<0.001), however the time from symptom onset until start date of first bDMARD was longer (7 (12) vs 6 (9) years; p=0.003). Multivariate analysis identified HLA-B27 positivity, dactylitis, enthesitis and tobacco exposure as independently associated with axPsA.

Conclusions: The prevalence of exclusive axPsA was 8.1%, increasing to 37.1% when considered concomitant with peripheral involvement. The most common diagnosis was physician-reported spondylitis; however, when considering imaging findings, radiographic SI was the most prevalent. We cannot rule out the possibility of underdiagnosis of axPsA, particularly cases of subclinical disease, given the limited availability of imaging exams. Patients with axPsA exhibited distinct clinical and laboratory features compared to peripheral PsA, with a higher prevalence of HLA-B27 positivity, enthesitis and tobacco exposure, and lower prevalence of dactylitis.

177 - REGRESSION OF RHEUMATOID NODULES WITH JAK INHIBITORS: A SINGLE-CENTER DESCRIPTIVE STUDY

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Introduction: Rheumatoid nodules (RNs) are the most common extra-articular manifestations among rheumatoid arthritis (RA) patients and are associated with a worse prognosis. Subcutaneous RNs are the most prevalent, being present in 20-30% of them. The most frequently affected non-subcutaneous site is the lung. Usually, RNs are asymptomatic but some complications can occur, such as ulceration and infection. The therapeutic options for RNs are limited. In the literature, there are some case reports of regression of pulmonary RNs with Janus kinase inhibitors (JAKi). To the authors' knowledge, no studies have yet demonstrated this effect on RNs in other locations, namely subcutaneous ones. The aim of this study was to evaluate the regression of RNs in RA patients with current or past therapy with a JAKi.

Methods: We developed a retrospective observational study, including all RA patients from our center, entered in the Reuma.pt registry, on current or past therapy with JAKi. The presence of RNs was searched through clinical records. The assessment of RNs regression was made by consulting clinical records and by patients' self-reports. Sociodemographic data, disease characteristics and previous therapies were also collected and analyzed with independent t-test and chi-square analyses. Differences were considered statistically significant at p<0,05.

Results: Our sample included 79 RA patients under JAKi therapy. Forty-four (55,70%) were taking upadacitinib, 27 (34,18%) baricitinib and 8 (10,13%) tofacitinib. The mean age was 56 (±8,61) years and 70 (88,6%) were female. The disease duration was on average 17 (±9,43) years. The majority of patients presented with bone erosions (62,50%) and high titers of anti-citrullinated protein antibodies (83,33%) and rheumatoid factor (70,51%). There were 25 (31,65%) patients with manifestation of RNs, one of them with pulmonary RNs. Fourteen (66,67%) patients showed regression of their RNs, compared to 7 (33,33%) without regression. Regression was observed with all JAKi, 8 (57,14%) with upadacitinib, 5 (35,71%) with baricitinib and 1 (7,14%) with tofacitinib. The patient with pulmonary RNs was on baricitinib and regression was documented on thoracic computed tomography. Eight (61,54%) patients with RNs regression were off any conventional synthetic disease-modifying anti-rheumatic drugs (csDMARD) in combination with JAKi. In the group without regression, only 2 (28,57%) were without any associated csDMARDs. No statistical differences were found between the groups with and without regression concerning sociodemographic data, disease characteristics or previous therapies.

Conclusion: JAKi are a recent treatment option for RA and in clinical practice they appear to be a promising drug for RNs reduction. No other therapy option, nor csDMARDs nor biological-DMARDs, has presented evidence of RNs regression. In our study, the majority of patients with RNs regression were in JAKi monotherapy. On the other hand, in the non-regression group, most patients were treated with JAKi in combination with a csDMARD. Although the differences were not statistically significant, these facts may indicate that the effect on RNs is not due to the synergism of any csDMARDs. Regression of RNs was observed with different JAKi, which can indicate a class effect. Research studies with JAKi in RA that include evalution of this outcome are greatly needed to better recognize and understand these effects.

TO 177 – TABLE 1. Descriptive analysis of RA patients medicated with Janus kinase inhibitors

	Rheumatoid Nodules			Rheumatoid Nodules Evolution		
	Present	Absent	Total	Regression	No change	Total
Characteristics	**N=25 (31,65%)**	**N=54 (68,35%)**	**N=79**	**N=14 * (66,67%)**	**N=7 * (33,33%)**	**N=21***
Age, years	56 (±8,54)	57 (±11,14)	56 (±10,34)	54 (±9,02)	59 (±7,00)	56 (±8,61)
Sex						
Female	21 (84,00%)	49 (90,74%)	70 (88,61%)	11 (84,62%)	7 (100,00%)	18 (85,71%)
Male	4 (16,00%)	5 (9,26%)	9 (11,39%)	3 (21,43%)	0 (0,00%)	2 (14,29%)
Disease duration, years	17 (±8,97)	17 (±10,47)	17(±9,95)	17 (±9,46)	18 (±10,08)	17 (±9,43)
Anti-citrullinated protein antibodies						
High	23 (92,00%)	42 (79,25%)	65 (83,33%)	13 (92,86%)	6 (85,71%)	19 (90,48%)
Low	2 (8,00%)	11 (20,75%)	13 (16,67%)	1 (7,14%)	1 (14,29%)	2 (9,52%)
Rheumatoid Factor						
High	19 (76,00%)	36 (67,92%)	55 (70,51%)	11 (78,57%)	5 (71,43%)	16 (76,19%)
Low	6 (24,00%)	17 (32,08%)	23 (29,49%)	3 (21,43%)	2 (28,57%)	5 (23,81%)
Bone erosion						
Present	15 (71,43%)	20 (57,14%)	35 (62,50%)	7 (63,64%)	5 (83,33%)	12 (70,59%)
Absent	6 (28,57%)	15 (42,86%)	21 (37,50%)	4 (36,36%)	1 (16,67%)	5 (29,41%)
DAS28 before JAKi	4,162 (±1,31)	4,802 (±1,15)	4,580 (±1,24)	3,826 (±1,48)	4,379 (±1,01)	4,017 (±1,34)
DAS28 after 6 month of JAKi	3,347 (±1,01)	3,604 (±1,12)	3,521 (±1,08)	3,191 (±1,06)	3,616 (±1,04)	3,340 (±1,05)
JAKi used						
Upadacitinib	14 (56,00%)	30 (55,56%)	44 (55,70%)	8 (57,14%)	4 (57,14%)	12 (57,14%)
Baricitinib	10 (40,00%)	17 (31,48%)	27 (34,18%)	5 (35,71%)	3 (42,86%)	8 (38,10%)
Tofacitinib	1 (4,00%)	7 (12,96%)	8 (10,13%)	1 (7,14%)	0 (0,00%)	1 (4,76%)
JAKi duration, years	2,1 (±1,63)	2,3 (±1,74)	2,2 (±1,69)	1,9 (±0,79)	1,9 (±0,90)	1,9 (±0,80)
bDMARD previous used						
Yes	20 (80,00%)	42 (79,25%)	62 (79,49%)	12 (85,71%)	6 (85,71%)	18 (85,71%)
No	5 (20,00%)	11 (20,75%)	16 (20,51%)	2 (14,29%)	1 (14,29%)	3 (14,29%)
csDMARDS therapy in association with JAKi						
Methotrexate	4 (16,67%)	22 (40,74%)	26 (33,33%)	1 (7,69%)	1 (14,29%)	2 (10,00%)
Leflunomide	5 (20,83%)	12 (22,22%)	17 (21,79%)	3 (23,08%)	2 (28,57%)	5 (25,00%)
Hydroxychloroquine	3 (12,50%)	2 (3,70%)	5 (6,41%)	1 (7,69%)	2 (28,57%)	3 (15,00%)
Sulfasalazine	0 (0%)	3 (5,56%)	3 (3,85%)	0 (0%)	0 (0%)	0 (0%)
No csDMARDs in simultaneous	12 (50,00%)	15 (27,78%)	27 (34,62%)	8 (61,54%)	2 (28,57%)	10(50,00%)

JAKi, Janus kinase inhibitors. DAS28, Disease Activity Score 28. csDMARD, conventional synthetic disease-modifying anti-rheumatic drugs. bDMARD, biological disease-modifying anti-rheumatic drugs. *Four of the patients with a clinical record of rheumatoid nodules did not have any information regarding their evolution, and contacting these patients was not possible.

179 - MEDICATION-RELATED OSTEONECROSIS OF THE JAW AND ATYPICAL FEMUR FRACTURES IN OSTEOPOROSIS: A SINGLE-CENTER CASE-SERIES

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Introduction: Medication-related osteonecrosis of the jaw (ONJ) and atypical femur fractures (AFF), although rare, are serious adverse events associated with osteoporosis (OP) antiresorptive therapy. The main OP antiresorptive drugs are bisphosphonates (oral and intravenous) and denosumab. The aim of this study is to present all the cases of ONJ and AFF secondary to OP antiresorptive treatment of our center in the last 10 years, evaluate the characteristics of the patients, the therapeutic options and following approach.
Methods: Our study is a retrospective descriptive analysis of ONJ and AFF cases secondary to antiresorptive therapy for OP in our center. Sociodemographic, presence of previous fractures, densitometric and therapeutic data were collected. Descriptive analysis was performed with mean and standard deviation for continuous variables and absolute and percentage frequency for categorical variables. Independent t-test and chi-square tests were performed. Differences were considered statistically significant at $p<0.05$.
Results: Twelve patients were included, 9 with ONJ and 3 with AFF. All patients were female with a mean age of 70±8 years. Mean OP duration was 12±5 years. Half of the patients had previous history of fragility fractures. Three patients (33.3%) developed denosumab-related ONJ and 6 (66,7%) bisphosphonates-related ONJ [3 (33.3%) with zoledronate, 2 (22.2%) with alendronate, and 1 (11.1%) with pamidronate]. All patients with AFF were on alendronate, one of them had previously been medicated with ibandronate. The duration of the therapy was longer in patients with AFF (11.3±6.5 years) compared to those with ONJ (3.6±2.7years). Denosumab showed a statistically significant (p=0,011) shorter therapy duration (1.7±0,289years) when compared to bisphosphonates (7.1±4,986years). Five (55.6%) of the patients with ONJ had rheumatoid arthritis and 1 (11.1%) had spondyloarthritis. None of the patients with AFF had other associated rheumatic diseases. All patients suspended immediately the drug when the adverse effect was observed. From the twelve patients, only four (33.3%) received subsequent therapy for OP. Subsequent therapy with teriparatide was used in 2 patients with ONJ and in 1 with AFF, while denosumab was used in 1 patient with AFF. Densitometric reevalution was performed only in 2 patients, both showing vertebral improvement.
Conclusion: In our sample, the drug associated to ONJ and AFF was suspended in all cases. However, in the literature, the recommendation to stop denosumab after the event is controversial. We realized that occurrence of ONJ with denosumab happened after few administrations and in a shorter time compared to bisphosphonates. After the occurrence of ONJ and AFF and when subsequent therapy for OP is mandatory due to high fracture risk of the individual, the choice between treatments is challenging. Teriparatide is the best alternative after bisphosphonate adverse event and some studies have shown benefits in tissue healing, either in ONJ and in AFF. Nevertheless, the use of teriparatide after denosumab has demonstrated bone mass loss and increased fracture risk. After 2 years of teriparatide, subsequent therapy is recommended to avoid rebound effects. Unfortunately, the therapy options at this point are even more limited. The question "how should we treat these patients?" remains unanswered.

180 - EXPLORING OCCUPATIONAL BACKGROUND IN SYSTEMIC SCLEROSIS – INSIGHTS FROM A SINGLE - CENTER STUDY

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Introduction: Systemic sclerosis (SSc) is a complex immune-mediated connective tissue disease, characterized by vasculopathy and fibrosis of the skin and internal organs, with significant morbidity and mortality. The pathogenesis of SSc is not fully understood but like other autoimmune disorders, is believed to be induced by an environmental trigger in a genetically predisposed host. Evidence of geographic clustering provides insight into the significance of environmental factors.
Our study aimed to describe SSc patients' occupational background and explore its association with clinical

TO 179 – TABLE 1. Descriptive analysis of osteonecrosis of the jaw and atypical femur fractures

	Osteonecrosis of the Jaw	Atypical Femur Fracture	Total
Characteristics	**N = 9**	**N = 3**	**N = 12**
Age, years	69 (±9)	74 (±4)	70 (±8)
Sex			
Female	9 (100,0%)	3 (100,0%)	12 (100,0%)
Male	0 (0%)	0 (0%)	0 (0%)
OP duration, years	11 (±5)	15 (±3)	12 (±5)
Previous fragility fractures			
Yes	6 (66,7%)	0 (0%)	6 (50,0%)
No	3 (33,3%)	3 (100,0%)	6 (50,0%)
Time between the OP diagnosis and the adverse event, years	7,94 (±4,92)	13,64 (±3,37)	9,37 (±5,13)
Medication involved			
Denosumab 60mg/every 6 months	3 (33,3%)	0 (0%)	3 (25,0%)
Zoledronate 5mg/annually	3 (33,3%)	0 (0%)	3 (25,0%)
Alendronate 70mg/weekly	2 (22,2%)	3 (100,0%)	5 (41,7%)
Pamidronate 90mg/every 3 months	1 (11,1%)	0 (0%)	1 (8,3%)
Duration of treatment prior to the adverse event, years	3,9 (±2,7)	11,3 (±6,5)	5,8 (±4,9)
Previous OP treatment			
Yes	4 (44,4%)	1 (33,3%)	5 (41,7%)
No	5 (55,6%)	2 (66,7%)	7 (58,3%)
With which drug was the previous OP treatment?			
Alendronate 70mg/weekly	3 (75,0%)	0 (0%)	3 (60,0%)
Ibandronate 150mg/month	1 (25,0%)	1(100,0%)	2 (40,0%)
Subsequent OP therapy			
Teriparatide 20mcg/day	2 (22,2%)	1 (33,3%)	3 (25,0%)
Denosumab 60mg/every 6 months	0 (0%)	1 (33,3%)	1 (8,3%)
No futher treatment	7 (77,8%)	1 (33,3%)	8 (66,7%)
Other Rheumatic Disease			
Rheumatoid Arthritis	5 (55,6%)	0 (0%)	5 (41,7%)
Spondyloarthritis	1 (11,1%)	0 (0%)	1 (8,3%)
None	3 (33,3%)	3 (100,0%)	6 (50,0%)
Bone Density (BMD) baseline			
Femur	0,796 (±0,076)	**	0,816 (±0,92)
Vertebral	0,948 (±0,162)	**	0,948 (±0,162)
Radius	0,463 *	**	0,463 *
t-score baseline			
Femur	-1,5 (±0,8)	-2,0 (±0)	-1,6 (±0,7)
Vertebral	- 1,9 (±1,3)	- 2,2 (±0,9)	-2,0 (±1,2)
Radius	-3,5 *	**	-3,5 *
BMD after the adverse event			
Femur	0,833 (±0,124)	0,841 (±0,065)	0,835 (±0,108)
Vertebral	1,031 (±0,186)	0,941 (±0,027)	1,001 (±0,152)
Radius	0,422 *	**	0,422 *
t-score after adverse event			
Femur	-1,4 (±1,0)	-1,3 (±0,6)	- 1,3 (±0,9)
Vertebral	- 1,4 (±1,5)	-1,8 *	- 1,5 (±1,3)
Radius	-4,2 *	**	-4,2 *

features.

Material and Methods: Retrospective cohort study including consecutive SSc patients, followed in a single Rheumatology Department. Patients were interviewed regarding their lifetime occupational history, with records made for any job held for more than six months before SSc diagnosis. Jobs were classified based on the International Labour Office classification. Demographic and clinical data were collected. Multimorbidity was defined as the co-occurrence of at least two chronic conditions in the same patient. Continuous data were presented as mean (standard deviation) or median (interquartile range) for variables with skewed distribution, and categorical variables as absolute and relative frequencies. Associations between the different categorical or dichotomic variables were tested using Chi-Square Test or Fischer's Exact Test, as appropriate. Associations of continuous variables with categorical or dichotomic variables were tested using Student's t-test. A p value ≤ 0.05 was considered statistically significant.

Results: We included 42 SSc patients (81.0% females, mean age 60.9 ± 10.2 years). Most patients (54.8%) had more than one different occupation in their lifetime. Table 1 provides a complete description of clinical characteristics and occupational history.

Sixteen patients (38.1%) had a history of manufacturing employment. Employment in ceramics factories was particularly common (16.7%), followed by textile factories (9.5%). History of work in metallurgy was present in 11.9% of patients (n=5) and work in construction was reported by 7.1% Employment in construction (corrected OR = 43.9, 95% CI [1.98, 970]; p =0.005), metallurgy (OR = 9.60, 95% CI [1.27, 72.5]; p = 0.040) and electrical work (corrected OR = 26.5, 95% CI [1.13, 6230]; p = 0.033) was more frequent in male patients. Previous work in manufacturing was associated with multimorbidity (OR = 4.52, 95% CI [1.19, 17.2]; p = 0.023). Work in construction was significantly associated with the diffuse SSc phenotype (corrected OR 33.9, 95% CI [1.39, 826]; p = 0.040) as opposed to the limited phenotype. No association was found regarding occupational history and clinical manifestations (Table 1), baseline and current modified Rodnan skin score, capillaroscopic pattern, immunology features, smoking and alcohol consumption, and current immunosuppressive treatment.

Conclusion: Previous manufacturing employment was common in our cohort, particularly in ceramic factories. Manufacturing work history was associated with multimorbidity. Work in construction was associated with diffuse SSc phenotype. Further nationwide studies are needed to explore the role of occupational factors in SSc.

TO 180 – TABLE 1. SSc patients characteristics (n = 42)

Table 1- SSc patients characteristics (n = 42)	
Sex (Female) – n (%)	34 (81.0)
Age - mean ± SD	60.9 ± 10.2
Age at symptoms onset – mean ± SD	50.1 ± 11.5
Disease duration – median (IQR)	7.0 (8.0)
Disease classification – n (%)	
Limited SSc	30 (71.4)
Diffuse SSc	8 (19.0)
VEDOSS	4 (9.5)
Capillaroscopic pattern – n (%)	
Non-scleroderma pattern	2 (4.9)
Early scleroderma pattern	22 (53.7)
Active scleroderma pattern	13 (31.7)
Late scleroderma pattern	4 (9.8)
Immunology – n (%)	
Anti-centromere	30 (71.4)
Anti-Scl70	7 (16.7)
Anti-RNA polymerase	4 (9.5)
Anti -PM-Scl75	1 (2.4)
Current mRss – mean ± SD	7.5 ± 7.6
Baseline mRss - median (IQR)	0.0 (6.0)
Clinical manifestations (ever) – n (%)	
Raynaud's phenomenon	42 (100)
Skin thickening	33 (78.6)
Telangectasias	25 (59.5)
Digital ulcers	11 (26.2)
Calcinosis	4 (9.5)
Contractures	2 (4.8)
Tendinous friction rubs	2 (4.8)
Arthralgia	9 (21.4)
Arthritis	1 (2.4)
Myositis	0 (0)
Esophageal involvement	8 (19.0)
Gastric involvement	13 (31.0)
Intestinal involvement	1 (2.4)
Cardiac involvement	2 (4.8)
Pulmonary involvement	12 (28.6)
Renal involvement	1 (2.4)
Current immunosuppressive treatment – n (%)	13 (31.0)
Multimorbidity – n (%)	17 (40.5)
Comorbidities – n (%)	
Arterial hypertension	18 (42.9)
Dyslipidemia	18 (42.9)
Diabetes mellitus	2 (4.8)
Cardiovascular disease	6 (14.3)
Chronic pulmonary diseases	2 (4.8)
Neurological diseases	3 (7.1)
Smoking history – n (%)	7 (16.7)
Alcohol consumption history – n (%)	3 (7.1)
Employment history – n (%)	
Manufacturing work	16 (38.1)
Ceramic industry	7 (43.8)
Textile industry	4 (25.0)
Metal and machinery (Metallurgy) work	5 (11.9)
Construction work	3 (7.1)
Mechanics	1 (2.4)
Electrical work	2 (4.8)
Cleaning work	3 (7.1)
Agricultural, Forestry and Fishery	6 (14.3)
Professionals (Health, Teaching, Administration...)	16 (38.1)
Services and sales workers	12 (28.6)

N- number; SD - Standard Deviation; IQR – Interquartile range; SSc – Systemic Sclerosis; VEDOSS – Very Early Diagnosis of Systemic Sclerosis; mRss – Modified Rodnan Skin Score

183 - VALIDATION OF THE PORTUGUESE VERSION QUALEFFO-41 IN PORTUGUESE PATIENTS WITH VERTEBRAL FRACTURES-PRELIMINARY DATA

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Introduction: Fragility fractures, such as vertebral fractures, significantly impact morbidity, mortality, and quality of life (QoL). The QUALEFFO-41, developed by the International Osteoporosis Foundation (IOF) is a widely recognized tool for assessing quality of life in patients with osteoporotic vertebral fractures. This study aims to validate the Portuguese version of QUALEFFO-41 in Portuguese patients with vertebral fragility fractures.

Methods and Materials: A multicenter study involving Portuguese osteoporotic patients from six nationwide Rheumatology and one Endocrinology centers was conducted

Patients aged 55-85 years with vertebral fractures who could understand and give informed consent were included. Those with other conditions impacting QoL or recent fractures were excluded. The questionnaires were applied twice in the same order, with a 2-week interval between answers.

The Portuguese version of the QUALEFFO-41 was tested for validity and reliability. Cronbach´s α and the internal validity index were calculated to verify the internal reliability and validity of the content. Cronbach´s α indicator, which can range from 0 to 1, with values above 0.70 considered ideal. Repeatability was assessed by the intraclass correlation coefficient (ICC) for all domain scores. Convergent validity was considered adequate when the correlation coefficient between each question score and the total domain score was > 0.40. Adequate discriminant validity was assumed when the correlation coefficient between each question score and the total domain score was higher than the correlation with the total scores of other domains. Lastly, the correlation between QUALEFFO-41 domains and those of SF-36 was investigated using the Spearman correlation coefficient.

TO 183 – TABLE 1. Results of the multitrait analysis of QUALEFFO-41

	Internal consistency alpha-Chronbach	repeability (ICC)
Pain (5)	0.89	0.98
Physical function (17)	0.88	0.76
Social function (7)	0.60	0.77
Genereal health perceptio	0.77	0.87
Mental function (9)	0.77	0.88
QUALEFFO-41 total	0.90	0.88

TO 183 – TABLE 2. Spearman correlation for each QUALEFFO-41 and SF-36 domain

QUALEFFO-41	SF-36	Correlation coeficient
Pain	Body Pain	-0.42
Physical function	Functional capacity	- 0.69
Social function	Social aspects	- 0.45
Genereal health perception	General health status	-0.85
Mental function	Mental health + vitality	-0.62

The validation study was initiated after the consent of the IOF, and the methodology followed the same criteria as the original validation study.

Results: 40 patients were enrolled in the study, 37 (92.5%) were female, with a mean age of 69.95 ±8.41 years. Eighteen patients (45%) had vertebral deformities and 15 (37.5%) had non-recent previous fractures. Katz's score was between 0-2 in most patients (25, 62.5%). The most common anti-osteoporotic treatment administered was bisphosphonates (19 patients, 47.5%), followed by denosumab (10 patients, 25%). All patients were under vitamin D and calcium supplementation, and most under anti-osteoporotic treatment (34 patients, 85%).

QUALEFFO-41 demonstrated adequate internal consistency for all domains (Cronbach´s α coefficient from 0.60 to 0.90) and repeatability (ICC for each domain: 0.76-0.98). Of the total questions, 85.40 % demonstrated satisfactory convergent and discriminant validity. (Table 1)

There was a good correlation among the QUALEFFO-41 domains and their corresponding SF-36 domains, except for social function and pain, which demonstrated poor to moderate correlation (r = -0.45 and -0.42, respectively). (Table 2)

Conclusion: With these preliminary data, our study demonstrated that the QUALEFFO-41 exhibits good reliability, repeatability, and validity in this sample of the Portuguese population. The next steps include comparing patients with vertebral fractures to age- and sex-matched controls to assess the questionnaire's ability to discriminate QoL in vertebral fracture patients and further corroborate its utility in this population.

184 - PERSISTENCE OF VEDOSS OVER TIME: INSIGHTS FROM A SINGLE-CENTRE

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TO 184 – TABLE 1. Demographic, clinical and serological characterisation of VEDOSS patients

Variables	Number of patients with available data	Number of patients (N = 72)
Female, n (%)	72	67 (93.1%)
Current age (years), mean ± sd (range)	72	59.2 ± 14.9 (26-89)
Caucasians, n (%)	54	49 (90.7%)
BMI (m2/Kg), mean ± sd (range)	33	26.0 ± 4.3 (18.3-37.7)
First symptom: Raynaud's phenomenon, n (%) Puffy hands/fingers, n (%)	72	 71 (98.6%) 1 (1.4%)
First symptom age (years), mean ± sd (range)	71	45.7 ± 15.5 (17-81)
VEDOSS age of diagnosis (years), mean ± sd (range)	72	52.9 ± 15.2 (20-83)
Time from first symptom to VEDOSS diagnosis (years), mean ± sd (range)	71	6.8 ± 7.9 (0-36)
Raynaud's phenomenon, n (%)	72	71 (98.6%)
Raynaud's Phenomenon age of onset (years), mean ± sd (range)	65	45.6 ± 15.6 (18-81)
Puffy hands/fingers, n (%)	72	16 (22.2%)
Puffy hands/fingers age of onset (years), mean ± sd (range)	16	48.4 ± 17.8 (23-71)
Onset of telangiectasias during follow-up, n (%)	72	6 (8.3%)
Onset of calcinosis during follow-up, n (%)	72	2 (2.8%)
Onset of digital ulcers during follow-up, n (%)	72	9 (12.5%)
Capillaroscopy changes at VEDOSS diagnosis, n (%)	68	39 (57.4%)
Capillaroscopic pattern, n (%) No changes Non-specific changes Early pattern Active pattern Late pattern	67	 28 (41.8%) 6 (9.0%) 27 (40.2%) 6 (9.0%) 0
Age of identification of capillaroscopic changes (years), mean ± sd (range)	40	52.2 ± 14.4 (23-83)
ANA positivity, n (%)	72	56 (77.8%)
ANA positivity age of identification (years), mean ± sd (range)	46	54.7 ± 15.1 (22-83)
SSc-related antibodies positivity, n/N (%)	72	63 (87.5%)
SSc-related antibodies positivity age of identification (years), mean ± sd (range)	53	55.2 ± 14.7 (22-83)
SSc-related antibodies, n (%) Anti-centromere Antitopoisomerase I Anti-Th/To Anti-PM/Scl Anti-NOR90 Anti-Ku Anti-fibrillarin Anti-Ro52 Anti-Ro60 Anti-RNA polymerase III None	72	 40 (55.5%) 5 (6.9%) 5 (6.9%) 4 (5.6%) 4 (5.6%) 3 (4.2%) 1 (1.4%) 1 (1.4%) 1 (1.4%) 1 (1.4%) 7 (9.7%)
Score in the 2013 EULAR/ACR SSc Criteria at VEDOSS diagnosis (points), mean ± sd (range)	72	6.4 ± 1.7 (3-8)
Current score in the 2013 EULAR/ACR SSc Criteria (points), mean ± sd (range)	72	6.5 ± 1.7 (3-8)
Follow-up period (years), mean ± sd (range)	69	7.0 ± 5.4 (1-34)
Loss of follow up, n (%)	67	5 (7.5%)
Loss of follow-up motive, n (%) Missed appointment	5	 5 (100%)

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Introduction: The very early diagnosis of systemic sclerosis (VEDOSS) applies to patients who do not fulfill the 2013 ACR/EULAR criteria for systemic sclerosis (SSc) but exhibit Raynaud's phenomenon, puffy fingers, abnormalities in nailfold capillaroscopy and/or possess SSc-specific antibodies. The clinical profile of VEDOSS patients varies significantly depending on the studied population.
Aims: To describe the demographic, clinical and serological profile of the VEDOSS patient population of a single centre.
Methods: The clinical records of all patients with VEDOSS followed in our department were reviewed. SSc patients or overlap syndromes with VEDOSS were excluded. We collected demographic, clinical and serological variables. T-student or Mann-Whitney tests were used, as appropriate, for continuous variables and chi-square or Fisher tests for categorical variables.
Results: A total of 72 patients (93.1% women, 90.7% Caucasian) were identified (Table 1). The mean age at first symptom and VEDOSS diagnosis was 45.7±15.5 and 52.9±15.2 years, respectively. Diagnosis of VEDOSS was delayed, with a mean time from the first symptom to diagnosis of 6.8±7.9 years.
Raynaud's phenomenon was the most frequent first symptom of VEDOSS (n=71, 98.6%), at a mean onset age of 45.6±5.6 years. During follow-up, puffy hands/fingers were identified in 16 patients (22.2%), telangiectasias in six (8.3%) and calcinosis in two (2.8%)
At VEDOSS diagnosis, 39 patients (57.4%) had changes in capillaroscopy, 27 (40.2%) with early pattern, 6 (9.0%) with non-specific changes and 6 (9.0%) with active pattern. Notably, 28 (41.8%) had normal capillaroscopy.
The mean score in the 2013 EULAR/ACR SSc Criteria at diagnosis of VEDOSS was 6.4±1.7. After a mean follow-up period of 7.0±5.4 years, the mean 2013 EULAR/ACR SSc Criteria score remained stable (6.5±1.7 points).
Regarding serologic findings, 56 patients (77.8%) had positive antinuclear antibodies (ANA), while 65 (90.3%) had positive SSc-related antibodies (the most frequent were: anti-centromere n=40 (55.5%); anti-topoisomerase-I n=5 (6.9%); anti-Th/To n=5 (6.9%)).
During the follow-up period, 5 patients (7.5%) were lost to follow-up and none had died.
Conclusion: VEDOSS presented mostly in middle-aged Caucasian women and had a delayed diagnosis from symptom onset. Raynaud's phenomenon was the first symptom in the majority of patients and one in five developed puffy hands/fingers. Presence of autoantibodies and capillaroscopic changes were common. Notably, there was not significant clinical evolution in this cohort over follow-up, possibly suggesting a low progression rate to SSc, at least in a selected subpopulation of patients with VEDOSS.

191 - WHAT IS THE ROLE OF BIOLOGIC'S IN PREVENTING PSORIATIC ARTHRITIS PROGRESSION IN PATIENTS WITH PSORIASIS? - A RETROSPECTIVE STUDY

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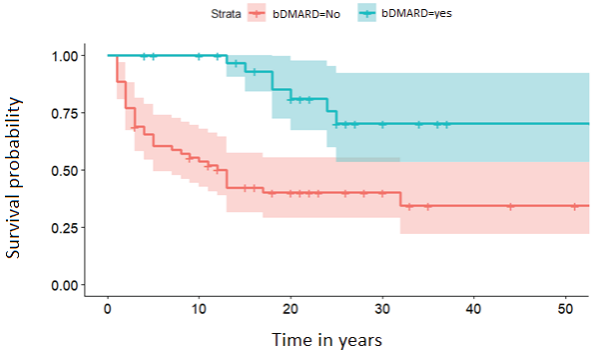
Introduction: The effectiveness of biologic treatments in slowing the progression of psoriatic arthritis (PsA) is well recognized. Nevertheless, and despite recent evidence suggesting the potential benefit of these treatments in the prevention of PsA in patients with psoriasis (PsO), this potential is less well established, and contradictory data remain, namely when comparing different biologic Disease Modifying Anti-Rheumatic Drugs (bDMARD) classes.
Our study aimed to compare the incidence of PsA in PsO patients treated with bDMARD and in patients under other systemic or topic treatments.
Methods: Retrospective study of patients with PsO followed in the Dermatology department of our Centre from 2012-2023. Patients were divided into two groups: PsO Vs PsA patients (according to CASPAR criteria). The risk of PsA development was compared between patients treated with bDMARDs and patients under other treatments (conventional Disease Modifying Anti-Rheumatic Drug (csDMARD) and topic treatment) and between different bDMARD classes. For those progressing to PsA, a survival analysis was performed, comparing the two previously mentioned treatment groups. A univariate followed by multivariate analysis was conducted to identify predictors of psoriatic arthritis in patients with psoriasis.



TO 191 – Figure 1. Survival curves for psoriatic arthritis for biologic Vs no-biologic treatment

Results: After excluding patients with relevant missing data, a total of 122 patients were included (69 with PsO and 53 with PsA). Mean age was 52.40 ± 11.25 years, 53.3% were female, with no differences encountered between groups. 87 patients (71.31%) were under topical treatments, 20 (16.39%) received csDMARDs, 52 (42.62%) received bDMARD, and 62 (50.81%) received systemic treatment (either bDMARD or csDMARD).
PsA patients were less frequently under bDMARD before arthritis onset compared to patients with PsO alone (p<0.001), had higher HAQ and PASI scores (p<0.001 and p=0.02, respectively), and higher prevalence of Spondylarthritis (SpA) family history (p<0.001). On survival analysis, the global mean time to arthritis progression was 32 years, with significant differences in patients with or without bDMARD, indicating that the average time of PsA incidence was approximately 24.1±3.3 years in patients without bDMARD, whereas, in patients with bDMARD, this average time is 44.5±3.4 years (p<0.001) (Figure 1).
On multivariate analysis, SpA family history was independently associated with a higher risk of PsA development [OR 2.80 CI(1.26; 6.23), p=0.01], while bDMARD treatment was a protective factor, with those not under these treatments experiencing a higher risk of progression [OR 10.31 CI (3.89; 27.33), p<0.001].
Conclusion: Our study indicates that bDMARDs exhibit a decelerating effect on PsA development in patients with PsO. Prospective observational cohorts with disease activity measures and randomized trials are required to confirm these findings.

193 - TAXA DE RETENÇÃO DOS BDMARD NA ARTRITE PSORIÁTICA – EXPERIÊNCIA DE UM HOSPITAL TERCIÁRIO

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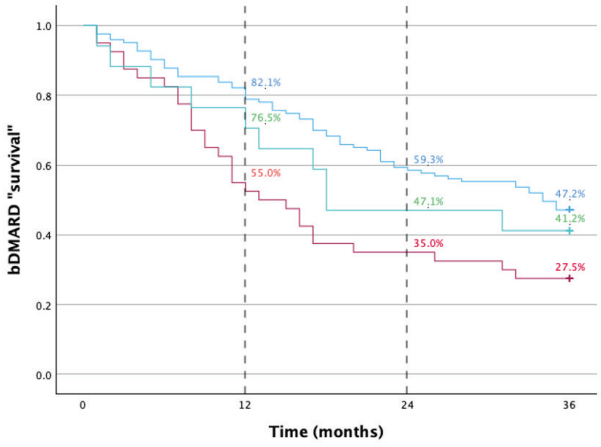
Introdução: A artrite psoriática (APso) é uma artropatia inflamatória muito comum associada a um elevado potencial para destruição articular e incapacidade funcional, sobretudo nos casos em que o tratamento é inadequado/iniciado de forma tardia. Caso o tratamento de primeira linha com cDMARDs não seja bem-sucedido, os bDMARDs são uma boa opção para controlo da doença.
A sua eficácia e tolerância pode ser avaliada através da taxa de retenção, que varia conforme a apresentação da doença, histórico terapêutico e características do doente.
Objetivos: Este estudo pretende fazer uma descrição sumária dos principais grupos de bDMARD utilizados como 1ª a 4ª linha terapêutica, bem como as causas que levaram à sua suspensão, numa população de doentes com APso de um hospital terciário. Secundariamente, determinou-se a taxa de retenção, de acordo com mecanismo de ação, aos 12, 24 e 36 meses, para comparação entre classes.
Metodologia: Foram incluídos todos os doentes com diagnóstico de APso (critérios de CASPAR) que se encontrassem medicados com bDMARD ± cDMARD, seguidos entre Junho/1990 e Dezembro/2023, na consulta de Reumatologia do Hospital de Braga.
Destes, foram excluídos todos os doentes com diagnóstico concomitante de outra patologia reumatológica, bem como dados ausentes do processo clínico, considerados essenciais para a descrição do doente – incluídos 123 doentes.
A comparação da taxa de retenção prendeu-se com uma análise do tipo "survival", através de testes Log-Rank (significância se p-value <0.05), sendo estas representadas na forma de curvas de Kaplan-Meyer.
Resultados: Em média, os pacientes eram seguidos há cerca de 93.6 ± 61.0 meses (tempo desde 1ª consulta de Reumatologia). As principais causas para suspensão de tratamento encontram-se listadas na tabela 1.
A taxa de retenção global para os bDMARD usados entre 1ª e 3ª linha encontra-se representada na figura 1 (4ª linha de bDMARD não avaliada uma vez que n pouco representativo). Na comparação entre diferentes bDMARD usados como 1ª linha, foram considerados apenas fármacos com n ≥ 10, tendo-se verificado uma maior taxa de retenção com Etanercept em relação ao Adalimumab, aos 12 (100.0% vs 79.3%; p=0.024), 24 (95.5% vs 51.2%; p<0.01) e 36 (95.5% vs 37.8%; p<0.01) meses.
A comparação entre bDMARD independentemente da linha terapêutica demonstrou uma taxa de retenção su-

TO 193 – TABLE 1.

	1º bDMARD	2º bDMARD	3º bDMARD	4º bDMARD
TNFi	119	30	5	-
IL12/23i	2	1	3	-
IL17i	1	8	5	2
Il23i	1	1	1	2
Jaki	-	-	2	-
CTLA4-Ig	-	-	1	-
Total	123	40	17	4
Motivos que levaram a suspensão do Tratamento				
Falência Primária	5	2	-	3
Falência Secundária	20	11	4	-
Remissão	2	-	-	-
Toxicidade/Efeito Adverso -Diagnóstico de Neoplasia -Reação Paradoxal -Infeção Grave/Recorrente -Toxic. Hepática/Hematológica -Reação Alérgica	17 3 4 4 3 3	5 - 2 2 1 -	-	-
Cirurgia	1	1	-	-
Desistência do doente	2	1	-	-
Gravidez	2	1	-	-
Total	49	21	4	3



TO 193 – Figura 1. Taxa de retenção de bDMARD aos 12,24 e 36 meses (azul - 1ª linha; vermelho - 2ª linha; verde - 3ª linha)

perior nos TNFi relativamente às restantes subclasses (restantes subclasses não comparadas entre si por n pouco representativo).

Conclusão: Em primeiro lugar, importa referir que este estudo apresenta algumas limitações, como algumas características dos doentes não serem incluídas na análise, como progressão da doença no momento de início do bDMARD, fenótipo (axial vs periférico), presença de fatores de mau prognóstico ou o uso de cDMARD concomitante, que pode influenciar significativamente a taxa de retenção, sobretudo nos TNFi.

Como esperado, a taxa de retenção é menor nos tratamentos de 2ª/3ª linha, o que pode implicar uma doença mais severa e/ou de curso mais refratário, nos doentes que apresentaram falência a 1º bDMARD.

Comparar estes resultados com a literatura implica alguma caução, uma vez que a maior parte dos estudos é relativamente heterogéneo, sendo difícil uma comparação 1:1. Apesar disso, este estudo avalia a taxa de retenção a 3 anos para várias linhas terapêuticas, fazendo também um levantamento dos principais motivos de falência terapêutica, cuja análise permite inferir que estes fármacos são relativamente seguros, eficazes, bem tolerados e constituem o futuro da abordagem terapêutica destes doentes.

195 - DEPRESSION DISORDER IN SPONDYLOARTHRITIS PATIENTS ON BIOLOGIC THERAPY REGISTERED IN REUMA.PT: PREVALENCE, ROLE OF DISEASE-RELATED FACTORS AND INFLUENCE OF BIOLOGIC THERAPY

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Introduction: Depression disorder prevalence is higher in chronic conditions like Spondyloarthritis (SpA), possibly linked to chronic inflammation, pain and functional impairment. Our aim was to determine Depression disorder prevalence in SpA patients at first biologic prescription (bDMARD) and to assess its effect on Depression disorder.
Methods: We conducted a multicenter, retrospective, observational study including adult patients registered in Reuma.pt with SpA treated with their first bDMARD. Patients that completed the Hospital Anxiety and Depression Scale (HADS) at baseline (T0), after 3 (T1) and/or 6 months (T2) of treatment were included. Socio-demographic, disease and treatment-related data were collected. Depression disorder was considered when subscale depression (HADS-D) ≥ 11. Pearson and Spearman correlations, ANOVA and T-tests were used.
Results: 141 patients were analyzed, 55.3% were female. Mean age at diagnosis was 37.4 ± 10.5 years. Adalimumab was the most frequently bDMARD prescribed (59.6%) of initial prescriptions, followed by ertanecept (23.4%). Depression symptoms at baseline were present in 13.5% (N=19) of patients.
Patients with depression had significantly higher BASDAI and ASDAS-CRP, but not BASMI, at T0 than those who didn't have DD (p of 0.045, 0.007 and 0.570, respectively).
Mean scores of HADS-D significantly differed between the three time points (F(2.000, 155.387) =12.440, p<0.001). Post hoc analysis with Bonferroni adjustment revealed that HADS-D significantly decreased from T0 to T1 (1.535 (95% CI, 0.621 to 2.449), p<0.001), from T0 to T2 (1.628 (95% CI, 0.630 to 2.626), p<0.001), but not from T1 to T2 (0.093 (95% CI, -0.664 to 0.851), p=1).
No significant differences were found in HADS-D at the three time points between patients with axial disease and those with peripheral disease (F(2.000, 52.000) =2.917, p=0.063) and between patients treated with anti-TNF and those with an anti-IL-17 (F(1.824, 153.221) =0.251, p=0.758).
Conclusion: Depression symptons improve with bDMARD therapy, probably due to control of disease activity and function improvement. Inflammation hypothesis in depression should be considered and further investigated.

197 - CARACTERIZAÇÃO DOS DOENTES COM DIAGNÓSTICO DE ARTERITE DE CÉLULAS GIGANTES: A EXPERIÊNCIA DA IMPLEMENTAÇÃO DE UMA CONSULTA DE FAST-TRACK

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A Arterite de Células Gigantes (ACG) é a vasculite de grandes vasos mais comum nos idosos. O seu tratamento precoce reduz o risco de complicações isquémicas, nomeadamente a perda visual permanente. A consulta de fast-track de ACG tem como objetivo a avaliação rápida dos doentes com suspeita de ACG, com possível realização de ecografia das artérias temporais e axilares, para que seja iniciada terapêutica adequada atempadamente. Além disso, evita a exposição desnecessária a corticoterapia (CCT) nos doentes sem confirmação do diagnóstico.
O objetivo deste trabalho é avaliar as características clínicas, tratamentos e outcomes dos doentes com diagnóstico de ACG, avaliados em consulta de fast-track

desde 2021.
Foram incluídos um total de 22 doentes com diagnóstico confirmado de ACG. Destes, 54,5% eram do sexo feminino e a média de idades ao diagnóstico de 79,2±5,79 anos. 81,8% dos doentes apresentava pelo menos 1 fator de risco cardiovascular (CV), sendo que apenas 1 doente tinha antecedentes de evento CV prévio. Apenas 1 dos 22 doentes tinha diagnóstico prévio de polimialgia reumática (PMR). A referenciação à consulta de fast-track foi feita pela Neurologia em 90,9% dos doentes, sendo que os restantes doentes foram referenciados pela Medicina Interna. O sintoma mais comum na apresentação inicial foi a cefaleia temporal (95,5%). 72,3% dos doentes apresentavam sintomas visuais, 68,2% referiam sintomas constitucionais, 22,7% tinham quadro concomitante de PMR e 9,1% apresentaram evento cerebrovascular isquémico. Analiticamente, a média da velocidade de sedimentação era de 79,6±27,16 mm/h, a média da proteína C reativa de 77,5±62,1 mg/L, 31,8% apresentavam hemoglobina <10g/dl e 31,8% apresentavam contagem de plaquetas >400000/ul. Todos os doentes realizaram ecografia das artérias temporais e axilares, sendo que o tempo médio desde o início dos sintomas até a realização da ecografia foi de 53,0±77,8 dias. Nos 19 doentes com resultado positivo, a média de segmentos com sinal do halo nas artérias temporais foi de 3,4±1,87. Em 2 doentes o resultado da ecografia foi equívoco e em 1 doente negativo. Quanto ao tratamento efetuado, 68,2% dos doentes realizaram pulsos de metilprednisolona endovenosa e todos os doentes realizaram prednisolona (PDN) oral (média de 50,5±13,64 mg/d). Em 6 doentes foi iniciado um DMARD no início do tratamento (metotrexato em 2 doentes e tocilizumab em 4 doentes). Dos 22 doentes, 4 experienciaram recidiva da doença, 6,5±2,29 meses após terem atingido a remissão, com uma dose de PDN de 8,75±2,80mg/d, sendo que nenhum destes tinha iniciado qualquer DMARD previamente. Em 2 destes 4 doentes foi iniciado tratamento com MTX no momento da recidiva. Até à data, a média de meses de follow-up dos 22 doentes foi de 22,27±14,81 meses, tendo-se observado 3 mortes, nenhuma delas relacionada com a ACG.
A ACG é uma doença que se pode apresentar com uma sintomatologia muito variada e por vezes inespecífica, podendo atrasar o diagnóstico. Na nossa população, a maioria dos doentes foi referenciada pela neurologia e apresentava sintomas isquémicos ao diagnóstico. Estes dados poderão ser explicados em parte pela iliteracia em saúde da população, que leva à desvalorização das queixas menos especificas, como sintomas constitucionais e cefaleia e não procura cuidados de saúde atempadamente e, por outro lado, pela falta de alerta dos médicos de família para esta patologia. A gestão destes doentes é um desafio, principalmente devido às comorbilidades apresentadas, optando-se cada vez mais pela utilização de DMARDs para uma redução mais rápida da CCT.

198 - RELATION BETWEEN FATIGUE AND OTHER PARENT REPORTED OUTCOMES IN A COHORT OF CHILDREN WITH JIA

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Introduction: Although fatigue is considered to be common in children and young people with juvenile idiopathic arthritis (JIA), the relevance of this symptom within the burden of the disease was poorly studied.
Objective: To assess correlation of fatigue with physician centered measures and other patient reported outcomes (PROs) in a cohort of JIA patients.
Methods: We enrolled in the study all JIA patient attending the outpatient clinic at the Study Unit in April 2024. Patients were asked to complete the Juvenile Arthritis Multidimensional Assessment Report (JAMAR) and the PROMIS® Item Bank v1.0 – Fatigue – Short Form 13a (FACIT-Fatigue), measuring the level of fatigue in 13 items with a 5-points Likert scales yielding a score of 0 to 52, with higher scores indicating more sever fatigue. Physician centered measures included the rheumatological examination form and the physician global assessment of disease activity (PhGA). We calculated Spearman rank correlation between fatigue score and quantitative measures included in the JAMAR, active joints count, and PhGA. Finally, we compared the levels of fatigue in patients considering themselves to be satisfied or not satisfied with current disease outcome.
Results: The questionnaires were proposed for completion before clinical examination to 51 JIA patients; 49 (87.2% females) completed both questionnaires and were included in the study. Patients had a median disease duration of 9.6 years (IQR 5.3-13.5) and were predominantly affected with oligoarticular JIA (72.3%). All JIA categories were represented. Median PhGA was 0 (0-2). Median fatigue score was 47 (38-51.5). Fatigue correlations were higher with JAMAR HRQoL tool (r = -0.67) and with JAMAR functional ability tool (r = -0.60). Correlation coefficients were between -0.4 and -0.6 with well-being VAS, patient disease activity VAS, pain VAS, morning stiffness duration, and cJADAS10.

Correlations were poor (r < -0.4) with PhGA and active joint count. Correlations with HRQoL items in the JAMAR that were suggested to explore the domain of fatigue were the highest (r = -0.70 between fatigue score and JAMAR HRQoL items 3 and 9). Fatigue score was 37 (35-44) in 21 patients who were not satisfied with current disease outcome and was 51 (47-52) in 26 patients who considered their disease status as satisfactory (p < 0.001).

Conclusion: Fatigue seems to have a relevant weigh in the disease perception of children with JIA in a cohort of children with a generally well controlled disease. It is strongly correlated with HRQoL score and functional ability score. Patients not satisfied with disease outcome had significantly higher level of fatigue.

REFERENCES

1. Nijhof LN, et al. Arthritis Care Res (Hoboken), 2016; 2. Filocamo G, et al. J Rheumatol, 2011; 3. Hewlett S, et al, Arthritis Care Res (Hoboken), 2011; 4. Consolaro A, et al, Pediatric rheumatology, 2012

201 - RELATIONSHIP BETWEEN SALIVARY GLAND ULTRASOUND FINDINGS AND IMMUNOLOGY IN SJÖGREN'S SYNDROME

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Introduction: Sjögren's Syndrome (SS) is a chronic inflammatory disease that typically involves the salivary glands. Most patients have positive immunology, namely antinuclear antibodies (ANAs), anti-SSA, and anti-SSB. Major salivary gland ultrasound (SGUS) evaluation has gained more attention in the last years, since it has shown associations with immunological profile and clinical outcomes in SS. The Outcome Measures in Rheumatology Clinical Trials (OMERACT) score includes a semi-quantitative evaluation of the parenchymal heterogeneity of the four major salivar glands, with scoring ranging from 0 (normal) to 3 (pathological parenchyma without normal areas). A score ≥2 is interpreted as SGUS compatible with SS. Despite its high sensitivity and specificity, there are still few studies using the OMERACT score to search association between SGUS and immunological findings. This study aims to characterize the SGUS abnormalities found in patients with SS, and evaluate their relationship with autoantibodies.

Methods: Our study is a retrospective observational study. Sociodemographic data and immunological profile were collected from all patients who underwent SGUS at our center between January 2023 and April 2024. SGUS included the evaluation of both parotids and submandibulars glands and was scored according the OMERACT Grey Scale Ultrasound Scoring System for SS. A GE Logiq S8 ultrasound machine with a ML6–15linear array transducer was used for all examinations. Only patients with primary or secondary SS were included in this study. Descriptive analysis was performed with mean and standard deviation for continuous variables, and absolute and percentage frequency for categorical variables. Independent t-test and chi-square test were performed. Differences were considered statistically significant at p<0.05.

Results: Forty-six patients were included in our study, 43 (93,5%) of whom were female. The mean age was 59 (±13) years. Most patients were ANA positive (84,8%; n=39). Anti-SSA antibodies were detected in 73,9% (n=34) and anti-SSB in 34,8% (n=16) of the SS patients. Twenty-six (56.5%) had SGUS suggestive of SS, while 14 (30.4%) showed no pathological SGUS findings. With regard to SGUS findings, the majority of patients presented bilateral involvement of the salivary glands (65.2%; n=30). This involvement was statistically significant in patients with anti-SSA (p=0.022). Eighteen (51.4%) patients had involvement of all glands, with no preference between parotid or submandibular involvement. When comparing the group of patients with abnormal versus normal SGUS, we found that most patients with abnormal SGUS were significantly associated with ANAs, Anti-SSA and Anti- SSB positivity (p=0.013, p=0,006, p=0,047, respectively). The OMERACT score was higher in patients with positive ANAs (p=0.003), anti-SSA (p=0.021), and anti-SSB (p=0.010). Furthermore, the SGUS findings associated with greater severity, such as gland atrophy (28,3%), irregular borders (28,3%), or hyperechoic bands (30,4%), were only observed in patients with positive ANAs.

Conclusion: SGUS is a non-invasive, easy to perform and effective technique for the evaluation of salivary glands in SS patients. Similarly to previous studies, our results suggest that the autoantibodies detected in the blood samples have a strong association with the salivary glandular pathology detected by SGUS, potentially as part of local inflammation and structural damage of the salivary glands.

202 - TRANSFORM- CORTICOSTEROID UTILIZATION PATTERNS AMONG SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS – CROSS-SECTIONAL STUDY IN THE PORTUGUESE COMMUNITY PHARMACIES

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Background: The management of patients with systemic lupus erythematosus (SLE) has changed significantly since the introduction of biologic therapies, which can contribute to improve patients' outcomes while allowing a reduction of the cumulative use of oral corticosteroid (OCS) and the long-term risk for comorbidities [1]. The EULAR 2023 management recommendations propose as treatment target to attain sustained remission while reducing OCS to a maximum maintenance dose of 5mg/day of prednisone equivalent, and when possible, its withdrawal [2].
Aim: The TRANSfORM study aims to characterize the use of OCS for treatment of SLE in Portugal. As secondary objectives, this study will aim to describe treatment patterns with other medications for SLE, assess treatment adherence, characterize the SLE population and estimate their utilization of healthcare resources.
Methods: Non-interventional, cross-sectional, multicentre study conducted through Portuguese community pharmacies affiliated in the National Association of Pharmacies (ANF) that use the Sifarma®2000 software. The study will be conducted nationwide (autonomous regions of Madeira and Azores included). According to a feasibility process based on the pharmacy database, a pool of pharmacies will be invited to participate. We aim to enrol around 250 pharmacies to include a regionally representative sample. Each enrolled pharmacy will assign at least one pharmacist to the study, who will be trained in the study procedures. After approval of the study protocol by a Research Ethics Committee, recruitment of participants will take place over 5 months at the participant pharmacies or until the estimated sample size (N=385) is reached. Patients that fulfil the study criteria will be included after signing an informed consent form. Data will be collected through a paper-based questionnaire at the time of the pharmacy visit. Anonymised data referring to patient reported outcomes, treatment regimen, adherence and exposure to OCS and other SLE medications will be collected.
Results: Description of participating pharmacies (including regional and setting comparison with the universe of pharmacies) and patients' demographics (including age and gender comparison with refusals) will be performed. The sociodemographic, patient reported outcomes, and medication will be described. Categorical variables will be summarized by absolute and relative frequencies. Numeric variables will be summarized using measures of central tendency and dispersion, as appropriate. The missing values will be stated in the corresponding summary table.
Conclusion: The study results will aim to estimate the treatment patterns with OCS and other SLE medications of patients with SLE and their adherence to treatment prescribed by clinicians. These results may contribute to generate new perspectives about unmet needs for improving clinical management of SLE in Portugal.

REFERENCES:

1. Tsoi, A., Nikolopoulos, D., & Parodis, I. (2024). Expert Opinion on Pharmacotherapy, 25(6), 705–716. https://doi.org/10.1080/14656566.2024.2354457
2. Fanouriakis A, Kostopoulou M, Andersen J, et al. Ann Rheum Dis 2024 Jan 2;83(1):15-29. doi: 10.1136/ard-2023-224762.

210 - RHEUMATOID ARTHRITIS REFERRAL CRITERIA: SYSTEMATIC REVIEW OF THE LITERATURE

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Introduction: Referral criteria for Rheumatoid Arthritis (RA) are useful to allow early disease diagnosis and treatment, leading to better outcome with less radiographic progression and need for orthopaedic surgery. Optimized RA referral criteria would allow the clinician and/or the healthcare artificial intelligence faster and better decision regarding patient access to a Rheumatologist, but there is a lack of a validated tool for referring patients.
Objective: To compile and analyse in terms of performance all available RA referral criteria since January 2000 until October 2023.
Material & Methods: We searched EMBASE, PubMed, Scopus and Web of Science in the considered period, using a defined set of strings. Studies were only included if they provided RA referral criteria or Inflamma-

tory/Early Arthritis criteria directed at RA diagnosis.
Results: We identified 19 publications, most of them including symptoms, either arthralgia or stiffness (63%), in referral criteria and only 16% considered constitutional symptoms such as fever, fatigue or weight loss. However, the most prevalent criterion for referral is swollen joints (79%), while tender joints are included in 63% and squeeze test assessment in 33%. Regarding laboratory tests rheumatoid factor is considered in 53% of referral criteria, while ACPA in 32%. Nearly half of the referral criteria consider inflammatory markers (CRP and/or ESR). Family history is present in 21% of the cases.
Discussion and Conclusion: Published RA referral criteria encompass several components (symptoms, signs, laboratory and family history), but none were validated in a large cohort of patients. The vast majority, are either recommendations, published guidelines or based in expert opinion. There is an unmet need for evidence-based validated RA referral criteria.

213 - RELIABILITY OF THE MYBACK PHYSICAL PERFORMANCE BATTERY

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Aims: During an episode of low back pain (LBP) physical performance impairments (PPI) can occur, and in remission of symptoms they can persist (1-4). To evaluate PPI related to cardiorespiratory capacity, motor control, trunk/lower limb resistance, and flexibility the literature suggests several Physical performance (PP) tests. However, the information on the psychometric properties of this tests when applied to individuals who have recovered from LBP is scarce or non-existent (5-28). The aim of this study was to evaluate the intra-rater reliability of a PP battery of tests to assess individuals who have recovered from LBP. Methods: A test-retest study was conducted with a sample of 22 individuals who have recovered from LBP. The participants performed the PP battery composed by 12 tests in two different moments, with an interval of 5.27±1.83 days. For tests scored as numerical variables the Intraclass Correlation Coefficient (ICC3,1) was determined; for tests scored as categorical variables Cohen's k was estimated (29-33). Measurement Error was estimated for all tests. Results: The CCI was >0.87, suggesting high intra-rater reliability. The measurement error was diverse between tests, with standard error of measurement % values varying between 4.47% for the 6-Minute Walk Test (6-MWT) to 19.43% for the Trunk Flexor Test (TFT). Minimal detectable change % varied between 12.38% for 6-MWT and 53.87% for the TFT. Prone Instability, Aberrant Movement Pattern and Modified Thomas tests showed high reliability values (0.71 ≤Cohen's k ≤1) and a high percentage of agreement (>86%). Conclusions: The results of this study showed high intra-rater reliability for the MyBack Physical Performance battery of tests, which supports its use when the aim is to evaluate cardiorespiratory capacity, motor control, trunk and lower limb resistance, and flexibility in individuals who recovered LBP.

220 - CLINICAL INSIGHTS AND DISEASE PROGRESSION FACTORS IN PALINDROMIC RHEUMATISM: A RETROSPECTIVE COHORT STUDY

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Background: Palindromic rheumatism (PR) features recurring self-limiting arthritis and peri-arthritis. A full understanding of its natural course, optimal management, and outcomes remains elusive.
Objectives: This study aims to describe the PR cases within our rheumatology department, focusing on disease profile, treatments, and outcomes, and to conduct a comparative analysis between patients who have and who have not progressed to chronic disease.
Methods: A retrospective observational study was performed using registry data of PR patients at a secondary level hospital. Demographic, clinical and laboratory data were collected. Complete remission, partial remission, and treatment failure were defined as a cessation of attacks for 12 weeks, a minimum 50% reduction in attack frequency, and less than a 50% decrease, respectively. Univariate analysis (significance at $p<0.05$) was performed using chi-square, Mann-Whitney, or t-test, as appropriate.
Results: Thirty-six patients were enrolled, 75.0% female, with mean age at diagnosis of 47.1 ± 10.3 years. Five patients had psoriasis, and 4 a first-degree relative with psoriasis or rheumatoid arthritis (RA). Most patients (52.9%) were diagnosed within a year of symptom onset. Median follow-up was 29.43 months (18.83 - 78.46). Most patients were non-smokers (75%), 58.3% experienced 1 or 2 attacks per month. Half had elevated erythrocyte sedimentation rate and C-reactive protein during attacks. Small joints of the hands, wrists, and knees were the most affected. Peri-arthritis

was present in 58.3%, rheumatoid factor was positive in 25%, anti-cyclic citrullinated peptide (ACPA) in 33.3%, antinuclear antibodies in 16.7%, and HLA-B27 in 13.9%. Data is outlined in Table 1.

Complete remission was achieved in 21 (58.3%) patients, 7 medicated with methotrexate (1 in association with hydroxychloroquine (HCQ)), 5 with nonsteroidal anti-inflammatory drugs or corticosteroids as needed, 5 with sulfasalazine, 2 with HCQ, 1 with deflazacort, and 1 with colchicine. Partial remission was observed in 5.6% and treatment failed in 13.9%. Persistent arthritis was evident in 8 (22.2%) patients, with 4 progressing to RA, 2 to undifferentiated polyarthritis, 1 to psoriatic arthritis, and 1 to systemic lupus erythematosus. Median time to progression was 73.5 (21.5 – 387) months.

Patients progressing to chronic conditions were more frequently positive for ACPA (71.4% VS 25.0%,

TO 220 – TABLE 1. Demographic, clinical and laboratory characteristics and Univariate analysis

Demographic, clinical and laboratory characteristics of total cohort (n = 36).	
Age at Symptom Onset, mean ± standard deviation	42.9 ± 10.7
Age at Diagnosis, mean ± standard deviation	47.1 ± 10.3
Symptom Duration before Diagnosis (years), n(%)	
≤ 1	19 (52.9)
1 to 5	7 (19.4)
≥ 5 to 10	4 (11.1)
≥ 10	6 (16.7)
Follow-up time (months), median (Q1-Q3)	29.4 (18.8 – 78.5)
Female Sex / Male Sex, n(%)	27 (75.0) / 9 (25.0)
Non-Smoker / Current or Former Smoker, n(%)	27 (75.0) / 9 (25.0)
Frequency of attacks before treatment (per month), n (%)	
< 1	7 (19.4)
1	12 (33.3)
2	9 (25.0)
≥ 3	7 (19.4)
Not recorded	1 (2.8)
Involved Structures, n(%)	
MCP joints	32 (88.9)
Hand PIP joints	28 (77.8)
Wrists	22 (61.1)
Knees	20 (55.6)
Hand DIP joints	18 (50.0)
Ankles	17 (47.2)
MTP joints	14 (38.9)
Shoulders	8 (22.0)
Elbows	5 (13.9)
Periarticular structures	21 (58.3)
Positive Rheumatoid Factor, n (%)	9 (25.0)
Positive ACPA, n(%)	12 (33.3)
Positive ANAs, n(%)	6 (16.7)
Positive HLA-B27, n(%)	5 (13.9)
Elevated ESR and CRP during attacks, n(%)	18 (50.0)
Psoriasis, n(%)	5 (13.9)
First-degree Relative with Psoriasis, n(%)	2 (5.6)
First-degree Relative with RA, n(%)	2 (5.6)
Number of Previous Treatments, mean ± standard deviation	1.4 ± 1.1
Previous or current treatment:	
with NSAIDs	36 (100)
with methotrexate	14 (38.9)
with sulfasalazine	12 (33.3)
with hydroxychloroquine	14 (38.9)
with colchicine	4 (11.1)

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TO 220 – TABLE 1. Continuation

Univariate analysis			
	Progression to Chronic Disease (n=8)	Non-Progression to Chronic Disease (n=28)	Analysis value
Age at Symptom Onset < 40 > 40	 5 (62.5) 3 (37.5)	 13 (46.4) 15 (53.6)	 NS
Age at Diagnosis, mean ± standard deviation	50.8 ± 15.2	46.0 ± 8.5	NS
Symptom Duration before Diagnosis, n(%) ≤ 1 year > 1 year	 5 (62.5) 3 (37.5)	 14 (50.0) 14 (50.0)	 NS
Female Sex, n(%) Male Sex, n(%)	6 (75.0) 2 (25.0)	21 (75.0) 7 (25.0)	NS
Non-Smoker, n(%) Current or Former Smoker, n(%)	4 (50.0) 4 (50.0)	23 (82.1) 5 (17.9)	NS
Frequency of attacks before treatment, n (%) ≤ 2 per month >2 per month	 6 (75.0) 2 (25.0)	 22 (81.5) 5 (18.5)	 NS
Involved Structures, n(%) MCP joints Hand PIP joints Wrists Knees Hand DIP joints Ankles MTP joints Shoulders Elbows Periarticular structures	 7 (87.5) 6 (75.0) 5 (62.5) 4 (50.0) 5 (62.5) 7 (87.5) 3 (37.5) 2 (25.0) 1 (12.5) 5 (62.5)	 25 (89.3) 22 (78.6) 17 (60.7) 16 (57.1) 13 (46.4) 10 (35.7) 11 (39.3) 6 (21.4) 4 (14.3) 16 (57.1)	 NS NS NS NS NS p=0.013 NS NS NS NS
Rheumatoid Factor Positivity, n (%)	3 (37.5)	6 (21.4)	NS
Anti-CCP Positivity, n(%)	5 (71.4)	7 (25.0)	p=0.033
ANAs Positivity , n(%)	2 (25.0)	4 (20.0)	NS
HLA-B27 Positivity, n(%)	1 (25.0)	4 (40.0)	NS
Elevated ESR and CRP during attacks, n(%)	4 (50.0)	4 (50.0)	NS
Psoriasis, n(%)	1 (12.5)	4 (14.3)	NS
First-degree Relative with Psoriasis, n(%)	0 (0.0)	2 (7.1)	-
First-degree Relative with RA, n(%)	2 (25.0)	0 (0.0)	-
Number of Previous Treatments, mean ± standard deviation	2.3 ± 0.9	1.2 ± 1.1	p=0.020
Previous or current treatment: with NSAIDs with methotrexate with sulfasalazine with hydroxychloroquine with colchicine	 8 (100.0) 4 (50.0) 3 (37.5) 5 (62.5) 0 (0.0)	 28 (100.0) 10 (35.7) 9 (32.1) 9 (32.1) 4 (14.3)	 NS NS NS NS NS
	Complete or Partial Remission (n=23)	Treatment Failure or Progression to Chronic Disease (n=13)	Analysis value
Current Treatment: with NSAIDs with methotrexate with sulfasalazine with hydroxychloroquine with colchicine	 6 (26.1) 7 (30.4) 6 (26.1) 3 (13.0) 1 (4.3)	 5 (38.5) 3 (23.1) 0 (0.0) 0 (0.0) 0 (0.0)	 NS NS - - -

ANAs, antinuclear antibodies; ACPA, anti-cyclic citrullinated peptide; CRP, C-reactive protein; DIP, distal interphalangeal; ESR, erythrocyte sedimentation rate; HLA-B27, Human Leukocyte Antigen B27; MCP, metacarpophalangeal; MTP, metatarsophalangeal; NSAIDs, Nonsteroidal Anti-Inflammatory Drugs; NS, non-significant; PIP, proximal interphalangeal; RA, rheumatoid arthritis.

p=0.016), displayed more ankle involvement (87.5% VS 35.7%, p=0.016) and underwent a higher number of prior treatments (2.3 VS 1.2, p=0.02). An intriguing but non- significant trend suggested a higher propensity for disease progression in patients with tobacco exposure (50.0% VS 17.9%, p=0.086). No differences were found regarding gender, age, diagnosis delay, presence of psoriasis, family history and treatment options. Comparative analyses are detailed in Table 1.

Conclusion: In summary, his retrospective cohort study offers a greater insight into the clinical nuances of PR and potential key factors associated with disease progression. While these findings enhance our understanding, it is crucial to acknowledge the study's limitations including a relatively small sample size, the retrospective nature of the analysis, and the utilization of univariate analysis, potentially overlooking complex interactions and confounding factors. Research with larger cohorts and prospective approaches are warranted to validate and extend these observations.

TO 228 – TABLE 1. Baseline characteristics of included patients

	N=98
Female sex, no.(%)	78 (79.6)
Age, years, mean ± SD	55.6 ± 11.7
Symptoms duration, months, median [IQR]	12 [0-446]
RF positivity, no. (%)	55 (56.1)
ACPA positivity, no. (%)	51 (52)
Erosive disease, no. (%)	19 (19.4)
csDMARD started at baseline, no. (%)	85 (86.7)
csDMARD monotherapy, no. (%)	82 (83.7)
csDMARDs combination, no. (%)	5 (5.1)
TJC28, mean ± SD	6.5 ± 5.2
SJC28, mean ± SD	6.7 ± 4.7
DAS-28-ESR, mean ± SD	4.8 ± 1.1

Values are presented as mean (SD) or median (IQR), as applicable.

228 - INITIAL GLUCOCORTICOID THERAPY IN NEWLY DIAGNOSED RA-PATIENTS

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Introduction: Glucocorticoids (GC) are currently recommended as bridging therapy in combination with conventional synthetic disease-modifying antirheumatic drugs (csDMARDs) in patients with rheumatoid arthritis (RA) but should be tapered as rapidly as clinically feasible for safety concerns about its long-term use.

Objectives: To evaluate initial GC therapy in newly diagnosed RA-patients and whether initial GC therapy influences the course of disease within 24 months after treatment onset.

Material and Methods: Longitudinal observational cohort study including newly diagnosed RA-patients who visited our outpatient clinic between May 2010 and May 2022 and were naïve for csDMARDs and GC. Descriptive analysis of continuous variables was presented as median and interquartile range (IQR) or mean and standard deviation (SD) according to normality. Categorical variables were presented as frequency or proportions. P-value < 0.05 was considered statistically significant.

Results: A total of 98 patients were included, of which 78 (79.6%) were female with a mean age at baseline (time of diagnosis) of 55.6±11.7 years. 83 patients (84.7%) received oral GC at baseline; 65.3% received a low dose (<7.5 mg prednisolone/day) and 19.4% received a moderate dose (7.5-20 mg). Fifty three percent of the patients who did not initiate GC at baseline ended up receiving it later. In a multivariate regression analysis, only higher DAS28-ESR (OR = 2.1, p=0.011) values showed association with prescription of any dose of GC at baseline and only higher DAS28-ESR (OR 1.8, p=0.048) values were associated with higher GC starting doses. No other baseline variables (sex, age, rheumatoid factor (RF) and anti-citrullinated protein autoantibodies (ACPA) positivity) showed association with either the prescription of GC or it's respective dose. Older age showed to be statistically significant predictor of GC therapy at 24 months (OR 1.1, p=0.026). Additionally, higher DAS28-ESR baseline values showed to be predictive of a lower probability of GC therapy at 24 months (OR=0.405, p=0.046). No other baseline variables such as age, sex, symptoms duration, RF or ACPA presence, erosions, tender (TJC) and swollen joints count (SJC) and csDMARDs initiation showed association with GC therapy at month 24. GC starting dose was not statistically significantly associated with either DAS28 scores at 3, 6, 12, 18 and 24 months (p=0.054) nor remission (DAS28<2.6) at month 24 (p = 0.113), which was reached by 56% of the patients.

Conclusion: These results verified that higher DAS28-VS values at baseline were associated with prescription of higher GC doses. We did not find that GC starting dose influenced DAS28-ESR at any time. In our cohort, a low GC dose was achieved within 6 months in most

of the patients (81.3%), irrespective of the initial GC dose, and about 30% were completely GC- free at 2 years. The main limitations of our study are, first, the relatively low number of included patients. A larger sample may depict differences between GC groups in a more reliable way. Second, we have no data on the GC doses between visits, so we can not exclude temporal GC adaptions and, therefore, provide precise cumulative GC doses.

231 - HOW USEFUL IS CONTRAST-ENHANCED ULTRASOUND IN THE STUDY OF SYNOVITIS? A SYSTEMATIC REVIEW WITH META-ANALYSIS ON THE COMPARISON BETWEEN CONTRAST-ENHANCED ULTRASOUND AND COLOUR OR POWER DOPPLER ULTRASOUND.

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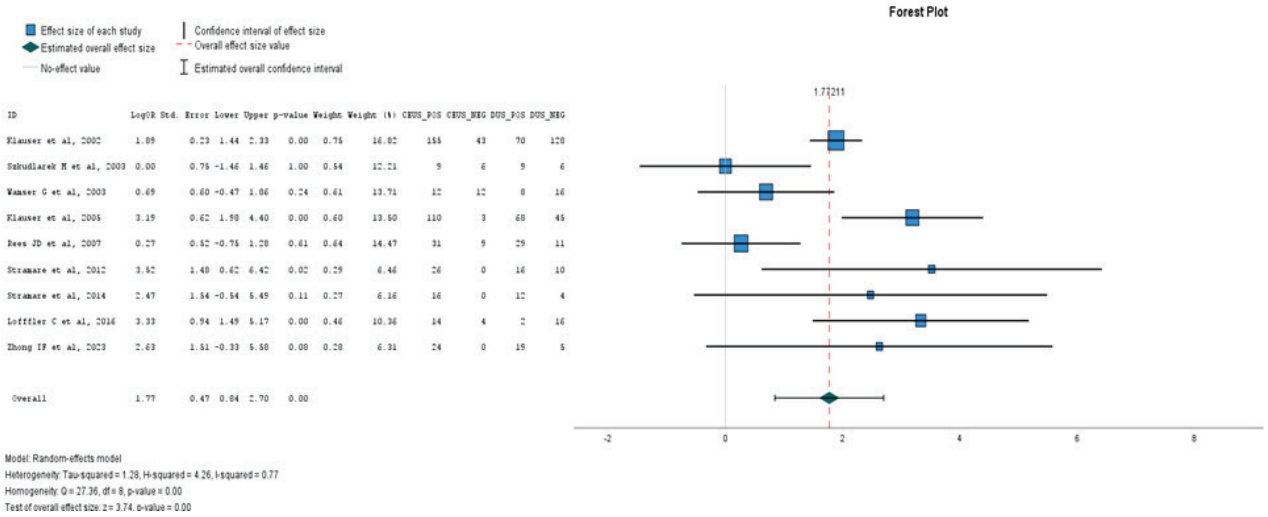
Objective: The primary objective was to evaluate the additional benefits of contrast-enhanced ultrasound (CEUS) for the study of synovitis in rheumatic diseases compared to unenhanced Doppler ultrasound (DUS). Secondary objectives were: 1) to explore the usefulness of CEUS in diagnosis, disease activity monitoring, response to treatment and prognosis; 2) to assess the safety of the use of contrast in this setting and 3) to elucidate CEUS findings in the joints of healthy subjects.

Methods: We searched PubMed/MEDLINE and EMBASE (February 2024) for all English-written published reports of human studies including the use of CEUS for the study of synovitis in rheumatic diseases and healthy subjects (HS). Comparators were clinical markers of disease activity, validated composite scores of disease activity, lab inflammatory markers, and other imaging methods such as radiography, DUS and magnetic resonance imaging (MRI), synovial fluid analysis, arthroscopy, and histology. The outcomes were diagnosis, disease activity monitoring, response to treatment and prognosis. The risk of bias in included studies was evaluated using the QUADAS-2 or QUADAS-C tools as appropriate. Whenever both the DUS and the CEUS were evaluated as a semiquantitative scale, data was transformed in binomial (either positive or negative), i.e. grade 0 was negative and grade>0 was positive, and this data was extracted for meta-analysis, per diagnosis. Results that we were unable to quantitatively aggregate were summarised for each diagnosis per outcome.

Results: Thirty-five studies using CEUS to evaluate 1554 joints from 1195 subjects were included, of which 1364 joints were from 1135 rheumatic patients. We performed a meta-analysis of 9 studies in active



TO 231 – Figure 1. Forest plot comparing PDUS/CDUS with CEUS in the study of synovitis in active RA patients

rheumatoid arthritis patients, showing that from 474 joints evaluated by either PDUS/CDUS and CEUS, the synovitis detection was 49.8% and 84%, respectively, corresponding to an OR of 1.77 (95% CI 0.84-2.7) favouring CEUS (Figure 1). The heterogeneity of the data was high.

CEUS correlated with lab inflammatory markers, clinical activity scores, treatment response, arthroscopy vascular pattern, pro-inflammatory T helper cells in the synovial fluid, and vessel markers in the synovium in patients with various inflammatory joint diseases (IJD), showing overall superiority to PDUS, although with more conflicting results in remission patients. CEUS aids in the differential diagnosis between RA and other IJD or pigmented villonodular synovitis (PVNS) to identify psoriatic arthritis (PsA) in psoriasis patients and coxitis in various rheumatic diseases. In HS, CEUS detected no vascularisation in 5 of 6 studies. CEUS adverse events were rare and mild (0.3% of subjects exposed).

Discussion: CEUS detects more microvascularisation than PDUS in active RA, with an OR of 1.77. The heterogeneity of the data evaluated was high, and the studies' methods were diverse, not permitting the aggregation of most of the data, which may hinder the conclusions.

234 - CLINICAL PROFILE OF VISUAL SYMPTOMS IN GIANT CELL ARTERITIS – INSIGHTS FROM A TERTIARY CENTER COHORT

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Background: Giant Cell Arthritis (GCA) is the most common form of systemic vasculitis in adults. It typically affects the extracranial branches of the carotid artery, and visual symptoms are one of its most feared complications. (1,2)

TO 234 – TABLE 1. Frequency of GCA-associated clinical manifestations, comorbidities, and serological characteristics in patients with and without visual symptoms

	Whole Cohort n = 37 (100%)	Visual Symptoms n = 12 (32.4%)	Without Visual Symptoms n = 25 (67.6%)	p-value
Age at GCA diagnosis median (IQR)	72.0 (10.5)	79.5 (12.8)	70.0 (10.5)	<0.001
Age ≥ 70 years at GCA diagnosis [n (%)]	26.0 (70.3)	12 (100.0)	14.0 (56.0)	0.007
Male [n (%)]	14.0 (37.8)	8.0 (66.7)	6.0 (24.0)	0.027
Clinical features at baseline [n (%)]				
- Constitutional symptoms	22 (59.5)	6 (50.0)	16 (64.0)	0.265
* Fever	6 (16.2)	1 (8.3)	5 (20.0)	0.640
* Weight loss	15 (40.0)	4 (33.3)	11 (44.4)	0.350
* Asthenia	11 (29.7)	1 (8.3)	10 (40.0)	0.060
* Anorexia	11 (29.7)	4 (33.3)	7 (28.0)	1.000
- Cranial symptoms	33 (89.2)	12 (100.0)	21 (84.0)	0.282
* Headache	30 (81.1)	10 (83.3)	20 (80.0)	1.000
* Scalp sensitivity	6 (16.2)	2 (16.7)	4 (16.0)	1.000
* Jaw claudication	18 (48.6)	7 (58.3)	11 (44.0)	0.414
- Polymyalgia rheumatica	14 (37.8)	1 (8.3)	13 (52.0)	0.013
- Abnormalities in the temporal artery	20 (54.1)	8 (66.7)	12 (48.0)	0.343
Other common comorbidities and clinical manifestations [n (%)]				
- Arterial hypertension	26 (70.3)	11 (91.7)	15 (60.0)	0.064
- Dyslipidemia	18 (48.6)	5 (41.7)	13 (52.0)	0.556
- Obesity	1 (2.7)	0 (0)	1 (4.0)	-
- Diabetes mellitus	7 (18.9)	2 (16.7)	5 (20.0)	1.000
Laboratory features at baseline [median (IQR)]				
- Hemoglobin (g/dL)	11.6 (1.5)	11.5 (2.6)	11.6 (1.4)	0.860
- Leukocytes (x109/L)	9.1 (2.9)	10.2 (2.1)	8.7 (2.3)	0.115
- Platelets (x109/L)	316.0 (119.3)	345.0 (128.0)	304.5 (99.3)	0.466
- C-Reactive Protein (mg/dL)	6.8 (8.1)	7.7 (7.0)	6.4 (7.5)	0.905
- Erythrocyte sedimentation rate (mm/h)	71.5 (32.0)	70.0 (24.5)	71.5 (42.3)	0.704
BVAS at baseline median (IQR)	3.0 (3.5)	6.5 (6.8)	2.0 (2.0)	0.002
Relapse [n (%)]	10.0 (27.0)	1.0 (8.3)	9.0 (36.0)	0.015

BVAS: Birmingham Vasculitis Activity Score; GCA: Giant Cell Arteritis; IQR: Interquartile range.

Objectives: To evaluate the prevalence and clinical correlations of visual symptoms in a cohort of GCA patients.
Methods: Cross-sectional single-center study including patients followed in our University Hospital fulfilling the 1990 ACR and/or 2022 ACR/EULAR GCA Classification Criteria. Demographic features and clinical and serological characteristics were compared between the patients with and without visual symptoms. The Birmingham Vasculitis Activity Score (BVAS) at baseline was used to assess disease activity. The continuous variables were described as means or medians, according to distribution. Categorical variables were expressed in percentages. The Chi-squared test/Fisher test and Mann–Whitney U-test were used to compare categorical and continuous variables. A p-value <0.05 was considered statistically significant.
Results: 37 GCA patients were included. Of these, 23 (62.2%) were female, and the median age at diagnosis was 72 (IQR 10.5) years. The median BVAS score at baseline was 3.0 (IQR 3.5). Visual symptoms were registered in 32.4% (n=12), including diplopia (n=5; 41.6%), and unilateral (n=4; 33.3%) and bilateral (n=5; 41.6%) amaurosis. Visual symptoms were associated with male sex (66.7% vs 24.0%, p=0.027), older age at diagnosis (79.5 [IQR 12.8] vs 70.0 [IQR 10.5] years), age ≥ 70 years at diagnosis (100.0% vs 56%, p=0.007) and higher BVAS at baseline (6.5 [IQR 6.8] vs 2.0 [IQR 2.0]). This group also presented a lower prevalence of Polymyalgia rheumatica (8.3% vs 52%, p=0.013) and a lower prevalence of relapse (8.3% vs 56%, p=0.015) (Table 1).
Conclusion: In our cohort of GCA patients, disease-related visual symptoms are significantly associated with male sex, older age at diagnosis (≥ 70 years), higher disease activity at baseline, and absence of polymyalgia rheumatica.

236 - CLINICAL AND IMMUNOLOGICAL FEATURES OF SYSTEMIC SCLEROSIS PATIENTS WITH TELANGIECTASIAS: RESULTS FROM A PORTUGUESE TERTIARY CENTER

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Background: Systemic sclerosis (SSc) is an immune-mediated rheumatic disease characterised by skin fibrosis and internal organs and vasculopathy. Telangiectasias are dilated, non-inflammatory, superficial postcapillary venules that occur in up to 70% of SSc patients and may be associated with greater systemic involvement.
Objective: To describe and compare the demographic features, SSc subsets, and main clinical and immunological features in SSc patients with and without telangiectasias.
Materials and methods: Cross-sectional single-center study including SSc patients fulfilling the ACR'1980, ACR/EULAR'2013 or LeRoy's classification criteria, followed in our University Hospital. Demographic features, SSc subtype, cumulative clinical manifestations, immunological characteristics, respiratory function tests and nailfold capillaroscopy pattern were collected. The chi-squared test/Fisher test and Mann-Whitney U test were used to compare the groups. P-value <0.05 was considered statistically significant.
Results: A total of 158 patients were included. Telangiectasias were found in 50 (31.6%) patients. Of these, 90.0% (n=45) were female, and the median age was 54.0 (IQR 14.3) years. Patients with telangiectasias had a longer disease duration [114.0 (IQR 96.0) vs 65.0 (IQR 105.0) months, p=0.017]. VEDOSS was the SSc subtype least associated with telangiectasias (6.8 vs 29.8%, p<0.001). Regarding clinical manifestations, there was a statistically significant higher proportion of Raynaud phenomenon (100.0% vs 90.7%, p=0.031), digital ulcers/pitting scars (40.0% vs 26.4%, p=0.040), articular involvement (36.0% vs 20.4%, p=0.036), gastrointestinal involvement (42.0% vs 21.3%, p=0.007), and a higher median in mRSS [7.0 (IQR 10.3) vs 4.1 (IQR 6.0), p=<0.001] in this group. No differences in demographic characteristics, immunological features, respiratory function tests and nailfold capillaroscopy pattern were found between the groups (Table 1).
Conclusion: In this cohort, telangiectasias were present in 50 (31.6%) patients. These patients represent a subset of SSc with a significantly higher frequency of Raynaud phenomenon, digital ulcers/pitting scars, articular and gastrointestinal involvement, and increased skin thickening. These findings suggest that telangiectasias may be a more severe SSc disease marker and potentially guide treatment decisions. However, further prospective studies are needed.

241 - RHEUMATOID ARTHRITIS ACTIVITY AND FUNCTIONAL CAPACITY WITH ACUPUNCTURE IN COMPLEMENTARY TREATMENT

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TO 236 – TABLE 1. Frequency of SSc-associated clinical manifestations and laboratory features in SS patients with and without telangiectasis

	Whole cohort n=158 (100%)	With telangiectasis n=50 (31.6%)	Without telangiectasis n = 108 (68.4%)	p-value
Age at diagnosis, years (median IQR)	52.0 (15.3)	54.0 (14.3)	51.5 (17.5)	0.284
Disease duration, month (median IQR)	11.1 (11.0)	114.0 (96.0)	65.0 (105.0)	0.017
Sex (n [%])				
- Female	135 (85.4)	45 (90.0)	90 (83.3)	0.269
- Male	23 (14.6)	5 (10.0)	18 (16.7)	
Subtype (n [%])				
- VEDOSS	37 (23.4)	4 (8.0)	33 (30.6)	0.002
- Limited cutaneous	91 (57.6)	34 (68.0)	57 (52.8)	0.072
- Diffuse cutaneous	30 (19.0)	12 (24.0)	18 (16.7)	0.274
Clinical features (n [%])				
- Raynaud´s Phenomenon	148 (93.7)	50 (100.0)	98 (90.7)	0.031
- Skin thickening (mRSS)	4.0 (9.0)	7.0 (10.3)	4.1 (6.0)	<0.001
- Digital ulcers or pitting scars	46 (29.1)	20 (40.0)	26 (24.1)	0.040
- Articular involvement	40 (25.3)	18 (36.0)	22 (20.4)	0.036
- Myositis	5 (3.2)	3 (6.0)	2 (1.9)	0.327
- Gastrointestinal involvement	44 (27.8)	21 (42.0)	23 (21.3)	0.007
- Renal involvement	1 (0.6)	1 (2.0)	0 (0)	-
- Interstitial lung disease	36 (22.8)	13 (26.0)	23 (21.3)	0.512
- Pulmonary arterial hypertension	22 (13.9)	10 (20.0)	12 (11.1)	0.133
Laboratory features (n [%])				
- Antinuclear antibodies	155 (98.1)	49 (98.0)	106 (98.1)	1.000
- Anti-centromere	94 (59.5)	31 (62.0)	63 (58.3)	0.662
- Anti-topoisomerase I	33 (20.9)	10 (20.0)	23 (21.3)	0.852
- Anti-RNA polymerase III	4 (2.5)	2 (4.0)	2 (1.9)	0.592
- C Reactive Protein (median IQR)	0.6 (0.6)	0.2 (0.6)	0.2 (0.6)	0.922
- Erythrocyte sedimentation rate (median IQR)	14 (16.0)	16.0 (17.0)	13.0 (16.0)	0.218
Respiratory Function Tests (median IQR)				
- DLCO (mmol/min/kpa)	89 (28.2)	88.6 (34.0)	89.3 (24.8)	0.781
- FVC (%)	100 (27.4)	112.9 (24.8)	109.5 (24.0)	0.170
Nailfold capillaroscopy pattern (n [%])*				
- Early	32 (20.3)	9 (18.0)	23 (21.3)	0.636
- Active	35 (22.2)	14 (28.0)	21 (19.4)	0.222
- Late	16 (10.1)	8 (16.0)	8 (7.4)	0.094
- Non-specific	58 (36.7)	14 (28.0)	44 (40.7)	1.121
- Normal	11 (7.0)	3 (6.0)	8 (7.4)	0.738

IQR: Interquartile range; VEDOSS: Very Early Diagnosis of Systemic Sclerosis. DLCO: Diffusing lung capacity for carbon monoxide; FVC: Forced vital capacity; *6 missings. Interstitial lung disease diagnosis was confirmed by chest high-resolution computed tomography. Pulmonary artery hypertension diagnosis was based on echocardiography and/or right heart catheterisation evaluation.

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Background: Nowadays, the RA treatment paradigm is based on the treat-to-target strategy, where the target is remission/decreased RA activity, as quickly and consistently as possible. The complex pathophysiological mechanism of RA is not yet fully understood, opening the door to new requirements and therapies. Acupuncture, based on Traditional Chinese Medicine, has emerged as a complement to the conventional treatments already in place and is recommended by the World Health Organization for its therapeutic effect.

Methods: Experimental, randomized, prospective and controlled study. Research question: How does acupuncture affect the functional status and activity phase of RA? The sample consists of 100 patients (50 Control Group "CG" and 50 Experimental Group "EG") who underwent 8 consecutive weeks of acupuncture (20 minutes). Two evaluations were carried out: 1st evaluation before the first complementary acupuncture treatment (tcA) and 2nd evaluation: one week after the last acupuncture treatment. Data collection: for 6 months.

Evaluation Instruments: Questionnaire characterization sample; Disease Activity Score 28-CRP (DAS-CRP); Health Assessment Questionnaire – Disability Index (HAQ-DI). Statistical analyses: Software Rstudio version 1.1.1003. Test hypotheses (significance level α= 0.05).

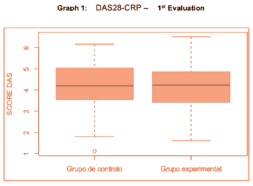
Results: In 2nd evaluation (after tcA), the average DAS28-CRP value in the sample, decreased 4.19 to 3.74, which was explained by the reduction in EG (4.13 to 2.95), since in CG we observed an increase from 4.25 to 4.54. For a significance level of 0.05, the hypothesis test applied enables us to reject the similarity between the mean DAS28-CRP values of the CG and EG after treatment, as the p-value is significantly lower than 0.05 (p <0.01).

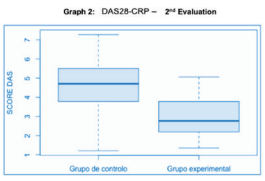
By counting the 28 joints, we verified that the average value of tender (TJ) and swollen joints (SJ) decreased overall from the 1st to the 2nd evaluation (TJ 6.81 to 5.52) and (SJ 6.55 to 4.77). The p-values of the hypothesis tests allow us to conclude that in the 1st evaluation the CG and the EG were similar (p 0.22 and p 0.93), i.e. for a significance level of 0.05 it is not possible to reject the null hypothesis that the means of the two groups were equal. For the means obtained in the 2nd evaluation and after the tcA, the p-value obtained (p <0.01) enables us to assume a difference between the means of the two groups, control and experimental.

After the tcA, the mean value of the HAQ-DI score fell (0.65 to 0.57) due to the decrease in the mean value in the SG (0.57 to 0.26), while in the CG there was an increase (0.74 to 0.88). The hypothesis test applied allowed us to rule out the similarity between the mean HAQ-DI values in the groups, as the p-value (p <0.01) was significantly lower than 0.05.

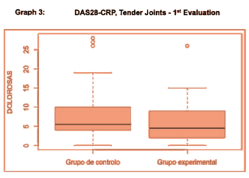
Conclusions: Acupuncture was shown to influence the results of the DAS28-CRP, with the final score changing from "Moderate Activity" to "Low Activity" after tcA. The count of the 28 joints obtained statistically significant results after tcA, with a decrease of 60% in TJ and 70% in SJ. Patients in the EG showed a 50% decrease in the HAQ-DI score, with a score close to 0, classified as "No Difficulty" in performing activities of daily living. Acupuncture was shown to influence the relief of acute pain complaints and over time, as well as contributing to a significant reduction in functional disability in RA patients undergoing tcA, highlighting its effectiveness in managing and controlling the disease, which could produce prolonged effects and contribute to the benefits of sustained RA remission.

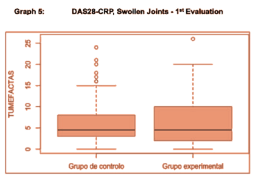
Variável	Amostra	Grupo		Teste de hipóteses
		Controlo	Experimental	
DAS28-CRP	Média (SD)	Média (SD)	Média (SD)	*Valor-p*
Primeira avaliação	4.192 (1.087)	4.245 (1.096)	4.139 (1.088)	0.629
Segunda avaliação	3.747 (1.371)	4.541 (1.279)	2.953 (0.936)	< 0.01

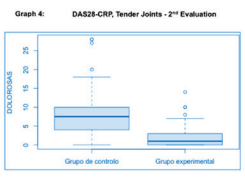


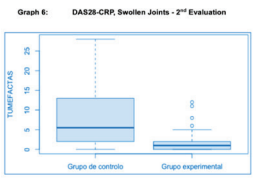


Variável	Amostra	Grupo		Teste de hipóteses
		Controlo	Experimental	
articulações DOLOROSAS (AD)	Média (SD)	Média (SD)	Média (SD)	*Valor-p*
Primeira avaliação	6.810 (6.260)	7.580 (7.186)	6.040 (5.131)	0.221
Segunda avaliação	5.520 (6.203)	8.660 (6.951)	2.380 (3.050)	< 0.01
articulações TUMEFACTAS (AT)	Média (SD)	Média (SD)	Média (SD)	*Valor-p*
Primeira avaliação	6.550 (5.783)	6.600 (5.945)	6.500 (5.676)	0.932
Segunda avaliação	4.770 (6.085)	7.720 (7.060)	1.820 (2.685)	< 0.01

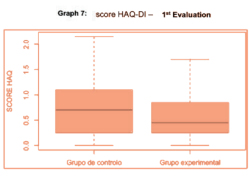


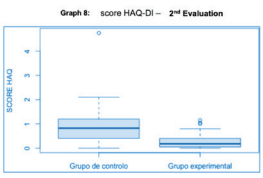






Variável	Amostra	Grupo		Teste de hipóteses
		Controlo	Experimental	
HAQ-DI	Média (SD)	Média (SD)	Média (SD)	*Valor-p*
Primeira avaliação	0.659 (0.513)	0.746 (0.563)	0.571 (0.445)	0.089
Segunda avaliação	0.577 (0.660)	0.888 (0.773)	0.265 (0.290)	< 0.01





TO 241 – Figure 1.

242 - PRODUCTIVITY LOSS IN PORTUGUESE PATIENTS WITH SYSTEMIC SCLEROSIS

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Introduction: Systemic Sclerosis (SSc) is a chronic autoimmune disease which can affect patients on working age and have a significant impact on work productivity and quality of life. In Portugal, no study evaluat-

TO 242 – TABLE 1. Clinical and demographic characteristics of patients with Systemic Sclerosis according to work status.

	Employed (n= 16)	Retired (n=21)	Unemployed, not retired (n=3)
Age (years), median (min-máx)	53.50 (29-67)	70 (56-83)	62 (59-63)
Female: n (%)	14 (41.2%)	17 (50%)	3 (8.8%)
Disease subtype			
Limited	13 (81.3%)	14 (66.7%)	2 (66.7%)
Diffuse	2 (12.5%)	6 (28.6%)	0 (0)
VEDOSS	0 (0)	(0)	1 (33.3%)
Sine scleroderma	1 (2.5%)	1 (2.5%)	0 (0)
Years of disease, median (min-máx)	4.50 (1-11)	10 (2-19)	7 (2-10)
Body Mass Index	25.72 (20.55-41.41)	25.71 (16.12-32.03)	24.46 (24.30-26.17)
Disease manifestations			
Digital ulcers	3 (18.8%)	10 (47.6%)	0 (0)
Calcinosis	1 (6.3%)	5 (23.8%)	0 (0)
Synovitis	0 (0)	3 (14.3%)	0 (0)
Interstitial Lung Disease	2 (12.5%)	8 (38.1%)	0 (0)
Pulmonary Hypertension	0 (0)	3 (14.3%)	0 (0)
Gastro-intestinal involvement	8 (50.0%)	13 (61.9%)	1 (33.3%)
Rodnan score (mRSS), median (min-max)	7 (0-28)	6 (0-24)	14 (0-18)
SHAQ			
Gastro-intestinal symptoms	0 (0-2.70)	0 (0-1.80)	0 (0-1.50)
Respiratory symptoms	0 (0-1.80)	0 (0-2.70)	0 (0-0.90)
Raynaud phenomenon	0.45 (0-2.40)	0.60 (0-2.70)	0.60 (0-0.90)
Ulcers	0 (0-0.90)	0 (0-2.70)	0 (0-0)
Global	0.30 (0-2.10)	1.80 (0-2.70)	1.20 (0-90-1.50)
EQ5D, median (min-máx)	0.8833 (0.2894-1.000)	0.4285 (0.1644-0.3547)	0.5546 (0.0068-0.6499)
FACIT, median (min-máx)	48 (24-52)	29 (15-50)	30 (14-38)
PGA VAS, median (min-máx)	1 (0-7)	6 (0-8)	5 (3-6)
WPAI			
Absenteeism	0 (0-14.29)		
Presenteeism	0 (0-70)		
OWI	0 (0-73.33)		
DAI	20 (0-70.0)		

ing productivity in SSc with a validated questionnaire has yet been performed.

Material and Methods: We conducted a cross-sectional study on patients, aged ≥18, who fulfilled the ACR/EULAR 2013 classification criteria for SSc. Socio-demographic and clinical data were collected. To assess work productivity, we applied the questionnaire Work Productivity and Activity Impairment General Health (WPAI), with the calculation of 4 productivity parameters: absenteeism (A), presenteeism (P), overall work impairment (OWI) and daily activity impairment (DAI). Patients also answered the Scleroderma Health Assessment Questionnaire (SHAQ), Patient Global Assessment through visual analog scale (VAS PGA) and the health-related quality of life questionnaire EQ-5D. Spearman's correlation (rs) was used to evaluate the validity of WPAI.

Results: Forty patients (median age of 62 (29-83) years old; 85% female) agreed to participate in this study. Most of the patients had limited systemic sclerosis. Table 1 summarizes clinical and demographic characteristics according to work status. Less than half of the patients were employed (n=16; 40%) and they were younger and had a shorter duration of disease. Retired patients had also more organ involvement.

Regarding SSs symptoms, gastro-intestinal symptoms (A: rs=0.833 (p<0.001); P: rs=0.574 (p=0.020); OWI: rs=0.598 (p=0.014); DAI: rs=0.345 (p=0.029), respiratory symptoms (A: rs= 0.894 (p<0.001); P: rs=0.615 (p=0.011); OWI: rs=0.640 (p=0.008); DAI: rs=0.686 (p<0.001)), and global impact (A: rs=0.663 (p=0.004); P: rs=0.687 (p=0.003); OWI: rs=0.689 (p=0.003); DAI: rs=0.796 (p<0.001)) correlated with all 4 parameters of WPAI. Raynaud Phenomenon correlated with A (rs=0.552 (p=0.022)) and DAI (rs=0.421 (p=0.007)) and digital ulcers with P and OWI (rs=0.507 (p=0.045)). FACIT and EQ5D correlated negatively with WPAI (FACIT - A: rs=-0.734 (p<0.001); P: rs=-0.790 (p<0.001); OWI: rs=-0.794 (p<0.001); DAI: rs=-0.709 (p<0.001); EQ5D – A: rs=-0.673 (p=0.003); P: rs=-0.585 (p=0.017); OWI: rs=-0.590 (p=0.016); DAI: rs=-0.834 (p<0.001)). HAQ (A: rs=0.966 (p<0.001); P: rs=0.857 (p=0.014); OWI: rs=0.857 (p=0.014); DAI: rs=0.559 (p=0.007)) and PGA VAS (A: rs=0.741 (p<0.001); P: rs=0.523 (p=0.037); OWI: rs=0.539 (p=0.031); DAI: rs=0.762 (p<0.001)) also showed association with WPAI.

Conclusion: This study is the first to describe SSc-related work impairment in Portugal, using a validated tool. Despite the limitations, moderate to strong correlations were found between WPAI parameters and different health outcome measures, strengthening the validity for use in SSc. Bigger cohorts are needed to confirm these results.

249 - INCREASED BODY MASS INDEX AND FAT MASS ARE ASSOCIATED WITH HIGHER SYMPTOM BURDEN AND REDUCED SALIVARY FLOW IN PATIENTS WITH SJÖGREN'S DISEASE

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Introduction: Sjögren's disease (SjD) oral symptoms cause major discomfort and reduce patient's quality of life. In addition, it can also impair dietary intake and impact the patient's nutritional status, leading to nutritional risk and body composition alterations. Limited evidence suggests that SjD patients have higher consumption of fatty foods and a lower carbohydrate intake, possibly related to difficulties in mastication and swallowing. This might increase the percentage

of body fat and body weight, while reducing muscle mass.

Objectives: We aimed to explore the relationship between nutritional status, dietary pattern and symptom severity in patients with SjD.

Methods: Cross-sectional study including patients fulfilling ACR/EULAR 2016 criteria for SjD. We assessed symptom burden and disease impact using validated questionnaires such as the EULAR Sjögren's Syndrome Patient Reported Index (ESSPRI; pain, fatigue, dryness), Xerostomia Inventory (XI; oral dryness degree/impact) and the EULAR Sicca Score (ESS; oral/ocular dryness severity). Bodyweight and height were determined using a digital scale and a stadiometer, respectively. Body mass index (BMI) was calculated as weight/hight2 (kg/m2) and classified according to the World Health Organization cut-off values. Body fat mass percentage (FM) was measured by bioelectrical impedance analysis using a Bodystat QuadScan 4000 equipment. Adherence to the Mediterranean Diet (AdhMD) was evaluated using the PREDIMED 14-item questionnaire, and classified as high (≥11 points), moderate (9-10 points), and low (0-8 points), low AdhMD. Further, we compared patients with normal or reduced unstimulated salivary flow using Pearson and Spearman correlation analyses, or Student's T-test / Mann-Whitney U Test, as appropriate. Mean± standard deviation or median (interquartile range) are displayed according to data distribution. P-value was deemed significant at <0.05.

Results: A total of 70 patients with SjD (98.6% women; median age 58.0(17.0) years) with a median disease duration of 10.5±8.5 years were included (Table 1). Mean BMI and FM were 27.0±4.9 and 39.1±7.3, respectively. Most patients in our sample were overweight (40.0%) and 22.8% were obese. Median (interquartile range) AdhMD was 8.5 (3) points. More than half of our sample had a low (51%) or moderate (40.0%) AdhMd. Only a minority had high AdhMD (8.6%). Higher FM was positively and significantly correlated with ESSPRI (r=0.307; p=0.010), ESS (r=0.286; p=0.017) and XI (r=0.292; p=0.014). A higher BMI was also correlated with ESSPRI (r=0.342; p=0.004) and XI (r=0.275; p=0.021) scores, and a trend was observed with ESS (r=0.234; p=0.052). Patients with reduced unstimulated salivary flow had higher FM (41.1±7.8 vs 37.3±6.4, p=0.029) and a trend for higher mean BMI (28.1±5.8 vs 25.9±3.5; p=0.056) compared to patients with preserved exocrine function. No correlations were found between AdhMD and ESSPRI, XI or ESS. Further, AdhMD was similar between patients with normal or reduced salivary flow (p=0.886).

Conclusion: Overall, higher body mass index and, especially, higher adiposity were associated with higher SjD symptom burden and reduced exocrine function. Adherence to a Mediterranean diet did not seem to decrease disease impact. These results suggest that body composition changes may be associated with worse clinical outcomes in SjD, an hypothesis that warrants further investigation in larger studies.

TO 249 – TABLE 1. Clinical and sociodemographic characteristics of the study participants

	Whole sample (n=70)	Normal salivary flow (n=36)	Reduced salivary flow (n=34)	p-value
Age (years), median(IR)	58.0(17.0)	54.0 (24.0)	59.0 (12.0)	0.106[a]
Female, n (%)	69 (98.6)	35 (97.2)	34 (100.0)	0.328[b]
Disease duration (years), median(IR)	8.0 (12.0)	9.0 (13.0)	8.0 (11.0)	0.509[a]
ANA, n (%)	63 (90.0)	34 (94.4)	29 (85.3)	0.202[c]
Anti-Ro, n (%)	60 (85.7)	33 (91.7)	27 (79.4)	0.143[c]
Anti-La, n (%)	37 (52.9)	23 (63.9)	14 (41.2)	0.057[c]
Rheumatoid factor, n (%)	40 (57.1)	20 (55.6)	20 (58.8)	0.888[c]
Schirmer's I test ≤5mm/5min, n(%)	26 (37.1)	9 (25.0)	17 (50.0)	**0.029[c]**
ESSDAI, median(IR)	1.0 (2.0)	1.5 (3.0)	0.0 (2.0)	**0.047[a]**
BMI, mean±SD	27.0±4.9	25.9±3.4	28.1±5.8	0.056[c]
BMI classification				
Underweight, n(%)	2 (2.9)	2 (5.6)	0 (0.0)	
Normal weight, n(%)	24 (34.3)	13 (36.1)	11 (32.4)	
Overweight, n(%)	28 (40.0)	16 (44.4)	12 (35.3)	0.148[b]
Obesity class I, n(%)	11 (15.7)	5 (13.9)	6 (17.6)	
Obesity class II, n(%)	4 (5.7)	0 (0.0)	4 (11.8)	
Obesity class III, n(%)	1 (1.4)	0 (0.0)	1 (2.9)	
Fat mass, mean±SD	39.1±7.3	37.3±6.4	41.1±7.8	**0.029[c]**
Fat mass classification*				
Normal, n(%)	5 (7.1)	3 (8.3)	2 (5.9)	0.691[b]
Excess, n(%)	65 (92.9)	33 (91.7)	32 (94.1)	
PREDIMED score, median (IR)	8.5 (3.0)	8.0 (3.0)	9.0 (2.0)	0.886[a]
PREDIMED classification				
Low AdhMD, n(%)	36 (51.4)	21 (58.3)	15 (44.1)	
Moderate AdhMD, n (%)	28 (40.0)	12 (33.3)	16 (47.2)	0.469[c]
High AdhMD, n (%)	6 (8.6)	3 (8.3)	3 (8.8)	
ESSPRI score, mean±SD	5.9±2.1	5.6±2.0	6.3±2.2	0.183[c]
ESS score, mean±SD	5.8±2.3	5.5±2.4	6.1±2.2	0.238[c]
XI score, mean±SD	38.0±9.7	35.9±10.3	40.2±8.7	0.062[c]

*FM was classified according to the Bodystat QuadScan 4000 equipment reference values.
[a]Mann-Whitney U Test
[b]Chi-squared test
[c]Student's T-test

Abbreviations: ANA: Antinuclear antibody; AdhMD: Adherance to the Mediterranean Diet; BMI: Body Mass Index (kg/m^2); ESS: EULAR Sicca Score; ESSPRI: EULAR Sjögren's Syndrome Patient Reported Index; FM: Fat mass percentage; PREDIMED: SD: standard deviation; IR: Interquertil range; RF: Rheumatoid factor; XI: Xerostomia. Missing data: FR (n=69); Schirmer's test (n=61); ESSDAI (n=62)

251 - PREDICTORS OF FLARE OF INTERSTITIAL LUNG DISEASE IN PATIENTS WITH INFLAMMATORY IDIOPATHIC MYOSITIS: A RETROSPECTIVE COHORT STUDY

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Background: Idiopathic Inflammatory Myopathies (IIMs) are heterogeneous systemic rheumatic disorders characterized by muscle inflammation. Interstitial lung disease (ILD) is the most common extramuscular manifestation, being present in 75%, and is the major

TO 251 – TABLE 1. Clinical and serological characteristics of patients with Idiopathic Inflammatory

Characteristics	ILD in remission (n=46)	Flare of ILD (n=17)	*p*-value
Age (years), med (min-max)	63 (34-83)	70 (52-81)	0.069
Disease duration (months), med (min-max)	69 (71-312)	74 (16-148)	0.282
Gender			0.903
Female	29 (63.0%)	11 (64.7%)	
Male	17 (37.0%)	6 (35.3%)	
Smoking status			0.119
Never	26 (56.5%)	14 (82.4%)	
Active smoker	8 (17.4%)	2 (11.8%)	
Ex-smoker	12 (26.1%)	1 (5.9%)	0.377
Age at diagnosis	**57 (30-80)**	**62 (77-78)**	**0.049**
Diagnosis			0.332
Dermatomyositis	11 (23.9%)	5 (29.4%)	
Polymyositis	1 (2.2%)	2 (11.8%)	
Anti-synthetase syndrome	31 (67.4%)	10 (58.8%)	
Escleromyositis	3 (6.5%)	0 (0.0%)	
Antibodies			
Anti-Mi2	0 (0.0%)	1 (100.0%)	0.270
Anti-SAE	1 (100.0%)	0 (0.0%)	0.540
Anti-MDA5	8 (17.4%)	4 (23.5%)	0.582
Anti-JO1	24 (52.2%)	10 (41.2%)	0.438
Anti-PL12	1 (2.2%)	0 (0.0%)	0.426
Anti-PL7	5 (10.9%)	3 (17.6%)	0.473
Anti-EJ	1 (2.2%)	0 (0.0%)	0.540
Anti-PM/Scl	4 (8.7%)	0 (0.0%)	0.567
Anti-Ro52	18 (39.1%)	6 (35.3%)	0.781
Seronegative	1 (2.9%)	1 (10.0%)	0.923
Other manifestations			
Myositis	28 (60.9%)	11 (64.7%)	0.781
Dysphagia	9 (20.0%)	5 (29.4%)	0.429
Diaphragm paralysis	2 (4.4%)	1 (5.9%)	0.814
Arthritis	29 (63.0%)	8 (47.1%)	0.253
Skin involvement			
Mechanic hands	17 (37.0%)	4 (23.5%)	0.316
Heliotrope rash	4 (8.7%)	3 (17.6%)	0.316
Gottron	13 (28.3%)	2 (11.8%)	0.172
V-neck sign	4 (8.7%)	1 (5.9%)	0.714
Raynaud phenomenon	16 (34.8%)	5 (29.4%)	0.688
Capillaroscopy			0.944
Normal	4 (22.0%)	1 (20.0%)	
Unspecific	5 (27.8%)	2 (40.0%)	
Early scleroderma pattern	1 (5.6%)	0 (0.0%)	
Active scleroderma pattern	7 (38.9%)	2 (40.0%)	
Late scleroderma pattern	1 (5.6%)	0 (0.0%)	
Digital ulcers	5 (10.9%)	1 (5.9%)	0.549
Cutaneous sclerosis	4 (8.7%)	0 (0.0%)	0.209
Myocarditis	2 (4.3%)	1 (5.9%)	0.800

determinant of morbidity and mortality in IIM. Since recurrence is not uncommon and may result in progression of pulmonary fibrosis, maintaining ILD in remission is critical. However, recurrence of ILD is usually unpredictable and little is known about its relevant risk factors. We aimed to identify risk factors for recurrence of ILD in patients with IIM.

Methods: This retrospective observational study included patients with IIM according to the 2017 ACR/EULAR Classification Criteria, with ILD, observed in the Rheumatology-Pneumology visits between March 2023 and March 2024. Patients with anti-aminoacyl-tRNA synthetase antibodies were diagnosed with Anti-Synthetase Syndrome (ASyS). Clinical and serological variables were collected. ILD flare was considered when patients had at least two of three changes: clinical worsening and/or radiological and/or functional progression, such as a worsening of 10% in FVC and/or 20% in DLCO, during a period of 24 months. The variables' associations were tested using the Chi-Square Test or Fischer's Exact Test, ANOVA or Kruskal-Wallis test, as appropriate. Independent predictors of ILD recurrence, adjusted for sex and age at diagnosis, were identified through binomial logistic regression modelling. SPSS v. 27 was used for statistical analysis, and the significance level will be defined as 2-sided p≤0.05.

Results: We included 71 patients, 45 (63.4%) females, of which 48 (67.6%) had ASyS, 17 (23.9%) had Dermatomyositis (DM), 3 (4.2%) had Polymyositis and 3 (4.2%) had Escleromyositis). The median age at diagnosis was 58 (min 27 – max 83) years, and the median disease duration at the last follow-up was 66.5 (min 8 – max 312) months. In our cohort, 17 (27.0%) patients had an ILD flare with the need for therapy: 10 (58.8%) had ASyS, 5 (29.4%) had DM and 2 (11.8%) had PM. We didn't find differences between IIM subgroups either in antibody status (Table 1). Compared to other IIM patients who didn't have a flare of ILD, patients with a flare of ILD were older at disease onset (57 (30-80) vs. 62 (77-78), p=0.049) and had more frequently cancer (2 (4.3%) vs. 4 (23.5%), p=0.041). We didn't find statistical differences between the disease duration at the last follow-up. Smoking status was not statistically different between groups. Clinically, there were no differences regarding the frequency of myositis, arthritis, cutaneous involvement, Raynaud's phenomenon, myocarditis, or pulmonary hypertension. In the multivariate analysis, active smoking (OR 18.99 (1.16-310.53), p=0.030) was an independent predictor of ILD flare in IIM patients, regardless of sex and age at diagnosis. ILD flares were treated with nintedanib (16.9%), cyclosporine (11.3%), rituximab (8.5%), mycophenolate (2.8%), azathioprine (2.8%), intravenous immunoglobulin (2.8%), and baricitib (1.4%).

Conclusion: In our cohort, active smoking was an independent predictor of ILD flare. We didn't find differences in myositis subgroups nor antibodies such as ARS, anti-MDA5, or anti-Ro52, as described in the literature. However, studies in larger cohorts are needed to confirm possible predictors of ILD flare in IIM. As in other rheumatological conditions, clinicians should be particularly alert to smoking status, which is a known cause of disease worsening, especially in lung involvement.

258 - ARE SYSTEMIC LUPUS ERYTHEMATOSUS AND SJOGREN SYNDROME TWO SIDES OF THE SAME DISEASE SPECTRUM? – A SINGLE CENTER RETROSPECTIVE STUDY

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Introduction: Lupus erythematosus (SLE) and Sjögren's syndrome (SjS) present overlapping clinical features and immunological biomarkers. They share several aspects from the immunopathology point of view and can concomitantly affect the same patient in 15% of cases. In some cases, primary SjS (pSjS) and SLE differential diagnosis is not always easy to be performed. Therefore, we aim to evaluate clinical, serological, immunological, and therapeutical differences between patients with SLE, pSjS, and SLE + SjS to better understand their differences, overlapping characteristics, and outcomes.

Methods: This retrospective observational study included patients with SLE according to the 2012 Systemic Lupus International Collaborating Clinics (SLICC) and/or 2019 ACR/EULAR Classification Criteria for SLE, and patients classified with pSjS according to the 2016 ACR-EULAR Classification Criteria for SjS, who were observed at the Rheumatology Department between March 2023 and March 2024. Patients who fulfilled both the SLE and SjS classification were classified as SLE-SjS. Variables' associations were tested using the Chi-Square Test or Fischer's Exact Test, ANOVA, or Kruskal-Wallis test, as appropriate, followed by post-hoc analysis in case of statistical significance, with Bonferroni correction, while considering SLE patients as the reference group. SPSS v. 27 was used for statistical analysis, and the significance level will be defined as 2-sided p≤0.05.

Results: We included 283 patients, of whom 121 had SLE (%), 131 had pSjS (%), and 31 SLE + SjS (2%). Patients' characteristics are described in Table 1. In comparison to SLE patients, the SLE-SjS group was older at inclusion and onset and had a longer disease course. Sicca syndrome, Raynaud phenomenon, and interstitial lung disease were more frequent in patients with primary SjS and SLE-SjS than SLE only. Contrariwise, patients with SLE and SLE-SjS had more cutaneous involvement, more precisely acute cutaneous lupus, alopecia, oral aphthous, glomerulonephritis, arthritis, and cytopenia. Serositis and hemolytic anemia were more prominent in the SLE-only group. Anti-SSA, anti-SSB, RF, and gammaglobulinemia were higher in the SjS and SLE-SjS groups. Conversely, hypocomplementemia, anti-dsDNA, and anti-phospholipid antibodies were observed more in SLE patients. When analyzing differences between SLE patients with and without SSA/Ro, we found that patients with SSA/Ro had more sicca syndrome, oral aphthous, hypergammaglobulinemia, anti-Sm, anti-SSB/La, and Rheumatic Factor. Conversely, lupus anti-coagulant was more seen in the group of SLE without anti-SSA/Ro. No differences were found in disease activity and damage scores between groups. Regarding treatment, more patients with SLE were treated with steroids, hydroxychloroquine, and immunosuppressants.

Discussion/Conclusion: As expected, despite having similar clinical and serological findings, patients with SLE and SjS present them in different proportions. Furthermore, SLE-SjS presented as a subgroup of patients with clinical and serologic features found both in SLE and SjS patients. Besides sicca syndrome and oral aphthous, we found that patients with SLE and anti-Ro/SSA were also more similar to SjS than patients without it. This raises the possibility of both conditions being part of the same spectrum, with possibly

TO 258 – TABLE 1. Clinical characteristics of patients with Systemic Lupus Erythematosus (SLE), Sjogren Syndrome (SjS), and SLE with secondary SjS

Characteristics	SLE (n=121)	SjS (n=131)	SLE-SjS (n=31)	*p*-value
Age at inclusion	49 (20-87)	63 (23-89)	57 (26-83)	<0.001*
	Ref	<0.001*	0.004*	
Female	105 (86.8%)	119 (90.8%)	31 (100%)	0.083
Age at onset	34 (15-75)	50 (16-85)	41.5 (18-72)	<0.001*
	Ref	<0.001*	0.224	
Disease duration	11 (1-40)	9 (1-37)	17 (3-44)	0.038*
	Ref	0.015*	0.112	
Manifestations				
Cutaneous involvement	51 (42.1%)	14 (10.7%)	16 (51.6%)	<0.001*
	Ref	<0.001*	0.003*	
Acute cutaneous lupus	46 (38.0%)	8 (6.1%)	16 (51.6%)	<0.001*
	Ref	<0.001*	<0.001*	
Subacute cutaneous lupus	2 (1.7%)	1 (0.8%)	0 (0.0%)	0.724
Chronic cutaneous lupus	4 (3.3%)	2 (1.5%)	1 (3.2%)	0.537
Oral ulcers	32 (26.4%)	8 (6.1%)	4 (12.9%)	<0.001
	Ref	<0.001*	0.689	
Nasal ulcers	5 (1.8%)	0 (0.0%)	0 (0.0%)	0.045*
	Ref	0.009*	0.424	
Sicca syndrome	10 (8.3%)	121 (92.4%)	29 (93.5%)	<0.001*
	Ref	<0.001*	<0.001*	
Alopecia	29 (24.0%)	5 (3.8%)	4 (12.9%)	<0.001
	Ref	<0.001	0.920	
Raynaud phenomenon	28 (23.1%)	25 (19.1%)	13 (41.9%)	0.026*
	Ref	0.110	0.009*	
Interstitial lung disease	1 (0.8%)	13 (9.9%)	3 (9.7%)	0.007*
	Ref	0.002*	0.009*	
Pulmonary hypertension	0 (0.0%)	1 (0.8%)	1 (3.2%)	0.207
Serositis	30 (24.8%)	3 (2.3%)	4 (12.9%)	<0.001*
	Ref	<0.001*	0.976	
Cardiac involvement	4 (3.3%)	1 (0.8%)	1 (3.2%)	0.259
Glomerulonephritis	43 (35.5%)	5 (3.8%)	9 (29.0%)	<0.001*
	Ref	<0.001*	0.190	
Interstitial nephritis	0 (0.0%)	0 (0.0%)	1 (3.3%)	0.106
Central nervous system involvement	5 (4.1%)	3 (2.3%)	1 (3.2%)	0.624
Peripheral nervous involvement	0 (0.0%)	5 (3.8%)	1 (3.2%)	0.071
Vasculitis	12 (9.9%)	11 (8.4%)	3 (9.7%)	0.912
Arthritis	97 (80.2%)	62 (47.3%)	22 (71.0%)	<0.001*
	Ref	<0.001*	0.390	
Myositis	1 (0.8%)	2 (1.5%)	0 (0.0%)	0.716
Gastrointestinal involvement	5 (4.1%)	4 (3.1%)	1 (3.2%)	0.899
Fever	23 (19.0%)	6 (4.6%)	3 (9.7%)	0.001*
	Ref	<0.001*	0.764	
Lymphadenopathy	12 (9.9%)	18 (13.7%)	4 (12.9%)	0.639
Weight loss	2 (1.7%)	3 (2.3%)	1 (3.2%)	0.718
Glandular tumefaction	0 (0.0%)	21 (16.0%)	3 (9.7%)	<0.001*
	Ref	<0.001*	0.803	
Fatigue	17 (14.0%)	16 (12.2%)	6 (19.4%)	0.580
Hemolytic anemia	21 (17.4%)	4 (3.1%)	0 (0.0%)	<0.001*
	Ref	0.001*	0.066	
Thrombocytopenia	30 (24.8%)	6 (4.6%)	5 (16.1%)	<0.001*

different immunological characteristics. More studies are needed to understand the underlying pathophysiology and biomarkers to improve the management of these patients.

259 - ACUTE PERICARDITIS AND POSITIVE ANTINUCLEAR ANTIBODIES – WHAT DIFFERENCE DOES IT MAKE?

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Background: Acute pericarditis is a common condition in the Emergency Room (ER). It is well known that several connective tissue diseases are associated with acute pericarditis and is a poor prognostic factor. We aim to assess whether the presence of antinuclear-antibodies (ANAs) in patients with acute pericarditis is associated with prognosis.
Methods: We performed a retrospective analysis of adult patients admitted to Cardiology ER in our hospital centre with the diagnosis of acute pericarditis and a request for ANA, from 2009 to 2019. ANAs levels were assessed by indirect immunofluorescence on HEp-2 (human epithelial type 2) cells, with ANA positivity defined as a titre of ≥ 1:160. ANA patterns were classified using the International Consensus on Antinuclear Antibody (ANA) Patterns (ICAP). We evaluated the duration of hospitalisation, decision of hospital follow-up, previous diagnosis of an auto-immune disorder (AID) and new AID diagnosis within 5-year follow-up. Quantitative data were expressed as either mean ± standard deviation or median and interquartile range and categorical data as frequencies and percentages. Groups were compared using the Student T-test or Mann-Whitney U test for continuous variables and Pearson's $\chi 2$ test for categorical variables.
Results: From the 192 patients admitted to the Cardiology UD with acute pericarditis, ANA was requested in 48 patients and came back positive in 25. Gender (73,3% female) and age (mean 48 years-old) were similarly distributed between the ANA positive and negative groups. In the positive ANA group (n=25), 10 had a previously known AID (3 undifferentiated connective tissue disease, 3 systemic lupus erythematosus, 1 Sjögren syndrome, 1 systemic sclerosis, 1 auto-immune hepatitis and 1 inflammatory bowel disease). In the ANA negative group (n=23), 4 patients had known AID (2 auto-immune thyroiditis, 1 rheumatoid arthritis and 1 pulmonary alveolar proteinosis).
Among patients without AID (n=34), ANA positivity (n=15) did not influence duration of hospitalization (p=0,433) or decision for hospital follow-up (p = 0,683). No new diagnosed episodes of pericarditis occurred in the subsequent 5 years in any of the patients. In the ANA-positive group one case of autoimmune hemolytic anaemia was observed within 5 years after the occurrence of pericarditis. No other new diagnosis of AID were made in this group. Surprisingly, in the ANA-negative group, one patient developed positive anti-centromere antibodies and was recently diagnosed with undifferentiated connective tissue disease, and a second one had recurring anterior uveitis.
Discussion: Positive ANA in acute pericarditis patients with no known AID do not correlate with worst outcomes at hospitalisation and 5-years follow-up. Interestingly, no new AID diagnoses were made in the ANA-positive group but one ANA-negative patient developed systemic sclerosis (and ANA positivity). These results indicate that, while ANAs are common in patients with acute pericarditis, their presence does not imply a worse prognosis, nor does their absence completely rule out the possibility of future AID.

260 - CALCIUM PYROPHOSPHATE CRYSTALS IN SYNOVIAL FLUID IN A TERTIARY HOSPITAL CENTRE – CLINICAL, BIOCHEMISTRAL AND IMAGIOLOGICAL CORRELATIONS

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Background: Calcium pyrophosphate deposition disease (CPPD) is caused by the deposition of calcium pyrophosphate dihydrate crystals (CPC) in the connective tissues, leading to acute or chronic arthritis and progression to osteoarthritis.
Methods: We requested records of all synovial fluid samples sent to our pathology laboratory from 2019 to 2023. We reviewed clinical records in search for secondary causes of CPPD and clinical and/or imagiological signs of CPPD.
Results: From a total of 1010 synovial fluid samples received in this period, search for crystals was requested in 654 (64,8%), with CPC being identified in 25 (2 from the same patient). Most patients were male (60%), aged 49 to 98 years, with mean of 77,2 years. Half the cases (52%) were observed in the Urgency Department, 28% in the Rheumatology outpatient clinic and 20% in different wards. Synovial fluid aspiration was done in the knee in all but one case (elbow). The aspiration was performed due to acute arthritis of the joint. Most patients (72%) presented with recurrent episodes of ar-

TO 260 – Table 1. Biochemistry parameters in patients with calcium pyrophosphate deposition disease

Biochemistry parameters	Availability (%)	Minimum	Maximum	Mean (±SD)	Reference values on our laboratory
Calcium (mg/dL) (N = 23)	92	8,3	9,9	9,1±0,5	8,8 - 10,6
Phosphate (mg/dL) (N = 7)	28	2,4	4,8	3,4±0,8	2,5 - 4,5
Magnesium (mg/dL) (N = 8)	32	1,6	2,2	1,9±0,21	1,9 - 2,5
Cholecalciferol (ng/mL) (N = 5)	20	7,6	26	18,1±7,1	Deficiency < 10 Insufficiency 10 -30 Sufficiency > 30
Parathormone (pg/mL) (N = 6)	24	49	335	114,7±112,6	9 - 72
Alkaline phosphatase (U/L) (N =)	84	32	637	124±138,8	30 - 120
Ferritin (ng/mL) (N = 4*)	16	63,6	378,1	180,6±136,5	30 - 300
Thyroid stimulating hormone (nUI/mL) (N = 14)	56	<0,004	3,8	1,9±1,1	0,4 - 40
Uric acid (mg/dL) (N = 13)	52	<1,0	7,7	5,1±1,8	2,6 – 6,0
C reactive protein (mg/dL) (N = 21*)	84	0,05	20,31	5,6±6,1	< 0,50
Erythrocyte sedimentation rate (mm/1st hour) (N = 10*)	40	6	64	32,7±18,6	1 - 20
Total cell count on synovial fluid (cell/ µL) (N = 21**)	84	152	135618	27075,5±32600,9	NA

N - number; NA – not applicable; SD - standard deviation; T - total. * Excluding 4 patients with simultaneous infections, * Excluding 2 patients with septic arthritis

thritis while the remainder had persistent polyarthritis. Three patients had another rheumatic disease associated (2 rheumatoid arthritis, 1 pulmonary sarcoidosis and 1 tophaceous gout). Two patients presented with simultaneous septic arthritis of the same joint. One patient had concomitant MSU crystals in the synovial fluid.

The most common biochemical disturbances found in these patients were: hypocalcemia (n=12), hypomagnesemia (n=3) and hyperparatirodism (n=2). Acute phase reactants excluding patients with concomitant infections) were elevated in most patients (Table 1). Chondrocalcinosis was observed in the knee menisci, triangular fibrocartilage complex and pubic symphysis in 17, 5 and 3 patients, respectively. Ultrasound examinations (availability 24%), had signs of chondrocalcinosis in all, except one case. Additionally, osteoarthrosis (defined as present if the Kellgren and Lawrence score is ≥2) of the knee or wrist was observed in all patients with available radiographs (76% and 36% of patients, respectively). Four patients had total knee replacement of at least one knee. Evidence of calcific tendinopathy of the rotator cuff was found in 3 cases. The diagnosis of DPPC was previously known in only 3 cases. Based on the presence of CPC in synovial fluid analysis in patients with acute arthritis of a joint, all patients met the 2023 ACR/EULAR classification criteria for CPPD. Interestingly, if the criteria were applied based solely on clinical history and imaging exams (without crystal analysis in synovial fluid), 80% of patients would still meet the classification criteria. Lastly, of the 18 patients not already in Rheumatology consultation, only 3 were referred for consult.

Discussion: Most patients where CPC was identified in the synovial fluid after arthrocentesis were middle-aged men with acute knee arthritis. The presence of another rheumatic disease and disturbances of phosphate-calcium metabolism are common. Imagiological evidence of chondrocalcinosis and osteoarthritis were prevalent, emphasising the importance of imaging in the assessment of CPPD. Indeed, most patients still met the 2023 ACR/EULAR classification criteria for CPPD even without considering the presence of CPC crystals. A thorough study of metabolic causes was rare, and occurred (with one exception) in patients followed in Rheumatology consultation. Despite these findings, referral to Rheumatology was limited, highlighting a need for increased awareness and diagnostic rigour.

261 - OSTEOPOROSIS IN CELIAC PATIENTS: INSIGHTS FROM A DESCRIPTIVE STUDY AT A PORTUGUESE CENTRE

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Introduction: Celiac disease (CD) is an autoimmune

disorder of the small intestine triggered by the ingestion of gluten in susceptible individuals. Osteoporosis (OP) is increasingly recognized as one of the most common extra-intestinal manifestations of CD, with calcium and vitamin D malabsorption, the release of inflammatory mediators, and the activation of osteoclasts being the main proposed pathophysiological mechanisms. The only effective treatment for CD is a gluten-free diet (GFD); however, even apparent control of the gastrointestinal disease does not prevent consistently low T-scores on dual-energy X-ray absorptiometry (DEXA).

Objective: To describe the characteristics of celiac patients who develop OP in a Portuguese hospital center.

Methods: We conducted a descriptive retrospective study. Patients diagnosed with CD who were subsequently diagnosed with OP and referred for a Rheumatology consultation at ULS São João were included in this study.

Results: A total of 7 patients were included in this study. Their characteristics at the time of CD diagnosis, as well as the therapeutic interventions they underwent and their T and Z-scores over a 3-year follow-up period, are summarized in Table 1. The mean age at diagnosis was 54.14 ± 3.00 years. The majority of patients were women (5/7, 71.4%), all of whom were postmenopausal. The mean BMI was 21.21 ± 0.92, and 2 patients were smokers (28.6%). Two patients had other autoimmune diseases: psoriasis and type 1 diabetes mellitus.

Diarrhea was the most common presenting symptom (n=4), followed by weight loss (n=3), oral ulcers (n=2), and nausea and vomiting (n=1), while 2 patients were asymptomatic. In terms of duodenal mucosa changes in biopsy, according to the Marsh-Oberhuber classification, only 1 patient did not present the typical changes of celiac enteropathy, while the others already showed villous atrophy (grade 3). One patient had a family history of low-energy hip fracture, and another had a personal history of a low-energy forearm fracture. At the initial DEXA, the lowest T-score and Z-score values were mainly observed at the lumbar spine (6/7, 85.7%), with mean values of -3.18 ± 0.34 and -2.08 ± 0.49, respectively.

Regarding laboratory tests, only 1 patient presented with anemia, but 100% had a vitamin D deficiency, with 2 of them having severe deficiency (<10 ng/mL). The mean calcium and alkaline phosphatase levels were 4.73 ± 0.10 mEq/L and 92.71 ± 11.58 U/L, respectively.

Five patients (71.4%) adhered to a gluten-free diet, and all were supplemented with calcium and/or vitamin D. Only 1 of the patients received pharmacological treatment for OP during the first 3 years of follow-up.

TO 261 – TABLE 1. Characteristics of patients diagnosed with celiac disease and osteoporosis

SOCIODEMOGRAPHIC DATA AT DIAGNOSIS OF CD	
Female sex - n/N (%)	5/7 (71.4)
Age (years) - mean ± SD	54.14 ± 3.00
Menopause_status, n/N (%)	5/5 (100)
BMI (kg/m^2) – mean ± SD	21.21 ± 0.92
Smoking - n/N (%)	2/7 (28.6)
Other AI diseases – n/N (%)	2/7 (28.6)
Psoriasis	1
Diabetes mellitus type 1	1
CLINICAL DATA	
Celiac disease	
Presenting symptoms – n/N (%)	
Diarrhea	4/7 (57.1)
Weight loss	3/7 (42.9)
Oral ulcers	2/7 (28.6)
Nausea, vomiting	1/7 (14.3)
Asymptomatic	2/7 (28.6)
Biopsy results (MMO classification) – n/N (%)	
0	1/7 (14.3)
3	6/7 (85.7)
Osteoporosis	
Family history of low energy fractures – n/N (%)	1/7 (14.3)
Personal history of low energy fractures – n/N (%)	1/7 (14.3)
Baseline DEXA results – mean ± SD	
Lowest T-score	-3.18 ± 0.34
Lowest Z-score	-2.08 ± 0.49
Location of lowest T/Z score – n/N (%)	
Lumbar spine	6/7 (85.7)
Femur	1/7 (14.3)
BASELINE LABORATORY TESTS	
Anemia – n/N (%)	1/7 (14.3)
25-hydroxyvitamin D deficiency	7/7 (100%)
Severe 25-hydroxyvitamin D deficiency	2/7 (28.6)
Total calcium (mEq/L) – mean ± SD	4.73 ± 0.10
Alkaline phosphatase (U/L) – mean ± SD	92.71 ± 11.58
INTERVENTION	
GFD adherence	5/7 (71.4)
Suplementation – n/N (%)	
Calcium	1/7 (14.3)
Vitamin D	1/7 (14.3)
Calcium + vitamin D	5/7 (71.4)
Pharmacological treatment	
IV zoledronate - n/N (%)	1/7 (14.3)
EVOLUTION	
Clinical improvement in CD, n/N (%)	5/5 (100)
Histologic remission, n/N (%)	3/4 (75)
3-year follow-up DEXA – mean ± SD	
T-score variation	+0.50 ± 0.25
Z-score variation	+0.53 ± 0.22
New fractures during follow-up	0/7 (0)

CD- celiac disease; SD- standard deviation; IQR- interquartile range; BMI- body mass index; AI- autoimmune; MMO- modified Marsh-Oberhuber; DEXA- Dual-energy X-ray absorptiometry; GFD- gluten-free diet.

Clinical improvement occurred in all symptomatic patients, and 3 of them achieved histological remission. The change in T and Z-scores at 3 years was generally positive, with a mean value of +0.50 ± 0.25 and +0.53 ± 0.22, respectively.

Conclusion: Given the significant prevalence of OP in celiac patients, it is essential for clinicians to always consider early screening with DEXA, regardless of the presence of other risk factors and the clinical severity or apparent control of CD. Even in the absence of nutritional deficiencies or laboratory changes, there is

sufficient evidence to conclude that these patients remain at risk. On the other hand, it is a diagnosis that rheumatologists must keep in mind, especially in the presence of severe OP, low BMI, and low vitamin D levels. This study results highlight the importance of multidisciplinary approaches to achieve maximum treatment efficacy.

263 - TREAT-TO-TARGET ATTAINMENT IN PATIENTS WITH SLE FROM THE REUMA.PT SLE REGISTRY: A MULTICENTER CROSS-SECTIONAL STUDY OF 795 PATIENTS

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Background: Treat-to-target (T2T) aiming for remission, or at least low disease activity (LDA), while tapering prednisone to ≤5mg/day is established as a pillar for management of systemic lupus erythematosus (SLE) in the 2023 updated EULAR recommendations for clinical practice.

Objective: To investigate the attainment of the T2T goal in the SLE population from the Rheumatic Diseases Portuguese Registry (Reuma.pt) and identify unmet treatment needs.

Methods: We performed a cross-sectional multicenter study of patients fulfilling the EULAR/ACR 2019 classification criteria for SLE in the Reuma.pt. The assessment was performed at the last visit occurring between June 2023 and March 2024. Disease activity and attainment of T2T were assessed with the SLE Disease Activity Score (SLE-DAS).1 Patients with no SLE-DAS scoring were excluded. Patients were classified as in remission (clinical items of SLE-DAS =0 and prednisone ≤5 mg/day), low disease activity (LDA) (SLE-DAS ≤2.48 and prednisone ≤5 mg/day) and, for those not attaining T2T, in categories of disease activity (mild: 2.08< SLE-DAS ≤7.64; moderate: 7.64< SLE-DAS≤ 9.90; severe: SLE-DAS >9.90). Descriptive analysis was performed, and a comparison between the remission vs. non-remission and LDA vs. non-LDA groups was performed using Student's t-test, Fisher's exact test, Chi-square or Mann-Whitney U tests, as appropriate. IBM SPSS Statistics software version 27 was used. p≤0.05 was considered statistically significant.

Results: There were 2459 patients with SLE from 37 centers registered in Reuma.pt. Of these, 1664 (67.7%) were excluded due to: missing data on fulfilment (n=1257, 51.1%) or no fulfilment (n=83, 3.4%) of EULAR/ACR classification criteria; missing data for SLE-DAS (n=324, 13.2%). A total of 795 patients from 18 participating centers were included [89.6% female; mean age 49.9 (SD 14.2) years] (table 1). Main cumulative SLE clinical features included: mucocutaneous (72.5%), hematological (66.7%), arthritis (63.6%), nephritis (38.9%), serositis (19.4%) and neuropsychiatric lupus (6.8%).

Remission was attained by 71.3% and an additional 3.0% were in LDA, meaning that the EULAR treatment target was achieved by 74.3%. The remaining patients presented active disease (mild 20%, moderate 2.6%, severe 3%). Hydroxychloroquine (HCQ) was prescribed to 88.3% of all patients. Patients not in LDA were more frequently treated with synthetic (69.1% vs 39.6%, p<0.001) and biologic (19.1% vs 8.5%, p<0.001) immunosuppressants. Of the patients with active SLE, 40.2% were receiving >7.5 mg/day of prednisolone-equivalent dose and 72.7% were treated with synthetic and/or biologic immunosuppressants.

Conclusions: This subgroup of patients with SLE registered in Reuma.pt present a high rate of attainment of the T2T, and most are treated with HCQ, in line with the EULAR recommendations. The high number of patients excluded preclude the generalization of these conclusions. This provides benchmarking indicators demonstrating high quality of care. Furthermore, un-

TO 263 – TABLE 1. Characteristics of the patients included (n=795)

	Total (n=795)	Remission (n=567)	Non-remission (n=228)	p value	LDA (n=591)	Non-LDA (n=204)	p value
Demographic characteristics							
Female, n (%)	712/795 (89.6)	508/567 (89.6)	204/228 (89.5)	0.960	531/591 (89.8)	181/204 (88.7)	0.651
European, n (%)	645/720 (89.6)	476/519 (91.7)	169/201 (84.1)	**0.003**	496/540 (91.9)	149/180 (82.8)	**<0.001**
Age at symptom onset (years), mean±SD (n)	31.1±13.1 (713)	31.6±13.1 (509)	29.8±12.9 (204)	0.098	31.6±13.2 (532)	29.8±12.8 (181)	0.101
Age at diagnosis (years), mean±SD (n)	33.3±13.7 (721)	33.9±13.6 (511)	31.9±13.8 (210)	0.061	33.9±13.6 (534)	31.7±13.8 (187)	**0.047**
Age at study visit (years), mean±SD (n)	49.9±14.2 (795)	50.7±14.2 (567)	47.7±14.1 (228)	**0.006**	50.7±14.2 (591)	47.3±14.2 (204)	**0.003**
Disease duration (years), mean±SD (n)	16.7±10.0 (721)	17.1±10.1 (511)	15.7±9.5 (210)	0.145	17.1±10.0 (534)	15.5±9.8 (187)	0.095
Cumulative SLE clinical features							
Mucocutaneous, n (%)	569/785 (72.5)	382/561 (68.1)	187/224 (83.5)	**<0.001**	397/584 (68.0)	172/201 (85.6)	**<0.001**
Arthritis, n (%)	497/782 (63.6)	343/559 (61.4)	154/223 (69.1)	**0.043**	353/583 (60.5)	144/199 (72.4)	**0.003**
Serositis, n (%)	151/778 (19.4)	104/556 (18.7)	47/222 (21.1)	0.432	108/580 (18.6)	43/198 (21.7)	0.341
Renal, n (%)	299/768 (38.9)	203/552 (36.8)	96/216 (44.4)	0.050 (ns)	208/574 (36.2)	91/194 (46.9)	**0.008**
Neuropsychiatric, n (%)	52/760 (6.8)	34/549 (6.2)	18/211 (8.5)	0.253	39/573 (6.8)	13/187 (7.0)	0.945
Hematological, n (%)	518/777 (66.7)	358/556 (64.4)	160/221 (72.4)	**0.033**	378/579 (65.3)	140/198 (70.7)	0.162
Cumulative SLE immunological features							
ANA, n (%)	795/795 (100)	567/567 (100.0)	228/228 (100.0)	-	204/204 (100)	591/591 (100)	-
Anti-dsDNA, n (%)	677/782 (86.6)	475/559 (85.0)	202/223 (90.6)	**0.038**	497/582 (85.4)	180/200 (90.0)	0.099
Anti-Sm, n (%)	44/419 (10.5)	35/326 (10.7)	9/93 (9.7)	0.769	35/339 (10.3)	9/80 (11.3)	0.808
aPL, n (%)	305/685 (44.5)	218/503 (43.3)	87/182 (47.8)	0.299	233/525 (44.4)	72/160 (45.0)	0.890
Low C3/C4, n (%)	617/771 (80.0)	430/552 (77.9)	187/219 (85.4)	**0.019**	450/575 (78.3)	167/196 (85.2)	**0.036**
Disease activity scores							
SLE-DAS, median (range) (n)	1.12 (0.37-29.51) (795)	0.37 (0.37-2.08) (567)	2.99 (0.37-29.51) (228)	-	0.37 (0.37-2.47) (591)	3.50 (0.37-29.51) (204)	-
SLEDAI-2K, median (range) (n)	2 (0-20) (782)	0 (0-8) (559)	3 (0-20) (223)	**<0.001**	0 (0-8) (583)	4 (0-20) (199)	**<0.001**
PGA, median (range) (n)	0.1 (0.0-2.5) (670)	0.1 (0.0-1.0) (490)	0.5 (0.0-2.5) (180)	**<0.001**	0.1 (0.0-1.5) (508)	0.6 (0.0-2.5) (162)	**<0.001**
SLE-DAS disease activity category							
Remission, n (%)	567/795 (71.3)	567/567 (100.0)	0/228 (0.0)	-	567/591 (95.9)	0 (0)	-
Low disease activity, n (%)	591/795 (74.3)	567/567 (100.0)	24/228 (10.5)	-	591/591 (100.0)	0 (0)	-
Mild, n (%)	159/795 (20.0)	0/567 (0.0)	159/228 (69.7)	-	0 (0.0)	159/204 (77.9)	-
Moderate, n (%)	21/795 (2.6)	0/567 (0.0)	21/228 (9.2)	-	0 (0.0)	21/204 (10.3)	-
Severe, n (%)	24/795 (3.0)	0/567 (0.0)	24/228 (10.5)	-	0 (0.0)	24/204 (11.8)	-
Damage							
SDI score ≥1, n (%)	286/772 (37.0)	201/554 (36.3)	85/218 (39.0)	0.483	210/576 (36.5)	76/196 (38.8)	0.562
Ongoing SLE treatment							
Hydroxychloroquine, n (%)	693/785 (88.3)	510/567 (89.9)	183/218 (83.9)	**0.019**	528/591 (89.3)	165/194 (85.1)	0.107
Synthetic immunosuppressants, n (%)	368/785 (46.9)	219/567 (38.6)	149/218 (68.3)	**<0.001**	234/591 (39.6)	134/194 (69.1)	**<0.001**
Mycophenolate mofetil or Mycophenolic acid, n (%)	136/785 (17.3)	82/567 (14.5)	54/218 (24.8)	**<0.001**	85/591 (14.4)	51/194 (26.3)	**<0.001**
Azathioprine, n (%)	129/785 (16.4)	73/567 (12.9)	56/218 (25.7)	**<0.001**	84/591 (14.2)	45/194 (23.2)	**0.003**
Methotrexate, n (%)	96/785 (12.2)	59/567 (10.4)	37/218 (17.0)	**0.012**	61/591 (10.3)	35/194 (18.0)	**0.004**
Cyclosporine, n (%)	11/785 (1.4)	7/567 (1.2)	4/218 (1.8)	0.522	7/591 (1.2)	4/194 (2.1)	0.479
Tacrolimus, n (%)	8/785 (1.0)	5/567 (0.9)	3/218 (1.4)	0.692	5/591 (0.8)	3/194 (1.5)	0.416
Leflunomide, n (%)	6/785 (0.8)	4/567 (0.7)	2/218 (0.9)	0.672	4/591 (0.7)	2/194 (1.0)	0.641
Intravenous immunoglobulin, n (%)	3/785 (0.4)	1/567 (0.2)	2/218 (0.9)	0.188	1/591 (0.2)	2/194 (1.0)	0.153
Cyclophosphamide, n (%)	2/785 (0.3)	0/567 (0.0)	2/218 (0.9)	0.077	0/591 (0.0)	2/194 (1.0)	0.061
Dapsone, n (%)	1/785 (0.1)	0/567 (0.0)	1/218 (0.5)	0.278	0/591 (0.0)	1/194 (0.5)	0.247
Biologics, n (%)	87/785 (11.1)	49/567 (8.6)	38/218 (17.4)	**<0.001**	50/591 (8.5)	37/194 (19.1)	**<0.001**
Belimumab, n (%)	52/785 (6.6)	33/567 (5.8)	19/218 (8.7)	0.144	33/591 (5.6)	19/194 (9.8)	**0.041**
Rituximab*, n (%)	33/785 (4.2)	16/567 (2.8)	17/218 (7.8)	**0.002**	17/591 (2.9)	16/194 (8.2)	**0.001**
Anifrolumab, n (%)	3/785 (0.4)	0/567 (0.0)	3/218 (1.4)	**0.021**	0/591 (0.0)	3/194 (1.5)	**0.015**
Synthetic immunosuppressants and/or biologics, n (%)	391/785 (49.8)	235/567 (41.4)	156/218 (71.6)	**<0.001**	250/591 (42.3)	141/194 (72.7)	**<0.001**
Oral corticosteroids, n (%)	390/785 (49.7)	217/567 (38.3)	173/218 (79.4)	**<0.001**	229/591 (38.7)	161/194 (83.0)	**<0.001**
Dose of PDN (mg/day), mean±SD (n)	3.5 ±6.5 (785)	1.6±2.1 (567)	8.6±10.3 (218)	**<0.001**	1.6±2.1 (591)	9.4±10.6 (194)	**<0.001**
Dose >0 and ≤5 mg/day of PDN, n (%)	280/785 (35.7)	217/567 (38.3)	63/218 (28.9)	**0.014**	229/591 (38.7)	51/194 (26.3)	**0.002**
Dose >5 and ≤7.5 mg/day of PDN, n (%)	32/785 (4.1)	0/567 (0.0)	32/218 (14.7)	-	0/591 (0.0)	32/194 (16.5)	-
Dose >7.5 mg/day of PDN, n (%)	78/785 (9.9)	0/567 (0.0)	78/218 (35.8)	-	0/591 (0.0)	78/194 (40.2)	-

*Ongoing or in the last 6 months.
ANA, antinuclear antibodies; Anti-dsDNA, anti-double stranded DNA antibodies; aPL, antiphospholipid antibodies; Anti-Sm, anti-Smith antibodies; LDA, low disease activity; Low C3/C4, low serum levels of complement fractions C3 and/or C4; PDN, prednisolone-equivalent; PGA, physician global assessment; SDI, Systemic Lupus International Collaborating Clinics/American College of Rheumatology Damage Index; SLEDAI-2K, Systemic Lupus Erythematosus Disease Activity Index 2000; SLE-DAS, Systemic Lupus Erythematosus Disease Activity Score.

met needs for new and improved therapies are demonstrated, with over a quarter of patients presenting active disease despite more intensive standard-of-care. Longitudinal studies are needed to assess if T2T goals are sustained over time. Optimized treatment regimens are needed to attain recommended T2T goals in many SLE patients. Improvement of registry in Reuma.pt is indispensable for a representative national appraisal.

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273 - LUNG DISEASE AS THE FIRST GLIMPSE OF AN INFLAMMATORY MYOPATHY: A CASE SERIES

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Introduction: Idiopathic inflammatory myopathies (IIM) typically present with proximal muscle weakness, but interstitial lung disease (ILD) can be a prominent initial manifestation. We present five cases highlighting ILD as the primary involvement in IIM.

Case 1: A 48-year-old female presented with progressive dyspnea, cough and fatigue. Imaging revealed NSIP (Nonspecific interstitial pneumonia) and PFTs (pulmonary function tests) showed a restrictive pattern, with greatly decreased DLCO. After initial corticosteroid treatment symptoms progressed and, 3 months later, she developed myalgia and elevated muscle enzymes. Anti-Jo-1 antibodies were positive and a muscle biopsy confirmed myositis. She improved with high-dose corticosteroids and azathioprine.

Case 2: A 53-year-old male smoker presented with cough, arthralgia and edema. He had bibasilar crackles and mild arthritis, but no muscle weakness. Elevated muscle enzymes and a positive anti-SSA52 antibody were found. Imaging revealed UIP (Usual interstitial pneumonia) and PFTs showed a restrictive pattern, with greatly decreased DLCO. Despite normal EMG (eletromyogram) and ultrasound, a muscle biopsy confirmed IIM. Corticosteroids and mycophenolate mofetil were effective.

Case 3: A 66-year-old female with fibromyalgia presented with dyspnea, cough, asthenia and night sweats. Examination revealed bibasilar crackles. A NSIP pattern was confirmed, PFTs showed a restrictive pattern, with moderately decreased DLCO and muscle enzymes were elevated. Anti-PL7 and anti-SSA52 antibodies were positive. Despite no muscle weakness, EMG showed myopathic changes. Treatment with corticosteroids, mycophenolate mofetil and rituximab led to clinical stability.

Case 4: A 38-year-old male presented with acute respiratory failure and arthralgias. Imaging revealed NSIP and PFTs showed a restrictive pattern, with greatly decreased DLCO. He had elevated creatine kinase and positive anti-Jo-1 and anti-SSA52 antibodies. Antisynthetase syndrome was diagnosed. Initial treatment with corticosteroids and cyclophosphamide, followed by mycophenolate mofetil, resulted in stable disease.

Case 5: A 53-year-old female with known pulmonary

fibrosis and restrictive PFTs developed polyarthralgias, Raynaud's phenomenon and fatigue. Although muscle enzymes were normal, strong positive anti-PL12 and anti-SSA52 antibodies suggested antisynthetase syndrome. Corticosteroids and cyclophosphamide had limited effect and she is being evaluated for lung transplantation.
Conclusion: While not typical, ILD can be the initial presentation of IIM in 13-37.5% of cases. In patients with ILD, especially with elevated muscle enzymes or myositis-specific antibodies, prompt investigation for myopathy is warranted. Moreover, almost all the presented patients had not only myositis-specific antibodies but also myositis-associated antibodies, like anti-SSA52, which, as described in the literature, seems to predispose to a worse prognosis, with more severe ILD.

277 - HOW UNIQUE IS SERONEGATIVE RHEUMATOID ARTHRITIS?

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Introduction: Rheumatoid arthritis (RA) is a chronic systemic immune-mediated disease with differences based on seropositivity for Rheumatoid Factor (RF) and Anti-cyclic citrullinated peptide antibodies (ACCP). Seronegative RA lacks a consistent phenotype and can be challenging to define, often resulting in the reclassification of patients with other rheumatic diseases.
Objectives: This project aims to explore the clinical, laboratory and radiographic presentation of patients with seronegative and seropositive RA at an academic hospital centre and to investigate characteristics that could be framed in other rheumatic pathologies.
Methods: Data on the participants was collected and registered into Reuma.pt, between July 2022 and January 2023. Patients were selected through convenience sampling based on their attendance at designated RA appointments. Additional clinical and imaging data was collected using a questionnaire (attachment 1) which assessed various variables typical of rheumatic diseases that make differential diagnosis with RA. Continuous variables were compared using the t-student or Mann-Whitney tests (depending on the distribution)

TO 277 – Table 1. Rheumatoid arthritis patient questionnaire

Spondylarthritis
1. do you have inflammatory spinal pain or pain in the gluteal region when you sit down? - Low back pain with onset before the age of 40, insidious, which improves with exercise and does not improve with rest, or night pain which improves when you get up; - Gluteal pain with an alternating inflammatory rhythm.
2. Enthesitis: pain in the heel or Achilles tendon when you wake up?
3. Have you ever had dactylitis / "sausage fingers"?
4. Personal or family history of psoriasis or inflammatory bowel disease?
5. Personal history of uveitis: sudden onset of red eye, pain, photophobia and blurred vision?
Diffuse connective tissue diseases
1. Have you ever had skin manifestations such as malar rash?
2. Photosensitivity: do skin complaints appear with sun exposure? If so, where?
3. Do you frequently get oral ulcers (multiple ulcers and > 1 x/month)?
4. Have you had hair loss?
5. Have you ever had Raynaud's phenomenon?
6. Have you ever noticed digital ulcers that appear spontaneously and take a long time to heal?
7. Do you have dysphagia or heartburn?
8. Have you noticed your eyes, skin or mouth becoming drier? What about the vaginal mucosa?
9. Has there been progressive proximal muscle weakness without pain: when climbing stairs, combing or bathing?

and categorical variables using the chi-square. Statistical significance was considered for p-values < 0.05.
Results: 60 patients were included in the study, comprising 30 with seropositive RA and 30 with seronegative RA. There were no significant differences in mean age (64.33 ± 14.17 years for seronegative RA vs. 60.83 ± 14.3 years for seropositive RA, p = 0.3532) or gender distribution (90% females in the seronegative group vs. 97% females in the seropositive group, p = 0.3006). Seronegative RA patients had significantly longer disease duration (19.43 ± 12.25 years) compared to those with seropositive RA (10.73 ± 7.76 years, p=0.0089).
In seronegative RA patients, the most common manifestations were sicca symptoms (57%) and bone erosions of the hand (50%) whose rates were comparable to those observed in seropositive RA (sicca symptoms: 60%, p = 0.7934; bone erosions: 47%, p = 0.7961).
Patients with seronegative RA and bone erosions of the hands have a longer average disease duration compared to patients with the same clinical manifestations and seropositive RA (seronegative RA: 24.47 ± 14.08 years; seropositive RA: 14.93 ± 7.26 years; p = 0.0363).
Enthesitis (40%) and chondrocalcinosis of the wrist (20%) were more common in seronegative RA, compared to seropositive RA, where enthesitis was present in 17% (p = 0.0449) and chondrocalcinosis in 3.3% (p = 0.0443).
There is no statistically significant age difference between seropositive and seronegative RA patients with enthesitis (seronegative: 61.58 ± 13.79 years; seropositive: 54 ± 13.27 years; p-value = 0.1711).
Conclusion: The comparable prevalence of erosive disease between seronegative and seropositive RA in this sample may be attributed to disease duration. Further investigation with more homogeneous groups of patients is necessary to ascertain whether there may exist additional prognostic factors, in addition to RF and ACPA, that contribute to greater aggressiveness and erosive disease. Enthesitis and chondrocalcinosis of the wrist are more prevalent in patients with seronegative RA, suggesting other possible etiologies, while other studied variables were not significantly different between seropositive and seronegative RA patients, which might be attributable to differences in disease duration or lack of power due to a small sample size.

279 - DOES THE AGREEMENT BETWEEN ANTINUCLEAR ANTIBODIES INDIRECT IMMUNOFLUORESCENCE PATTERNS AND MYOSITIS ANTIBODIES CORRELATE WITH THE FULFILMENT OF IDIOPATHIC INFLAMMATORY MYOPATHIES CLASSIFICATION CRITERIA? – A MULTICENTRIC, PILOT STUDY

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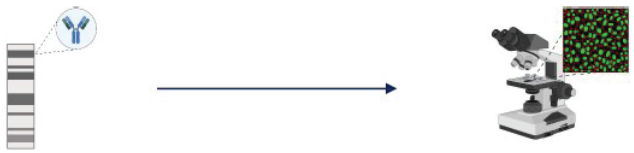
Introduction: Idiopathic inflammatory myopathies (IIM) are a heterogeneous group of systemic autoimmune disorders in which chronic skeletal muscle inflammation leads to muscle weakness[1]. Although helpful for establishing the diagnosis of IIM in the appropriate clinical setting, myositis antibodies (MAs) don't always predict the occurrence of connective tissue diseases[1]. Commonly used techniques such as line blot are known to have high rates of false positivity, especially for rare MAs[2]. The accuracy of MAs tests such as line blot may be improved by cross-checking its results with antinuclear antibodies (ANA) patterns on HEp-2 indirect immunofluorescence (IIF)[3].
Objective: To determine whether the agreement between MAs and ANA IIF is associated with a higher rate of 2017 European League Against Rheumatism/American College of Rheumatology classification criteria fulfilment for adult IIM.
Methods: In this cross-sectional, multicentric study, adult patients with a clinical diagnosis of IIM were enrolled. Sociodemographic and clinical data were collected from the patient's medical records. Patients were divided into three groups: total (detected MAs entirely matching the complete description of a compatible ANA IIF pattern), partial (detected MAs matching the broad description of a compatible ANA IIF pattern), or no agreement between IIF pattern and MAs – figure 1. One-way ANOVA and Chi-Square test were performed to assess differences between groups, with a statistically significant p<0.05. Logistic regression analysis was performed to ascertain the effects of the agreement between IIF and MAs on the likelihood of fulfilment of IIM classification criteria. Sensitivity analysis was performed, using different combinations of possible confounders in the multivariate analysis.
Results: We included 86 patients, 74.4% females, with

TO 279 – TABLE 1. Clinical and sociodemographic characteristics of IIM patients, divided according to total, partial or no agreement between antinuclear antibodies immunofluorescence patterns and myositis antibodies.

	Total Agreement (n=33; 38.4%)	Partial Agreement (n=23; 26.7%)	No Agreement (n=30; 34.9%)	p
Female, n (%)	24 (72.7)	16 (69.6)	24 (80.0)	0.662
Age at diagnosis, years (SD)	54.0 (17.6)	47.6 (14.7)	56.7 (15.7)	0.244
Form of IIM, n (%)				0.674
- Dermatomyositis, n (%)	18 (54.5)	11 (47.8)	11 (36.7)	
- Amyopatic dermatomyositis, n (%)	2 (6.1)	2 (8.7)	6 (20.0)	
- Inclusion bodies myositis, n (%)	1 (3)	0 (0)	0 (0)	
- Nonspecific myositis, n (%)	2 (6.1)	2 (8.7)	3 (10.0)	
- Immune mediated necrotizing myositis, n (%)	3 (9.1)	4 (17.4)	5 (16.7)	
- Polymyositis, n (%)	4 (12.1)	4 (17.4)	3 (10.0)	
- Not specified, n (%)	3 (9.1)	0 (0)	2 (6.7)	
Fulfilment of 2017 EULAR/ACR classification criteria for adult IIM, n (%)	22 (66.7)	18 (78.3)	17 (56.7)	0.257
ANA high titer (1/1280 or more), n (%)	30 (90.9)	6 (26.1)	2 (6.7)	0.080

N: number of patients. SD: standard deviation. IIM: Idiopathic inflammatory myopathies. EULAR/ACR: European League Against Rheumatism / American College of Rheumatology. ANA: antinuclear antibodies.



Myositis Antibodies	ANA IIF Code	ANA IIF Staining Pattern – Complete Description	ANA IIF Staining Pattern – Broad Description
Anti-SRP	AC-19	Cytoplasmic dense fine speckled	Cytoplasmic
Anti-HMGCR	AC-0	Difficult to recognize Negative	Negative
Anti-Mi2	AC-4	Nuclear fine speckled	Nuclear speckled
Anti-MDA5	AC-0 AC-19 AC-20	Inconstant: Negative Cytoplasmic speckled Cytoplasmic dense fine speckled	Cytoplasmic
Anti-TIF1γ	AC-4	Nuclear fine speckled	Nuclear speckled
Anti-NPX2	AC-4, AC-6	Nuclear fine speckled and/or multiple nuclear dots	Nuclear speckled (or multiple nuclear dots)
Anti-SAE	AC-4 AC-5	Nuclear fine speckled Nuclear coarse speckled	Nuclear speckled
Anti-CN1A	-	Undefined	-
Anti-PL-7	AC-19	Cytoplasmic dense fine speckled	Cytoplasmic
Anti-PL-12	AC-19	Cytoplasmic dense fine speckled	Cytoplasmic
Anti-EJ	AC-19 AC-20	Cytoplasmic speckled	Cytoplasmic
Anti-OJ	AC-19 AC-20	Cytoplasmic speckled	Cytoplasmic
Anti-Jo-1	AC-20	Cytoplasmic fine speckled	Cytoplasmic
Anti-U1RNP	AC-5	Nuclear coarse speckled	Nuclear speckled
Anti-Ku	AC-4	Nuclear fine speckled	Nuclear speckled
Anti-Ro52	AC-0 AC-4 AC-19 AC-20	Negative Nuclear fine speckled Cytoplasmic speckled	Nuclear speckled (or cytoplasmic)
Anti-PM/Scl	AC-8	Nucleolar homogeneous	Nucleolar
Anti-mitochondrial antibody	AC-21	Cytoplasmic reticular/AMA	Cytoplasmic

TO 279 – Figure 1. Myositis autoantibodies and compatible antinuclear antibodies immunofluorescence staining patter. ANA: antinuclear antibodies. IIF: indirect immunofluorescence

a mean age of 59.1±17.6 years (Table 1). From these, 33 patients (38.4%) had total, 23 (26.7%), partial, and 30 (34.9%) no agreement between IIF pattern and MAs. The IIM classification criteria were fulfilled in 57 patients (66.3%). We found no statistically significant differences between groups concerning the rate of fulfilment of the IIM classification criteria, even after adjusting for confounders. Additionally, no differences between groups were found concerning the type of IIM, age at diagnosis, gender distribution, or ANA titers.

Conclusion: In our cohort of IIM patients, there was no association between the agreement of IIF patterns and MAs and the fulfilment of IIM classification criteria. These results may be due to the highly selected population used (all patients had IIM confirmed by a Rheumatologist, two-thirds fulfilling classification criteria), and to the dimension of our cohort. We plan to expand this project to all Portuguese Rheumatology Departments willing to participate and to further analyse the impact of the agreement between MAs and IIF patterns in patients with IIM or a suspected IIM.

REFERENCES:

1. Dourado, E., et al., Idiopathic inflammatory myopathies: one year in review 2022. Clin Exp Rheumatol, 2023. 41(2): p.199-213.
2. Loganathan, A., et al. Agreement between local and central anti-synthetase antibodies detection: results from the Classification Criteria of Anti-Synthetase Syndrome project biobank. Clin Exp Rheumatol, 2024. 42(2):p. 277-287.
3. He, J., et al. Concordance between myositis autoantibodies and anti-nuclear antibody patterns in a real-world, Australian cohort. Rheumatology, 2022