COMUNICAÇÕES ORAIS

Comunicações Orais

010 - IMMUNE RESPONSES TO VACCINATION AGAINST HERPES ZOSTER IN PATIENTS WITH RHEUMATIC DISEASES UNDER TREATMENT WITH JAK-INHIBITORS: OUR PRELIMINARY RESULTS

Cristiana Sieiro Santos[1], Alejandra López[1], Carolina Álvarez[1], Sara Calleja[2], Clara Moriano[1], Elvira Díez[1], Jose Ruiz de Morales[2]

[1]Reumatología, Complejo Asistencial Universitario de León, León, Spain, [2]Immunology, Complejo Asistencial Universitario de León, León, Spain

Background: Shingrix is a recombinant inactive vaccine available for prevention of herpes zoster (HZ) infection, recently approved for patients with rheumatic diseases under treatment with JAK-inhibitors. Because of its novelty, scant information is available on Shingrix vaccine responses in patients with immune mediated rheumatic diseases (IMRD).

Objectives: To investigate the antibody responses to the HZ vaccine in selected rheumatic diseases treated with JAK-inhibitors (JAKi) pre- and post-Shingrix vaccination (weeks 4–6) and to identify factors associated with reduced immunogenicity.

Methods: Patients with selected rheumatic diseases under treatment with JAKi underwent two series of IM Shingrix vaccine (0.5mL each) administered 2 months apart. Bloodwork was performed 4-8 weeks following 2nd dose of vaccine to assess post- vaccination antibody response. Pre and post vaccination response was then compared using Anti Varicella Zoster Virus IgG Multiplex Flow Immunoassay (MFI). Blood lymphocyte distributions (CD3+, CD4+, CD8+, CD19+) and NK cells, total serum IgG and IgM levels, and VZV-IgG and IgM, were investigated pre- and post-Shingrix vaccination (weeks 4–6).

Results: 38 patients were included, 79% were female with a median age at inclusion of 57±10.5 years. 21% of patients had rheumatoid arthritis, 8% had systemic lupus erythematosus, 16% had psoriatic arthritis, 16% had ankylosing spondylitis and 5% had dermatomyositis. VHZ IgG antibody levels and distributions of lymphocyte subpopulations in peripheral blood pre- and post-immunization are represented in table 1. Positive humoral responses were observed in 86% of patients. Mean changes VHZ-IgG antibody between post- and pre-vaccination sera were 4.6±0.33 and 1.8±1.98 ($p<0.0001$), respectively. After immunization, the number of T cells (CD3+, CD4+, CD8+) and NK cells remained relatively unchanged however, the number of B cells (CD19+) increased significantly from 147.4±156 to 780.62±475.5 significantly ($p<0.0001$). There was a 2.5-fold increase in antibody titers after immunization in RA patients, a 2.4-fold increase in SLE patients, a 2-fold increase in AP patients, a 2.5-fold increase in AS patients and a 3.5-fold increase in DM, with no significant difference between the different patients' subgroups. Results from multivariate regression analysis adjusted by age, gender, disease duration, treatments and glucocorticoid and MTX cumulative dose, showed a negative correlation between VHZ-IgG and age >65 years (β=-0.34, p 0.03), disease duration (β=-0.58, p 0.04) and glucocorticoid cumulative dose (β=-0.42, p 0.02).

Conclusions: Our preliminary results show preserved seroconversion rates and VHZ-IgG antibodies and concurrently increase the number of CD19+ B-cells

TABLE 1. Varicella zoster virus IgG Ab levels and distributions of lymphocyte subpopulations in peripheral blood pre- and post-immunization

	Pre-vaccination	Post-vaccination	P value
Total serum IgG (mg/dL)	958±300	1328.5±308	0.04
Total serum IgM (mg/dL)	126±98	118.5±156	0.84
VZV IgG Ab (Ab Index)	1.8±1.98	4.6±0.33	0.0001
VZV IgM Ab (Ab Index)	0.32±2.1	1.3±3.4	0.72
CD3 (cells/mm3)	1065.5±716.5	1128±721	0.63
CD19 (cells/mm3)	147.4±156	780.62±475.5	0.0001
CD4 (cells/mm3)	653±584	783±475.5	0.34
CD8 (cells/mm3)	386.2±260	392.7±297.5	0.42
NK (cells/mm3)	281.05±189	284.52±145.5	0.42

and total serum IgG in patients with rheumatic diseases on treatment with JAKi. Age>65 years, disease duration and glucocorticoid cumulative dose are negatively correlated with humoral response.

049 - AXILLARY ARTERIES ULTRASOUND IN THE DIAGNOSIS OF GIANT CELL ARTERITIS WITH PREDOMINANTLY CRANIAL SYMPTOMS

Martins-Martinho J [1, 2], Matilde Bandeira[1, 2], Lija James[3], Ayna Verdiyeva[3], Tomás Fontes[1, 2, 4], Ana Rita Lopes[1, 2], Nikita Khmelinskii[1, 2], Raashid Luqmani[3], Cristina Ponte[1, 2]
[1]Serviço de Reumatologia, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [2]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Nuffield Department of Orthopaedics, Rheumatology and Musculoskeletal Sciences, University of Oxford, Oxford, United Kingdom, [4]Rheumatology Department, Hospital do Divino Espírito Santo, Ponta Delgada, Portugal

Background: Giant cell arteritis (GCA) is the most common form of primary systemic vasculitis in patients aged >50 years. It predominantly affects the cranial arteries; however, extra-cranial disease involving the aorta and its major branches can also be present. According to the 2018 EULAR recommendations, the ultrasound of temporal arteries (TAs), with or without axillary arteries (AXs), should be the first imaging modality performed in patients with suspected predominantly cranial GCA. This recommendation reflects that the value of the AXs ultrasound in GCA diagnosis is still questionable, especially in patients who present with cranial symptoms of the disease. This study aims to assess the value of AXs ultrasound in the ultrasonographic diagnosis of GCA, particularly in patients with predominantly cranial symptoms of GCA.
Methods: Observational retrospective study involving the Rheumatology Departments of Centro Hospitalar Universitário Lisboa Norte (CHULN), Lisbon-Portugal, and the Nuffield Orthopaedic Centre (NOC), Oxford-UK. We included patients with a clinical diagnosis of GCA and a positive ultrasound for GCA at diagnosis, i.e., the presence of a non-compressible halo sign in the TAs or AXs. Information regarding the demographics, clinical manifestations at disease presentation, and therapeutics were collected. Cranial symptoms included headache, temporary or permanent loss of vision, diplopia, blurred vision, jaw or tongue claudication, scalp tenderness or paraesthesia, stroke, or transient ischaemic attack (TIA).
Results: We included 230 patients, 139 (60.4%) from the NOC and 91 (39.6%) from CHULN. One hundred and thirty-six (59.1%) patients were females, and the mean ± standard deviation age at diagnosis was 75.3 ± 8.5 years. The ultrasound was performed after ten days of treatment with prednisolone ≥30mg/day in 70/230 (30.4%) patients. The presence of halo sign in the TAs was found in 207/230 (90.0%) patients and in the AXs in 57/230 (24.8%) patients. According to the ultrasound pattern presented, 173/230 (75.2%) patients had positive TAs and negative AXs ultrasound, 23/230 (10.0%) had negative TAs and positive AXs ultrasound, and 34/230 (14.8%) had both positive TAs and AXs ultrasound. Cranial symptoms were reported in 207/230 (90.0%) patients, in whom AXs involvement on ultrasound was detected in 43/207 (20.8%) of cases. Concerning only the patients with negative TAs and positive AXs ultrasound, 13/23 (56.5%) reported cranial symptoms. Headache was reported in 10/23 (43.5%), visual symptoms in 6/23 (26.1%), jaw or tongue claudication in 4/23 (17.4%), scalp tenderness or paraesthesia in 4/23 (17.4%), and stroke or TIA in 1/23 (4.3%) patients. Constitutional symptoms were present in 13/23 (56.5%), polymyalgia rheumatica in 9/23 (39.1%), and limb claudication in 1/23 (4.3%) patients. A total of 10/23 (43.5%) patients were on glucocorticoid treatment at the time of the diagnosis, but only 4/23 (17.4%) were on prednisolone ≥30mg/day for at least ten days.
Conclusions: Axillary involvement in GCA is frequent, affecting around 1/4 of the patients at diagnosis. The additional assessment of the AXs improved the ultrasound diagnostic sensitivity by 10% compared to only assessing the TAs. Patients with AXs involvement were reported to have cranial symptoms in 43/57 (75.4%) of cases. More than half of the patients with negative TAs and positive AXs ultrasound presented with cranial symptoms. Our results support the need to assess the AXs in patients with suspected GCA, regardless of the presence or absence of cranial symptoms.

067 - RELIABILITY STUDY OF THE SLE-DAS, SLEDAI-2K AND PGA INSTRUMENTS FOR MEASURING SLE DISEASE ACTIVITY

Beatriz Mendes[1], Carolina Mazeda[2], Diogo Jesus[3, 4], Helena Assunção[5], Anabela Barcelos[2, 6], Luisa Brites[3], Daniel Carvalho[7], Carlos Costa[5], Emanuel Costa[8], Ana Bento da Silva[9], Margarida Faria[7], Gisela Eugénio[2], Mariana Luis[1, 10], Ana Isabel Maduro[1], Margarida Lucas Rocha[11], Beatriz Samões[12], Filipe Cunha Santos[13], André Saraiva[1], Susana P. Silva[2], Cláudia Vaz[4, 13], Paulo Vilas-Boas[2], JAP da Silva[1, 10], Ines L[1, 4]
[1]Rheumatology Department, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [2]Rheumatology Department, Centro Hospitalar do

Baixo Vouga, Aveiro, Portugal, [3]Serviço de Reumatologia, Centro Hospitalar de Leiria, Leiria, Portugal, [4]Faculdade de Ciências da Saúde, Universidade da Beira Interior, Covilhã, Portugal, [5]Rheumatology Department, Centro Hospitalar Trás-os-Montes e Alto Douro, Vila Real, Portugal, [6]Comprehensive Health Research Centre (CHRC), NOVA Medical School | Faculdade de Ciências Médicas, NMS|FCM, Universidade Nova de Lisboa, Lisboa, Portugal, [7]Rheumatology Department, Hospital Dr. Nélio Mendonça, Funchal, Portugal, [8]Serviço de Reumatologia, Hospital de Braga, Braga, Portugal, [9]Rheumatology Department, Centro Hospitalar de Lisboa Ocidental, Hospital Egas Moniz EPE, Lisboa, Portugal, [10]Faculdade de Medicina, Universidade de Coimbra, Coimbra, Portugal, [11]Rheumatology Department, Centro Hospitalar Universitário do Algarve, Faro, Portugal, [12]Rheumatology Department, Centro Hospitalar de Vila Nova de Gaia/ Espinho, Vila Nova de Gaia, Portugal, [13]Rheumatology Department, Unidade Local de Saúde da Guarda - Hospital Sousa Martins, Guarda, Portugal

Background: The Systemic Lupus Erythematosus Disease Activity Score (SLE-DAS) has been recently developed and validated, providing improved accuracy and sensitivity for changes in SLE disease activity in comparison to SLE Disease Activity Index 2000 (SLEDAI-2K). New recommendations to standardize the Physician Global Assessment (PGA) scoring may improve its reliability.

Objectives: To assess the intra- and interrater reliability of SLE-DAS, SLEDAI-2K and PGA for measuring SLE disease activity.

Methods: A set of 24 clinical vignettes were abstracted, each from a real clinical visit of patients followed at an academic lupus clinic. These vignettes were selected to include a wide spectrum of SLE manifestations, organ-system involvements, and global severity of disease activity. Abstracted data were presented in a standardized format, including demographic, past medical history, current clinical picture and treatment, laboratory, and other workup assessments. A group of 19 raters were recruited as a random multicenter sample of Rheumatologists. All raters completed a preliminary training session on scoring rules for SLE-DAS, SLEDAI-2K and PGA. Each rater scored each clinical vignette with SLE-DAS, SLEDAI-2K, and PGA through an online survey. The scoring was repeated in a second round 7-14 days after the first one. The clinical vignettes were randomly ordered for each round. Inter and intra-rater reliability of each instrument was estimated using the intraclass correlation coefficient (ICC) with 95% confidence intervals (95%CI), based on single-measurement, absolute agreement, with a two-way random effect or two-way mixed-effects model, respectively.

TABLE 1. Interrater and intra-rater reliability of SLE-DAS, SLEDAI-2K and PGA

	ICC (95%CI)	
	Interrater reliability	Intra-rater reliability
SLE-DAS	0.885 (0.819-0.939)	0.918 (0.902-0.931)
SLEDAI-2K	0.812 (0.717-0.896)	0.892 (0.871-0.909)
PGA	0.704 (0.578-0.828)	0.900 (0.881-0.916)

CI: confidence interval; ICC: intraclass correlation coefficient.

Results: The 19 raters included 8 rheumatologists and 11 rheumatology trainees from 11 hospitals, with a mean of 12.1±7.1 and 3.6±0.5 years of rheumatology practice, respectively, and 78.9% of the participants assess ≤5 SLE patients per week in their regular clinical practice. The 24 clinical vignettes included 83.3% female patients, with a mean of 36.5±17.9 years of age. Active SLE organ involvement included: skin rashes (20.8%); arthritis (12.5%); nephritis (12.5%); thrombocytopenia (12.5%); cardiac/pulmonary involvement (12.5%); mucocutaneous vasculitis (8.3%); serositis (8.3%); neuropsychiatric lupus (8.3%). Systemic vasculitis, myositis, alopecia, hemolytic anemia, and leukopenia were each present in 4.2% of the vignettes. Hypocomplementemia and/or high anti-dsDNA were present in 75.0%. Twenty-one percent of the cases were in remission.

All raters completed the survey, totaling 912 case assessments. Scores attributed by the raters ranged from 0.37 to 49.53 in SLE-DAS, 0 to 24 in SLEDAI-2K, and 0.0 to 3.0 in PGA. The interrater reliability was good for SLE-DAS and SLEDAI-2K, and moderate for PGA. The intra-rater reliability was excellent for SLE-DAS, and good for SLEDAI-2K and PGA (Table 1).

Conclusions: SLE-DAS presents high intra- and interrater reliability for measuring SLE disease activity. The high reliability of SLE-DAS is an important quality both in clinical practice and research, allowing consistent scoring among different clinicians including those who are not SLE experts.

Acknowledgements: Beatriz Mendes and Carolina Mazeda contributed equally and share first authorship.

075 - CAN WE PREDICT THE RISK FACTORS FOR SWITCHING IN THE FIRST YEAR OF THERAPY WITH BDMARD IN PATIENTS WITH RHEUMATOID ARTHRITIS?

Ana Martins[1, 2], Daniela Oliveira[1, 2], Rafaela Nicolau[2,]

[3], Alexandra Bernardo[1], Teresa Martins-Rocha[1, 2], Sofia Pimenta[1, 2], Miguel Bernardes[1, 2], Lúcia Costa[1]
[1]Serviço de Reumatologia, Centro Hospitalar Universitário de São João, Porto, Portugal, [2]Departamento de Medicina, Faculdade de Medicina da Universidade do Porto, Porto, Portugal, [3]Rheumatology Unit, Centro Hospitalar Tondela-Viseu, Viseu, Portugal

Background: Biological Disease-modifying Antirheumatic Drugs (bDMARD) have improved the clinical course and quality of life of patients with rheumatoid arthritis (RA). However, some patients failed to respond or have an insufficient response to bDMARD, early in the course of the treatment and the reasons why this happens aren't fully understood.
Objectives: To determine the percentage of RA patients who failed to respond to bDMARD and need to switch in the first year of treatment, describe their characteristics, and identify specific baseline features as possible predictors of bDMARD early failure.
Methods: A monocentric retrospective cohort study was conducted with RA patients (according to 2010 ACR/EULAR criteria), registered in the national database (Reuma.pt) that started their first bDMARD and had a minimum follow-up of 12 months. Demographic data, disease characteristics, laboratory parameters and treatment at baseline were collected. Disease activity scores (CDAI, SDAI and DAS-28-CRP) and functional scores (HAQ) were collected at baseline and 12 months after the start of bDMARD (T12). Clinical response to treatment (according to EULAR and ACR response criteria) was collected at T12. The proportion of patients who failed to respond (according to treat-to-target strategies) and who switched to another bDMARD was calculated. Patients who discontinued treatment in the first year due to adverse events were excluded. Chi-square test, t-test and Mann-Whitney U test were conducted. A multivariate logistic regression analysis was performed. Odds ratio (OR) and 95% confidence interval (CI) were calculated. A p-value <0.05 was considered statistically significant.
Results: A total of 437 (364 females, 83.3%) patients with RA were included. The mean age was 52.4±11.4 years and the disease duration was 11.8±8.8 years. Regarding therapy, 315 (72.1%) patients started an anti-TNF-α agent, 66 (15.1%) rituximab, 51 (11.7%) tocilizumab and 5 (1.1%) abatacept. Forty-eight (11.0%) patients failed to respond to the bDMARD in the first year of treatment (mean duration of bDMARD treatment was 0.75±0.3 years) and needed to switch to another bDMARD. Demographic characteristics were similar in the group of patients that switch due to ineffectiveness and patients that didn't switch in the first year. DAS-28-CPR, CDAI, SDAI and HAQ at baseline were also similar in the two groups. There were significantly more current or former smokers and patients with depression in the group of patients that needed to switch (p=0.03 and p=0.003 respectively). Positivity for rheumatoid factor at baseline was also significantly more frequent in the switch group (p=0.014). Regarding the type of bDMARD, patients in the switch group were more frequently treated with anti-TNF-α agents than patients that didn't switch (p<0.001).
In the multivariate analysis, anti-TNF-α agents use (versus non-anti-TNF-α agents) (OR 8.3, 95%CI 2.4-28.8, p=0.001), tobacco exposure (OR 2.3, 95%CI 1.1-4.8, p=0.02) and history of depression (OR 3.1, 95%CI 1.3-7.7) seem to predict the bDMARD early failure and the need to switch in the first year of treatment.
Conclusion: In our study, anti-TNF-α agents were associated with a higher switch rate due to ineffectiveness in the first year of treatment in RA patients, when comparing to non-anti-TNF-α agents. Moreover, current and former smokers, patients with depression and with positive rheumatoid factor had a higher switch rate due to ineffectiveness.

079 - MICROSPA: GUT MICROBIOTA AND AXIAL SPONDYLOARTHRITIS: THE IMPACT ON ANTI-TNF THERAPY

Diana Rodrigues[1], Rita Pinheiro Torres [2, 3], Ana Bento da Silva[2, 3, 4], Jaime C. Branco[1, 2, 3, 5], Diana Teixeira[1, 5], Conceição Calhau[1, 6], Ana Faria[1, 5], Pimentel-Santos FM[1, 2, 3]
[1]NOVA Medical School|Faculdade de Ciências Médicas, Universidade NOVA de Lisboa, Lisboa, Portugal, Lisboa, Portugal, [2]iNOVA4Health, Rheumatology Lab, NOVA Medical Research, Lisboa, Portugal, Lisboa, Portugal, [3]Rheumatology Department, Centro Hospitalar de Lisboa Ocidental, Hospital Egas Moniz EPE, Lisboa, Portugal, [4]Rheumatology, Leiden University Medical Center, Leiden, Netherlands, [5]Comprehensive Health Research Centre (CHRC), NOVA Medical School | Faculdade de Ciências Médicas, NMS|FCM, Universidade Nova de Lisboa, Lisboa, Portugal, [6]Centro de Investigação em Tecnologias e Serviços de Saúde (CINTESIS), Lisboa, Portugal

Background: The etiopathogenesis of axial spondyloarthritis (axSpA) is not fully understood, and emerging evidence suggests that the gut microbiota could have a significant role in the initiation and progression of axSpA. The advent of anti-Tumor Necrosis Factor (TNFi) therapy has revolutionized the treatment of axSpA. However, a substantial proportion of patients do not respond to these expensive medications. Therefore, it is crucial to comprehend the factors that influence treatment response and identify novel predictors of therapeutic efficacy.
Aim: To assess the relationship between fecal micro-

biota composition and TNFi therapy in axSpA patients with high levels of disease activity. Furthermore, we characterized the composition of the fecal microbiota and investigated associations between bacterial taxa and disease activity.

Methodology: This 14-week prospective observational study included axSpA patients, who had an indication for TNFi therapy. Each patient provided two fecal samples: one at baseline and another after 14 weeks of TNFi treatment. Bacterial DNA was extracted from the samples, and specific bacterial groups were identified and quantified using 16S rDNA sequencing. The activity of axSpA was assessed at each time point using the Ankylosing Spondylitis Disease Activity Score with C-Reactive Protein (ASDAS-CRP) and Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) scores.

Results: We observed that TNFi, has an impact on the composition of the fecal microbiota, leading to shifts in the abundance of specific genera. Specifically, we observed an increase in the abundance of Bifidobacterium, Butyrivibrio, Pseudobutyrivibrio, Eubacterium, and Sutterella, and a decrease in the abundance of Porphyromonas. Notably, we identified Porphyromonas as a biomarker of disease activity. These findings reinforce previous observations that patients with higher levels of inflammation and disease activity are more likely to respond to TNFi.

Our investigation suggests that the fecal microbiota could potentially serve as a tool for predicting the treatment response to TNFi in axSpA patients. Specifically, we found that patients with fecal microbiota characterized by a higher abundance of Anaerovorax, Coprococcus, Lautropia, Rothia, Saccharothrix, Succiniclasticum, and Veillonella were associated with a failure to respond to treatment. On the other hand, it appears that axSpA patients may respond more effectively to anti-TNF treatment if they exhibit increased bacterial richness and increased abundances of Gemmatimonadetes, Atopobium, Burkholderia, and Escherichia.

Conclusion: This pilot study provides interesting preliminary data on the fecal microbiota characteristics of axSpA patients. It offers insights into the dynamic alterations of the microbiota following 14 weeks of TNFi and identifies microbial biomarkers that can potentially guide the development of precision therapy in axSpA.

082 - DOES AGE AT DIAGNOSIS OF GIANT CELL ARTERITIS INFLUENCE THE CLINICAL PHENOTYPE AND OUTCOMES?

Matilde Bandeira[1, 2], Diana Raimundo[3, 4], Martins-Martinho J [1, 2], Eduardo Dourado[1, 2], Adriana Carones[5], Jorge Pestana Lopes[6], Mariana Santos[7], Susana P. Silva[8], Catarina Soares[9], Vítor Teixeira[10], Ana Rita Cruz-Machado[1, 2], Sofia C Barreira[1, 2], Nikita Khmelinskii[1, 2], Carla Macieira[2], Ines L[5], JE Fonseca[1, 2, 3], Vasco C Romão[1, 2, 3], Cristina Ponte[1, 2, 3]
[1]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [2]Serviço de Reumatologia, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Clínica Universitária de Reumatologia, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, Lisboa, Portugal, [4]Rheumatology Department, Hospital Beatriz Ângelo, Lisboa, Portugal, [5]Serviço de Reumatologia, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [6]Serviço de Reumatologia, Hospital Garcia de Orta, Almada, Portugal, [7]Rheumatology Department, Centro Hospitalar de Lisboa Ocidental, Hospital Egas Moniz EPE, Lisboa, Portugal, [8]Serviço de Reumatologia, Centro Hospitalar do Baixo Vouga, E.P.E., Aveiro, Portugal, [9]Serviço de Reumatologia, Unidade Local de Saúde do Alto Minho, Ponte de Lima, Portugal, [10]Serviço de Reumatologia, Centro Hospitalar e Universitário do Algarve, Faro, Portugal

Background: Giant Cell Arteritis (GCA) is the most prevalent primary systemic vasculitis affecting individuals ≥50 years old. Previous studies suggest that age at diagnosis influences the clinical features and prognosis of GCA. A better identification of patients at a higher risk of worse outcomes may prompt a more tailored treatment approach. Our work aims to assess how age at diagnosis of GCA influences its phenotype and disease course.

Methods: Multicentre open cohort study, including all patients with GCA registered in the Rheumatic Diseases Portuguese Register (Reuma.pt) up to January 2023. Two groups were established using the median age at diagnosis of 75 years as the cut-off. Patients who underwent ultrasound (US) evaluation were divided into three groups according to their baseline US results: exclusive large vessel GCA (LV-GCA) in cases of axillary and/or subclavian artery involvement, exclusive cranial-GCA in cases of temporal artery involvement, and mixed pattern when both arterial territories were involved. Eye involvement was defined as arteritic ischemic optic neuropathy (AION), transient amaurosis and/or permanent vision loss. Independent predictors of different clinical manifestations were identified through binomial logistic regression. Kaplan-Meier with the Log Rank test was performed to evaluate maintenance on glucocorticoid (GC) therapy and time to first relapse.

Results: A total of 294 patients were included, 65.6% females, with a median age at diagnosis of 75.2 (IQR 11.0) years (range 51-91 years). Patients most com-

TABLE 1. Clinical and laboratorial characteristics in younger and older patients

	Younger patients, ≤75 years old (n=145)	Older patients, >75 years old (n=149)	Univariate analysis
Age at diagnosis, mean ± SD years (N)	67.42 ± 5.85 (145)	80.86 ± 4.09 (149)	-
Female sex, n/N (%)	92/145 (63.4)	101/149 (67.8)	p=0.463
Diagnosis delay, mean ± SD years (N)	0.37 ± 0.87 (145)	0.38 ± 1.14 (149)	p=0.983
Patients who fulfil the classification criteria for GCA, n/N (%)			
ACR 1990	125/144 (86.8)	133/147 (90.5)	p=0.359
ACR / EULAR 2022	111/117 (94.9)	118/120 (98.3)	p=0.168
Clinical data			
Pattern of disease involvement according to ultrasound results, n/N (%)			
Exclusive cranial-GCA	75/99 (75.8)	88/109 (80.7)	p=0.404
Exclusive large vessel-GCA	17/99 (17.2)	7/109 (6.4)	p=0.017
Mixed pattern GCA	7/99 (7.1)	14/109 (12.8)	p=0.249
Symptoms/signs, n/N (%)			
Night sweats	15/128 (11.7)	5/134 (3.7)	p=0.019
Fever	26/128 (20.3)	11/132 (8.3)	p=0.007
Weight loss	61/127 (48.0)	64/133 (48.1)	p=1.000
Arthralgia	45/129 (34.9)	42/135 (31.1)	p=0.600
Polymyalgia rheumatica	63/130 (48.5)	58/136 (42.6)	p=0.389
Jaw claudication	43/128 (33.6)	66/136 (48.5)	p=0.017
Tong claudication	6/128 (4.7)	4/134 (3.0)	p=0.533
Arm claudication	5/128 (3.9)	0/134 (0.0)	p=0.027
Leg claudication	2/128 (1.6)	0/134 (0.0)	p=0.238
Scalp tenderness	24/128 (18.8)	22/135 (16.3)	p=0.629
New-onset frontal headache	36/129 (27.9)	49/135 (36.3)	p=0.150
New-onset temporal headache	62/128 (48.4)	79/137 (57.7)	p=0.141
Ischaemic cerebral event	36/95 (37.9)	82/113 (72.6)	p<0.001
Transient amaurosis	9/126 (7.1)	26/134 (19.4)	p=0.006
Permanent vision loss	21/125 (16.8)	59/138 (42.8)	p<0.001
Eye involvement*	27/127 (21.3)	75/138 (54.3)	p<0.001
Signs/physical examination changes, n/N (%)			
Loss of pulses	7/125 (5.6)	1/132 (0.8)	p=0.032
Abnormalities on temporal arteries	41/130 (31.5)	52/134 (38.8)	p=0.247
Abnormalities on other arteries	14/128 (10.9)	5/134 (3.7)	p=0.031
Arteritic ischemic optic neuropathy (AION)	15/127 (11.8)	48/135 (35.6)	p<0.001
Laboratory examination at baseline, mean ± SD (N)			
C-reactive protein (mg/dL)	7.52 ± 6.61 (103)	6.40 ± 5.87 (108)	p=0.194
Erythrocyte sedimentation rate (mm/hr)	81.83 ± 29.24 (105)	85.33 ± 36.08 (113)	p=0.431
Haemoglobin (g/dL)	11.67 ± 1.62 (94)	11.53 ± 1.61 (103)	p=0.540
Platelets (/uL)	381000 ± 142048 (87)	368410 ± 148745 (94)	p=0.561
Treatment, n/N (%)			
IV methylPDN pulses	27/88 (30.7)	60/102 (58.8)	p<0.001
Methotrexate	74/145 (51.0)	58/149 (38.9)	p=0.046

continues on the next page

TABLE 1. Continuation

	Younger patients, ≤75 years old (n=145)	Older patients, >75 years old (n=149)	Univariate analysis
Azathioprine	3/145 (2.1)	1/149 (0.7)	p=0.366
Tocilizumab	20/145 (13.8)	11/149 (7.4)	p=0.088
PDN cumulative dose/disease duration, mean ± SD grams per year (N)	8.82 ± 22.32 (77)	18.02 ± 58.30 (87)	p=0.195
Disease follow-up, n/N (%)			
Disease follow-up, mean ± SD years (N)	5.05 ± 4.77 (145)	3.25 ± 3.65 (149)	p<0.001
Patients who relapsed	46/90 (51.1)	43/93 (46.2)	p=0.555
Time to first relapse, mean ± SD years (N)	1.88 ± 3.02 (46)	1.15 ± 1.15 (43)	p=0.140
Number of disease relapses in the first two years	0.47 ± 0.69 (90)	0.49 ± 0.70 (92)	p=0.828
Number of relapses throughout follow-up	0.84 ± 1.09 (90)	0.77 ± 0.98 (93)	p=0.647

Abbreviations: n – number of patients positive for the variable of interest, N – number of patients without missing information regarding the variable of interest, PDN – prednisolone, SD – standard deviation;
* Eye involvement: presence of transient amaurosis, permanent vision loss or AION

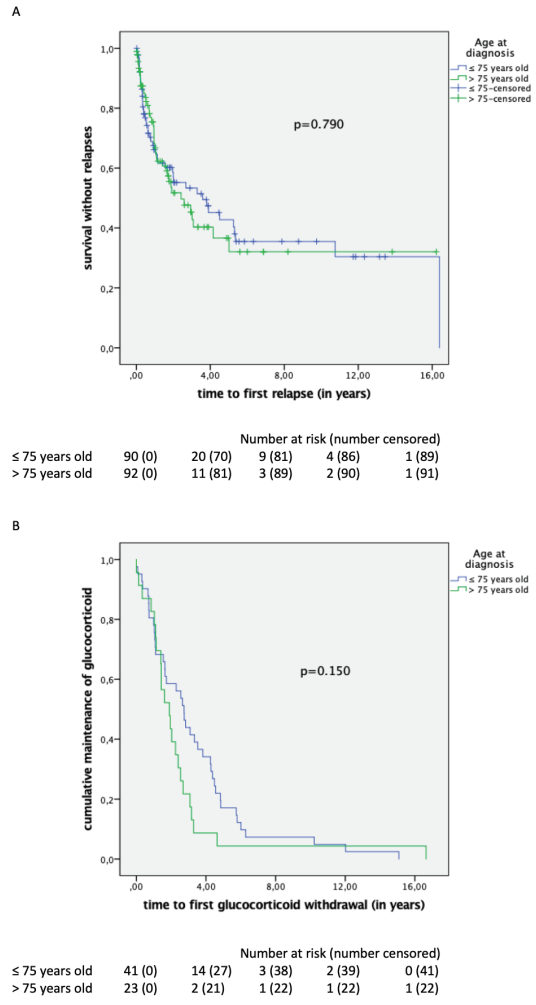

Figure 1. Survival analysis for time to first relapse (A) and time to first complete GC withdrawal (B)

monly presented with new onset temporal headache (53.2%), weight loss (48.1%), polymyalgia rheumatica (PMR; 45.5%), fatigue (45.1%), jaw claudication (41.3%) and eye involvement (33.8%). Exclusive cranial-GCA was identified in 78.4% of patients, exclusive LV-GCA in 11.5% and mixed pattern in 10.1%. Jaw claudication, transient amaurosis, permanent vision loss, AION and cerebral ischaemic events were more likely to be present in older patients (Table 1). In contrast, younger patients were more likely to have fever, night sweats, arm claudication and exclusive LV-GCA. On multivariate analysis, older age at diagnosis (OR 1.07, 95%CI: 1.03-1.12) was an independent predictor of cerebral ischemic events related to GCA, regardless of sex, disease duration, exclusive LV-GCA, PMR, and fatigue. The same model identified exclusive LV-GCA (OR 0.19, 95%CI: 0.06-0.63) and PMR (OR 0.40, 95%CI: 0.20-0.81) as protective factors. Age at diagnosis (OR 1.07, 95%CI: 1.02-1.11) was also an independent predictor of eye involvement, regardless of sex, disease duration, exclusive LV-GCA, fatigue and night sweats. Exclusive LV-GCA (OR 0.15, 95%CI: 0.03-0.67) was a protective factor. The mean disease duration of our cohort was 4.13±4.33 years. Patients ≤75 and >75 years old had a similar proportion of relapsing disease (p=0.555) and number of relapses (p=0.828) (Table 1). Furthermore, on survival analysis no differences were found in time to first relapse (p=0.790, Figure 1A) and time to first complete GC withdrawal (p=0.150, Figure 1B) between groups.

Conclusions: Our study shows that older patients with GCA have more cranial symptoms and cerebral ischaemic complications, whereas younger patients present with a more systemic disease phenotype. Old-

er age at diagnosis is an independent predictor of eye involvement and cerebral ischaemic events related to GCA. These findings support the hypothesis that age at diagnosis in patients with GCA influences disease manifestations and outcomes.

115 - MATERNAL AND PERINATAL OUTCOMES IN WOMEN WITH VASCULITIS - A 13-YEAR EXPERIENCE FROM A PORTUGUESE TERTIARY CENTRE

Ana Rita Lopes[1, 2], Ana Teodósio Chícharo[1, 2, 3], Ana Rita Cruz-Machado[1, 2], Sofia C Barreira[1, 2], Patrícia Martins[1, 2], Cristina Ponte[1, 2], Luísa Pinto[4], Susana Capela[1, 2]
[1]Rheumatology and Bone Metabolic Diseases Department, Centro Hospitalar Lisboa Norte, EPE - Hospital de Santa Maria, Lisbon Academic Medical Centre, Lisboa, Portugal, [2]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Rheumatology Department, Centro Hospitalar Universitário do Algarve, Faro, Portugal, [4]Departamento de Ginecologia, Obstetrícia e Medicina da Reprodução, Centro Hospitalar Universitário Lisboa Norte, CHULN, Lisboa, Portugal

Background: Vasculitides are rare conditions that may affect women of childbearing age potentially increasing their risk for developing adverse pregnancy outcomes (APO). Maternal and perinatal outcomes as well as their optimal management during pregnancy remain poorly understood in this population.
Objectives: This study aimed to describe maternal and perinatal outcomes in pregnant women with vasculitis.
Methods: Observational retrospective study including pregnant women with vasculitis who were followed at our rheumatology-obstetric clinic from 01/2009 to 11/2022. We collected and analysed data from past cases to identify patterns and outcomes related to vasculitis in pregnancy.
Results: We identified 31 pregnancies in 24 women with vasculitis. Table 1 summarizes demographic and clinical data. The most frequent diagnosis were Behçet's disease (BD) (18; 58%), polyarteritis nodosa (PAN) (4; 13%) and Takayasu arteritis (TAK) (4; 13%). The mean ± SD age at conception was 33 ± 6 years with a median disease duration of 6 (IQ range 3-12) years, 29 (94%) were in remission by the time of conception. We documented 30 live births, 1 early miscarriage (BD) and there was no perinatal mortality or birth defect. The mean ± SD gestational age at delivery was 38.2 ± 2.1 weeks. Five (17%) viable pregnancies resulted in preterm births - 3 BD, 1 TAK and 1 relapsing polychondritis (RP). Fetal growth restriction was detected in 2 (6%) pregnancies (TAK and RP). Eight (30%) newborns were small for gestational age (SGA), most of them from

TABLE 1. Maternal and perinatal outcomes in women with vasculitis followed at a rheumatology-obstetric clinic

Main diagnosis	N (%)	Gestational age at delivery (mean ± SD weeks)	BW (mean ± SD grams)	Miscarriages N (%)	SGA N (%)	FGR N (%)	Preterm births N (%)	Pregnancy flares N (%)	Post- partum flares N (%)
Behçet's disease	18 (58)	38.3 ± 1.5	3013 ± 494	1/18 (6)	4/14 (29)	0	3/17 (18)	4/17 (24)	4/14 (29)
Polyarteritis nodosa	4 (13)	37.4 ± 0.6	2783 ± 175	0	1/4 (25)	0	0	2/4 (50)	0
Takayasu arteritis	4 (13)	38.0 ± 2.1	2725 ± 364	0	1/4 (25)	1/4 (25)	1/4 (25)	1/4 (25)	1/4 (25)
IgA vasculitis	1 (3)	40.3	3435	0	0	0	0	0	0
ANCA-PR3 cutaneous vasculitis	1 (3)	40.9	2845	0	1	0	0	1	0
Relapsing polychondritis	1 (3)	36.0	2040	0	1	1	1	1	1
Cryoglobulinemic vasculitis	1 (3)	35.9	3020	0	0	0	0	0	0
Cryoglobulinemic vasculitis associated with Sjögren's syndrome	1 (3)	39.0	3000	0	0	0	0	0	0
	31 (100)	38.2 ± 2.1	2925± 566	1/31 (3)	8/27 (30)	2/31 (6)	5/30 (17)	9/25 (36)	6/27 (22)

Legend: BW - birthweight; SD - standard deviation; SGA - small for gestational age; FGR - fetal growth restriction

mothers with BD (4; 29%). No cases of preeclampsia or eclampsia were recorded. Cesarean was performed in 8 (26%) patients – 2 (TAK) due to vasculitis activity. Nine (36%) patients relapsed during pregnancy (4 BD; 2 PAN; 1 TAK; 1 RP; 1 ANCA-PR3 cutaneous vasculitis) while 6 (22%) relapsed during the post-partum period (4 BD; 1 TAK and 1 RP). A total of 24 (77%) patients were treated with glucocorticoids (GC), most of them (63%) at low doses (≤5mg/day of prednisolone or equivalent). Seven (23%) patients were treated with conventional DMARDs: 2 TAK and 3 BD with azathioprine and 2 PAN with azathioprine and hydroxychloroquine. Biological DMARDs were prescribed in 2 (6%) patients, both with the diagnosis of TAK: one with a multisegmental aortic disease who received tocilizumab during the first 30 weeks of gestation (WG) and had an uneventful pregnancy, and another patient with an ascending aortic aneurism (AAA) who also received tocilizumab, but from the 24th week onwards showed a gradual worsening of the AAA dimensions (maximum 56 mm). An elective cesarean at 32 WG was performed in the latter case due to the high risk of aneurysm rupture and a healthy baby with 1900g was born. The occurrence of flares during pregnancy increased the risk for APO (OR 13, 95 CI 2-80; p=0.007). No association was found concerning maternal age or use of DMARDs/GC and the risk of APO (p>0.05).

Conclusion: Most pregnant women with vasculitis who were managed at out multidisciplinary unit had successful gestations. However, they may still be at risk for developing APO, namely if they experience disease flares during gestation. SGA was the main APO recorded. The results highlight the importance of close monitoring and management of vasculitis in pregnant women to minimize the risk of complications.

151 - A TWO-YEAR COMPARISON OF QUALITY OF LIFE BETWEEN AXIAL SPONDYLOARTHRITIS AND NON-AXIAL SPONDYLOARTHRITIS CHRONIC BACK PAIN PATIENTS IN THE SPONDYLOARTHRITIS CAUGHT EARLY (SPACE) COHORT

Ana Bento da Silva[1, 2, 3], Anne Boel[3, 4], Miranda van Lunteren[3], Mary Lucy Marques[3, 5], MGH van de Sande[6], Sofia Ramiro[3, 7], Désirée van der Heijde[3], F vanGaalen[3]
[1]Rheumatology Department, Centro Hospitalar de Lisboa Ocidental, Hospital Egas Moniz EPE, Lisboa, Portugal, [2]Comprehensive Health Research Centre (CHRC), NOVA Medical School | Faculdade de Ciências Médicas, NMS|FCM, Universidade Nova de Lisboa, Lisboa, Portugal, [3]Rheumatology, Leiden University Medical Center, Leiden, Netherlands, [4]UCB Pharma B.V., Breda, Netherlands, [5]Rheumatology Department, Centro Hospitalar Universitário de Coimbra, Coimbra, Portugal, [6]Department of Rheumatology & Clinical Immunology, Amsterdam University Medical Center, Amsterdam, Netherlands, [7]Rheumatology Department, Zuyderland Medical Center, Heerlen, Netherlands

Background: Treatment of axial spondyloarthritis (axSpA) has shown to improve patient outcomes including health-related quality of life (HRQoL). This is one of the reasons for which many efforts have been made to diagnose axSpA earlier. We set out to compare HRQoL at baseline and after 2 years in early axSpA versus (vs) non-axSpA chronic back pain (CBP) patients.

Methods: The population consisted of adults (≥16 years) with CBP of unknown origin lasting ≥3 months and ≤2 years, starting before 45 years, included in the SPondyloArthritis Caught Early (SPACE) cohort. Patients had a diagnosis of axSpA or non-axSpA at 2 years with a high level of confidence by the treating rheumatologist (Marques ML, Ann Rheum Dis 2023;82:3-4). The diagnosis was established according to clinical practice, based on patient's medical history, physical examination, laboratory tests and imaging findings. HRQoL was assessed using the Medical Outcomes Study 36-Item Short-Form Health Survey (SF36). Age, sex and country-matched scales were calculated for each of the 8 subscales, ranging from 0 (worst health) to 100 (best health). The physical (PCS) and mental component summary (MCS) scores were calculated from the adjusted scores on each subscale and transformed to enable comparison to the general population mean of 50. The proportion of patients with an improvement or worsening of the PCS and MCS above the minimal clinically important difference (MCID ≥3 points; Davis JC, Arthritis Rheum 2007;57:1050-1057) was also evaluated. Only patients with complete data at baseline and 2 years were considered. Paired t-tests were used to compare baseline and 2-year results within groups. For the comparison axSpA vs non-axSpA, linear regression models were built, with either PCS or MCS as the outcome adjusting for the baseline value of the respective outcome, gender, age and use of nonsteroidal anti-inflammatory drugs (NSAIDs).

Results: A total of 366 patients (71% axSpA) were included. AxSpA patients (vs non-axSpA patients) were more frequently male (53% vs 25%) and had more SpA features (mean (SD): 5 (2) vs 3 (1)), including HLA-B27 positivity (73% vs 30%). Age (mean (SD): 30 (8) vs 31 (8) years) and symptom duration (mean (SD): 13 (7) vs 13 (7) months) were similar between groups.

PCS significantly improved after 2 years in both groups (Table 1, p<0.001), even though it remained considerably impaired, mainly in non-axSpA patients (mean (SD) scores at 2 years: 39.3 (12.5) for axSpA and 35.8

TABLE 1. Health-related quality of life: comparison between axSpA and non-axSpA chronic back pain patients at baseline and after 2 years.

	AxSpA (N=261)			Non-axSpA (N=105)		
	Baseline	2 years	p-value (over time)	Baseline	2 years	p-value (over time)
SF36: PCS, mean (SD)	28.6 (14.6)	39.3 (12.5)		26.6 (13.6)	35.8 (15.4)	
Change over time, mean (SD)		10.7 (14.4)	p<0.001*		9.1 (14.1)	p<0.001*
Improvement ≥MCID, n (%)		185 (71%)			71 (68%)	
Worsening ≥MCID, n (%)		39 (15%)			17 (16%)	
SF36: MCS, mean (SD)	46.4 (14.0)	47.7 (12.1)		47.7 (10.8)	49.3 (10.1)	
Change over time, mean (SD)		1.3 (13.1)	p=0.114		1.6 (10.9)	p=0.132
Improvement ≥MCID, n (%)		108 (41%)			46 (44%)	
Worsening ≥MCID, n (%)		91 (35%)			35 (33%)	

* Statistical significance (p-value <0.05). axSpA: axial spondyloarthritis; MCS: mental component summary of the SF36; MCID: minimal clinically important difference (≥3 points); PCS: physical component summary of the SF36: SF36: Medical Outcomes Study 36-Item Short-Form Health Survey.

(15.4) for non-axSpA; mean difference between groups: 3.6 points, p=0.022). The proportion of patients with a MCID improvement or worsening were similar in axSpA and non-axSpA (71% vs 68% and 15% vs 16%, respectively). In adjusted multivariable analysis, axSpA (vs non-axSpA) was an independent predictor of higher PCS scores at 2 years (adjusted coefficient 3.3, 95% CI (0.3; 6.4); p=0.034). In contrast, no relevant impact on the MCS was found for either group, with scores very close to the general population mean of 50 points.

Conclusion: Over 2 years, the physical component of HRQoL improves in both axSpA and non-axSpA patients. While it remains impaired in both groups compared to the general population, worse scores are observed in patients with non-axSpA compared to those with axSpA. Compared to non-axSpA, axSpA is an independent predictor of larger improvement in physical HRQoL. No significant impact is found for mental HRQoL in either group.

155 - MIOPATIA INFLAMATÓRIA VS. DISTROFIA MUSCULAR: UM CASO DE DOENÇA ASSOCIADA AO ANTICORPO ANTIPM/SCL-100

Carlos Marques-Gomes[1, 2], Mariana Diz-Lopes [1, 2], Luís Braz[3], Miguel Bernardes[1, 2], Lúcia Costa[1]

[1]Serviço de Reumatologia, Centro Hospitalar de São João, Porto, Portugal, [2]Departamento de Medicina, Faculdade de Medicina da Universidade do Porto, Porto, Portugal, [3]Serviço de Neurologia, Centro Hospitalar de São João, Porto, Portugal

Introdução: A positividade para o anticorpo antiPM/Scl-100 associa-se, frequentemente, à síndrome de sobreposição esclerose sistémica/polimiosite (ES-PM). Contudo, o diagnóstico pode não ser óbvio na ausência de critérios suficientes para enquadrar o doente numa das doenças e perante a coexistência de uma distrofia muscular.

Caso Clínico: Sexo masculino, 44 anos, referenciado à consulta do nosso centro (setembro de 2019) dois anos após ter sido diagnosticado com esclerose sistémica (ES), no contexto de artralgias de ritmo inflamatório e edema difuso das mãos. Ao exame objetivo, apresentava atrofia marcada dos músculos periescapulares (Figura 1), força proximal grau 3/5 do membro superior esquerdo e esclerodactilia nas falanges intermédias dos dedos das mãos. Estava medicado com prednisolona (PDN) 5 mg/dia e azatioprina (AZA) 200 mg/dia e aguardava 1º retratamento com rituximab. Do estudo analítico, destacavam-se aumento das enzimas musulares (CPK 611 UI/L; aldolase 8,7 UI/L; mioglobina 225 ng/mL), anticorpos (ac) antinucleares (ANA) 1/160 (padrão nucleolar), ac antiPM/Scl-100 positivo, com restante imunologia negativa (incluindo painel de miopatias e esclerose sistémica). Trazia o relatório da biópsia muscular diagnóstica, onde apresentava atrofia de fibras musculares do tipo 2 e expressão focal de antigénios do complexo principal de histocompatibilidade classe I, sem infiltrado inflamatório, sugerindo miopatia inflamatória ou doença genética com contributo inflamatório. Numa EMG recente revelava características miopáticas em músculos da cintura escapular esquerda, compatíveis com miopatia proximal e o seu fenótipo levantava a hipótese de distrofia muscular das cinturas (LGMD). Após estudo genético do painel para LGMD, foi confirmado o diagnóstico de titinopatia [2 VUS (em trans; HTZ composta) gene TTN – Ile659Val e Arg9489Gln]; em maio de 2021, por decisão multidisciplinar com a neurologia, a imunossupressão foi sus-

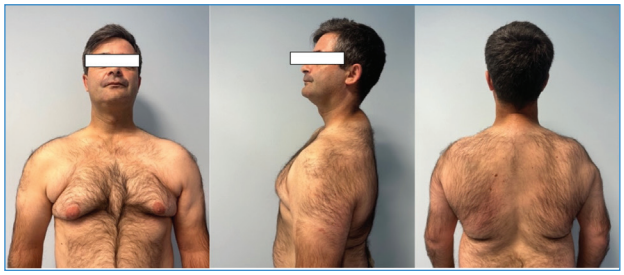

Figura 1. Atrofia muscular da cintura escapular

pensa. Contudo, após 1 ano, o doente reiniciou artralgias inflamatórias das mãos com sensação de tumefação difusa dos dedos e mialgias proximais dos membros superiores, apresentando nova elevação das enzimas musculares (CPK 1203 UI/L; CPK-MB 62,4 ng/mL; aldolase 23,2 UI/L; mioglobina 460,6 ng/mL). Assim, foi retomada AZA até à dose de 200 mg/dia, com franca melhoria clínica e com diminuição das enzimas musculares para níveis inferiores aos prévios à suspensão da AZA. A videocapilaroscopia do leito ungueal manteve-se sem alterações patológicas.

Discussão: Embora a presença do ac anti-PM/Scl-100 se associe à síndrome de sobreposição ES-PM, o doente não preenche critérios de classificação que possam enquadrar os sinais clínicos objetiváveis numa outra doença específica do tecido conjuntivo (esclerose sistémica), o que pode sugerir a existência de um fenótipo característico relacionado com o referido ac. Além disso, o diagnóstico de polimiosite tem que ser considerado, independentemente da coexistência de uma distrofia muscular, dados os achados da biópsia muscular e o agravamento clínico do doente uma vez suspensa a imunossupressão com AZA. Este caso clínico reforça, assim, a importância da revisitação diagnóstica sempre que os sinais clínicos não são específicos de uma determinada entidade patológica. Fica, também, patente a importância do estudo dos marcadores imunológicos nas conectivites em que, porventura, só com a evolução das caraterísticas clínicas ao longo do tempo é possível obter o diagnóstico reumatológico definitivo.

158 - EFFICACY AND SAFETY OF ULTRASOUND-GUIDED MUSCULAR BIOPSY IN THE DIAGNOSIS OF IDIOPATHIC INFLAMMATORY MYOPATHIES

Filipa Marques Costa[1, 2], Matilde Bandeira[1, 2], Eduardo Dourado[1, 2], Rafael Roque[3], Raquel Campanilho-Marques[1, 2], Fernando Saraiva[1, 2]

[1]Serviço de Reumatologia, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [2]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Laboratório de Neuropatologia, Serviço de Neurologia, Centro Hospitalar Universitário Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Introduction: Idiopathic inflammatory myopathies (IIM) are a group of multisystemic diseases, and the differential diagnosis usually requires clinical, analytical, imaging, and histological evaluation. Muscle biopsies can be performed surgically or percutaneously. Since myositis can affect only some muscles in the same anatomical region, non-guided methods can be associated with lower sensitivity.

Objectives: To assess the safety and efficacy of ultrasound-guided muscle biopsy (UGMB) in obtaining adequate muscle samples for histological analysis to establish or rule out an IIM diagnosis in patients with suspected myositis.

Methods: We included patients followed at the Rheumatology outpatient clinic of a tertiary centre, with a UGMB performed from January 2022 to June 2023. All patients had a suspected IIM diagnosis. Patients with conditions that potentially increased the risk of complications (e.g., coagulation disorders, pregnancy) were excluded. Before the biopsy, a muscle was selected through a brief muscle ultrasound (US) examination. Then, local anaesthetic was injected into the soft tissues up to the muscle fascia under US guidance. Afterwards, US was used to guide a 14 G biopsy needle to an appropriate biopsy site. Finally, five or more muscle samples were collected, placed in a dry-cooled medium and delivered to the Neuropathology laboratory within one hour.

Results: Twenty-five biopsies were performed on 25 patients, of whom 64% (n=16) were female. Vastus lateralis was the muscle most frequently biopsied (n=14). In total 88% (n=23) of the samples allowed a thorough histologic evaluation. From all the samples, 32% (n=8) were compatible with myositis in patients with muscle weakness (two allowed to define a histological subtype, polymyositis and dermatomyositis, respectively), and 28% (n=7) revealed healthy muscle tissue without pathologic features. About 32% (n=8) of the biopsies showed nonspecific alterations of the muscle. One of those patients was diagnosed with polymyositis, the rest, although having a raised CK (creatine kinase), remained without an established IIM diagnosis. Finally, 8% (n=2) did not have enough material to perform a complete histologic diagnosis, and 4% (n=1) presented histological artifacts related to the biopsy procedure/sample manipulation. The histological findings were compatible with the final diagnosis in 44% of the pa-

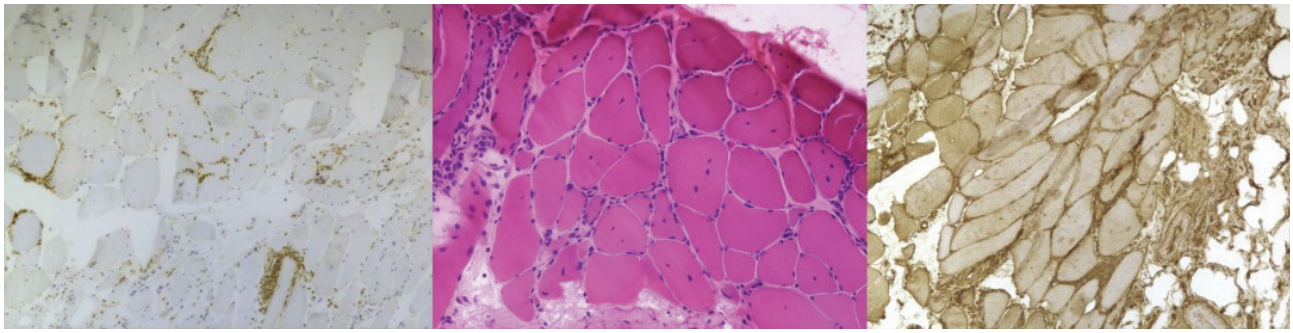

Figure 1. Polymyositis - atrophic fibres and internal nuclei (Middle, HEx20), (Left, CD3x10), (Right, MHC-Ix20)

TABLE 1. Clinical, histologic and final diagnosis

Gender	Clinical diagnosis	Indication for biopsy	Histologic diagnosis	Final diagnosis	IMS before biopsy	Statin before biopsy
F	Suspected inflammatory myopathy	DC	Inflammatory myopathy	Inflammatory myopathy	-	No
F	Sjogren's syndrome	DC	Inflammatory myopathy	Inflammatory myopathy	PDN	No
F	Antysyntethase syndrome	DC	Inflammatory myopathy	Antysyntethase syndrome	CYC	No
M	Dermatomyositis	DC	Inflammatory myopathy	Dermatomyositis	PDN	No
M	SLE	DC	Inflammatory myopathy	Inflammatory myopathy	MTX	No
F	Dermatomyositis	DC	Inflammatory myopathy	Dermatomyositis	PDN	No
F	Dermatomyositis	DC (disease progression)	Inflammatory myopathy/ dermatomyositis	Dermatomyositis	PDN, MTX	No
M	Polymyositis	DC	Inflammatory myopathy/ polymyositis	Polymyositis	-	No
M	Dermatomyositis	DC	Multiple artifacts	Dermatomyositis	PDN, MTX	Yes
F	Suspected inflammatory myopathy	DC	Normal	Normal	-	No
F	Clinically amyopathic dermatomyositis	DC (previous biopsy outside SNS)	Normal	Clinically amyopathic dermatomyositis	MTX	No
F	Steroids myopathy	DC	Normal	Steroids myopathy	-	Yes
F	Fibromyalgia with raised CK and inflammatory markers	DC	Normal	Fibromyalgia	-	No
F	Clinically amyopathic dermatomyositis	DC (atypical cutaneous findings)	Normal	Clinically amyopathic dermatomyositis	PDN, MTX	No
F	Dermatomyositis	DC (previous biopsy outside SNS)	Normal	Dermatomyositis	AZT	Yes
F	Dermatomyositis	DC	Normal	Dermatomyositis	PDN, MTX	No
M	Suspected inflammatory myopathy	DC	Unspecific findings	Normal	-	No
F	Necrotizing myopathy	DC	Unspecific findings	Necrotizing myopathy	PDN, MTX	No

continues on the next page

TABLE 1. Continuation

Gender	Clinical diagnosis	Indication for biopsy	Histologic diagnosis	Final diagnosis	IMS before biopsy	Statin before biopsy
M	Antysyntethase syndrome	DC (previous biopsy outside SNS)	Unspecific findings	Antysyntethase syndrome	-	Yes
F	Suspected inflammatory myopathy	DC	Unspecific findings	Inflammatory myopathy	AZT	No
M	Suspected inflammatory myopathy	DC	Unspecific findings	Normal	PDN	No
M	Suspected inflammatory myopathy	DC	Unspecific findings	Normal	PDN, MTX	No
F	Polymyositis	DC (disease progression)	Unspecific findings	Polymyositis	PDN, MTX	No
F	Suspected inflammatory myopathy	DC	Insufficient material	Myopathy non- IIM	PDN	No
M	Polycythemia with raised CK and positive MSA	DC	Insufficient material	Normal	-	No

Abbreviations – AZT- azathioprine; CYC – cyclosporine; CK – creatine kinase; DC – diagnostic confirmation; F – female; IIM – idiopathic inflammatory myopathy; IMS – immunosuppressant; M – male; MSA – myositis specific antibody; MTX – methotrexate; PDN – prednisolone; SNS – Serviço Nacional de Saúde

tients (n=11), with discrepancies happening more frequently in patients with treated disease/amyopathic forms. Considering the eight patients without immunossupressant treatment, two had an IIM diagnosis (concordant with histological findings). The final diagnosis was changed after the biopsy in 24% (n=6) of patients (Table 1).

The UGMB was generally well-tolerated, with an average Visual Analogical Scale for pain of 4.5/10, concerning the procedure. There were no moderate or severe adverse events. Only one patient (4%) reported a mild long-term adverse event (mild pain), which lasted for twelve weeks after the procedure.

Conclusion: UGMB is a safe method to collect muscle samples. Despite the limitations of this study, namely the small number of biopsies performed, the results are promising. Histological analysis was possible in nearly 90% of the samples, suggesting this is an effective way to collect muscle samples for IIM differential diagnosis. The discrepancy between histological findings and clinical diagnosis suggests that biopsies in patients treated with immunomodulators or clinically amyopathic forms have lower rentability than in untreated patients with muscle weakness.

163 - NINTEDANIB EFFICACY IN INFLAMMATORY RHEUMATIC DISEASE-ASSOCIATED INTERSTITIAL LUNG DISEASES – A MULTICENTER NATIONWIDE STUDY

Augusto Silva[1,2], Ana Martins[3,4], Beatriz Samões[5], Carla Campinho Ferreira[6], C. Pinto Oliveira[7], João Aguiar[1,2], Laura Gago[8], Carlos Costa[9], Ana Catarina Duarte[10], Filipe Araújo[11], Maria João Salvador[12], Natália Melo[13], Joana Leite Silva[6], JE Fonseca[1,2], Maria João Gonçalves[8], Nikita Khmelinskii[1,2], P Pinto[5], Ana Rita Prata[7], Vasco C Romão[1,2], Miguel Bernardes[3,4], Cristina Ponte[1,2], Sofia C Barreira[1,2], Catarina Resende[1,2]

[1]Rheumatology and Bone Metabolic Diseases Department, Centro Hospitalar Lisboa Norte, EPE - Hospital de Santa Maria, Lisbon Academic Medical Centre, Lisboa, Portugal, [2]Serviço de Reumatologia e Doenças Ósseas Metabólicas do Hospital de Santa Maria, Centro Hospitalar Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [3]Serviço de Reumatologia, Centro Hospitalar São João, Porto, Portugal, [4]Faculdade de Medicina da Universidade do Porto, Porto, Portugal, [5]Rheumatology Department, Centro Hospitalar de Vila Nova de Gaia/Espinho, Vila Nova de Gaia, Portugal, [6]Serviço de Reumatologia, Hospital de Braga, Braga, Portugal, [7]Rheumatology Department, Centro Hospitalar do Baixo Vouga, Aveiro, Portugal, [8]Rheumatology Department, Centro Hospitalar de Lisboa Ocidental, Hospital Egas Moniz EPE, Lisboa, Portugal, [9]Serviço de Reumatologia, Centro Hospitalar Trás-os-Montes e Alto Douro, Vila Real, Portugal, [10]Serviço de Reumatologia, Hospital Garcia de Orta, Almada, Portugal, [11]Serviço de Reumatologia, Hospital CUF Cascais, Cascais, Portugal, [12]Rheumatology Department, Centro Hospitalar e Universitário de Coimbra, Coimbra, Portugal, [13]Serviço de Pneumologia, Centro Hospitalar de São João, Porto, Portugal

Background: Interstitial lung disease (ILD) is a severe manifestation of inflammatory rheumatic diseases (IRD). Conventional treatments have shown modest and short-lived success in controlling ILD(1). Nintedanib (NTD) was approved for systemic sclerosis (SSc)-associated ILD and progressive fibrosing ILD(1,2). However, its experience in other IRD-ILD is still scarce.
Objectives: To compare the disease course of patients with IRD-ILD who were treated with NTD to those who had no exposure to NTD.

TABLE 1. Clinical features of the patients with Inflammatory Rheumatic Disease-associated Interstitial Lung Disease

	Treated with NTD, N = 25	Control group, N = 24	p value
Female, n (%)	15 (60.0%)	18 (75.0%)	0.263
Smoking status, n (%) - Never smoked - Smoker - Ex-smoker	12 (48.0%) 6 (24.0%) 7 (28.0%)	13 (54.2%) 2 (8.3%) 9 (37.5%)	0.321
Age at IRD diagnosis, mean±SD (years)	59.2±12.6	57.1±12.5	0.545*
Inflammatory rheumatic disease (IRD), n (%) - Systemic sclerosis - Rheumatoid arthritis - Sjögren's syndrome - Mixed connective tissue disease - Microscopic polyangiitis - Systemic lupus erythematosus - Dermatomyositis - Polymyositis	11 (44.0%) 7 (28.0%) 4 (16.0%) 1 (4.0%) 1 (4.0%) 1 (4.0%) 1 (4.0%) 0 (0.0%)	10 (41.7%) 10 (41.7%) 2 (8.3%) 0 (0.0%) 0 (0.0%) 0 (0.0%) 0 (0.0%) 1 (4.2%)	0.514
Age at ILD diagnosis, mean±SD (years)	62.8±11.2	60.8±10.3	0.186*
Interstitial lung disease (ILD) pattern, n (%) - Usual interstitial pneumonia - Nonspecific interstitial pneumonia - Organizing pneumonia	15 (60.0%) 9 (36.0%) 1 (4.0%)	15 (62.5% 9 (37.5%) 0 (0.0%)	0.613
Time from IRD diagnosis to ILD diagnosis, mean±SD (years)	3.6±7.2	3.7±6.0	0.972
Autoantibodies, n (%) - Antinuclear antibody - Anti-Scl70 - Rheumatoid Factor - Anticyclic-citrullinated protein antibody - Anti-Ro52 - Anticentromere - Anti-RNP - Anti-dsDNA - Anti-Myeloperoxidase - Anti-RNA polymerase III - Anti-PM/Scl75 - Anti-PL12	19 (76%) 5 (20%) 9 (36%) 6 (24%) 4 (16%) 1 (4%) 1 (4%) 1 (4%) 1 (4%) 1 (4%) 1 (4%) 0 (0%)	18 (75%) 10 (41.7%) 10 (41.7%) 10 (41.7%) 3 (12.5%) 1 (4.2%) 1 (4.2%) 0 (0%) 0 (0%) 0 (0%) 0 (0%) 1 (4.2%)	0.618
Glucocorticoids over the 24 months, n (%), and mean dose (mg) - Prednisolone - Deflazacorte	17 (68.0%), 6.4 mg 1 (4.0%), 12 mg	18 (75.0%), 10.6 mg 2 (8.3%), 12 mg	0.560
csDMARD over the 24 months, n (%) - Mycophenolate mofetil/ Mycophenolic acid - Methotrexate - Cyclophosphamide - Azathioprine - Hydroxychloroquine	9 (36.0%) 4 (18.2%) 2 (9.1%) 1 (4.5%) 4 (18.2%)	7 (29.2%) 6 (29.2%) 6 (23.1%) 3 (11.5%) 3 (11.5%)	0.762 0.321 0.138 0.349 1.000

continues on the next page

TABLE 1. Continuation

	Treated with NTD, N = 25	Control group, N = 24	p value
bDMARD in the 24 months (Rituximab), n (%)	10 (40.0%)	7 (29.2%)	0.551
FVC, mean±SD % (n/N) - baseline - 6 months - 12 months - 18 months - 24 months	 66.6±18.6 (24/25) 67.9±17.9 (12/25) 76.9±22.3 (13/25) 73.6±27.2 (5/25) 72.1±23.7 (8/25)	 81.8±17.4 (14,24) 90.2±21.7 (11/24) 85.0±21.1 (16/2) 80.7±18.4 (10/24) 82.5±22.1 (18/24)	 0.018 0.013 0.237 0.557 0.289
FVC variation compared to baseline, mean±SD % (n/N) - 6 months - 12 months - 18 months - 24 months	 2.2±8.8 (12/25) 7.5±14.5 (13/25) 5.6±21.3 (5/25) 1.5±10.9 (8/25)	 4.0±10.5 (9/24) -3.2±11.5 (12/24) -1.3±9.9 (7/24) -2.5±13.1 (7/24)	 0.161 0.052 0.464 0.488
DLCOc SB, mean±SD % (n/N) - baseline - 6 months - 12 months	 36.9±18.1 (17/25) 45.5±15.7 (11/25) 36.9±14.6 (10/25)	 55.6±17.1 (9/24) 55.4±21.8 (11/24) 51.0±18.3 (12/24)	 0.018 0.236 0.063
- 18 months - 24 months	43.3±15.0 (3/25) 32.7±7.1 (6/25)	42.2±12.4 (10/24) 49.3±15.8 (16/24)	0.892 0.023
DLCOc SB variation compared to baseline, mean±SD % (n/N) - 6 months - 12 months - 18 months - 24 months	 11.3±17.1 (9/25) 7.7±14.6 (9/25) 6.7±10.2 (3/25) 7.4±8.0 (5/25)	 -1.4±11.5 (8/24) -2.8±12.6 (7/24) -4.5±14.4 (4/24) -6.1±10.5 (8/24)	 0.181 0.145 0.321 0.024
Radiographic evaluation (1) at 12 months by HRCT, n/N (%) - Improvement - Stabilisation - Deterioration	 0/21 (0%) 16/21 (76.2%) 5/21 (23.8%)	 2/24 (8.3%) 14/24 (58.4%) 8/24 (33.3%)	 0.941
Number of deaths, n (%)	1 (4%)	3 (12.5%)	0.227

Abbreviations: bDMARD - biological disease modifying anti-rheumatic drugs; csDMARD - conventional disease-modifying antirheumatic drugs; DLCOc SB - single-breath diffusing capacity of the lungs for CO; FVC - forced vital capacity; HRCT - High-resolution computed tomography; ILD - Interstitial Lung Disease; IRD - Inflammatory Rheumatic Disease; NTD - Nintedanib; SD - Standard Deviation.
* The Shapiro-Wilk normality test revealed a normal data distribution (t-student test used), in the remaining cases the Mann-Whitney test was used.
(1) Improvement or worsening defined as a change of at least 10% in pulmonary fibrosis by an experienced radiologist.

Methods: A retrospective observational study with patients from ten Portuguese rheumatology centers was conducted. Patients with less than 3 years of IRD-ILD duration who were treated with NTD and a control group, also with an IRD-ILD duration less than 3 years, and no exposure to NTD were included. Patients and data from the control group was related to the period before NTD approval in these diseases. Categorical variables are presented as frequencies, while continuous variables are presented as mean ± standard deviation (range) or median (interquartile range). Chi-square, Mann-Whitney U and t-test were used to assess differences between groups. A linear regression analysis was used to evaluate independent predictors of forced vital capacity (FVC) and single-breath diffusing capacity of the lungs for CO (DLCO) progression.

Results: 25 cases (60% women) and 24 controls (75% female) were included, with a mean age at ILD diagnosis of 62.8±11.2 and 60.8±10.3 years, respectively (Table 1). Baseline features were similar between groups and mean follow-up was 24.2±14.3 months. In both groups, the most frequently underlying IRD was SSc (11/25 vs 10/24). Usual interstitial pneumonia (15/25 and 15/24) and nonspecific interstitial pneumonia (9/25 and 9/24) were the most common ILD patterns in both groups. The most frequently used immunosuppressants were mycophenolate mofetil (MMF; 36.0% vs 29.2%) and rituximab (RTX; 40.0% vs 29.3%). At baseline, pulmonary function tests showed a lower DLCO and FVC in patients who were treated with NTD compared to controls (36.9±18.1% vs 55.6±17.1%, p=0.018 and 66.6±18.6 vs 81.8±17.4, p=0.018; respectively). Median (IQR) treatment duration was 15 (11-36) months. Only 12/25 (48%) patients were treated with NTD for

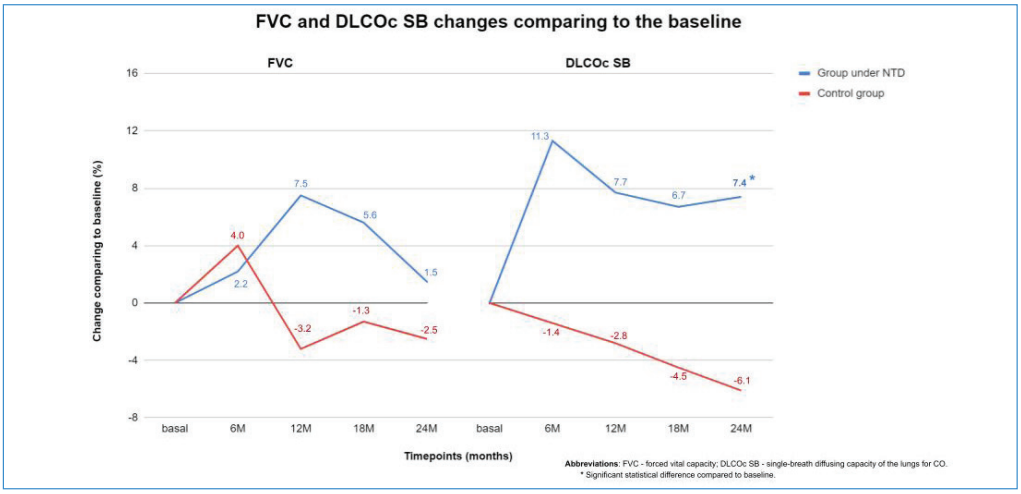

Figure 1. Mean forced vital capacity and single-breath diffusing capacity of the lungs for CO variations up to 24 months of follow-up

at least 24 months (including one deceased patient) and the remaining have not yet completed this follow-up period. There was an improvement in the DLCO variation at 24 months for patients treated with NTD compared to controls (7.4±8.0 vs -6.1±10.5, p=0.024), but no differences were found between groups at 6, 12 and 18 months (p=0.181, p=0.145 and p=0.321, respectively). Regarding FVC variation, there was only a trend for a benefit of NTD at 12 months (7.5±14.5 vs -3.2±11.5, p=0.052), which was not observed at months 6, 18 and 24 (p=0.161, p=0.464 and p=0.488, respectively) (Figure 1). In the multivariate linear regression analysis, adjusted for concomitant use of RTX and MMF, NTD was associated with a positive DLCO variation at 24 months (HR 13.60 CI 95% 2.46-24.76, p=0.022). A significant improvement of the FVC variation was only observed in patients with less than one year of ILD duration (n=13) and at 12 months (10.6±8.0 vs -3.2±11.5, p=0.014).

Conclusion: NTD seems to be a promising and effective therapeutic option for patients with IRD-ILD. In our cohort, treatment with NTD was associated with higher FVC and DLCO improvements after one and two years, respectively. However, the former was only verified in cases of early NTD initiation. Further replication in larger and prospective cohorts is warranted.

174 - HIPOFOSFATASIA DO ADULTO - UMA CAUSA RARA DE DOR MÚSCULO-ESQUELÉTICA CRÓNICA

Catarina S Cortesão[1], Barbara Lobão[1], Gonçalo Boleto[1], Cândida Silva[1], Helena Santos[1]
[1]Rheumatology Department, Instituto Português de Reumatologia, Lisboa, Portugal

Introdução: A hipofosfatasia congénita (HC) é uma doença genética rara do metabolismo ósseo, causada por defeitos na síntese ou função da isoenzima fosfatase alcalina (FAL) essencial na mineralização óssea e uma das causas raras de osteomalácia no adulto. A osteomalácia é o estadio clínico e histológico final da inadequada mineralização da matriz óssea, promovida por diferentes mecanismos patológicos.

A ação da FAL no tecido ósseo relaciona-se com a conversão do pirofosfato inorgânico em fósforo inorgânico, tornando-o disponível para formação dos cristais de hidroxiapatite, integrados pelos osteócitos no processo de mineralização óssea. Mais de 400 mutações foram já identificadas no gene ALPL que codifica a FAL, condicionando grande variabilidade clínica na apresentação da doença(1).

Caso clínico: Homem, 31 anos, com antecedentes de psoríase gutata, que iniciou há 3 anos um quadro de parestesias das mãos e pés e há cerca de 1 ano lombalgias e cervicalgias de ritmo mecânico, artralgias dos punhos, mialgias e fadiga. Ao exame objetivo não apresentava artrite, lesões cutâneas ou anomalias na dentição. Como medicação habitual o doente fazia AINEs em SOS. Na avaliação analítica destacava-se FAL 13U/L (ref:40-150U/L). Em avaliação subsequente mantém FAL baixa, fosfato inorgânico, cálcio sérico e vitamina D normais e marcadores de fase aguda negativos. RM lombar e sacro-ilíacas sem evidência de espondilartrite. Densidade mineral óssea normal para a faixa etária. Peranto o caso clínico e FAL baixa, o diagnóstico de hipofosfatasia do adulto foi considerado. Este foi verificado após teste genético para mutações no gene ALPL, que revelou a variante c.809G>A em heterozigotia, confirmando o diagnóstico em estudo.

Discussão: No contexto de um homem jovem com um quadro de dor musculo-esquelética crónica difu-

sa, sem evidência de espondilartrite e após exclusão de neoplasia, as formas clínicas congénitas e adquiridas com impacto na mineralização óssea têm de ser consideradas no diagnóstico diferencial. Neste caso, o défice de FAL sérica teve um papel essencial para encaminhar o estudo para a hipótese diagnóstica de HC, confirmada com estudo genético(2). As variantes genéticas na HC condicionam manifestações clínicas heterogéneas, com hipomineralização como denominador comum. As formas heterozigóticas simples, como neste caso, parecem corresponder a apresentações clínicas mais inespecíficas e já na idade adulta. Outras formas genéticas mais complexas formam um espectro de apresentações mais graves de doença.

A asfotase alfa, é atualmente a única terapêutica enzimática de substituição da ação da FAL e está indicada para tratar as manifestações ósseas das formas de apresentação de HC infanto-juvenil, tendo demonstrado bons perfis de eficácia e segurança. A administração de teriparatide off label no adulto tem sido reportada apenas em casos clínicos e com eficácia limitada(3). Por outro lado, os bifosfonatos e o denosumab estão contraindicados e a suplementação com vitamina D e cálcio deve ser ponderada caso a caso.

Conclusão: A apresentação de HC no adulto, ao contrário da criança, pode ser apenas de dor músculo-esquelética difusa. Neste contexto, a HC, apesar de rara, deve fazer parte do diagnóstico diferencial, principalmente se na avaliação analítica inicial a quantificação da FAL seja baixa. Nesta situação, se a determinação subsequente da FAL se mantiver negativa, pede-se teste genético para confirmar o diagnóstico.

REFERÊNCIAS

1. Cianferotti (2022) Int.J.Mol.Sci.23,14896; 2. Feurstein et al (2022) OrphanetJ.Rare.Dis,17,435; 3. Camacho et al (2016) Endocr.Pract.22,941

183 - ADIPOSITY TRAJECTORY IN CHILDHOOD AND PAIN EXPERIENCES IN EARLY ADOLESCENCE

Francisco Fernandes[1], Nare Navasardyan[1], Makram Talih[1], Catarina Pires[1], Raquel Lucas[1]

[1]Departamento de Epidemiologia, ISPUP, Porto, Portugal

Background: Pain and obesity present serious public health concerns. Pain conditions are associated with several adverse health outcomes, being responsible for decreased quality-of-life, increased use of health services and out-of-pocket health expenses.

There are different mechanisms that link obesity to pain. On the one hand, obesity is associated with a pro-inflammatory state which may produce a hyperalgesic state, on the other hand, increased mechanical stresses on the body in obesity are believed to result in an increased risk of musculoskeletal pain.

Even though evidence shows an association between obesity and a higher prevalence of pain syndromes in adulthood, little is known regarding adiposity and pain experienced during childhood and adolescence.

We aim to assess the association between adiposity trajectory during childhood and pain experiences at age 13 in a population-based birth cohort.

Methods: We used data from Generation 21, a population-based birth cohort established in 2005/6, in all public maternities in the Porto metropolitan area.

We focused on the individual trajectory of body fat percentage, measured via bioimpedance, at ages 4, 7, 10, and 13. We obtained individual trajectories by extracting the random effects from mixed-effects models, considering both the intercept and slope.

At age 13, we assessed the outcomes of chronic pain (pain persisting for more than 3 months) and multisite pain (pain experienced in two or more sites) using the Luebeck pain-screening questionnaire. We used logistic regression models to evaluate the association between the body fat percentage trajectory and the outcomes, stratified by sex at birth.

Results: We included a total of 2211 girls and 2354 boys. Among girls, we observed a significant association between the individual trajectory of adiposity and chronic and multisite pain at the age of 13. We have found that a higher initial body fat percentage was associated with increased odds of experiencing chronic pain at age 13 (odds ratio [OR] 1.11, 95% confidence interval [CI] 1.00 - 1.22). Additionally, a steeper slope of variation in body fat percentage over time was also associated with higher odds of chronic pain at age 13

	Girls						Boys					
	Chronic Pain at age 13			Multisite Pain at age 13			Chronic Pain at age 13			Multisite Pain at age 13		
Fat Mass Percentage	*Odds Ratios*	*CI*	*p*	*Odds Ratios*	*CI*	*p*	*Odds Ratios*	*CI*	*p*	*Odds Ratios*	*CI*	*p*
Initial	1.11	1.00 – 1.22	**0.049**	1.03	0.94 – 1.13	0.504	1.02	0.89 – 1.17	0.770	1.05	0.94 – 1.18	0.377
Slope	1.15	1.05 – 1.26	**0.002**	1.12	1.03 – 1.21	**0.007**	1.01	0.89 – 1.15	0.840	1.02	0.91 – 1.13	0.777

Figure 1. Association between body fat percentage trajectories and chronic and multisite pain at age 13, stratified by sex at birth.

(OR 1.15, 95% CI 1.05 - 1.26).
For multisite pain, we observed that a greater slope of variation in body fat percentage over time was associated with an increased likelihood of experiencing pain in multiple sites at age 13 (OR 1.12, 95% CI 1.03 - 1.21). In contrast, among boys, we did not find any statistically significant associations between the adiposity trajectory and the occurrence of chronic pain or multisite pain.
Conclusion: Our study suggests a positive association between adiposity trajectory during childhood and pain experiences in early adolescence, particularly among girls. Higher body fat percentage and steeper increase in adiposity over time were associated with higher odds of chronic pain and multisite pain at age 13. These findings underline the importance of considering adiposity as a potential risk factor for pain conditions during this critical developmental stage.

203 - COMPARISON OF CLINICAL FEATURES, DISEASE ACTIVITY AND SAFETY BETWEEN ELDERLY AND NON-ELDERLY PATIENTS WITH PSORIATIC ARTHRITIS TREATED WITH FIRST BIOLOGIC AGENT: A MULTICENTRIC RETROSPECTIVE COHORT STUDY OVER TWO YEARS

Daniela Oliveira[1,2], Teresa Martins-Rocha[1,3], Rita Amaral[2,4,5,6], Ana Martins[1], Elsa Vieira-Sousa[7,8], Carolina Ôchoa-Matos[7], Susana Fernandes[9], Beatriz Mendonça[9], José Tavares-Costa[10], Maria Pontes Ferreira[10], Ana Catarina Moniz[11], Ana Chícharo[11,12], Ana Catarina Duarte[13], Catarina Abreu[13], Patrícia Nero[14], João Rovisco[15], Pedro Teixeira[16], Marcos Cerqueira[17], Graça Sequeira[12], Paula Valente[18], Patrícia Pinto[19], Teresa Nóvoa[20], Lígia Silva[21], Carlos Vaz[1,2,3], Lúcia Costa[1] and Miguel Bernardes[1,3]
[1]Rheumatology Department, Centro Hospitalar Universitário S. João, Porto, Portugal; [2]Center for Health Technology and Services Research (CINTESIS), Faculty of Medicine, University of Porto, Porto, Portugal; [3]Medicine Department, Faculty of Medicine, University of Porto, Porto, Portugal; [4]Department of Community Medicine, Information and Health Decision Sciences (MEDCIDS), Faculty of Medicine, University of Porto, Porto, Portugal; [5]Department of Cardiovascular and Respiratory Sciences, Porto Health School, Polytechnic Institute of Porto, Porto, Portugal; [6]Department of Women's and Children's Health, Paediatric Research, Uppsala University, Stockholm, Sweden; [7]Rheumatology Department, Centro Hospitalar Universitário Lisboa Norte; [8]Instituto de Medicina Molecular- João Lobo Antunes Centro Académico de Medicina de Lisboa, Lisboa, Portugal; [9]Instituto Português de Reumatologia, Lisboa, Portugal; [10]Rheumatology Department, Unidade de Saúde Local do Alto Minho, Ponte de Lima, Portugal; [11]Rheumatology Department, Centro Hospitalar Lisboa Ocidental, Lisboa, Portugal; [12]Rheumatology Department, Centro Hospitalar Universitário do Algarve, Faro, Portugal; [13]Rheumatology Department, Hospital Garcia de Orta, Almada, Portugal; [14]Hospital CUF Descobertas, Lisboa, Portugal; [15]Rheumatology Department, Centro Hospitalar Universitário de Coimbra, Coimbra, Portugal; [16]Rheumatology Department, Centro Hospitalar do Baixo Vouga, Aveiro, Portugal; [17]Rheumatology Department, Hospital de Braga, Braga, Portugal; [18]Rheumatology Department, Centro Hospitalar de Entre Douro e Vouga, Santa Maria da Feira, Portugal; [19]Rheumatology Department, Centro Hospitalar Vila Nova de Gaia/Espinho, Gaia, Portugal; [20]Rheumatology Department, Hospital do Divino Espírito Santo, Ponta Delgada, Portugal; [21]Rheumatology Department, Centro Hospitalar Trás-os-Montes e Alto Douro, Vila Real, Portugal

Introduction: The number of elderly (EL) patients with psoriatic arthritis (PsA) has gradually increased due to improved healthcare and increased life expectancy for these patients. Biologic disease-modifying antirheumatic drugs (bDMARD), represents an important challenge in EL PsA patients due to the increased frequency of comorbidities and risk of drug interactions. Moreover, these patients are commonly excluded from large clinical trials due to their higher risk of adverse events (AEs) and hence, previous research report that bDMARDs are used less frequently in clinical practice in this population. Thus, little is known about the effectiveness and safety of bDMARD in EL PsA patients. Therefore, this study aimed to assess clinical features, disease activity, clinical response and AEs in PsA EL patients compared with non-elderly (N-EL) patients under bDMARD therapy. Methods: A multicentric observational retrospective cohort study was conducted involving patients with PsA (axial and peripheral disease) treated with bDMARD. Patients diagnosed with PsA, according to CASPAR criteria, treated with the first bDMARD and registered on the Rheumatic Diseases Portuguese Register (Reuma.pt) were included. Patients were classified into two groups: patients with age of first bDMARD from 18 to 64 years old (N-EL) and patients with age of first bDMARD ≥65 years old (EL). For each age group, sociodemographic data, laboratory findings and disease activity scores were obtained at baseline, 12 and 24 months (M). Disease activity scores, clinical response, switching to another bDMARD and AEs were obtained at 12 and 24 M. Continuous variables are presented with mean, standard deviation (SD), median and interquartile range (IQR). Categorical variables are presented with absolute and relative frequencies. To examine the differences between groups, independent samples t-tests, Mann-Whitney U-tests and chi-square

tests were performed. Results: A total of 1255 PsA patients (mean age of 48±12.1 years old, 50.4% female, 9.0% EL patients) under bDMARD were included. Sociodemographic and clinical characteristics, disease activity and function scores, clinical response, switch rate and safety features are described in Table 1. At baseline, the EL group had a lower prevalence of smokers (p=0.039), higher BMI (p<0.036), more comorbidities and higher prevalence of dactylitis (p=0.005) compared to the N-EL group. Furthermore, EL patients had a later age of PsA onset and diagnosis (p<0.001 for both). EL PsA patients had significantly lower activity scores at baseline and 12M compared to N-EL patients. There were no differences between age groups regarding therapeutic options at baseline, disease activity scores at 24M, clinical response, overall switch rate and AEs over time. Conclusion: These findings showed that in EL patients with PsA under bDMARD the activity scores were lower at baseline and 12M. As well and significant for clinical practice is the fact that clinical response, switch rate and the number of patients with AEs were similar between age groups. Further research is needed to assess the true benefit-risk balance of bDMARD therapy in EL PsA patients in order to explore a more tailored treatment strategy.

206 - CORTICOSTEROID IRRIGATION OF PAROTID GLANDS IS AN EFFECTIVE AND WELL-TOLERATED TREATMENT FOR ORAL DRYNESS IN PATIENTS WITH SJÖGREN'S AND NON-SJÖGREN'S SICCA SYNDROME: A LONGITUDINAL STUDY

Gonçalo Cunha Coutinho[1, 2], Vasco C Romão[3, 4], Rita Simão[1, 2], José Coutinho[1, 2], JE Fonseca[3, 4], Francisco Salvado[1, 2]
[1]Serviço de Estomatologia, Hospital de Santa Maria, CHLN, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [2]Clínica Universitária de Estomatologia, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, Lisboa, Portugal, [3]Serviço de Reumatologia e Doenças Ósseas Metabólicas, Hospital de Santa Maria, Centro Hospitalar e Universitário de Lisboa Norte, Centro Académico de Medicina de Lisboa, Lisboa, Portugal, [4]Unidade de Investigação em Reumatologia, Instituto de Medicina Molecular, Faculdade de Medicina, Universidade de Lisboa, Centro Académico de Medicina de Lisboa, Lisboa, Portugal

Introduction: The best recent evidence supports the application of sialendoscopy in the treatment of Sjögren's syndrome (SS) salivary disease. However, further studies are needed to validate this approach and provide a consistent therapy protocol, especially regarding the complementary benefit of intraductal irrigation with corticosteroids.

Objective: To assess the effect of sialendoscopy associated with corticosteroid irrigation of parotid glands on clinical oral dryness and xerostomia in patients with a suspicion of SS.

Methods: We included patients who were seen at our SS Multidisciplinary Clinic of the Stomatology, Rheumatology and Ophthalmology Departments, for suspicion of SS, who were submitted to a protocol of irrigation of the parotid glands between 2020 and 2022. Briefly, under local anesthesia the ductal orifices of both parotid glands were dilated and on the first session, sialendoscopy with washing of mucous material and dilation of strictures was performed. On subsequent sessions, based on patient demand, 1ml of 2mg/ml prednisolone was injected through a sialography catheter for 2 minutes. We collected demographic and baseline clinical characteristics, including unstimulated whole salivary flow (UWSF), Clinical Oral Dryness Score (CODS), Xerostomia Inventory (XI), EULAR SS Patient Reported Index (ESSPRI), EULAR SS Disease Activity Index (ESSDAI), Salivary Gland Ultrasonography (SGUS), Salivary Gland Biopsy, anti-SSA and anti-SSB. CODS and XI were evaluated after sialendoscopy as measures of procedure efficacy. Tolerability (visual analogue scale [VAS] 0-10, with 10 as very well tolerated) and safety were also assessed.

Results: A total of 17 patients were submitted to the parotid irrigation protocol, two of whom were lost to follow-up and two had missing post-procedure data. 13 female patients with mean age of 67.3±9.9years (range, 50–83 years) were included for analysis, 9 of whom with SS (Table 1). A total of 54 procedures of sialendoscopy/ irrigation were performed, for a mean of 5.2±2.5 procedures per patient (range 1-8). The mean interval between irrigations was 6.2±5.2 weeks. The procedure was well tolerated with a mean VAS of 8.4±1.0 and there were no reported adverse events. The majority of patients had an improvement in objective (CODS, n=9, 69.2%) and subjective (XI, n=10, 76.9%) measures of oral dryness. This corresponded to a significant decrease of CODS (1,15±0.32, p=0.002) and XI (5,62±2.16, p=0.012) after the irrigation protocol. More severe baseline xerostomia translated by higher baseline XI was correlated with greater decreases of oral dryness assessed by CODS (Pearson r=-0.641, p=0.009) and XI (Pearson r=-0.598, p=0.016). Baseline UWSF was not associated with post-procedure xerostomia variation. A higher number of irrigations was associated with a greater decrease of XI (Pearson r=-0.525, p=0.033)., but not CODS (Pearson r=-0.285, p=0.173). Patients with lower SGUS scores underwent more irrigations (Pearson r=-0.519, p=0.035).

Conclusion: Corticosteroid irrigation of the parotid glands resulted in a significant decrease in subjective complaints and clinical manifestations of dry mouth.

TABLE 1. Clinical and demographic characteristics of the study population

N=13	Mean ± SD or n (%)
Female sex, n (%)	13 (100%)
Age (years)	67.3 ± 9.9
Diagnosis	
Sjögren's syndrome	9 (69.2%)
Non-Sjögren's sicca syndrome	4 (30.8 %)
Anti-SSA	7 (53.8%)
Anti-SSB	5 (38.5%)
Positive salivary gland biopsy	4 (36.4%)
Salivary gland ultrasound score[1]	2.31 ± 0.95
UWSF (ml/15min)[2]	3.07 ± 1.33
ESSPRI (all domains) [2]	6.67 ± 1.43
ESSPRI dryness[2]	7.08 ± 2.57
ESS[2]	7.44 ± 1.92
ESSDAI[2]	3.38 ± 4.03
XI	
- baseline	43.46 ± 7.28
- final	37.85 ± 6.78
CODS	
- baseline	6.15 ± 1.52
- final	5.00 ± 1.41
Number of irrigations	5.15 ± 2.48
Tolerability (VAS 1-10)	8.32 ± 0.73

[1]according to Cornec et al
[2]at baseline

A higher symptom burden and the number of parotid irrigations were associated with greater improvement in measures of oral dryness. Patients with less parenchymal changes as evaluated by SGUS sought more parotid irrigation sessions, possibly suggesting a higher potential for improvement.

207 - SUBTRATAMENTO DAS FRATURAS DE FRAGILIDADA DA EXTREMIDADE PROXIMAL DO FÉMUR NOS CUIDADOS DE SAÚDE PRIMÁRIOS: PERSPECTIVA NACIONAL DE 3 ANOS

Mariana Castro Guimarães[1], Vítor Trindade Pedrosa[2], Beatriz Oliveira Pinto[3], Raquel Batista Leite[3], Filipe Araújo[4]
[1]ARSLVT - ACES Cascais - USF KosmUS, Cascais, Portugal, [2]Cascais - ARSLVT - ACES Cascais - USF São João do Estoril, Cascais, Portugal, [3]Cascais - ARSLVT - ACES Cascais - UCSP Parede, Cascais, Portugal, [4]Hospital Ortopédico de Sant 'Ana, Cascais, Portugal

Objetivos: O objetivo deste estudo foi determinar a taxa de tratamento farmacológico na sequência de fraturas da Extremidade Proximal do Fémur (EPF) no contexto dos Cuidados de Saúde Primários (CSP) em Portugal de 2020 a 2022, comparativamente com o período homólogo de 2018 e 2020 e caracterizar os padrões de prescrição de medicamentos anti-osteoporóticos durante este período.
Materiais e métodos: Foi realizado um estudo observacional, transversal e retrospectivo utilizando dados da base de dados do Bilhete de Identidade - Cuidados de Saúde Primários (BI-CSP). O BI-CSP é uma base de dados nacional de cuidados de saúde primários (excepto Madeira e Açores) que apresenta informação demográfica, indicadores de saúde, prescrição de medicamentos e gestão e organização de unidades de cuidados de saúde primários. Dados agrupados e anónimos em qualquer variável pré-especificada durante um período de tempo (por exemplo, diagnóstico ou tratamento) podem ser obtidos por meio da aplicação de filtros. A ferramenta de classificação usada é a Classificação Internacional de Cuidados Primários, 2ª Edição. Incluímos no nosso estudo todos os indivíduos inscritos nos CSP de Portugal que sofreram uma fractura da anca após os 50 anos. As taxas de tratamento foram determinadas considerando qualquer prescrição de medicamento antiosteoporótico para esses pacientes nos CSP, de 2018 a 2022. Também foi avaliada a frequência de prescrição para cada tipo de medicamento em cada ano. Todos os resultados de interesse foram extraídos para uma folha de extração de dados criada para esse fim. Estatísticas descritivas foram geradas usando o Microsoft Office Excel®2016. Trata-se de um prolongamento de um estudo cujo protocolo de investigação foi aprovado pela Comissão de Ética para a Saúde da ARS LVT em 2021.
Resultados: Identificamos um total de 48.532 fraturas da EPF em Portugal entre 2020 e 2022, 36.384 (74,9%) em pacientes do sexo feminino. A maioria das fraturas da anca foi registada nas Administrações Regionais de Saúde (ARS) do Norte - (19076) e Lisboa e Vale do Tejo - (15559). Apenas 7.391 (15,2%) pacientes foram tratados com terapias anti-osteoporóticas após fratura da EPF, um ligeiro aumento face ao estudo inicial de 2018 a 2020 (14,0%). A ARS com maior taxa de tratamento foi o Algarve (17,6%) e a mais baixa o Alentejo (12,2%). Os padrões de prescrição nacionais e regionais são apresentados na Tabela 1. Os bifosfonatos continuam a ser as terapias mais utilizadas, principalmente o ácido alendrónico (58,1%), o ácido ibandrónico (18,1%) e o ácido zoledrónico (8,0%) embora tenham tendência decrescente, com o Denosumab a aumentar (de 5.1% para 11.9%).
Na Tabela 1 apresentamos o padrão de prescrição na fratura da EPF nos Cuidados de Saúde Primários entre

TABELA 1. Padrão de prescrição na fratura da extremidade proximal do fémur nos Cuidados de Saúde Primários

	AA (%)	AI (%)	AZ (%)	C (%)	D (%)	R (%)	RS (%)	RE (%)	T (%)	Total (%)
N	1502 (56,2%)	535 (20,0%)	245 (9,2%)	0 (0%)	266 (10,0%)	30 (1,1%)	68 (2,5%)	0 (0%)	27 (1,0%)	2673
C	901 (52,6%)	227 (14,2%)	98 (6,1%)	0 (0%)	344 (21,4%)	9 (0,6%)	19 (1,2%)	0 (0%)	6 (0,4%)	1604
LVT	1521 (61,7%)	427 (17,3%)	171 (6,9%)	0 (0%)	243 (9,9%)	14 (0,6%)	55 (2,2%)	0 (0%)	35 (1,4%)	2466
ALT	163 58,2%)	81 (28,9%)	12 (4,3%)	0 (0%)	14 (5,0%)	2 (0,7%)	5 (1,8%)	0 (0%)	3 (1,1%)	280
ALG	210 (57,1%)	70 (19,0%)	65 (17,7%)	0 (0%)	15 (4,1%)	1 (0,3%)	4 (1,1%)	0 (0%)	3 (0,8%)	368
Total (n)	4297 (58,1%)	1340 (18,1%)	591 (8,0%)	0 (0%)	882 (11,9%)	56 (0,8%)	151 (2,0%)	0 (0%)	74 (1,0%	7391

Administração Regional de Saúde: N - Norte; C - Centro; LVT - Lisboa e Vale do Tejo; ALT - Alentejo; ALG - Algarve. Terapêuticas: AA - Ácido Alendrónico; AI - Ácido Ibandrónico; AZ - Ácido Zoledrónico; C - Calcitonina de salmão; D - Denosumab; R - Raloxifeno; RE - Ranelato de estrôncio; RS - Risedronato de Sódio

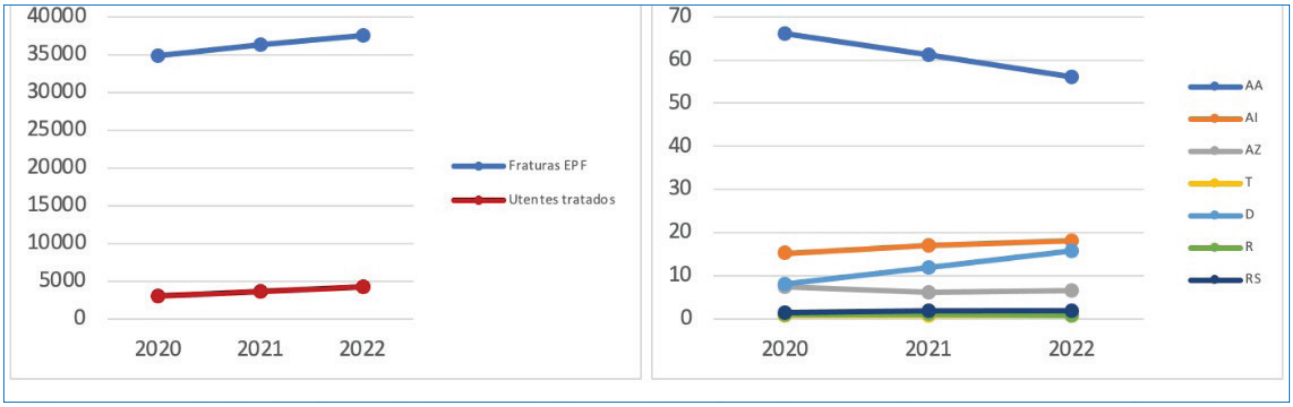

Figura 1. A) Evolução do número de fraturas do fémur codificadas e da prescrição com anti-osteoporóticos de 2020 a 2022. B) Evolução da taxa de prescrição de cada fármaco de 2020 a 2022.
AA – Ácido Alendrónico; AI Ácido Ibandrónico; AZ – Ácido Zolendrónico; C – Calcitonina de salmão; D – Denosumab; R – Raloxifeno; RE – Ralenato de estrôncio; RS – Risedronato de sódio.

2020 e 2022.
No Gráfico 1A podemos observar a evolução do número de fraturas do fémur codificadas e do seu tratamento de 2020 a 2022 e no 1B a evolução da taxa de prescrição de cada fármaco no mesmo período.
Conclusões: Apenas uma minoria dos doentes recebeu tratamento anti-osteoporótico na sequência de fracturas da anca por fragilidade nos Cuidados de Saúde Primários em Portugal. As terapias mais frequentemente utilizadas continuam a ser os bifosfonatos, nomeadamente o ácido alendrónico.
Nos últimos 3 anos a taxa de prescrição tem vindo a aumentar, mas ainda com um crescimento pouco marcado. Em relação aos fármacos utilizados, verificamos que a taxa de prescrição do Denosumab tem aumentado ao longo dos anos, contrariamente à do ácido alendrónico.

240 - INFLAMMATORY POLYARTHRALGIA WITH VERY HIGH TITTER OF RHEUMATOID FACTOR – IS IT RHEUMATOID ARTHRITIS?

Fernando Albuquerque[1], Marcelo Neto[1], Maria João Cadório[1], João Alexandre Oliveira[1], Ana Isabel Maduro[1], André Saraiva[1], Cátia Duarte[1]
[1]Rheumatology Department, Centro Hospitalar Universitário de Coimbra, Coimbra, Portugal

Introduction: The combination of chronic polyarthritis and high titter of rheumatoid factor is very suggestive of rheumatoid arthritis. However, there are many other diseases that may have similar presentation, so a careful clinical assessment is essential.
Clinical Case: We report the case of a 45-year-old

female that had been suffering for 2 months of inflammatory polyarthralgia, with a migratory pattern, affecting the knees, wrists, hands and feet. At clinical examination, there was one swollen joint (5º right PIP) and most of PIPs were painful. Additionally, she referred a significant weight lost and anorexia and a past medical history significant for asthma and allergic rhinitis. She also had an history of recurrent bacterial pneumonias a few years before.

Blood tests revealed a mild normocytic/normochromic anaemia, thrombocytosis, high level of ESR (43 mm/h) and CRP (1,98 mg/dL), a negative anti-CCP and a high titter of rheumatoid factor (445 IU/mL). A thoracic radiography showed a cavitated nodule in the left lung.

Thorax CT scan demonstrated a lung abscess. Sputum cultures were positive for S. aureus while blood cultures were negative. At this point antibiogram-guided antibiotherapy was started, with the addition of metronidazole.

Following the start of this treatment, the lung abscess diminished significantly in size and acute phase reaction normalized completely, but the arthritis persisted. Antibiotherapy was stopped after 4 weeks. 1 month after discharge, a new thorax CT scan revealed 3 nodules in the left lung (Figure 1), and acute phase reactants were once again raised. She also reported dysesthesic pain on her feet. Culture of bronchoalveolar lavage revealed Aspergillus fumigatus. Testing for ANCA revealed a strongly positive c-ANCA and a positive anti-PR3 (17 IU/mL). A transthoracic biopsy was performed, revealing phenomena of endotelitis compatible with Wegener Granulomatosis. A diagnosis of Granulomatosis with polyangiitis was established. After the treatment of the superinfected nodules with guided therapy, Methotrexate was started, with resolution of her symptoms and normalization of acute phase reactants.

Discussion: Rheumatoid Factor is a useful tool when evaluating polyarthritis, but despite having a good specificity for Rheumatoid Arthritis (around 85%), the clinician must always perform a complete anamnesis and physical exam, as well as excluding other possible mimics.

The Rheumatoid Factor is present in up to 40% of ANCA associated vasculitis, being more associated with chest, renal and nervous system involvement as well as higher serologic inflammatory markers.

Therefore, it is important to be aware of the possibility of primary vasculitis and other aetiologies, when assessing a patient with inflammatory polyarthralgia and high titter of rheumatoid factor.

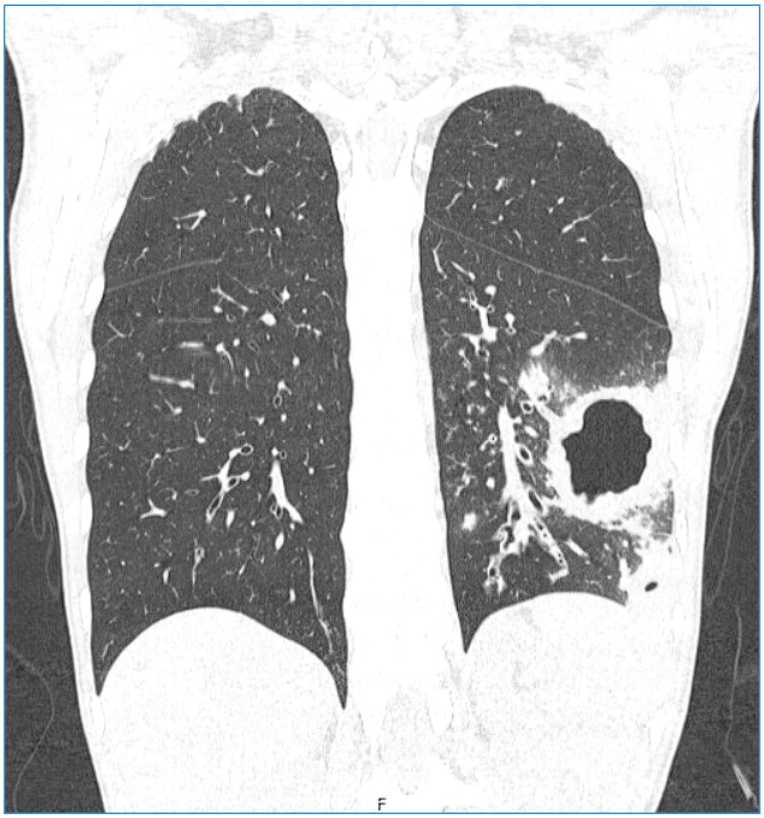

Figure 1. CT scan showing 3 new cavitated nodules 1 month after completion of antibiogram-guided antibiotherapy. The larger lesion was biopsed, with pathological fundungs compatible with granulomatosis with polyangiitis.

Table 1. Clinical characteristics, disease activity scores and function at baseline and following 12 and 24 months of bDMARD treatment.

Variables	Total (N=1255)	Elderly patients (n=113)	Non-Elderly patients (n=1142)	p_{value}
Sociodemographic characteristics				
Female, n (%)	632(50.4)	66(58.4)	566(49.6)	NS
Caucasian, n (%), n=881	865(98.2)	70(97.2)	785(97)	NS
Current smoker, n (%), n=826	145(17.6)	6(8.6)	139(18.4)	0.039
Current alcohol consumer, n (%), n=928	148(15.9)	16(19.5)	132(15.6)	NS
BMI (Kg/m^2), median (IQR), n=718	27.9(4.8)	28.9(5.2)	27.2(5.9)	0.036
Comorbidities, n (%), n=922				
Hypertension	244(26.5)	41(53.2)	203(24.0)	<0.001
Hypercholesterolemia	43(4.7)	9(11.7)	34(4.0)	0.007*
Diabetes Mellitus	85(9.2)	16(20.8)	69(8.2)	<0.001
Cardiovascular disease	35(3.8)	10(13.0)	25(3.0)	<0.001*
Involvement, n (%), n=1058				
Peripheral	887(83.8)	78(88.6)	809(83.4)	NS
Axial	171(16.2)	10(11.4)	161(16.6)	
Disease pattern, n (%), n=1058				
Symmetric polyarthritis	555(52.5)	59(67.0)	496(51.1)	0.042
Asymmetric oligoarthritis	274(25.9)	15(17.0)	259(16.7)	
Axial pattern	171(16.2)	10(11.4)	161(16.6)	
Predominant distal interphalangeal joint	45(4.3)	2(2.3)	43(4.4)	
Mutilans arthritis	13(1.2)	2(2.3)	11(1.1)	
Disease characteristics				
Age of symptoms onset, mean±SD, n=1048	38.6±12.5	55.6±11.5	37.1±11.4	<0.001
Age at diagnosis, mean±SD, n=1046	42.1±12.2	60.4±10.0	40.3±10.9	<0.001
Disease duration until first bDMARD, years, median (IQR), n=1048	6.9(9.9)	10.3(16.8)	6.7(9.7)	<0.001
HLA-B27, n (%), n=999	123(12.3)	7(8.1)	116(12.7)	NS
Extra-articular manifestations, n (%)	694(55.3)	56(49.6)	638(55.9)	NS
Skin psoriasis (ever)	651(51.9)	52(46.0)	599(52.5)	NS
Nail psoriasis (ever)	208(16.6)	15(13.3)	193(16.9)	NS
Enthesitis (baseline)	302(24.1)	20(17.7)	282(24.7)	NS
Dactylitis (ever)	222(17.7)	9(8.0)	213(18.7)	0.005
Uveitis (ever)	39(3.1)	6(5.3)	33(2.9)	NS
Inflammatory bowel disease	4(0.3)	0	4(0.4)	-
Treatment at baseline, n (%)				
Glucocorticoid	457(36.4)	48(42.5)	409(35.8)	NS
NSAIDs	382(30.4)	36(31.9)	346(30.3)	NS
cDMARD	895(71.3)	83(73.5)	812(71.1)	NS
bDMARD				
TNFi	1144(91.2)	99(87.6)	1045(91.5)	NS
Anti IL12/23	32(2.5)	3(2.7)	29(2.5)	
Anti-IL17	78(6.2)	10(8.8)	68(6.0)	
Anti-CD20 (rituximab)	1(0.1)	1(0.9)	0	
Disease activity (peripheral and axial) and function				
Baseline				
Tender joints 68, median (IQR), n=787	6.0(10.0)	3.0(7.0)	6.0(10.6)	0.001

continues on the next page

Table 1. Continuation.

Variables	Total (N=1255)	Elderly patients (n=113)	Non-Elderly patients (n=1142)	p_{value}
Swollen joints 68, median (IQR), n=778	3.0(6.0)	3.0(4.0)	4.0(6.0)	NS
ESR (mm/1st hour), median (IQR), n=772	22.0(32.0)	25.0(32.5)	22.0(32.0)	NS
CRP (mg/dL), median (IQR), n=754	0.9(1.7)	1.0(2.0)	0.9(1.7)	NS
Patient VAS, mean±SD, n=702	61.1±23.1	51.6±21.7	61.9±23.1	0.002
Physician VAS, mean±SD, n=650	49.5±20.6	47.0±19.0	49.6±20.7	NS
Pain VAS, mean±SD, n=563	59.5±23.9	55.6±23.1	59.7±23.9	NS
DAS28 4V CRP, mean±SD, n=552	4.3±1.2	3.9±1.2	4.3±1.3	NS
CDAI, median (IQR), n=544	18.0(14.1)	15.6(8.3)	18.5(14.1)	0.024
SDAI, median (IQR), n=519	20.1(15.0)	16.9(9.9)	20.3(14.8)	NS
DAPSA, median (IQR), n=497	25.2(1.8)	20.6(9.7)	25.5(16.9)	0.029
BASDAI, mean±SD, n=295	6.2±1.9	5.2±2.6	6.2±1.9	NS
ASDAS CRP, mean±SD, n=280	3.6±1.0	3.3±1.3	3.6±1.0	NS
BASFI, median (IQR), n=272	5.7(3.9)	1.8(6.6)	5.7(3.8)	NS
HAQ, median (IQR), n=463	1.0(1.0)	0.9(1.2)	1.0(1.0)	NS
12 Months				
Tender joints 68, median (IQR), n=593	0(2.0)	0(0.8)	0(2.0)	0.036
Swollen joints 68, median (IQR), n=590	0(1.0)	0(0)	0(1.0)	NS
ESR (mm/1st hour), median (IQR), n=576	10(16.0)	5.0(14.0)	10.0(16.0)	0.005
CRP (mg/dL), median (IQR), n=567	0.3(0.5)	0.2(0.4)	0.3(0.5)	0.001
Patient VAS, mean±SD, n=516	30.9±26.2	16.2±20.3	31.7±26.3	<0.001
Physician VAS, mean±SD, n=464	15.5±17.8	11.0±14.2	15.6±16.9	NS
Pain VAS, mean±SD, n=420	29.5±26.2	17.4±21.4	30.4±26.3	0.004
DAS28 4V CRP, mean±SD, n=398	2.3±1.1	1.7±0.9	2.4±1.1	0.002
ΔDAS28, mean±SD, n=306	2.3±1.4	3.1±1.5	2.1±1.5	0.030
CDAI, median (IQR), n=394	4.6(8.5)	2.0(3.3)	5.0(8.6)	0.004
SDAI, median (IQR), n=369	4.9(8.5)	2.2(4.5)	5.1(8.7)	0.012
DAPSA, median (IQR), n=376	6.6(11.0)	2.1(7.4)	6.8(11.1)	0.005
BASDAI, mean±SD, n=192	3.3±2.3	3.0±3.3	3.3±2.3	NS
ΔBASDAI, mean±SD, n=146	-2.8±2.4	-2.2±-2.8	0.2±2.5	0.010
BASFI, median (IQR), n=169	3.0(4.0)	4.3(4.0)	3.0(3.9)	NS
HAQ, median (IQR), n=330	0.5(1.0)	0(0.4)	0.5(1.1)	0.025
24 Months				
Tender joints 68, median (IQR), n=444	0(1.0)	0(1.0)	0(1.8)	NS
Swollen joints 68, median (IQR), n=444	0(1.0)	0(1.0)	0(1.0)	NS
ESR (mm/1st hour), median (IQR), n=418	10(18.0)	11.0(20.0)	10.0(17.0)	NS
CRP (mg/dL), median (IQR), n=413	0.2(0.4)	0.2(1.2)	0.2(0.4)	NS
Patient VAS, mean±SD, n=387	28.4±25.8	23.3±19.2	28.7±26.0	NS
Physician VAS, mean±SD, n=341	12.4±15.2	11.4±16.6	12.4±15.2	NS
Pain VAS, mean±SD, n=301	26.9±25.5	24.4±23.5	27.0±25.6	NS
DAS28 4V CRP, mean±SD, n=282	2.2±1.0	2.0±0.8	2.2±1.0	NS
ΔDAS28, mean±SD, n=220	2.3±1.4	3.0±1.2	2.3±1.4	NS
CDAI, median (IQR), n=275	4(7.1)	3.5(5.6)	4.0(7.1)	NS
SDAI, median (IQR), n=253	4.4(7.8)	4.0(4.6)	4.5(7.8)	NS
DAPSA, median (IQR), n=258	5.6(10.5)	6.0(7.4)	5.5(10.8)	NS
BASDAI, mean±SD, n=138	3.0±2.1	3.0±3.2	3.0±2.1	NS

continues on the next page

Table 1. Continuation.

Variables	Total (N=1255)	Elderly patients (n=113)	Non-Elderly patients (n=1142)	p_{value}
ΔBASDAI, mean±SD, n=100	-2.7±2.1	-2.9±1.8	-2.7±2.1	NS
BASFI, median (IQR), n=117	2.7(4.0)	0.2(0)	2.7(4.0)	NS
HAQ, median (IQR), n=240	0.4(0.9)	0.4(0.9)	0.4(0.9)	NS
Clinical response (peripheral and axial)				
12 Months				
ASDAS CRP, mean±SD, n=182	2.1±0.9	1.6±1.0	2.1±0.9	NS
ΔASDAS CRP, mean±SD, n=134	-1.6±1.2	1.3±0.5	-1.6±1.2	NS
PsARC response (yes), n=348	258(74.1)	16(88.9)	242(73.3)	NS
24 Months				
ASDAS CRP, mean±SD, n=123	2.0±0.9	2.4±2.2	1.9±0.9	NS
ΔASDAS CRP, mean±SD, n=85	-1.6±1.2	-	-1.6±1.2	NC
PsARC response (yes), n=244	192 (78.7)	6(66.7)	186(79.1)	NS
Switch over 24M (yes), n (%)	459(36.6)	33(29.2)	426(37.3)	NS
Patients with AE, n (%)	135(10.8)	14(12.4)	121(10.6)	NS
bDMARDs discontinuation due to an AE, n (%), n=135	75 (55.6)	9/14(64.3)	66/121(54.5)	NS

*Fisher test. BMI-body mass index; IQR-interquartile rate; SD-standard deviation; bDMARD-biologic disease-modifying antirheumatic therapy; NS– Non significant; NSAIDs- Non-steroidal anti-inflammatory drugs; cDMARD- Conventional Disease Modifying Anti-Rheumatic Drugs; TNFi- tumour necrosis factor inhibitors; ESR- erythrocyte sedimentation; CRP- C Reactive Protein; VAS- Visual analogue scale; DAS-28-Disease Activity Score 28; CDAI-Clinical Disease Activity Index; SDAI-Simplified Disease Activity Index; DAPSA-Disease Activity in PSoriatic Arthritis; BASDAI- Ankylosing Spondylitis Disease Activity Index; BASFI- Bath Ankylosing Spondylitis Functional Index; HAQ- Health Assessment Questionnaire; ASDAS-Ankylosing Spondylitis Disease Activity Score; PsARC- Psoriatic Arthritis Response Criteria; AE-adverse events. NS-not significant; NC-not calculated.