Spondylodiscitis (Andersson lesion) in psoriatic spondyloarthritis: a rare event successfully treated with anti-TNF therapy

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To the Editor,

A 58 year-old woman, suffering from diffuse psoriasis since the age of 22 years, was referred to our Unit due to rheumatic symptoms. Eight year before, she complained of an episode of dactylitis at the right hand, successfully treated with non-steroidal anti-inflammatory drugs (NSAIDs). Two years later arthritis involving hand-fingers (II, III, IV and V proximal and distal interphalangeal bilateral joints) and wrists, feet (II and III proximal and distal interphalangeal bilateral joints), ankles and knees, with severe functional impairment occurred. In addition, a severe pain at the bilateral sacroiliac and coxo-femoral regions developed. The magnetic resonance imaging (MRI) of the pelvis showed bone oedema at the left femoral head and in the subchondral bone of the right acetabulum with coxo-femoral effusion and chondropathy. The diagnosis of psoriatic arthritis (PsA) was formulated. She was then treated for 1 year with NSAIDs monotherapy achieving only a temporary relief, and thereafter with methotrexate (15 mg/week) and methylprednisolone (16 mg/day). However, due to an inadequate control of disease (Bath Ankylosing Spondylitis Disease Activity Index, BASDAI: 7.1; Visual Analog Scale VAS: 7/10; Disease Activity Score 28, DAS28: 5.3), 2 years later anti-TNF monotherapy (adalimumab 40 mg/every 2 weeks) was started, achieving an improvement for 1 year (DAS: 2.8). Unfortunately, a worsening and spreading of psoriasis occurred, requiring adalimumab interruption. Therefore, methylprednisolone (16 mg/day) therapy was then reintroduced for 1 year. Despite psoriasis improvement (Psoriasis Area Severity Index (PASI) reduction from 30 to 10) a rapidly progressive worsening of peripheral and axial arthritis occurred, with nagging and piercing pain at the dorsal rachis causing functional impairment. A spine MRI scan showed spondylodiscitis at D11-D12 (i.e. Andersson lesion), with partial fusion of the vertebral bodies and intraspongiosal oedema (Figure 1A). She was not able to walk or perform her usual daily activities. When the patient was referred to our clinic, the BASDAI scored 9.0, the VAS was of 8/10 and the DAS 28 was of 5.6. The HLA B 27 was negative. The patient underwent another MRI scan of the spine, which showed further worsening of spondylodiscitis with fusion of the vertebral bodies and parenchymal tissue invasion of the vertebral channel (Figure 1B). We started a biologic therapy with infliximab (5 mg/Kg) according to the conventional induction scheme, plus methotrexate (15 mg/week). A marked improvement occurred within 3 months, with no evidence of psoriasis patches (PASI: 0), and improvement of axial and peripheral symptoms (BASDAI: 4.1, VAS: 4, and DAS 28: 3.1). At 10 months, a new spine MRI scan showed absence of both intraspongiosal oedema at D11-D12 vertebral bodies and intracanalicular tissue, with an almost total fusion of the vertebral bodies (Figure 1C).

Our case shows some peculiar aspects. Firstly, a dramatic worsening of psoriasis during adalimumab monotherapy was observed, requiring treatment interruption. This is an infrequent paradoxical side effect of biologic therapy reported in the literature[1]. Secondly, Andersson lesions usually occur in ankylosing spondylitispatients[2]. This is a very exceptional event in PsA patients, since only few cases of Andersson lesions were reported[3]. Third, efficacy of biologics on these lesions in PsA patients was scantly tested. We previously described a case of Andersson lesions successfully treated with anti-TNF therapy, and two further cases were reported[4-6]. Therefore, the present report would indicate that Infliximab therapy could be effective in treating

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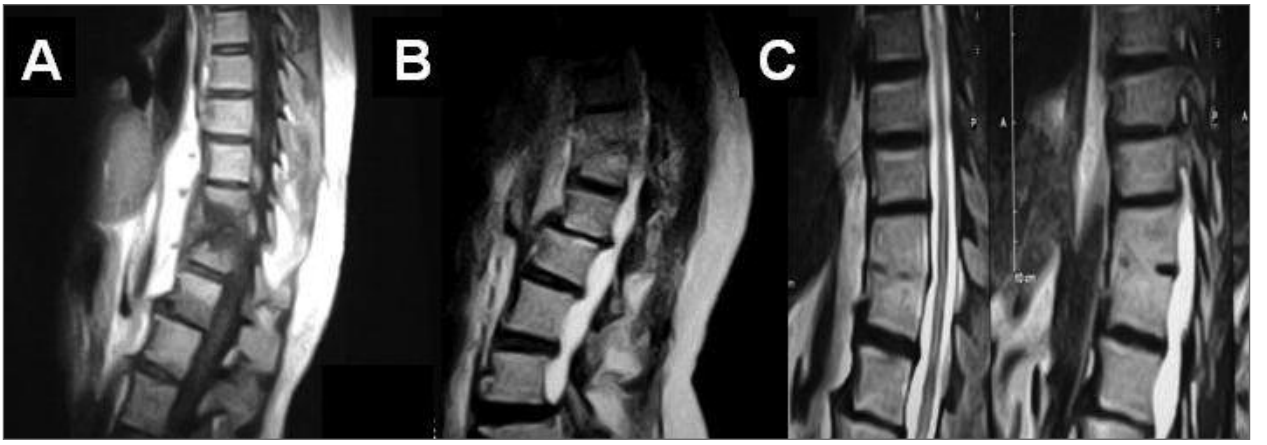

FIGURE 1. (A) Andersson lesion; (B) Parenchymal tissue in the canal; (C) Complete intravertebral fusion

Andersson lesions in PsA patients. Finally, the addition of methotrexate to anti-TNF therapy might have prevented the worsening of psoriasis, as initially occurred during adalimumab monotherapy in our patient, suggesting that the class effect may be eliminated[1].

In conclusion, Andersson lesions may occur in PsA patients, which may be successfully managed with anti-TNF therapy.

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