Dorso-Lumbar Pain in a Woman Diagnosed With Ankylosing Spondylitis

Raquel Almodóvar González,[a] Jorge Juan Prada,[b] Pedro Zarco Montejo,[a] Francisco Javier Quirós Donate,[a] and Ramón Mazzucchelli Esteban[a]

[a]Unidad de Reumatología, Fundación Hospital Alcorcón, Madrid, Spain
[b]Medicina Familiar y Comunitaria, Fundación Hospital Alcorcón, Madrid, Spain

Clinical Case

A 41-year-old woman with a history of smoking 5 cigarettes a day was diagnosed at 27 years of age with ankylosing spondylitis with a positive HLA-B27, according to the modified New York criteria. She was initially treated with analgesics and sulphasalazine, receiving up to 1000 mg/day, not tolerating a higher dose due to gastrointestinal complications, achieving good control of her disease. In March 2004, the patient presented intense dorsolumbar pain that increased with rest and improved with movement. The pain partially improved with the use of paracetamol and muscle relaxants. She did not present fever nor constitutional symptoms and had no history of trauma. The physical examination evidenced pain of the spinous processes of the dorsolumbar spine, a modified Schober test of 1.3 cm, a finger-floor distance of 5 cm, BASFI (Bath Ankylosing Spondylitis Functional Index) of 51, and BASDAI (Bath Ankylosing Spondylitis Disease Activity Index) of 48.

The laboratory testing showed: hemoglobin, 11.6 g/dL (12-15); mean corpuscular volume, 83.20 fl (78-100); erythrocyte sedimentation rate, 25 (020); and CRP, 5.4 mg/L (0-5). The blood chemistry, blood proteins, and urine tests were normal. PPD was positive (11 mm), with a normal chest x-ray. Blood cultures, serology for *Brucella* and acid-alcohol resistant bacilli in urine were negative. The patient had a lumbar spine x-ray that showed a wedge-like appearance of D-12 which had not been observed on previous x-rays as well as osteopenia and squaring of the vertebral bodies. The study was completed with a STIR magnetic resonance (MR) of the spine (Figure 1) which showed a hyperintense image in T2 with fat suppression, compatible with a grade C1 Anderson lesion (edema of the vertebral body) at the level of D7 and D8; a hyperintense lesion in T2 at the level of L1-L2 compatible with a grade C2 Anderson lesion (fatty exchange) and an anterior wedging due to osteoporosis at D12 without signs of edema. A bone densitometry was carried out and it showed lumbar (*T-score,* –2.2; *Z-score,* –0.86) and femoral osteopenia (*T-score,* –1,8; *Z-score,* –0,62).

Diagnosis and Progression

Faced with these findings, the diagnosis of aseptic spondylodiskitis at the level of D7-D8 and L1-L2 was made, with vertebral crushing of D12 due to osteoporosis. Treatment with anti-TNF was begun in July 2004, with etanercept at a dose of 25 mg twice a week, when no

Correspondence: Dra. R. Almodóvar González.
Fundación Hospital Alcorcón.
Budapest, s/n. 28922 Madrid. España.
E-mail: ralmodovar@fhalcorcon.es

Manuscript received December 26, 2006; accepted for publication Juny 29, 2007.

Figure 1. *Magnetic resonance of the dorsal spine, STIR sequence sagital plane showing a hyperintense image in T2 compatible with a grade C1 Anderson lesion (vertebral body edema) at the level of D7 and D8; hyperintense lesion at L1-L2 compatible with a grade C2 Anderson lesion (fat exchange) and an anterior osteoporotic wedging of D12 without signs of edema.*

Figure 2. *Control magnetic resonance image of the dorsal spine, sagital plane STIR sequence in which a complete resolution of spondylodiskitis at the D7-D8 level, with fatty substitution and no signs of interdisk inflammatory activity can be seen. The rest of the image shows no changes with respect to the previous one.*

response was seen to non-steroidal anti-inflammatory drugs (NSAIDs) and sulphasalazine. Concomitant chemoprophylaxis with isoniazide was carried out for 9 months due to the positive PPD reading and treatment with calcium and vitamin D was added due to the densitometric findings. After 4 weeks of treatment the patient showed clinical improvement with a disappearance of dorsolumbar pain and the normalization of acute phase reactants. On physical examination the patient presented a modified Schober of 1.5 cm, a finger-floor distance of 0 cm, BASFI of 11 and BASDAI of 0. In January 2005 a control dorsal spine STIR MR was performed (Figure 2) in which no signs of bone or disk inflammatory activity was seen at the level of D7-D8. After 1 year of treatment there was improvement of the bone densitometry findings (lumbar: *T-score*, –1.35, and femoral: *T-score*, –0.02) with respect to the previous one. The patient continues to be asymptomatic, receiving anti-TNFα therapy.

Discussion

Ankylosing spondylitis (SA) is considered as the prototype of spondyloarthropathies and is a chronic inflammatory rheumatic disease which presents between the second and third decades of life, with a prevalence of 0.1%-1.1%.[1] The disease is characterized by an inflammation of the sacroiliac joints, as well as enthesis and peripheral joints. Axial skeleton affection can be present in the form of spondylitis, spondylodiskitis, and arthritis of the joint apophysis, with lumbar and/or buttock inflammatory pain as the main symptom.[1-3] Aseptis diskitis was described by Andersson in 1937, and is an infrequent manifestation of SA characterized by erosive lesions of the disk-vertebrae union and its pathogeny is unknown but several inflammatory factors have been implicated, as well as severe progressive enthesopathy and mechanical factors such as vertebral displacements due to the instability originated by interapophiseal joint affection or pseudoarthritis in relation to foci of adjacent fractures[4]. Diskitis is usually associated to advanced stages of disease and grade III sacroillitis[5]. It is important to perform a differential diagnosis of aseptic spondylodiskitis with infections (pyogenic, brucellosis, tuberculosis), trauma (osteoporotic fractures, psudoarthritis after a poorly mended or undiagnosed fracture), neoplasia (myeloma, bone metastasis), sarcoidosis, chondrocalcinosis, intervertebral osteochondrosis, and neuropathic osteoartropathy. In our case, all of these causes were reasonably discarded through laboratory testing (general analysis, cultures serology and PPD) and imaging (chest and lumbar spine x-ray and spinal MR).

Therapeutic options for SA have been limited during the past decades. NSAIDs are considered standard treatment. With respect to disease-modifying agents, there is no evidence that shows they are useful in SA axial affection,[6] and sulphasalazine is partially useful in peripheral and axial arthritis.[7] But currently, treatment with anti-TNFα has revolutionized the treatment of active SA. Etanercept is a recombinant fusion TNF-IgG1 receptor protein, with an efficacy that has been demonstrated by significant improvement in different clinical, functional, quality of life, and disease activity parameters, both in the short and the long terms.[8-10]

Conventional x-rays only visualize chronic changes after 2 years of progression of SA, but is unable to detect acute inflammatory lesions. On the contrary, MR is a powerful tool used to see and follow acute inflammatory lesions, both spinal and sacroilliac, allowing for an early diagnosis of SA and a better evaluation of response to treatment. Two MR techniques are used to detect active inflammatory lesions. On one hand gadolinium and fat suppression techniques and on the other, STIR, which has a greater sensitivity.[10-12]

There are different studies on the efficacy of etanercept treatment in SA spinal lesions, all of them showing improvement of 53% to 73% of acute spinal lesions evaluated by MR after treatment.[12] A significant regression of the acute spinal lesions at 6 weeks of treatment has been seen, and this response is seen after 2 years of continuous therapy. With respect to chronic spinal lesions (sclerosis, syndesmophites, and bony bridging), there is no significant change after treatment.[10-12] Similar results have been published with infliximab.[13] Acute inflammatory lesions of the sacroiliac joints show

a moderate regression, not a significant one, after treatment with etanercept.[10-12]

The interest of the case resides in the relevant role that MR plays in the diagnosis and follow-up of patients with seronegative spondyloarthroopathies, which allows deciding upon the most adequate treatment option. It must be emphasized, on the other hand, the clinical efficacy and safety of etanercept treatment in inducing regression of inflammatory activity in patients with ankylosing spondylitis.

References

1. Braun J, Bollow M, Remlinger G, Eggens U, Rudwaleit M, Distler A, et al. Prevalence of spondylarthropathies in HLA-B27 positive and negative blood donors. Arthritis Rheum. 1998;41:58-67.
2. Braun J, Sieper J. The sacroiliac joint in the spondyloarthropathies. Curr Opin Rheumatol. 1996;8:275-87.
3. Braun J, Bollow M, Sieper J. Radiology and pathology of the spondyloarthropathies. Rheum Dis Clin North Am. 1998;24:697-735.
4. Rasker JJ, Prevo RL, Lanting PJ. Spondylodiscitis in ankylosing spondylitis, inflammation or trauma? A description of six cases. Scand J Rheumatol. 1996;25:52-7.
5. Langlois S, Cedoz JP, Lohse A, Toussirot E, Wendling D. Aseptic discitis in patients with ankylosing spondylitis: a retrspective study of 14 cases. Joint Bone Spine. 2005;72:248-53.
6. Roychowdhury B, Bintley-Bagot S, Bulgen DY, Thompson RN, Tunn EJ, Moots RJ. Is methotrexate effective in ankylosing spondylitis? Rheumatology (Oxford). 2002;41:1330-2.
7. Braun J, Alten R, Burmester G, Ebner W, Gommor B, Grasedyck K, et al. Efficacy of sulfasalazine in undifferentiated spondyloarthritis and eraly ankylosing spondylitis: a multicenter randomized controlled trial. Berlin: Annual European Congress of Rheumtology; 2004.
8. Brandt J, Khariouzov A, Listing J, Haibel H, Sorensen H, Grassnickel L, et al. Six-month results of a double-blind, placebo-controlled trial etanercept treatment in patients with active ankylosing spondylitis. Arthritis Rheum. 2003;48:1667-75.
9. Davis J, van der Heijide D, Braun J, Dougados M, Cush J, Clegg DO, et al. One year efficacy of etanercept in ankylosing spondylitis [abstract]. Ann Rheum Dis. 2004;63 Suppl 1:390.
10. Baraliakos X, Brandt J, Listing J, Haibel H, Sorensen H, Rudwaleit M, et al. Outcome of patients with active ankylosing spondylitis after two years of therapy with etanercept: clinical and magnetic resonance imaging data. Arthritis Rheum. 2005;53:856-63.
11. Rudwaleit M, Baraliakos X, Listing J, Brandt J, Sieper J, Braun J. Magnetic resonance imaging of the spine and the sacroiliac joints in ankylosing spondylitis and undifferentiated spondyloarthritis during treatment with etanercept. Ann Rheum Dis. 2005;64:1305-10.
12. Baraliakos X, Davis J, Tsuji W, Braun J. Magnetic resonance imaging examinations of the spine in patients with ankylosing spondylitis before and after therapy with the tumor necrosis factor alpha receptor fusion protein etanercept. Arthritis Rheum. 2005;52:1216-23.
13. Baraliakos X, Wanders A, Listing J, Brandt J, Rudwaleit M, Sieper J, et al. X–ray progression in patients with ankylosing spondylitis (AS) afeter 2 years of infliximab therapy [Abstract]. Arthritis Rheum. 2004;50 Suppl 9:S450.