Austin Journal of Orthopedics & Rheumatology

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Austin Publishing Group

Case Report

Andersson Lesion Presenting Itself as Spondylodiscitis and Evolving to Pseudoarthrosis in a Patient with Ankylosing Spondylitis: A Case Report

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Received: December 08, 2014; **Accepted:** March 01, 2015; **Published:** March 03, 2015

Abstract

This is a case report of a patient with an advanced stage Andesson lesion presenting itself as a spondylodiscitis which evolved to a full pseudoarthrosis. A 57-year-old Caucasian woman with a known history of ankylosing spondylitis was admitted due to worsening pain in her back and lower limbs that did not respond to conservative treatment. A series of surgical procedures were needed. Although ankylosing spondylitis is a well-known disease, Andersson lesions are not well known nor fully understood. Orthopaedic surgeons must be familiar with the condition in order to make the right diagnosis.

Keywords: Andersson lesion; Ankylosing spondylitis; Discovertebral lesion; Aseptic spondylodiscitis; Spondylodiscitis

Abbreviations

AL: Andesson Lesion; AS: Ankylosing Spondylitis; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; CT: Computer Assisted Tomography; MRI: Magnetic Resonance Imaging; NSAID: Non Steroid Anti-Inflammatory Drugs; anti-TNF: anti Tumour Necrosis Factor

Introduction

Ankylosing spondylitis is an idiopathic inflammatory disorder that primarily affects the sacroiliac joints and the spine. The disease is characterized by the ossification of the spinal ligaments, joints and intervertebral discs. These processes lead to a fused and brittle spine susceptible to fractures. Mechanical forces and inflammation result in focal discal or discovertebral lesions that fail to heal and eventually result in pseudoarthrosis. First described by Andersson in 1937, they are named Andersson lesions [1]. Although well known by the specialist community, AL is relatively rare and awareness of the condition has to be spread.

Case Presentation

A 57-year-old patient with history of HLA B27 positive ankylosing spondylitis under continuous ambulatory management by rheumatologists was admitted to the rheumatology department because of worsening pain in the thoracic spine unresponsive to NSAIDs. In the past she was tested positive on the Mantoux skin test and also had a positive Quantiferon test. X-ray of the thoracic spine showed changes suspicious of inflammation. An MRI scan was performed that showed signs consistent with spondylodiscitis of the Th11-Th12 intervertebral disc (Figure 1). The patient was afebrile the whole time during hospitalization and her blood work was unremarkable for inflammation. The idea arose to start treating her with biopharmaceuticals; however, due to a suspected spondylodiscitis and positive Mantoux and Quantiferon tests, treatment was held off.

The patient was referred to the orthopaedic department, where a biopsy of the affected disc was performed. The material was sent for histological and bacteriological assessment. During hospitalization laboratory results were basically normal, leukocytes 8.45 and CRP 33. Upon consultation with the infectious disease specialist no antibiotics were administered. Bacteriology results were negative and the patient was discharged in a good condition.

She was scheduled for a follow-up examination two weeks after discharge. At that time the results of histology were known and showed a nonspecific osteomyelitis. The patient was still afebrile, pain free and subjectively in a good condition.

She was referred to a pulmonology specialist who recommended prophylactic tuberculostatic treatment. When the treatment was completed, the patient was continued with the biopharmaceutical treatment with etanercept. Because of an allergic reaction, it was later substituted for adalimumab. The treatment was successful, as the patient reported subjective relief in pain, better quality of life and

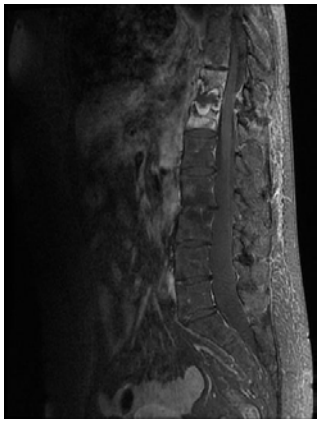

Figure 1: MRI showing spondylodiscitis of the Th11 – Th12 intervertebral discus.

Citation: Pen M, Milcic M, Krajnc A, Vogrin M and Recnik G. Andersson Lesion Presenting Itself as Spondylodiscitis and Evolving to Pseudoarthrosis in a Patient with Ankylosing Spondylitis: A Case Report. Austin J Orthopade & Rheumatol. 2015;2(1): 1011.

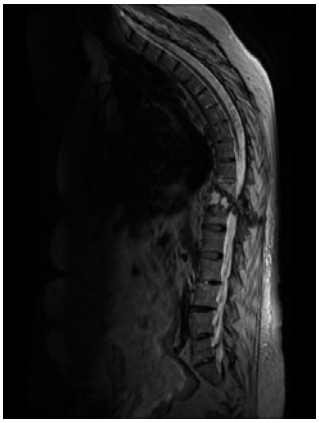

Figure 2: MRI showing pseudoarthrosis of the involved spinal bodies.

less impairment. At examination her performance scores (BASDAI, Schober) improved. Followup was unremarkable.

Three years later she presented to the rheumatologist with a four-week long history of pain in the lower thoracic spine, painful cramps in her left lower limb, muscular weakness and a sensation of numbness. There were no signs of an acute infection. Since the last hospitalization she was diagnosed with osteoporosis and was receiving bisphosphonates and vitamin D. She was admitted to the hospital because of a suspected fracture of the 11th and 12th thoracic vertebrae. Because the patient was unfit to perform an MRI scan due to intense pain, a CT scan was performed and showed an uneven transverse fracture at the level of Th11-Th12, with stenosis of the spinal canal. The next day the patient's pain allowed for an MRI scan, which showed a pseudoarthrosis between the 11th and 12th thoracic vertebrae (Figure 2). In wake of these results, the original MRI scan from 3 years ago was revised to had shown a transverse fracture at the same level and the suspected spondylodiscitis was classified as an Andersson lesion (Figure 3).

The patient was again transferred to the orthopaedic department, where a decompression and posterior stabilisation from the 9th thoracic to the 2nd lumbar vertebra was performed. Because of the inveterate state of the fracture with pseudoarthrosis and ongoing inflammation, the dural sac was degenerated and partly non-existent. Dural reconstruction was needed.

The day after surgery a spinal fluid leak through the drainage wound was observed. The patient complained of headaches and sickness. Conservative management was attempted. CRP spiked at 283, but spontaneously fell to 182 the next day; leukocytes fell from 10 to 5.9. The decision to start administering antibiotics was made, a combination of amoxicillin and clavulanic acid was given. A control MRI scan showed a large fluid collection, residual spinal stenosis and a dural tear at the Th11-Th12 level.

Revision surgery with additional decompression had to be performed. There were no intraoperative signs of infection.

After the revision surgery her CRP stayed elevated at around 200, while leukocytes were at a normal range. Infectious disease specialist recommended exchanging the antibiotic for meropenem. A few days later her CRP started falling.

The patient was then prepared for her third surgery, an anterior stabilisation at the Th11-Th12 level with an autologous bone graft form the iliac crest. During the procedure tissue samples were taken for bacteriology. After the surgery she was placed in our perioperative intensive care unit for one night and later transferred to the ward. All tissue samples stayed sterile. With the help of extensive physiotherapy work, she was verticalized. After 87 days she was discharged ambulatory and in a subjectively good condition.

At follow-up visit CT scan showed good positioning of the instrumentation (Figure 4 and 5). The patient still complained of slight femoralgia, but was fully ambulatory. Rehabilitative programme was yet to begin.

Discussion

According to a review article from 2009 by Bron et al. the prevalence of AL varies from 1.5% to over 28% [2]. This discrepancy might be explained by the lack of proper diagnostic criteria. The exact etiology is not yet known, but is believed to be non-infectious. There are two main theories, one is inflammatory and is the other mechanical/traumatic. Traditionally, three fields of specialists were involved in AL. Rheumatologists explained the lesions as an aseptic spondylodiscitis. Cawley et al. (1972) reported that these lesions can either be focal or affecting more levels at once [3]. Orthopaedic surgeons and radiologists on the other hand described it as a pseudoarthrosis due to non-union of a stress fracture [4,5]. Today it seems that inflammation is the main culprit of AL in early ankylosing

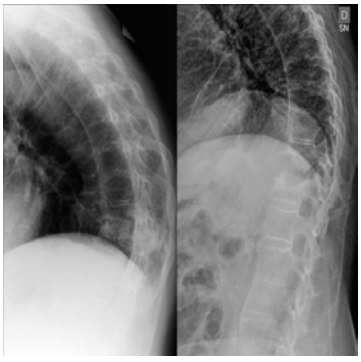

Figure 3: X – rays showing instability of the involved spinal segment.

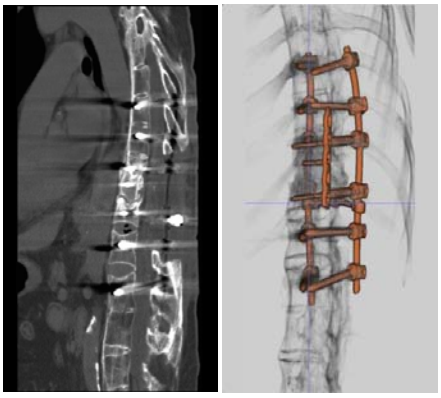

Figure 4 and 5: CT showing final results – spinal fusion with bone graft and instrumentation.

spondylitis, while traumatic origin is believed to be responsible for AL occurring in fully ankylosed spines.

During the course of AS, there is progressive ossification of the annulus fibrosus, anterior longitudinal ligament, apophyseal joints, interspinous ligaments and the ligamentum flavum. This causes the spine to become rigid. Because of ongoing inflammation there is also concurrent osteoporosis of the vertebral bodies. Thus the nucleus pulposus can herniate through the vertebral endplate. This causes a florid inflammatory response due to the antigenicity of nucleus pulposus, as avascular nucleus tissue meets the vascularized bone [2].

Another theory is that during the course of the disease the extent of inflammation and spinal fusion are not equally distributed throughout the spine. Areas with increased inflammation but decreased fusion allow for an excessive degree of movement (mobile segment), while areas of increased fusion but decreased inflammation make the spine stiff. The result is an ankylosed spine prone to stress fractures and moves around the mobile segment. Once a fracture develops, it is unlikely to reunite as it is the only mobile segment and a pseudoarthrosis develops [2].

AL can develop after traumatic events, which include direct blows, repetitive mechanical stress or series of minor trauma. Acute fractures most commonly involve the thoracolumbar junction, especially in an ankylosed kyphotic spinal column. Fractures may be transvertebral or transdiscal and usually involve the posterior elements.

A correlation between AL and heavy labour was reported. Because bone is more susceptible to distraction forces, posterior spinal elements may fail during repetitive hyperflexion loads. Also, there are reports of AL in patients who became more mobile after hip arthroplasty. Anti-inflammatory drugs often enable patients with AS to be more mobile, new anti-TNF "biopharmaceutical" drugs have profound effects on mobility and pain control. As a result patients do not feel pain and stress fractures with pseudoarthrosis can develop [2].

Conservative treatment is the first choice, but there are no developed protocols yet. Stabilisation with plaster has been tried and reported successful, but once pseudoarthrosis develops, fusion is unlikely. If the spine is stable and there are no signs of pseudoarthrosis, bed rest, NSAIDs and anti-TNF drugs seem to be the proper treatment. It has been suggested that in thoracic spine an intact ribcage can provide enough stability to prevent kyphosis [6].

Wang et al. published a study of 8 cases (2011) in which they postulated surgical instrumentation with fusion as treatment of choice in cases with traumatic etiology [6]. Curettage of the lesion, anterior fusion and posterior fixation has been proposed as the golden standard. However, there are reports of successful fusion with posterior fixation only, without any curettage of the lesion and without anterior fusion. For lesions with obvious vertebral body destruction bone grafting is recommended [6].

Hanson and Mirza [7] reported of fractures below the level of instrumentation as a late complication in patients after series of minor trauma [8]. Careful evaluation of imaging data of such patients is of great importance.

References

1. Andersson O. X-ray image at spondylarthritis ankylopoetica. Nord Med Tidskr. 1937; 14: 2000–2002.
2. Bron JL, de Vries MK, Snieders MN, van der Horst-Bruinsma IE, van Royen BJ. Discovertebral (Andersson) lesions of the spine in ankylosing spondylitis revisited. Clin Rheumatol. 2009; 28: 883–892.
3. Cawley MI, Chalmers TM, Kellgren JH, Ball J. Destructive lesions of vertebral bodies in ankylosing spondylitis. Ann Rheum Dis. 1972; 31: 345–358.
4. Wu PC, Fang D, Ho EK, Leong JC. The pathogenesis of extensive discovertebral destruction in ankylosing spondylitis. Clin Orthop. 1988; 230: 154–161.
5. Chan FL, Ho EK, Fang D, Hsu LC, Leong JC, Ngan H. Spinal pseudarthrosis in ankylosing spondylitis. Acta Radiol. 1987; 28: 383–388.
6. Wang G, Sun J, Jiang Z, Cui X. The Surgical Treatment of Andersson Lesions Associated With Ankylosing Spondylitis. Orthopedics. 2011; 34: 302-306.
7. Hanson JA, Mirza S. Predisposition for Spinal Fracture in Ankylosing Spondylitis. Trauma Cases from Harborview Medical Center. AJR. 2000; 174: 150.
8. Dhakad U, Das SK. Anderson lesion in ankylosing spondylitis. BMJ Case Rep. 2013.

Austin J Orthopade & Rheumatol - Volume 2 Issue 1 - 2015
ISSN: 2472-369X | www.austinpublishinggroup.com

Citation: Pen M, Milcic M, Krajnc A, Vogrin M and Recnik G. Andersson Lesion Presenting Itself as Spondylodiscitis and Evolving to Pseudoarthrosis in a Patient with Ankylosing Spondylitis: A Case Report. Austin J Orthopade & Rheumatol. 2015;2(1): 1011.