

## Case Report



# Pyogenic Andersson Lesion in a Patient With Ankylosing Spondylitis

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## OPEN ACCESS

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### Conflict of Interest

The authors have no financial conflicts of interest.

## ABSTRACT

Although the exact etiology of the Andersson lesion (AL) remains unclear, it is known to occur mostly in patients with long-standing ankylosing spondylitis (AS). Among the various theories for the etiology of AL, repetitive trauma and inflammatory causes are the most common. The histopathological appearance of the AL in this report was consistent with that of chronic inflammation without any infection. Pyogenic ALs in the context of AS are extremely rare; to the best of our knowledge, positive cultures of this lesion in bone biopsies have never been reported. Herein, we report a rare case of a pyogenic AL with a positive culture and discuss a relevant review of the literature.

**Keywords:** Discovertebral lesion; Ankylosing; Spondylitis; Infection

## INTRODUCTION

Ankylosing spondylitis (AS) is a seronegative arthropathy that primarily affects the sacroiliac joints and spine, causing progressive back stiffness and pain.<sup>6,8)</sup>

In the advanced state of AS, progressive ossification of the whole spine often results in a fixed and rigid ankylosed spine. Spinal pseudoarthrosis, a well-known complication of AS, was first described by Andersson.<sup>1)</sup> Since Andersson's first report, various names have been used to refer to these spine lesions, including 'Andersson lesion' (AL), 'discovertebral lesion,' 'vertebral lesion,' 'destructive vertebral lesion,' 'spondylodiscitis,' 'sterile discitis,' and 'pseudoarthrosis.'<sup>4,9,12)</sup>

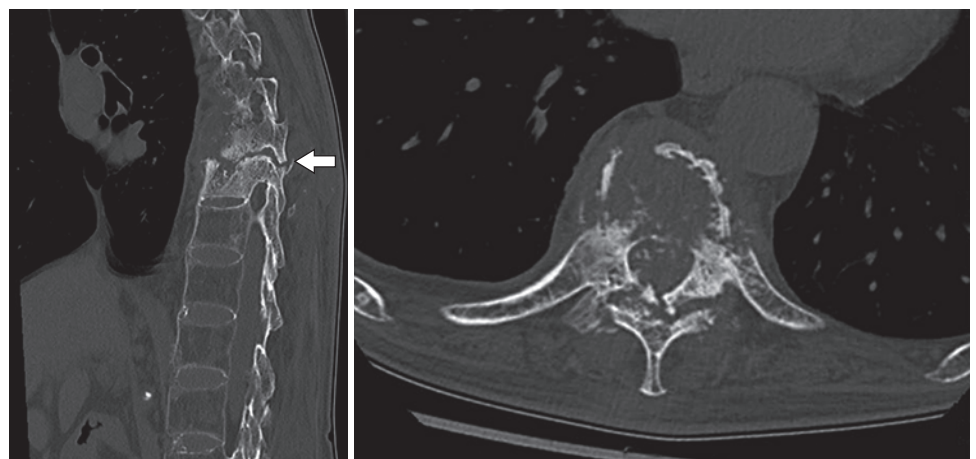
The naming of different terms for the same lesion may reflect the existence of an enduring debate regarding the exact etiology of AL. Among the various hypothesis to explain the etiology of AL, the most commonly accepted theories is repetitive trauma or inflammatory causes.<sup>13)</sup> Although radiological findings mimic pyogenic or tuberculous infection, there has been no evidence of a pyogenic or tuberculous infection in AL.

Herein, we describe a rare case of pyogenic spondylitis with AL, which revealed a positive culture of the lesion histopathologically, including radiological studies and a review of the relevant literature.

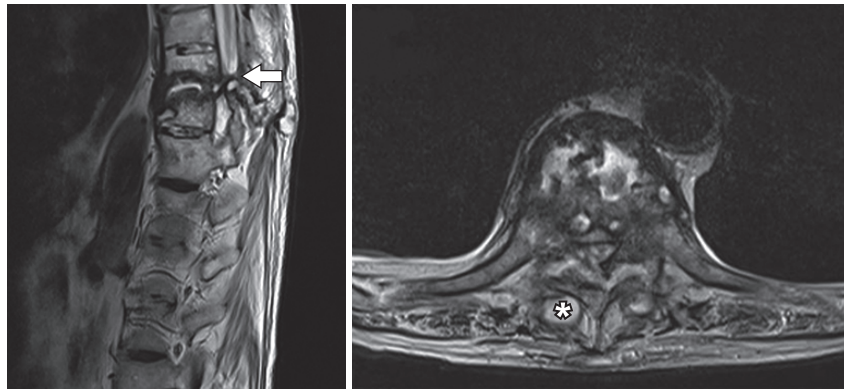
## CASE REPORT

A 68-year-old man with a history of AS diagnosed 3 months ago was admitted to the emergency room (ER) because of weakness of the lower extremities that had developed approximately 2 weeks prior. He was diagnosed with AS 3 months before admission at a local clinic. Before his diagnosis, he had also experienced chronic back pain and limited spinal motion, but motor weakness had just occurred 2 weeks ago; he was not able to walk by himself. He had received conservative treatment under the diagnosis of AS, including exercise and non-steroidal anti-inflammatory drugs (NSAIDs), but denied any history of recent trauma or previous pyogenic spondylitis. He had not received any pain block or newer agents for AS, such as tumor necrosis factor (TNF)- $\alpha$  antagonists. At the time of admission, the patient had a clear mental status, but physical examination confirmed a muscle strength of grade 2 and grade 3 for the right and left legs, respectively. He also complained of a febrile sensation and intractable back pain. At the time of admission to the ER, however, his body temperature was not high and noted to be 36.9°. Laboratory tests performed upon admission revealed elevated erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) levels of 81 mm/h (normal range <20mm/h) and 2.57 mg/dL (normal range <0.05 mg/dL), respectively, and mild leukocytosis with a total white blood cell count of 11.500/ $\mu$ L. Computed tomography (CT) revealed central osteolysis of the T10 and T11 bodies, surrounded by an irregular sclerotic zone. The lesion extended to posterior elements (**FIGURE 1**). In the T2-weighted image of the thoracolumbar magnetic resonance imaging (MRI), a decrease in signal due to disc inflammation at the T10–T11 level was noted. Erosion in the intervertebral disc and adjacent endplate between T10 and T11 with severe cord compression was also confirmed. Additionally, a paraspinous abscess was noted at the T10–T11 level (**FIGURE 2**).

Emergency decompressive surgery was performed on the day of admission. A midline incision through a posterior approach was performed from the level of T9 to T12, while a decompressive laminectomy was performed at the T10 and T11 levels to relieve cord compression. During the operation, a large amount of pus was observed at the paraspinous site and epidural space. The pus was collected; thus, bacterial culture was performed for microbial identification. A bone biopsy was also performed for histological and microbiological confirmation. Irrigation was performed using a large amount of vancomycin containing normal saline solution. After massive irrigation, to prevent instability,



**FIGURE 1.** Sagittal and axial computed tomography scans show prominent osteolysis surrounded by an irregular sclerotic zone and transdiscal fracture. The lesion extends into the posterior elements (arrow).



**FIGURE 2.** T2-weighted sagittal and axial magnetic resonance images reveal involvement of the surrounding structures. The erosion in the intervertebral disc and in the adjacent endplate between T10 and T11 is observed (arrow). Severe cord compression with paraspinous abscess (asterisk) is also observed.

transpedicular screw fixation was performed at the T9, T11, and T12 vertebrae. The operation was performed successfully, while no surgery-related complications were also observed. Methicillin-sensitive *Staphylococcus aureus* was identified in the collected specimens from bone biopsy and blood cultures.

At the 2-week follow-up, radiologic findings showed that the screws were well maintained; in addition, the ESR and CRP levels had markedly dropped to 37 mm/h and 0.6 mg/dL, respectively. The paraparesis improved progressively after surgery. He was transferred to the rheumatology department for further treatment of pyogenic spondylitis with AL. After 4 months of follow-up, he was able to walk on his own without any assistance.

## DISCUSSION

ALs are discovertebral lesions that mainly occur in patients suffering from long-standing AS, which were first described by Andersson in 1937. Since then, several possible etiologies for the development of AL in patients with AS have been postulated, including infection, inflammation, repetitive trauma, or mechanical stress.

Cawley et al.<sup>4)</sup> analyzed these lesions into 3 categories according to whether there is an involvement of the discal surface of the vertebral rim (types A and B), the cartilaginous part of the end plate (types C and D), or both (type E). The risk of instability of lesions increases incrementally from type A to type E. Cawley also categorized AL into 2 types: localized and extensive lesions. These may cause unstable 3-column lesions, causing pseudoarthrosis in the fused spine.

The histopathological appearance of AL is known to be similar to that of chronic inflammation without pyogenic bacterial or tuberculous infection.<sup>5,11)</sup>

Due to the radiographic similarities with pyogenic spondylitis or tuberculous osteomyelitis, an infectious origin was initially suggested by some authors, but this has not gained much popularity in the literature because positive cultures of causative organism had not been identified.<sup>2)</sup>

So far, as far as the authors' knowledge, there have been few case reports of an infectious spondylitis with AL or septic AL like this case. Lohr et al.<sup>10)</sup> reported the case of a patient with a history of intravenous drug abuse who had AS, T11-T12 spondylodiscitis, and a positive culture of *S. aureus* in the urine, pleural fluid, and blood culture. However, there have been no reports that showed positive cultures by biopsy of the lesion, as present in our patient.

Histopathological findings of AL are similar to pseudoarthrosis with microbleeding, fibrous tissue, a consistent with callus portion, and sclerosis of the adjacent levels. Moreover, microscopic findings are similar to that of non-union of the bone, naming this lesion 'pseudoarthrosis.'<sup>13)</sup>

Inflammatory markers are usually elevated in most patients with AL, suffering from AS similar to infectious conditions. These points, together with inflammatory biopsy findings in AL patients, may indicate that inflammation could be an early contributor to AL of AS.<sup>12)</sup>

Other studies on biopsies in AL during the late course of the disease showed a reactive new bone, and a cartilage node or pseudoarthrosis about a micro-fracture have suggested that repetitive mechanical stress can be an initiating and potential factor for the development of AL; however, repetitive stress of micro-traumas cannot explain early AL or the development of AL in children.<sup>15)</sup>

In addition to the use of simple radiographs, CT scans are more sensitive in demonstrating fractures of the posterior elements and discovertebral osteolysis with reactive sclerosis. However, MRI is considered the best diagnostic modality for revealing AL with the highest sensitivity.<sup>7)</sup> Moreover, MRI allows better differentiation between neural elements and surrounding soft tissues.

Conservative treatment is generally the first method of treatment of AL with AS. NSAIDs form the initial basis of pharmacological management of AS during the active stages of the disease. Given its efficacy in AS treatment, newer agents such as TNF- $\alpha$  antagonists have reduced recovery period and prevented the complete fusion of vertebrae in AS. However, there is no evidence that these drugs actually work in the treatment of symptomatic AL. Moreover, there is an increased risk of infection when TNF- $\alpha$  antagonists and steroids are used in patients with rheumatic diseases including AS.

Surgical treatment is indicated in patients with intractable back pain, progressive kyphosis, or prominent neurological deficits. The surgical procedure depends on the localization of the lesion, alignment of the spine, and the degree neurological deficits.<sup>14)</sup>

## CONCLUSION

Herein, we report a rare pyogenic spondylitis with AL complicating an AS, which was proven positive culture of the lesion. A trial to understand pyogenic spondylitis complicated by AS is mandatory to improve clinical results of this rare condition.

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