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Case Report

Andersson lesion in ankylosing spondylitis misdiagnosed as spinal tuberculosis: two cases

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Abstract: Andersson lesion (AL) is a rare complication of ankylosing spondylitis, which can be easily misdiagnosed in clinical practice as an infectious disease (such as spinal tuberculosis) or as a neoplastic disease. Here we report two cases of AL which had been misdiagnosed as spinal tuberculosis. We hope to alert all clinicians to tuberculosis and ankylosing spondylitis, and help them to identify these diseases more accurately.

Keywords: Andersson lesion ankylosing spondylitis spinal tuberculosis

Introduction

Andersson lesion (AL) is a rare complication of ankylosing spondylitis (AS), first described by Andersson in 1937 [1-3]. AL is a lesion of the intervertebral disc-vertebral interface at the later stages of AS, and can be characterized as a combination of bone hyperplasia and bone destruction. AL can be easily misdiagnosed in clinical practice as an infectious disease (such as spinal tuberculosis) or as a neoplastic disease. Here we report two cases of AL which had been misdiagnosed as spinal tuberculosis.

Two case reports

Patient one

Male, 36 years old, was admitted to hospital due to "repeated back pain for 18 years, aggravated with lumbar kyphosis for six years". The patient also complained of nocturnal pain and morning stiffness over the previous 18 years. However, he was misdiagnosed as suffering from "spinal tuberculosis" six years ago after multiple clinical visits and MRI and CT scans because his lower back pain was significantly aggravated by kyphotic deformity, especially after activity or standing for a long time. The anti-tuberculosis treatment had no effect in improving his symptoms. Upon referral to our clinic, his condition was diagnosed as AS according to 1984 NY criteria. He is HLA-B27 positive, spinal motion is significantly limited, and both sacroiliac joint spaces have disappeared. **Figure 1** shows the patient's films, including full spine X-ray, lumbar CT plain film, and plain MR scan of the lumbar spine (T1-weighted and T2-weighted). In the full spine X-ray, the shape of the L2/3 vertebral bodies was abnormal, and the L2 vertebra was displaced slightly. The L3 vertebral body showed patchy high-density shadows and bamboo-like changes, and some of the intervertebral space had narrowed. The bilateral sacroiliac joint space had disappeared and the bilateral hip joint space had narrowed. From the lumbar CT plain film, the L2 and L3 vertebrae were also found to be slightly displaced backwards, there was relative marginal bone destruction, increased density of the adjacent bone marrow cavity and shadow indicating partial high-density regions in the soft tissue. This was confirmed in the enhanced scan, which also showed L2/3 stenosis. The plain MR scans of the lumbar spine (T1-weighted and T2-weighted) indicate that the L2 and L3 vertebrae are slightly displaced posteriorly, and are relatively disrupted in the marginal bone mass, showing abnormal mass signals of areas with low signal in T1WI. In T2WI, there was hyperintensity in the central part of the lesion, and the edge show-

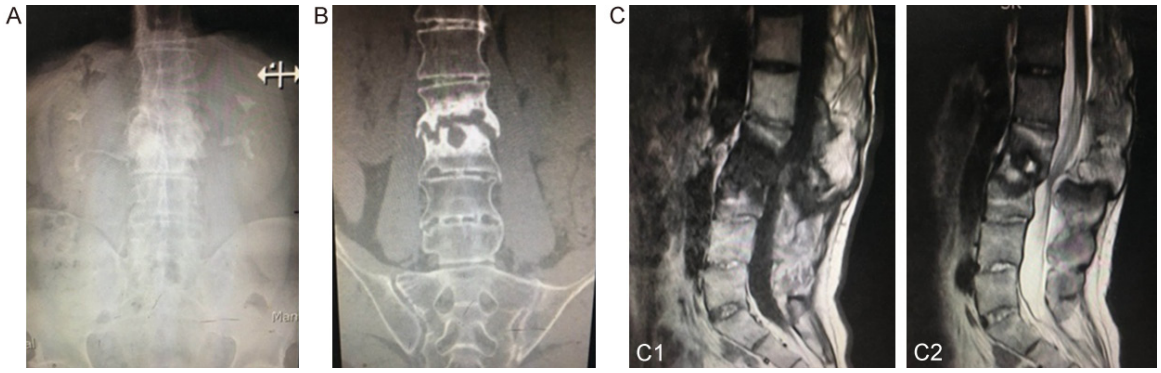

Figure 1. A. Full spine X-ray; B. Lumbar CT plain film; C. Plain MR scans of the lumbar spine (C1. T1-weighted; C2. T2-weighted).

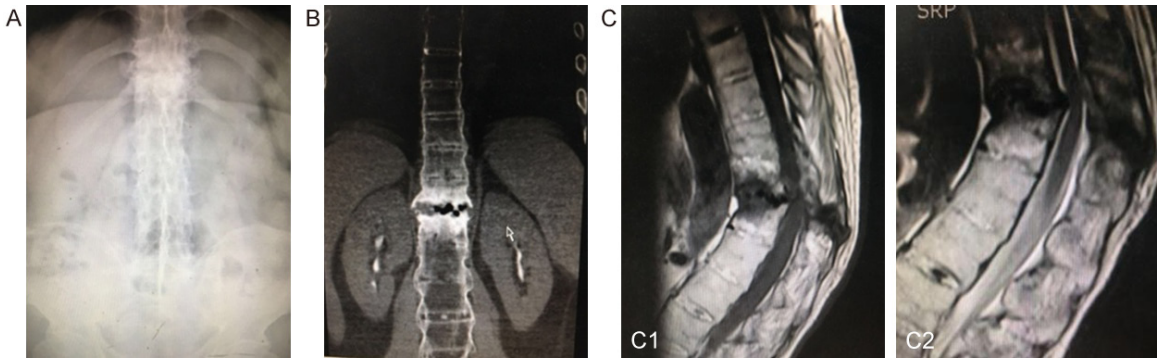

Figure 2. A. Full spine X-ray; B. Lumbar CT plain film; C. Plain MR scans of the lumbar spine (C1. T1-weighted; C2. T2-weighted).

ed linear low signal. The formation of soft tissue mass could be seen, and the enhanced scan showed heterogeneous enhancement.

Patient two

Male, 44 years old, was admitted to hospital because of "lower back pain for more than 10 years with numbness in both lower limbs for six months". In his local clinic, his MRI and CT scans showed "T11/12 vertebral bone destruction, pathological fracture and thoracolumbar kyphosis malformation", and "thoracic tuberculosis" had been suggested. After the regular anti-tuberculosis treatment, his symptoms did not improve. Upon referral to our clinic, his condition was diagnosed as AS according to 1984 NY criteria. He is HLA-B27 positive, spinal motion is significantly limited, and both sacroiliac joint spaces have disappeared. **Figure 2** shows the patient's spinal changes. From the full spine X-ray, thoracic kyphotic deformity and thoracolumbar spine bamboo-like changes can be clearly seen. The lower edge of the T11 vertebral body and the upper edge of the T12 vertebral body show bone destruction and T12 has compressive deformation. The structure of the bone in the attachment area is disordered and density is uneven. The bilateral sacroiliac joint spaces have disappeared and the bilateral hip joint spaces are narrowed. In the lumbar CT plain film, thoracic spine with kyphosis and bamboo-like changes can be very clearly seem. The lower edge of the T11 vertebral body and the upper edge of the T12 vertebral bone appears destroyed, while T12 has compression deformation. The plain MR scans of the lumbar spine (T1-weighted and T2-weighted) indicate that long T1, short T2 signal and short T1, long T2 signal shadows are visible in T11 and T12. Long T1 and slightly longer T2 signals are seen in the T12 vertebra. A reduction of the thoracic and lumbar segments of the disc is seen in T2WI.

Currently, both patients are using non-steroid anti-inflammatory drugs on demand, and can manage this by themselves in daily life. Mean-

while, spine surgeons advise that surgery should be performed at an appropriate time (in the event of pain intolerance, nerve compression, etc.).

Discussion

The coexistence of both hyperostosis and bone destruction can be manifested in AL, which can be easily misdiagnosed as tuberculosis. Dave et al reported in 2011 that 10 out of 14 patients studied were misdiagnosed as having spinal tuberculosis and received anti-tuberculosis treatment [4]. Since AL and spinal tuberculosis may affect intervertebral discs, which may lead to bone destruction and intervertebral space stenosis, as well as hyperostosis and osteosclerosis in later phases, clinicians should pay more attention to identifying them [5]. However, spinal tuberculosis is often characterized by toxic symptoms of tuberculosis, including low fever and night sweats. MRI manifestations include destruction in intervertebral discs and adjacent vertebrae, vertebral collapse, wedge change, severe parastylar damage, thoracolumbar kyphosis and abscess next to the vertebrae or in the psoas major muscle. Meanwhile, proactive and regular anti-tuberculosis treatments are quite effective [6]. When AL affects the vertebrae, such symptoms can be manifested as universal dissolution of the vertebrae complicated with tissue swelling and osteotylus formation, which shares similarities with neoplastic damage. The key points for differentiation lie in the fact that AL is not only limited to the vertebrae of the adjacent intervertebral discs, but also often afflicts intervertebral discs and terminal plates. Typically, pseudoarthrosis formed through generalized AL often afflicts the posterior column, and unhealed bone fractures can be seen [7-9]. Combined with a history of AS, a diagnosis of AL can be clearly made.

Before coming to our hospital, both of the patients described here suffered from recurring and deteriorating lumbago and backaches. Meanwhile, they were also afflicted with numbness in both lower limbs. In previous hospitals their conditions were diagnosed as spinal tuberculosis but no improvement was made through regular anti-tuberculosis therapy. After they were admitted to our hospital, comprehensive relevant examinations were completed. According to the characteristics of the patients' histories, including male, chronic course, HLA-B27 positive, sacroiliac joint fusion and bamboo-like changes to the spine, AS diagnosis is very clear. Lesions of the spine identified through imaging features, pathological biopsy and the ineffectiveness of previous anti-tuberculosis therapy which allowed us to exclude infections, tumors and other diseases, were clearly diagnosed as AS with AL.

Up to now, the pathogenesis of AS remains unclear. Although various biological agents have achieved certain results in the treatment of AS, there are still many patients with unpredicted pathological changes and the disease progresses rapidly [10-12]. For AL, we need to determine the pathogenesis through more clinical cases so as to be able to predict the possibility of this complication earlier. Furthermore, proactive internal medical measures should be adopted to control and postpone the development of these diseases so that it is mostly possible to avoid the progression of AL to the surgical stage.

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Disclosure of conflict of interest

None.

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