CASE REPORT

Andersson lesion in ankylosing spondylitis

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SUMMARY

A middle-aged male patient developed acute back pain and a lumbar vertebral lesion following trivial physical trauma. The lesion was considered as tuberculous on vertebral x-rays and MRI. After biopsy of the lesion and spinal fixation, the patient was kept on empirical antituberculous treatment (ATT) to which he did not respond. On re-evaluation he was diagnosed to have an Andersson lesion in ankylosing spondylitis (AS). ATT was stopped and he was successfully managed by rest, steroids, methotrexate and sulfasalazine. A careful look at the patient's plain x-ray spine and awareness about the lesion can avoid misdiagnosis of this characteristic vertebral lesion found in AS.

BACKGROUND

In a patient with an ankylosed spine having localised vertebral/disc/vertebral lesions on imaging, an Andersson lesion should be the first diagnosis to suspect. However, this lesion can be misdiagnosed due to various reasons like lack of awareness about the lesion and appearance of lesion on MRI suggestive of infection. Often, it is misdiagnosed as Pott's spine especially in high endemic areas for tuberculosis.

CASE PRESENTATION

A 45-year-old man presented to a neurosurgeon with acute low pain. Pain had started after a jerky motorbike ride and patient had become bed bound within 5 days. Neurological examination was normal, but movements of whole spine were restricted. Percussion tenderness was present over T12-L1 spine. Medical history included back stiffness for 5 years and diabetes mellitus for 3 years. There was no history of prior tuberculosis, weight loss, fever, cough or night sweats.

INVESTIGATIONS

Erythrocyte sedimentation rate and C reactive protein were 45 mm per hour and 10 mg/l, respectively. Blood counts and other serum chemistries were normal. x-Ray spine had syndesmophytes at lumbar, thoracic and cervical areas and an irregularity/destruction of the upper part of L1 vertebrae. MRI spine showed abnormally raised T2 hyperintensity in T12/L1 disc with erosions of adjacent endplates (figure 1A), subchondral bone marrow oedema of L1 and extradural cord compression without any myelopathic changes.

Suspecting it to be a tuberculous lesion, the neurosurgeon had done spinal fixation (figure 1B) by biopsy of the lesion and started him on empirical antituberculous treatment (ATT) with non-steroidal anti-inflammatory drugs (NSAIDs) and

Figure 1 (A) MRI spine showing abnormally raised T2 hyperintensity in T12/L1 disc with erosion in adjacent endplates, subchondral bone marrow oedema of L1 and extradural cord compression without any myelopathic changes. (B) x-Ray spine showing changes of ankylosing spondylitis in the spine, an Andersson lesion at T12/L1 and spinal fixation.

rest. Biopsy showed necrotic fibrocartilagenous tissue along with scanty inflammatory infiltrate comprising plasma cells, lymphocytes and macrophages. There were no epithelioid cell granulomas, multinucleated/Langhans giant cells, acid-fast bacilli (AFB) or malignant cells. Cultures were negative for aerobic bacteria and acid-fast bacilli.

DIFFERENTIAL DIAGNOSIS

- ▶ Andersson lesion.
- ▶ Pott's spine.
- ▶ Pyogenic infection.
- ▶ Malignant lesion.

TREATMENT

There was no significant improvement at 8 weeks on ATT. Patient visited Rheumatology department for a second opinion. On reviewing history and investigations (particularly, x-ray spine which showed clear changes of AS), patient was diagnosed to have ankylosing spondylitis (AS) with an Andersson lesion and ATT was stopped. Patient was started on intramuscular methylprednisolone acetate (80 mg/week for six doses), methotrexate (7.5 mg/week, which was increased to 15 mg/week at 2 weeks), sulfasalazine (1000 mg twice daily), folic acid (5 mg/week) and oral NSAIDs (naproxen 500 mg twice daily).

OUTCOME AND FOLLOW-UP

After 3 weeks of starting this treatment, patient had significant improvement in pain and started walking with support. At 6 weeks, he started

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walking without support. At 24 months follow-up, the patient is doing well on methotrexate 22.5 mg/week and sulfasalazine 1000 mg twice daily.

DISCUSSION

Localised vertebral or discovertebral lesion of the spine described by Andersson in 1937 is a known complication of AS with a reported prevalence of 1.5% to over 28%. Aetiology is either aseptic inflammation or mechanical factors (stress fractures). Different terms used to refer to these lesions include 'Discovertebral lesion', 'Vertebral lesion', 'Destructive vertebral lesion', 'Spondylodiscitis', 'Discitis', 'Diskitis', 'Sterile diskitis', 'Pseudoarthrosis' or '(stress) Fracture'. Lesions can be either 'Localised' or 'Extensive'. Radiologically lesions may be pseudodystrophic, pseudotuberculous, extensive erosions, bone condensation or isolated narrowing of intervertebral spaces. Anderson lesion usually involves only a single spinal level and most common affected site is thoracolumbar junction. Often it is misdiagnosed as tuberculous lesion.¹

Spinal tuberculosis is more common in children and young adults and is usually associated with constitutional symptoms (fever, weight loss and night sweats). Cold abscess around the lesion is another characteristic feature. MRI may show destruction of intervertebral disc and adjacent vertebral bodies, collapse of spinal elements and anterior wedging of vertebrae. Abscess formation, collection and expansion of granulation tissue adjacent to vertebral body are highly suggestive of spinal tuberculosis. Imaging-guided needle biopsy is the gold standard technique for early histopathological diagnosis. Histology confirms diagnosis in 60% cases by finding epithelioid cell granuloma, granular necrotic background, lymphocytic infiltration, scattered multinucleated and Langhans giant cells. Smear may be positive for AFB in 52% cases and culture is positive in about 83% cases. PCR assay for mycobacterium tuberculosis is another useful test for rapid diagnosis of spinal tuberculosis. The reported sensitivity of PCR ranges from 61% to 90% with a specificity of 80–90%.² PCR offers better accuracy than smear and can be performed at greater speed than cultures.²

Andersson lesion on MRI can mimic a malignant lesion. In case of metastatic spinal lesions intervertebral disc height is usually preserved but this may be affected in lymphoma and multiple myeloma. Vertebral endplates are also distinct and usually regular. The posterior vertebral segments are more extensively affected early in malignant lesions.

An acute presentation, no prior diagnosis of AS, appearance of lesion on MRI suggestive of infection, high prevalence of Pott's spine in endemic areas (suspicion bias) and lack of awareness about the Andersson lesion, can account for misdiagnosis at various levels. Misdiagnosis leads to unnecessary financial and psychological burden in form of unwanted investigations, biopsy, treatment with antibiotics, ATT and surgical intervention.

Learning points

- ▶ Localised vertebral/discovertebral lesions in an ankylosed spine on x-ray are characteristic of Andersson lesion.
- ▶ In endemic areas of tuberculosis, awareness about Andersson lesion can avoid misdiagnosis to tuberculous discitis/Pott's spine.
- ▶ Diagnosis of Andersson lesion can be well established by x-ray spine although CT or MRI may provide greater details of the lesion.
- ▶ Biopsy is not required for diagnosis of Andersson lesion in a typical case scenario.
- ▶ Conservative management is mainstay of treatment and surgical intervention is only required for progression of symptoms, unbearable pain, kyphotic deformity or neurological symptoms.³

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