CASE REPORT

Andersson Lesion in Ankylosing Spondylitis

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Abstract

Andersson lesions are destructive foci that appear at the discovertebral junction in ankylosing spondylitis. We report three cases of ankylosing spondylitis with such lesions. These lesions simulate an infection and in our country, mimic spinal tuberculosis.

Key words

Ankylosing spondyilitis; Andersson lesion

Introduction

Ankylosing spondylitis is a chronic inflammatory disease involving the axial skeleton and the peripheral joints with extraarticular manifestations. Radiological changes are observed in the spine throughout the course of the disease and discovertebral lesions are one amongst them (1). The common sites involved are the lower thoracic and upper lumbar vertebrae. Cawley and colleagues classified them into 3 types. Type1 involves the central portion of the discovertebral junction that is covered by cartilage, Type 2 lesions involve the peripheral portion of discovertebral junction not covered by cartilaginous endplate and Type 3 involves both the peripheral and central portions (2). All three types manifest in ankylosing spondylitis.

Case No 1

A 34 year old male, a known case of HLA B27 positive ankylosing spondylitis of fifteen years duration had a trivial fall following which he developed pain localized to the lower dorsal region, urinary incontinence and difficulty in walking. X-ray pelvis showed bilateral sacroiliitis. X-ray of the dorsolumbar spine showed squaring of vertebrae, presence of syndesmophytes, widening of disc space at D11-12 level with adjacent end plate irregularity and ill defined sclerosis suggestive of discitis and spondylitis with fracture of the ankylosed spine at D 11-12 (Andersson lesion- Type 3).

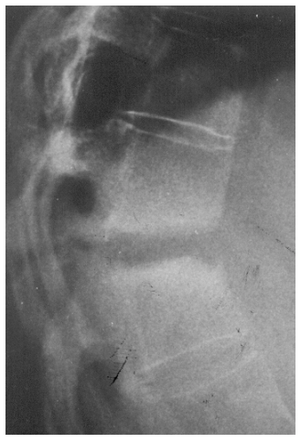

Fig. 1: X-ray dorsolumbar spine showing Andersson Type-3 lesion at D11-12 with associated fracture of ankylosed spine.

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Case No 2

A 43 year old male symptomatic for 13 years with insidious onset of low backache subsequently developed progressive kyphosis, neck pain, breathlessness and pain over the thoracolumbar junction. He had no peripheral joint involvement. X-ray pelvis showed bilateral sacroiliitis. X-ray dorsolumbar spine showed squaring of vertebrae with lateral syndesmophytes and calcification of anterior spinal ligament consistent with ankylosing spondylitis. In the superior margin of D11 vertebral body, central form of disco-vertebral erosion (Type1 Andersson lesion) was observed. At the D12- L1 level, mild wedge compression with anterior disco-vertebral erosion (Type2 Andersson lesion) was seen.

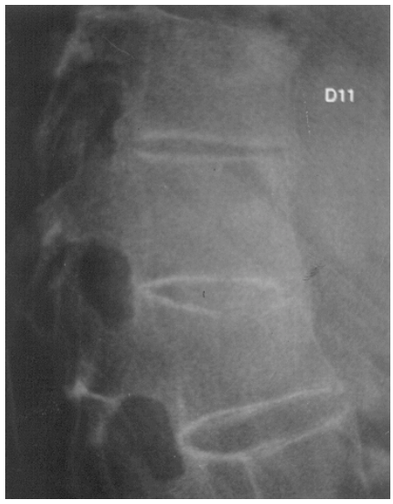

Fig. 2: X-ray dorsolumbar spine showing Andersson Type-1 lesion at D11-12 and Type-2 lesion at D12-L1 level.

Case No 3

A 37 year old male, a known case of juvenile onset ankylosing spondylitis of 25 years duration with peripheral joint involvement, developed neck pain with restricted movements and severe lumbar pain following a trivial fall. X-ray pelvis showed bilateral sacroiliitis and joint space reduction in both hip joints. X-ray of the lumbar spine showed squaring of the vertebrae and presence of syndesmophytes. L4-L5 disc space showed widening, irregularity of adjacent end plates centrally and erosions anteriorly consistent with Andersson Type 3 lesion and fracture in the posterior segment.

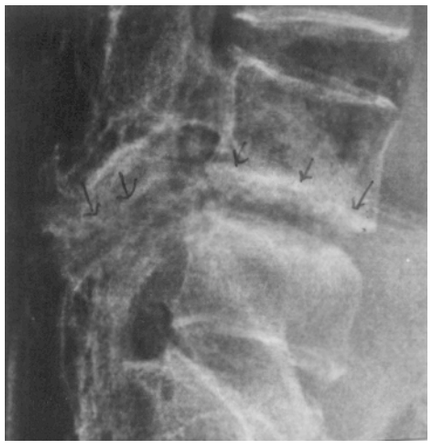

Fig. 3: X-ray lumbar spine showing Andersson Type-3 lesion at L4-L5 with fracture in the posterior segment.

Discussion

Discovertebral lesions are frequently termed Andersson lesions who described them in two patients with ankylosing spondylitis in 1937. Cawley *et al* classified them into three types (2). Many reports have emphasised on destructive abnormalities at discovertebral junction in this disorder. These lesions have been observed in the early and late phases of the disease and occur in traumatic and nontraumatic situations (3).

Type 1 lesions are localised to the central subchondral portions of the discovertebral junction and this can occur in both ankylosed and non-ankylosed spines. Osteoporosis of vertebrae is marked in ankylosing spondylitis. This results in weakening of the subchondral bone and displacement of the discal contents through the cartilaginous end plate into the vertebral body. Apophyseal joint involvement may lead to instability and recurrent traumatic insult to the disc-bone interface. This eventually produces infarction of cartilaginous endplate allowing the discal material into the vertebral body. Inflammatory changes in subchondral bone itself may

lead to osseous weakening and discal displacement (4). Radiologically the lesions appear as irregularity of central portion of the superior and inferior vertebral margins with surrounding sclerosis.

Type2 -These are peripheral localized lesions occuring in the anterior or posterior part of discovertebral junction. The anterior lesion is attributed to collapse of osteoporotic anterior vertebral margin as occurs in osteoporotic kyphosis and also alternatively to injury to the anterior fibres of annulus fibrosus leading to invasion and replacement of discal material by vascular fibrous tissue as in senile kyphosis.The mechanism of the localised posterior lesions though not clear may be due to osteoporotic collapse or cartilagenous nodes. Inflammation of the outer fibres of the annulus fibrosus related to spondylitic process may also play a role. Radiologically Type 2 lesions are seen as intervertebral disc space narrowing with bony sclerosis, irregularity of discovertebral junction and anterior or posterior discovertebral erosion with intact apophyseal joints.

Type3 Destruction of the whole discovertebral junction of two adjacent vertebral bodies occurs in patients with advanced ankylosis. There may be a history of trauma and associated fracture through the ankylosed portion. There may be improper healing with callus formation, heamorrhage and minimal inflammatory changes.The radiological appearance of this type is a combination of Type1 and Type 2 lesions.

The radiological features of all types of Andersson lesion including disc space narrowing, destruction of vertebral end plate and sclerosis of adjacent bone mimic infective spondylitis particularly due to tuberculosis (5).

Among the 3 patients of established ankylosing spondylitis the first patient had a disease duration of 15years with postural deformities. He had a trivial fall and subsequently developed localized pain, gibbus and neurological symptoms. The second patient had a disease duration of 13 years, with kyphosis and no history of fall but developed localized dorsal pain. The third patient who had a juvenile onset disease of 25 years duration had a minor trauma and developed localized lumbar pain.

Infection was ruled out in all three cases. Case 1 who had neurological symptoms was subjected to decompression surgery. The material removed showed lamellar bony spicules and areas of heamorrhage consistent with Type3 lesions. Case 2 and 3 were managed conservatively with immobilization and analgesics.

The significance of Andersson lesion is that it occurs frequently in anklylosing spondylitis of longer disease duration and mimics infection. Knowledge of these lesions and thorough screening including radiography help us to avoid subjecting these patients to treatment strategies for infection. Rest is desirable in this situation and physiotherapy is not.

References

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