

SUDDEN INCREASE OF NECK FLEXION IN ANKYLOSING SPONDYLITIS: QUO VADIS?

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To the editor,

A 55-year-old man with ankylosing spondylitis (AS) was seen for a control visit during anti-TNF- α treatment. On detailed questioning, he described a sudden increase in the neck flexion after a fall episode two weeks ago. Other than his ongoing mild spinal pain and morning stiffness, he denied having any other painful complaints. The medical history was consistent with AS for 23 years and bilateral hip prosthesis surgery. He was using sulphasalazine 2 g/day, etanercept 25 μ g twice weekly and a nonsteroidal anti-inflammatory drug. On physical examination, all spinal motions were completely limited except a slight neck flexion (not present in his previous visits). A substantial neurological examination was performed to rule out any spinal cord compromise and it was unremarkable. Laboratory evaluations including complete blood count, liver and renal function tests, erythrocyte sedimentation rate and C-reactive protein were normal. Cervical radiographs were noncontributory and as one of the hip prostheses of the patient was magnetic resonance incompatible, computed tomography was performed. Disruption of the anterior longitudinal ligament at the level of T1-T2 vertebrae was detected (Fig. 1). Cervical collar was prescribed along with reassurance of the patient with regard to spinal trauma.

As the disease progresses over several years, the spine becomes more rigid and deformed in AS, susceptible to major injuries even from minor trauma.¹ Accompanying osteoporosis definitely contributes to the eventual scenario of vertebral fractures. The findings that suggest a spinal fracture in a patient with AS would include pain precipitated by activity, change in posture or in their field of view.² Further, neurological deficits which may even require extensive surgery for neural decom-

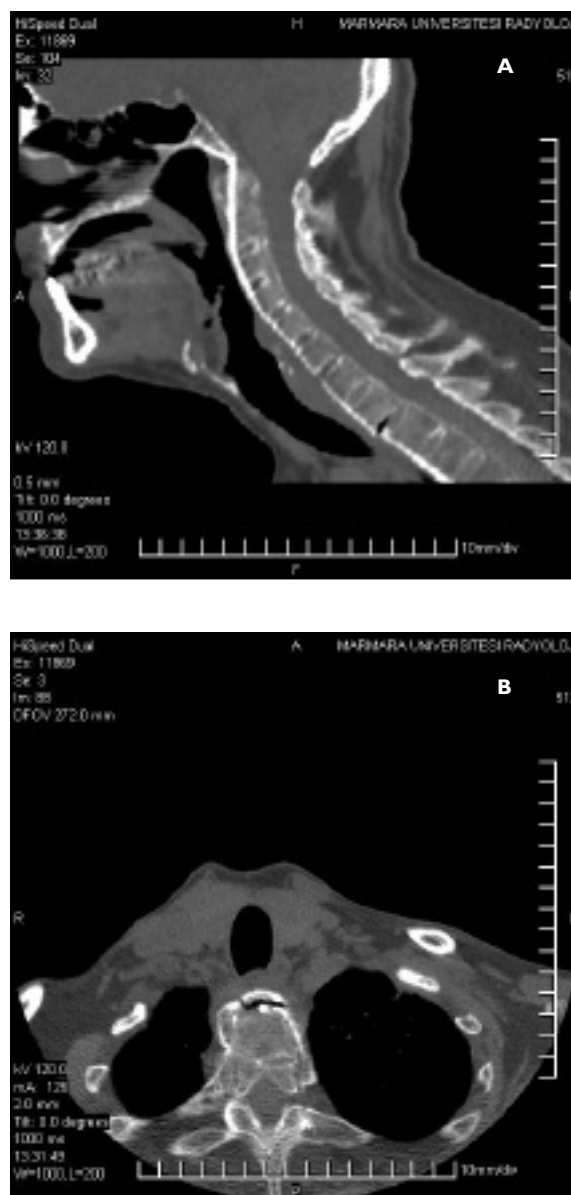


Figure 1. Computed tomography of the patient, reformatted sagittal (A) and axial (B) views demonstrating disruption of the anterior longitudinal ligament at T1-T2 level.

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pression and spinal stability may also follow.³

Overall, the reason we report our patient was two-fold. First, we would like to call attention of physicians to the increased risk of vertebral fractures and ligament disruptions in AS patients. Second, as in our case, it is noteworthy that these fractures might well be asymptomatic other than an increased spinal motion. Therefore, they may otherwise be easily overlooked unless patients are thoroughly evaluated with detailed questioning and prompt physical examination. Last but not least, computed tomography or MRI seems to be promising for better demonstration of these fractures once the diagnosis is on the agenda.

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