Images in...

Discitis with vertebral body destruction in a 28-year-old intravenous drug user

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DESCRIPTION

A 28-year-old hepatitis C positive, intravenous drug user presented with a 2-month history of sharp pain over the thoracic vertebrae that was referred to the upper epigastrium. There was tenderness over the upper epigastrium and thoracic vertebrae. No neurological deficit was found on examination. The patient's temperature was 37.5 °C. Initial blood results showed a white cell count of $12.0 \times 10^9/l$, haemoglobin of 87 g/l and a C reactive protein of 240. Blood cultures revealed a *Staphylococcus aureus* bacteraemia.

Following the initial presentation, the patient's Glasgow Coma Scale dropped to 13/15. A CT of the chest, abdomen and pelvis revealed destructive change affecting T8 and T9 and the T8/9 disc space with over half of the T8/9 vertebral body being destroyed consistent with a T8/9 discitis (figure 1A). An urgent MRI spine revealed surrounding inflammatory changes extending into both adjacent vertebral bodies at T8/9 (figure 1B). The posterolateral ligament was displaced posteriorly reducing the spinal canal diameter without focal changes in the spinal cord. Treatment was with bed rest, CT guided disc drainage and a 6-week

course of intravenous antibiotics with orthopaedic follow-up (figure 1B).

While discitis usually presents in patients over the age of 65, it is important to consider the diagnosis in high risk individuals such as alcoholics, diabetics and intravenous drug users who present with spinal tenderness even if there is no neurological deficit.¹ The most common causative organism is *S aureus*.² The value of CT and MRI must also be emphasised in providing information about the extent of damage due to the discitis, especially because if antimicrobial therapy is unsuccessful, surgery should be considered.¹

Competing interests None.

Patient consent Obtained.

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Figure 1 (A) CT image showing vertebral destruction at the level of T8/9. (B) MRI image showing vertebral collapse and spinal cord displacement without intrinsic cord signal change.

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