An unusual case of sepsis? A rare presentation of a common disease

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DESCRIPTION

A 22-year-old Algerian man attended hospital with a 20-day history of non-specific abdominal pain and vomiting. Concurrently, the patient reported non-intentional weight loss and fevers/night sweats, but denied any neurological, respiratory or cardio-vascular symptoms. The patient had an extensive travel history throughout North Africa and Europe.

Clinical examination revealed general upper abdominal and bilateral flank pain. Respiratory, cardiovascular and neurology examinations were normal. Vital observations revealed fever of 39.3°C, but were otherwise normal. Initial blood tests demonstrated an elevated C reactive protein (CRP) of 92 mg/L, but normal white cell count and full blood cell count, liver function tests, and urea and electrolytes. The serology for HIV was negative. Admission chest X-ray was normal. CT of the abdomen/pelvis was undertaken: a large paravertebral, septated collection (figure 1A) and scalloping of the T10 and T11 vertebrae were demonstrated. A subsequent CT of the chest revealed cranial extension of the previously described paraspinal collection to the level of

the T6/7 disc space (figure 2A). A 10-french pigtail drain was placed under CT guidance: PCR of the drained fluid revealed *Mycobacterium tuberculosis* DNA and standard quadruple *M. tuberculosis* treatment (encompassing pyrazinamide, isoniazid, ethambutol and rifampicin) was started. Later, *M. tuberculosis* culture revealed full sensitivity to the four chemotherapeutic agents. MRI of the thoracic and lumbar spine illustrated a large multilocular perivertebral abscess (figures 1B, 2B and 3), but no spinal cord compression.

Two days after starting standard quadruple *M. tuberculosis* therapy, the patient deteriorated haemodynamically with fever, hypotension and tachycardia, requiring intensive care unit admission for aggressive fluid resuscitation, however, inotropes were not required. The white cell count elevated to 25.1×10⁹/L, CRP to 233 mg/L and lactate to 3.5 mmol/L despite fluid resuscitation. Clinically,



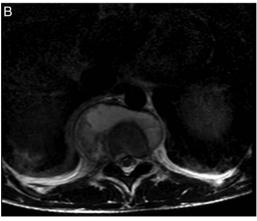
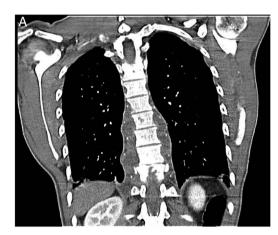


Figure 1 (A) Axial CT and (B) axial T2-weighted MRI demonstrating a large, septated prethoracic vertebral abscess. Anterior displacement of the oesophagus is illustrated.



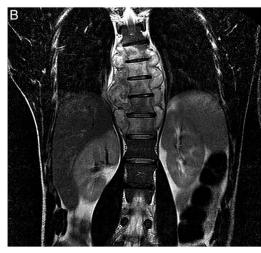


Figure 2 (A) Coronal CT and (B) coronal T2-weighted MRI illustrating a paravertebral abscess of the lower thoracic vertebrae.



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Images in...

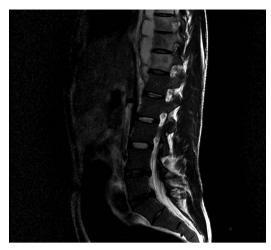


Figure 3 Sagittal T2-weighted MRI demonstrating a prevertebral abscess of the lower thoracic and lumbar vertebrae; anterior displacement of the anterior vertebral ligament is illustrated.

the patient reported malaise and fevers, but systemic examinations were normal and a septic screen (encompassing a chest X-ray, and blood, sputum and urine cultures) was negative. Multidrug resistant/extensive-drug resistant *M. tuberculosis* (sensitivities were not known at this time), a paradoxical response to the *M. tuberculosis* treatment, an unidentified source of bacterial sepsis or *M. tuberculosis* sepsis were differential diagnoses.

More than 2 billion people worldwide are infected with *M. tuberculosis*, including 9.3 million new cases and 1.7 million fatal cases per annum.¹ It is postulated that *M. tuberculosis* sepsis pathophysiology is mediated by lipoarabinomannan, a component of the *M. tuberculosis* cell wall, which stimulates tumour necrosis factor while inactivating macrophages.²

In light of the haemodynamic changes, proximity of the *M. tuberculosis* abscess to the central nervous system and nonspecific symptoms potentially indicative of *M. tuberculosis* meningitis, the patient was treated with high-dose steroids. After continued standard quadruple *M. tuberculosis* treatment and abscess drainage, the patient improved and was discharged.

Learning points

- Mycobacterium tuberculosis sepsis is rare, especially in immunocompetent patients; it occurs most often in immunocompromised patients, notably patients with HIV.²
- ▶ Initiating M. tuberculosis chemotherapy can evoke worsening of disease or new lesions, termed paradoxical reactions, postulated to be due to the liberation of tuberculoproteins.³

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