

CLINICAL VISTAS BRIEFS

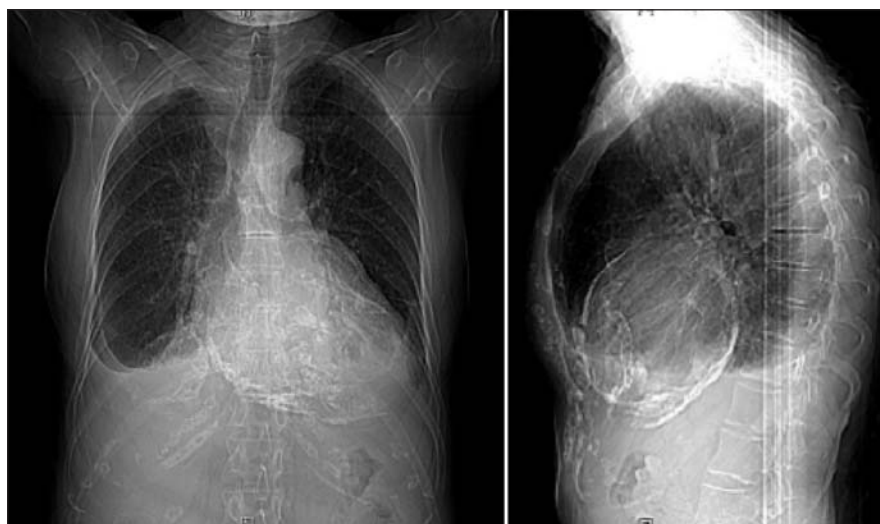
What's your call?



CT scan (left) and chest radiograph (right) of a 37-year-old homeless man whose pulmonary tuberculosis had been treated in hospital 5 months before presentation. He returned to hospital after losing 5 kg. Over the preceding 2 months, he had experienced anorexia, low-grade intermittent fevers and a productive cough.



CT scan of the lumbar spine of a 26-year-old man from India who presented with low back pain, fever and weight loss of 3 weeks' duration.



Chest radiographs of a 76-year-old woman with previous tuberculosis, progressive dyspnea and peripheral edema.

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Disseminated tubercular osteitis

A 26-year-old man from India who had lived in France for 1 year experienced low back pain, fever and weight loss. After 3 weeks of it, he came to hospital. His medical history included kidney stones and alcohol abuse, but was negative for tuberculosis. He denied having any weakness or numbness of the lower limbs, and the results of a neurologic examination were normal. HIV test results were negative. Plain radiographs appeared normal. However, a CT scan of the spine revealed multiple lytic, well-defined osseous lesions, with surrounding sclerosis and central opacity disseminated in his lumbar vertebrae and sacrum (Figure 1; additional images available online at www.cmaj.ca/cgi/content/full/177/2/148-a/DC1). We observed no disc extensions or soft-tissue abscesses. A technetium-99 bone scan showed no hyperfixation of the lesions, although some found on the sacrum and right ilium via CT showed hyperfixation with positron emission tomography.



Figure 1: An axial reconstruction of a CT scan of the lumbar spine (the L3 vertebra, in this image), with use of a soft-tissue filter. Small, round, lytic bone lesions were found with well-defined margins, surrounding sclerosis and central opacity (arrowheads). This central opacity is called a button sequestrum sign.

Specimens of the L5 vertebral body obtained via percutaneous biopsy showed several tuberculous granulomas with a central mass of epithelioid cells, some giant cells and surrounding lymphocytes with no caseous focus. No acid-fast bacilli were found in the specimen, and cultures remained negative. Nevertheless, a specimen biopsied from an enlarged cervical lymph node yielded acid-fast bacilli later identified as *Mycobacterium tuberculosis*. A diagnosis of disseminated tubercular osteitis (cystic tuberculosis) with sequestra was made.

The patient underwent 2 months of daily chemotherapy with isoniazid (300 mg), pyrazinamide (15–30 mg/kg), rifampin (600 mg) and ethambutol (15 mg/kg), followed by 22 months of daily isoniazid (300 mg) plus rifampin (600 mg). He also wore external bracing for the first 8 months of his treatment. His clinical course was favourable, and his back pain completely resolved within 5 weeks. Although a CT scan 6 months after the biopsies showed the same bone lesions at the same size, their sclerotic margins were thicker, a possible sign of cicatrization. At that time, there had been no sign of recurrence.

Osteoarticular tuberculosis occurs in 2.5%–5% of tubercular infections; of these, 50%–60% are vertebral, most frequently in lower thoracic and lumbar regions. They result from an arterial hematogenous seeding.

An opaque, radiodense centre in a lytic lesion is called a button sequestrum sign. Originally described as a manifestation of eosinophilic granuloma,¹ the sign may also be seen in cases of osteomyelitis, fibrosarcoma or lymphoma.² The pathophysiology of tuberculous osteomyelitis is believed to involve granulation tissue initially formed in the marrow, with secondary resorption of trabeculae. Caseous necrosis creates an abscess cavity that contains pus and small granules of bone. The abscess is surrounded by granulation tissue, connective tissue with cellular elements and sclerotic trabeculae.^{2,3} Radiographs show foci of osteolysis, with condensation and periostitis. Sequestrum formation may manifest as a central increase in radioden-

sity within the lytic area, producing the button sequestrum sign.

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Cystic tuberculous constrictive pericarditis

A 76-year-old woman with a history of childhood tuberculosis was examined before admission to a tuberculosis sanatorium. Over the past 4–5 years, she had been experiencing peripheral edema and episodes of shortness of breath upon exertion. Her medical history included long-standing atrial fibrillation. Her family doctor had prescribed diuretics, an angiotensin-converting enzyme inhibitor and a β -blocker.

Although she was normotensive upon examination, her neck veins were grossly distended and peripheral edema was obvious. Kussmaul's sign was present, and she had vague distant heart sounds and a distinct pericardial knock. Angiography showed that her coronary arteries were normal. Although her systolic function was also normal, she had an end-diastolic filling impairment consistent with constrictive pericarditis.

A CT scan revealed calcification on the undersurface of the heart, along its posterior and anterior aspects, and over