

CLINICAL VISTAS BRIEFS

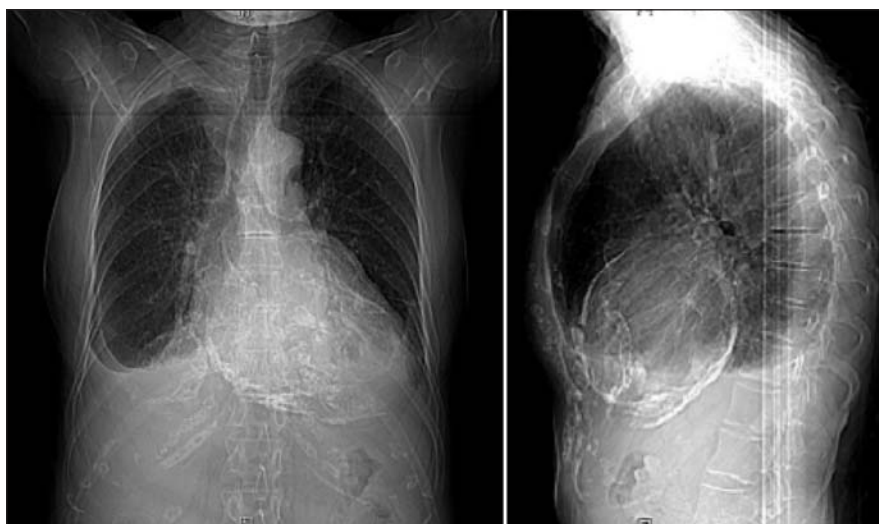
What's your call?



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.

See page 151 for diagnoses.

CLINICAL VISTAS BRIEFS

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.

Pierre Sellier MD

Department of Internal Medicine

Annabelle Wyler MD

Department of Radiology

Lariboisière Hospital

Paris, France

This article has been peer reviewed.

Competing interests: None declared.

Acknowledgements: We thank Drs. Myriam Diemer, Karine Champion and Valerie Bousson for their help in the preparation of this manuscript.

REFERENCES

1. Rosen IW, Nadel HI. Button sequestrum of the skull. *Radiology* 1969;92:969–71.
2. Krasnokutsky MV. The button sequestrum sign. *Radiology* 2005;236:1026–7.
3. Resnick D. Osteomyelitis, septic arthritis, and soft tissue infection: organisms. In: *Diagnosis of bone and joint disorders*. 4th ed. Philadelphia: WB Saunders; 2002. p. 2535–8.

DOI:10.1503/cmaj.070146

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

the cardiac apex (Figure 1). On the anterior cardiac surface a lenticular cystic mass, 8 cm in its longest dimension, was visibly compressing the right ventricular outflow tract.

During a median sternotomy, the cyst was explored in the anterior mediastinal area (Appendix 1; available online at www.cmaj.ca/cgi/content/full/177/2/148-b/DC1). The cyst contained a brown caseous liquid, which was evacuated. Radical pericardectomy without cardiopulmonary bypass was performed, with no intraoperative complications. The patient's postoperative course was uneventful. A specialist in infective diseases was consulted; since the resected tissue showed no active bacteria and the patient had no signs of active disease, no further interventions were suggested.

After discharge from hospital, the patient was no longer bothered by shortness of breath on exertion. At follow-up, she exhibited no physical signs of heart failure.

Constrictive pericarditis, often the result of fibrosis and calcification, can be a long-term consequence of pericarditis, either acute or chronic. Rigid, heavily fibrosed or even calcified pericardium restricts the myocardium and prevents adequate ventricular filling after an initial expansion. Tuberculous pericarditis, which occurs in 1%–2% of cases of pulmonary tuberculosis, progresses to a constrictive form even more rarely.¹ Constrictive tubercular pericarditis commonly arises via extension from pulmonary disease; however, miliary spread to the pericardium can also occur.

Although this patient's symptoms arose because of untreated pulmonary tuberculosis, constrictive calcific pericarditis can result from many processes, including chest trauma, the aftereffects of radiation, connective tissue diseases (e.g., lupus, rheumatoid arthritis) and infections (e.g., tuberculosis, histoplasmosis). In 30% of cases, a cause is not identified.² Most patients with constrictive pericarditis arrive with signs of heart failure, including dyspnea and peripheral edema. The treatment of choice in symptomatic patients is pericardectomy.

Kristopher Patrick Croome MD BScH

Department of Surgery

Mark S. Landis MD MSc

David Bach MD

Department of Diagnostic Radiology
and Nuclear Medicine

Michael W.A. Chu MD MEd

Department of Cardiac Surgery

Neil McKenzie MD

Department of Cardiac Surgery

University of Western Ontario

London, Ont.

This article has been peer reviewed.

Competing interests: None declared.

REFERENCES

1. Larrieu AJ, Tyers GF, Williams EH, et al. Recent experience with tuberculous pericarditis. *Ann Thorac Surg* 1980;29:464-8.
2. Bertog SC, Thambidorai SK, Parakh K, et al. Constrictive pericarditis: etiology and cause-specific survival after pericardectomy. *J Am Coll Cardiol* 2004;43:1445-52.

DOI:10.1503/cmaj.061494

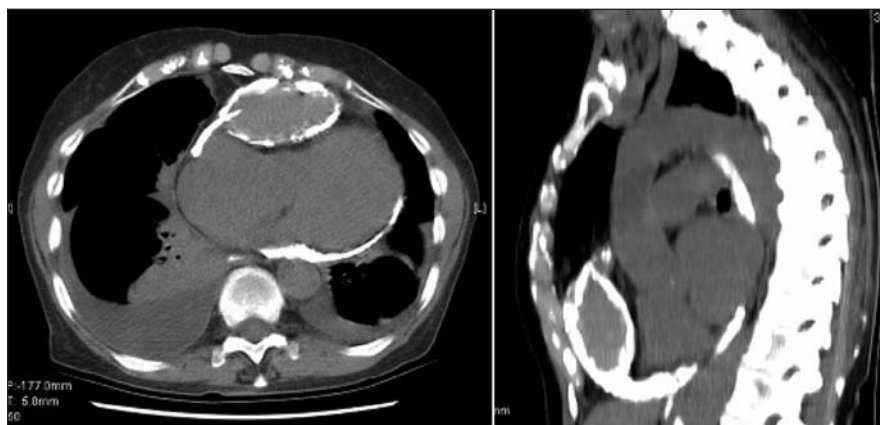


Figure 1: A CT scan revealing a large, anterior calcified pericardial abscess and circumferential pericardial calcification.

Canadian Adverse Reaction Newsletter Bulletin canadien des effets indésirables

To receive the Newsletter and health product Advisories free by email,
join Health Canada's **MedEffect** mailing list.
Go to www.healthcanada.gc.ca/medeffect

Inscrivez-vous à la liste **MedEffet** de Santé Canada pour recevoir gratuitement par
courriel le Bulletin et les Avis au sujet des produits de santé. Rendez-vous à l'adresse
www.santecanada.gc.ca/medeffet

**Report adverse reactions toll free to Health Canada
Signaler sans frais des effets indésirables à Santé Canada**

Tel./Tél. : 866 234-2345 • Fax/Télec. : 866 678-6789

