Case report

Acute vertebral osteomyelitis complicating
Streptococcus sanguis endocarditis

CHRISTINE DEMERS, MICHEL TREMBLAY, AND YVES LACOURCIÈRE
From the Departments of Medicine, Pathology and Nuclear Medicine, Le Centre Hospitalier de l’Université Laval, Québec, Canada G1V 4G2

SUMMARY The first well documented case of acute pyogenic vertebral osteomyelitis presenting as the initial manifestation of Streptococcus sanguis endocarditis is reported. The importance of suspecting vertebral osteomyelitis in the presence of disc infection and the diagnostic value of imaging procedures are underlined.

Although low back pain accounts for 25–33% of musculoskeletal manifestations of bacterial endocarditis, the finding of pyogenic vertebral osteomyelitis is extremely rare.1–3 As highly sensitive screening procedures designed for infectious bone disease enhance the diagnostic capabilities of the clinician, and most patients with this condition respond well to antibiotic therapy, very few cases have been confirmed either by biopsy or at necropsy.

We report the first case recorded at necropsy of a patient who presented with vertebral osteomyelitis as the primary manifestation of S sanguis endocarditis.

Case report

A 64 year old man with no significant previous illnesses and no history of recent injury or dental manipulation was admitted to hospital in August 1985 with a four week history of severe low back pain exaggerated by standing and motion and only partially relieved by recumbency. Neurological symptoms were absent. One month previously he had developed chills, sweating, fatigue, anorexia, and a 15 pound weight loss. On examination the patient was pale and prostrated. His temperature was 38.7°C. Mouth examination showed no evidence of dental caries or gingivitis. Pertinent cardiac findings included supine blood pressure 150/70 mmHg, pulse 88/min, and a grade 3/6 systolic ejection murmur along the left sternal border radiating to the neck. There was no peripheral oedema and the lung bases were clear. Examination of the lower back showed exquisite tenderness on palpation and percussion of L2, L3, L4, spasm of the paravertebral musculature, and moderate limitation of back motion in all directions. Neurological examination was unremarkable.

INVESTIGATIONS Investigations showed haemoglobin 94 g/l, white cell count 10.7×10⁹/l with a normal differential, platelets 198×10⁹/l, erythrocyte sedimentation rate 50 mm/h.

Renal function was normal, but there was a mild derangement of the liver enzymes. Chest x ray was normal and the electrocardiogram showed left ventricular hypertrophy. Lumbar spine x ray findings were consistent with degenerative disc disease and computed tomography suggested discitis of L3, L4. Increased uptake in the area of L2, L3, L4 compatible with osteomyelitis was observed on both technetium-99m labelled methylene diphosphonate (¹⁷⁰Tc MDP) and gallium-67 (⁶⁷Ga) scans (Fig. 1). An alpha-haemolytic streptococcus identified as S sanguis type I was isolated from the aerobic bottles of six blood cultures taken at intervals of 60 minutes over a two day period.

An echocardiogram disclosed left ventricular hypertrophy and vegetations on the aortic valve.

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Correspondence to Dr Yves Lacourcière, Department of Medicine, Le Centre Hospitalier de l’Université Laval, 2705, boulevard Laurier, Ste-Foy, Québec, Canada G1V 4G2.
COURSE OF DISEASE

Therapy was instituted with penicilline G, 24 million units a day intravenously and gentamicin 240 mg a day intravenously. Rapid defervescence and amelioration of back pain was noted. Twelve days after initiation of antibiotics the patient developed left ventricular failure with bilateral pleural effusion which responded temporarily to medical treatment. Aortic valve replacement was considered, but he died two days later of intractable acute pulmonary oedema.

NECROPSY

The heart showed typical lesions of infective endocarditis with both mitral and aortic valves showing vegetations and ulcerations affecting the margins of mitral valve and the cusps of aortic valve (Fig. 2). Gross findings in the lumbar vertebrae (L2, L3...

Fig. 1 The $^{99m}$Te MDP scan shows increased uptake in the second, third, and fourth vertebra. The $^{67}$Ga scan also demonstrates enhanced uptake in the area.

Fig. 2 Gross findings in the heart with aortic cusps ulcerated and covered by friable vegetations.
Vertebral osteomyelitis complicating $S$ sanguis endocarditis

Fig. 3a

Fig. 3 (a) Longitudinal section of lumbar vertebrae with irregular cavity destroying the bone; (b) high magnification showing granulation tissue and necrosis with irregular bony trabecula.

L3, L4) showed a 5 cm cavity surrounded with friable and necrotic bone (Fig. 3a). Histology showed subacute osteomyelitis (Fig. 3b). Bacterial cultures of the specimens were negative.

Discussion

This is the first case recorded at necropsy of $S$ sanguis endocarditis in a patient who presented with acute lumbar vertebral osteomyelitis. We identified 49 cases in the world published reports of vertebral osteomyelitis during the course of endocarditis and found that only one (which involved $S$ aureus) had been fully documented at necropsy. The failure to culture the causative agent in valvular and bone specimens in our patient is not surprising as he had received antibiotic therapy for 15 days. Both macroscopic and histological findings, however, were compatible with the diagnosis of endocarditis and osteomyelitis. Many features of this case are similar to those of previously published reports. In fact low back pain antedating the diagnosis of endocarditis by one month, weight loss, characteristic physical findings, and rapid improvement after institution of antibiotic therapy are consistent with previous descriptions.

Discitis and osteomyelitis are usually considered in the literature as separate complications of bacterial endocarditis. In a review of bacterial endocarditis disc space infection was thought to be present in five of 84 (6%) patients with musculoskeletal manifestation and osteomyelitis in none. As the vascular supply of the intervertebral disc is lost with aging it seems reasonable to suggest that pyogenic infection of this structure occurs after osteomyelitis has spread beyond the vertebrae. Thus disc space narrowing or discitis, or both, disclosed by radiological studies in the course of bacterial endocarditis may suggest the presence of an associated vertebral osteomyelitis.

Pyogenic vertebral osteomyelitis must be recognised rapidly because delays in diagnosis and
treatment can result in unnecessary surgery, serious neurological complications, and a high mortality.  

Spine x rays are usually consistent with the diagnosis in 50% of cases with abnormalities such as narrowing of the disc space, sclerosis, or erosion of the end plates and destruction of the vertebral body. Fifty per cent of patients' x rays are normal, however, or consistent with degenerative arthritis, as in our patient. In the early stage of the disease, computed tomography may be negative or disclose disc hypodensity, an early sign of vertebral osteomyelitis. The diagnostic value of radionuclide bone imaging has been well established, with a sensitivity of 100% for a 99mTc MDP bone scan. A positive 67Ga scan increases the specificity when pyogenic vertebral osteomyelitis is suspected in the course of bacterial endocarditis.

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References