

sion produced by adenosine decreased perfusion to a compromised end-artery. Concomitant localised vascular steal may have contributed to regional hypoperfusion of the cerebellum, although any relationship between adenosine and neurologic abnormalities remains conjectural.

Learning point

Adenosine perfusion imaging is a useful diagnostic technique with a very low rate of significant adverse effects. Heightened suspicion for an ischaemic event is, however, warranted in patients with known or suspected vascular disease whose adverse symptoms do not resolve promptly

- 1 Wilson RF, Wyche K, Christensen BV, Zinuner S, Laxson DD. Effects of adenosine on human coronary arterial circulation. *Circulation* 1990; **82**: 1950-66.
- 2 Watt AH, Penny WJ, Sigh H, Routledge PA, Henderson AB. Adenosine causes transient dilatation of coronary arteries in man. *Br J Clin Pharmacol* 1987; **24**: 665-8.
- 3 Ogilby JD, Iskandrian AS, Untereker WJ, Reo J, Nguyen TN. Effect of intravenous adenosine infusion on myocardial perfusion and function. *Circulation* 1992; **86**: 887-95.
- 4 Belardinelli L, Linden J, Berne RM. The cardiac effects of adenosine. *Prog Cardiovasc Dis* 1989; **32**: 73-97.
- 5 Patterson RE, Kirk ES. Coronary steal mechanisms in dogs with one-vessel occlusion and other arteries normal. *Circulation* 1983; **67**: 1009-15.
- 6 Epstein SE, Cannon RO, Talbot TL. Hemodynamic principals in the control of coronary blood flow. *Am J Cardiol* 1985; **56**: 4E-10E.
- 7 Cerqueira MD, Verani MS, Schwaiger M, Heo J, Iskandrian AS. Safety profile of adenosine stress perfusion imaging: results from the Adenoscan Multicenter Trial Registry. *J Am Coll Cardiol* 1994; **23**: 384-9.
- 8 Coyne EP, Belvedere DA, Vande Streek PR, Weiland FL, Evans RB, Spaccavento LJ. Thallium-201 scintigraphy after intravenous infusion of adenosine compared with exercise thallium testing in the diagnosis of coronary artery disease. *J Am Coll Cardiol* 1991; **17**: 1289-94.
- 9 Gupta NC, Esterbrook DJ, Hillman DE, Mohiuddin SM. Comparison of adenosine and exercise thallium-201 single photon emission computed tomography myocardial perfusion imaging. *J Am Coll Cardiol* 1992; **19**: 248-57.
- 10 Klabunde RD. Dipyridamole inhibition of adenosine metabolism in human blood. *Eur J Pharmacol* 1983; **93**: 21-6.

Symmetrical Brodie's abscess

AFW Chamblor, PJ Chapman-Sheath, MF Pearse, J Hollingdale

Summary

Chronic recurrent multifocal osteomyelitis is often confused with symmetrical Brodie's abscess as it has a similar pathogenesis. We report an otherwise healthy 17-year-old boy presenting with a true symmetrical Brodie's abscess. We conclude that a symmetrical Brodie's abscess presenting in an otherwise healthy patient is a separate clinical condition with a different management protocol.

Keywords: tibia, Brodie's abscess, osteomyelitis

Brodie's abscess is defined as a centrally placed, sharply circumscribed, lytic lesion in the metaphysis adjacent to the growth plate. Sir Benjamin Brodie first described a subacute bone abscess in 1832. Chronic recurrent multifocal osteomyelitis is often given the name of symmetrical Brodie's abscess in error, although they have a similar pathogenesis. We report an otherwise healthy 17-year-old boy presenting with a true symmetrical Brodie's abscess.

Case report

An Asian boy aged 17 years attended orthopaedic out-patients complaining of pain in both lower legs. He located the pain to the distal end of both tibiae. The pain had become more severe recently, stopping him from participating in sporting activities. He de-

scribed the pain as dull in nature with no radiation, and troubling him at rest and at night. There was no history of trauma or foreign travel. There were no hereditary haematological disorders within his family.

On examination he was afebrile, there were no local signs of inflammation but there was marked tenderness over the distal end of both tibiae. There was full range movement of lower limb joints. He showed a normal gait, but walking or running caused discomfort. Blood tests revealed a raised erythrocyte sedimentation rate (ESR) (53 mm/h). Plain X-rays showed lytic lesions at the distal end of both tibiae (figures 1 and 2).

Under general anaesthetic, the lesions were explored under tourniquet control. Free fluid was evacuated from the lesions and sent for

Department of
Orthopaedics,
Central Middlesex
Hospital NHS Trust,
Acton Lane, London
NW10 7NS, UK
AFW Chamblor
PJ Chapman-Sheath
MF Pearse
J Hollingdale

Correspondence to:
Mr AFW Chamblor,
22 Queensmill Road,
London SW6 6JS, UK

Accepted 22 January 1997



Figure 1 X-ray of left ankle

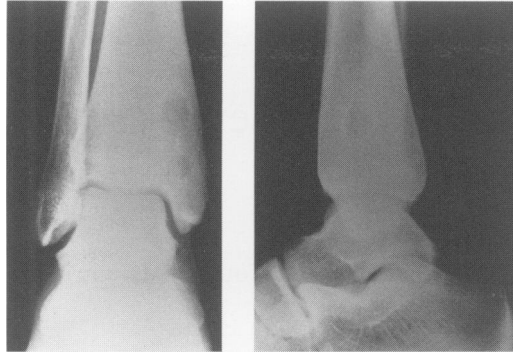


Figure 2 X-ray of right ankle

culture which grew *Staphylococcus aureus*. The cavities were curetted and lavaged. Post-operatively he was treated with antibiotics for six weeks. He was discharged from out-patients' clinic after an 18-month follow-up completely asymptomatic, pain-free while playing sporting activities and no tenderness on palpation. Blood results showed an ESR of 7 $\mu\text{m/h}$, C-reactive protein $<5\text{ mg/l}$, and white cell count of $6 \times 10^9/\text{l}$.

Discussion

Sir Benjamin Brodie first described a subacute bone abscess in 1832.¹ Brodie's abscess is defined as a centrally placed, sharply circumscribed, lytic lesion in the metaphysis adjacent to the growth plate. It is generally found in adolescents, with males most at risk.² Patients often present with mild pain with exacerbations, local warmth and occasional soft tissue swelling. Laboratory findings may show a raised ESR and leucocytosis. Frequently, however signs and symptoms may be absent, which presents a diagnostic dilemma. Differential diagnosis of uninfected causes include Ewing's sarcoma, giant cell tumour, aneurys-

mal bone cyst, fibrous dysplasia, eosinophilic granuloma, brown tumours of hyperparathyroidism and fibrocortical defects. Radiological investigation using MRI and/or radioisotope bone scan can recognise these malignancies, but ultimately, histological analysis may be required. Biochemical profiles would be useful to elicit the rare brown tumour.

Pathogenesis is due to an insidious bacteraemia with septic embolic producing subacute osteomyelitis. The patient may be septic or possess an infection remote from the symptomatic area. Consequently, 90% of Brodie's abscesses are located in the lower limb, at the distal ends of the tibia (70% of tibial lesions) or in the femur (60% of femoral lesions).³ *Staphylococcus* is the commonest causative organism, although 25% of subacute osteomyelitis cases have negative cultures.² Unusual organisms such as *Salmonella* and tuberculosis are found in patients with sickle cell disease or who are immunocompromised.

There are several radiographic findings associated with subacute osteomyelitis and authors have attempted to classify these appearances.^{2,4} However, in all reported studies, Brodie's abscess has been the most commonly encountered form.

It is rare for the abscess to present bilaterally (1 in 1000). Symmetrical Brodie's abscess is often confused with chronic recurrent multifocal osteomyelitis,⁵ as it has the same pathogenesis. In chronic recurrent multifocal osteomyelitis, patients have been systemically unwell with multiorgan dysfunction associated with a bacteraemia giving rise to the multifocal osteomyelitis. We conclude that a symmetrical Brodie's abscess presenting in an otherwise healthy patient is a separate clinical condition with a different management protocol. This report is the first recorded in the English literature.

1 Brodie BC. An account of some cases of chronic abscess of the tibia. *Medico-Chirug Trans* 1832; 175: 239–49.

2 Stephens MM, MacAulay P. Brodie's abscess. A long-term review. *Clin Orthop* 1988; 234: 211.

3 Brailsford JF. Brodie's abscess and its differential diagnosis. *BMJ* 1938; 2: 119–23.

4 Harris NH, Kirkaldy-Willis WH. Primary subacute pyogenic osteomyelitis. *J Bone Joint Surg* 1965; 47B: 526.

5 Day DL, Griffiths H. Bilateral Brodie's abscess. *J Orthop* 1989; 12: 885–8.