Subacute osteomyelitis (Brodie’s abscess) is essentially a problem of diagnosis, and there may be considerable difficulty in distinguishing it from other benign and malignant bone lesions. Though reported in the metaphyseal region of the femur, Brodie’s abscess is rarer in the femoral neck. The authors present a case of Brodie’s abscess in the femoral neck, which clinico-radiologically simulated an osteoid osteoma. Retrospectively, the presence of a cortical sinus tract should have aroused suspicion.

Keywords: Brodie’s abscess; subacute osteomyelitis; osteoid osteoma; hip infection.

INTRODUCTION

Known as the great masquerader in bone diseases, osteomyelitis mimics various benign and malignant conditions, resulting in delayed diagnosis and treatment (5,7-16). The diagnosis may be easier in the acute and chronic stage, when typical signs and symptoms are apparent. It is most often in the subacute state, which may include partially treated or previously undiagnosed disease, that the picture becomes more confusing. As much as 90% of the cases are initially misdiagnosed, with a mean delay of 3 months to correct diagnosis (12). Fifty percent of the cases are wrongly suspected to be of tumoral origin (12). The tibia is the most common site of a Brodie’s abscess (4,12,14). Although also reported in the metaphyseal region of the femur, Brodie’s abscess is rarer in the femoral neck (1, 11,14).

CASE REPORT

A 17-year-old boy was seen in the clinic because of increasing, dull, non-radiating pain in the left hip. The pain had begun insidiously three months earlier and was made worse by walking and by exercise. It did not affect his activities of daily living, but it prevented him from practising sports. The pain was relieved by lying down and awoke him occasionally from sleep. It was often partially relieved by non-steroidal anti-inflammatory drugs. The parents had noticed a slight limp. There was no fever, malaise, loss of appetite, weight loss, trauma, or any other musculoskeletal symptom. The personal and family histories were unremarkable.

At examination a healthy boy was seen in no acute distress and in a good physical condition. A slight limp was noted. There was diffuse tenderness in the left hip region with some fullness medially. There was some local warmth, but no erythema or significant inguinal lymphadenopathy. There was

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no discrete palpable mass. The active mobility was limited. The passive range of motion was terminally painful in all planes, especially in adduction and internal rotation. There was no limb length discrepancy and no distal neurovascular deficit. The rest of the orthopaedic examination was unremarkable. The biochemistry was non-contributory and included a haematocrit of 42% (range: 40 to 54), a white blood-cell count of 10,100/mm$^3$ (4300-10800), a neutrophil count of 67% (48-73), an ESR of 24 mm/hr (1-20), a CRP of 3.6 mg/L (0-60) and a negative HIV test. Plain radiographs (fig 1) and a CT scan (figs 2, 3) showed a geographic IA lesion in the medial cortex of the femoral neck, with a radiodense nidus-like center. The bone scan showed diffuse intense uptake in the left hip region (fig 4). Magnetic resonance images showed a hypointense nidus as well as surrounding bone oedema and joint effusion (fig 5).

After discussing the risks and benefits of the procedure, the lesion was exposed using a Ludloff approach. There was about 10 ml of clear serous fluid in the joint. After fluoroscopic localisation, curettage material was obtained and sent for frozen sections. These demonstrated necrotic bone with inflammatory cells, without evidence of osteoid osteoma. The lesion was curetted and the defect was grafted with autogenous bone. No organisms, including tubercular, fungal or anaerobes were grown from the joint fluid or from the tissue samples. The histopathological examination showed necrotic bone trabeculae, and granulation tissue with aggregates of acute and chronic inflammatory cells, most consistent with a subacute osteomyelitis. Parenteral cloxacillin and gentamicin were administrated for three weeks, followed by three weeks of oral cloxacillin with weekly monitoring. Limited weight bearing with crutches was advised for 3 months; high-impact activities were
forbidden for 6 months. At the last follow-up at 3 years, the patient was asymptomatic and pursued his previous life style, including sports.

**DISCUSSION**

Ever since its first description by Sir Benjamin Brodie in 1832 (4), Brodie’s abscess presents not only a difficult clinical picture, but also an interesting spectrum relative to its radiographic manifestations (1-16). Although the definition has been inconsistent, most agree that any infectious process in the bone for more than 2 weeks without any evidence of acute illness should be called subacute osteomyelitis (7). The cause for such a course is thought to be related to a combination of good host resistance and low virulence of the infective organism, often modified with the use of antibiotics (5, 7,12). It may be difficult to assess an origin of infection such as haematogenous seeding, local invasion from surrounding infected structures or direct inoculation of the bone from trauma or surgery (7). The diagnosis is made on the basis of imaging, biopsy and microbiology. Subacute osteomyelitis may mimic various benign and malignant conditions, which delays a correct diagnosis (1-16).

Although osteoid osteoma has been a differential diagnosis of subacute osteomyelitis in the metaphyseal region of the tibia or femur (4,5,9), it has mimicked Brodie’s abscess in the femoral neck only rarely (1,11). The clinico-radiological manifestations may be similar, but the incidence of Brodie’s abscess is lower than osteoid osteoma. Both may present with pain only, which may generate a delay in diagnosis. A few distinguishing radiographic features have been described, though not pathognomonic. A serpentine tract may be appreciated in Brodie’s abscess on CT and plain radiographs (3). In Miller’s series of 25 cases (11) lesions in the femoral neck were least defined with buttressing of the calcar in all cases. A sequestrum, typical for osteomyelitis, may look like the nidus of an osteoid osteoma, as evident in this case. According to Mahboubi (10), the inner aspect of the nidus is smooth in osteoid osteoma, and there may be calcification that is generally round, smooth, and centrally located in contrast to osteomyelitis where sequestration is seen off center in a lucency that has an irregular border. A soft tissue mass is often seen in osteomyelitis. The presence of a sinus or cloaca points towards an infection. Although magnetic resonance images can identify the nidus, they may be non specific and in fact misleading in osteoid osteoma because of bone marrow and soft tissue changes, which may be extensive (8). Grey et al (6) have described a ‘penumbra sign’ on T1-
weighted MRI scans. The penumbra is a discrete peripheral zone of marginally higher signal intensity than the abscess cavity and surrounding marrow oedema/ sclerosis, and of lower signal intensity than fatty bone marrow, and often enhancing with contrast. This has been postulated to be due to the presence of active, vascular, inflammatory granulation tissue around the abscess. Although this sign was also found in single isolated cases of eosinophilic granuloma and chondrosarcoma, the authors stated a sensitivity of 75% and a specificity of 99% with an accuracy of 99%, a positive predictive value of 92% and a negative predictive value of 99%. Scintigraphically, osteoid osteoma may show moderate tracer uptake with a central area of more focal uptake (double density sign), but this may not be easily interpreted. The use of 111-In labelled white cells has been shown to be more accurate than 99m-Tc labelled phosphonates or 67-Ga citrate scans in localisation of infection, but may still be non specific (6). In a recent report, Turkolmez et al (15) have shown that Tc-99m polyclonal human immunoglobulin scintigraphy may be further helpful in this differentiation as there is no increased uptake in osteoid osteoma.

Eosinophilic granuloma can be highly variable radiographically, thus providing another possibility in the differential diagnosis. Solitary lesions predominate over multiple lesions; approximately 10% of solitary lesions eventually develop into multiple lesions (2). They have well defined irregular margins, and are typically lytic. With time, they may show expansion of the cortex with periosteal new bone formation. Histologically, they may still be confused with osteomyelitis unless attention is given to the presence of Langerhans type histiocytes and absence of plasma cells.

Chondroblastoma has a predilection for epiphysis and apophysis, with a characteristic mineralisation pattern and osteoclastic giant cells. Periosteal chondroma usually sits on the surface of the bone, producing a superficial indentation that has been described as similar to an egg in a cup. Intracortical chondromas may still be difficult to distinguish radiologically. Cortical haemangiomas, although very rare, may present with a central homogenous nidus surrounded by sclerosis, but usually have fewer symptoms. In typical cases, neither chondroma nor haemangioma will produce reactive changes in surrounding structures. Malignant tumours like Ewing’s sarcoma/primitive neuroectodermal tumour and osteosarcoma would be expected to show a rapid clinical course with more pronounced perifocal oedema on MRI and characteristic histological features. Haematological tumours like leukaemia and lymphomas present as cohesive tumorous lesions containing mainly lymphocytes and few histiocytes. Radiologically there is a sharp transition between a ‘bone infarct’ and surrounding bone. The gross specimen appears as a chalky white area embedded within the normal cancellous bone. Histologically, the area shows empty lacunae in necrotic bone with calcified fibrosis. Other rarer possibilities like ganglions, fibrous defects, or bone cysts may also be indistinguishable radiologically. The possibility of a tubercular or fungal infection should also be considered. If the lesion is multifocal and recurrent, the possibility of chronic recurrent multifocal osteomyelitis should also be considered, although abscess formation, periosteal reaction and sequestrum are unusual in the latter and the patients are unwell for some time (2).

Based on anatomic location, morphology, and similarity to other bony lesions, Roberts et al (12) have expanded the Gledhill’s system (5) of radiographic classification of subacute osteomyelitis. Type Ia lesions present as a punched out radiolucency, often suggestive of eosinophilic granuloma. Type Ib is similar to Ia but has a sclerotic margin, the classical Brodie’s abscess. Type II lesions erode the metaphysis and resemble osteogenic sarcoma. These three lesions may extend to the epiphysis. Type III lesions have a localised diaphyseal cortical lesion with periosteal reaction, often mimicking osteoid osteoma. Type IV diaphyseal lesions often resemble round cell tumours like Ewing sarcoma with onion skin periosteal reaction. Type V lesions occur in the epiphysis and appear as a concentric radiolucency like in a chondroblastoma or chondromyxoid fibroma. Type VI lesions are vertebral based with an erosive or destructive process as in tuberculosis. In the classification of King et al (7) lesions in the calcaneum are considered separately.
This case reemphasises the old dictum “culture every tumour and biopsy every infection”. The importance of biopsy in addition to the clinical picture and radiographic studies to differentiate between subacute osteomyelitis and other pathologic entities including bone tumours cannot be overemphasised. Culturing the surgical specimen may or may not (as in this case) grow organisms: cultures of tissue samples are positive in no more than 50-60% of the cases\(^{(16)}\). Blood cultures are rarely positive. Although the most common infective organism is still \textit{Staphylococcus aureus}, others like \textit{Streptococcus}, \textit{Pseudomonas}, \textit{Haemophilus}, \textit{Kingella kingae}, and certain anaerobes may be seen\(^{(2,5,7,9,11-14,16)}\). \textit{Salmonella}, fungi and tuberculosis may be found in immunocompromised patients.

It is still debated whether medical treatment alone can cure this disease. Ross and Cole\(^{(13)}\) reported a cure rate of 87\% with a single course of medical treatment in children with non-aggressive lesions without pus formation. Others prefer additional surgery. Though medical treatment may be acceptable in children, literature favours surgical treatment in adults\(^{(2,7,11,12,14)}\). Failure of medical treatment or worsening symptoms warrant surgical exploration. Other indications for surgery are impending sinus formation or drainage into a pathological fracture and need for bone graft.

in the series of Stephens and MacAuley\(^{(14)}\) recurred after surgical excision, two of these requiring a re-operation and one of these recurring. They advocated that these lesions should be approached from the side where they are closest to the cortex. This will minimise bone resection, pathological fracture and need for bone graft.

\begin{thebibliography}{16}
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