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Case Report

Primary Subacute Osteomyelitis of the Greater Trochanter

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Primary subacute osteomyelitis is difficult to diagnose, because of its insidious onset, mild symptoms, lack of a systemic reaction, and similarity with tumoral conditions. This condition is seen mostly in the epiphysis or metaphysis of tibia. Although it has been reported at various bones, its occurrence in the greater trochanter is rare. We describe two patients, 4 and 14 years old, with mild pain of the hip joint and limping without history of acute bone infection. Radiography showed a lytic lesion in the greater trochanter resembling Brodie abscess. Both patients were treated completely with antibiotics.

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Introduction

Subacute osteomyelitis was first described by Sir Benjamine Brodie in 1836.[1] It results from an altered host-disease interaction, so that the bone though can control the extent of the infection, cannot eradicate it. In this condition, constitutional symptoms are absent, and the usual presentation is mild to moderate pain, swelling, localized tenderness, and limping for a long duration of weeks or months.[2, 3] Its incidence has been increased since the time when antibiotics have been used to treat osteomyelitis. The common age of affliction is 2 – 15 years. Males are affected slightly more than females. White blood cell (WBC) count and C-reactive protein (CRP) are usually normal or slightly elevated. Erythrocyte sedimentation rate (ESR) is often elevated.[1, 4]

Blood culture is usually negative. However, culture of the lesion is positive in 50% of the patients. Staphylococcus is the most commonly identified organism (30 – 60%).[1]

This condition may mimic all of the signs, symptoms, and radiographic findings of bone tumors — both benign and malignant — resulting in delayed diagnosis and treatment. Tuberculous abscess of the greater trochanter is another condition, which should be considered in the differential diagnosis of this disease.[5 – 7]

Bone scans are often positive but nonspecific. Computerized tomography (CT) is of value in detecting the lesions in the difficult anatomic locations, and can differentiate it from osteoid osteoma.[8] Nonetheless, magnetic resonance imaging (MRI) is the most sensitive diagnostic tool for this condition.[9]

Herein, we describe two patients with primary subacute osteomyelitis of the greater trochanter, which apparently has not been reported previously.

Case Reports

Case 1: A 14-year-old boy was referred in January 2005 to Imam Khomeini Hospital in Ahwaz, with mild pain of the right hip and limping for two months. The initial laboratory and radiographic work-ups were normal. After one month, he was admitted to the Orthopedic Ward because of more intense pain, limp, and mild fever. Examination showed tenderness over the right greater trochanter, WBC count of 10,500/mm^3, a negative CRP, and an ESR of 40 mm after one hour. Radiography revealed a lytic lesion with sclerotic border 7 – 10 mm in size in the greater trochanter of the right femur (Figure 1). Aspiration of the hip was normal. Gallium scan was positive

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Figure 1. Lytic lesion of the greater trochanter.

Figure 3. Lytic lesion before treatment.

(Figure 2). With the diagnosis of subacute osteomyelitis, treatment with intravenous antibiotics (cephalotin+gentamicin) for two weeks, and oral for four weeks was started. After five months of follow-up, the patient was symptom free, and the size of lytic lesion was decreased in radiography.

Case 2: A 4-year-old boy was admitted to the same hospital with pain of the left hip and limping for three weeks without a history of trauma or known infection. Range of motion and aspiration of the hip were normal. Tenderness over the left greater trochanter was noted.

Laboratory studies showed a WBC count of 7100/mm^3, a negative CRP, and an ESR of 50 mm after one hour. An ill-defined lytic lesion was seen in the radiography of the greater trochanter of the left femur (Figure 3). Bone scans were positive (Figure 4). As a case of subacute osteomyelitis, treatment with intravenous antibiotics for three weeks and oral for four weeks was started. After two months, the symptoms relieved and the lytic lesion became smaller with a more defined border in the radiography (Figure 5).

Discussion

The most widely used classification of primary subacute osteomyelitis in the literature is of Gledhill (1973), modified by Roberts in 1982, which consisted of six types, among which, type 1B, as the classic Brodie abscess, is the most frequent type.[10] Several recent reports advocate modifying this classification to epiphyseal, meta-

Figure 2. Bone scan shows increased uptake.

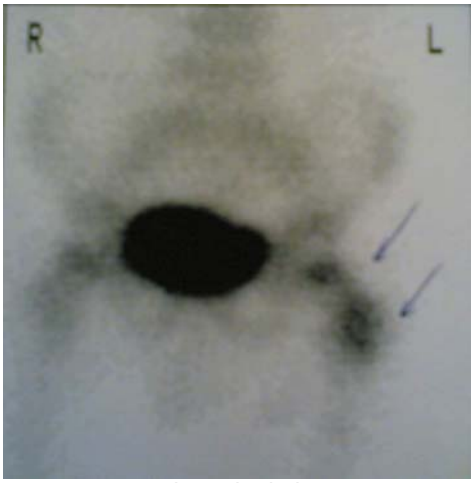

Figure 4. Increased uptake in bone scan.

Figure 5. The lesion after treatment.

physeal, diaphyseal, and metaphyseal-equivalent lesions. The latter is defined as the portion of a flat or irregular bone that borders cartilage or epiphysis, like the vertebrae, pelvis, clavicle, and small bones.[11, 12] The lesions in these two patients were similar to the Brodie abscess or metaphyseal-equivalent type lesion.

Ross and Cole divided the lesions either as aggressive or cavities. Aggressive lesions in the diaphysis or metaphysis with periosteal onion-skin bone formation do require biopsy for diagnosis, and to differentiate from bone tumors or tuberculous abscess, although some authors believe that we should perform biopsy as a routine investigation in all of the cavities or lytic lesions.[13]

Most cases of subacute osteomyelitis resolve with administration of antibiotics alone, at least in the pediatric age group. So, if the diagnosis can be made with assurance, no intervention other than aspiration is needed. Because of typical findings and good therapeutic response to antibiotics, no surgical intervention was performed for our two patients.

Failure of symptoms to resolve after six weeks of antibiotic therapy, aggressive lesions, sinus formation, drainage into a joint, or clinical signs of subperiosteal pus, are indications for surgery.

Although the symptoms of our patients were relieved by antibiotics, because the radiologic healing is much slower than clinical healing that usually occurs 12 months or longer, long-term follow-up is needed for reliable assessment of patients.

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