Foot Pain Arising From Subacute Osteomyelitis in a Child

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Osteomyelitis is known as the "great masquerader," and this report explores the differential diagnosis that attends cases of possible subacute osteomyelitis.

Case Presentation

An 11-year-old girl with a 2-year history of pain about the right foot was referred to our orthopedic service. She rated the severity of her pain at 3 on a 1-to-10 scale. The pain, well localized to the medial border of the right foot and not radiating proximally or distally, was not associated with night pain, weight loss, or night sweats. There had been no recent trauma and no overt local or systemic infection. Past history was remarkable for some nodular deformity about the distal phalanx joint of her left-hand fourth finger. Biopsy results for that lesion were positive for enchondroma.

On examination, the child was afebrile. There was some local tenderness to direct palpation over the base of the first metatarsal on the right foot. On initial examination, there was no obvious swelling, redness, or warmth over the area. The patient was able to walk tiptoe and on her heels without difficulty. There was no restriction in movement of forefoot, midfoot, subtalar, or ankle joints on the right side. Distal pulses, capillary refill, and sensations were intact. There was no associated lymphadenopathy. Limb lengths were equal. Gait pattern was normal. Systemic and neurovascular examination results were all normal.

On hematologic examination, leukocyte count was 5274, hemoglobin was 12.3 g/dL (normal, 11.5–15.5 g/dL), C-reactive protein was 1.01 mg/dL (normal, <1.0 mg/dL), and blood cultures were negative. Erythrocyte sedimentation rate was 16 mm/h (normal, 0–20 mm/h), and purified protein

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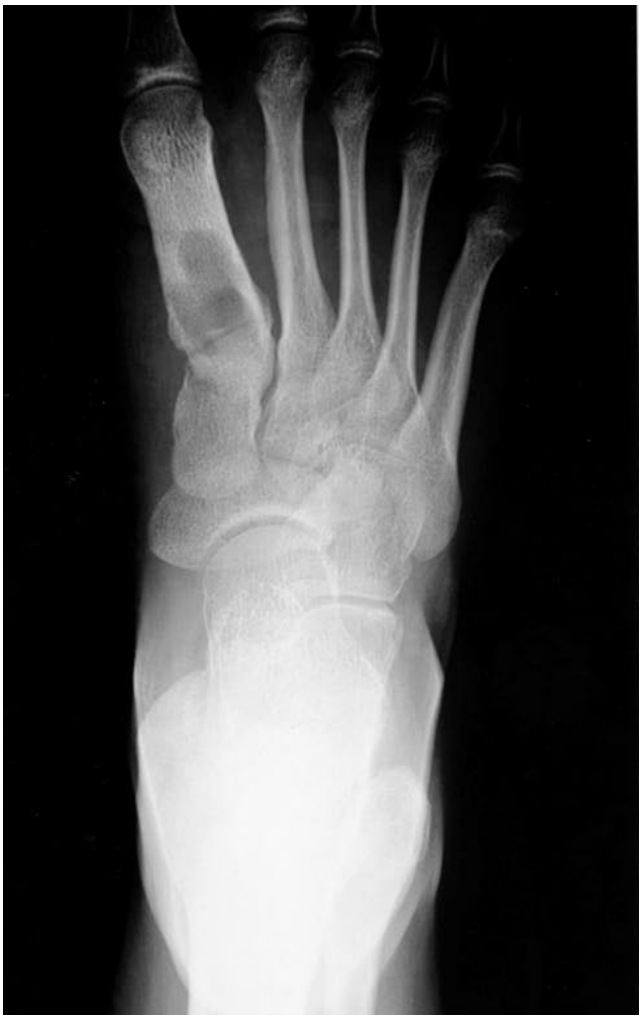

Figure 1. Plain radiograph of right foot.

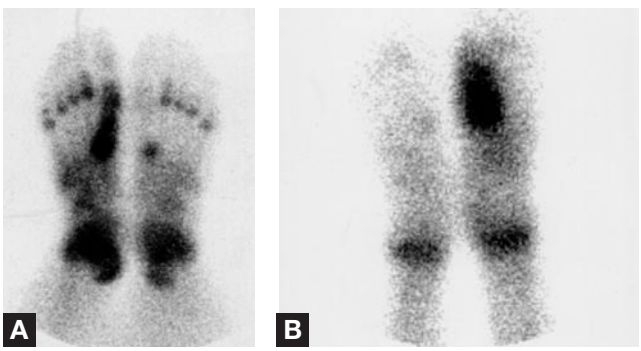

Figure 2. **(A)** Radionuclide angiogram image from Tc99m-methylene diphosphonate radioisotope bone scan. **(B)** Bone scan with image in static phase.

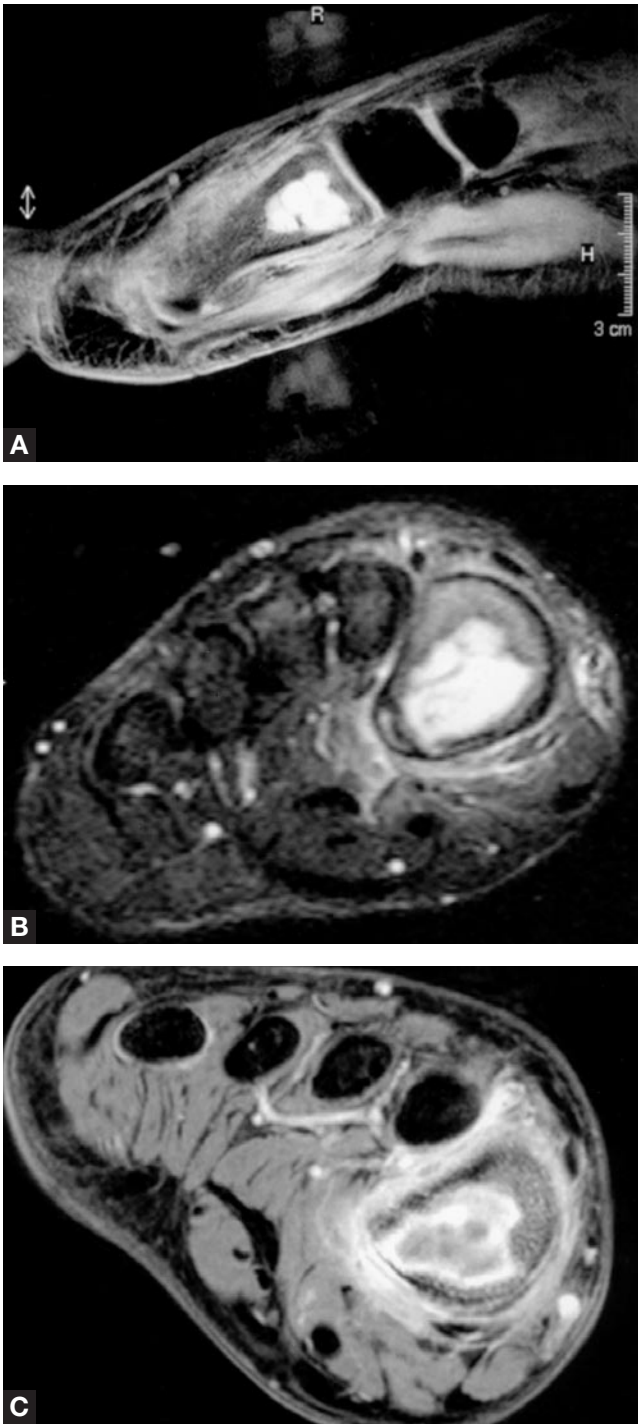

Figure 3. **(A)** Sagittal T_2-weighted magnetic resonance imaging (MRI) scan. **(B)** Axial T_1-weighted MRI scan. **(C)** Axial short τ inversion recovery image.

derivative skin test was negative. Radiographic investigations included plain radiographs, magnetic resonance imaging (MRI) scans, and bone scans (Figures 1–3).

Radiographic Interpretation

Plain radiograph (Figure 1) showed a cystic-appearing lucency in the proximal right first metatarsal with overlying soft-tissue shadow medially. The margins of the cystic lesion were well-defined and sharp. The adjacent bone was slightly sclerotic. There was no evidence of any periosteal reaction involving the base of the right first metatarsal. The rest of the foot appeared within normal limits. Follow-up plain radiograph of the foot showed an expanding lesion without diaphyseal or metaphyseal widening, evidence of loculation, and continued lack of periosteal reaction. The joint space was preserved.

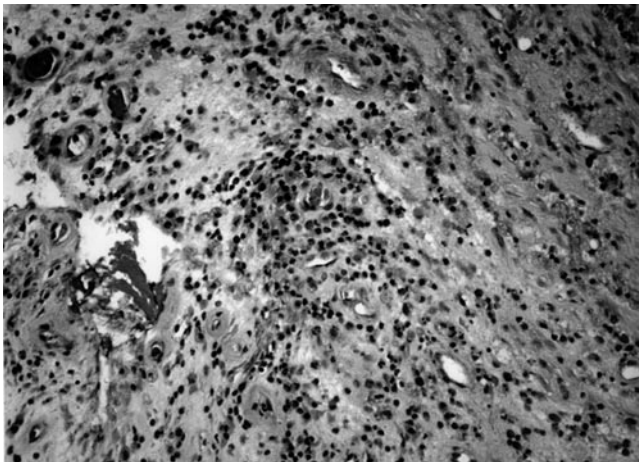

Figure 4. Photomicrograph (hematoxylin & eosin; magnification ×40).

A radionuclide angiogram image from a Tc99m-methylene diphosphonate radioisotope bone scan showed increased perfusion of the right first metatarsal (Figure 2A). The static image (Figure 2B) showed increased radiotracer uptake in the proximal right first metatarsal. Bone scan showed no other lesions.

A sagittal T_2-weighted MRI scan (Figure 3A) showed evidence of marrow edema, cortex thickening, periosteal reaction, and intramedullary focus of heterogenous signal. An axial T_1-weighted scan (Figure 3B) showed intermediate intramedullary signal intensity surrounded by cortical thickening and periosteal reaction. An axial short τ inversion recovery image (Figure 3C) on MRI showed an intramedullary focus of heterogenous but predominantly high-signal with thickened high-signal-intensity periosteal reaction.

The differential diagnosis included osteomyelitis, osteoid osteoma, enchondroma, aneurysmal bone cyst, unicameral bone cyst, Langerhans cell histiocytosis, and Ewing sarcoma.

An open biopsy was performed.

Histology

A histologic photomicrograph (Figure 4) showed multiple fragments of viable and focally nonviable bone with evidence of new bone formation. The marrow space contained aggregates of chronic inflammatory cells, including lymphocytes and plasma cells. There were some stromal changes, including early fibrosis. There was no evidence of any nidus, malignant cells, or aggregates of Langerhans histiocytosis.

Diagnosis

Subacute osteomyelitis of the base of the first metatarsal.

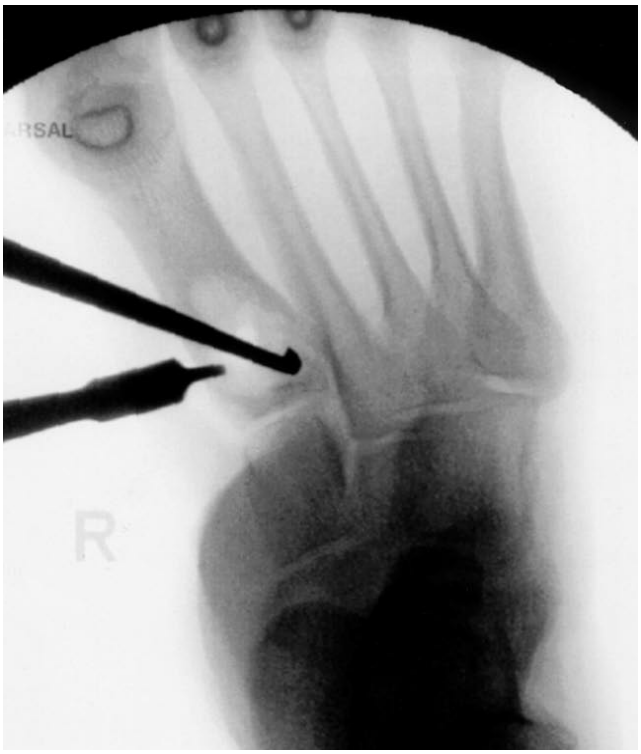

Figure 5. Intraoperative picture of lesion after curettage.

Treatment

The patient's lesion was treated surgically. Exploration of the first metatarsal base was done through a longitudinal skin incision. There was no evidence of periosteal elevation or inflammation and no subperiosteal collection. A cortical window was then carefully made, and the cystic lesion was curetted (Figure 5). The lesion was then thoroughly irrigated with saline, and the wound was closed in layers. The patient was given postoperative antibiotics and her postoperative course was uneventful. Monitoring was done with weekly office visits. The patient's wound healed well, and the scar was supple.

Discussion: The Diagnostic Process

Osteomyelitis has been long known as the "great masquerader" owing to its varied presentations.

Acute Versus Subacute Osteomyelitis

Acute hematogenous osteomyelitis (AHO) usually presents with a relatively short history, elevated temperatures, and a fulminant course. Subacute osteomyelitis may include partially treated or previously undiagnosed disease and has a less clear presentation. Comparative features of acute and subacute osteomyelitis are listed in the Table.

Up to 50% of patients with subacute osteomyelitis are initially suspected to have tumors, given the radiographic and clinical presentation.[1] Subacute osteomyelitis most likely occurs because of an altered host–pathogen relationship,[2] and possible origins of infection include hematogenous seeding, local invasion from surrounding infected structures, and direct inoculation of the bone from trauma or surgery.[3]

At presentation in subacute osteomyelitis, plain radiographs may show bone lesions. Unlike acute hematogenous osteomyelitis, subacute osteomyelitis often crosses the physis, though growth plates are rarely permanently damaged. *Staphylococcus* is the common pathogen in most cases of subacute osteomyelitis; in some cases, *Streptococcus* is found. Often, as in our case, organisms are not isolated.

Radiographic classification of subacute osteomyelitis was first described by Gledhill and McIntyre[4] in 1973. The Brodie abscess may be considered a form of subacute pyogenic osteomyelitis.[5] Most Brodie abscesses occur in the metaphyseal region (Gledhill type I lesions). When the cortex is destroyed (Gledhill type II) or when there is extensive cortical reaction (Gledhill types III and IV), tumors such as eosinophilic granuloma, Ewing sarcoma, and osteogenic sarcoma should be considered in the differential diagnosis.

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Clinical correlation is the most important factor in diagnosing osteomyelitis. Fever with unexplained bone pain should be considered osteomyelitis unless proved otherwise. Subacute osteomyelitis may be difficult to interpret clinically. Moreover, laboratory data may be well within

Table. Comparison of Acute and Subacute Osteomyelitis

Feature	Acute Osteomyelitis	Subacute Osteomyelitis
Pain	Severe	Mild
Fever	Majority of cases	Few cases
Loss of function	Marked	Minimal
Prior antibiotic therapy	Occasional	Often (30%–40% of cases)
Elevated white blood cell count	Majority of cases	Few
Elevated erythrocyte sedimentation rate	Majority of cases	Majority of patients
Blood cultures	50% positive	Few positive
Bone cultures	85% positive	60% positive
Initial radiographs	Often normal	Frequently abnormal
Site	Usually metaphysis	Any location (may cross physis)

normal limits. Also, blood cultures are positive in fewer than 50% of subacute osteomyelitis cases.[6]

Subacute osteomyelitis simulates a wide range of differential diagnoses, including Ewing sarcoma, chondroblastoma, osteoid osteoma, chondromyxoid fibroma, osteogenic sarcoma, and eosinophilic granuloma.[7] Primary subacute osteomyelitis, described by King and Mayo[3] in 1969, can be thought of as a Brodie abscess of the epiphysis. Most cases of epiphyseal osteomyelitis are subacute. The Brodie abscess was first described as a localized tibial metaphyseal abscess without associated systemic illness.[5] The tibia is a common site for this lesion; other sites are the metaphysis and the epiphysis of other long tubular bones.[8] On MRI, Brodie abscesses have low signal intensity on T_1-weighted scans and high signal intensity on T_2-weighted scans.[8] These lesions usually respond well to surgical débridement and postoperative antibiotics.[2–4,9] Sclerosing osteomyelitis of Garré, which differs from the Brodie abscess, is characterized by thickening and expansion of bone without sequestration or suppuration.[10]

Chronic Osteomyelitis Versus Ostoid Osteoma

Chronic osteomyelitis is defined by most authors as osteomyelitis with symptoms that have persisted for more than 1 to 3 months. Interestingly, chronic osteomyelitis with a localized sequestrum may mimic an osteoid osteoma clinically and radiologically. Both osteoid osteoma and subacute osteomyelitis can present with pain only, and thus a clinically overlapping presentation can lead to delay in diagnosis.[3] Computed tomography (CT) features that are characteristic of both conditions were outlined by Mahboubi.[11] In osteoid osteoma, the inner aspect of the nidus is smooth and centrally located. In osteomyelitis, the sequestration is typically off-center in a lucency having an irregular inner border; an adjacent soft-tissue mass may also be found.[11]

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Chronic Recurrent Multifocal Osteomyelitis

Chronic recurrent multifocal osteomyelitis (CRMO) may initially present as pain, swelling, and tenderness at a single locus. Psoriasis, palmoplantar pustulosis, and arthritis are linked to chronic multifocal osteomyelitis and so should raise suspicion of CRMO even in the presence of a single bone lesion.[6] Bone lesions tend to develop sequentially and are most often found in the long-bone metaphyses. Radiographic features include lucency with or without sclerosis.[6,12] Our patient had local pain and a metaphyseal lesion consistent with osteomyelitis. There were no additional symptomatic lesions and no other foci evident on bone scan. By 12 months of postoperative follow-up, no other foci had become involved.

Sclerosing Osteomyelitis Versus Ewing Sarcoma and Osteosarcoma

The sclerosing variant of osteomyelitis (Garré osteomyelitis) can be differentiated from Ewing sarcoma and osteosarcoma on the basis of the number of subperiosteal bone layers visible on radiographs.[10,13] Whereas systemic signs are often lacking with sclerosing osteomyelitis, fever and elevated erythrocyte sedimentation rate can be present with Ewing sarcoma and osteomyelitis. Ewing sarcoma, the second most common malignant tumor of childhood, has its peak incidence in the early second decade of life. Essentially all bones are at risk, though the most common site is the medullary portion of the metadiaphyseal region of long bones. Interestingly, when Ewing sarcoma occurs in the bones of the hands and feet, active periosteal reactions are infrequent. Biopsy is usually needed for definitive diagnosis.

Osteomyelitis Versus Enchondroma

Enchondromas are benign tumors of mature hyaline cartilage. The short, tubular bones of the hands and feet are common sites, followed in frequency by the femora, humeri, tibiae, and ribs.[14,15] Lesions are often asymptomatic, unless fracture occurs. Radiographs show a circumscribed lytic defect, with a central region of rarefaction, within the metaphysis or diaphysis of the involved bone. Calcification, if present, is usually stippled. Mature enchondromas may have peripheral enchondral ossification. Cortical expansion is common. Histology is diagnostic and includes lobules of hyaline cartilage with varying cellularity and blue matrix.[14]

Osteomyelitis Versus Aneurysmal Bone Cysts

Aneurysmal bone cysts arise either centrally or eccentrically within the medullary cavity, though intracortical and subperiosteal lesions are also found. Early lesions are often small and radiolucent with relatively permeative borders and they gradually expand the contour of the bone as they grow. Classical evolved lesions have a marked distortion of the bony contour, internal trabeculations, and peripheral ossification. Isotope scans typically show intense uptake peripherally and relatively less uptake centrally. On MRI and CT scans, fluid-filled levels are evident in approximately 80% to 90% of aneurysmal bone cysts, a situation attributed to the layering of fluids and cellular elements of different specific gravities. Aneurysmal bone cysts have characteristic gross and microscopic features. Morphologically, they look like a blood-filled sponge; histologically, they are a combination of blood and collapsed septa consisting of fibrous tissue with numerous capillaries, multinucleated giant cells, and inflammatory cells.[14,15]

Osteomyelitis Versus Langerhans Cell Histiocytosis

Langerhans cell histiocytosis can be highly variable in radiographic appearance and can involve single or multiple

lesions. These well-defined, lytic-appearing lesions may cause cortex expansion and periosteal new bone formation over time. Presence of Langerhans cell histiocytes and absence of plasma cells are characteristic features on histology. Biopsy is confirmatory for diagnosis.[14,15]

Conclusions

In this report, we highlight the importance of conducting a thorough clinical evaluation and assessing radiographic features and biopsy results to differentiate infections like subacute osteomyelitis from other conditions, including tumors of the bone.

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