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Canadian Association of Radiologists Journal 60 (2009) 99–102

CANADIAN ASSOCIATION OF RADIOLOGISTS JOURNAL

www.carjonline.org

Canadian Residents' Corner / Coin canadien des résidents en radiology

Answer to Case of the Month #146
Osteoid Osteoma

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Clinical Presentation

A 17-year-old boy presented with a 6-month history of unrelenting right groin pain causing night awakenings. There was mild response of pain to naproxen although other medications were ineffective. The patient had no fever. There was local discomfort on palpation and pain on internal rotation (Figures 1–4)

Diagnosis

Osteoid osteoma—intra-articular.

Radiologic Findings

Anteroposterior radiograph of the right hip shows a 6-mm, sclerotic focus surrounded by a thin, uniform, lucent rim projecting over the femoral neck. No gross peripheral sclerosis or periosteal reaction is shown.

Technetium-99m–labeled methylene diphosphonate triple-phase bone scan (flow, 1 min; blood pool, 5 min and delayed 3 h) shows no abnormality in the flow phase, increased delivery of radiotracer to the right hip region in the blood pool phase, and more focal increased uptake at the right femoral neck on delayed phase.

Thin-slice, low-dose computed tomography (CT) localizes the lesion to the anterior cortex of the femoral neck. The lesion has a densely mineralized core with a surrounding thin, uniform, lucent rim and a lack of a periosteal reaction. There is a very thin margin of sclerosis in the adjacent cancellous bone.

A magnetic resonance image (MRI) shows the center of the lesion to be of low signal on both T1W and T2W images in keeping with mineralization. This is surrounded by intermediate-signal rim on fat suppressed proton density weighted images, which represents the noncalcified rim of the nidus. Intermediate to high signal seen on the fat-suppressed T2W image surrounding the nidus is consistent with marrow oedema. A small hip joint effusion also is present.

Discussion

Osteoid osteoma (OO) is a benign skeletal neoplasm characterized by a lucent nidus surrounded by a variable amount of sclerosis and new bone formation. It accounts for approximately 12% of all benign skeletal neoplasms. It most commonly occurs in patients between the ages of 10 to 25 years with a male to female ratio of 3:1 [1]. There are no reports of malignant transformation or metastatic spread.

There is significant predilection of OOs for the lower extremity. About 70% of them occur in the femur and tibia, typically involving the diaphyseal or metadiaphyseal regions. The remaining 30% are distributed in the spine, hands, and feet. In the spine the neural arches of the lumbar spine commonly are involved, and in the hands and feet the distal phalanges are slightly more commonly involved than the proximal phalanges [2].

Macroscopically, OO appears as a nidus surrounded by abundant reactive sclerotic bone. The tumour has limited

Key Words: Bone neoplasms/radiography; Diagnosis, differential; Osteoma, osteoid/radiography; Osteoma, osteoid/radionuclide imaging; Magnetic resonance imaging; Osteoma, osteoid/diagnosis; Tomography, x-ray computed.

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0846-5371/$ - see front matter
doi:10.1016/j.carj.2009.02.016

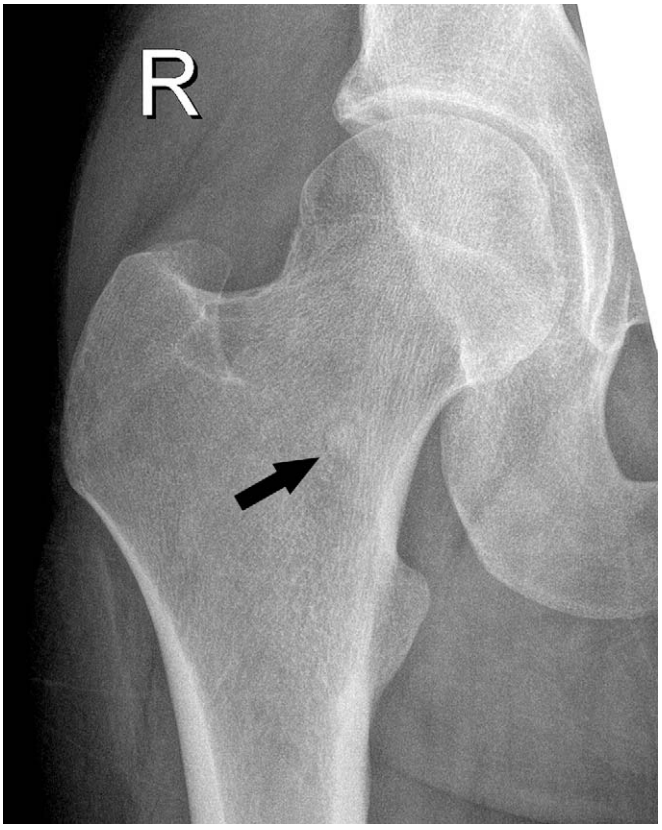

Figure 1. Anteroposterior radiograph of the right hip shows a 6-mm, sclerotic focus surrounded by a thin, uniform, lucent rim projecting over the femoral neck. No gross peripheral sclerosis or periosteal reaction is shown.

growth potential and it rarely exceeds a diameter of 1.5 cm. Microscopically, the nidus is composed mainly of highly vascular stroma interspersed with various degrees of osteoid and woven bone. The surrounding reactive bone mainly contains varying mixtures of woven and lamellar bone.

The most common presenting symptom is pain, which progressively worsens over time, becoming severe in many cases. It often is worse at night, and typically is relieved by acetylsalicylic acid. However, if the lesion is intra-articular, night pain is atypical and it tends to be less responsive to anti-inflammatory medications [3]. Intra-articular lesions may present with joint pain and swelling [4]. Referred pain may make it difficult to localize the lesion to the joint [5]. Rarely OO may be painless, especially if it is located in the phalanges, for which patients may present with a palpable soft-tissue mass. Patients with lesions in the spine typically present with painful scoliosis. Prolonged hyperemia (if the symptoms are present for at least 3 months) can cause premature fusion of the growth plate, which can lead to structural alterations. Patients may present with a deformity, muscle atrophy, or a painless limp caused by growth disturbances [2].

OO are classified into 3 subgroups based on the location of the nidus within the bone. The groups are as follows. Group 1: cortical (most common)—the epicenter of the nidus is within the cortex, with surrounding fusiform sclerosis.

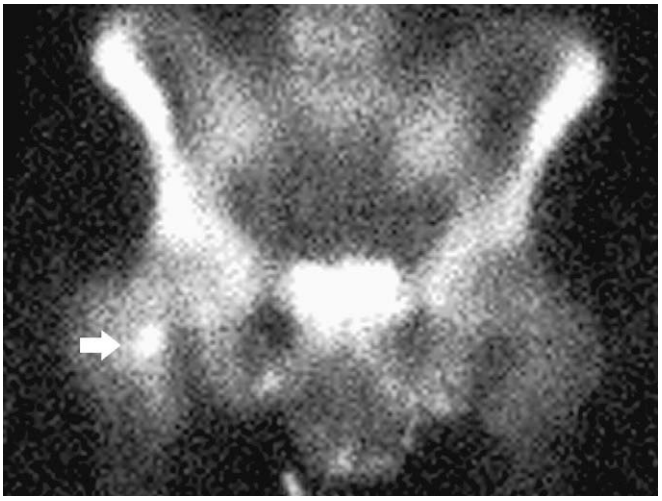

Figure 2. Technetium-99m–labeled methylene diphosphonate triple-phase bone scan (delayed phase scan—3 hours) shows focal increased uptake at the right femoral neck.

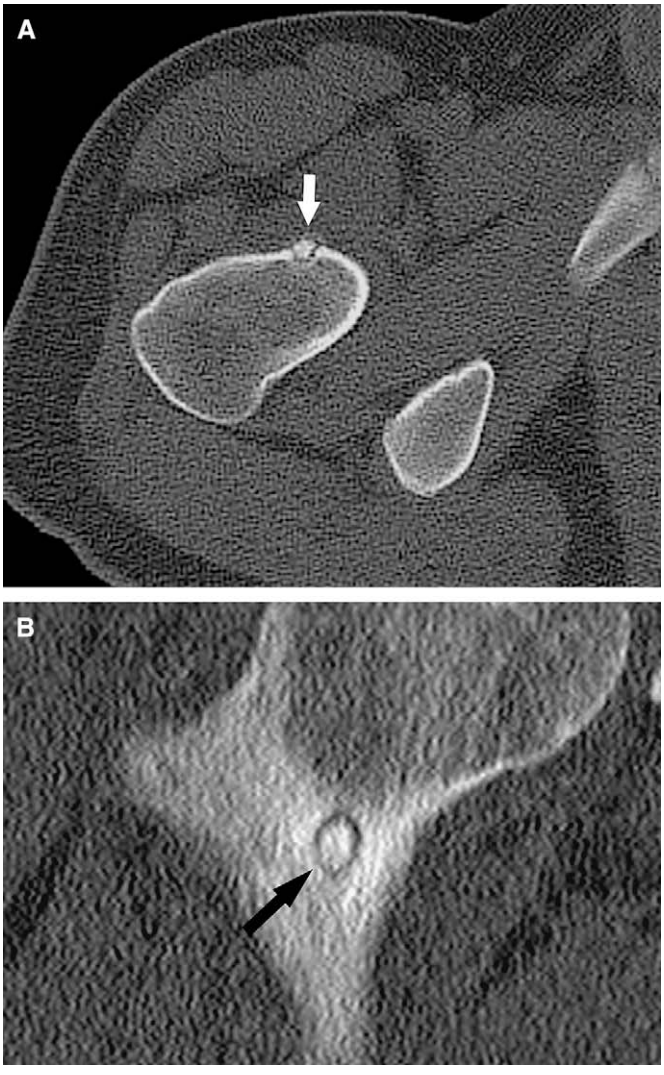

Figure 3. (A) Thin-slice, low-dose computed tomography axial image and (B) coronal reformat localize the lesion to the anterior cortex of the femoral neck. The lesion has a densely mineralized core with a surrounding thin, uniform, lucent rim and lack of periosteal reaction. There is a very thin margin of sclerosis seen in the adjacent cancellous bone.

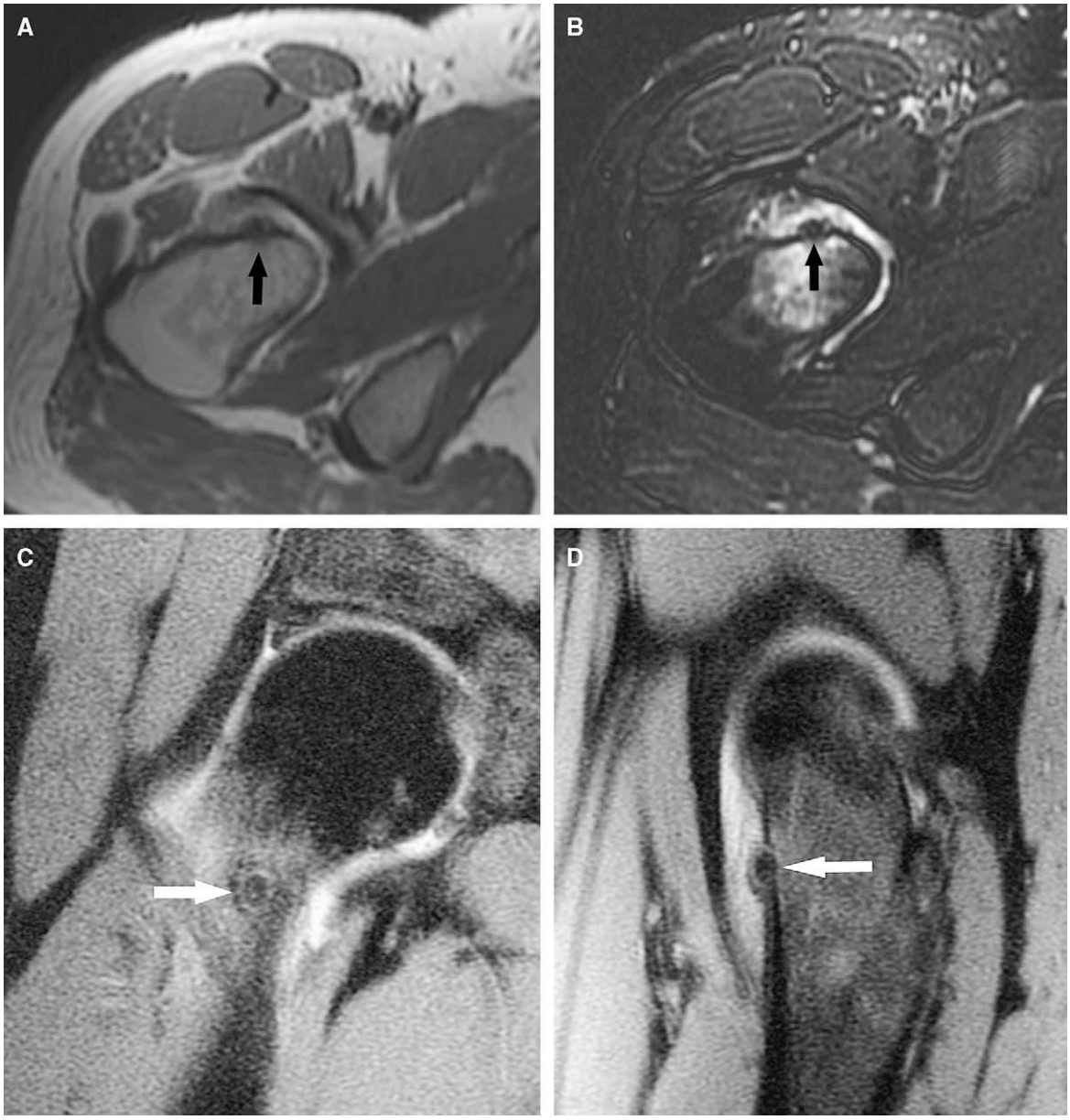

Figure 4. Magnetic resonance imaging shows the center of the lesion to be of low signal on both (A) T1W and (B) T2W images in keeping with mineralization. This is surrounded by intermediate-signal rim on (C and D) fat suppressed proton density weighted images, which represents the noncalcified rim of the nidus. High signal seen on the (B) fat-suppressed T2W image surrounding the nidus is consistent with marrow oedema. A small hip joint effusion also is present.

OOs of the long bones, such as the femur and tibia, typically are cortical in location. Group 2: medullary—the epicenter of the nidus is within cancellous bone, typically juxta-articular or intra-articular. Medullary OOs occur in the femoral neck, in the small bones of the hands and feet, and the posterior elements of the vertebrae. These lesions may generate mild to moderate surrounding sclerotic reaction, which can be distant from the lesion. The nidus may be eccentrically situated relative to the zone of sclerosis. Group 3: subperiosteal (rarest)—the epicenter of the nidus appears to be extra-osseous, causing pressure erosion of the adjacent bone. It can occur in the small bones of the hands, feet (commonly in the talar neck), and the femoral neck. The subperiosteal variant results in minimal reactive sclerosis.

The radiographic appearance of the classic cortical OO is of a small radiolucent nidus surrounded by an area of reactive sclerosis and new bone formation. The nidus can have variable mineralization, which can progress over time. The periosteal reaction usually is dense and solid, but may be lamellated.

Intra-articular lesions (typically cancellous or subperiosteal) can be difficult to detect on radiographs unless there is high clinical suspicion. They induce a weaker periosteal reaction than the more common cortical variant. This is caused by functional differences between the intracapsular and extracapsular periosteum, the former being incapable of a proliferative response [6]. However they may show greater reactive sclerotic changes in the adjacent cancellous bone. The degree of sclerosis varies and tends to be mild in hip joints, but

moderate to severe in the elbow [2]. Periosteal reaction may occur beyond the confines of the joint capsule and on both sides of the articulation [2]. The joint space may be widened secondary to effusion and/or synovitis. Histologically, the synovitis may be lymphofollicular, similar to that seen in inflammatory arthritis [5]. The regional hyperemia induced by OOs, along with disuse, can lead to radiographic osteopenia. On triple-phase nuclear scintigraphy, extra-articular OOs may show localized tracer activity in the flow phase. Activity seen within the nidus in the blood pool phase is caused by the presence of a vascular stroma. Delayed phase shows intense uptake centrally, corresponding to the nidus and a less intense yet still increased uptake surrounding the nidus related to the reactive bone reaction. This is referred to as a *double-density sign*. However, this sign is not specific [7]. Intra-articular lesions also show increased local radiotracer uptake, albeit less intense than the extra-articular form. In addition, the other patterns of uptake described for intra-articular lesions are diffuse uptake at the epiphysis and increased uptake in the adjacent growth plate [5]. In general, the degree of uptake in OOs varies with the degree of lesion vascularity [3].

On CT, OOs appear as a low-attenuation nidus with variable degrees of internal mineralization and perinidal sclerosis. When compared with other diagnostic modalities, CT is very sensitive in detecting and localizing OOs owing to its high spatial resolution and contrast between bone and soft tissues. Location, extent of surrounding bone reaction, and cortical thickening are best determined by CT. It also may show gross regional soft-tissue inflammatory changes such as oedema and, in the setting of intra-articular OOs, joint effusion. CT is the investigation of choice for characterizing intra-articular lesions because the radiographic findings may be quite subtle or nonspecific. CT thus also can aid in the planning of interventional treatment of the lesion. It is the modality of choice to differentiate OOs from stress fractures, which can have overlapping features on radiographs.

The MRI signal of the nidus depends on the size, vascularity, and amount of calcification present within the nidus [3]. The nidus typically show low to intermediate signal on T1−WI sequence and low or high signal on fluid- sensitive sequences such as fat-suppressed T2W or short tau inversion recovery (STIR). The nidal signal intensity on the fat-suppressed, fluid-sensitive sequences is dependent on the mineral content—mineralized nidi are of lower signal and nonmineralized nidi show higher signal. The nidus usually is surrounded by a variable amount of bone marrow and soft-tissue oedema. The degree of bone marrow oedema is greater in early, active lesions than in relatively older, quiescent lesions. The use of anti-inflammatory medications can diminish the surrounding marrow oedema. The extent of bone reaction and surrounding marrow oedema is greatest in younger patients [3]. Enhancement of the nidus is typical after intravenous gadolinium administration, although the degree depends on the vascularity of the lesion. MRI excels at detecting associated soft-tissue and bone marrow oedema. However, soft-tissue findings on MRI are not always specific and the extensive inflammatory change may be misinterpreted as a soft-tissue mass. MRI falls behind CT in identifying the central nidus. Davies et al [8] found that in 50% of cases, the nidus is either not identified or is poorly defined by MRI. For these reasons, it is helpful to correlate the findings of MRI with those of CT.

Extra-articular lesions often are suspected on radiographs, with confirmation and further characterization achieved with CT. Intra-articular lesions are often difficult to identify on radiographs, which may lead to a delay in the diagnosis. If there is a high index of clinical suspicion, bone scan is suggested to identify the abnormal area for subsequent imaging with CT to identify the nidus. The hip is the most common site for intra-articular OOs. Thickening of the medial calcar (the medial cortex, just proximal to the lesser trochanter) on radiographs is an additional finding that is highly suggestive of intracapsular OO of the hip if there is supporting clinical history [5].

Differential diagnosis for a cortical osteoid osteoma includes Brodie's abscess, stress fracture, osteoblastoma, and early stages of osteosarcoma and Ewing's sarcoma. Inflammatory and septic arthritis are the main differential considerations for intra-articular OOs.

Relief of pain symptoms is the goal of therapy. If initial conservative treatment with anti-inflammatory medications fails, then image-guided intervention should be considered. The most commonly used treatment is CT-guided percutaneous radiofrequency ablation of the lesion, provided there is a typical clinical, scintigraphic, and CT presentation. It is a highly effective, efficient, minimally invasive, and safe method of treating OO. Clinical success ranges from 76% to 100%. Laser photocoagulation is an alternative ablation technique in some centers. Surgery remains the standard treatment in cases in which histology of the lesion is in doubt, if neurovascular structures are in close proximity to the lesion such that heat from an ablative process might damage them, in repeated failure of minimally invasive ablative techniques [9], or if there is a contraindication to an image-guided procedure. Incomplete resection can result in recurrence of symptoms.

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