Osteonecrosis: a pictorial review

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Learning objectives

To review the principal causes and pathophysiology of osteonecrosis (ON), the common sites affected, and illustrate the spectrum of radiologic findings on different imaging techniques.
Background

Osteonecrosis (ON) results from a reduction or total loss of blood supply to a region of bone, followed by sclerosis, collapse and secondary osteoarthritis. The terms *avascular* or *aseptic necrosis* have been applied to subarticular or epiphyseal bone commitment.

Usually, ON is a non-symptomatic condition and therefore its true prevalence is underestimated. The clinical significance depends on the grade of articular collapse. The core causes for bone ischemia are trauma, corticosteroids and alcoholism, although many have no evident explanation [1,2].

Osteonecrosis is a common situation among world community and the most typical affected sites are femoral head, humeral head and scaphoid bone [3].

Legg-Calvé-Perthes (LCP) disease is an idiopathic avascular necrosis of the pediatric hip, with a male predominance and seen most frequently between the age of 4 and 8 years, when the femoral head is most at the vascular supply risk [4]. Children, when symptomatic, usually have limping gait or just refer knee pain on the affected side.

Older age at the time of diagnosis and an involvement of more than 50% of the femoral head are two indicators of a bad prognosis [4].

After the hip, the humeral head is the second most common affected site by osteonecrosis [5,6]. It is one of the most important causes for shoulder joint pain, but is an uncommon condition.

Although trauma is a common cause, atraumatic necrosis can develop in patients with some risk factors as corticosteroid administration or heavy alcohol intake, and some systemic disease as sickle cell disease, rheumatoid arthritis and systemic lupus erythematos [7].

The diagnosis is made based in clinical and radiographic findings. The treatment depends on the severity and chronicity of symptoms as the degree of radiographic progression.

The exact etiology of ON involving the lunate bone (Kienbock disease) is unknow but, in some cases, has been associated with history of trauma months before presentation[8]. A single blood supply on palmar or dorsal surface or a dual blood supply with no intraosseous anastomoses may explain some of the vulnerability of the lunate to avascular necrosis [9]. There is also a significant association between ulnar minus
variance and Kienbock disease, as a distal ulna is few milimeters shorter than the distal radius transmitting unequal forces to the lunate bone [3,4].

Conservative management with immobilisation and non-steroidal anti-inflammatory drugs is typically the initial management [9,10]. A radial shortening procedure to correct negative ulnar variance is the most typical surgical therapy as it reduce the pressure transmitted to the lunate at the wrist [9].

The Freiberg’s disease is another cause of avascular necrosis affecting the metatarsal head with consequent subchondral collapse. It occurs commonly on the second and third metatarsal heads [4,11] and is more frequently seen in younger females, related to the trauma of bearing weight in high-heeled shoes [4,12]. It can be bilateral in up to 10% of the cases [13].

Despite the fact that the first ON radiographic sign takes several weeks to occur, the characteristic imaging features can avoid additional radiologic evaluation. The initial evaluation starts with a fast and easy method of imaging as radiography, and the typical radiographic appearance of a compromised bone is patchy areas of lucency and sclerosis. Occasionally, radiography may also show early areas of articular collapse [3].

Magnetic Resonance (MR) imaging is generally referred as the most sensitive and specific image modality for diagnosis of ON [14-16]. Computerized Tomography (CT) scanning is not commonly used for the assessment of this condition, but can be useful for describing the subchondral fractures [17].
Findings and procedure details

We will review the typical radiologic findings of some sites that can be affected by avascular necrosis presenting selected cases from our Imaging Department: femoral head in adults and children, humeral head, scaphoid and metatarsal bone.

All the cases were first studied by radiography and some followed by MR and CT. The clinical history and possible associated cause were correlated with the imaging findings. The pathology progression was documented by radiography imaging.

The first radiographic sign in avascular necrosis of the hip occurs weeks or months after the bone infarct. Sclerosis is the initial radiologic finding, first because of the hipervascularized bone surrounding the necrotic bone and later due to the new bone formation. The classic radiographic appearance is patchy areas of lucency and sclerosis, with the sclerotic bone showing a undulating morphology over the lesion rim (Fig. 1, arrow). A linear subchondral fracture termed "crescent sign" (Fig.2, arrow) is the typical finding developed during bone healing that often progresses to subchondral fragmentation, flattening and deformity. These complications can evolve to secondary osteoarthritis [3,4].

CT scan can demonstrate some later changes of a ischemic bone as serpentine and undulating sclerotic margin (Fig.3, arrow), although CT is not the best choice for evaluation and detecting early stages of ON [18].

MR is the most sensitive image modality for diagnosis avascular necrosis. The initial findings can be nonspecific as diffuse marrow edema. As the condition progresses, the caracteristic imaging of a serpiginous low-signal-intensity rim with all pulse sequences appears corresponding to a line of sclerosis (Fig. 4, arrow). The surrounding yellow marrow maintains the signal intensity as viable and devitalized adipose tissue have an indentical intrinsic MR imaging appearance.

On T2 sequences, a "double-line" sign can be seen with the outer low-signal-intensity rim representig the sclerosis and the inner rim of high signal intensity representing the reparative granulation tissue of the reactive interface (Fig.5, arrow) [19,20].

The chosen initial exam for suspected LCP is radiography with frontal and frog-leg lateral projections [21]. The first radiographic sign may be the presence of effusion, which can mimic infection. Later on, due to the loss of blood flow, the ossific nucleus of the affected femoral head fails to grow resulting in smaller appearance than the contralateral normal side. The combination of mechanical forces and weakness of the femoral epiphyses by the necrosis and healing process account for fragmentation and flattening of the femoral head (Fig.6). Growth abnormality results in metaphyseal irregularity with consequent
shortness and a wide femoral neck (coxa magna) and a superiorly located ("high-standing") greater trochanter [3,4] (Fig.6 (A), arrow).

The main imaging modalities used in the diagnosis of humeral head osteonecrosis are radiography and MR imaging. The standard radiography is commonly used to stage disease progression, although, MR imaging is more sensitive and useful in the diagnosis of the humeral head osteonecrosis. In earlier stages, the sclerosis which may be focal or diffuse, results from subchondral microfracture without articular collapse. It typically appears in the superior portion of the humeral head and as an hyposignal on T1-weighted images (Fig. 7). A double-line sign representing lines of alternating high and low signal intensity on T2-weighted images, as described before in the femoral head, is observed in over 50% of patients with osteonecrosis of humeral head [22]. The inner hypersignal line corresponds to hyperemic granulation tissue and the outer dark band corresponds to fibrosis or sclerosis (Fig. 8). In later stages, fragmentation and articular collapse can occur and be accompanied by joint effusion [23].

To diagnose Kienbock disease, as expected, radiography imaging is usually the initial imaging. Sclerosis, fracture, flattening and loss of volume of the lunate bone is possible to see on a radiographic exam (Fig. 9).

MR is the most sensitive and specific imaging modality to detect early disease and to diagnose the cause of wrist pain in patients with persistent clinic suggesting Kienbock disease but with normal radiographs [24]. The diagnosis is made when diffusely decreased T1-weighted signal intensity involves the entire lunate (Fig.10, arrow) or suggested when hypointense is seen centrally and within the radial aspect of the lunate. T2-weighted or STIR signal intensity is variable, and in the acute phase high T2 and intermediate T1 signal can be associated with bone oedema (Fig.11, arrow).

As the other suspected cases of osteonecrosis, radiography is often the initial imaging modality to diagnosis Feiberg’s disease. In early stages, metatarsal head flattening and decreased bone density is seen, followed by metatarsal head sclerosis, fragmentation and deformation (Fig.12, arrow). Later on, the presence of metatarsophalangeal osteoarthrosis with intra-articular loose bodies is seen [25].
**Fig. 1:** Antero-posterior (AP) projection radiography of a 55 year old man doing corticosteroids treatment with left hip pain shows patchy areas of bone lucency and sclerosis (arrow).

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**Fig. 2:** Frog-leg lateral view radiography of left femoral head avascular necrosis (same case showed in Fig.1) demonstrating the classic linear subchondral fracture, the "crescente sign".

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Fig. 3: Coronal CT (same case showed in Fig.1 and 2) of left femoral head avascular necrosis showing subchondral sclerosis with geodic cysts associated to left hip arthrosis (arrow).

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Fig. 4: Coronal T1-weighted MR image of the hips in a 60 year old male patient demonstrating the classic hypointense serpiginous line in both femoral heads (arrow) and an area of osteonecrosis with maintained adipose signal intensity (*).

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Fig. 5: Coronal T2-weighted MR image of the hips (same case showed in Fig.4) showing the double-line sign representing a rim of sclerosis (arrows) as an outer border with low signal intensity, and an inner rim of high signal intensity corresponding to the reactive interface or zone of creeping substitution (circle).

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Fig. 6: AP (A) and frog-leg lateral (B) projections radiography of an 11 year old boy showing a Legg-Calvé-Perthes Disease of the right femoral head.
**Fig. 8:** Coronal DP T2-weighted MR image (same case showed in Fig.7) shows a bright band, corresponding to hyperemic granulation tissue in the superior articular surface of the subchondral necrotic bone (arrow). It is also evident a hypersignal along the supraspinous tendon, corresponding to inflammation of the tendon (*).
Fig. 7: 57 year old woman with shoulder pain doing corticosteroids therapy. Coronal MR T1-weighted image demonstrates hyposinal of the superior articular surface corresponding to the ischemic and sclerotic subchondral bone (arrow).

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**Fig. 9:** 67 year old woman with a long standing painful right wrist. AP projection radiography of both wrist showing flattening, loss of volume and some sclerosis of the right lunate bone (arrow) suggesting Kienbock disease.

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**Fig. 10:** Coronal T1-weighted MR image (same case showed in Fig.9) of the wrist shows diffuse decreased signal intensity through the lunate associated with diffuse sclerosis and osteonecrosis (arrow).

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**Fig. 11:** Coronal T2 FAT SAT MR image (same case showed in Fig.9 and 10) demonstrating a diffuse hypersignal of the lunate bone associated with bone oedema (arrow).

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Fig. 12: AP foot projection (A) and oblique foot projection (B) radiography of a 30 year old woman with left foot pain that increases with weigh-bearing. Both radiographic images show flattening and sclerosis of the left second metatarsal head with widening of the metatarsophalangeal joint (arrow).

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Conclusion

Osteonecrosis is a frequent musculoskeletal abnormality that needs correct diagnosis for an optimal and successful patient management.

The typically imaging appearance of a serpentine or undulating sclerotic rim on radiographs is suggestive of underlying pathology.

MR is considered the most sensitive and specific imaging modality due to its ability to prevent future associated complications brought by early diagnosis.
References


