Acta Biomed 2016; Vol. 87, Supplement 3: 6-12 © Mattioli 1885

Review

Imaging of osteonecrosis of the femoral head

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Summary. Osteonecrosis of the femoral head is a common disease affecting both children and adults causing acute hip pain and functional impairment. Among the various techniques allowing a correct diagnosis, MRI represents the gold standard for an early detection, the latter being useful for a positive outcome. The purpose of this review is to describe the imaging findings of the osteonecrosis of the femoral head. (www.actabiomedica.it)

Key words: osteonecrosis, femoral head, imaging

Introduction

Osteonecrosis (ON) is a common disease caused by an ischemic injury toward the epiphyseal and subarticular bone with sparing of the adjacent hyaline cartilage (1); a reduction in blood supply leads to a progressive bone loss and subsequently to a impaired structure, the latter being responsible for complications, such as fractures and end-stage secondary osteoarthritis. The etiology of osteonecrosis is uncertain: the idiopathic form of osteonecrosis is frequent (37% of all cases), although is can be associated with various risk factors (systemic diseases, trauma, therapy with corticosteroids or alcohol abuse) (2). The skeletal regions most frequently affected are femoral head, humeral head, knee (distal femur and proximal tibia) and talus of patients aging between 30 and 50 years (3).

Etiology and pathogenesis of osteonecrosis of the femoral head

Osteonecrosis of the femoral head is a common disease - usually idiopathic and often bilateral - affecting both children (Legg – Calvè – Perthes disease) and adults (Chandler disease); it is newly diagnosed in up to 30.000 Patients yearly in the United State (4). Early diagnosis is difficult because of subtle and non specific initial imaging features but mandatory; indeed, a prolonged ischemic injury causes a progressive femoral head collapse that leads to secondary osteoarthritis, treatable only by total hip arthroplasty.

A reduced oxygen inflow - caused by arterial ischemia or venous congestion - determines the death of various cellular lineage: precisely, the firstly affected are the hematopoietic stem cells, followed by bone cells and - lastly - fat cells. Venous congestion is more dangerous than arterial ischemia because of its quick onset: an excessive storage of toxic catabolites leads to persistent cellular damage (5, 6).

After cellular necrosis, reparative mechanisms undergo three subsequent steps (5, 6):

1. Revascularization: a neuro-mediated increase of blood-flow in the periphery of the damaged bone raises Oxygen availability thus leading to osteopenia because of the osteoclasts' activation.

2. Cellular migration: the increased blood flow activates also osteoblasts, therefore new bony tissue is formed. Moreover, totipotent stem cells migrate from the periphery of the lesion onto the necrotic centre to replace and repair the ischaemic area.
3. Complications: those processes lead to an abnormal bone structure prone to subchondral fractures and trabecular collapse.

Clinical features

Main symptoms are acute hip pain and functional impairment; apyrexia and negative inflammatory markers differentiate ON from acute arthritis (e.g. rheumatoid and septic arthritis); additionally, the absence of specific imaging features at the plain hip radiograph excludes the diagnosis of coxarthrosis (3).

Classification

Many classifications have been proposed for the staging of hip osteonecrosis; the system most widely used is the one proposed by Ficat and Arlet in 1964 and amended in 1985 to include invasive testing procedures (such as measurement of bone marrow pressure, intramedullary venography and core biopsy) and a preradiographic and asymptomatic stage. Such system has been modified to include bone scan and MRI findings rather than the invasive testing procedures; Steinberg/University of Pennsylvania extended the Ficat classification including the quantification of the head's articular involvement (mild: less than 15% - moderate: 15-30% - severe: more than 30%). The Association Research Circulation Osseous (ARCO) proposed, in 1992, a new classification based on combined histologic findings and radiographic, MRI and bone scan imaging. Those classifications are not suitable enough in terms of prognosis, reliability, practicality, and communication among researchers (7, 8).

Imaging

X-Rays

Plain radiographies are usually the initial approach for Patients suffering from hip pain because of their high availability and low cost. Although hip x-rays present low sensibility and specificity, especially in early stages, they can exclude other causes of hip pain (such as hip arthrosis or inflammatory diseases) (9).

X-Rays finding in the Modified Classification of Ficat (7):

- Stage 0: normal plain radiography. This stage can be considered as a pre-clinical and pre-radiological condition; indeed, diagnosis is suspected in one hip when the opposite is affected.
- Stage I: normal plain radiography with hip acute pain.
- Stage II: hip x-ray shows areas of femoral sclerosis associated with diffuse increased porosity and/or cystic changes (Fig. 1); the joint line and the shape of the femoral head are preserved.
- Stage III: the femoral head is now disrupted, although the joint space may be normal or even augmented. After the first 4 weeks, the "crescent" sign (Fig. 2) may be detected, as a linear lucency surrounded by osteosclerosis, expression of subchondral fracture; this sign can be detected in either axial and frog-leg view.
- Stage IV: progressive flattening and sclerosis of the femoral head, expression of subchondral collapse (Fig. 3) leading to secondary end-stage osteoarthritis.

Bone Scan

As well as MR, bone scintigraphy may detect the early stages of osteonecrosis of the femoral head, especially in patients with unilateral diagnosis and negative opposite radiographs. Its high sensitivity is useful for the identification of multifocal lesions, although it is not routinely performed because of its low spatial resolution, low specificity, and inability to quantify the lesion. Bone scintigraphy can be used to detect inflammatory activity in the femoral head when MRI is contraindicated (10).

Table 1. Table presenting the various classifications of hip osteonecrosis commonly used

STAGE	FICAT-ARLET	STEINBERG/UNIVERSITY OF PENNSYLVANIA		ARCO		
0	• Hip pain - • Plan X-Ray - • CT - • RM - • Bone scan -	• Hip pain - • Plan X-Ray - • CT - • RM - • Bone scan -		• Hip pain - • Plan X-Ray - • CT - • RM - • Bone scan -		
1	• Hip pain + • Plan X-Ray - • CT - • RM + • Bone scan +	• Hip pain + • Plan X-Ray - • CT - • RM + • Bone scan +	Head affected •A: <15% •B: 15%-30% •C: >30%	• Hip pain + • Plan X-Ray - • CT - • RM + • Bone scan +	Location	Quantitation
2	Sclerosis and porosity and/or cystic	Femoral head lucency/sclerosis	Head affected •A: <15% •B: 15%-30% •C: >30%	Sclerosis in femoral head, no collapse	medial central lateral	Area •A: <15% •B: 15%-30% •C: >30% Length of crescent •A: <15% •B: 15%-30% •C: >30%
3	Femoral head flattening, subchondral collapse, "crescent sign"	Subchondral collapse without femoral head flattening, "crescent sign"	Involvement of the articular surface •A: <15% •B: 15%-30% •C: >30%	Femoral head collapse, "crescent sign", no joint space narrowing		Surface collapse Dome depression •A: <15% •B: 15%-30% •C: >30%
4	Osteoarthritic joint space narrowing, degenerative changes	Subchondral collapse, femoral head flattening, normal joint space	•A: <15% and <2 mm depression •B: 15%-30% or 2-4 mm depression •C: >30% or >4 mm depression	Osteoarthritic degenerative changes		
5		Flattening with joint space narrowing, acetabular changes, or both	Average of femoral head involvement and estimated acetabular involvement			
6		Osteoarthritic degenerative changes				

Computed tomography

Computed tomography (CT) cannot detect the early vascular and bone marrow alterations of osteonecrosis, although it can highlight osteoporosis in the first stages of disease. Compared with MR and x-rays, CT can easily detect subchondral fractures and small structural collapse. The sclerotic interface between ne-

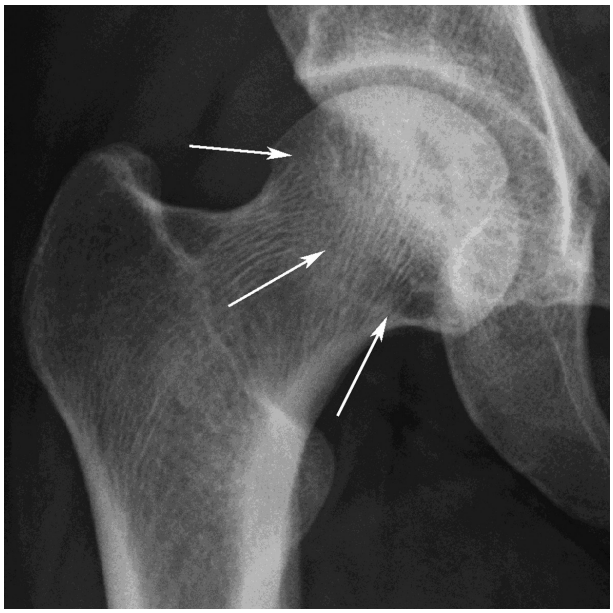

Figure 1. Antero-Posterior view of the right hip x-ray showing alternating regions of sclerosis and lucency on superior aspect of femoral head (stage 2 according to the classification of Ficat e Arlet). Sclerosis indicates areas of new bone on dead trabeculae. Lucency indicates resorption of dead marrow and trabecular meshwork

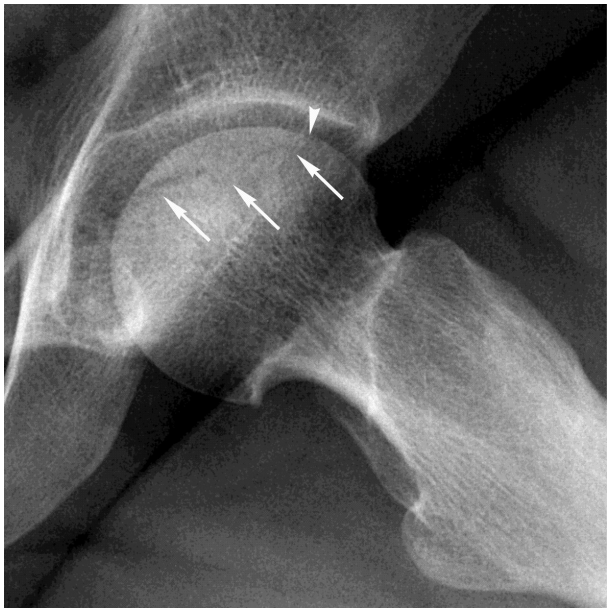

Figure 2. Axial view of the left hip x-ray showing a subchondral fracture line (the "crescent sign", arrows) and a cortical discontinuity (arrowhead) expression of the collapse of the subchondral bone in a 28 years old woman, in dialysis for lupus nephritis, suffering from hip pain

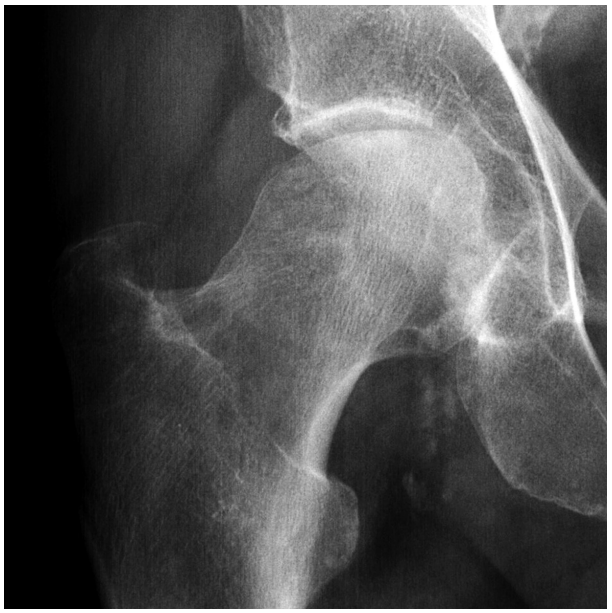

Figure 3. Antero-posterior view of the right hip x-ray show collapse with flattening and sclerosis of the femoral head in a young male.

crotic and viable bone can be demonstrated on CT as well as on MR as a low-signal line surrounding the necrotic bone (Fig. 4) (10).

Magnetic resonance

Magnetic resonance (MR) presents the highest sensitivity and specificity in both diagnosis and follow-up compared to x-rays, CT and bone scintigraphy; it is useful for an accurate quantification and staging, particularly during the phases with negative hip x-rays and as well as during follow-up (11).

MR Classification sec. Mitchell (12) (Tab.2)

- The first **adipose** phase - characterized by arterial ischaemia causing cellular necrosis – results in the death of cellular elements apart from adipocitic cells. The central necrotic area shows a preserved fat-signal (T1w: high signal intensity; T2w: middle-high signal intensity) and is surrounded by a sclerotic-reactive rim (T1w: single density hypo intense 'band-like' lesion; T2w:

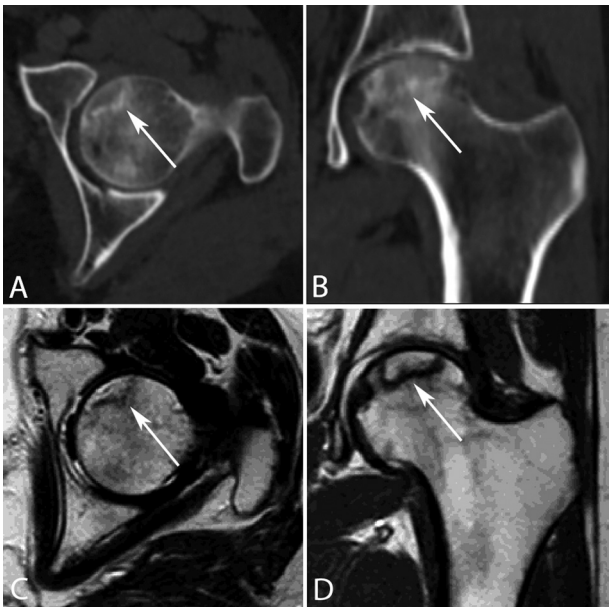

Figure 4. xial and coronal CT Images shows sclerotic interface between necrotic and viable bone (A,B) and analogous line of low signal surrounding the necrotic bone seen on TSE T2 MRI axial and coronal images (C,D)

'double line sign', an hypo-intense fibrotic rim surrounded by an hyper-intense ipervascular rim). (Fig. 5)

- The **haematic** phase is characterized by the presence of micro-haemorrhagic foci within the necrosis (T1w and T2w: high signal intensity).
- The **fluid** phase is caused by the venous congestion that determines a persistent cellular damage leading to the death of all cellular lineage; inflammation, hyperaemia and fibrosis are irregularly distributedT1w: irregular low signal intensity; T2w: irregular high signal intensity).
- The **fibrous** phase shows the predominance of sclerotic and fibrotic alterations (T1w and T2w: low signal intensity).

MR with intravenous injection of paramagnetic contrast media does not provide further information, because the impaired flow within the necrotic area allows a partial and irregular contrast enhancement of the femoral head; however, in the fibrotic phase, imaging with contrast media can show an enhanced peripheral rim surrounding the central necrotic area (13).

Table 2. Mitchell's MRI staging shows the different T1-w and T2-w characteristics signal, according to the tissue present in osteonecrosis area

STAGE	T1-w	T2-w	TISSUE
A	HIGH	MIDDLE	FAT
B	HIGH	HIGH	BLOOD
C	LOW	HIGH	LIQUID
D	LOW	LOW	FIBROSIS

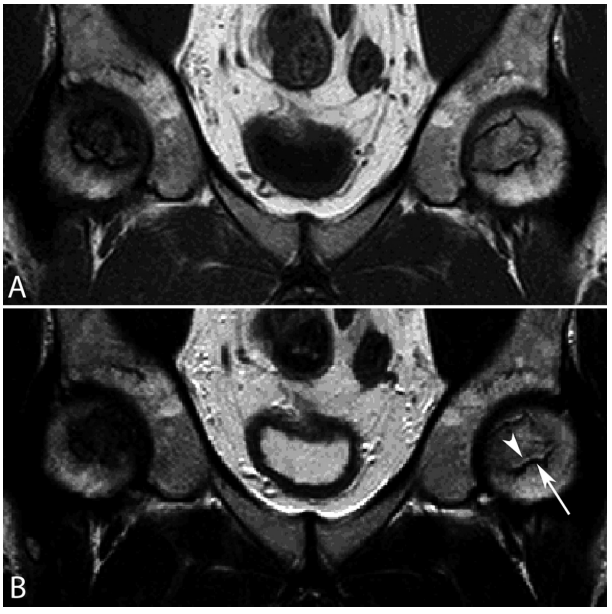

Figure 5. Coronal FSE T1-w (A) and coronal FSE T2-w (B) image of a male patient suffering of bilateral osteonecrosis of the femoral head in different stages of evolution, namely the fibrosis-(on the left) and the fatty-phase (on the right) where shows high signal intensity line in the inner zone (arrow head) and a low-signal-intensity line (arrow) in the parallel outer zone

Differential diagnosis

Once various conditions such as osteoarthritis, osteomyelitis, trauma and septic arthritis are excluded in accord with anamnestic and clinical information, the differential diagnosis of osteonecrosis comprises

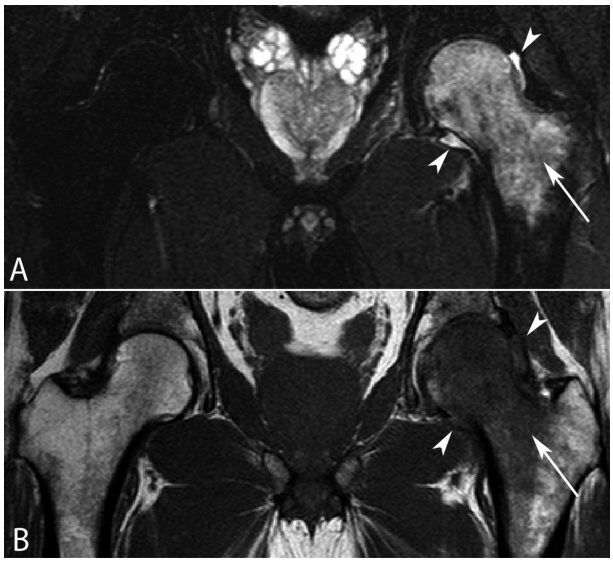

Figure 6. Coronal view of hip-MR in a young male. Patient suffering from algodystrophy demonstrates the typical segmental distribution of bone marrow oedema of femoral head, neck and intertrocanteric region (arrow). (A) DP-SPAIR sequence highlights bone marrow oedema as hyperdense; DP-SPAIR is also useful to detect intra-articular effusion (arrowhead). (B) FSE T1-w image shows the same distribution of bone marrow oedema (arrow), nevertheless intra-articular effusion is not easily identified

"sympathetic dystrophy syndrome" (RSDS or algodystrophy), given their clinical and radiological overlaps (14-19).

Some Authors includes RSDS into a group of pathologies named "bone marrow oedema syndrome", which includes transient osteoporosis of the hip and migrant regional osteoporosis; those idiopathic diseases show a common MR finding, namely bone marrow oedema; nevertheless, their clinical and radiological expression is self-limiting (usual resolution within 6-9 months). Typical MR feature of hip's RSDS is represented by a diffuse trabecular oedema of femoral head and neck, associated to abundant joint effusion, the latter being usually absent in osteonecrosis (Fig. 6). After intravenous contrast media injection, segmental RSDS may show an intense contrast enhancement in the synovial membrane of the joint capsule suggesting a reactive synovitis. The high signal of oedema detected in T2-w images is usually homogenous in RSDS, whereas it is heterogeneous in osteonecrosis due the presence of granulation tissue and micro fractures. RSDS's oedema is accompanied by osteoclastic hyperactivity causing osteopenia. Moreover, trabecular oedema of RSDS involves both femoral head and neck, whereas in osteonecrosis it involves only the proximal third of the femoral head. Some Authors consider algodystrophy as an initial stage of osteonecrosis because they observed that some cases of algodystrophy evolved into osteonecrosis.

Conclusions

Magnetic resonance imaging plays a crucial role in the early detection and staging of osteonecrosis; it allows clear quantification and evaluation of lesions that are undetectable on plain radiographs.

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