



Diagnosis of Aseptic Osteonecrosis of the Femoral Head (AONTH)

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Abstract: Aseptic osteonecrosis is a frequent pathology. It corresponds to epiphyseal bone necrosis, secondary to ischemic and/or cytotoxic mechanisms. It is not a specific disease, but rather the final outcome of various pathological situations in which there is a disturbance of the intraosseous circulation of the femoral head. This explains the usual names of avascular necrosis or aseptic necrosis. It may be unilateral, bilateral or multifocal. More than 75% of aseptic osteonecrosis involve the femoral head. Many risk factors must be considered, such as corticosteroid therapy, alcoholism, dyslipidemia or sickle cell disease. It is important to diagnose osteonecrosis in the potentially reversible stages of the disease. It is a condition that is all the more disabling because it affects the young adult, in the middle of an active life, in the presence of pain in the groin crease of a young man who tends to take steroids, when the femoral head is still spherical on standard radiography. Magnetic resonance imaging has profoundly changed our diagnostic possibilities, showing pathognomonic signs while the radiograph may still be normal. Aseptic osteonecrosis of the femoral head (AONTH) often poses a problem of differential diagnosis with other coxopathies, which is a source of diagnostic delay in medical practice.

Keywords: Osteonecrosis, Aseptic Osteonecrosis, Etiologies, Diagnosis

1. Introduction

Osteonecrosis can be defined as the cell death of the different components of the bone, i.e. bone tissue but also bone marrow. It is not specific disease, but the consequence of an interruption of blood flow caused by various local or general conditions. The exact incidence of AON is not well known because of its pauci-symptomatic or asymptomatic nature, which is the main factor in delayed diagnosis. AONFH is a multifactorial pathology. A wide variety of etiologies such as massive alcohol consumption, dyslipidemia, hypercorticism, blood flow disorders or thromboembolic pathologies have been proposed as responsible for this disease. In all cases, an abnormality of

femoral head perfusion combining platelet aggregation and intravascular coagulation is found [1-3]. In about 30% of cases, no underlying etiology is found after a long negative etiological workup, and AONFH is considered idiopathic (iAON) [4, 5]. The diagnosis is guided by an interrogation and a meticulous clinical examination, the paraclinical explorations allow us an early diagnosis of certainty. The consequence of this disease development is leading to loss subchondral tissue and an loss of the femoral head sphericity [5].

Physiopathology (figure 1)

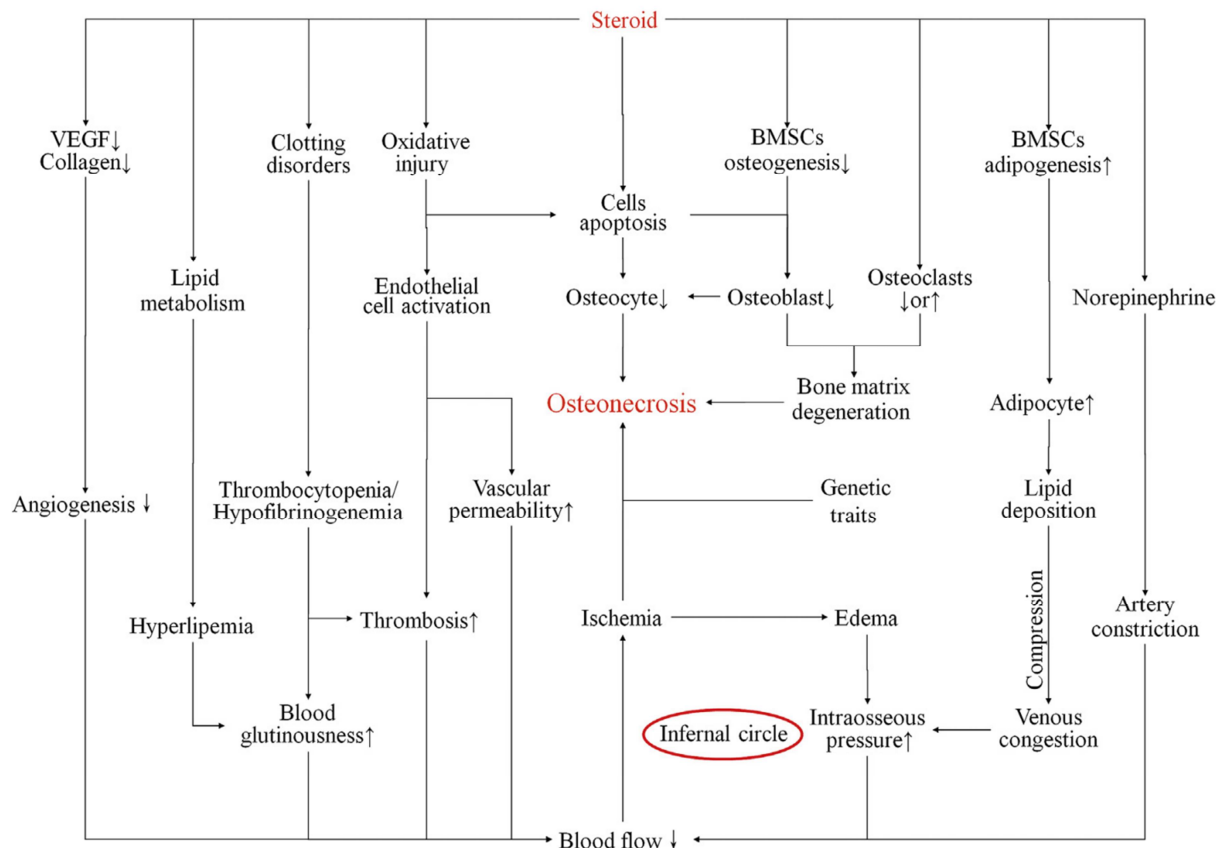


Figure 1. Pathophysiological diagram of osteonecrosis [6].

2. Etiological Diagnosis

2.1. Individualized Causes

Traumatic.

2.1.1. Fracture

Fractures of the femoral neck, especially subcapital, interrupt most of the vascularization of the femoral head. The frequency of AONFH is at least 30% and increases in displaced fractures, especially in young adults.

2.1.2. Dislocation

AONFH is common in hip dislocation because of possible vascular compression, and the factors favoring AONFH are the delay in reduction (a reduction performed 6 hours after the dislocation occurs will increase the risk of ON by vascular compression) [7].

2.1.3. Caisson and Diver's Disease

It is due to a sudden atmospheric decompression, the mechanism would be the formation of nitrogen bubbles in the tissues, its accumulation in the organism allows it to appear in intra and extra vascular bubbles that can lead to circulatory arrest [8].

2.1.4. Gaucher Disease

Gaucher disease is a rare autosomal recessive genetic disorder caused by the abnormal accumulation of glucocerebroside in

cells. secondary to mutations in the gene (GBA) encoding β -glucocerebrosidase [9, 10]. However, different mechanisms seem to be involved. First, Gaucher cells infiltrate and alter trabecular bone. Secondly, the inflammatory process intrinsic to the disease leads to activation of osteoclasts and inactivation of osteoblasts, resulting in the appearance of large bones that can compress the vessels. Thus, AONFH is one of the most frequent complications of left-handed disease [11-13].

2.1.5. Sickle Cell Disease

This is the most common associated disease. Osteonecrosis of the femoral head has been reported in 30-50% of patients depending on the type of hemoglobinopathy. AONFH is the consequence of a major alteration of sickle cell blood with a decrease in the deformability of red blood cells linked to the polymerization of hemoglobin S. In case of hypoxia, polymers appear and the initially discoid red blood cell loses its deformability leading to the obstruction of certain vascular territories [14].

2.1.6. Radiation Necrosis

Observed after irradiation for pelvic cancers. The necrosis is related to the direct effect of radiation on the bone cells and indirect effect by vascular lesions.

2.2. Risk Factors

2.2.1. Traumatic

Minor trauma (contusion).

2.2.2. Non-traumatic

Corticosteroids (dose and duration dependent), alcoholism (dose dependent), disturbances of lipid metabolism, hyperuricemia and gout, pregnancy, organ transplants, systemic lupus erythematosus, other collagenoses, arteriosclerosis, other occlusive vascular diseases, diabetes mellitus, carbon tetrachloride intoxication, hypofibrinolysis syndromes, hypercoagulation syndromes, leukemia, chemotherapy, smoking, HIV, genetic predisposition. The two main precipitating factors are alcoholism and corticosteroid therapy [13]. The risk of AONFH following corticosteroid therapy is also dose-dependent, but cases have been reported with high or low doses of short duration, or even after simple prolonged local administration (infiltrations, spray, creams) [15]. ONATF has been reported in HIV-positive subjects, but its occurrence seems to be more related to known risk factors (corticosteroids in particular) than to HIV itself [16] or to antiretrovirals [17]. Smoking is a recognized risk factor [18]. A few dozen cases have been reported during pregnancy [19].

Idiopathic or primary aseptic osteonecrosis (NAOI): no etiology is found in 10 to 40% of cases. Preferably men in their forties. Bilateral in 70% of cases.

3. Positive Diagnosis

3.1. Functional Signs

There is nothing specific about them. The patient can often remain asymptomatic (majority of cases) and the disease can be diagnosed on the X-ray because of pain in the opposite hip ("silent hip" of Marcus). On the other hand, sometimes the patient complains of pain for weeks or months without the X-ray showing any abnormality. As with all hip pain, it is most often found in the groin (93%), but may also be in the buttock (34%), thigh (36%), greater trochanter (9%), and lumbar region (8%), and even in the knee (68%). It is usually increased by weight-bearing, but may persist at rest. Subsequently, the patient may experience lameness and then limitation of motion. The examination is also aspecific. Hip motion is often within normal limits, even when the radiograph shows advanced ONATF. When the femoral head has collapsed, joint limitation is more marked and painful.

3.2. Standard Radiography

Often performed for other reasons, is always behind other imaging methods. If the earliest radiological sign is a densification within the femoral head, standard radiography is especially interesting to look for a subchondral dissection. Indeed, it is the most reliable examination to look for the classic eggshell or loss of sphericity of the femoral head [20]. A classification of radiological signs has been proposed by Arlet and Ficat according to the stage of discovery (figure 2).

Hip pain in a young person with a normal radiograph requires an MRI of the hip.

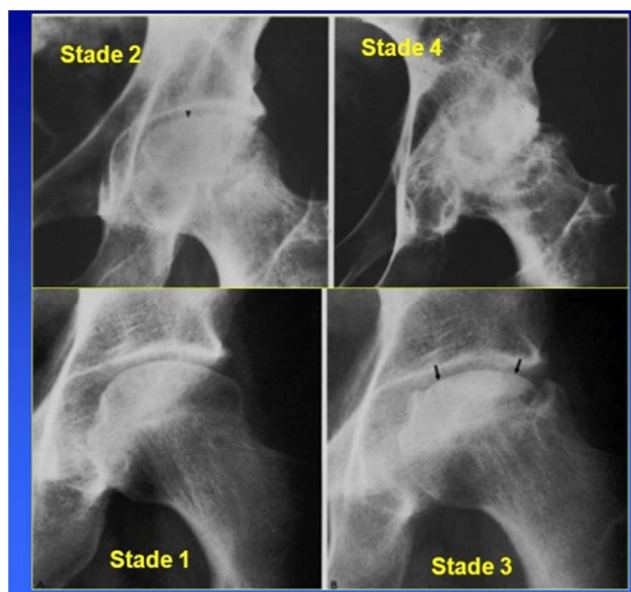


Figure 2. AONFH radiological classification [21]).

3.3. MRI (Magnetic Resonance Imaging)

Only MRI, with a sensitivity and specificity of almost 100%, is diagnostic and provides information on the status of the contralateral hip, which may be asymptomatic. MRI is usually performed on both hips, as the disease is frequently bilateral. On the images obtained, the analysis seeks to specify [22].

3.4. The Focus of Necrosis

Normal signal at the beginning, which may become heterogeneous late on, or even entirely obscure/ may merge with the border.

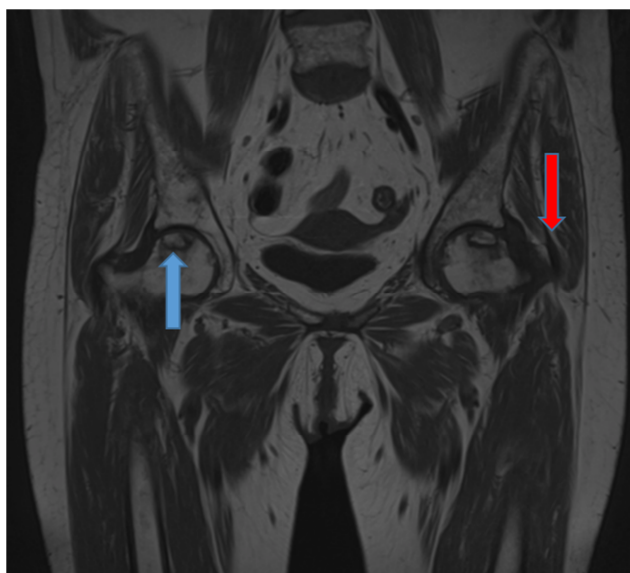


Figure 3. MRI bilateral osteonecrosis of the femoral heads (23). The necrotic area is circumscribed by a T1 hyposignal border. The radiograph showed a stage II of Arlet and Ficat. MRI stages are subdivided into. A: Necrotic area < 15%. B: Necrotic area= 15-30%. C: Necrotic area > 30%.

3.5. The Border (Essential Sign)

Hypo-signal line in T1 and T2, which often has a circular, irregular, roughly upward concave shape and whose ends connect seamlessly with the contour of the femoral head. In half of the cases, this line is matched by a duplicate: a hyper-signal border of the same contour (figure 3).

3.6. Joint Effusion

Frequent, marked in hypo-signal in T1 and especially clear in hyper-signal in T2.

3.7. Intraosseous Edema

Inconsistent, blurs the signal of the fat, decreased in T1.

3.8. Computed Tomography (CT)

Sometimes used because of its accessibility compared to MRI, shows the same lesions as radiography, but in greater detail (figure 4). Its value lies in its ability to better identify subchondral fractures (a major complication of AONFH) and early epiphyseal collapse [23].

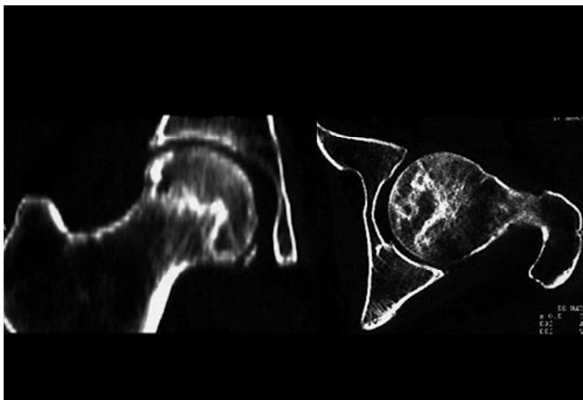


Figure 4. Hip scan, osteonecrosis of the left femoral head [24].

3.9. ^{99m}Tc Bone Scintigraphy

Specific, it may show a cocoon-shaped area with perilesional hyperfixation, surrounding a hypofixing area, testifying to the attempted repair (figure 5). Its interest lies in the possibility of identifying in a single examination other recent or semi-recent AON locations on the entire skeleton, but it remains less sensitive than MRI [25].

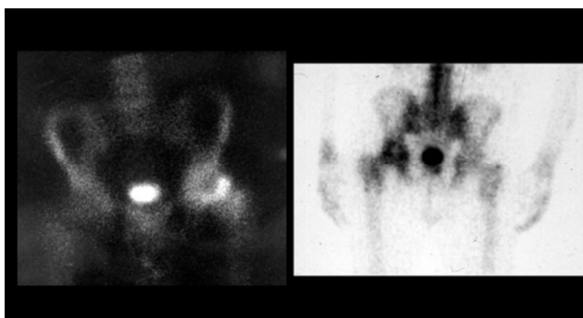


Figure 5. Bone scan of the pelvis showing hyperfixation at the right femoral head in the setting of AONFH [26].

4. Differential Diagnosis

4.1. Regional Complex Pain Syndrome (RCPS)

Formerly called femoral head algodystrophy, it sometimes occurs in the same clinical setting as ONATF, the radiograph may be normal or show localized demineralization, without the classic "evanescent hip" appearance. MRI shows diffuse spinal cord edema; an area of hyposignal T1, which changes an area hypersignal T2 or fat-suppressed sequences (figure 6) without the T1 and T2 hyposignal border that limits ON [27].

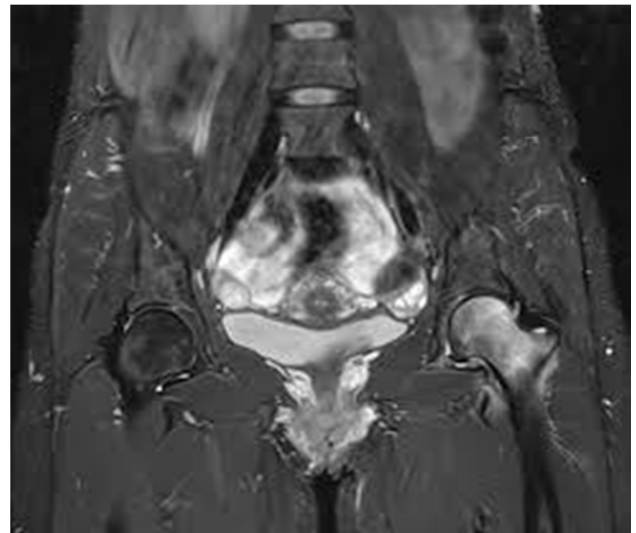


Figure 6. RCPS of the left hip [28].

4.2. Subchondral Tired Fracture

This is the most difficult diagnosis. Its confusion with AONFH explains a certain number of so-called osteonecroses that heal spontaneously! This subchondral fatigue fracture heals on its own and should be recognized on MRI images: within a medullary edema (same MRI signs as algodystrophy), the foci of trabecular impaction, located at variable distances from the cephalic contour, are manifested by linear or arciform bands of hyposignal, perpendicular to the axis of the weight-bearing trabeculae, better visible in high-resolution sagittal section and in T2 weighting. Because these bands may join the subchondral bone contour of the femoral head, they may sometimes simulate the reactive interface of osteonecrosis. In this case, differentiation with a complete demarcation line may be tricky. On T2-weighted sequences, a hypersignal line frames the fractured area [29].

4.3. Chondroblastoma of the Head of the Femur

Exceptionally localized, it may give the same MRI images as ON, especially when intra-tumoral calcifications (on CT) and lobular architecture (on MRI) suggestive of cartilage tumors are lacking [30].

4.4. Other

Coxarthrosis and other coxopathies, Paget's disease, cruralgia.

5. Evolution

In the absence of treatment, the natural course of AONFH is subchondral fracture, followed by irreversible joint surface deformity (collapse), with a poor joint prognosis. This is particularly true because the hip is a weight-bearing joint. If this collapse of the joint surface occurs, it usually takes place within two years of the diagnosis of AONFH. Between 32 and 79% of ONFH result in joint surface collapse [31, 32].

6. Treatment

The study of treatments for aseptic femoral head osteonecrosis, whether drug, physical, or surgical, suffers from weak trial methodology. The treatment options for AONFH depend essentially on the stage (before or after the subchondral fracture, assessed by X-ray or, better, by CT scan) and the extent of the lesion on MRI. Prevention of AONFH is primarily based on minimizing risk factors, including the use of the lowest possible doses of corticosteroids. Lipid-lowering drugs and anticoagulants may have a role, but this has not been established. Once AONFH has occurred, the goal is to avoid progression to subchondral fracture. None of the treatments considered (bisphosphonates, extracorporeal shock waves, pulsed magnetic fields, hyperbaric chamber, cervicocephalic drilling) has clearly demonstrated its effectiveness. Drilling with autologous bone marrow stem cells seems to be the most promising. At the stage of subchondral fracture, total arthroplasty remains the reference method if the functional discomfort justifies it. Osteotomies are a little-used alternative, as they are relatively heavy and technically difficult in some cases [33].

7. Conclusion

AONFH is a multifactorial and relatively disabling condition. Its little or asymptomatic clinical feature and the large number of differential diagnoses constitute the fundamental elements of the diagnostic delay. From a practical point of view, it is important to make the diagnosis of ON in the potentially reversible stages of the disease, standard radiography shows the lesions only in the early stages of the disease, and CT shows the radiographic lesions more clearly; thus, MRI is the best examination for early diagnosis of certainty, and is very sensitive, as it shows hyperfixations even in the pre-radiological stage of the disease. The management depends on the evolutionary stage and is based on the unloading of the limb, symptomatic treatment in the early stages and surgery for the advanced stages.

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