REVIEW ARTICLE

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Non-traumatic necrosis of the femoral head

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Non-traumatic, avascular, aseptic or ischemic necrosis of the femoral head (ON) is characterised by death of cells in the cancellous bone. Its etiology is unknown. It usually leads to collapse of the load bearing segment of the hip joint, and eventually to severe osteoarthritis.

Epidemiology

The incidence of ON is unknown. Many early cases are probably not diagnosed, and many late cases are probably misdiagnosed as osteoarthritis. About 5–10% of total hip replacements (THA) are performed due to ON [25].

Jacobs [17] found that treatment with corticosteroids (28%) and alcoholism (39%), and diseases like Gauchers disease, sickle cell disease, decompression sickness and primary Cushings disease are the most common conditions associated with ON. In 25% no associated conditions or diseases could be found. More than 3/4 of the patients are men in the third to fifth decade, and in more than half the cases both hips are affected [17].

Pathogenesis

Several theories about the pathogenesis of ON have been suggested, all leading to a common pathway of interruption of the blood circulation to the antero-lateral part of the femoral head.

At histological examination, an early finding in ON is disappearance of haematopoietic marrow and interstitial edema and haemorrhage. Next, the marrow cells become necrotic, and the marrow is invaded by fibrous tissues and granulation tissues. The osteocytes of the trabeculae become necrotic, and gradually, the trabeculae

might become resorbed. A subchondral fracture line may appear just beneath the bony end plate (the “crescent sign”), and loss of structural support may lead to collapse of the femoral head. At the transitional zone, between normal bone and the necrotic lesion, new bone formation in apposition to the dead trabeculae is seen (“creeping substitution”). Until degenerative changes appears, the articular cartilage of the joint remains unaffected. [2, 10].

Jones [18], suggested that destabilisation or coalescence of plasma lipoproteins, hyperlipemia or disruption of marrow fat might lead to fat embolism in the small subchondral vessels of the femoral head. These fat globules might then trigger a process of endothelial damage, focal intravascular coagulation and platelet aggregation, eventually leading to thrombosis, ischemia and ON.

Most diseases and conditions that are associated with ON might become complicated by accumulation of fat or disturbed fat metabolism, and experimentally, necrosis of marrow fat cells and fat embolism in the small vessels of the femoral head was found in steroid treated rabbits [7, 13]. Recently, coagulation disorders like thrombophilia and hypofibrinolysis was found in patients with osteonecrosis [12].

Hungerford [16] regarded the femoral head as a closed, non-distensible compartment crossed by thin walled compressible vessels (like a Starling resistor). An increase in the intraosseous, extravascular pressure due to expansion of the marrow tissues (fat cells and haematopoietic tissues) would cause compression of the vessels, and thus a reduced blood flow to the femoral head. This theory found clinical support as an increased intraosseous pressure, and a delayed run-off of contrast in intraosseous phlebography was documented in several studies [4, 15, 22]. Experimentally, an increase in the size of marrow fat cells in steroid treated rabbits was found [36].

Recently, Starklint et al. [33] found that the intraosseous veins and venules in the transitional zone at the rim of the necrotic sequester were occluded due to intravascular fibrin thrombi of different ages. This finding could

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Fig. 1a–c Steroid induced osteonecrosis of both femoral heads. **a** Plain radiography, computer tomography, magnetic resonance imaging and ^{99}Tc -MDP bone scintigraphy. **b** Coronal section of the femoral head shows the different zones of ON, featuring the characteristic subchondral fracture. **c** The soft area antero-lateral on the femoral head without subchondral support has been delineated

explain the increased intraosseous pulsatile pressure, the decreased microvascular blood flow and the decreased partial pressure of oxygen that has been found in femoral heads with ON [20, 23].

These different theories of the pathogenesis of ON are to some degree supporting rather than contradicting each other, and they are probably reflecting that ON is a multifactorial disease with several different pathogenic pathways.

Symptoms and diagnosis

The onset of ON is often gradual, but might be sudden, with progressive pain in the groin, radiating to the thigh and often worse at night. Usually, there is a limitation of

movement of the joint, specially of the internal rotation. The natural history of ON is usually that of relentless progression, and more than 80% of the patients will experience a collapse of the femoral head in a few years after its onset [10, 25]. The diagnosis is mainly based on radiography, ^{99}Tc -bone scan and, in recent years, on magnetic resonance imaging (MRI) (Fig. 1). The most widely accepted classification of ON of the hip has been suggested by Ficat [10]:

Stage 0

No clinical symptoms. Radiographs are normal, and ^{99}Tc -bone scan is normal or might show reduced uptake

of isotope, or even a “cold-in-hot sign”. Intraosseous pressure will be increased, run off at intraosseous phlebography will be delayed, and at histology characteristic features of ON is found. This is “the silent hip” of ON [24], that might be suspected in the contralateral hip of patients with ON.

Stage 1

The patient is having pain in the groin, radiating to the thigh, often worse at night. The onset is sudden or may be more insidious. Radiographs are still normal or subtle osteoporosis might be seen. ^{99}Tc -bone scan shows increased uptake of isotope.

Stage 2

The pain in the hip is persistent or increasing in severity. Radiographs shows mixed osteoporosis and osteosclerosis, and, as a transition to the next stage, a typical antero-lateral wedge-shaped sclerosis may occur. There might be slight flattening of the femoral head and the pathognomonic “crescent sign”, a fine subchondral fracture line, might appear. ^{99}Tc -bone scan shows increased uptake of isotope in the femoral head.

Stage 3

Usually a rapid increase in clinical symptoms occurs. Radiographs reveals flattening and even collapse of the femoral head, and a typical sclerotic sequestrum appears antero-laterally. The joint space is maintained, but it is uneven and eccentric. ^{99}Tc -bone scan shows increased uptake of isotope.

Stage 4

Clinical symptoms are severe. On the radiographs, collapse of the antero-lateral part of the femoral head are seen, the joint space is narrowed, and there is involvement of the acetabulum, indicating that secondary arthritis has now developed due to the mechanical collapse of the femoral head.

Thus, plain antero-posterior radiographs are essential in the diagnosis of ON. These should be supplemented with radiographs in the “frog position” with externally rotated hips, and with ^{99}Tc -bone scan in early cases.

If available, MRI is now considered to be the most sensitive diagnostic tool in the early stages of ON, and the sensitivity and specificity of MRI was between 80 and 100 per cent in several studies. In very early stages, a subchondral hypodense zone or a band-like zone of decreased signal intensity in the upper part of the femoral head might be seen on T1-weighted spin-echo images.

Later, this demarcation might become more pronounced, and a double band on T2-weighted images which is pathognomonic of ON, might appear [1, 14].

The degree of involvement of the femoral head as calculated on sagittal and coronal images was found to be prognostic for the development of ON, and an involvement of more than half of the weightbearing portion of the femoral head predicted subsequent segmental collapse [21, 31]. However, a considerable inter- and intra-observer variability in quantifying the extent of the necrosis has been found [19].

Treatment

Several studies have reported on the results of conservative treatment of ON with analgesics and non-weight-bearing, or no treatment at all. In general, the results were poor, as the disease progressed clinically and radiographically within a few years in more than 80% of the patients, irrespective of the underlying cause of ON [27].

Core decompression has proven successful in the treatment of ON in the early stages in many studies. By removing an 8 mm core of cancellous bone from the femoral neck and head the intraosseous pressure is normalised, and this is supposed to restore the microcirculation in the capillaries and sinusoids [10]. Further, vascularized tissue might invade the tunnel of the core, initiating repair of the necrotic bone [32].

In a megastudy of 24 reports on core decompression, a success rate of 84% for stage 1, 65% for stage 2 and 47% for stage 3 was found, at a mean duration of 30 months postoperatively [26]. Beside the stage of the disease, size of the lesion as estimated on MRI, and whether the lesion is predominantly cystic or sclerotic is probably predictive of the outcome [3].

Core decompression is an easy procedure that carries little risk of complications, and in case of progression of the disease it has no adverse influence on performing a THA. However, it is mandatory, that the procedure is performed at an early stage before any collapse of the femoral head has occurred. Also, it is very important to place the entry point of the coring device correctly, just distally to the flare of the trochanter, to avoid it acting as a stress riser [9].

In the classical *Phemister operation*, a non-vascularized cortical bone graft from the fibula or from the iliac crest is inserted into the femoral head and neck to provide structural support, and prevent collapse of the subchondral bone [28]. In some studies, the graft has been vascularized [37], or combined with pulsed electromagnetic fields [34]. *Intertrochanteric osteotomy* with flexion and adduction of the femoral neck or rotation of the femoral head has also been used in order to remove the necrotic part of the femoral head from the weightbearing dome of the hip joint [35]. Good results were obtained in some of these reports, but as yet, the cumulat-

ed experience and the reported long-term results are limited.

By and large, the result of these operations compares with core decompression and non-vascularized bone grafting, but as a high rate of complications has been reported, these demanding procedures are probably not justified for treatment of ON in the ordinary, non-specialised clinic at present.

Total hip replacement has been the treatment of choice for late stage ON during recent years. The results have been disappointing in several reports, with an early failure rate of up to 38% probably reflecting the relatively young age of these patients who are often suffering from concomitant diseases. Also, the possibility of an ongoing progression of ON distally into the metaphysis of the femur has been proposed [6, 30]. However, using contemporary techniques, other studies have reported results after THA in patients with ON that compares with patients with osteoarthritis [5, 11, 29]. Also, uncemented prosthesis has proven successful in these patients, although premature loosening of the acetabular component has caused concern [8].

Non-traumatic osteonecrosis of the femoral head remains a challenging disease, and many questions about epidemiology, pathogenesis, early diagnosis and treatment are still unsolved. In the future, screening for coagulation disorders might select patients with an added risk for having ON [12], and further knowledge of the pathogenesis will probably emerge. Developments in MRI with and without contrast might add to the early diagnosis of ON and we will probably be able to predict which lesions will heal spontaneously and which will progress to collapse of the femoral head. Adjuvant use of specific bone stimulating cytokines might promote healing of the early lesions [25], and further improvement in design and materials of THA will probably extend the longevity of the prosthesis, even in patients with ON.

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