Osteonecrosis of the knee: a concise review of the current literature

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ABSTRACT

Osteonecrosis of the knee is a progressive disease that can lead to subchondral collapse and end-stage osteoarthritis of the knee. Originally described as a single disease, it includes three different pathologic entities: spontaneous osteonecrosis of the knee, secondary osteonecrosis of the knee, and post-arthroscopic osteonecrosis of the knee. This article reviews the current literature of these three different conditions by describing their epidemiology, etiology and pathogenesis, clinical presentations and radiographic findings. Various treatment option (e.g., core decompression, bone grafting, stem cells implantation, tibial osteotomy and arthroplasty), available for each entities are reviewed.

KEYWORDS

Osteonecrosis, spontaneous osteonecrosis, SONK – secondary osteonecrosis, atraumatic osteonecrosis, avascular necrosis, post-arthroscopic osteonecrosis, knee.

Introduction

Osteonecrosis (ON) is cell death by ischemia of bone and bone marrow tissue. Avascular necrosis frequently affects the epiphyses of long bones: the femur is statistically the most affected bone, both in the hip (the most common location) and in the knee (the second most common). Osteonecrosis of the knee can be a devastating disease that leads to end-stage osteoarthritis of the knee. Ahlbäck *et al*. [1] first described ON of the knee in 1968. It was initially presented as a spontaneous condition typically involving the medial femoral condyle and showing greater prevalence in women over 60 years of age, following minor trauma. Later studies identified patients whose characteristics and symptoms did not match these initial descriptions, and this led to the recognition, by Zywiel *et al*., of three different entities: secondary ON, spontaneous ON, and post-arthroscopic ON [2,3].

Primary osteonecrosis or spontaneous osteonecrosis of the knee

Epidemiology

Spontaneous osteonecrosis of the knee (SONK) is the most common of the three entities. Its actual prevalence may be underestimated, as many patients who present with end-stage osteoarthritis may have had unrecognized SONK [4].

Pape et al. reported a 3.4% incidence of SONK in persons aged over 50 years, rising to 9.4% after the age of 65 years [5].

This disease more often affects women than men, is typically unilateral, and mainly affects the medial femoral condyle in 94% of cases. It has also been reported to affect the lateral femoral condyle, tibial plateau, and patella [6,7].

The predominant involvement of the medial femoral condyle may be explained by local differences in blood supply between the medial and lateral condyles [8].

Article history
Received 16 Nov 2021 – Accepted 16 Mar 2022

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Etiology and pathogenesis

For many years, the pathogenesis of SONK was considered to be related to vascular impairment, leading to necrosis. Recent pathologic findings have suggested that it may be secondary to subchondral insufficiency fractures in osteopenic bone, associated with subsequent edema leading to focal ischemia and eventually necrosis [9]. In support of this, a recent study demonstrated an association between low bone mineral density and the onset of SONK in women over 60 years of age [10].

Moreover, meniscal tears were found to occur in 50 to 100% of patients with SONK. It could be that disruption of the posterior medial meniscus root increases tibiofemoral contact pressures, and that resulting alteration of normal knee biomechanics causes the subchondral insufficiency fractures seen in SONK [11].

Berger *et al*. [12] prospectively assessed bone turnover in 22 patients with SONK, demonstrating that mean serum concentrations of the main biochemical markers of bone metabolism, such as bone-specific alkaline phosphatase (bone ALP), osteocalcin (OC), procollagen type I N-terminal propeptide (PINP), and C-terminal cross-linking telopeptide (ICTP), were not different from those found in healthy persons and hence were of no value in the diagnosis of spontaneous ON. Moreover, elevated markers in samples obtained from cancellous bone indicated increased turnover both in knee ON and in osteoarthritis patients compared with healthy cases.

Clinical presentation

SONK can be distinguished from secondary ON of the knee by its insidious onset and lack of identifiable etiology.

The pain is severe and often localized to the medial side of the joint, with lesions of the medial femoral condyle causing pain mimicking that of a medial meniscus tear. There is usually no notable history of trauma. Pain both at rest and at night, as well as with weight bearing, is common and can be quite debilitating. Focal tenderness to palpation over the medial femoral condyle is the most common finding on physical examination [7,13].

Classification

Osteonecrosis of the knee can be staged to assess the severity and to guide treatment. The Koshino classification of 1979, originally developed for SONK but subsequently extended to the other types, consists of four stages (Table I). Stage I is that of a patient with knee symptoms but normal X-ray findings. Stage II consists of condyles flattening and subchondral radiolucencies without collapse. In stage III there is subchondral collapse, while stage IV consists of further degenerative changes with osteosclerosis and osteophyte formation [14].

Another classification is the modified Ficat and Arlet staging system, adapted for the knee from the original version describing ON of the femoral head. It is based on radiological findings (Table II). A stage I patient has normal X-ray findings. In stage II there are cystic or osteosclerotic lesions with a normal contour of the distal part of the femur, while in stage III a crescent sign or subchondral bone collapse is detected. Stage IV consists of osteoarthritic changes [15].

Diagnosis

At the initial evaluation, anteroposterior, lateral, and oblique view radiographs should be performed, although in the early course of the disease they are often negative (and in some cases remain negative) or can demonstrate radiolucency of the subchondral bone surrounded by an area of sclerosis or flattening of the affected condyles, and collapse of the subchondral bone in later stages of the disease [16].

Magnetic resonance imaging (MRI) is useful in the early stages of the disease due to its high sensitivity in detecting bone edema. Initial MRI findings include bone marrow edema localized to the medial femoral condyle, and a subchondral crescent of a linear focus of low signal intensity on T1- and T2-weighted sequences, located on the central weight-bearing aspect of the femoral condyle; the overlying articular cartilage is intact. The subchondral bone collapse phase is characterized by the presence of focal depressions of the epiphyseal contour and a fracture line invaded by subchondral fluid. Concurrent meniscal tears are common [17,18].

The prognosis depends on the size of the lesion: larger lesions indicate an increased risk of osteoarthritis, as demonstrated by Juréus *et al*., who found that the development of osteoarthritis was likely when 40% or more of the joint surface was affected [19].

Small lesions (<3.5 centimeters squared) usually regress with non-surgical management, while large lesions (>5 centimeters squared or > 50% of femoral condyle) usually lead to condyle collapse and osteoarthritis [20].

Treatment

Non-operative management is usually reserved for smaller lesions (<3.5 cm^2) and consists of lateral wedge insoles, non-steroidal anti-inflammatory drugs, analgesia, protected weight bearing, and bisphosphonates; some authors have used pulsed electromagnetic fields therapy [4,21,22].

Recently, Hernigou *et al*. performed a literature review analyzing the role of implantation of fresh autologous subchondral

Table I Koshino classification.

STAGE	RADIOLOGICAL FINDINGS
1	Normal appearance
2	Weight-bearing area with radiolucent oval shadow in the medial femoral condyle, flattening of the condyle
3	Collapse of subchondral bone plate
4	Osteoarthritic changes such as osteosclerosis and osteophyte formation, with a shallow concave articular surface at the osteonecrotic region

Table II Modified Ficat and Arlet staging system.

STAGE	RADIOLOGICAL FINDINGS
I	Normal X-ray findings
II A	Cystic or sclerotic lesion (absence of subchondral cystic formation)
II B	Subcondral collapse (crescent sign) and/or subchondral aliasing
III	Irregular femoral contour
IV	Collapse of the femoral head, acetabular involvement and osteoarthritis changes

bone marrow aspirate concentrate (BMAC) or/and of scaffolds loaded with BMAC containing mesenchymal stem cells in an osteoarthritic knee, and demonstrated that it could improve pain and survivorship of the joint, and could delay the need for arthroplasty [23].

The same group suggested that computer navigation may be safely used in a basic procedure for the injection of stem cells in knee ON, allowing improved precision with less radiation [24].

Surgical management should be considered when patients do not improve clinically or radiographically after three months of non-operative treatment, as well as in patients who present with osteonecrotic lesions larger than 5 centimeters.

In the pre-collapse stages (Ficat I and II), joint-preserving surgical techniques such as arthroscopy, core decompression, bone grafting, and tibial osteotomy may be used.

Arthroscopy for knee ON remains undefined; however, it allows additional assessment of ON lesions, and coexisting meniscal tears or chondral lesions can be addressed at the same time [25].

The therapeutic benefit of core decompression is the result of reduced medullary pressure and increased neovascularization, driving healthy bone formation. Marulanda et al. introduced a small diameter (3.2 mm pin) drilling technique, based on a similar procedure previously reported for the hip; it is a percutaneous approach performed under fluoroscopic guidance and has shown a success rate > 90% [26].

Bone grafting is used in patients with early-stage knee ON: the osteonecrotic lesion is removed and then the bone defect is reconstructed with a bone autograft or fresh frozen allograft introduced through an extraarticular cortical window [27,28].

In the pre-collapse stage, core decompression gives good results in 90% of cases; autologous grafting gives good results in 100% in the pre-collapse and in 90% in the post-collapse stage [29].

Tibial osteotomy is indicated in younger and more active patients, who wish to avoid prosthetic replacement. It is performed to offload the proximal tibial metaphysis, thereby unloading the affected femoral condyle and potentially promoting healing of the lesion. A valgus-producing medial opening wedge osteotomy may be utilized in SONK, as the disease typically affects the medial compartment of the knee [4].

In the collapse stages (Ficat III and IV), patients may benefit from osteochondral autograft or mosaicplasty, due to restoration of the cartilage surface; localized lesions are filled using autologous osteochondral tissue harvested from uninvolved articular surfaces that undergo less weight bearing [30].

Moreover, one study suggested that osteochondral autograft transplantation mosaicplasty is superior to bone marrow stimulation by drilling as a concomitant procedure with opening-wedge valgus high tibial osteotomy for SONK of the medial femoral condyle, leading to successful cartilage repair [31].

These procedures may successfully postpone the need for joint arthroplasty. However, evidence for the use of joint-preserving techniques is limited because most studies present an uncontrolled, retrospective design and include small numbers of patients [4].

Unicompartmental knee arthroplasty (UKA) is a well-known treatment for unicompartmental osteoarthritis. In patients who have extensive disease affecting multiple compartments or in those whom more conservative measures have failed, total knee arthroplasty (TKA) may be the only successful alternative. Both UKA and TKA have shown favorable results, comparable to those seen after TKA for osteoarthritis, so long as surgical indications are followed [32].

Secondary (or atraumatic) osteonecrosis

Epidemiology

Secondary osteonecrosis (SON), also called atraumatic ON, is the second most common type in the knee. It usually affects patients younger than 45 years of age and frequently involves multiple lesions in several joints concurrently [2,33].

Etiology and pathogenesis

SON has been associated with numerous risk factors that can be divided into *direct causes*, such as sickle cell disease, caisson disease, Gaucher's disease, myeloproliferative disorders; and *indirect causes*, including alcohol, corticosteroids, tobacco, obesity [2].

Some evidence has shown that alcohol and corticosteroid abuse cause bone marrow adipose cell enlargement, which increases intra-osseous pressure leading to bone ischemia [34]. This theory has been extended to other conditions, such as Gaucher's disease, dysbaric disorders, and some myeloproliferative disorders. Other risk factors, such as tobacco use and sickle cell disease, may cause SON due to their vaso-occlusive effects [35].

Clinical presentation

Patients usually describe a gradual onset of pain over the affected area. The pain is often focused over either the medial or lateral femoral condyle and may also involve the tibial condyle. The pain may occur in other joints, too, because SON frequently involves multiple joints [4].

Diagnosis

As in SONK, radiographs and MRI are main diagnostic tools for SON.

Unlike what is seen in SONK, multiple lesions may be observed in SON and 80% of cases will have bilateral involvement. Furthermore, while SONK lesions are isolated to the epiphysis, SON lesions may be seen in the epiphysis, metaphysis, and/or diaphysis of the femur [2,33].

Additionally, T1-weighted images often show a hypointense serpentine line with a well-defined border and a rim or double halo sign, adjacent to the proximal border of the osteonecrotic bone [17,36,37].

As in SONK, SON of the knee is staged using the Koshino classification and the Modified Ficat and Arlet staging system.

Treatment

Nonsurgical management is recommended for patients who are asymptomatic, and the therapeutic options are similar to those available for SONK treatment. Symptomatic patients will need surgery [2].

There are several joint-preserving methods that have attracted increasing interest in recent years, primarily to delay the need for TKA in a patient group who are often young and active, and are more likely to be immunocompromised secondary to a concurrent disease process. If the cartilage surface is not depressed or arthritic, joint-preserving techniques such as core decompression and bone grafting are available.

Subchondral autologous bone marrow concentrate was shown to be an effective procedure for treating young patients with SON of the knee with a lower complication rate and a quicker recovery as compared with TKA [38].

The authors avoided the use of osteochondral grafts in patients with SON because the lesions usually involve multiple condyles, which do not lend themselves to a single osteochondral graft. Neither is UKA recommended for SON, due to the frequent involvement of multiple condyles [27,28].

Post-arthroscopic osteonecrosis

Epidemiology

Post-arthroscopic ON of the knee is the rarest type of ON. It occurs after these procedures in 2 out of 50 patients (4%) [39]. Most reported cases involve the medial femoral condyle. The lateral femoral condyle is the second most frequently affected site. In rare cases, the lateral tibial plateau, medial tibial plateau, or patella is affected [4].

Etiology and pathogenesis

Most early studies hypothesized that increased bone contact pressure after meniscectomy could cause pathologic fracture of subchondral bone and synovial fluid leakage, and eventually the onset of ON [40].

Another hypothesis is that post-arthroscopic ON is actually subchondral fracture, and not pure ON as traditionally described [41].

Some authors have suggested that thermal energy may directly damage bone tissue or that photoacoustic shock may be the cause of knee ON [42].

Clinical presentation

These patients usually present with sudden onset of knee pain at around 24 weeks after the arthroscopic procedure (range: 4 to 92 weeks) [43].

Diagnosis

Radiographs and MRI evaluation are recommended in patients with suspected post-arthroscopic ON. Bone marrow edema is typically seen in the compartment where the arthroscopic procedure was performed, with no evidence of edema seen prior to the arthroscopy [4].

Treatment

The treatment indications are the same as for other forms of ON and similar surgical procedures are used [4].

Differential diagnosis

A careful history and physical examination can help differentiate between ON and similar clinical entities, including osteochondritis dissecans, osteochondral lesions, bone marrow edema, bone contusion, meniscal lesions, osteoarthritis, tumor, and infection.

An infection or tumor can simulate avascular necrosis, and it is important to consider systemic signs.

Ostheochondritis dissecans (OCD) is often a difficult differential diagnosis with ON of the knee. OCD is an idiopathic, focal anomaly in the formation of the subcondral bone that may cause instability or detachment of a bone fragment and cartilage, with progression towards arthrosis. This condition affects male teenagers, and in 50% of cases is post traumatic. The patients may have mechanical pain during sport or sub-continuous pain with swelling and joint lock. The differences with respect to ON are the age at onset and the correlation with trauma, as well as the site involved, namely the lateral surface of medial condyle.

Conclusion

Osteonecrosis of the knee is an umbrella term covering different entities with a similar clinical presentation and similar imaging findings. Treatment is guided by corroborated classifications. In addition to the long-established techniques, treatment options now include stem cell and osteochondral autograft transplantation therapies, which are aimed at delaying joint replacement, and show promising outcomes.

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Authors' Notes: *No part of this study has been submitted or duplicated elsewhere. This study has been read and approved by all authors and each author believes that the manuscript is valid and represents honest work.*

Declaration of Conflicting Interests: *The authors declare that they have no competing interests.*

Funding sources: *This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.*