The Diagnosis and Management of Spontaneous and Post-Arthroscopy Osteonecrosis of the Knee


Abstract

Spontaneous osteonecrosis of the knee (SPONK) and osteonecrosis in the postoperative knee (ONPK) are two clinical entities that have the potential to cause significant morbidity in affected patients. In addition to the knowledge of the patient population at risk and the classic presentation and imaging characteristics of SPONK and ONPK, the treating orthopaedic surgeon needs to maintain a high index of suspicion for these disorders since early diagnosis and treatment may allow for an improved clinical outcome. The following review presents the current knowledge regarding these two pathological processes of the knee.

Since its first description in 1968 by Ahlback and colleagues,1 spontaneous osteonecrosis of the knee has been recognized as a distinct clinical entity with the potential to cause significant morbidity to affected patients.1,2 In contrast to the osteonecrosis associated with alcohol consumption, corticosteroid use, sickle cell disease, and other described risk factors, spontaneous osteonecrosis of the knee affects a different patient population with a different pattern of bony involvement. More recently, osteonecrosis of the knee following arthroscopic surgery has been described, typically following arthroscopic meniscectomy. Initially reported by Brahme and associates in 1991, post-arthroscopic osteonecrosis has also been noted to occur subsequent to anterior cruciate ligament reconstruction and chondroplasty procedures.4,5

Spontaneous osteonecrosis of the knee (SPONK) and osteonecrosis in the postoperative knee (ONPK) both have the potential to progress to end stage degenerative changes, causing significant symptomatology that may eventually require operative intervention. The following review will present the current knowledge regarding these two entities, describing their clinical and radiographic presentation, hypothesized etiology, and strategies for management.

Spontaneous Osteonecrosis of the Knee (SPONK)

Spontaneous osteonecrosis of the knee is a disorder of uncertain etiology, classically described as a focal lesion occurring in the medial femoral condyle of a patient in their fifth or sixth decade of life, with females affected almost three to five times more than males.1,3,6-10 Patients typically present with the sudden onset of severe pain localized at the medial aspect of the knee just proximal to the joint line. Although a traumatic etiology has been implicated in spontaneous osteonecrosis of the knee, only a minority of patients recall a specific injury that precipitated their symptoms.9 In the acute phase of the disease, patients will often report pain with weightbearing activities and an increase in the severity of their pain at night. Depending on the stage of their lesion and its size, this acute phase pain will either gradually resolve or become chronically debilitating.

Clinical Evaluation

Examination of the affected knee in the acute phase of SPONK, typically the first 6 to 8 weeks following symptom onset, will demonstrate a small to moderate effusion with limitation of range of motion secondary to pain and associated muscle spasm. Palpation will often elicit a localized area of tenderness over the medial femoral condyle just proximal to the joint line in the flexed knee. Although the medial femoral condyle is most commonly affected in
spontaneous osteonecrosis of the knee, lesions involving the medial tibial plateau, the lateral femoral condyle, and rarely the patella have also been reported.2-8-15 Identifying the area of maximal tenderness to palpation can serve as a guide to localizing the involved area. Ligamentous examination of the affected knee is typically normal in cases of SPONK.

**Etiology**

Similar to the proposed mechanisms associated with osteonecrosis of the femoral head, two main etiologies have been suggested in the pathogenesis of spontaneous osteonecrosis of the knee: traumatic and vascular. With the majority of affected patients being elderly females with osteoporotic bone, some investigators believe that SPONK develops as a consequence of microfractures occurring in weak subchondral bone secondary to minor trauma.15 It has been suggested that following an episode of trauma to the knee, fluid enters the intercondylar region filling the potential space created by the subchondral microfractures in the femoral condyle.8,16,17 This fluid increases the intraosseous pressure in the area leading to focal osseous ischemia and eventual necrosis. Recently, researchers have questioned this theory as the mechanism of SPONK. Mears and coworkers performed histopathologic evaluation of specimens taken from 24 patients diagnosed with spontaneous osteonecrosis of the knee and found that only 1 case had evidence of bone necrosis present.18 Seventy-five percent of specimens in this study had demonstrable osteoporosis, implying that osteonecrosis was more of a secondary phenomenon following insufficiency fracture rather than the primary mechanism of the disease. Other investigators have supported the insufficiency stress fracture theory as the etiology of SPONK: believing that when bony necrosis develops, it occurs as a consequence of physiologic resorption and remodeling following fracture.8,9,10,20

Where a vascular etiology continues to be the dominant theory for osteonecrosis of the femoral head, with up to three-quarters of affected patients showing evidence of an underlying thrombophilia or coagulopathy, these predisposing factors have yet to be consistently demonstrated in patients with SPONK.2,21 In the hip, many investigators believe that the presence of a coagulation disorder, including resistance of activated protein C, low tissue plasminogen activator activity, and hypofibrinolysis, causes intraosseous venous occlusion that culminates in the hypoxic death of bone. Evaluation of the coagulation profiles of patients affected with SPONK is necessary to determine whether this mechanism is present in the pathogenesis of the disease.

Recently, the presence of a medial meniscal tear has been proposed as a potential third etiology behind the development of spontaneous osteonecrosis of the knee.22-24 Case series have identified medial meniscal tears in 50% to 78% of patients of patients with SPONK with a recent series by Robertson and colleagues24 noting that tears, specifically in the area of the meniscal root, coexisted with spontaneous osteonecrosis in 24 of their 30 patients (80%). These investigators theorize that in elderly patients with osteoporotic bone, discontinuity of the medial meniscus results in loss of hoop stress distribution in the medial compartment thus increasing the load experienced in the femoral condyle and potentially predisposing patients to the development of subchondral insufficiency fracture.

**Radiographic Evaluation/Staging**

Cases of suspected spontaneous osteonecrosis of the knee should be initially evaluated with a plain x-ray series of the knee including an anteroposterior, lateral, and skyline or Merchant view. Early in the disease process, plain x-rays may fail to identify any abnormalities despite the presence of significant symptomatology. As the condition progresses, plain film findings may include a radiolucent lesion with a surrounding sclerotic halo in addition to subtle flattening of the involved femoral condyle (Fig. 1). In advanced cases with significant subchondral collapse, secondary degenerative changes may be evident with loss of joint space, sclerosis in the medial tibial plateau and osteophyte formation (Fig. 2).

Several staging systems have been described for spontaneous osteonecrosis of the knee based on plain x-ray appearance.2,3,8,25,26 In the four-tiered system described by Koshino and associates, stage I disease is defined as incipient, with patients reporting pain with activity; however; plain x-rays are negative for pathology. In stage II SPONK, or the avascular stage, a round to oval subchondral lucency in the weightbearing area is present with associated increased density in the surrounding femoral condyle. During the collapse or developed stage of disease (stage III), x-rays demonstrate a sclerotic halo bordering the radiolucent lesion. Subchondral collapse heralds stage III SPONK. Further subchondral collapse with associated development of arthritic changes in the affected compartment define stage IV disease.26

Aglietti and coworkers25 modified the Koshino staging,

**Figure 1** Anteroposterior radiographs of early stage spontaneous osteonecrosis (SPONK) affecting the right medial femoral condyle developing in a 67-year-old male.
system to include five stages of disease. In stage I the x-rays are normal in appearance. Subtle flattening of the affected femoral condyle characterizes stage II SPONK, which indicates the potential for subsequent collapse. Stage III describes the characteristic radiolucent lesion with a circumferential sclerotic border, and stage IV disease is heralded by an increase in the size of the sclerotic halo as the subchondral bone begins to collapse. Stage V SPONK includes continued subchondral collapse with the development of associated secondary degenerative changes.

Prognostic implications can be made based on the plain x-ray appearance of the lesion, primarily based on its size. The width of the lesion can be measured on the anteroposterior view, with those measuring less than 1 cm in size classified as small, and those greater than 1 cm classified as large. In many of the early studies of SPONK, the area of the lesion within the condyle was used to predict which cases would progress to severe degenerative arthritis. Cases where the lesion was less than 2.5 cm² were unlikely to progress, whereas those with an area of greater than 5 cm² were considered to have a poor prognosis. Another useful plain x-ray measure is the ratio of the width of the lesion compared to the overall width of the femoral condyle on the anteroposterior view. This measure is not impacted by differences in magnification of the view and has been shown to correlate with prognosis. Studies have demonstrated that good outcomes were common in lesions with a size ratio of less than 0.45, while those greater than 0.5 typically progress to severe degenerative arthritis.

In both the hip and the knee, magnetic resonance imaging (MRI) has become the standard imaging modality for the detection of osteonecrosis. Magnetic resonance is both sensitive and specific in the evaluation of SPONK, often demonstrating more extensive involvement than was evident on plain radiography. T1 imaging in cases of spontaneous osteonecrosis of the knee shows a discrete low signal area often surrounded by an area of intermediate signal intensity. A serpiginous low signal line is often present at the margin of the lesion, delineating the necrotic area from the adjacent area of bone marrow edema. T2 images will typically show a high signal intensity at lesion edge in the region of the bone marrow edema. Some investigators suggest using gadolinium enhanced MRI in the evaluation of cases of SPONK. The addition of gadolinium is believed to provide information on the extent of osseous activity and turnover at the edges of the lesion, with enhanced adjacent changes.
activity believed to be a positive prognostic sign indicative of healing potential.3

**Clinical Course**

The course and prognosis of patients with spontaneous osteonecrosis of the knee is dependent on the size and stage of the lesion. The majority of patients present with a similar history and physical examination. Pain is often severe at symptom onset, present with weightbearing with a typical increase in severity at night, and has a significant impact on the patients daily activities. The intense pain associated with the acute phase of SPONK may last up to 6 weeks at which point the extent of the patients’ symptoms divides them into two main groups.3 Those who will end up with a satisfactory outcome will typically report improvement in their pain and intermittent swelling after the 6 week time point, although mild symptoms with activity may continue for up to 12 to 18 months. These patients most commonly have smaller lesions evident on imaging studies, with lesions usually less than 40% of the width of the involved femoral condyle. Despite significant improvement in their symptoms and the ability to resume normal daily activities, the vast majority of patients with SPONK will eventually develop osteoarthritic changes in the involved compartment. Insall and colleagues reported that at 2 years of follow-up, almost all patients with osteonecrosis of the knee had evidence of at least grade I osteoarthritis with joint space narrowing.33 Patients whose symptoms fail to improve after 6 weeks tend to follow a more relentless and progressive disease course. They are more typically those with large lesions encompassing greater than 50% of the width of their femoral condyle. These patients in the poor prognosis group often never report improvement in their knee function or extent of pain. Serial imaging will often demonstrate a rapid progression with collapse and the subsequent development of degenerative changes in the affected compartment.

**Post-Arthroscopy Osteonecrosis of the Knee**

First reported by Brahme and associates4 in a series of 7 patients who developed radiographic evidence of osteonecrosis following the arthroscopic treatment of meniscal pathology, osteonecrosis in the postoperative knee (ONPK) has been recognized to be a rare, potential complication of arthroscopic surgery.4,6,8 Many of the early case reports describing this entity found it subsequent to arthroscopic meniscectomy, leading it to be referred to as post-meniscectomy osteonecrosis of the knee. However, more recently, osteonecrosis lesions have been noted to occur following other arthroscopic procedures including chondroplasty and anterior cruciate ligament reconstruction.4,8,34

Considering the large number of arthroscopic proce-

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**Figure 3**

A, Pre-operative MRI demonstrating medial meniscal tear with no evidence of pathology affecting the medial femoral condyle. B and C, Intra-operative arthroscopic images demonstrating radial tear of the posterior horn of the medial meniscus. D and E, Postoperative MRI demonstrating changes within the medial femoral condyle indicative of osteonecrosis in the postoperative knee (ONPK).
tures performed annually and the relatively few reports of cases of post‐arthroscopy osteonecrosis, the prevalence of ONPK is very low. At the present time, ONPK following arthroscopic meniscectomy has been described in 9 clinical studies including a total of 47 patients. In all 47 cases, postoperative MRI demonstrated evidence of osteonecrosis that was not present in preoperative imaging studies (Fig. 3). In contrast to the patient population typically affected by spontaneous osteonecrosis of the knee (SPONK), ONPK tends to affect younger patients (mean: 58 years; range: 21 to 82 years) with an equal gender distribution (23 females and 24 males).

Lesions of ONPK predominantly affect the medial femoral condyle (39 cases, 82%), followed by the lateral femoral condyle (4 cases), lateral tibial plateau (2 cases), and the medial tibial plateau (1 case). In each of the reported cases, osteonecrosis developed in the geographic location of the patient’s pathology and arthroscopic procedure, with none arising in the contralateral compartment postoperatively. Concomitant chondral lesions in the region of the meniscal tear were reported to exist in 65% of patients who went on to ONPK, with chondromalacia of the medial compartment noted to exist in 33 of the 47 published cases.

In patients who develop osteonecrosis of the knee following arthroscopic surgery, symptoms of pain, swelling, and limited range of motion may persist or even worsen postoperatively despite the fact that an adequate resection of the meniscal tear was performed. Cases of persistent or worsening symptoms after knee arthroscopy need to be considered for the possibility of an evolving osteonecrosis lesion; a diagnosis that needs to be distinguished from SPONK, bone marrow edema syndrome, and recurrent meniscal tear.

Clinical Evaluation
Patients with ONPK typically report continued or increased pain in the medial aspect of their knee postoperatively. Examination of the affected knee will often demonstrate a small to moderate effusion with limitation of range of motion secondary to pain and associated muscle spasm, similar to what was seen preoperatively. Palpation will elicit localized tenderness over the medial joint line and medial femoral condyle. Ligamentous exam will often be normal.

Etiology
At the present time, the exact etiology of osteonecrosis in the postoperative knee has yet to be fully elucidated. Similar to the previously described correlation of meniscal tears with the development of spontaneous osteonecrosis of the knee, some investigators believe that altered knee biomechanics following meniscectomy are responsible for the pathogenesis of the disease. Previous studies have shown that approximately 50% of joint compressive forces are transmitted through the meniscus in extension and up to 85% of the load in 90° of knee flexion. Partial meniscectomy increases tibiofemoral contact pressures in the treated compartment, potentially leading to subchondral insufficiency fractures from altered load transmission. Histopathologic evaluation of specimens from cases of ONPK have supported this theory, demonstrating evidence of subchondral insufficiency fractures with bony necrosis present distal to the fracture site. A corollary to the insufficiency fracture theory of ONPK is the possibility that overly aggressive postoperative rehabilitation contributes to the development of this condition. In an attempt to restore function, rapid resumption of weightbearing activities and exercise are often started within days of the operative procedure. It is possible that if aggressive therapy is resumed prior to bony remodeling in response to the altered load distribution that occurs post‐meniscectomy, insufficiency fractures may develop.

Others hypothesize that the pathologic articular cartilage in the affected compartment has increased permeability to arthroscopic fluid. This increase in fluid permeability may also occur following the instrumentation of the articular surface, during shaving chondroplasty, or with inadvertent contact of arthroscopic instruments with the femoral condyle during meniscectomy. Influx of arthroscopy fluid may cause subchondral edema and subsequent osteonecrosis from increased intraosseous pressure. Localized osteoarticular injury from the use of a laser or radiofrequency probe during the arthroscopic procedure has been described as a third potential cause of ONPK. It has been proposed that direct thermal injury or injury from photoacoustic shock from these instruments induces an inflammatory response leading to bony edema, increased local intraosseous pressure, and eventual osteonecrosis.

Radiographic Evaluation/Staging
In the early stages of osteonecrosis in the postoperative knee, plain x-rays are of limited value in the initial workup because the disease primarily involves the bone marrow. While a bone scan will often be positive in cases of ONPK, with a high level of sensitivity for changes in local osseous vascularity, its specificity and spatial resolution is poor. The diagnosis of osteonecrosis in the postoperative knee is dependent on an MRI of the affected joint, with two specific criteria that need to be filled: 1. the absence of osteonecrosis on preoperative MRI performed 4 to 6 weeks after the onset of symptoms and 2. a time association between the arthroscopic procedure and the development of a suspicious bone marrow edema pattern on postoperative MRI.

In order to distinguish cases of ONPK from those of SPONK, the preoperative MRI must be normal with respect to the condition of the bone and bone marrow of the femoral condyle and tibial plateau. However, it is important to acknowledge that in the very early stages of spontaneous osteonecrosis of the knee, MRI of the affected knee may be devoid of findings—described as the “window period” of SPONK, between symptom onset and MRI evidence of signal changes. Most investigators report using a period of
4 to 6 weeks following the development of symptoms as sufficient time for radiographic evidence of SPONK to be present.4,34 This is largely based on an animal study by Nakamura and coworkers,41 in which MRI changes developed in all specimens by 4 weeks following surgically induced femoral head osteonecrosis. Distinction between SPONK and ONPK may not be possible with imaging studies performed prior to 4 to 6 weeks.

A temporal association between the arthroscopic procedure and postoperative MRI signal changes must be present for the diagnosis of ONPK to be made. In the nine clinical studies reporting cases of osteonecrosis in the postoperative knee, the mean time between arthroscopy and MRI establishing the diagnosis of ONPK was 18 weeks (range: 3 to 176 weeks).4 This criterion is more difficult to assess and qualify, as bone marrow edema commonly occurs following arthroscopic knee procedures. In a study of 93 patients with a mean age of 36.6 years undergoing arthroscopic meniscectomy, Kobayashi and colleagues44 found that 34% had MRI evidence of bone marrow edema in the operative compartment within 8 months of their procedure. Although it may be related to the age of the patients in Kobayashi’s study, none progressed to ONPK.

Magnetic resonance images obtained in the early stages of ONPK will demonstrate a non-specific, large area of bone marrow edema in the femoral condyle, ipsilateral to the prior meniscectomy with heterogeneous signal present on T2 imaging. By 3 months postoperatively, the extent of edema typically decreases and MRI findings in cases of ONPK are similar to those seen in cases of SPONK with T1 imaging showing a discrete low signal area surrounded by an area of intermediate signal intensity. A line of low signal is often present at the margin of the lesion, delineating the necrotic area from the adjacent area of bone marrow edema. T2 images will typically show a high signal intensity at lesion edge in the region of the bone marrow edema. As the lesion progresses to its final stages, bone sequestration may be present with a surrounding high signal rim along with condylar flattening and the possibility of loose body development.4,45

**Clinical Course**

Review of the 47 reported cases of ONPK shows that 93.6% (44/47 cases) had permanent lesions evident on MRI or post-arthroscopy osteonecrosis include a younger patient age, primary involvement of the lateral compartment of the knee, as well as disease affecting multiple joints. Areas commonly affected by secondary osteonecrosis include the femoral head, knee, and proximal humerus.14 Additionally, approximately 60% of secondary osteonecrosis cases have the contralateral joint affected, an uncommon finding in cases of SPONK or ONPK.

**Pathology Findings**

Osteonecrosis is defined by the death of a portion of the weightbearing area of the joint with associated fracture and collapse of the subchondral bone. Regardless of the etiology, all forms of osteonecrosis result from ischemia and share the same pathologic features. Early in the disease process, the overlying articular cartilage is usually intact, as it is nourished and kept viable by the synovial fluid of the joint. As the disease progresses, continued necrosis of the marrow contents leads to osteocyte death, with empty lacunae evident on microscopic evaluation. Breakdown products from the necrotic osteocytes provoke an inflammatory response that represents the initiation of the body’s attempt at repair of the infarcted area.

During the healing response, osteoclasts resorb the necrotic trabeculae creating space for an ingress of vascularized fibrous tissue. In the remaining trabeculae, creeping substitution occurs where the necrotic trabeculae serve as a scaffold for the deposition of new bone by osteoblasts. The process of creeping substitution and new bone formation fails to keep pace with bone resorption, resulting in loss of subchondral bony support.19 Continued weightbearing loads lead to subchondral fracture and collapse with eventual disruption of the overlying cartilage, heralded as the crescent sign on radiographs. Abnormal loading secondary to a flattened, incongruous articular surface leads to the development of degenerative joint disease and the pathologic changes characteristic of osteoarthritis.

**Comparison to Secondary Osteonecrosis of the Knee**

In the evaluation of patients with suspected SPONK or ONPK, the treating orthopaedic surgeon must be also be aware of and consider other potential causes of osteonecrosis affecting the knee. Clinical findings that are more consistent with secondary osteonecrosis than spontaneous or post-arthroscopy osteonecrosis include a younger patient age, primary involvement of the lateral compartment of the knee, as well as disease affecting multiple joints. Areas commonly affected by secondary osteonecrosis include the femoral head, knee, and proximal humerus.14 Additionally, approximately 60% of secondary osteonecrosis cases have the contralateral joint affected, an uncommon finding in cases of SPONK or ONPK.

There are many secondary causes of osteonecrosis of the knee, as well as a few established pathways leading to this outcome, but the unifying theme among all of them is ischemia to the affected bone. Some of the pathways leading
to ischemia include thrombotic or embolic events, increased intra-osseous pressure, vessel injury, and venous hypertension. The most common causes of secondary osteonecrosis are chronic alcohol consumption and high dose corticosteroid use (Table 1).

### Table 1  Secondary Causes of Osteonecrosis

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<td>Alcoholism</td>
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<td>Chemotherapy</td>
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<td>Sickle Cell Disease (and other hemoglobinopathies)</td>
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<td>Smoking</td>
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<td>Systemic Lupus Erythematosus (and other connective tissue disorders)</td>
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<td>Tumors</td>
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**Treatment Options for SPONK and ONPK**

Various treatment options are available for the management of cases of spontaneous osteonecrosis of the knee and osteonecrosis in the postoperative knee. These range from non-operative and pharmacologic treatment to joint-preserving operative procedures and joint arthroplasty. As these diseases are relatively rare, a validated treatment algorithm has yet to be developed, and management is taken on a case-individualized basis.

**Non-Operative Treatment/Pharmacologic Therapy**

Once a lesion is identified, an attempt at nonoperative management is often undertaken. Protected weightbearing with crutches coupled with analgesics and anti-inflammatory medication is the mainstay of nonoperative treatment. Typically, the restrictions on weightbearing are maintained for a 4 to 8 week period. As the patient’s symptoms improve, a resumption of normal activities of daily living in addition to the use of physical therapy for quadriceps and hamstring strengthening is allowed.

In cases of SPONK, good to excellent results have been reported following non-operative management if the lesion size is small (less than 40% of the width of the femoral condyle). In a series of 79 cases of medial femoral condyle SPONK, Lotke and colleagues reported that 32 of 36 (88.9%) patients with stage I disease had resolution of their symptoms after a period of protected weightbearing and analgesic treatment. In Lotke’s series, only 1 patient with stage I disease went on to require total knee arthroplasty. Similar good results of non-operative treatment were reported by Yates and coworkers in their series of 20 cases of stage I SPONK. Resolution of the lesion was evident on follow up MRI in 19 of the 20 patients at a mean of 8 months (range: 3 to 18 months). As the lesion size and stage increases, the success of non-operative treatment for cases of SPONK becomes less reliable with most investigators reporting a slow, relentless progression to degenerative arthritis.

### Arthroscopic Debridement

The use of arthroscopic debridement in the management of SPONK and ONPK has limited applications. As the primary pathology is intraosseous, arthroscopic debridement has little likelihood of altering the course of the disease process; however, it may lead to symptomatic improvement in cases where mechanical symptoms are present secondary to unstable chondral fragments or loose bodies. In a series of 5 cases of SPONK treated with arthroscopic debridement and chondroplasty, Miller and associates reported
good postoperative outcomes in 4 cases at a mean follow up of 31 months with HSS knee scores improving from 52 to 82.55 However, the natural history and progression of the osteonecrosis lesions in these patients was not altered by the arthroscopic procedure. Some investigators have reported performing arthroscopic retrograde drilling for cases of SPONK or ONPK.56 While retrograde drilling may stimulate revascularization within the lesion, the potential for damage to the intact articular surface and the difficulty associated with accurately localizing the focus of the lesion in the precollapse stage makes antegrade drilling or core decompression a more attractive treatment option.

**Core Decompression**

Relief of elevated intraosseous pressure via extra-articular drilling has been used frequently in early stage, precollapse cases of osteonecrosis of the femoral head with variable results. Core decompression as a treatment for osteonecrosis of the knee was first described in 1989 by Jacobs and coworkers in their series of 28 patients.57 These investigators reported good results in their stage I and II cases (7 patients) and in 52% of their stage III cases. More recently, in a series of 16 patients with a mean age of 64 years, Forst and coworkers found that core decompression provided symptom relief and successful healing (normalization of bone marrow signal on MRI) in 15 stage I cases and 1 stage II case of SPONK at 3 year of follow-up.3,58 Based on their findings, these investigators recommended core decompression as a useful treatment option for early stage osteonecrosis of the knee (Fig. 4). However, it is important to note that these reports lacked control groups, with the possibility that these cases of early stage disease may have improved without intervention.

![Figure 4 A-C](image1) Pre-operative coronal, sagittal, and axial MRI cuts of SPONK affecting the lateral femoral condyle. **D-H**, Arthroscopic assisted, fluoroscopic-guided core decompression of the lateral femoral condyle.
**High Tibial Osteotomy**

Appropriately selected patients with SPONK or ONPK may be managed with a high tibial osteotomy as a joint-preserving treatment option. Typically reserved for younger, active patients, high tibial osteotomy can function to offload the affected femoral condyle by shifting the weightbearing axis laterally. In their series of 105 cases of SPONK diagnosed and treated over a 20-year period, Soucacos and colleagues reported using high tibial osteotomy as an effective treatment for patients with stage III disease. Although no details regarding the technique or patient outcome were described, the investigators reported better results with patients younger than 65 and for lesions less than 50% of the width of the femoral condyle. Koshino and associates, in a study of 37 cases of SPONK managed with high tibial osteotomy with or without a concomitant drilling and bone grafting procedure found that the outcomes were best when the combined procedures were performed and the mechanical axis was corrected to at least 10° of valgus alignment. Follow-up radiographic evaluation in this series demonstrated that the lesion improved in 17 patients and resolved completely in 13 patients. In a study including 10 patients with SPONK managed with either high tibial osteotomy (6 patients) or non-operative treatment (4 patients), Marti and colleagues found that patients treated with HTO had a higher incidence of improvement in the appearance of their lesion on follow up MRI (83% vs 25%) and a higher incidence of symptom improvement (100% vs 50%). Johnson and associates reported 2 cases of ONPK treated with high tibial osteotomy, 1 case was performed 8 months and the other 10 months after the index arthroscopic medial meniscectomy; however, clinical outcome and follow up were not described.

**Knee Arthroplasty**

For patients in whom joint-preserving treatments fail to provide symptomatic improvement and in those with large or advanced lesions, knee arthroplasty is the treatment of choice. Depending on patient factors, lesion characteristics and the condition of the remainder of the joint, unicompartmental arthroplasty or standard total knee arthroplasty may be utilized. Unicompartmental arthroplasty is an effective treatment method for those with disease isolated to a single femoral condyle or tibial plateau, with the benefit of preserving the patient’s bone stock and functioning cruciate ligaments. Extensive cases of SPONK or ONPK occurring in patients with evidence of degenerative change in the contralateral compartment or patellofemoral joint are better managed with tricompartmental replacement.

Bonutti and coworkers reported the outcome of arthroplasty in 19 patients with osteonecrosis in the postoperative knee, with 4 patients undergoing UKA and 15 undergoing TKA. At a mean follow-up of 62 months, 18 patients had a good or excellent outcome based on Knee Society Score (mean score: 92; range: 60 to 100). Data on total knee arthroplasty in cases of osteonecrosis of the knee demonstrate good outcomes; however, in the long term, they do not appear to function as well as TKA performed for cases of osteoarthritis. In a series of 32 knees with SPONK of the medial femoral condyle, Ritter and colleagues reported results inferior to those seen in a comparison group treated for osteoarthritis. Patients treated for SPONK had worse pain relief (82% vs 90%) and a higher incidence of revision surgery (17% vs 0%) compared to the OA patients. Similar results were reported by Bergman and Rand in their series of 36 cases of osteonecrosis of the knee managed with total knee arthroplasty. Good to excellent results were reported to occur in 87% of the study patients at a mean of 4 years of follow-up. While implant survivorship at 5 years was predicted to be 85% with revision surgery defined as the end-point, when moderate or severe pain was used as the end-point, survivorship was predicted to be 68%. These investigators theorized that if foci of osteonecrosis exist in the supporting subchondral or metaphyseal bone, persistent pain may complicate the outcome of total knee arthroplasty. Other studies have shown better outcomes of TKA for cases of SPONK. In a series of 32 total knee arthroplasties performed in 30 patients, of which 8 were done as treatment of SPONK, Mont and associates reported excellent results with a Knee Society Score of 98 at a mean of 108 months postoperatively.

**Conclusion**

Spontaneous osteonecrosis of the knee and osteonecrosis in the postoperative knee are two clinical entities that have the potential to cause significant morbidity in affected patients. In addition to the knowledge of the patient population at risk, the classic presentation and imaging characteristics of SPONK and ONPK, the treating orthopaedic surgeon needs to maintain a high index of suspicion for these disorders because early diagnosis and treatment may allow for an improved clinical outcome. Continued study of patients with SPONK and ONPK is needed in an effort to identify specific risk factors that predispose certain patients to their development. In the future, it is possible that pharmacologic intervention and alterations in post-arthroscopy rehabilitation protocols in susceptible patients may alter the course of disease for those who develop SPONK and ONPK.

**Disclosure Statement**

None of the authors have a financial or proprietary interest in the subject matter or materials discussed, including, but not limited to, employment, consultancies, stock ownership, honoraria, and paid expert testimony.

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