



45 Y old woman had a trivial work injury 2 wks ago. Could continue to work and worked for 3 years. After 3 wks: increasing pain in the knee and limp.

**Diagnosis?**

Diagnosis: SPONK [spontaneous osteonecrosis knee]

### **Abstract**

Spontaneous osteonecrosis of the knee (SPONK) and osteonecrosis in the post-operative knee (ONPK) are two clinical entities that have the potential to cause significant morbidity in affected patients. Needs to maintain a high index of suspicion for these disorders since early diagnosis and treatment. Secondary osteonecrosis associated with alcohol consumption, corticosteroid use, sickle cell disease, and other described risk factors.

### **Spontaneous Osteonecrosis of the Knee (SPONK)**

A disorder of uncertain etiology classically described as a focal lesion occurring in the medial femoral condyle in V/VI decade. Females affected almost three to five times more than males. Sudden onset of severe pain localized at the medial aspect of the knee. 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. Acute phase pain will either gradually resolve or become chronically debilitating.

### **Clinical Evaluation**

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.

### **Etiology**

? traumatic and vascular. As a consequence of micro fractures occurs in weak subchondral bone secondary to minor trauma. 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 micro fractures in the femoral condyle. 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 histopathology 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. 75% 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.

Where a vascular etiology continues to be the dominant theory: 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 and arthroscopic surgery has been proposed as a potential third etiology behind the development of spontaneous osteonecrosis of the knee [6,7,8]. It has been suggested that increasing the load experienced in the femoral condyle and predispose patients to the develop ONPK.

### **Radiographic Evaluation/Staging**

X ray : AP, lateral, and skyline or Merchant view.

Halo in addition to subtle flattening of the involved femoral condyle.



2. CT Scan

3. **MRI:** All the cases of post arthroscopy osteonecrosis were associated with chondral and meniscal, mainly medial meniscal pathology.

MRI, may be facing arthroscopic surgery with an undiagnosed early onset spontaneous osteonecrosis of the knee. In the MRI examination that was performed subsequently, edema was revealed as well as a minute fracture in the joint surface which exactly corresponds to the painful area

### **Prognosis**

1. On AP view, < 1 cm is better
2. Greater than 5 cm were considered to have a poor prognosis.
3. A serpiginous low signal line, delineating the necrotic area from the adjacent area of bone marrow edema.

It has been suggested using gadolinium enhanced MRI suggest the extent of osseous activity and turnover at the edges of the lesion indicative of healing potential.

### **Clinical Course**

A typical increases in severity at night, and has a significant impact on the patient's 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. 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.

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.

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**

Arthroscopic meniscectomy, leading to ON 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. At the present time, ONPK following arthroscopic meniscectomy has been described in 9 clinical studies including a total of 47 patients [4].

ONPK tends to affect younger patients (mean: 58 years) An equal gender distribution (23 females and 24 males). Lesions of ONPK predominantly affect the medial femoral condyle 82%. Concomitant chondral lesions in the region of the meniscal tear were reported to exist in 65% of patients who went on to ONPK.

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**

At the present time, the exact etiology of osteonecrosis in the postoperative knee has yet to be fully elucidated. Altered knee biomechanics following meniscectomy: Demonstrating evidence of subchondral insufficiency fractures with bony necrosis present distal to the fracture site.

2. 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.

### **Radiographic Evaluation/Staging**

1. The absence of osteonecrosis on preoperative MRI  
MRI performed 4 to 6 weeks post operative showing lesion.
2. A time association between the arthroscopic procedure and the development of a suspicious bone marrow edema pattern on postoperative MRI. "window period" of SPONK, between symptom onset and MRI evidence of signal changes.

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.

In contrast to the correlation of clinical course and prognosis with the size of the lesion in SPONK, this correlation has been less reliable in cases of ONPK.

### **Pathology Findings**

Pathology is same as SPONK. On continued weight bearing loads lead to subchondral fracture and collapse with eventual disruption of the overlying cartilage [the crescent sign]. Abnormal loading secondary to a flattened, incongruous articular surface leads to the development of degenerative joint disease.

### **Treatment Options for SPONK and ONPK**

1. Protected weight bearing with crutches
2. Coupled with analgesics and anti-inflammatory medication

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). 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.

Alendronate has shown efficacy in relieving pain and reducing the incidence of collapse in cases of femoral head osteonecrosis.

### **Arthroscopic Debridement**

Debridement in the management of SPONK and ONPK has limited applications.

While retrograde drilling may stimulate revascularization within the lesion: usually does not work.

### **Core Decompression**

Some investigators recommended core decompression as a useful treatment option for early stage osteonecrosis of the knee. However, it is important to note that these reports lacked control groups.

### **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 weight bearing axis laterally. 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.

Studies have shown better outcomes of TKA for cases of SPONK. In a series of 32 total knee arthroplasty performed in 30 patients, of which 8 were done as treatment of SPONK

### **AN UNSOLVED DILEMMA**

Arthroscopic debridement of a torn degenerative meniscus in an elderly arthritic patient may not only be of no benefit, but may make the patient much worse. The clinical combination of early onset osteonecrosis and an adjacent torn meniscus remains an unsolved dilemma for the arthroscopic surgeon, and poses many questions:

Is osteonecrosis a predictable and preventable outcome of arthroscopic debridement of a torn degenerative meniscus in an arthritic knee? Is the osteonecrosis the main and pre-existing pathology, and the torn meniscus an incidental finding? Is the torn meniscus the main pathology, and the osteonecrosis a consequence of weight bearing on the torn meniscus?

Although spontaneous onset osteonecrosis and post arthroscopic osteonecrosis appear to have many clinical similarities, review of the published cases showed some significant differences. Both conditions have similar symptoms, signs and imaging studies.

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### Comparison of Clinical Presentation and Etiology in Knee Osteonecrosis

Characteristic	Secondary ON	Spontaneous ON of the Knee	Postarthroscopic ON
Age	Typically <45 years	≥50 years	Any
Sex	More likely in men than women	Female-to-male ratio of 3:1	No predilection
Bilaterality	>80%	<5%	Never
Other joint involvement	>90% (hip, shoulder, ankle)	No	No
Associated risk factors	Direct causes: trauma, caisson disease, chemotherapy, Gaucher disease, radiation. Indirect causes: alcohol abuse, coagulation abnormalities (thrombophilia, hypofibrinolysis), corticosteroid use, inflammatory bowel disease, organ transplant, systemic lupus erythematosus, smoking.	Idiopathic, chronic mechanical stress, or microtrauma	Meniscectomy, cartilage débridement, anterior cruciate ligament reconstruction, laser or radiofrequency-assisted surgery
Proposed pathogenic mechanisms	Direct cell injury Restriction or occlusion of blood supply Increased intraosseous pressure	Weight-bearing articular surface subjected to altered stresses as the result of subchondral fracture Vascular compromise to subchondral bone, resulting in osseous ischemia and subsequent edema Osteoarthritis variant	Abnormal loading leading to chondral injury, inflammation, edema, and intraosseous pressure Abnormal loading leading to microfracture and abnormal blood circulation Direct thermal or photoacoustic injury via laser or radiofrequency-assisted arthroscopy
Pathologic findings	Necrotic bone	Fibrotic bone, healing fracture, osteopenia, osteoarthritis, necrosis found only at the distal end of the fractured segment	Fibrotic bone and healing fracture. Necrotic bone after direct thermal or acoustic injury.