"AVASCULAR NECROSIS OF THE KNEE: A Comprehensive Review", Dr. Harpal S. Khanuja, MD

JOHNS HOPKINS MEDICINE

EDUCATIONAL OBJECTIVES

Osteonecrosis of the knee is a rare disorder. The purpose of this exhibit is to highlight our current understanding of osteonecrosis about the knee. After viewing this exhibit the participant should be able to:

1. Differentiate between secondary osteonecrosis and spontaneous osteonecrosis of the knee.
2. Describe the associated radiographic and MRI findings.
3. Understand various treatment options and their outcomes.

INTRODUCTION

The knee is the second most common joint affected by osteonecrosis (ON). Ten percent of patients with osteonecrosis of the hip will have knee joint involvement. Two entities are commonly referred to as "osteonecrosis", but they are two distinct diseases. Spontaneous osteonecrosis of the knee (SPONK) is likely related to an insufficiency fracture or a vascular insult. Atraumatic ON is sometimes referred to as secondary osteonecrosis. It is most commonly steroid induced, but is seen with other disorders. The exact incidence of these two disorders is not known, but atraumatic ON appears to be more common.

Little is known about the pathophysiology of either process. Histologically both disorders demonstrate areas of necrotic bone. The two can be distinguished by history and imaging studies. The treatment options depend upon the stage and size of the lesion. A comparison of the two disorders is provided in order to aid the clinician in identifying the disease and choosing treatment.
The following information is color coded Grey boxes refer to SPONK and Yellow boxes refer to secondary ON.

SPONK

Clinical Presentation

•Typically occurs in patients greater than 55 years old.

•Women are more commonly affected than men (3:1).

•Acute onset knee pain over condyle or tibia depending on location of the lesion. May become chronic pain.

•May have tenderness over condyle or posterior tibia. Not tender over joint line.

•A small effusion may be present.

•May have restricted motion due to pain.

Risk Factors

•No defined risk factors as for secondary osteonecrosis.

•Possible theories include vascular injury or antecedent trauma.

Secondary ON

Clinical Presentation

•Typically occurs in patients less than 55 years old.

•Also more common in females. 3:1

•Individuals complain of gradual onset of knee pain over one or both condyles.

•The knee exam is usually unremarkable.

Risk Factors

•Associated with systemic corticosteroid use.

•Seen with systemic lupus erythematous, alcoholism and blood dyscrasias.

•Less common risk factors include Caisson's Disease, Gaucher's Disease and trauma.

SPONK

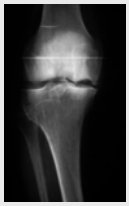
Location of Lesions

•Epiphyseal lesions.

•Medial femoral condyle is most commonly involved.

•Lesions may also appear in tibial plateau.

•No association with contralateral knee or hip involvement.



Radiograph and MRI showing typical femoral lesion of SPONK.

Etiology

Exact etiology remains unclear. May not be true osteonecrosis.

Of 24 pathologic specimens after TKA for SPONK, 23 had no evidence of necrosis. Osteopenia seen in 75% and Osteoarthritis seen in 70% of pathologic specimens.

SPONK may be precursor to osteoarthritis.

Traumatic or vascular injury is believed to be responsible for SPONK.

Vascular injury

Vascular insult may lead to edema and bone ischemia in a localized region. This may eventually progress to cell death and progression of the lesion.

Disorders of coagulation have not been demonstrated in patients with SPONK.

Traumatic Injury

Trauma may occur to the subchondral plate, particularly in osteoporotic bone. Fluid from the joint can then enter the marrow space leading to increased edema and eventual necrosis.

A similar phenomenon is proposed with osteonecrosis that develops after arthroscopy. The thermal effects of laser may also play a role.

Secondary ON

Location of Lesions

•There is greater area of involvement compared to SPONK . Often usually both condyles, extends from diaphysis through metaphysis and into epiphysis.

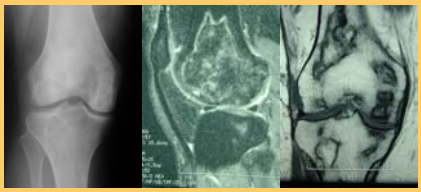
•Secondary ON predominantly affects the Femoral Condyles.

•The tibial plateau can be involved 20-30% of the time.

•80% of patients have bilateral knee involvement.

•90% of patients have hip involvement.

•10% of patients with secondary ON of the hip will have knee involvement.



Radiograph and MRI showing secondary osteonecrosis in patient on corticsteroid treatment for asthma. Note the extensive metaphyseal involvement compared to SPONK.

Etiology

Exact etiology remains unclear. Much of the understanding comes from ON of the Hip. Likely multifactorial.

Steroids may be involved in a variety of ways:

1. They may be directly cytotoxic.
2. They have been shown to increase intracellular fat.
   - Intracellular fat may be cytotoxic and cause increased bone marrow pressure. Increased pressure may restrict blood flow.

Other Possible Factors Include the Following:

Coagulation disorders.

More than 70 % of patients of ON have some coagulation abnormality that predispose to clot formation or slows clot dissolution

Cellular Hypertrophy

Enlarged cells in marrow may cause increased pressure.

Disorders resulting in cellular hypertrophy are associated with ON include: Gaucher's Dz, Leukemia, and Caisson's Dz

Lipid Emboli

Lipid particles in microvasculature cause ischemia.

Hyperlipidemia is seen with corticosteroids, gout and alcohol.

Immune complex deposition

In autoimmune disorders this may be responsible for vascular injury predisposing to ON.

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SPONK

Pathology

•Necrosis of bone occurs, initially. The overlying cartilage is spared.

•Necrotic bone becomes replaced with fibrous tissue.

•With advanced stages the cartilage may flatten.

•A cartilage defect forms. Secondary arthritic changes then develop.

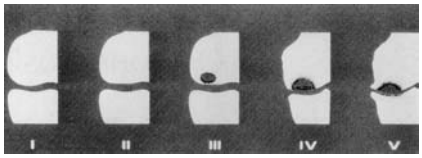
•A central area of necrotic bone is surrounded by reparative bone and bands of fibrovascular granulation tissue.

Plain Radiographs

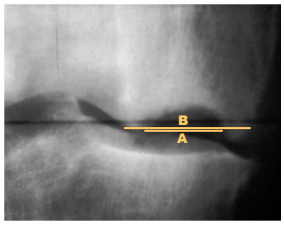
Early stage is negative.

Staging: 5 radiographic stages for SPONK of the distal femur

Stage I: normal radiographs
Stage II: flattening of condyle seen
Stage III: subchondral radiolucency surrounded by sclerotic halo
Stage IV: sclerotic halo thickens with collapse of bone
Stage V: arthritic changes



Radiograph stages of SPONK, reprinted with permission, Osteonecrosis of the Knee in Surgery of the Knee, 3rd edition, ed Insall and Scott 2001



Stage III lesions can be quantified by the ratio of the width of the lesion, line a, to the width of the condyle, line b. Knees with a ratio of less than 0.45 have a better prognosis.

MRI

T1: low signal surrounded by intermediate signal
T2: area of low signal surrounded by high signal intensity
Has been shown to miss SPONK identified with bone scan

Bone Scan

Must be positive for diagnosis.

Static phase imaging shows increased focal uptake over lesion.

Lesions on both sides of joint more indicative of osteoarthritis.

Secondary ON

Pathology

•Large area of necrosis. Centrally avascular, but peripherally good vascular supply.

•Necrotic bone becomes replaced with fibrous tissue. Osteoblasts attempt to lay new bone over this. Small lesions may heal.

•Subchondral bone fails leading to altered stress on overlying articular cartilage.

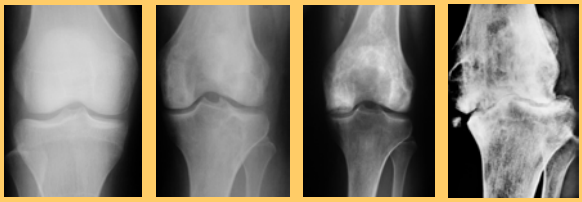
•Eventually chondrocyte death occurs and secondary degenerative changes ensue.

Plain Radiographs

Early stage is negative.

Staging: Based on Ficat and Arlet for ON of the Hip

Stage I: negative radiographs, positive MRI
Stage II: sclerotic and cystic changes on radiographs
Stage III:subchondral collapse
Stage IV:arthritic changes



Stage I- normal radiographs

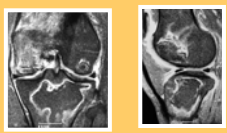
Stage II- sclerosis in medial femoral condyle

Stage III- evidence of collapse in medial condyle

Stage IV- degenerative changes, extensive in this case

MRI

99% sensitive for ON.
T1: low signal surrounded by intermediate signal in zone of ON
T2: area of low signal surrounded by high signal intensity



Bone Scan

Not useful for secondary ON.

In 24 patients 89 lesions by MRI and histology, 55% detected by bone scan. (Healy WL:*Orthopaedics* 1991).

Treatment

SPONK

Prognosis

•Dependent upon size of lesion.
•6 to 8 weeks of acute pain, then becomes a low grade pain.
•Smaller Stage I lesions may resolve in 1 to 1.5 years.
•Lesions found on plain film that occupy <45% of the condyle also gradually resolve.
•If greater than 50% of condylar width lesion progresses with eventual joint destruction.

Non-operative

•For early stages analgesics and protected weight bearing.
•Good prognosis less than 45% of articular surface.
•Need to watch lesion for progression.

Arthroscopic Debridement

•Does not address intraosseous pathology.
•May be good for unstable chondral flaps and mechanical symptoms.

Osteochondral Allografting

•Only a few reported cases of Allografting in SPONK lesions. See Table.

•Fresh allografting may be a reasonable alternative in these patients as there are good results with traumatic defects in the knee.

•Results are more predictable with arthroplasty in the elderly population (Lotke ICL).

•Mont and colleagues experience with osetochonrdral autografts (OATS) 90% success at 3 years in 20 patients.

Author	Result in SPONK lesions	Result in Steroid Induced lesions
Bayne *et al*	5 of 6 unsatisfactory	3 of 3 unsatisfactory
Meyers *et al*	5 lesions, 3 excellent 2 lost to f/u	NA
Lotke *et al*	57% success rate	76% success rate
Flynn	67% success rate	75 % success rate

Core Decompression

•Role of core decompression in early SPONK not known.

•Forst and associates reported that core decompression provided immediate pain relief in 16 patients with SPONK. Fifteen lesions were stage I, and 1 had radiographic flattening. However, these lesions may have resolved with nonoperative management.

•At this time core decompression should be used for steroid induced ON and not spontaneous lesions.

Secondary ON

Prognosis

•Poor results with non-operative management.
•Likely due to extensive involvement with secondary ON.
•Of 32 patients at 6 years only 18% did not require total knee arthroplasty (Mont).
•Surgery recommended for painful lesions.

Non-operative

•For non-painful lesions analgesics and protected weight bearing.
•Need to watch lesion for progression. 80% asymptomatic knees will not progress.

Arthroscopic Debridement

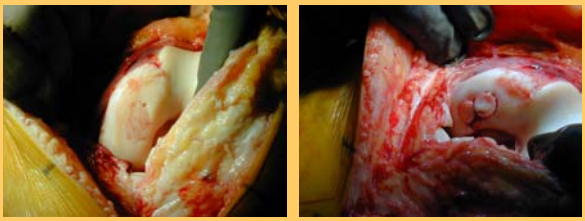
•Does not address intraosseous pathology.
•Mixed Results in literature
- Miller
- Weidel
•May combine with core decompression if intraarticular pathology.

Osteochondral Allografting

•Osteochondral allografts are not extensively studied in secondary ON of the Knee.

•Not practical for the extensive involvement with secondary ON.

•The underlying disorder is not addressed and this likely limits the results for allografting procedures.



Example of autograft from contralateral TKA use to treat SPONK lesion.

Core Decompression

•Based on relieving increased intraosseous pressure.

•Successful in delaying total knee arthroplasty in steroid associated ON.

•Usually provides immediate pain relief. Prevents progression if performed in early stages.

•34 of 42 knees good or excellent result(*Mont Tomek Hungerford)* usually immediate relief.

•74% still successsful at 10.4 years.

Femoral Core Decompression

Tibial Core Decompression

The area of pain is determined on physical exam. Core decompression is performed under fluoroscopy with 3-6mm Michele trephines.
The trephine is advanced two within a few millimeters of subchondral bone. For femoral lesions begin above the flare of the epicondyle.
For tibial lesions start just medial to the tibial tubercle.

Treatment

SPONK

Osteotomy

Osteotomy is an option in SPONK for younger active patients with loss of the medial compartment and change in mechanical axis.

Soucacos and collegues reported their results in stage III and IV SPONK. Results were better in Stage III lesions in younger patients.

Koshino reported his experience with high tibial osteotomy (HTO) for SPONK with and without bone grafting and drilling of the lesion. 37 knees were followed (average 67 months). The results were: malalignment was corrected to the results of HTO in primary arthritis.

Unicompartmental Arthroplasty

This may play a role in SPONK.

Attention needs to be paid to the underlying bone stock.

Marmor reported 88% success in 34 knees

2 failures occurs because of ON developing in opposite compartment.

2 failures occurred because of subsidence.

Total Knee Arthroplasty

Treatment of Choice for advanced stages of SPONK with Collapse.

Often good option as most patients are elderly females with osteoporotic bone.

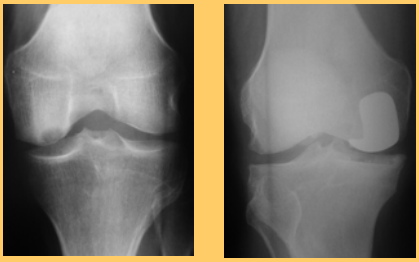
Results are less favorable than TKA for other diagnoses
Ritter and associates found at 7 years 17% of TKA for SPONK required revision. None for osteoarthritis needed to be revised.

New technique in total knee arthroplasty and the use of stems and augments will likely lead to better results in this patient population

Secondary ON

Osteotomy and Unicompartmental Arthroplasty

Given the extensive nature of lesions found in secondary ON, limited procedures such as osteotomy and unicompartmental knee replacement are not likely to be successful.



SPONK lesion of the distal femur
treated with unicompartmental replacement.

Total Knee Arthroplasty

Treatment of choice for Ficat stage III and IV secondary ON or when more conservative treatments fail.

Historically poor results compared to TKA for other diagnoses.

Author	Success Rate of TKA in ON	Years Follow up (year of study)
Bergman and Rand	68% successful	5 years (1993)
Mont *et al*	55% successful	6 years (1997)
Seldes, Lotke	81% successful	4 years (1999)
Mont *et al*	75 % successful	5 years (1999)

More recently Mont et al. reported a 97% success rate in 32 knees in 30 patients at average follow-up of 9 years.

- 22 patients had secondary ON and 8 had SPONK

Less than optimal results may have occurred in previous studies because of technique and fewer implant options than modern TKA.

Routine cementation is recommended in TKA in ON.

The use of stems and augments may be necessary for compromised bone stock.

SUMMARY

•SPONK and Secondary ON are two different entities that have different outcomes for various forms of treatment. Of utmost importance is the recognition of either condition and early intervention.

•For small lesions of SPONK (<45% of the affected condyle) symptomatic treatment is all that is necessary. Larger lesions may require reconstructive procedures include allografting or prosthetic replacement. Core decompressions are not effective for SPONK.

•Secondary osteonecrosis has poor outcomes with nonoperative treatment of symptomatic lesions. Core decompression is the treatment of choice in symptomatic lesions without collapse. Limited procedures such as osteotomy and unicompartmental arthroplasty will not do well in this generalized disorder. Total knee arthroplasty can have improved results with modern techniques, including cementation & stemmed prosthesis.

SELF-TEST

1.	Two distinct diseases have been called "osteonecrosis". Name them. In what age group does each occur? Do these disorders occur more commonly in one sex?
2.	With which disorder are other joints affected, and what is the likelihood of contralateral knee involvement? What is the likelihood of hip involvement?
3.	Coagulation disorders have been associated with which disease?
4.	In which disorder are the chondrocytes initially spared while the underlying bone is affected?
5.	Are the MRI images more consistent with SPONK or Atraumatic Osteonecrosis?
6.	For which entity are bone scans more useful
7.	Is there a way to radiographically predict which SPONK lesions may progress?
8.	Core decompression is a viable treatment for which entity: SPONK or Atraumatic Osteonecrosis?
9.	Which pulse sequence is best used to look for pathologic tissues and what does the signal change generally represent?
10.	What role does total knee arthroplasty play in SPONK and secondary ON?