Avascular Necrosis of Femoral Head

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Overview

- Introduction
- History
- Anatomy
- Epidemiology
- Etiology
- Pathogenesis
- Diagnosis and staging
- Management
Introduction

• Osteonecrosis, also referred to as avascular necrosis (AVN), aseptic necrosis, and ischemic necrosis, is not a specific disease but rather a condition in which a circumscribed area of bone becomes necrotic as a result of a loss of its blood supply

• Currently, 18% of all total hip arthroplasties performed in the United States are done for osteonecrosis
ONFH

• Refers to death of osteocytes with subsequent structural changes leading to femoral head collapse and secondary degen. arthritis of hip joint
History

• First described by Munro (1738)

• Curveilhier (1835) depicted femoral head morphological changes secondary to interruption of blood flow

• Ficat (1985) stated this condition resulted from blockage of osseous microcirculation with intramedullary stasis and increased pressure
Applied anatomy

• Proximal end of femur is supplied by three groups:
  - Extracapsular arterial ring
  - Ascending cervical branches of retinacular arteries
  - Arteries of ligamentum teres
Extracapsular arterial ring

- By medial femoral circumflex artery posteriorly and lateral circumflex femoral artery anteriorly

- Minor contribution from superior and inferior gluteal arteries
Ascending cervical branches

- Arises from extracapsular arterial ring
- Branches pass upwards under the synovial reflections and fibrous prolongations of capsule towards the articular cartilage
- Risk for injury due to its proximity to the bone
- Branches – Anterior, posterior, medial & lateral
  - **Lateral** supplies most to the femoral head and neck
Epidemiology

- True incidence of atraumatic AVN unknown
- Mean age of onset: 5th decade
- M:W = 4:1
- Atraumatic AVN is bilateral in 30-70% but typically asymmetrical
Etiology

- Trauma
- Alcohol consumption
- Corticosteroid intake
  - Hypercortisolism
  - Cushing disease
  - Hemoglobinopathies (SCD;Hb S/C;Polycythemia)
  - Caisson disease (*Dysbaric osteonecrosis*)
  - Pancreatitis
  - Neoplasms
  - CRF
  - Hemodialysis
  - Cigarette smoking
  - Collagen Vascular dis.
  - SLE

- Gout and hyperuricemia
- Hypercholesterolemia
- Hypercoagulable states
- Hyperlipidemia
- Hyperparathyroidism
- Intravascular coagulation
- Organ transplantation
- Pregnancy
- Congenital dislocation Hip
- Ehlers-Danlos synd
- Heredity dysostosis
- Legg-Calvé-Perthes dis
- Fabry disease
- Gaucher disease
- Giant cell arteritis
- Thrombophlebitis

*Idiopathic*
Traumatic ON

- Dislocation of hip (10-25%) or fracture neck of femur (15-50%)

- Prompt relocation helps in reducing incidence of ONFH

- 52% hips dislocated for more than 12hrs developed ONFH compared with 22% of those reduced within 12hrs
**Dysbarism**

- Tunnel workers and others associated with deep sea diving (1-4%)
- Formation of nitrogen bubbles within vessels precipitating infarction
Alcoholism

• 10-40% incidence

• Risk increases with cumulative dose of alcohol expressed as drink years

• Hyperlipidemic state – enhances thrombus

• Fat embolism from fatty liver and increased marrow fat
Haemoglobinopathies

• Mainly sickle cell disease (20-68%) and thalassaemia (4-12%)

• Sludging, thrombosis and eventual infarction at capillary level
Drug induced

- Steroids, phenytoin, indomethacin
- Steroid induced (10-30%)
- Mechanism – release of fat embolism from fatty liver, micro fracture secondary to osteoporosis and decreased intraosseous blood supply due increased fat cell mass
Collagen disease

- RA and systemic lupus erythematosus

- Inflammation of small peripheral blood vessels promotes the formation of vascular thrombosis and tissue infarction

- Therapeutic use corticosteroids also causes ON
Radiation

• Mechanism is unknown

• Apparent result is a combination of obliterative endarteritis and cellular death

• A threshold dose of 3000 rads
Gout

Sodium urate crystals enhance clotting by activation of Hageman factor, an initial protein component in the intrinsic coagulation mechanism
Idiopathic

- Most common category with no known cause

- Factors rendering bone liable to infarction at its articular edge
  - Small diameter of terminal vessels in subchondral region
  - Lack of collateral circulation
  - Reduced blood flow in bone with high marrow fat
  - In-expandable nature of bone tissue
Etiopathogenesis

• The bony compartment function essentially as closed compartment within which one element can expand only at expense of others

• Vascular occlusion and venous stasis leading to osteocyte necrosis play a central role in pathogenesis of AVN
Etiology

Pathogenesis

Trauma

Vascular interruption

Intravascular coagulation

Thrombotic occlusion

Decreased blood flow

Ischemia

Osteocyte necrosis

Repair

Loss of structural integrity

Collapse

Pathophysiology

Histopathology

Alcohol

Corticosteroids

Fat emboli

Extravascular compression
Pathogenesis

Hypoxia

Increased cell membrane permeability, which allows fluid and electrolytes to enter the cell, causing it to swell.

Lysosomal enzymes are released

Auto digestion or coagulation necrosis and cell rupture

Vascular injury leads to tissue edema and haemorrhage
Inflammatory response ensues

Disappearance of osteocytes from within their lacunae

Centre of the necrotic lesion remains avascular and repair is not possible

Repeated stresses

Dead trabeculae undergo micro fractures that cannot be repaired
Transition zone at the periphery, an active process of repair begins

Macrophages and osteoclasts remove dead marrow elements and bone

Granulation and fibrous tissue are formed
Osteoblasts form new bone, laid down directly on remnants of dead trabeculae

The resulting trabeculae are much thicker than normal

Responsible for the sclerotic margin that surrounds the lesion
• Small lesion (not in a major weight-bearing region) -
  Revascularization and completely replaced with
  viable bone

• Larger lesions, particularly those in a region of major
  weight bearing, have a poor prognosis - Gradual
  collapse
If the contour of the articular surface remains intact, a fluid-filled space beneath the cortical subchondral bone develops, which gives the appearance of a crescent sign on radiographs.
The mechanical stresses on the collapsed and irregular articular surface → Damage to and death of chondrocytes

These abnormal stresses are transferred to the otherwise normal cartilage of the acetabulum - Secondary degenerative changes.
Joint space narrowing

Typical changes of degenerative joint disease appear and include sclerosis, cyst formation, and marginal osteophytes

End-stage arthritis of the hip eventually ensues
Histopathological staging

STAGE 1

Cut Section:

Necrotic wedge shaped (dull, chalky, opaque and yellow), sub articular lesion, well demarcated

Micro:

Cartilage normal, subchondral bone shows eosinophilic changes lacking cellular elements, osteocytic lacunae empty
• Margin of infarct: increased osteoclastic activity with infiltration of granulation tissue (thin red rim)

• Beyond infarct and hypervascular zone; bone and marrow remain unchanged
STAGE 2

Articular surface remains intact

Cut section: rim of bony sclerosis at the boundary between necrotic zone and unaffected zone
Microscopy: advancing front of granulation tissue following which second front of osteoblasts (creeping substitution)

- Increased vascularity with osteoblastic activity and new bone formation
**STAGE 3**

Alteration in shape of articular bone
Gross: buckling

Cut section: fracture just below
articular end plate or on the necrotic
side of advancing sclerosis in
reparative front

- Fracture occurs due to weakening
  of trabeculae due to increased
  osteoclastic activity
STAGE 4

Articular deformity

Cut section: residual fragments of articular cartilage and dense fibrous connective tissue in the area of infarction

- Articular surface dense sclerotic eburnated
- OA changes
Clinical features

• Pain usual presenting symptom
• Intense and sudden in onset as in infarct or it can be insidious and chronic
• Groin pain but radiating to anterior and anteromedial thigh less common to buttocks
• Pain present at rest worsens with motion and weight bearing

• O/E – Antalgic gait
  Decreased ROM particularly flexion and IR
SECTORAL SIGN

The range of internal rotation is less in hip flexion compared to when hip in extension
Imaging

• **RADIOGRAPHY**
  (AP view and frog leg lateral views)

• Initial Radiographs will be normal

• Typical: mottled sclerosis and lucency usually in the anterosuperior segment of femoral head

• Progression into subchondral fracture and eventual collapse

• Advanced cases: secondary OA
Bone scan

• With technetium labelled phosphate analogue used for early detection of ON

• Not as sensitive as MRI

• During acute phase decreased uptake of bone tracer associated with vascular compromise

• Increased accumulation in chronic venous stasis in repair and revascularisation

• Can be useful, especially in assessing the status of multiple joints
CT scan

- Can visualize a small lesion not easily seen on routine radiographs, and it may demonstrate small areas of articular surface collapse that are not apparent on plain films.

- It may also be used to help quantitate the extent of femoral head involvement.
MR imaging

• Abnormalities in femoral head on MRI can be made out as early as 7 to 10 days after the onset of symptoms

• Better precision

• Low intensity signal band on both T1 weighted and T2 weighted images – early abnormality

• In more advanced lesions – T1 images continue to show low intensity signal but T2 images may exhibit signals of alternating high and low intensity (double line sign)
### Staging

<table>
<thead>
<tr>
<th>STAGE</th>
<th>SYMPTOMS</th>
<th>RADIOGRAPHY</th>
<th>BONE SCAN</th>
<th>PATHOLOGICAL FINDINGS</th>
<th>BIOPSY</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>None</td>
<td>Normal</td>
<td>Decreased uptake?</td>
<td>Infarction of weight-bearing portion of femoral head</td>
<td>Abundant dead marrow cells, osteoblasts, osteogenic cells</td>
</tr>
<tr>
<td>1</td>
<td>None/mild</td>
<td>Normal</td>
<td>Cold spot on femoral head</td>
<td>Spontaneous repair of infarcted area</td>
<td>New bone deposited between necrotic trabeculae</td>
</tr>
<tr>
<td>2</td>
<td>Mild</td>
<td>Density change in femoral head</td>
<td>Increased uptake</td>
<td>Spontaneous repair of infarcted area</td>
<td></td>
</tr>
<tr>
<td>2A</td>
<td></td>
<td>Sclerosis or cysts, normal joint line, normal head contour</td>
<td>Increased uptake</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2B</td>
<td>Flattening (crescent sign)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Mild to moderate</td>
<td>Loss of sphericity, collapse</td>
<td>Increased uptake</td>
<td>Subchondral fracture, collapse, compaction and fragmentation of necrotic segment</td>
<td>Dead bone trabeculae and marrow cells on both sides of fracture line</td>
</tr>
<tr>
<td>4</td>
<td>Moderate to severe</td>
<td>Joint space narrowing, acetabular changes</td>
<td>Increased uptake</td>
<td>Osteoarthritic changes</td>
<td>Degenerative changes in acetabular cartilage</td>
</tr>
</tbody>
</table>
Steinberg University of Pennsylvania

Stage 0  Normal or nondiagnostic radiograph, bone scan, and magnetic resonance imaging

Stage I  Normal radiograph; abnormal bone scan and/or magnetic resonance imaging
   A  Mild (<15% of head affected)
   B  Moderate (15% to 30% of head affected)
   C  Severe (>30% of head affected)

Stage II  Lucent and sclerotic changes in femoral head
   A  Mild (<15% of head affected)
   B  Moderate (15% to 30% of head affected)
   C  Severe (>30% of head affected)

Stage III  Subchondral collapse (crescent sign) without flattening of femoral head
   A  Mild (<15% of articular surface)
   B  Moderate (15% to 30% of articular surface)
   C  Severe (>30% of articular surface)

Stage IV  Flattening of femoral head
   A  Mild (<15% of surface and <2-mm depression)
   B  Moderate (15% to 30% of surface or 2- to 4-mm depression)
   C  Severe (>30% of surface or >4-mm depression)

Stage V  Joint narrowing and/or acetabular changes
   A  Mild
   B  Moderate
   C  Severe

Stage VI  Advanced degenerative changes
<table>
<thead>
<tr>
<th>STAGE</th>
<th>MRI FINDINGS</th>
<th>STRUCTURE</th>
<th>CONTOUR</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Abnormal</td>
<td>Normal</td>
<td>Normal</td>
</tr>
<tr>
<td>A</td>
<td>&lt;30%</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>B</td>
<td>&gt;30%</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>II</td>
<td>Abnormal</td>
<td>Abnormal</td>
<td>Normal</td>
</tr>
<tr>
<td>A</td>
<td>&lt;30%</td>
<td>&lt;30%</td>
<td>—</td>
</tr>
<tr>
<td>B</td>
<td>&gt;30%</td>
<td>&gt;30%</td>
<td>—</td>
</tr>
<tr>
<td>III</td>
<td>Abnormal</td>
<td>Abnormal</td>
<td>Abnormal</td>
</tr>
<tr>
<td>A</td>
<td>&lt;30%</td>
<td>&lt;30%</td>
<td>&lt;2 mm</td>
</tr>
<tr>
<td>B</td>
<td>&gt;30%</td>
<td>&gt;30%</td>
<td>&gt;2 mm</td>
</tr>
</tbody>
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Kerboul Necrotic Angle

- Determined by measuring arc of the articular surface overlying the lesion on AP and lateral radiographs

- These two are added and referred to as the combined necrotic angle
Goals of management

- Relief of pain
- Arrest the progression of disease
- Prevent the collapse of head
- Prevent secondary degenerative arthritis
Treatment modalities

- Conservative / non surgical treatment
- Core decompression
- Bone grafting
  - Cancellous bone graft
    - Autogenous
    - Allogenic
  - Osteochondral graft
  - Muscle pedicle bone graft
  - Free vascularized bone graft
- Osteotomy
- Electrical stimulation
- Joint reconstruction
TREATMENT ALGORITHM FOR OSTEONECROSIS OF THE FEMORAL HEAD BASED ON RADIOGRAPHIC EVALUATION

RADIOGRAPHIC EVALUATION OF LESION

SMALL, PRE-Collapsed

ASYMPTOMATIC

CONSIDER OBSERVATION, PHARMACOLOGICAL TREATMENT, OR CORE DECOMPRESSION

LARGE, COLLAPSED

OPERATIVE TECHNIQUE

FEMORAL HEAD CARTILAGE DAMAGE ONLY

LIMITED FEMORAL RESURFACING

TOTAL HIP ARTHROPLASTY

MEDIUM PRE-Collapsed TO EARLY POST

SYMPTOMATIC

BONE GRAFTING (VASCULARIZED OR NON-VASCULARIZED) OSTETOMY

CORE DECOMPRESSION WITH OR WITHOUT BONE GRAFTING
<table>
<thead>
<tr>
<th>Radiographic Stage</th>
<th>Symptoms</th>
<th>Procedure</th>
</tr>
</thead>
<tbody>
<tr>
<td>I and II</td>
<td>Asymptomatic</td>
<td>Observation, pharmacological treatment, possible core decompression ± bone-grafting</td>
</tr>
<tr>
<td>IA, IB, IC, IIA, IIB, and IIC</td>
<td>Symptomatic</td>
<td>Core decompression ± bone-grafting, vascularized graft</td>
</tr>
<tr>
<td>IC, IIC, IIIA, IIIB, IIIC, and IVA</td>
<td>Symptomatic</td>
<td>Bone-grafting (vascularized or nonvascularized), osteotomy, limited femoral head resurfacing, total hip arthroplasty</td>
</tr>
<tr>
<td>IVB and IVC</td>
<td>Symptomatic</td>
<td>Limited femoral head resurfacing, total hip arthroplasty</td>
</tr>
<tr>
<td>V and VI</td>
<td>Symptomatic</td>
<td>Total hip arthroplasty</td>
</tr>
</tbody>
</table>
Observation and medical management

• Pre-collapse lesion, asymptomatic – observation

• Symptomatic and asymptomatic lesions without radiographic changes – pharmacological (vasodilators, anticoagulants, and lipid lowering agents)
• Hyperlipidaemia and hypercholesterolemia – lipid lowering agents like lovastatin

• Hypertension from renal disease – antihypertensive like verapamil

• Coagulation disorders – anticoagulants eg. Stanazol

• Anabolic steroid in elevated lipoprotein A

• Systemic alendronate by decreasing osteoclastic activity
Protected weight bearing

• Decreases the degree of discomfort in patients who are symptomatic, they have not been shown to alter the natural course of this disorder.

• Following certain types of surgical procedures, such as core decompression, grafting, and osteotomies, where it is used as an adjunct.

• Protects the weakened regions from fracture, and perhaps protects the femoral head as well, until the healing processes have progressed satisfactorily.
Electrical stimulation

Two fundamental mechanisms of action:

1. Important role in control of local inflammation

2. Favours repair activity and can potentiate the healing process by stimulating neo-vascularization and new bone formation
Radiographic progression in Ficat stage II. Hips treated with core decompression (CD) plus pulsed electromagnet fields (PEMF) exhibit 33% less radiographic progression than hips treated with CD alone.
Bone marrow infiltration

• The small no. of progenitor cells in the proximal extremity of the femur with ONFH causes insufficient creeping substitution after osteonecrosis

• Red bone marrow graft contains osteogenic precursors, which help in the reparative process

• Used in adjunct to Core Decompression
Core decompression

• Rationale – relieves intraosseous pressure, improves vascularity thus slows the disease progression

• Can be combined with placement of non vascularised, non structural bone grafts or graft substitutes

• Recently insertion of porous tantalum rods has been advocated

• Ficat stage 1 and 2a

• 30% patient even with early stage disease likely to end up with THR in 4-5yrs of core decompression

Bone School @ Bangalore
Core Decompression

• The best indications are hips with osteonecrosis without collapse

• In some patients who had Steinberg stage III (subchondral crescent, no collapse), successful outcomes have been obtained between 5 and 10 years

• Therefore, in selected patients, even more advanced disease can be considered for core decompression
Post op care: Partial weight bearing 50% on crutches for at least 6 weeks
Bone grafting

• Various authors have claimed success rate of 50 – 80% after CD with structural BG

• Non vascularised BG – Ficat 1 and 2

• Accurate placement of the graft within the lesion and under the subchondral bone

• Standard core technique, Lightbulb technique, Trap door technique
Vascularised fibular graft

• Rationale:
  - Decompression of femoral head
  - Excision of the sequestrum
  - Filling the defect with osteoinductive cancellous graft and viable cortical strut

• Longer recovery period and less uniform and less complete relief of pain

• Success rate 80-91% for younger symptomatic patients without collapse
Proximal femoral osteotomy

• Rationale – to move the necrotic segment of femoral head away from the weight bearing area

• Small and medium sized (<30% of head involvement/necrotic angle less than 200)
• Valgus/Flexion osteotomy – anterolateral lesion

• Varus/Extension - Central or superomedial lesion
Sugioka transtrochanteric anterior rotational osteotomy (see text). Transposition of the necrotic portion of the femoral head anteroinferiorly away from the weight-bearing area is accomplished by anterior rotation of the femoral head. A, Before rotation. B, After rotation.
THR and Bipolar Hemiarthroplasty
Thank you