Avascular Necrosis of Femoral Head

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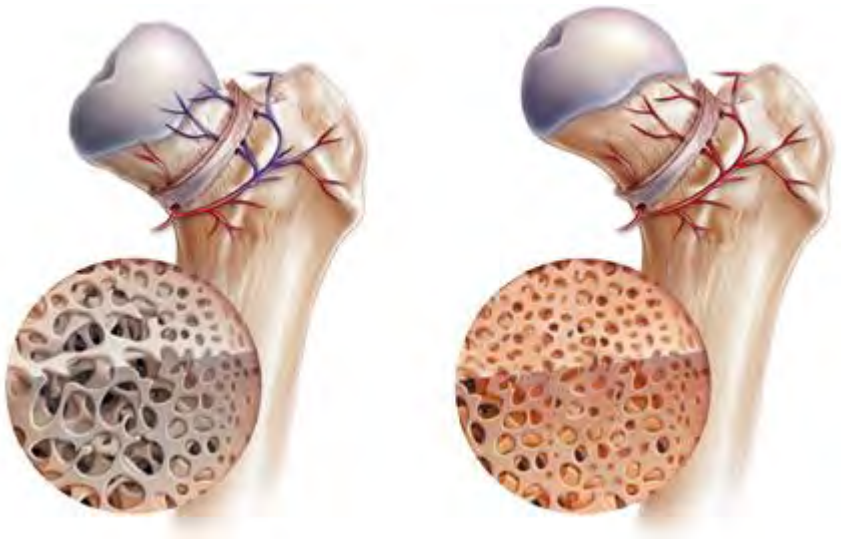
Overview

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

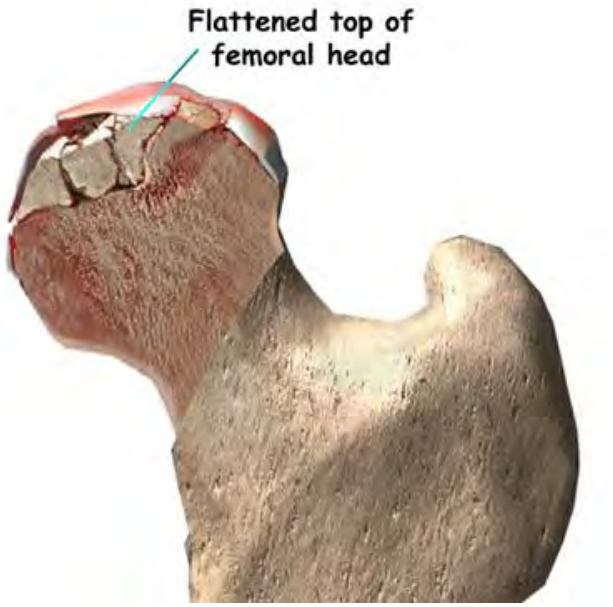


Introduction

- 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

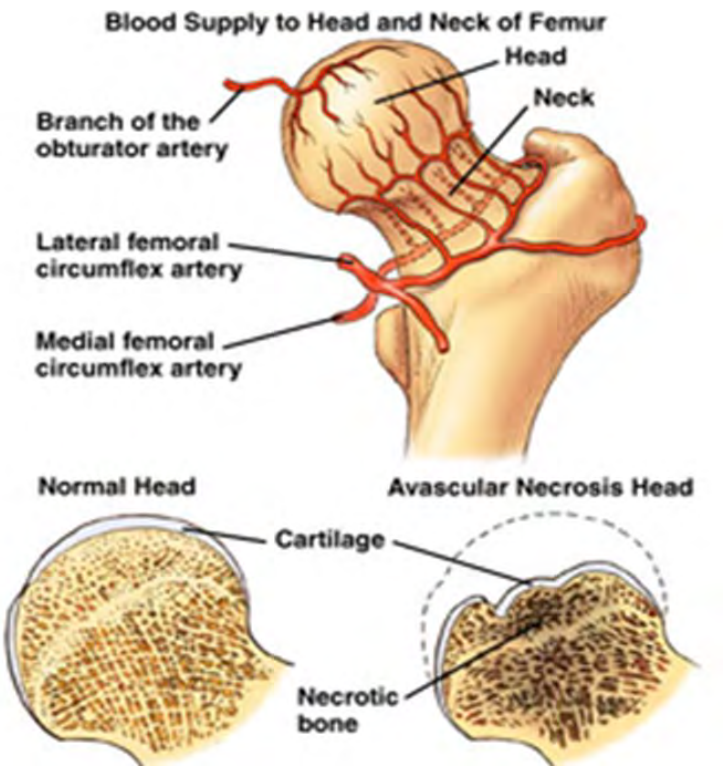
Applied anatomy

- Proximal end of femur is supplied by three groups :

- Extracapsular arterial ring

- Ascending cervical branches of retinacular arteries

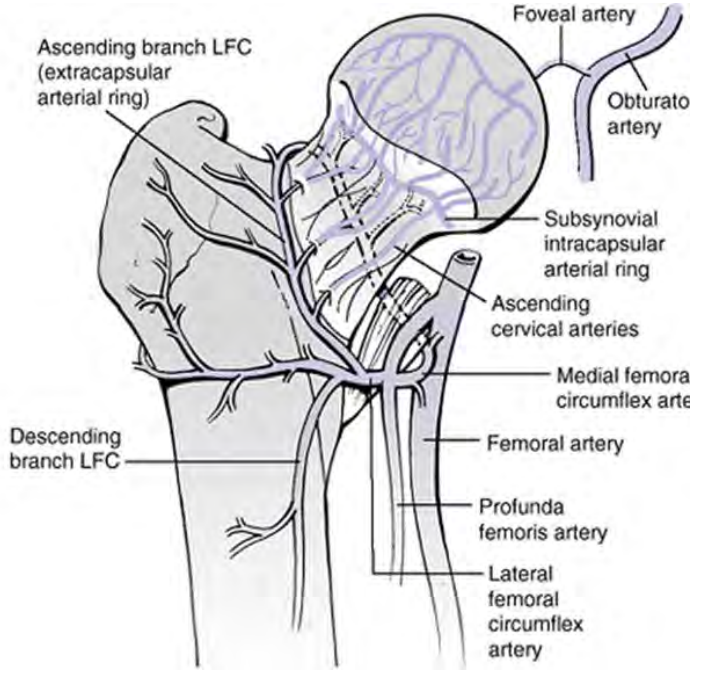
- Arteries of ligamentum teres



Extracapsular arterial ring

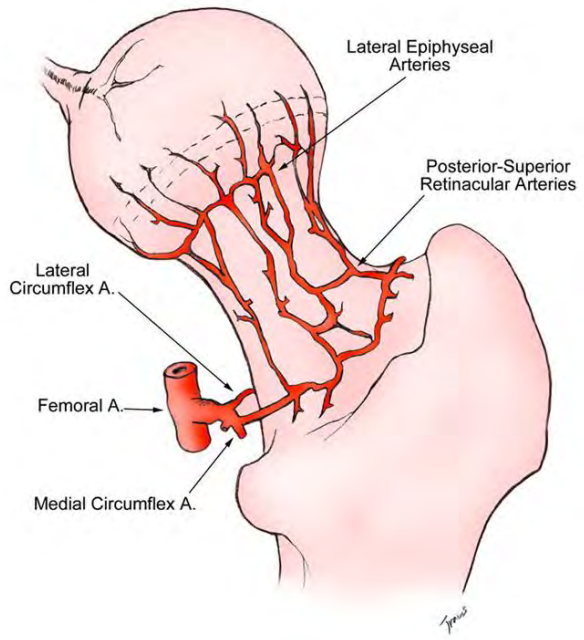
- By medial femoral circumflex artery posteriorly and lateral circumflex femoral artery anteriorly

- 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

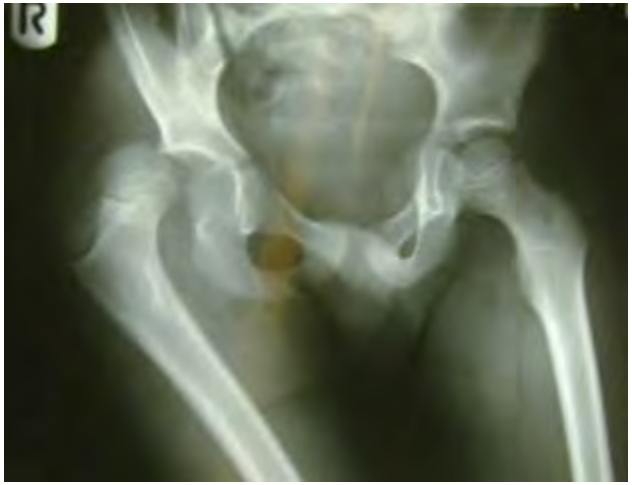
Etiology

- **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

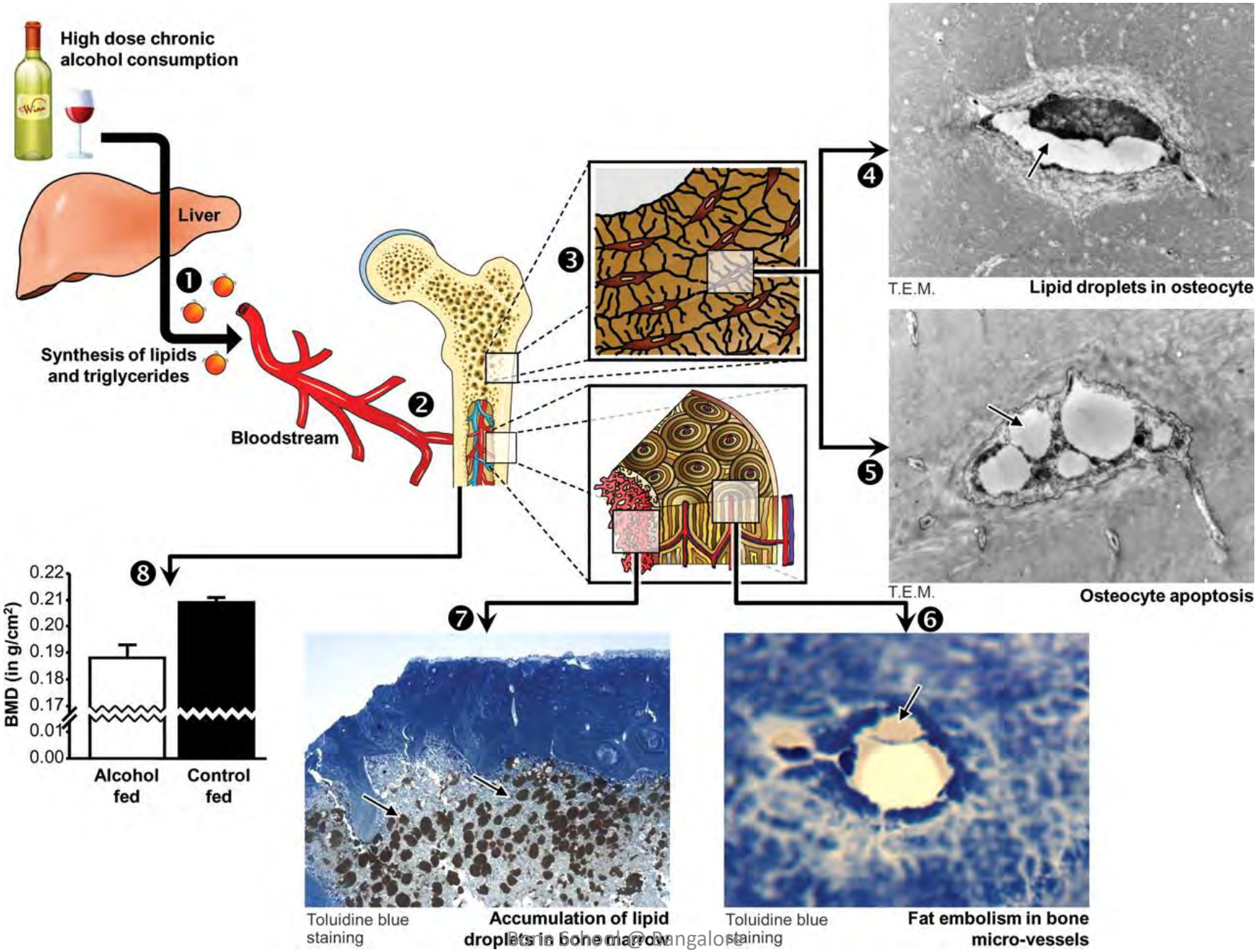


Dysbarism

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

Alcoholism

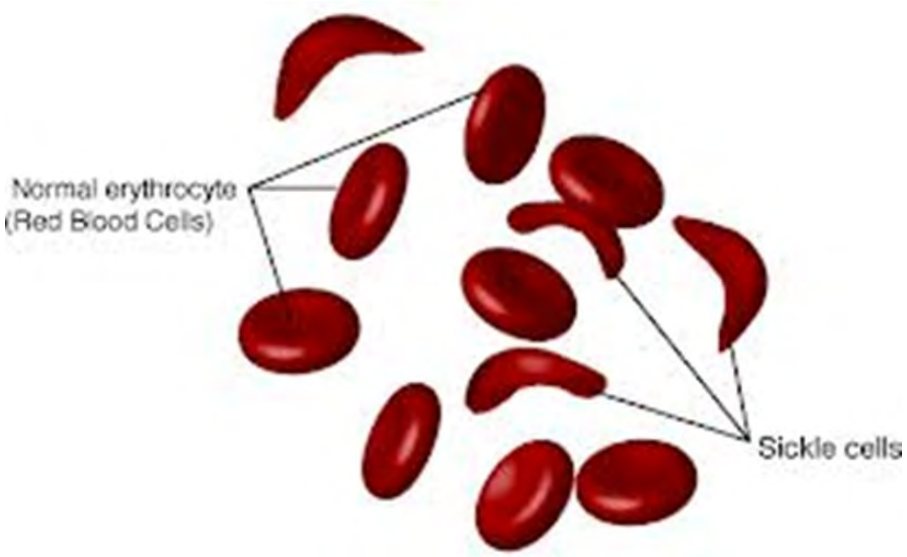
- 10-40% incidence
- Risk increases with cumulative dose of alcohol expressed as drink years
- Hyperlipidemic state – enhances thrombus
- Fat embolism from fatty liver and increased marrow fat



Haemoglobinopathies

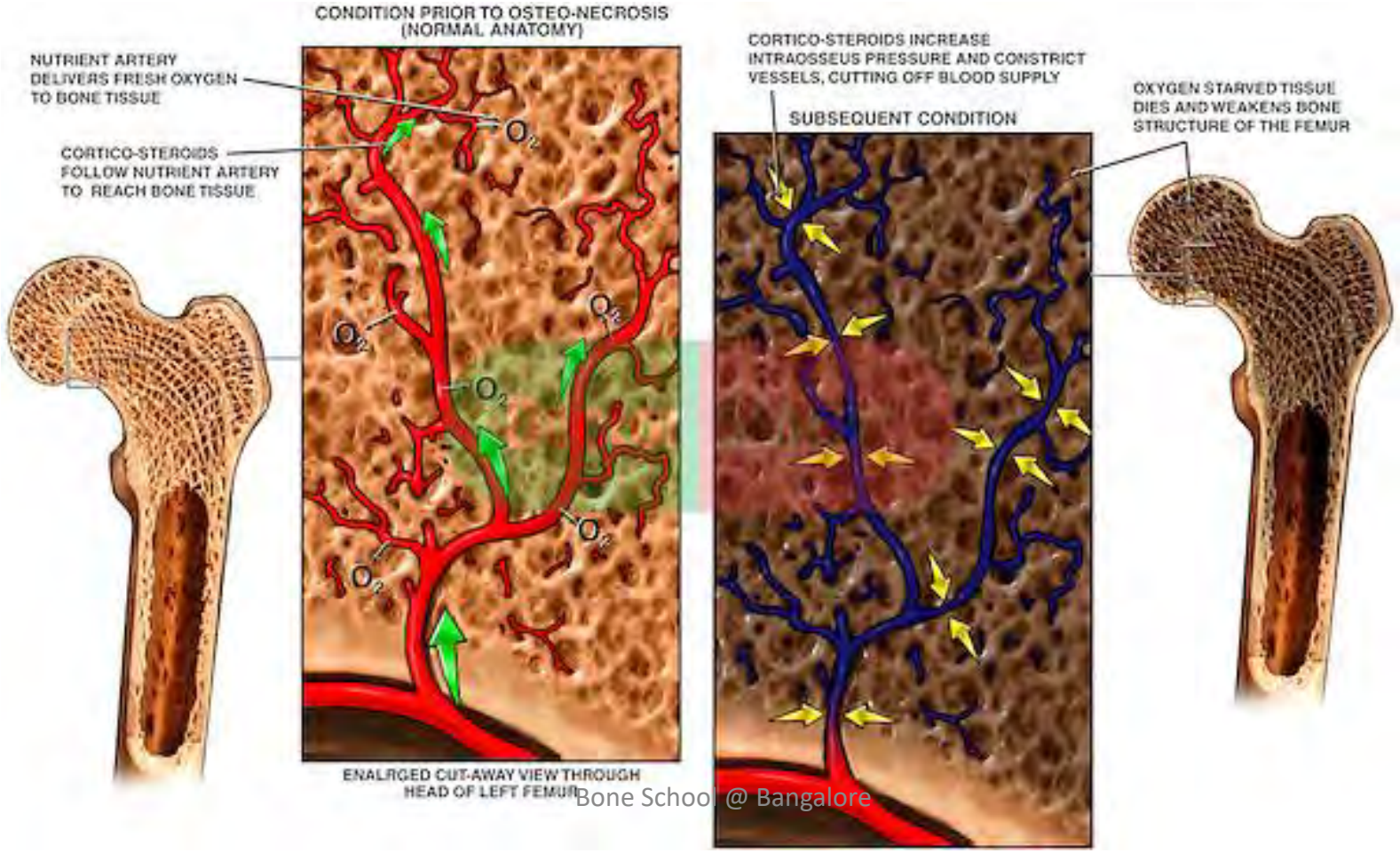
- Mainly sickle cell disease(20-68%) and thalassaemia (4-12%)

- 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

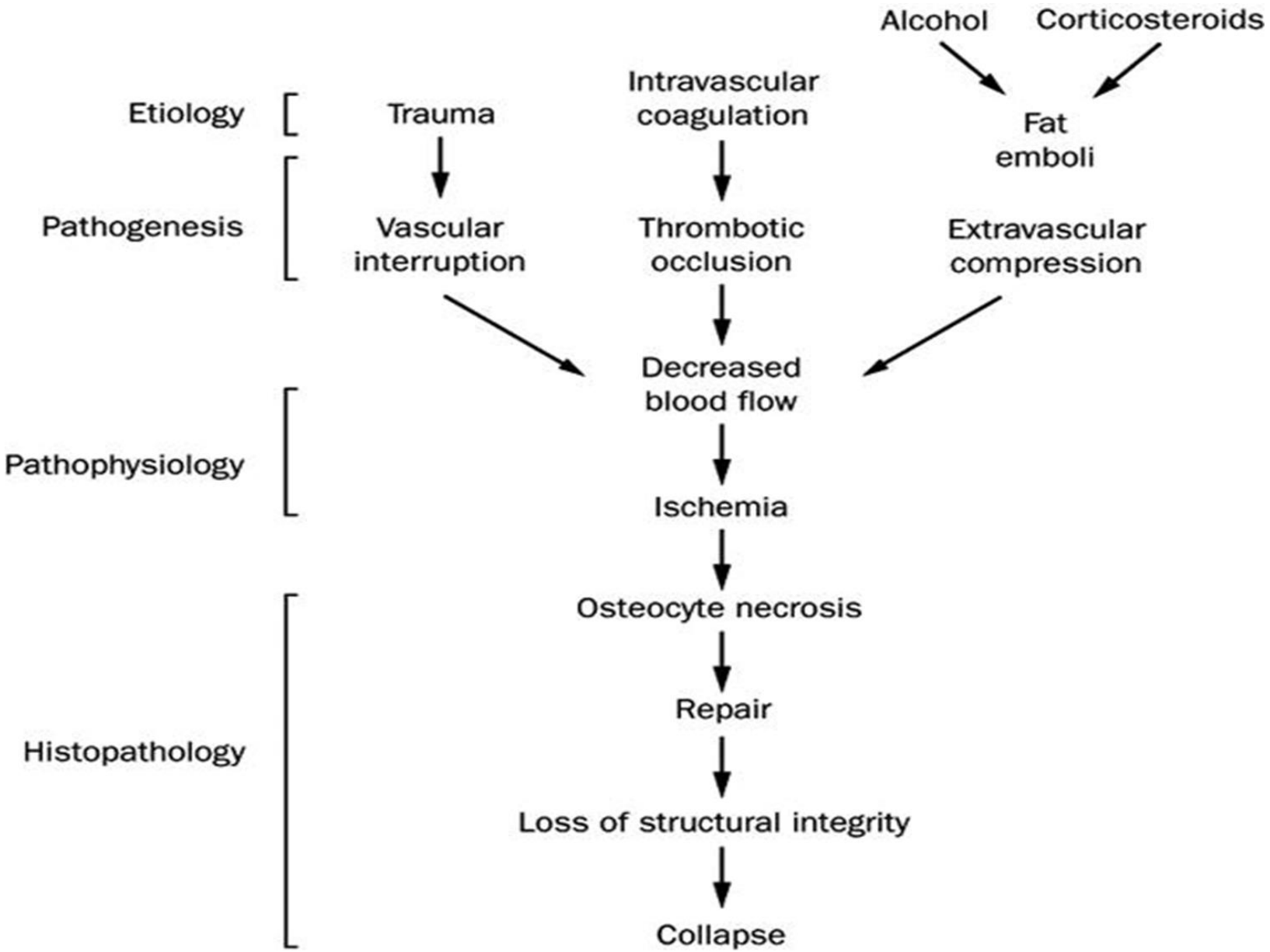
  - lack of collateral circulation

  - 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


Alcohol
Corticosteroids
Etiology
Trauma
Intravascular coagulation
Fat emboli
Pathogenesis
Vascular interruption
Thrombotic occlusion
Extravascular compression
Decreased blood flow
Pathophysiology
Ischemia
Osteocyte necrosis
Repair
Histopathology
Loss of structural integrity
Collapse

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

⬇

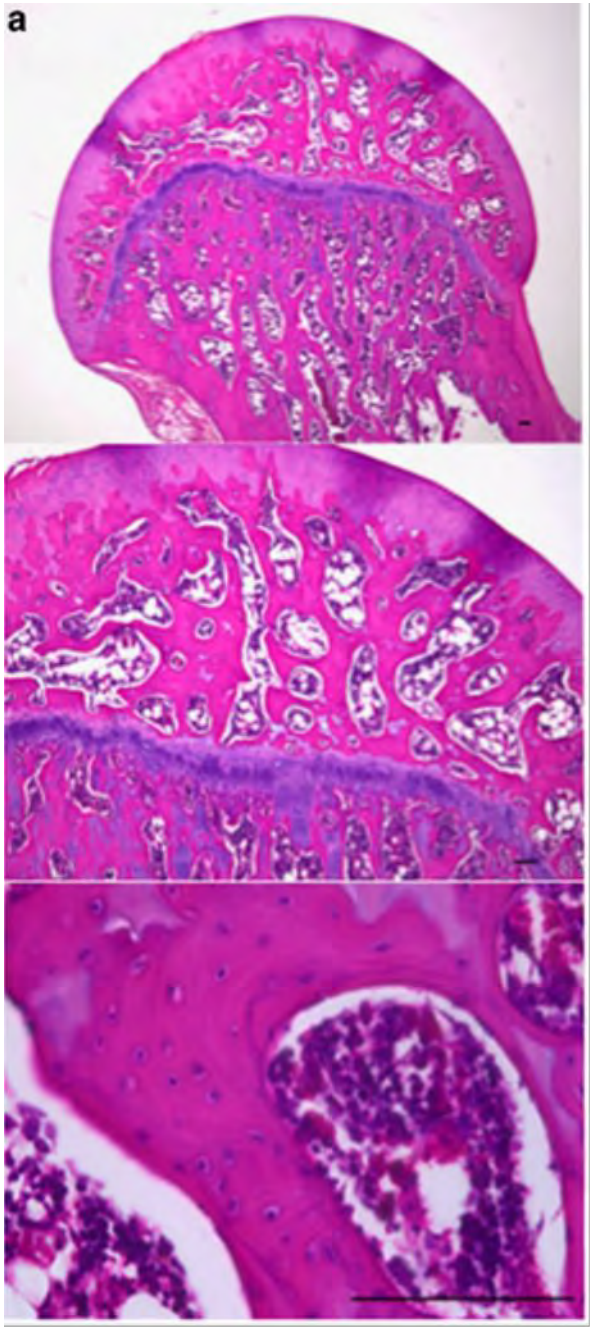
Centre of the necrotic lesion remains avascular and repair is not possible

⬇

Repeated stresses

⬇

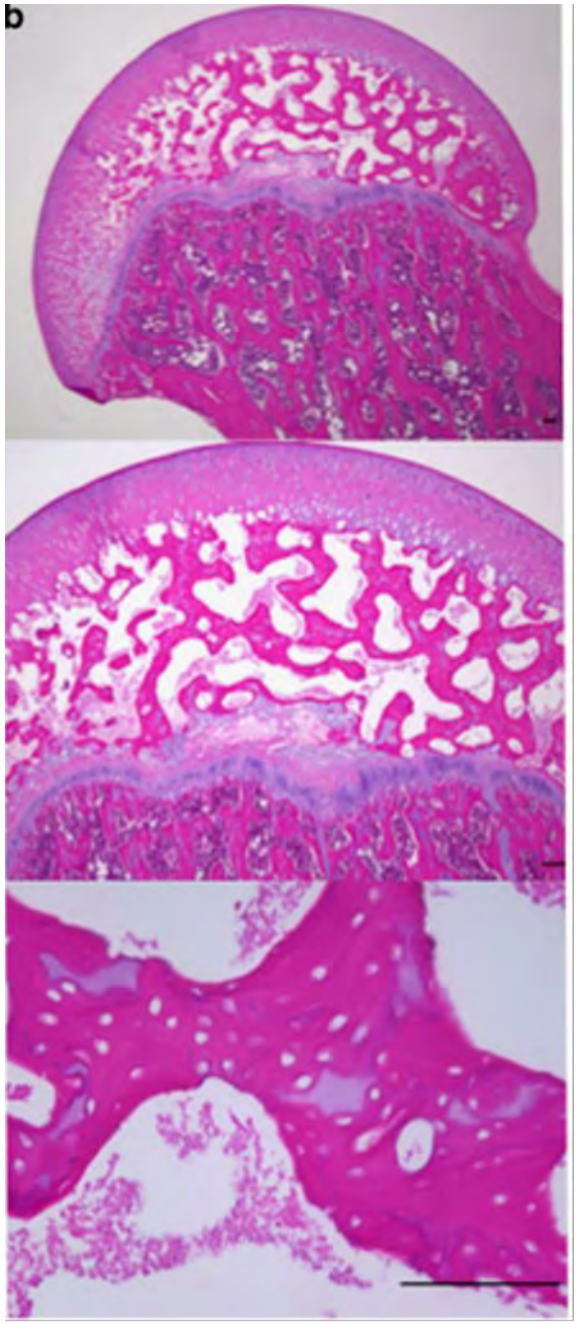
Dead trabeculae undergo micro fractures that cannot be repaired



Transition zone at the periphery, an active process of repair begins

Macrophages and osteoclasts remove dead marrow elements and bone

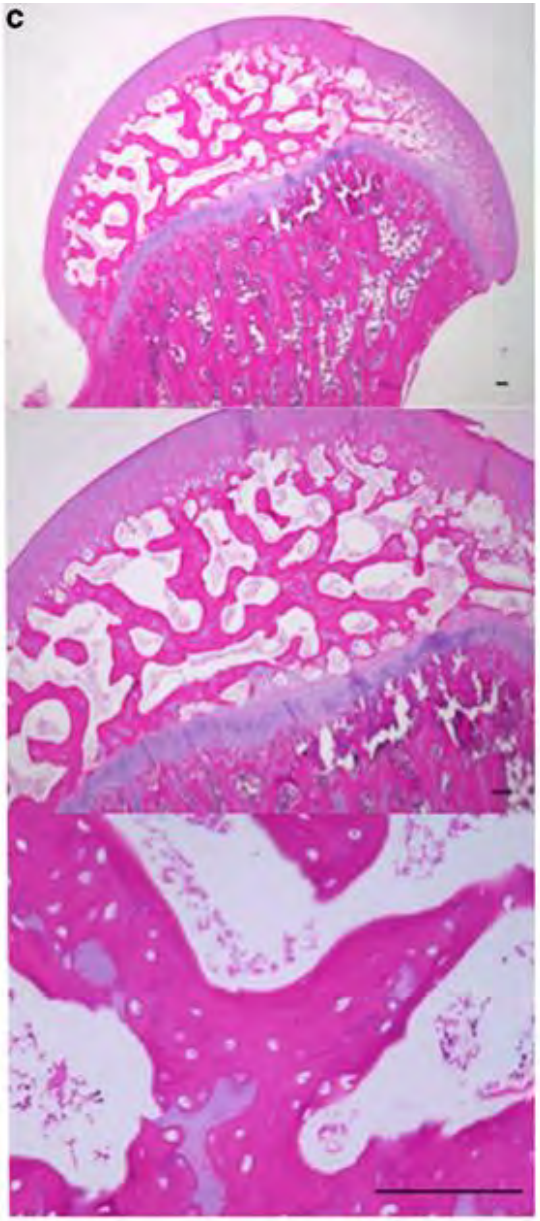
Granulation and fibrous tissue are formed



Osteoblasts form new bone, laid down directly on remnants of dead trabeculae

The resulting trabeculae are much thicker than normal

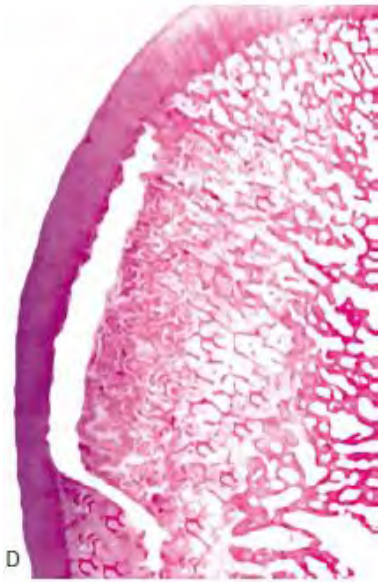
Responsible for the sclerotic margin that surrounds the lesion



- 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.

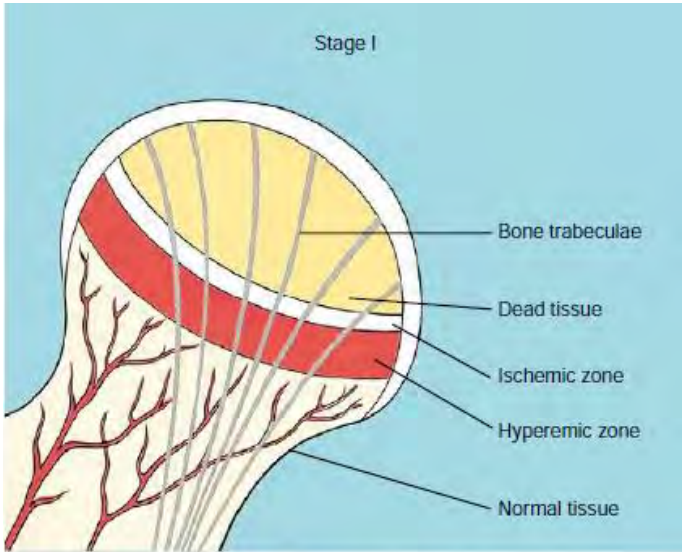
Joint space narrowing

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

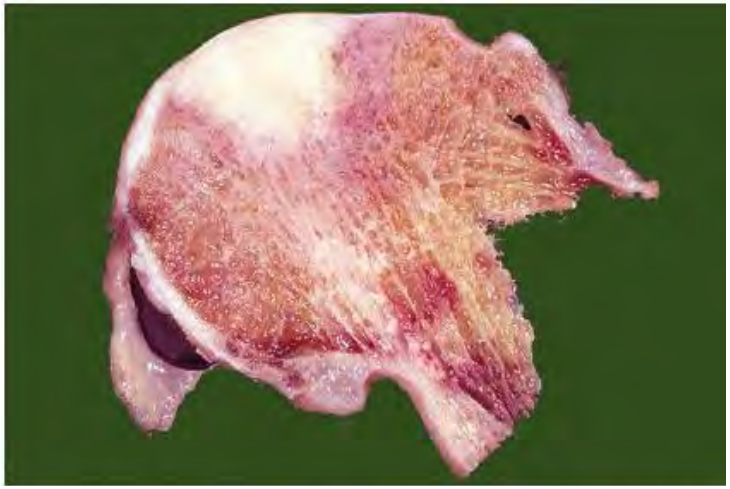
STAGE 1



Cut Section:

Necrotic wedge shaped (dull, chalky, opaque and yellow),

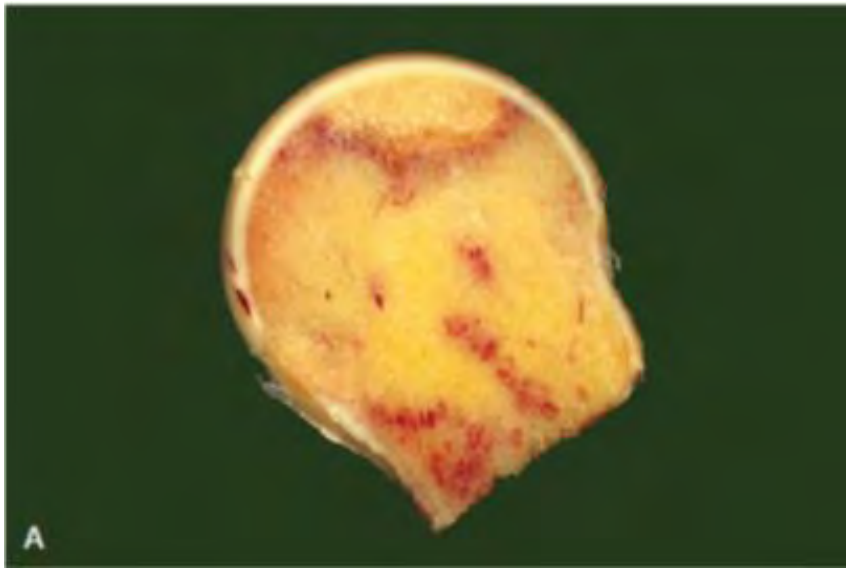
sub articular lesion, well demarcated



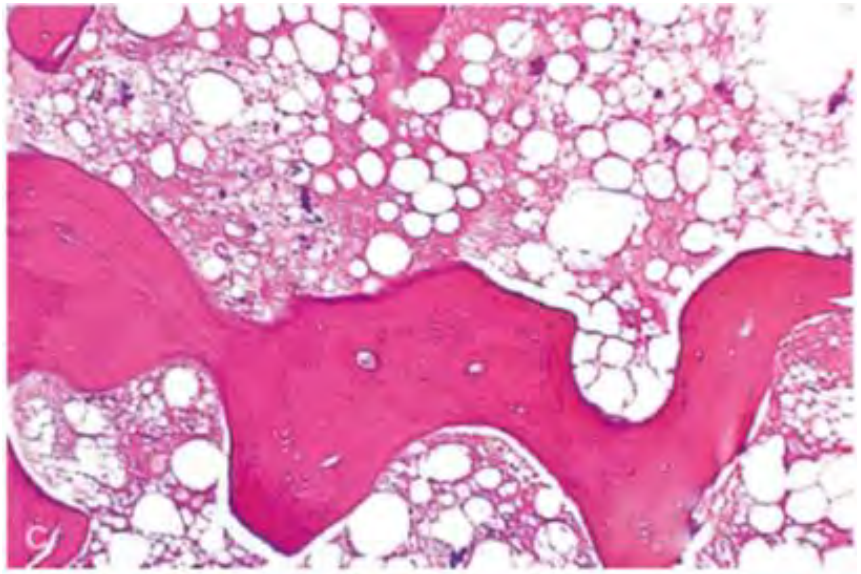
Micro :

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

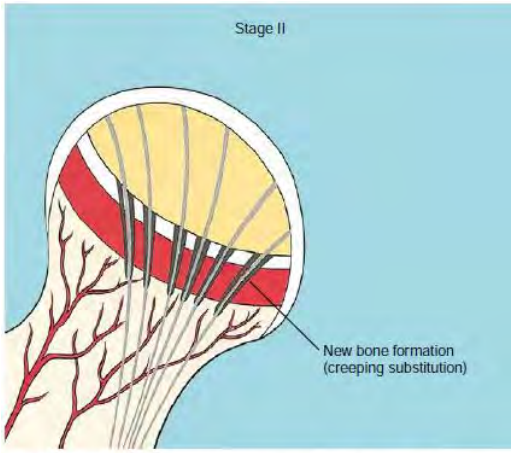
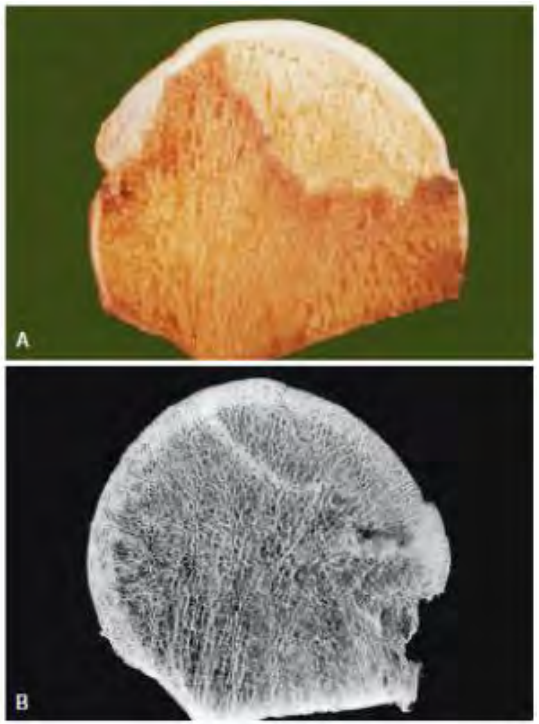
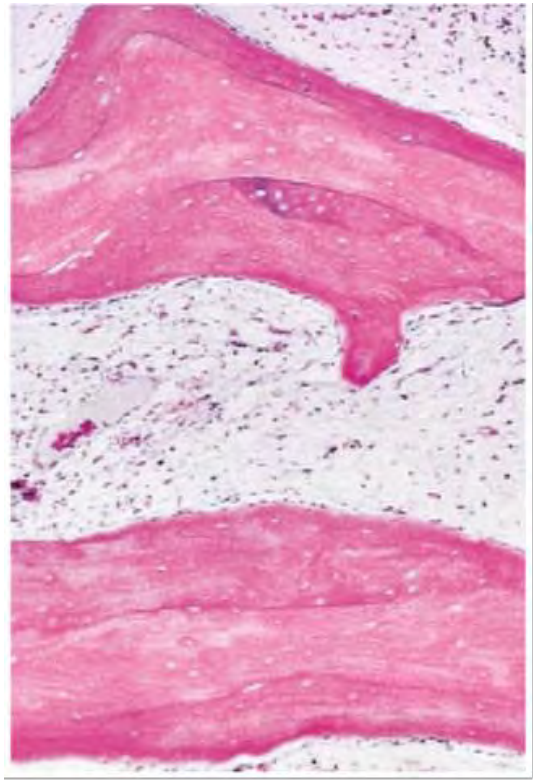


FIGURE 15-18 Diagrammatic representation of the changes that occur in stage II subchondral avascular necrosis.

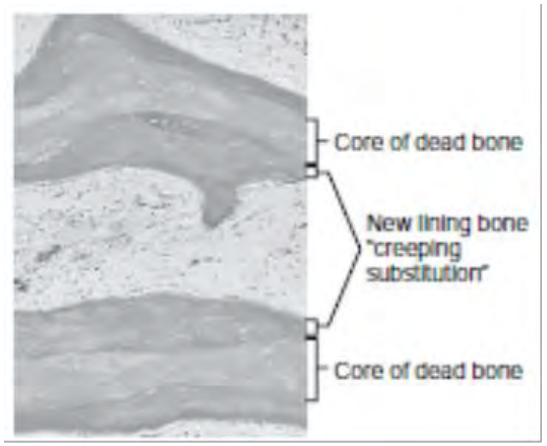
Cut section : rim of bony sclerosis at the boundary between necrotic zone and unaffected zone



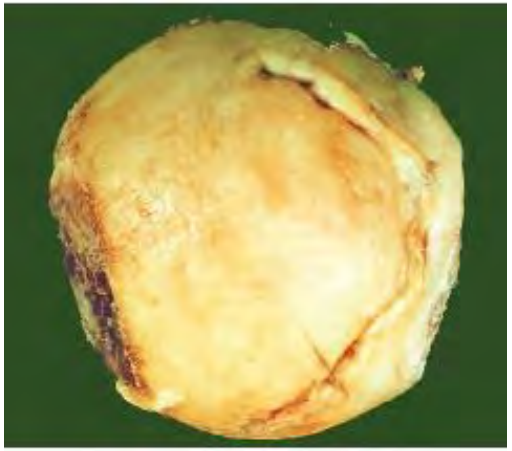
Microscopy : advancing front of granulation tissue following which second front of osteoblasts (creeping substitution)

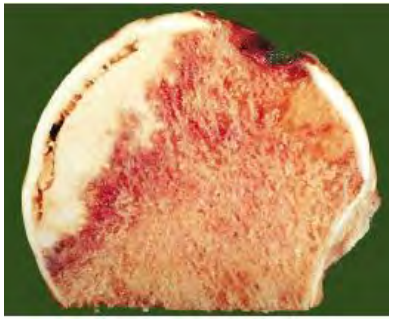


- Increased vascularity with osteoblastic activity and new bone formation



STAGE 3

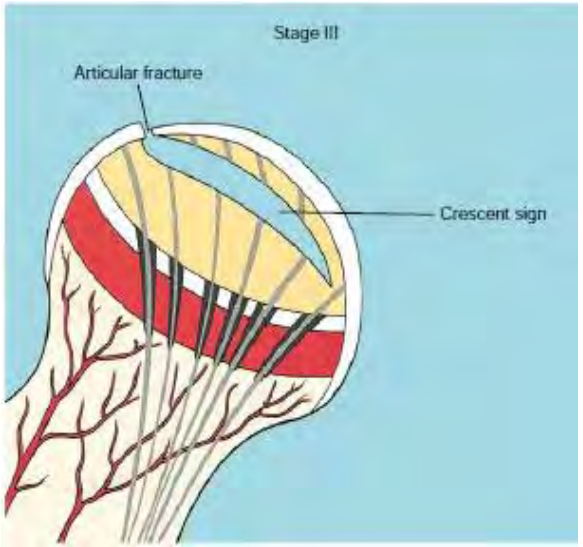




Alteration in shape of articular bone
Gross : buckling

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

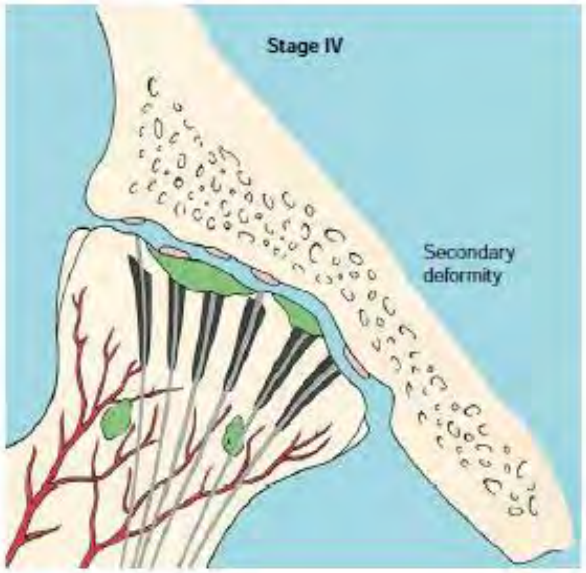


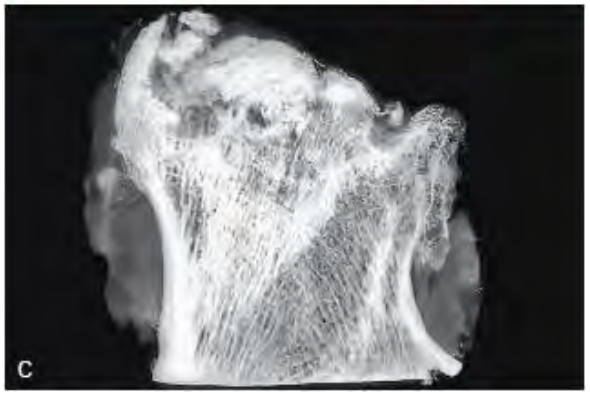
STAGE 4

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





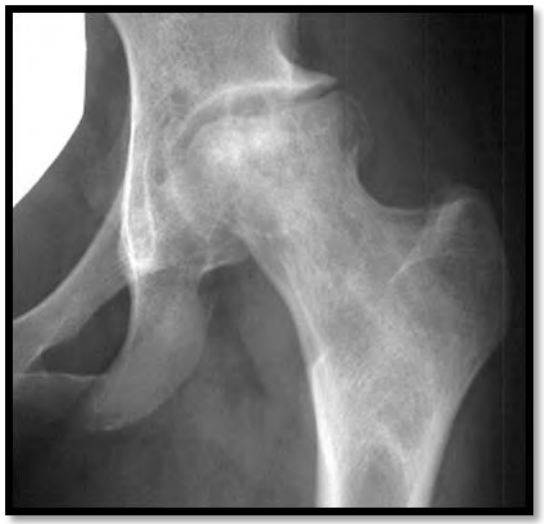
Clinical features

- 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

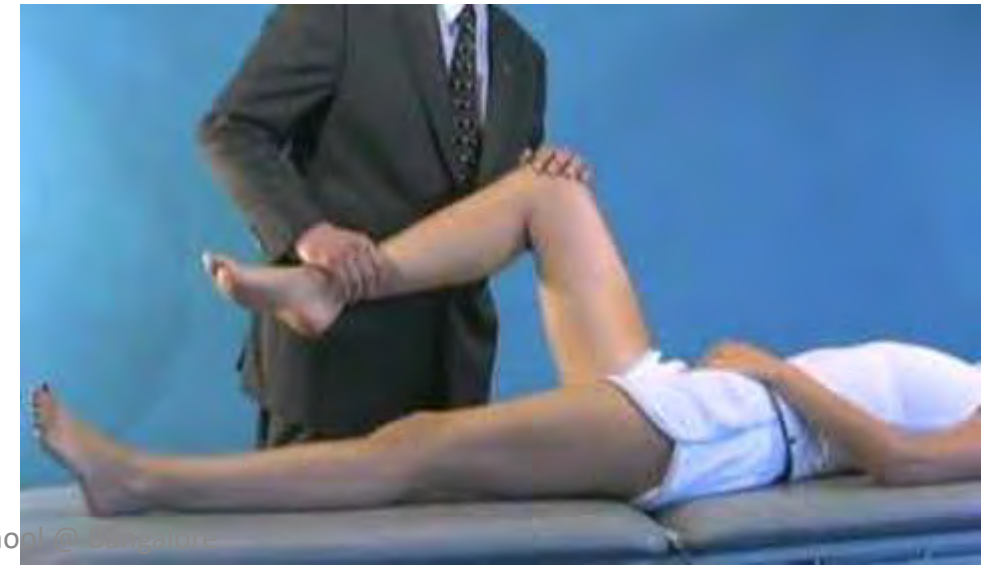
SECTORAL SIGN



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 to10 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 6-1 Ficut and Arlet Classification of Osteonecrosis of the Femoral Head

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

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

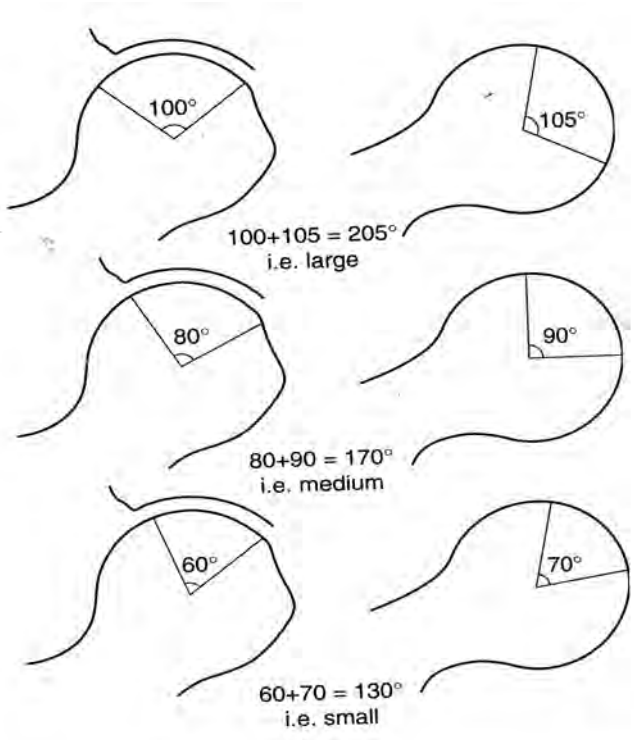
TABLE 6-3 Pittsburgh Classification

		PLAIN RADIOGRAPHIC FINDINGS	
STAGE	**MRI FINDINGS**	**STRUCTURE**	**CONTOUR**
I	Abnormal	Normal	Normal
A	<30%	—	—
B	>30%	—	—
II	Abnormal	Abnormal	Normal
A	<30%	<30%	—
B	>30%	>30%	—
III	Abnormal	Abnormal	Abnormal
A	<30%	<30%	<2 mm
B	>30%	>30%	>2 mm

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



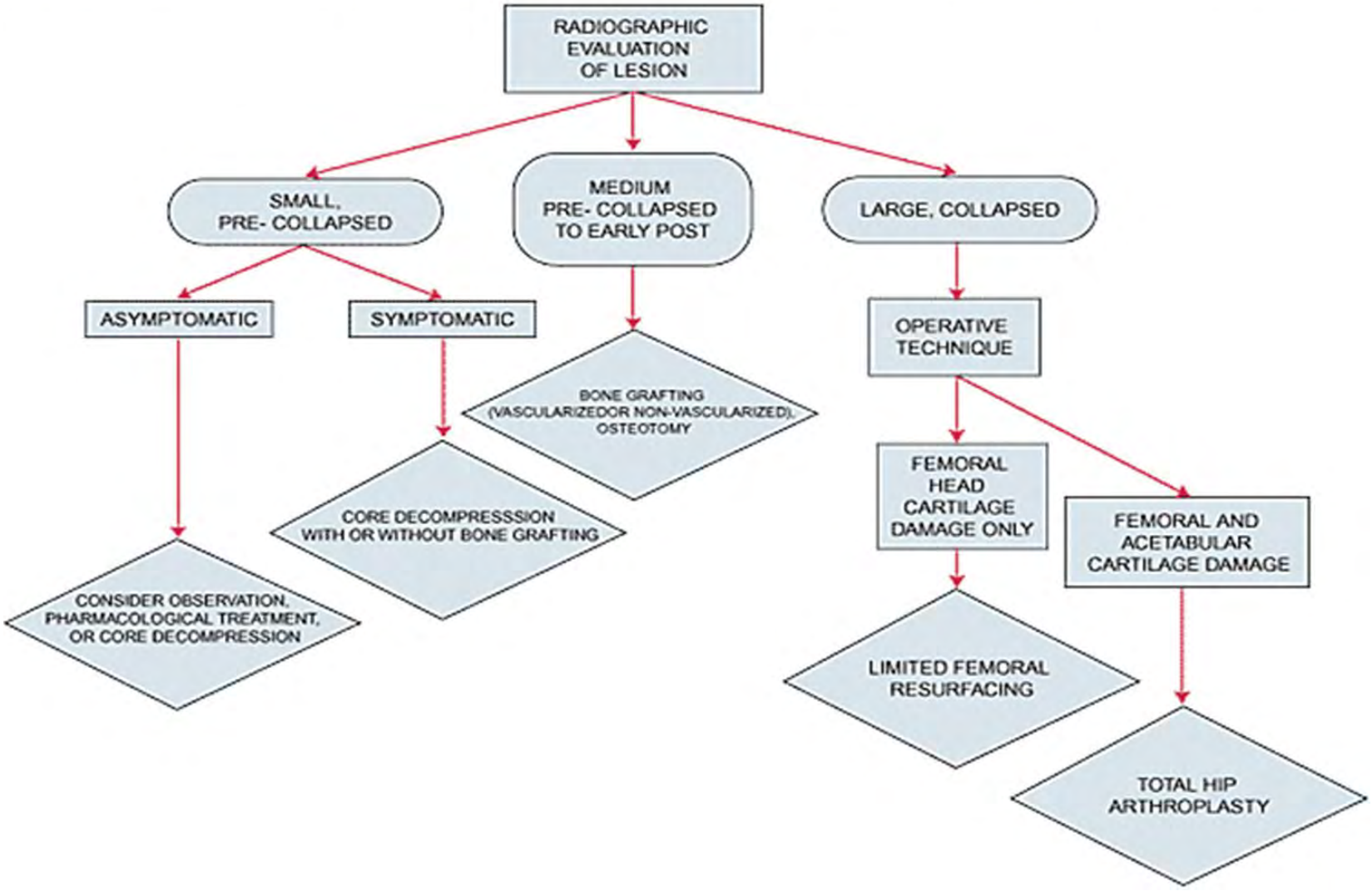
Goals of management

- 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
    - Allogenous
  - 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 Stage	Symptoms	Procedure
I and II	Asymptomatic	Observation, pharmacological treatment, possible core decompression ± bone-grafting
IA, IB, IC, IIA, IIB, and IIC	Symptomatic	Core decompression ± bone-grafting, vascularized graft
IC, IIC, IIIA, IIIB, IIIC, and IVA	Symptomatic	Bone-grafting (vascularized or nonvascularized), osteotomy, limited femoral head resurfacing, total hip arthroplasty
IVB and IVC	Symptomatic	Limited femoral head resurfacing, total hip arthroplasty
V and VI	Symptomatic	Total hip arthroplasty

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.

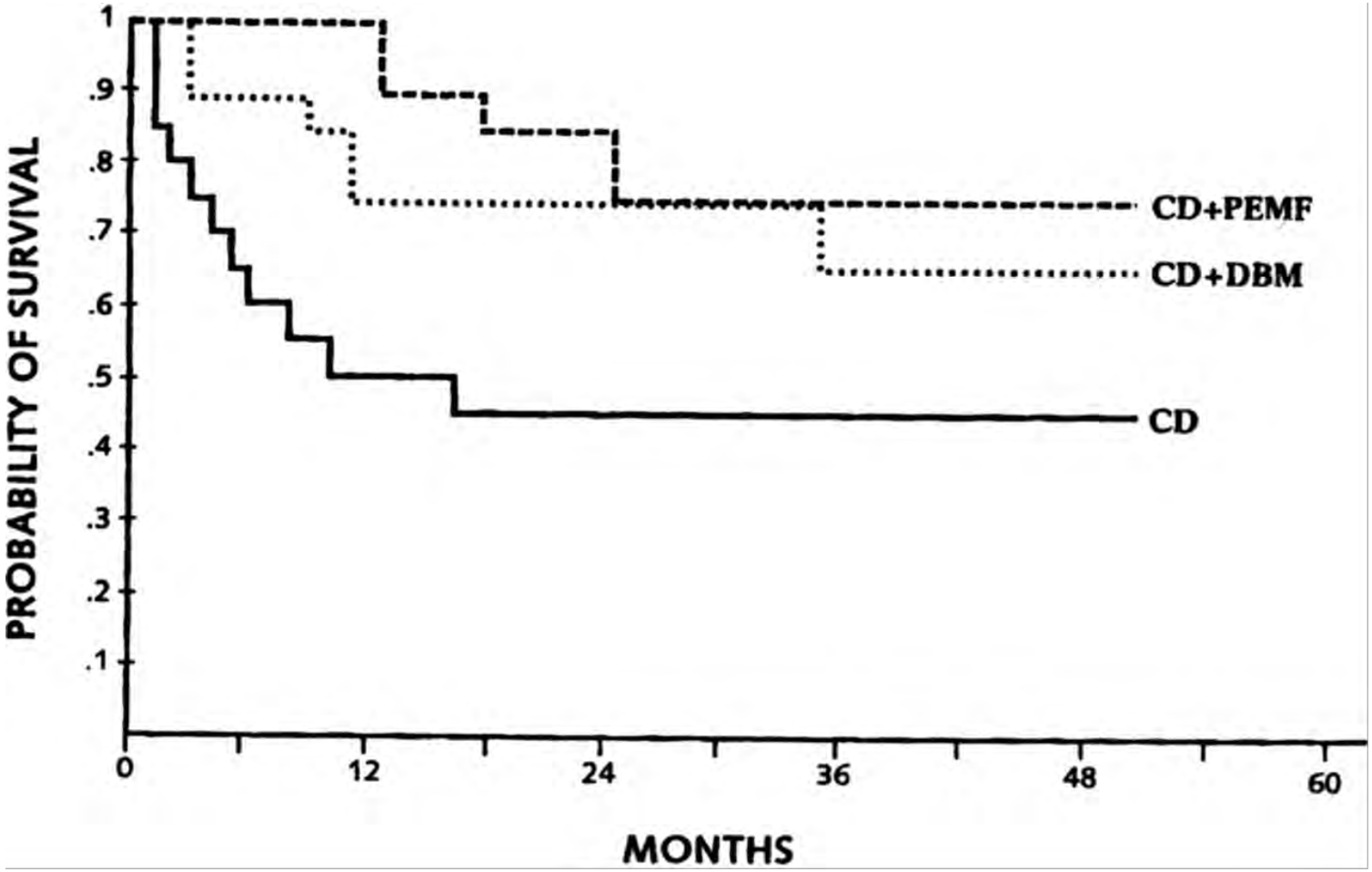
- Protects the weakened regions from fracture, and perhaps protects the femoral head as well, until the healing processes have progressed satisfactorily

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

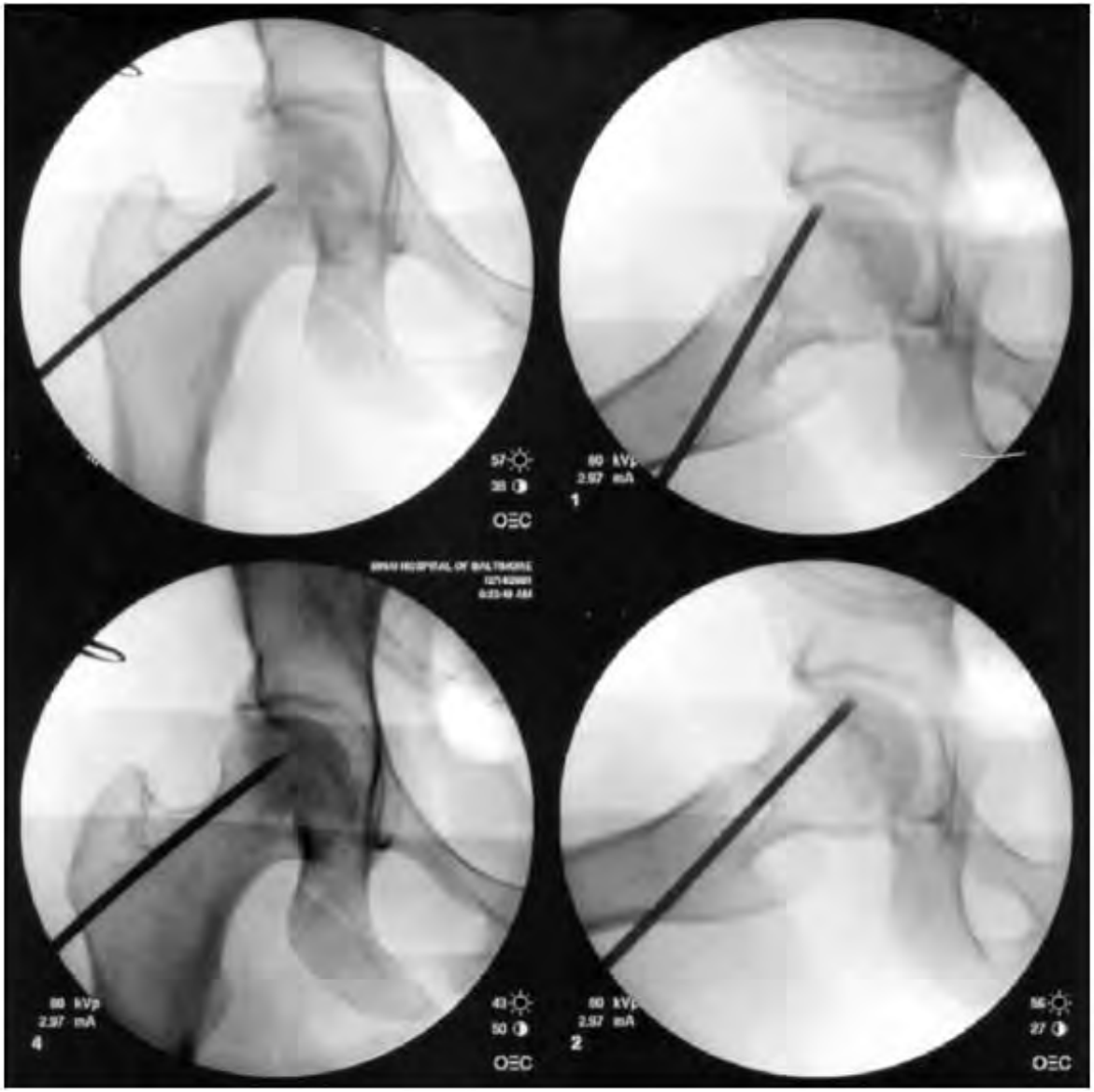
- The small no. of progenitor cells in the proximal extremity of the femur with ONFH causes insufficient creeping substitution after osteonecrosis
- Red bone marrow graft contains osteogenic precursors, which help in the reparative process
- Used in adjunct to Core Decompression

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

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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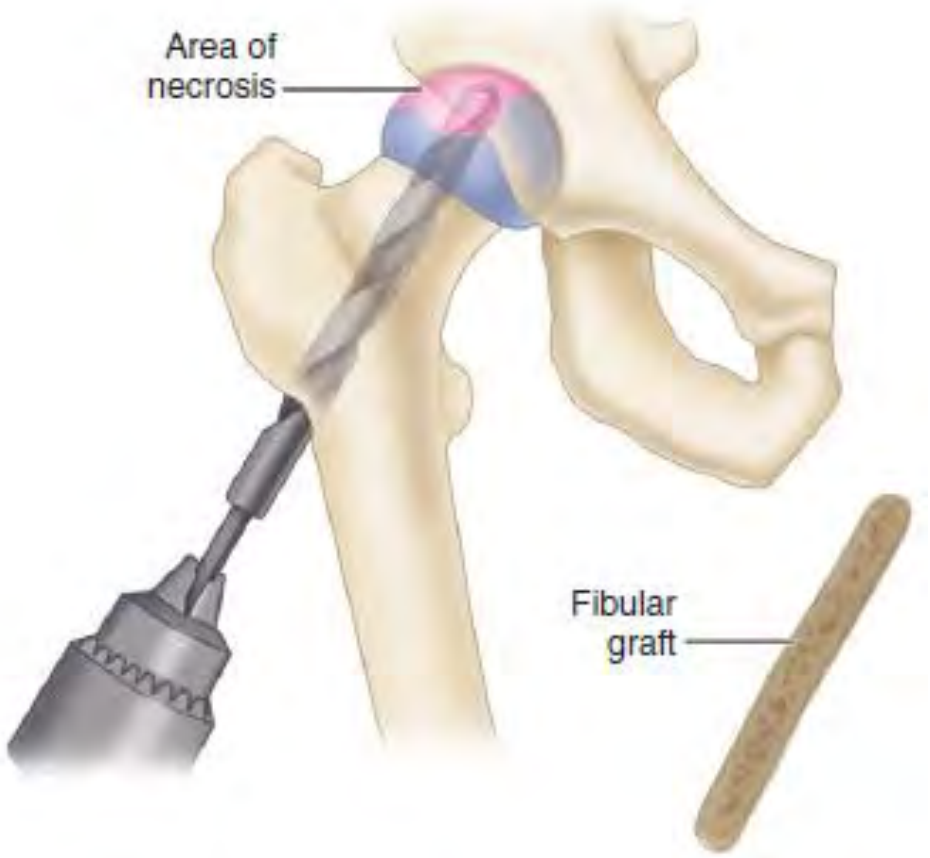


Post op care : Partial weight bearing 50% on crutches for at least 6 weeks

Bone grafting

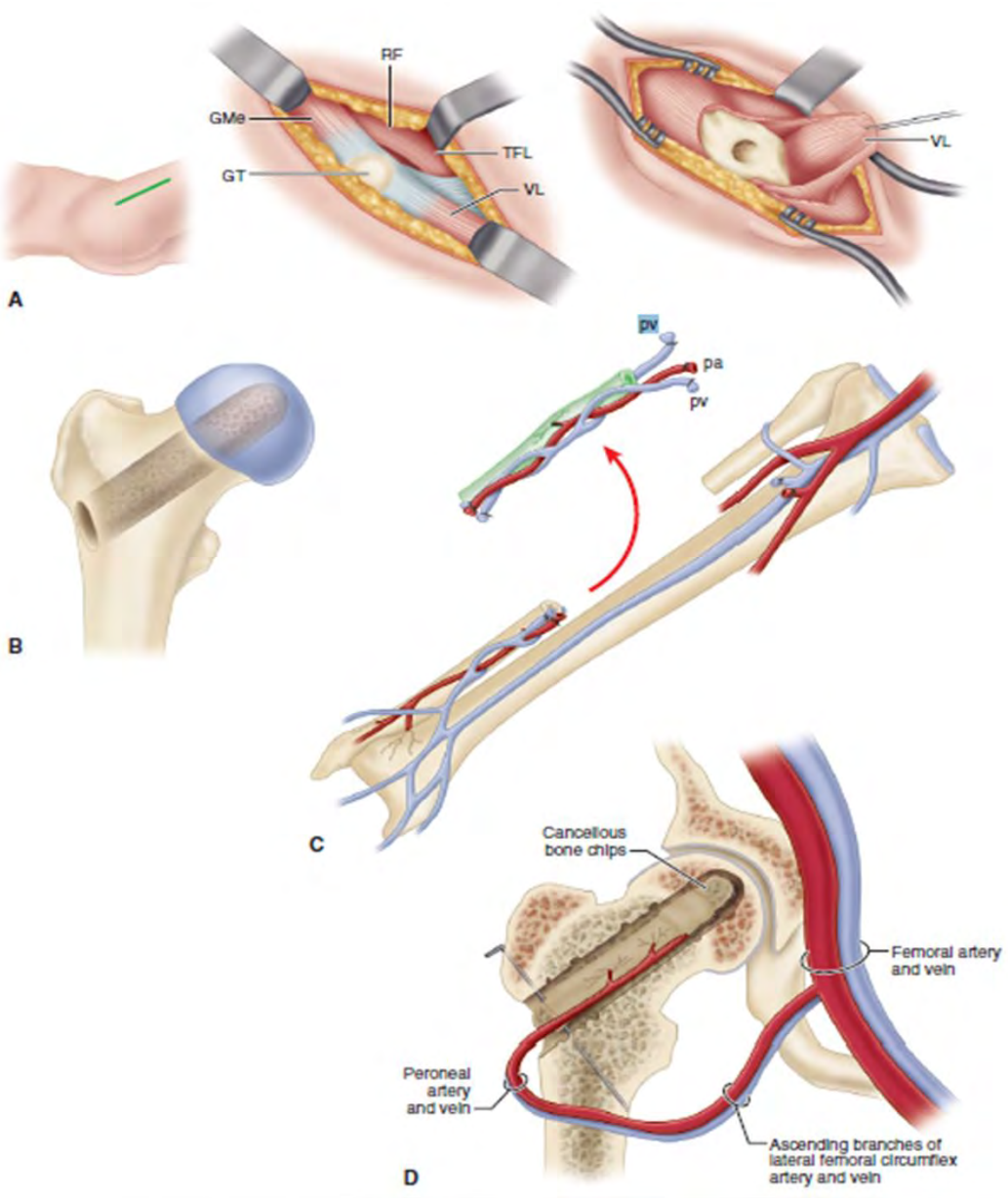
- Various authors have claimed success rate of 50 – 80% after CD with structural BG
- Non vascularised BG – Ficat 1 and 2
- Accurate placement of the graft within the lesion and under the subchondral bone
- Standard core technique, Lightbulb technique, Trap door technique

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

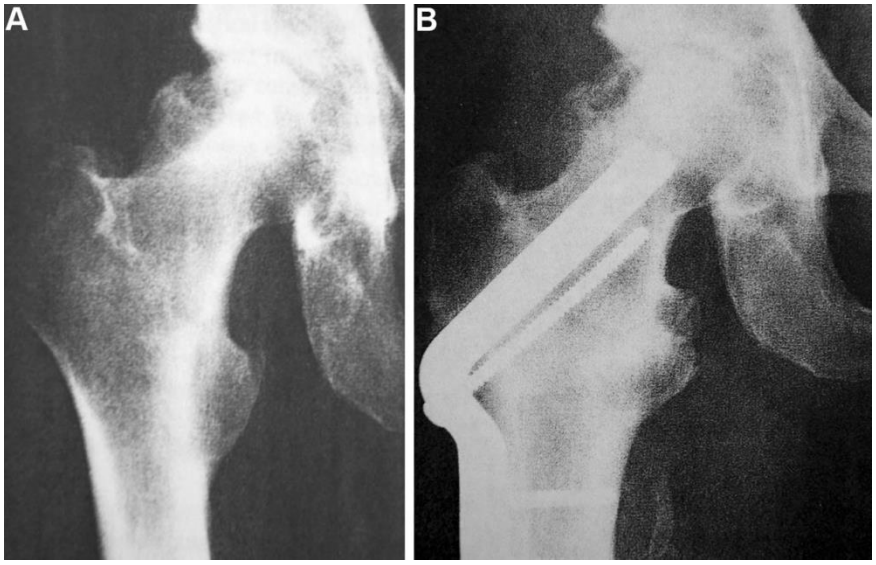

RF
GMe
TFL
GT
VL
A
VL
pv
pa
pv
B
C
Cancellous bone chips
Femoral artery and vein
Peroneal artery and vein
Ascending branches of lateral femoral circumflex artery and vein
D

Proximal femoral osteotomy

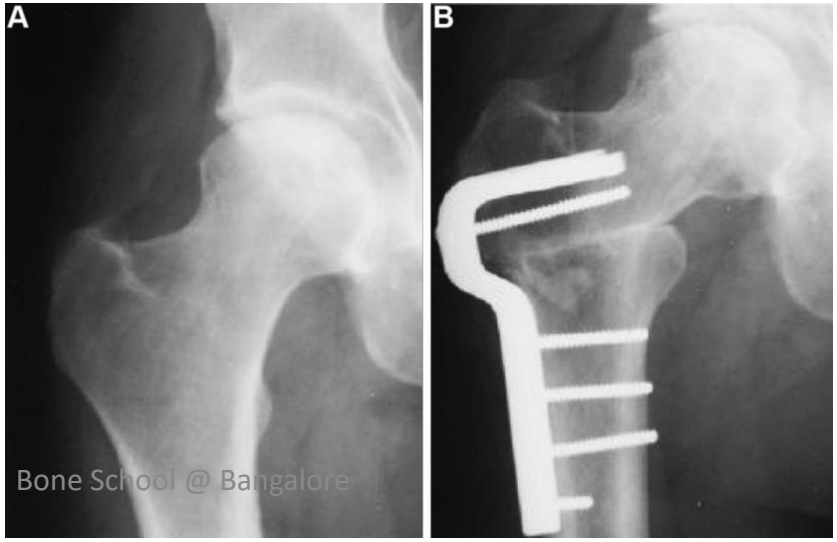
- Rationale – to move the necrotic segment of femoral head away from the weight bearing area

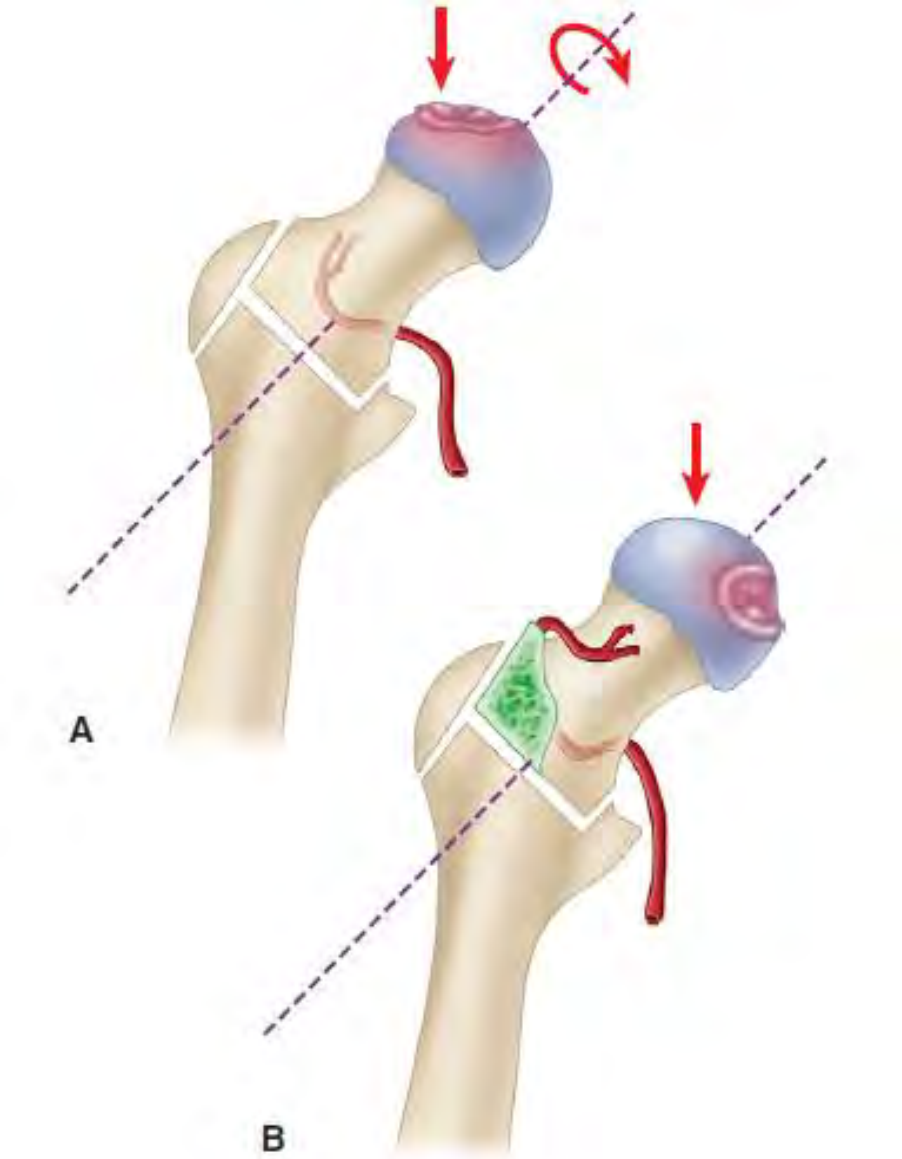
- Small and medium sized (<30% of head involvement/necrotic angle less than 200)

- Valgus/Flexion osteotomy – anterolateral lesion



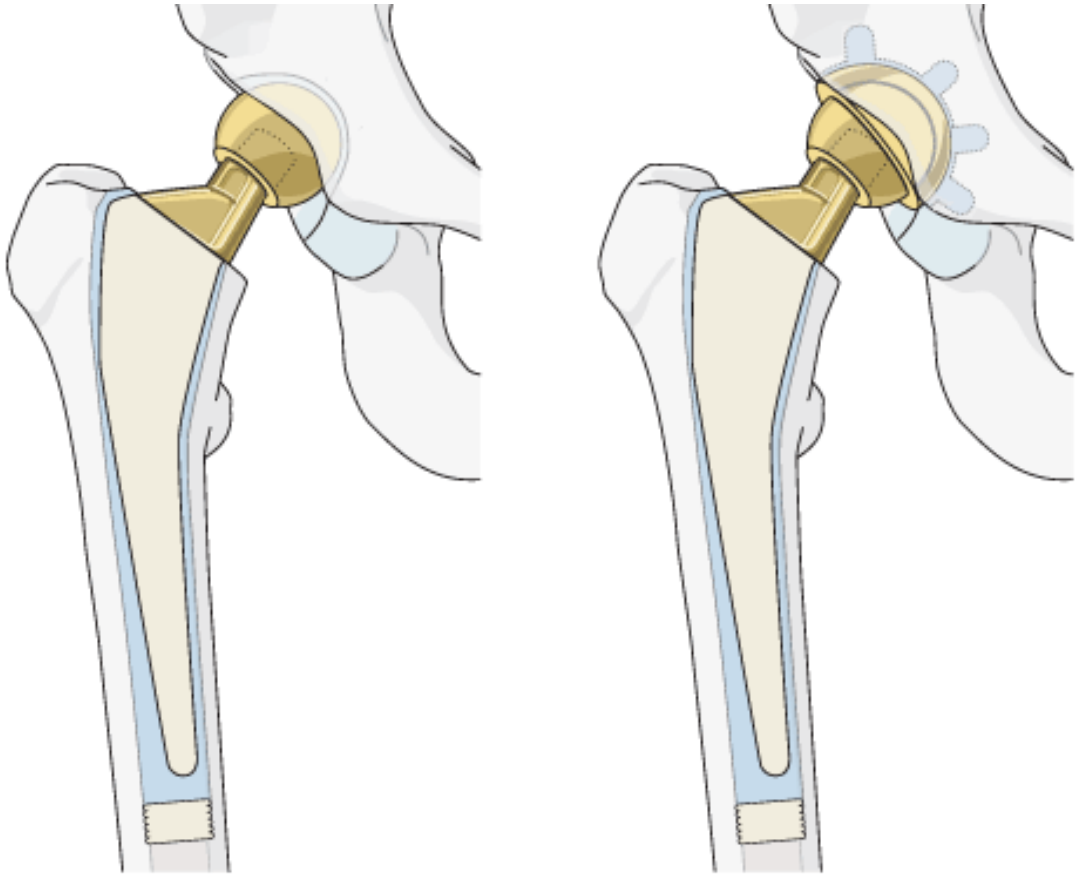
- Varus/Extension - Central or superomedial lesion





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