Avascular Necrosis

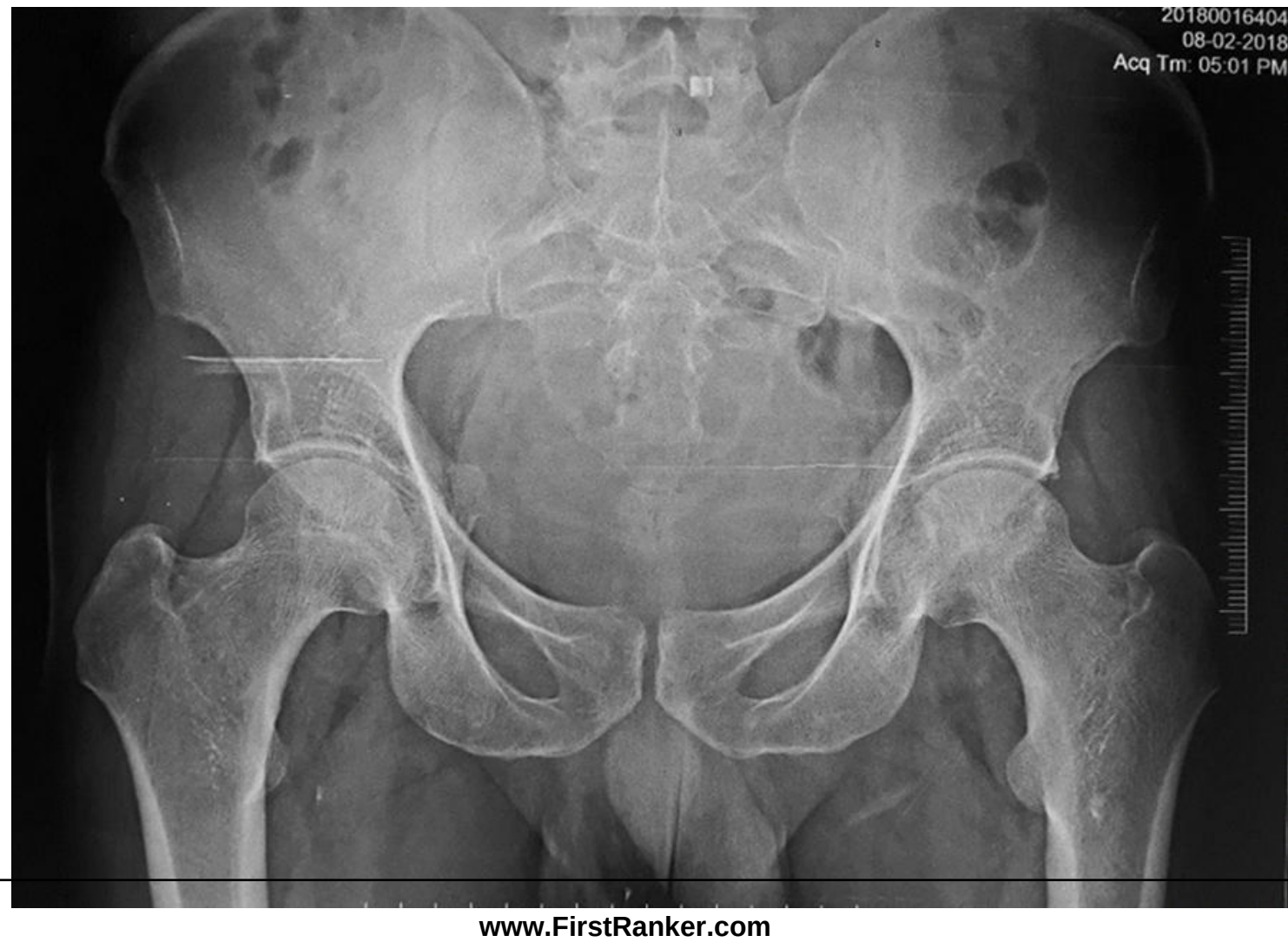
Learning Objectives

- Atraumatic /Traumatic AVN
- Understand the pathology of AVN
- Progression of disease
- Classification of Hip AVN
- Principles of treatment

27 years old ,pain right groin

- Non smoker, occasional alcohol intake, no medical history
- On examination.
  - No Tenderness
  - Restricted internal rotation ,painful
  - No limp
- CBC Normal, Rheumatoid factor –ve, ESR 18mm,CRP -6

Radiographs



Q1 Which of the following is an inappropriate diagnosis?

a) Early Sero-Negative Rheumatoid arthritis
b) Avascular necrosis right hip
c) Tubercular arthritis right hip
d) Torn acetabular labrum

Q 2 The most appropriate investigation for him is?

a) Tc 99 bone scan
b) MRI scan
c) MARS MRI
d) Contrast enhanced CT Scan

Q3 This is classifiable as Ficat-Arlet-

a) Stage 1
b) Stage 2
c) Stage 3
d) Stage 4

Q 4 The most appropriate treatment ?

a) Non operative with follow up MRI at 6 weeks
b) Non weight bearing and analgesics
c) Bed rest, traction and analgesics
d) Core decompression

Q 5 The patient comes after 2 years and has severe pain in his hip, decreased ROM and a pronounced limp. Radiographs reveal collapse of the head with decreased joint space suggestive of secondary OA. The appropriate management is-

a) Arthroscopic debridement and irrigation
b) Osteochondral grafting
c) Bipolar hip arthroplasty
d) Total hip arthroplasty

Overview

- AVN is a major cause of hip pain
- Traumatic/Atraumatic in etiology
- Evaluation is difficult
- Treatment is hip salvage/Replacement
- Other common areas are –Humerus, scaphoid, talus and distal femur

Avascular necrosis- Traumatic

- Femoral neck fractures- severance of the blood supply to the femoral head.
- The capitulum
- Femoral condyles
- Proximal parts of the scaphoid and talus.

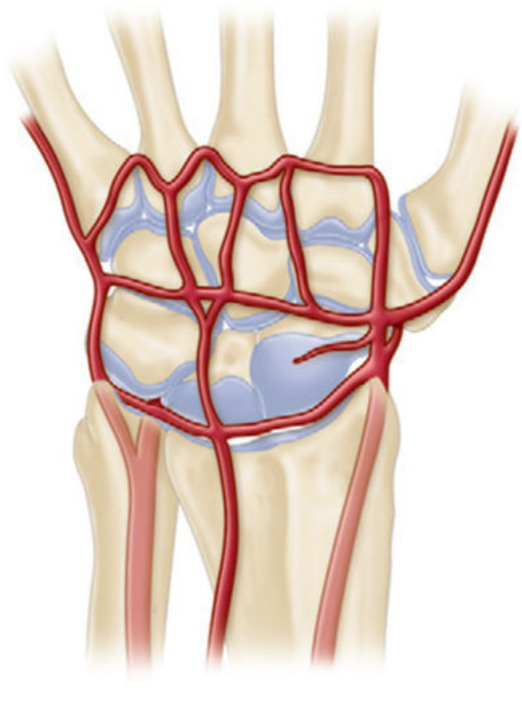
Distant parts of the bone's vascular territory

Largely enclosed by cartilage- restricted access to local blood vessels.

TRAUMATIC OSTEONECROSIS

- Fractures and dislocations of the hip
- Tear of retinacular vessels supplying the femoral head
- Displaced fractures of the femoral neck – AVN 20%.

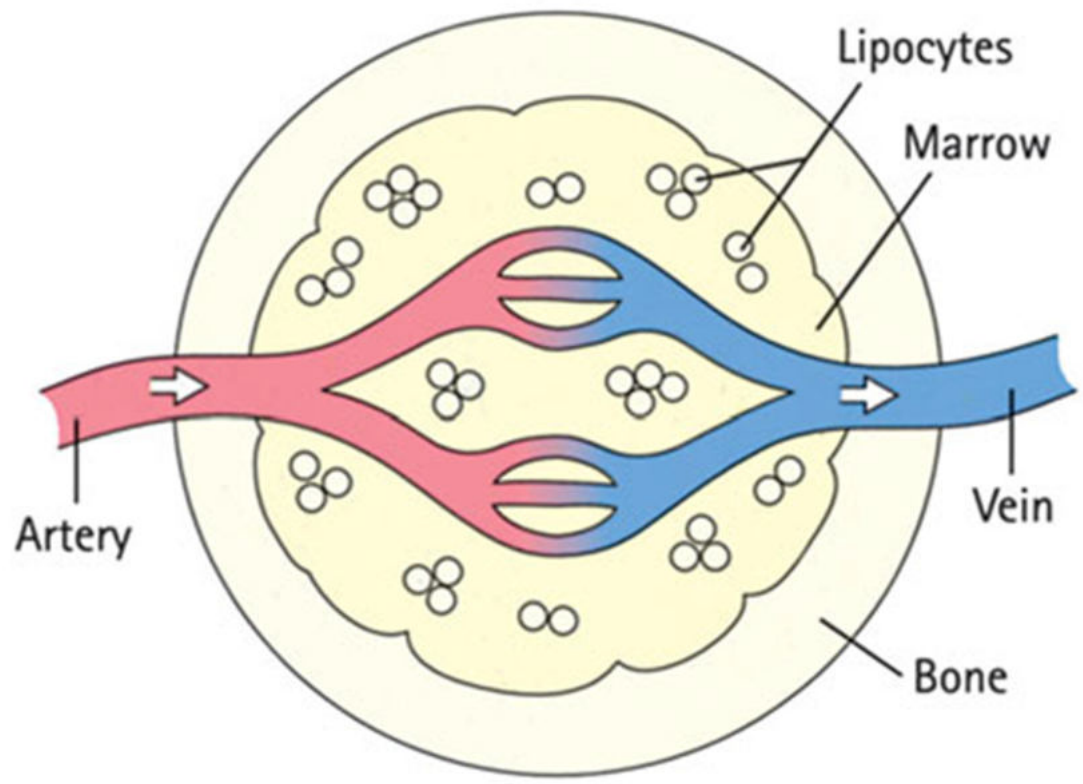
fractures of the scaphoid and talus- AVN



- Principal vessels enter their distal ends
- Intraosseous course from distal to proximal.

Closed compartment-Bone

- Vascular sinusoids- no adventitial layer
- Patency - volume and pressure of the marrow tissue
- Marrow is encased in unyielding bone.
- One element can expand-other gets compressed



NON-TRAUMATIC OSTEONECROSIS

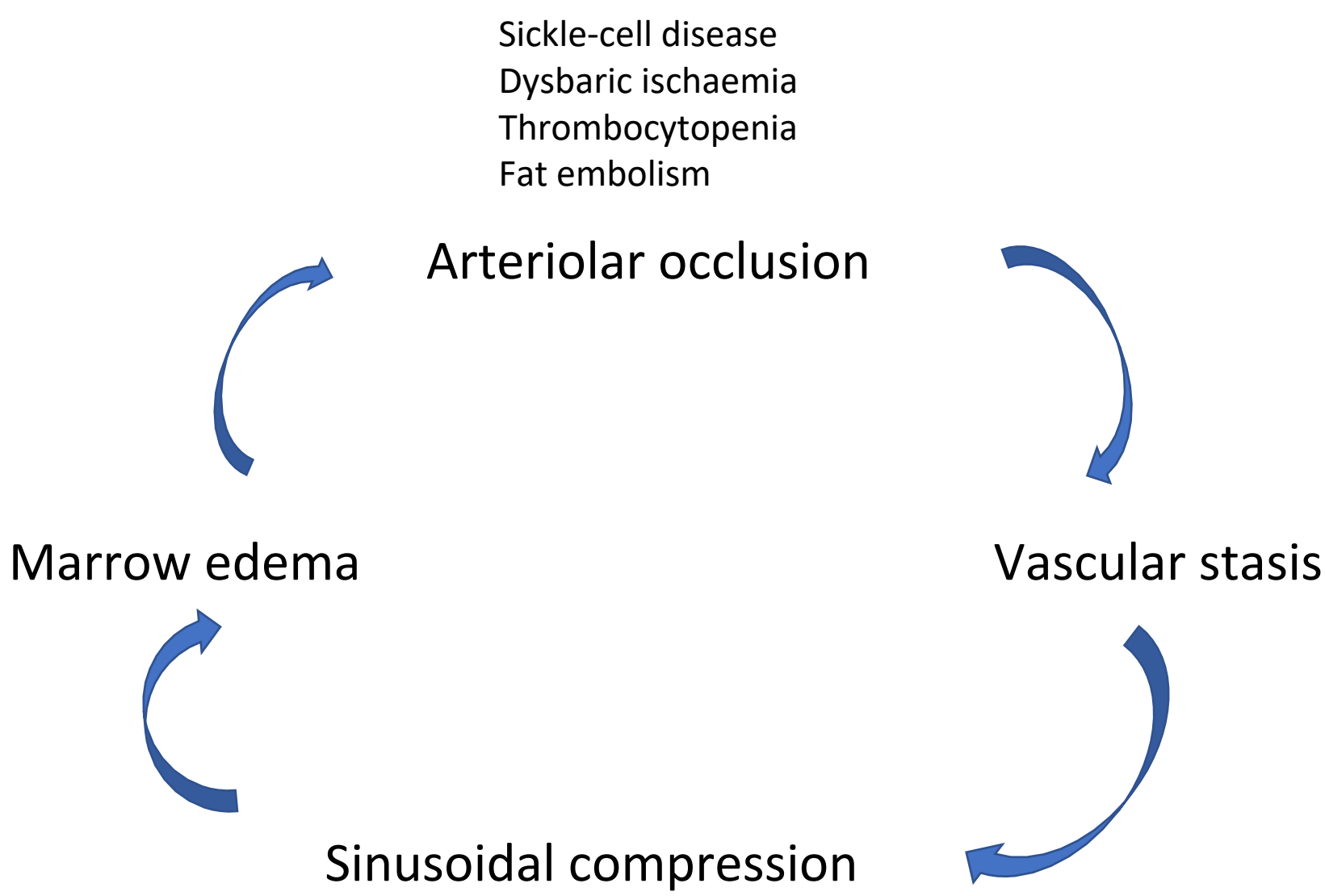
- Intravascular stasis
- Thrombosis
- Extravascular swelling and capillary compression.

Ischaemia- Multifactorial

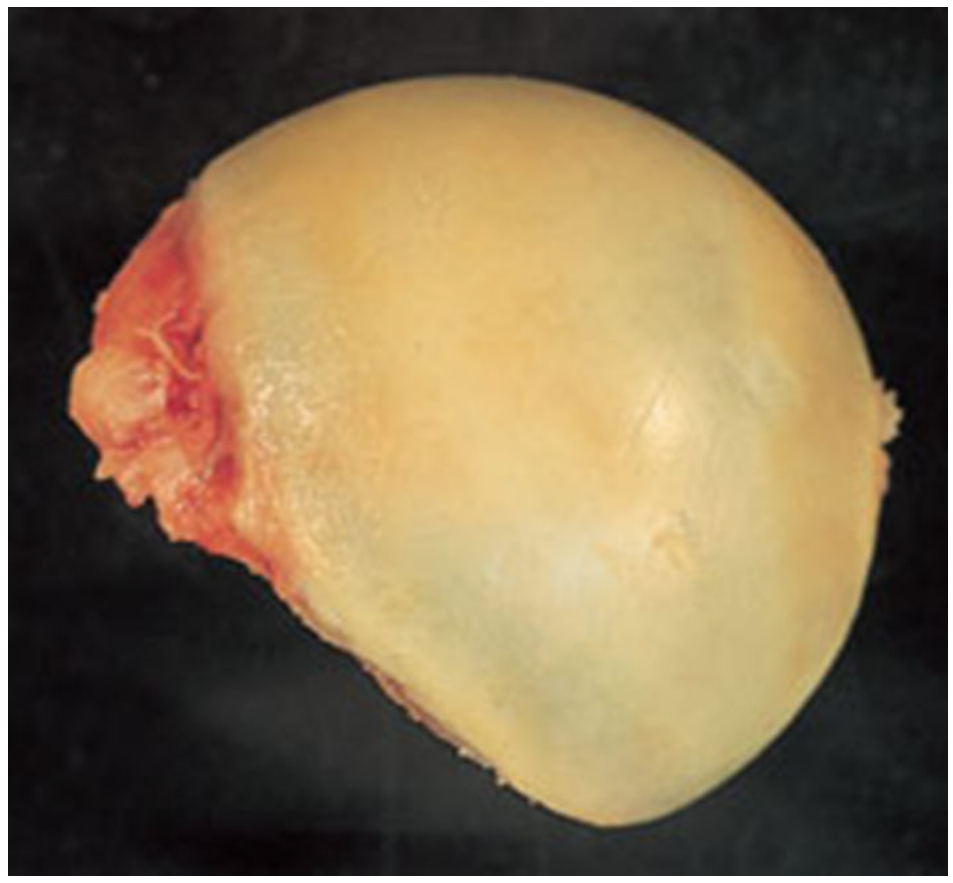
- Severance of the local blood supply
- Venous stasis and retrograde arteriolar stoppage
- Intravascular thrombosis
- Compression of capillaries and sinusoids by marrow swelling.

Nontraumatic -AVN

- Perthes' disease,
- Caisson disease
- Gaucher's disease
  systemic lupus erythematosus

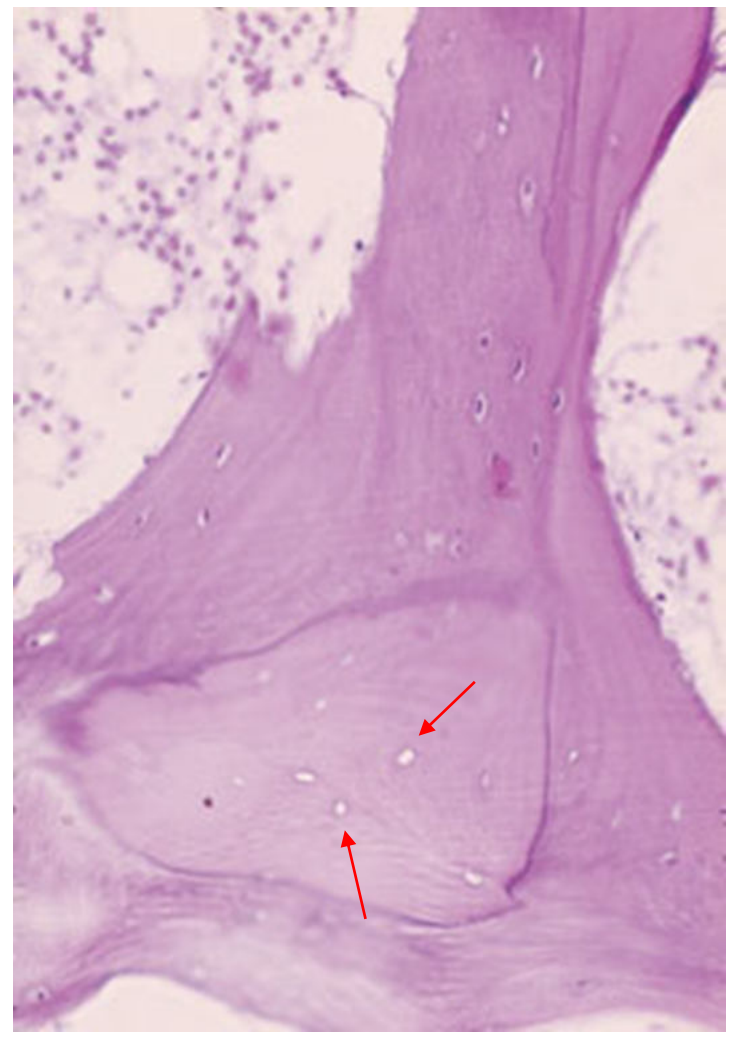


Normal head femur



Pathology and natural history

- Prolonged anoxia – Osteocyte death
- Gross appearance remains unaltered
- Striking histological changes in the marrow
  - Loss of fat cell outlines
  - Inflammatory cell infiltration
  - Marrow oedema
  - Replacement of necrotic marrow- mesenchymal tissue.



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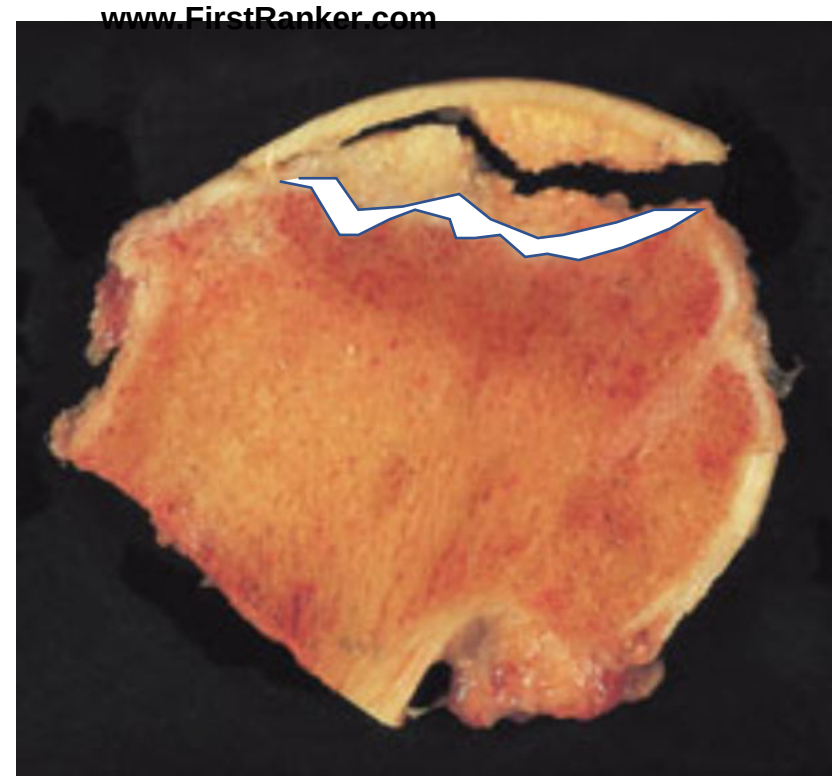
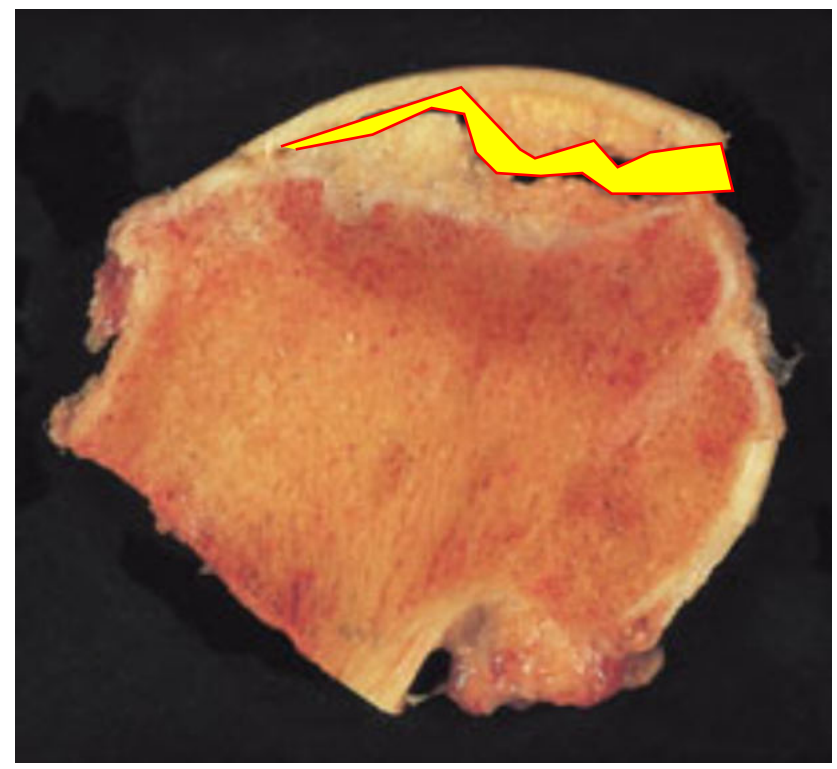
Bone repair?



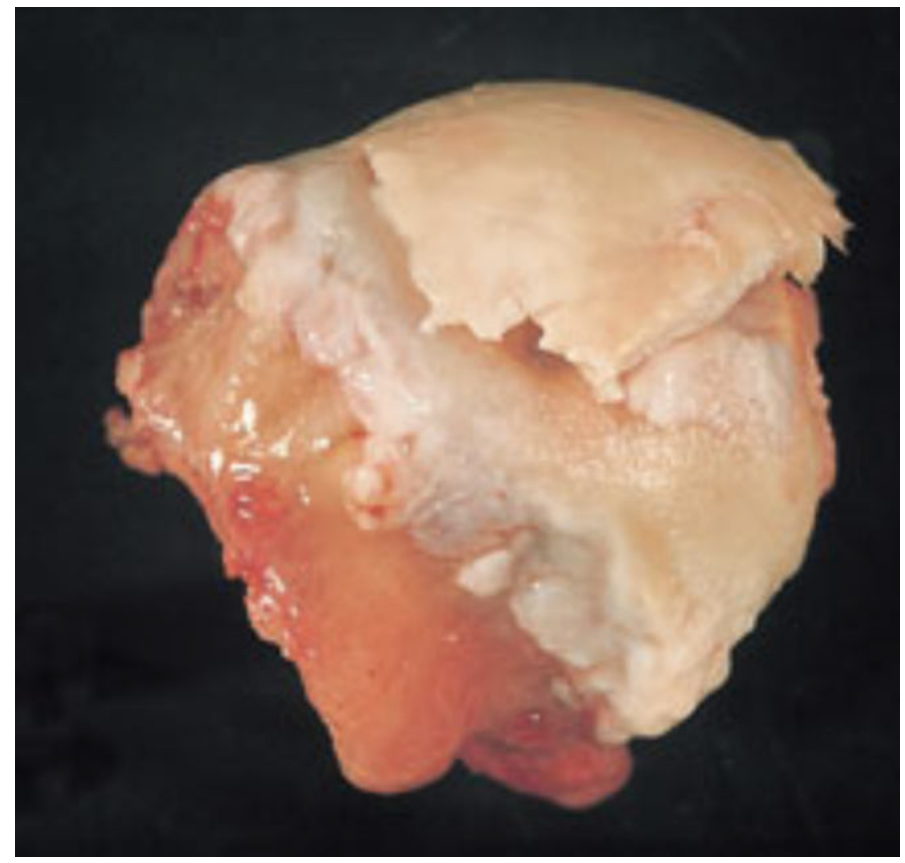
- New blood vessels and osteoblastic proliferation at the interface between ischaemic and live bone.
- Vascular granulation tissue advances from the surviving trabeculae
- New bone is laid down upon the dead- creeping substitution
- Increase in mineral mass - increased density or ‘sclerosis’.

Further progress



- Reparative formation proceeds slowly
- Advances 8–10 mm into the necrotic zone.
- Structural failure begins- most heavily stressed part of the necrotic segment.
- Linear tangential fracture close to the articular surface.

Stage of arthritis



- Articular cartilage retains its thickness and viability for a long time.
- In the final stages- fragmentation collapse of the necrotic bone
- Progressive deformity and destruction of the joint surface.

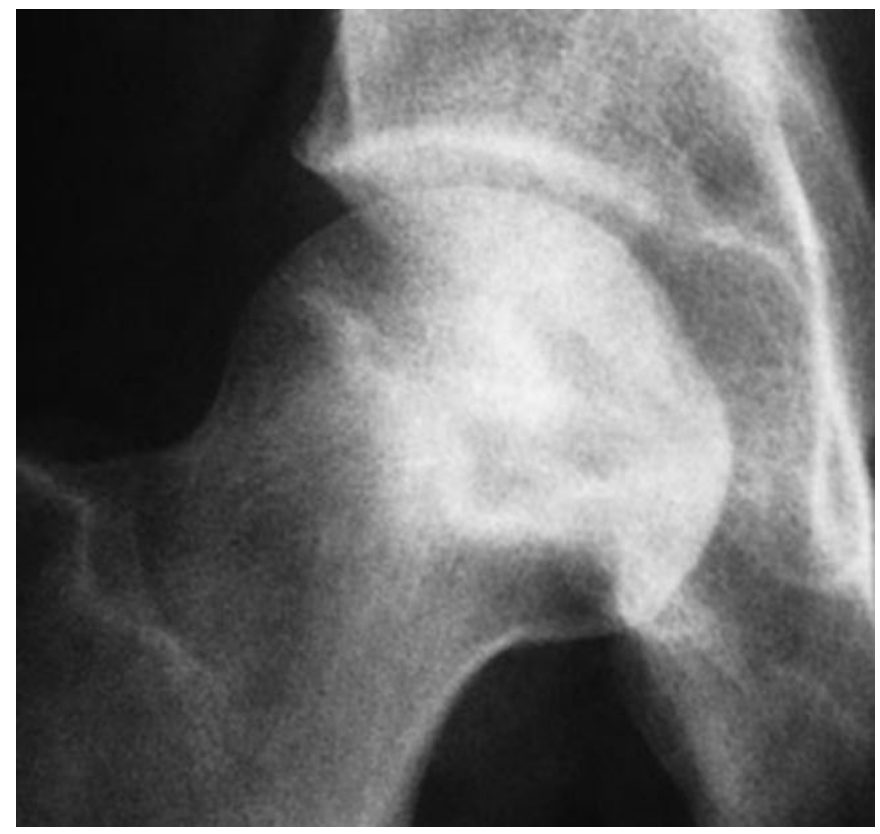
Clinical features

- The earliest stage of bone death is asymptomatic
- Patient presents with pain - lesion is usually well advanced.
- Pain in or near a joint
- Few complain of a ‘click’ in the joint- due to snapping or catching of a loose articular fragment.

Stage of arthritis

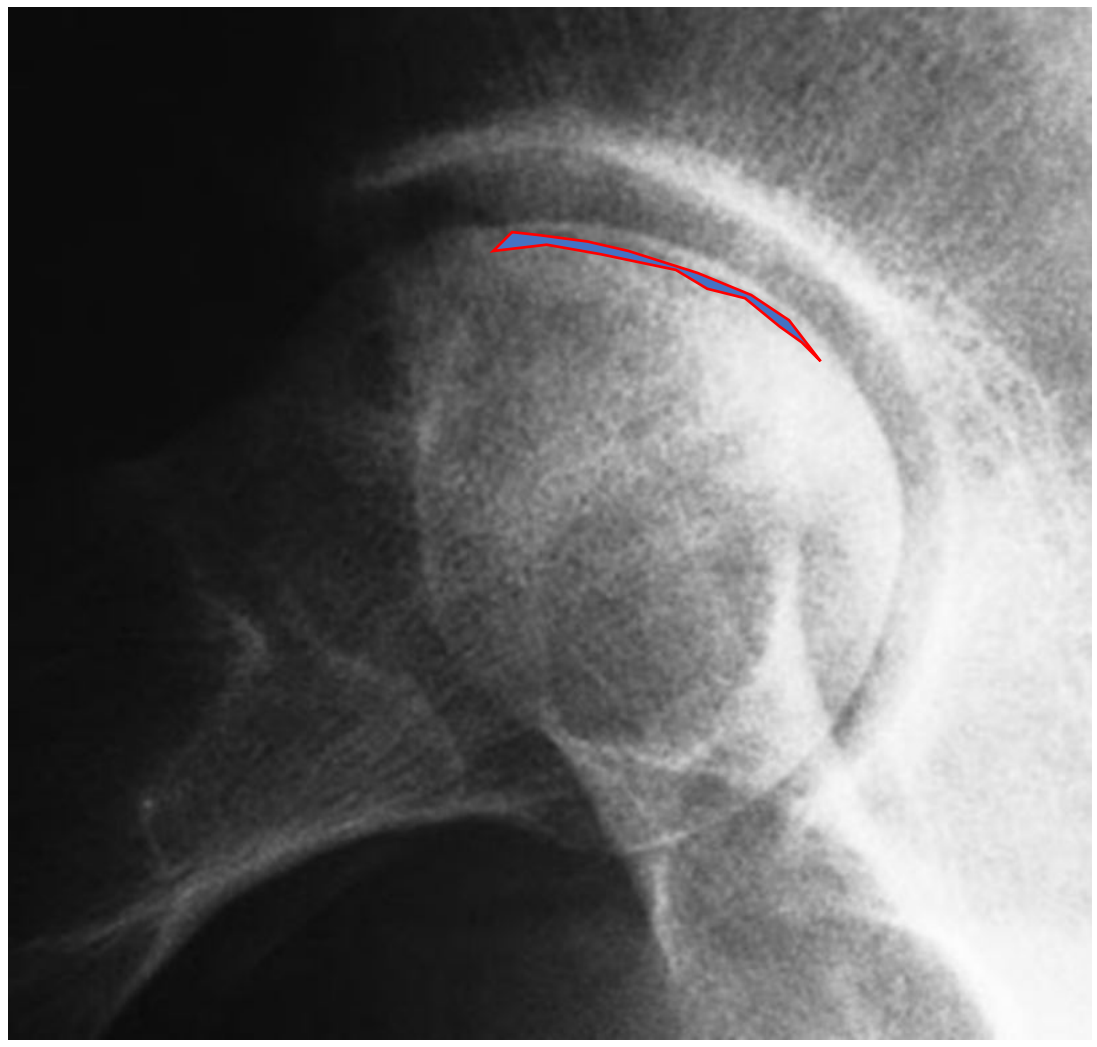
- Joint becomes stiff and deformed.
- Local tenderness may be present
- Superficial joints- effusion.
- Movements –may be restricted
- Advanced cases- fixed deformities.

Radiographs

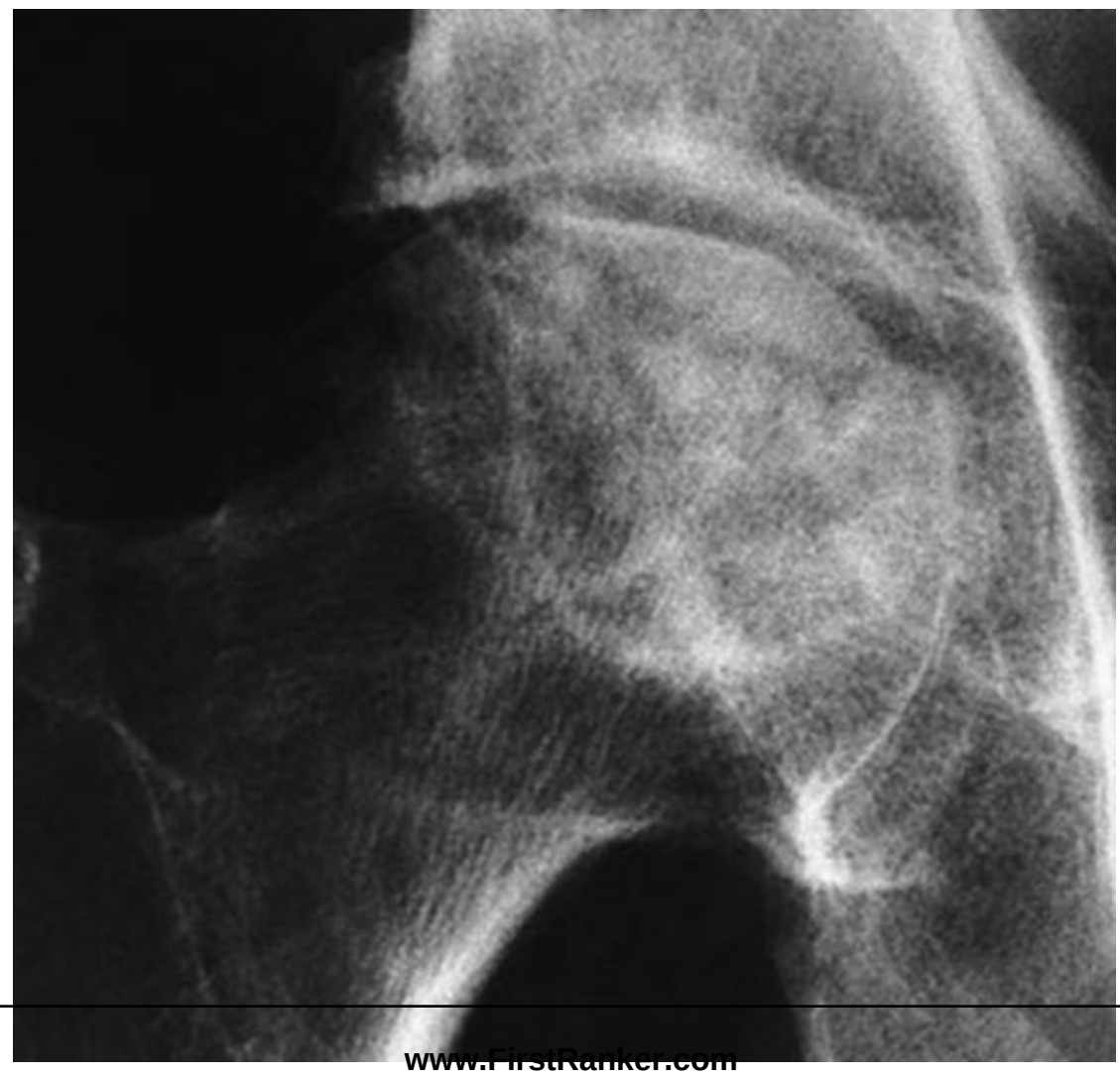


- The early signs of ischaemia -bone marrow and cannot be detected.
- 3 months after the onset of ischaemia- first sign
- Reactive new bone formation at the boundary of the ischaemic area - sclerosis

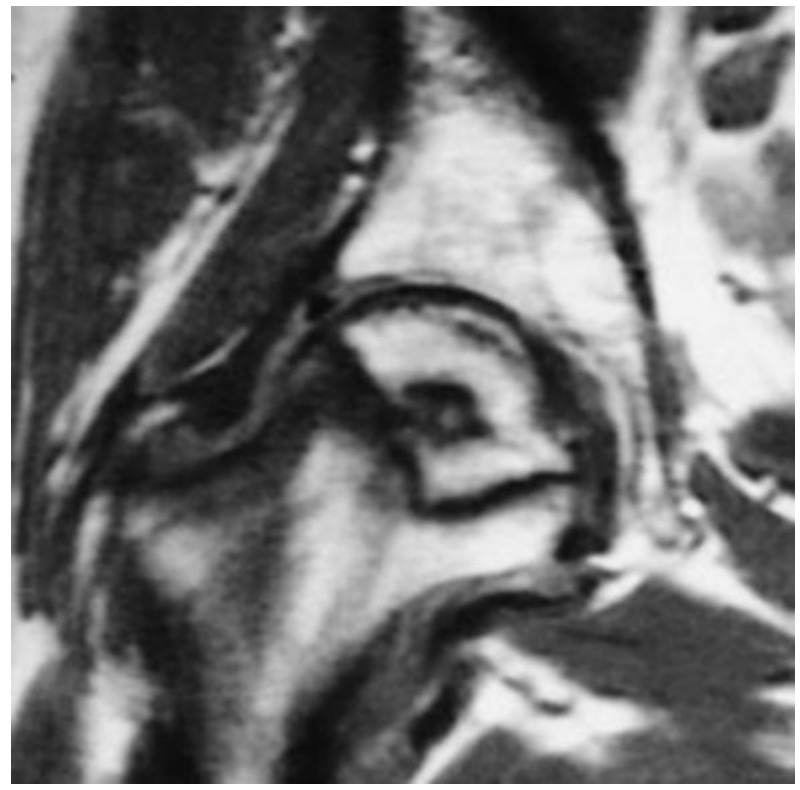
Thin tangential fracture line just below the articular surface –the *'crescent sign'*.



Late -collapse and distortion of the articular surface

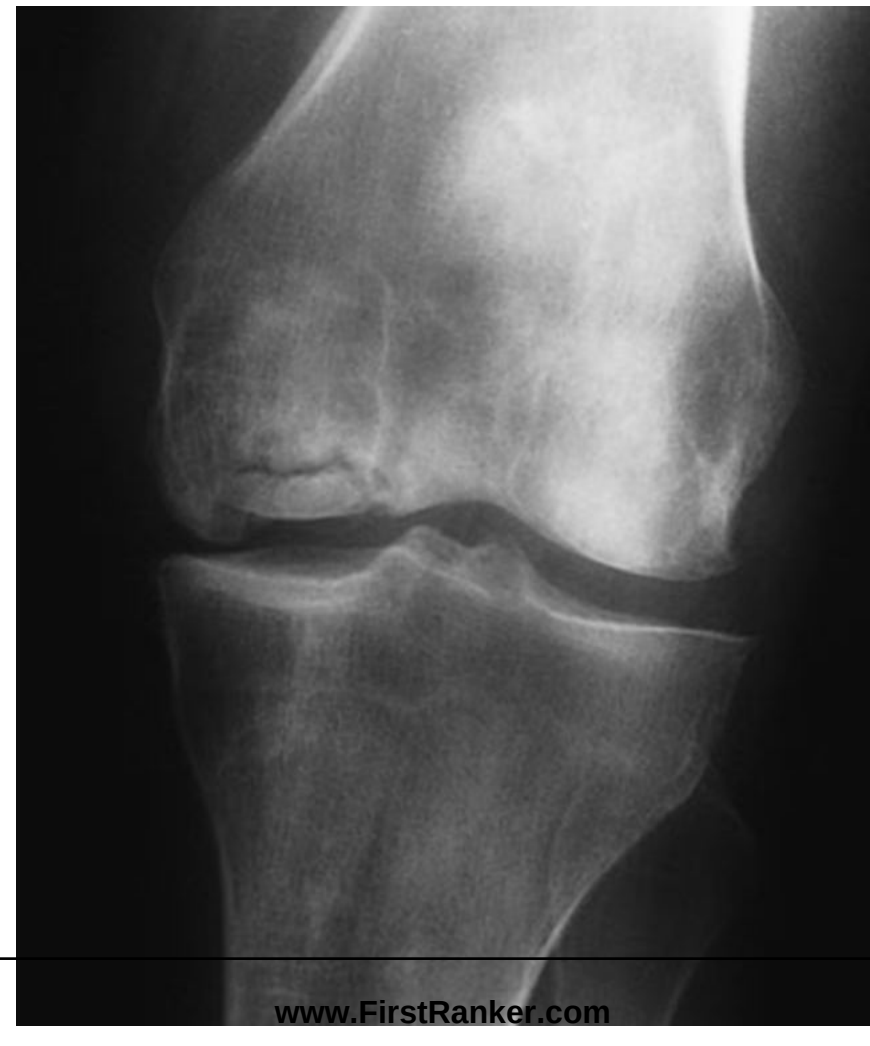


MRI Scan

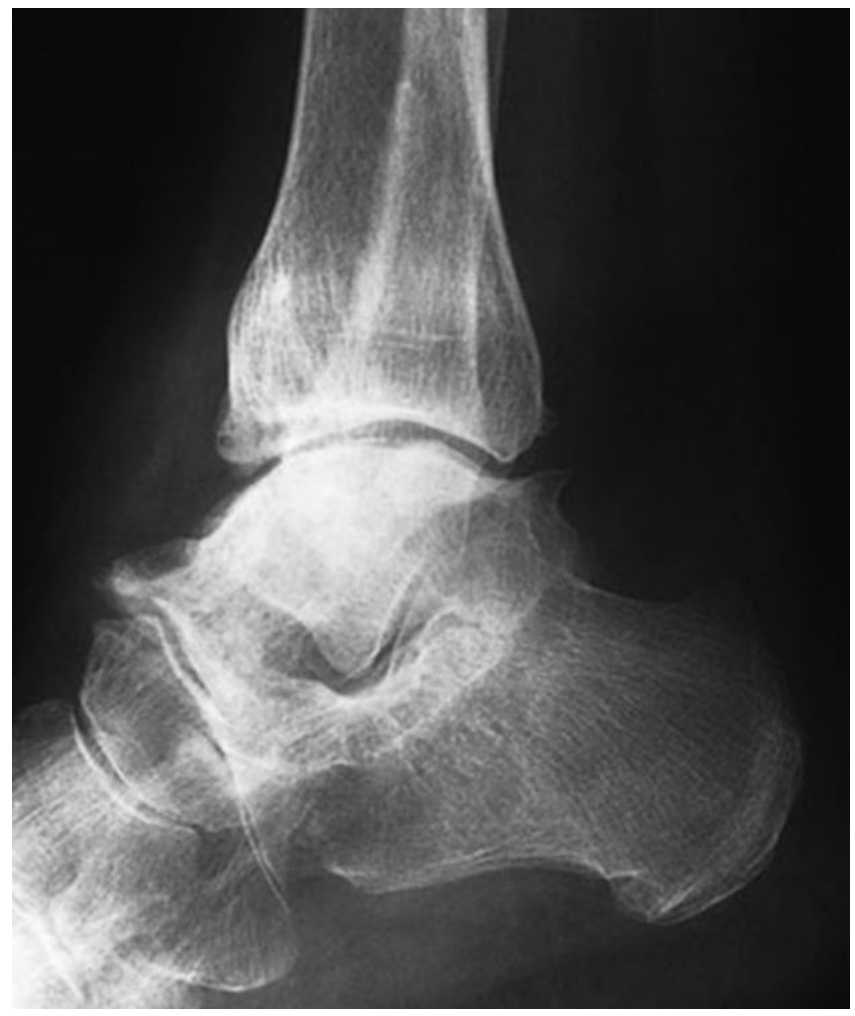


- The most sensitive modality – marrow changes are discernable
- The size of the necrotic segment-hypo-intense band in the T1,MRI

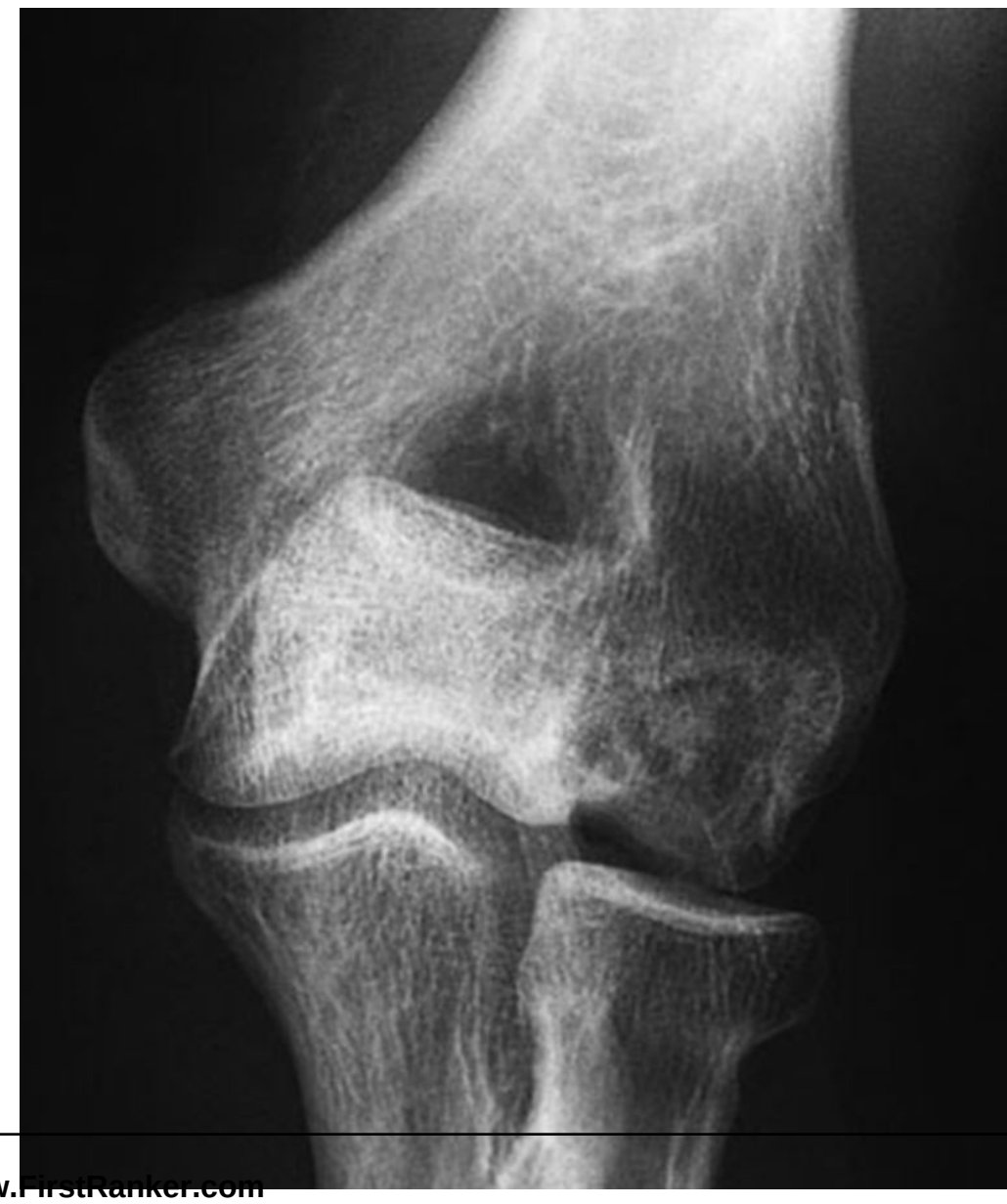
AVN –Distal femur



AVN Talus



AVN -Capitulum



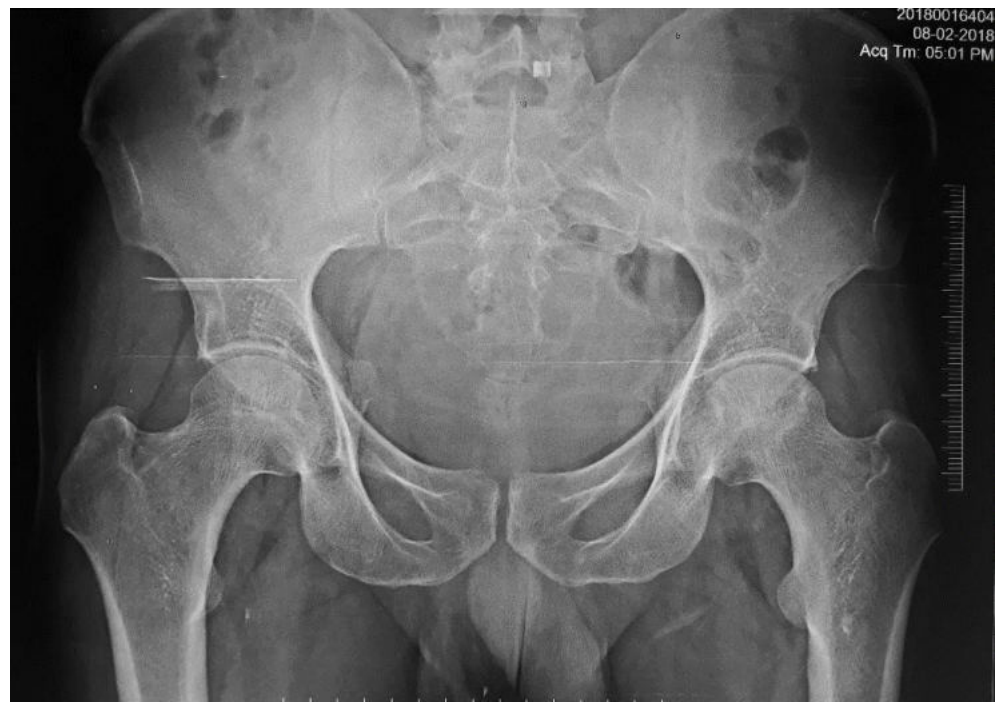
Radionuclide scanning

- 99mTcsulphur colloid- taken up in myeloid tissue.
- Useful in traumatic avascular necrosis- large segment of bone is involved.
- Sickle-cell disease - ‘cold’ area contrasts significantly with the generally high nuclide uptake due to increased erythroblastic activity.

Staging the lesion

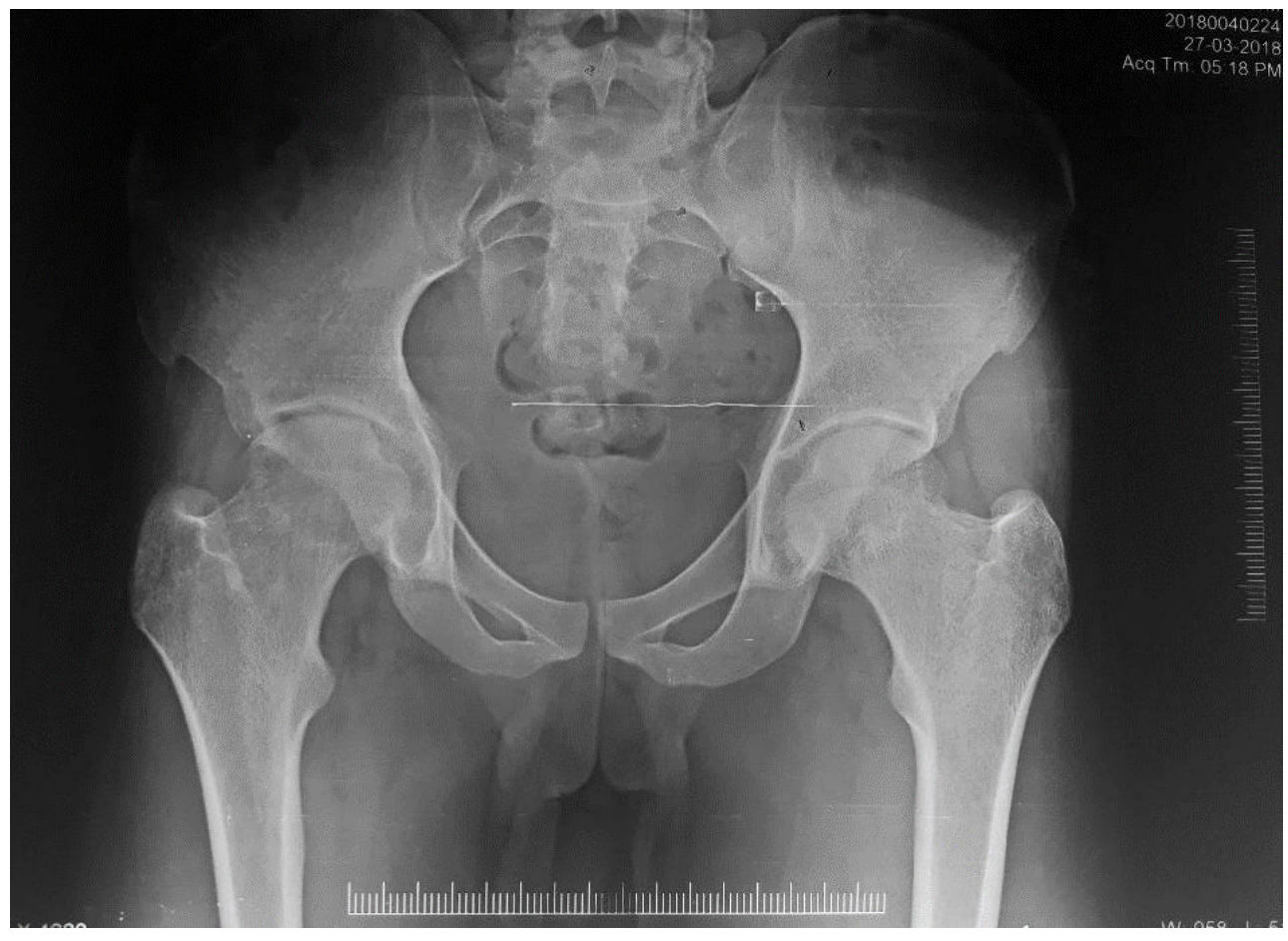
- Ficat and Arlet (1980) introduced the concept of *radiographic staging* for osteonecrosis of the hip
- Early (pre-symptomatic) signs- sclerosis, crescent sign.
- Later features- progressive demarcation and collapse of the necrotic segment in the femoral head.

Stage 1 – No radiological changes



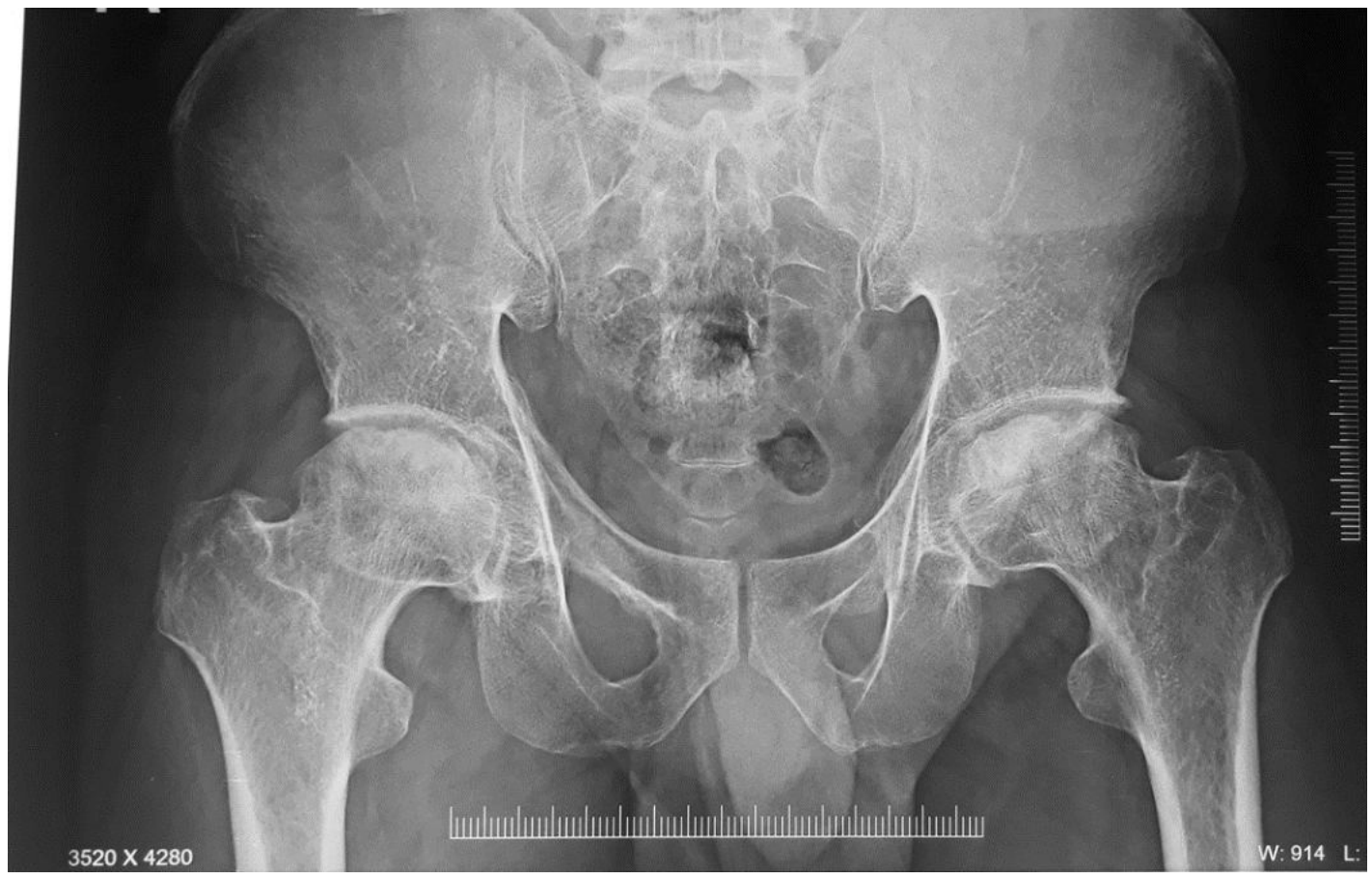
- Diagnosis was based on measurement of raised intraosseous pressure
- Histological features of bone biopsy
- MRI

Stage II



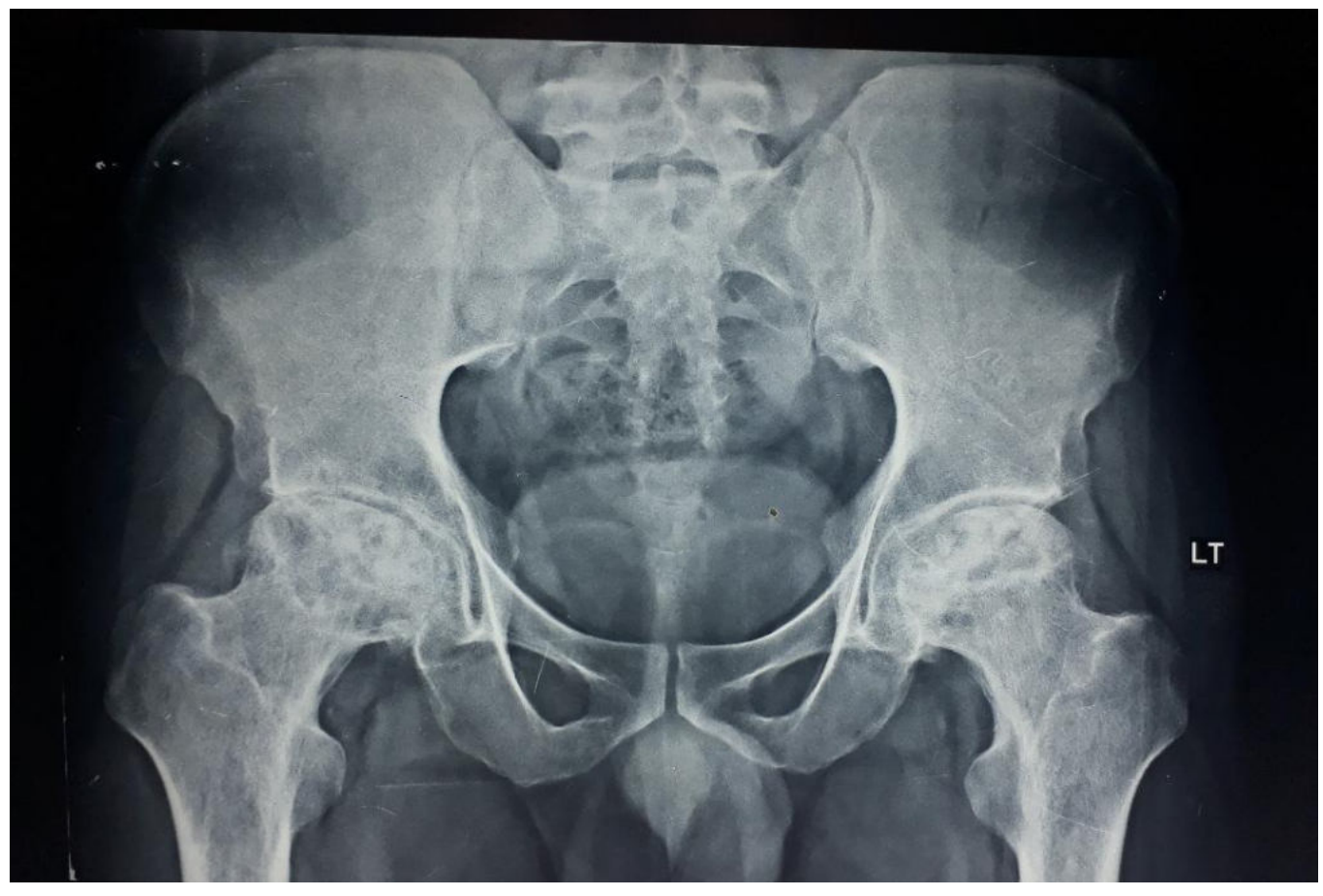
- The femoral head contour was still normal
- Early signs of reactive change in the subchondral area

Stage 3



- Signs of osteonecrosis with evidence of structural damage and distortion of the bone outline.
- Collapse of the necrotic segment

Stage 4



- Collapse of the articular surface and signs of secondary OA.

Diagnosis of the underlying disorder

- Episode of trauma- obvious
- Occupation- deep-sea diving or working under compressed air
- Family background of Gaucher's disease or sickle-cell disease.
- High-dosage corticosteroid administration; renal transplantation.
- Low dose use –quacks, inappropriate use
- Alcohol abuse is often difficult to determine
- SLE- antiphospholipid antibodies may be measured.

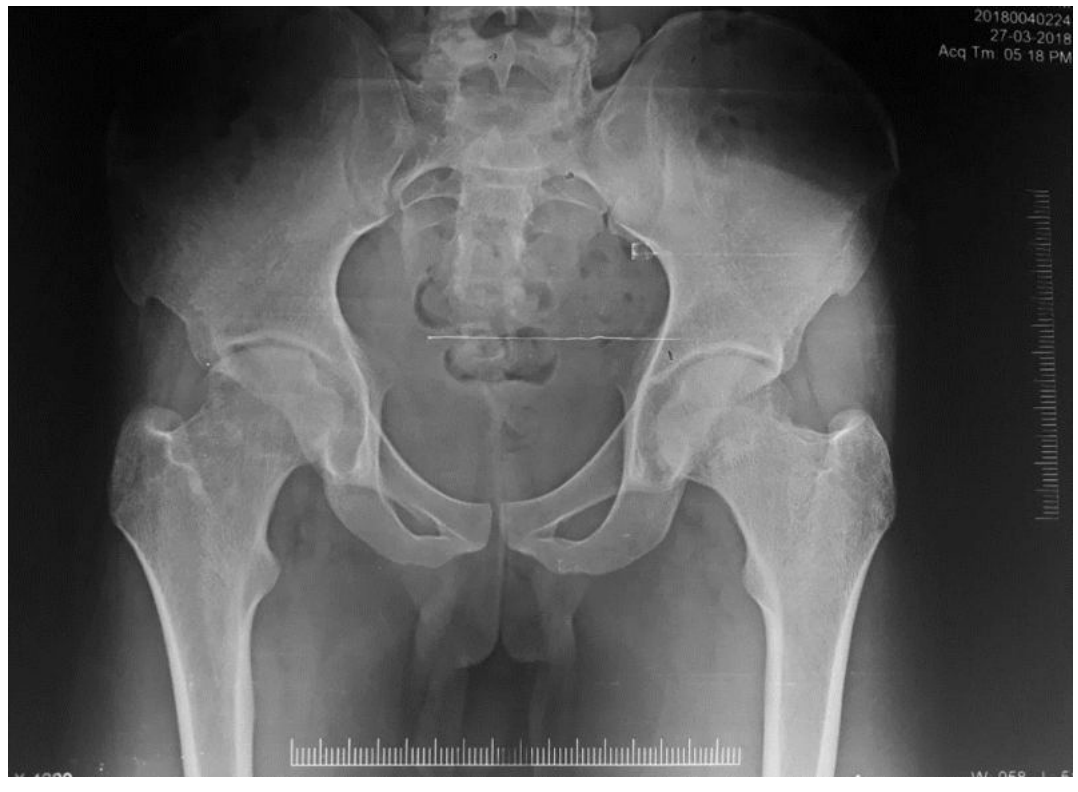
EARLY OSTEONECROSIS

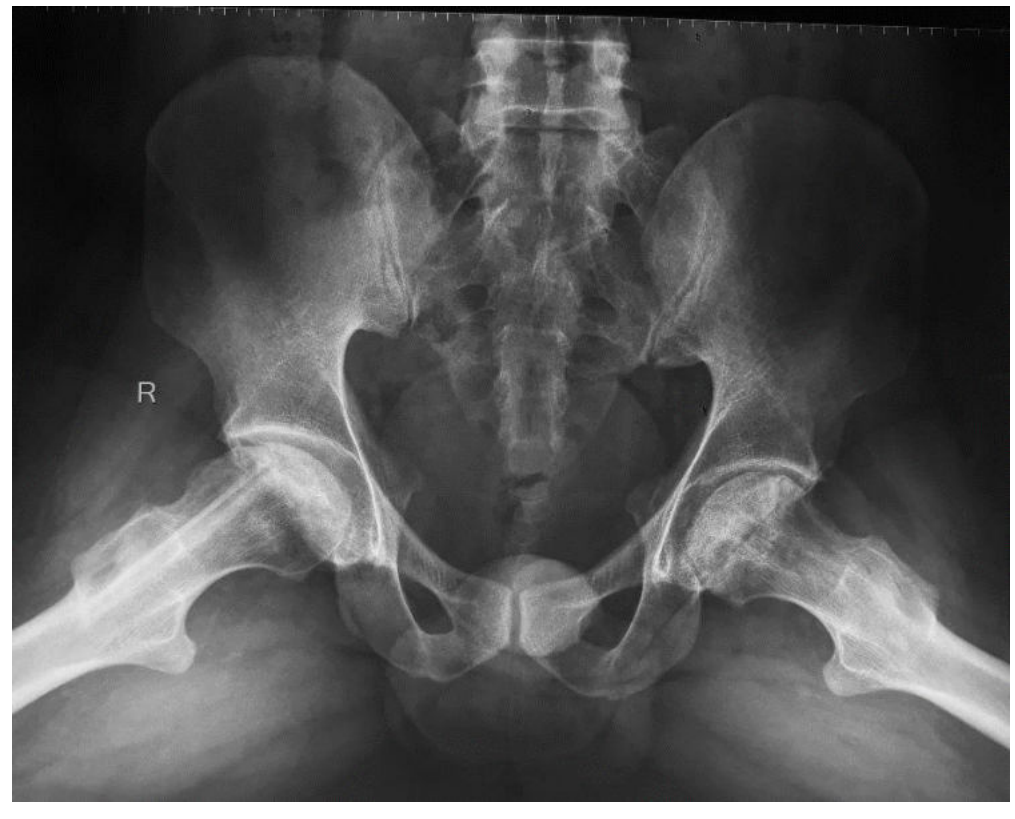
- Bone contour is intact- structural failure can be prevented.
- Some lesions heal spontaneously and with minimal deformity;
  - Non-weightbearing joints
  - Superomedial part of the femoral head
  - Non- weight bearing surfaces of the femoral condyles and talus.

Weight bearing joints

- Poor prognosis-l probably end in structural failure
- Simple measures like non weight bearing- reduce loading.
- If the bone contour is still intact,an ‘unloading’ osteotomy
- Help to preserve the anatomy while remodelling proceeds.
- Medullary decompression and bone grafting may have a place

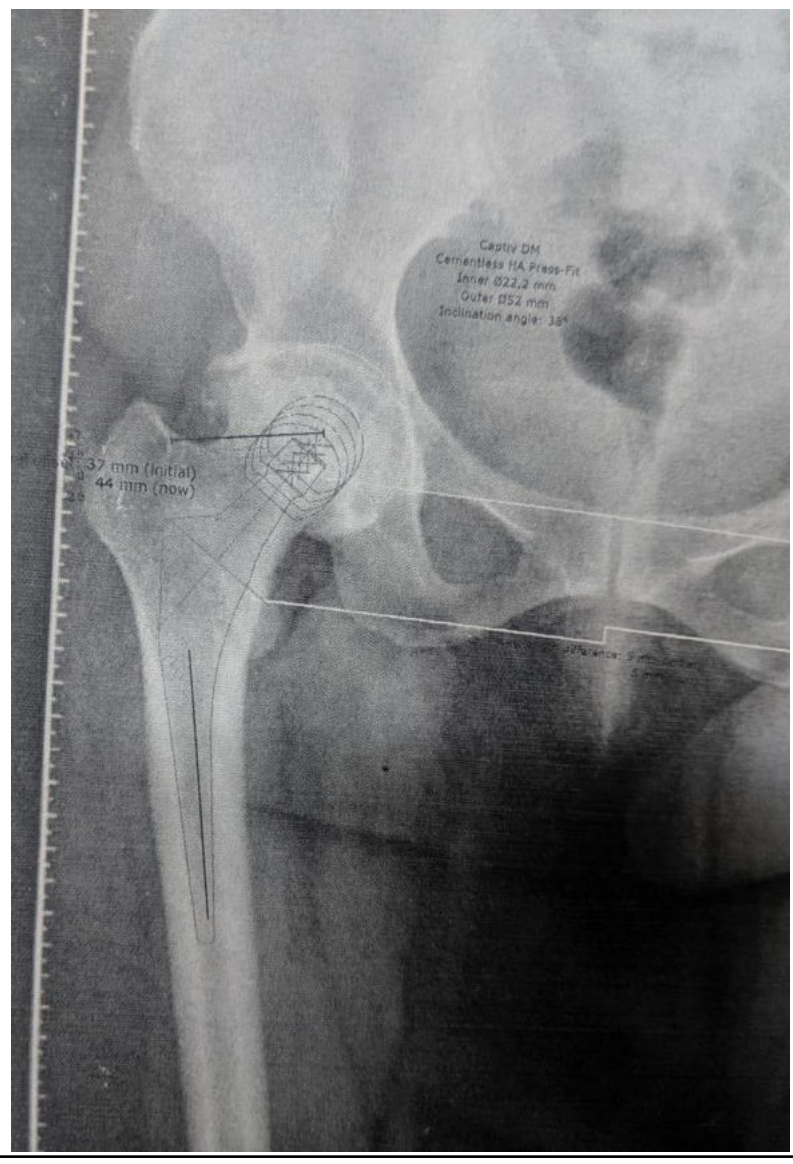
Stage II- Core decompression B/L

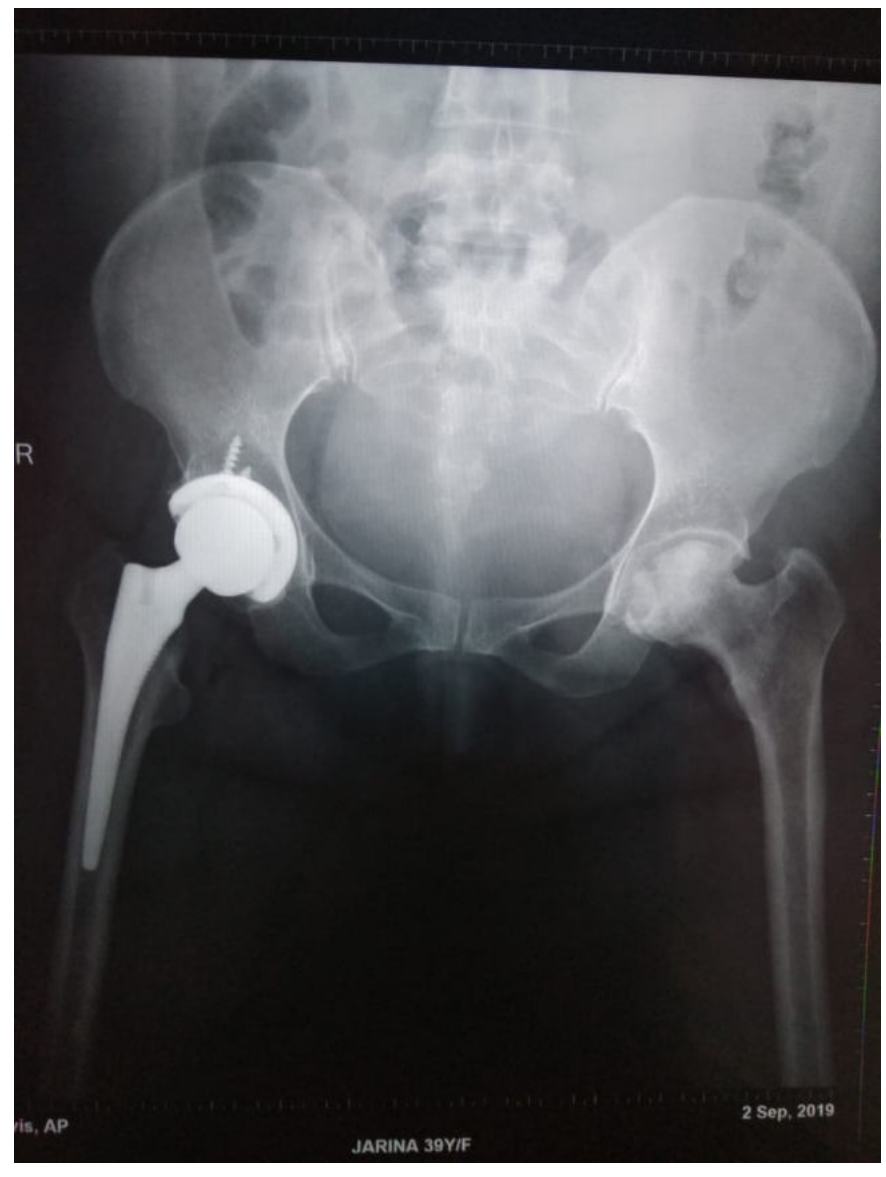




LATE STAGE OSTEONECROSIS

- Destruction of the articular surface may be give rise to pain and severe loss of function.
- non-operative management, concentrating on pain control, modification of daily activities and appropriate, splintage of the joint
- Arthrodesis of the joint, e.g. the ankle or wrist
- Total joint replacement- shoulder, hip and knee.





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Conclusion

- AVN is difficult to diagnose early- High degree of suspicion
- Radiographs in early stages are normal- Trust your findings more!
- Best modality for early diagnosis –MRI
- Salvage can be tried for Stage I,II
- Advanced stages require – Total hip arthroplasty