Osteonecrosis

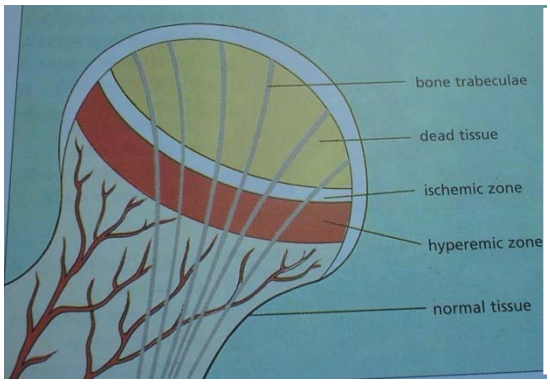
Osteonecrosis, or bone death, occurs as a result of either impaired blood supply (eg. due to trauma) or severe marrow and bone cell damage.

The hip joint is commonly affected, causing eventual collapse and flattening of the femoral head. Other susceptible sites include the femoral condyles, head of humerus, capitulum, scaphoid, lunate and talus.

In majority the aetiology of osteonecrosis is uncertain, but various factors have been implicated.

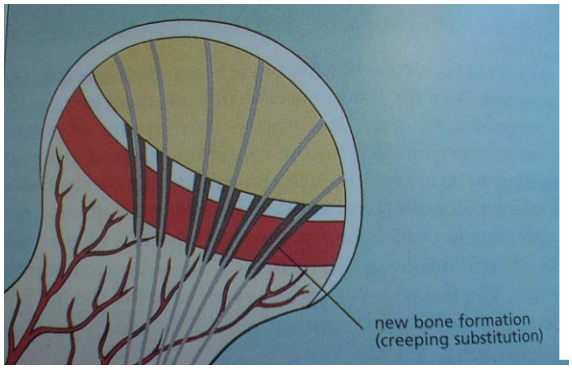
It is associated with steroids and heavy alcohol consumption (both causing fatty infiltration capillary compression), and also with blood dyscrasias (such as Sickle-cell Disease), decompression sickness (Caisson Disease), vasculitis, excessive radiation therapy, and Gaucher's Disease (abnormal accumulation of glucocerebride in the reticuloendothelial system causes pressure on bone sinusoids, thus necrosis). In majority. It is idiopathic.

b) Pathologic Changes: 4 stages



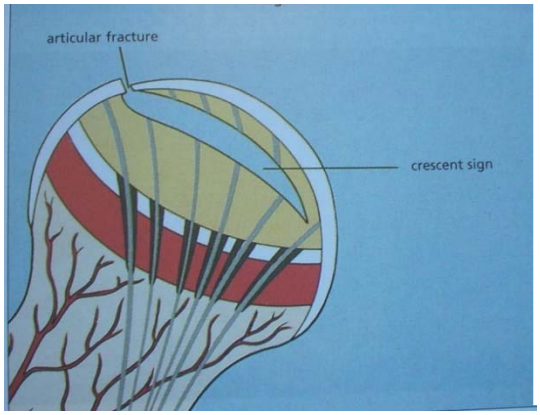
Stage I

- Viable articular cartilage
- Ischaemic zone
- Osteocytic death in the lacunae
- X ray: normal



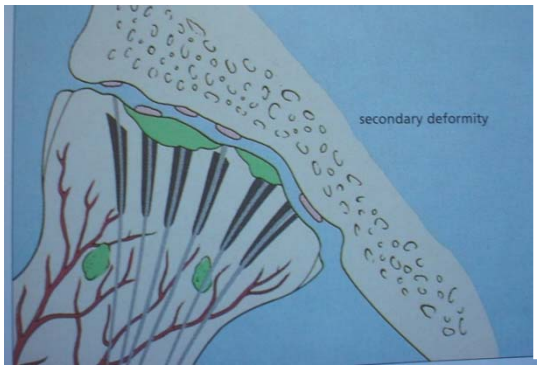
Stage 2:

- Inflammation occurs, with a vascular reaction.
- New bone is laid down upon the dead trabeculae [Creeping substitute]



Stage 3

- Resorption of necrotic trabeculae
- The bone is weakest during this phase
- Crescent sign and collapse
  [Stress at thick (creep) and necrotic junction]
- Fragmentation may occur.



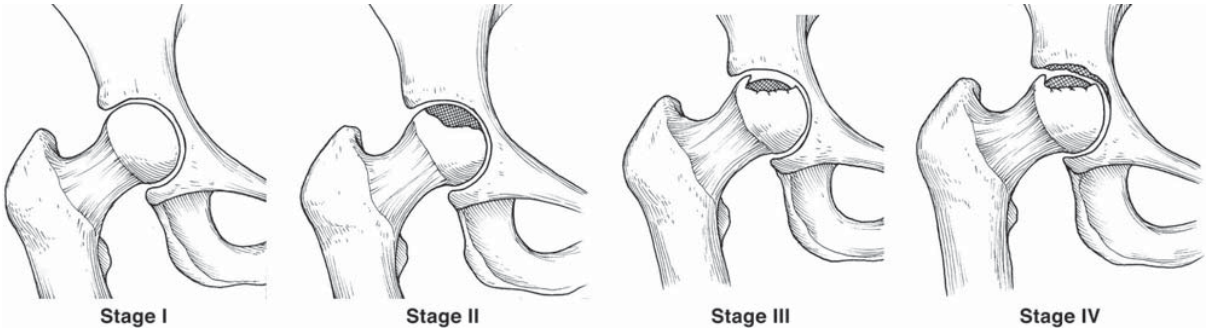
Stage 4

- Articular destruction
- However, severe distortion of the surface eventually results in cartilage destruction.

c) **Evaluation**
Detailed history-taking
In 50% of cases of idiopathic osteonecrosis
Physical examination: range of movement, pain, stiffness

Classification

1. **Ficat's**



II Steinburg's classification

- same as above for stages 0-3
- Stage III Subchondral fracture
- Stage IV Flattening of the femoral head
- Stage V Joint narrowing with or without acetabular involvement
- Stage VI Advanced degenerative changes

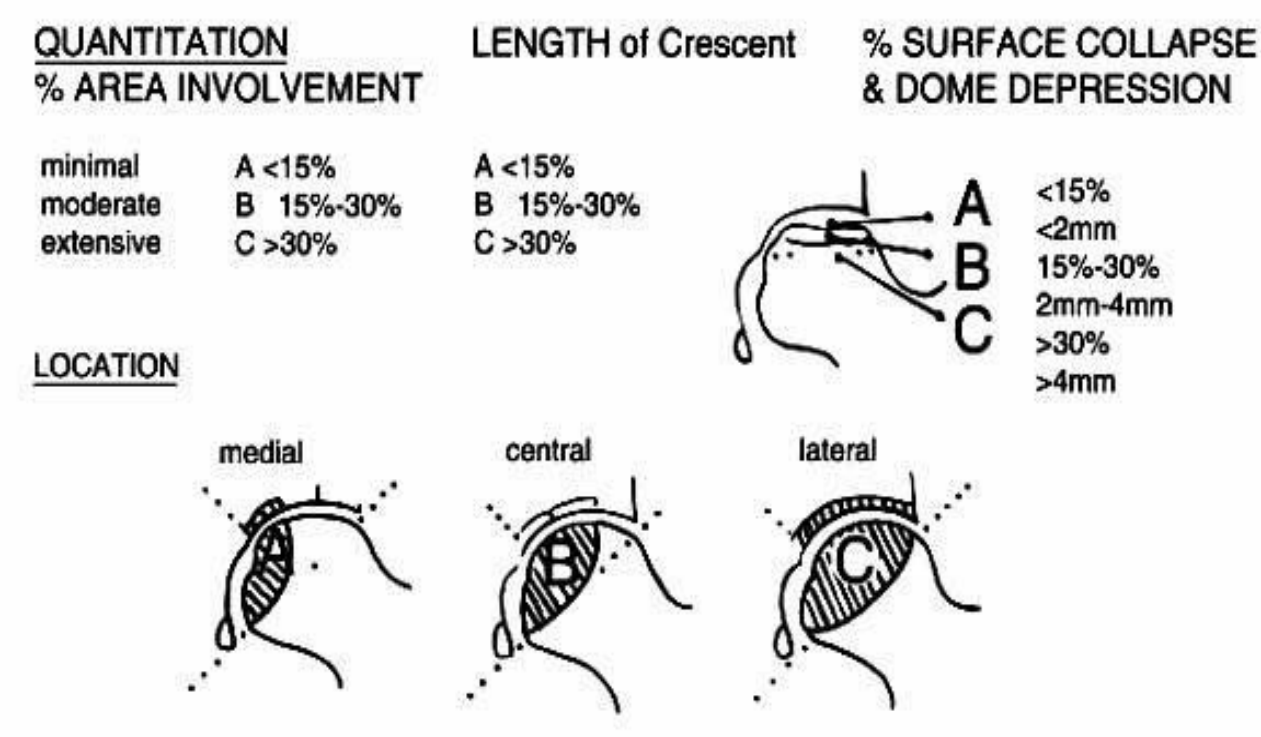
These stages are further divided into mild (A), moderate (B) or severe (C)

III Association Research Circulation Osseous [ARCO] modification of Ficat Staging

Stage	C/F	X ray	Hemodynamic	B Scan	Biopsy
0	Preclinical	0	+	Decreased	Only histo
I	PreXray	0	++	Increased +	+
II	Stage of AVN	Sclerosis or cysts	++	+	+
III	Collapse	Crescent Flattening NO OA	+	+	+
IV	OA	OA	+	+	+ with arthritis

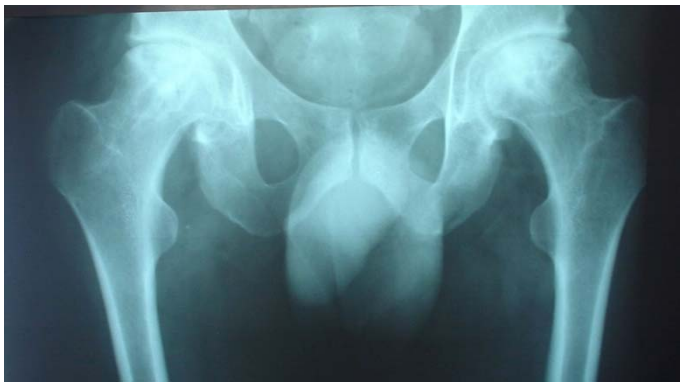
5 stage system suggested by ARCO

1. Modified Ficat Staging
2. Area Involvement: [MRI]
   Minimal <15% and Extensive >30% of the head
   [50% in AP and 50% Lateral = 50X50 =25%]
3. Length of crescent
   [Minimal <15% and Extensive >30% of the head]
4. Dome depression < 2mm depression
   2-4mm depression
   > 4mm depression



Investigations

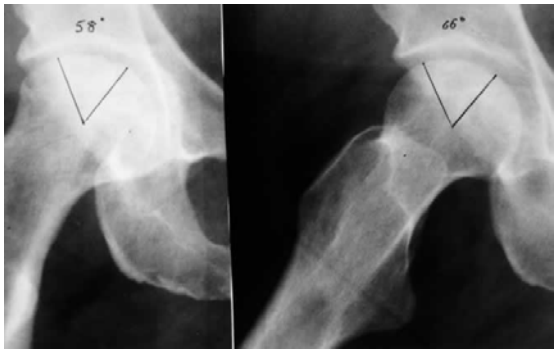
X-ray features



- Normal in stage 0 and I
- Osteopenia and sclerosis in stage II
- Subchondral collapse “crescent sign” in stage III ;
- Osteoarthritis in IV

Kerboull Necrotic angle:

- Sum of this angle in AP and Lateral
- More than 200 degrees means extensive AVN



Functional exploration of bone: not done

FEB: intraosseous venography, intraosseous pressure measurements
Rarely done

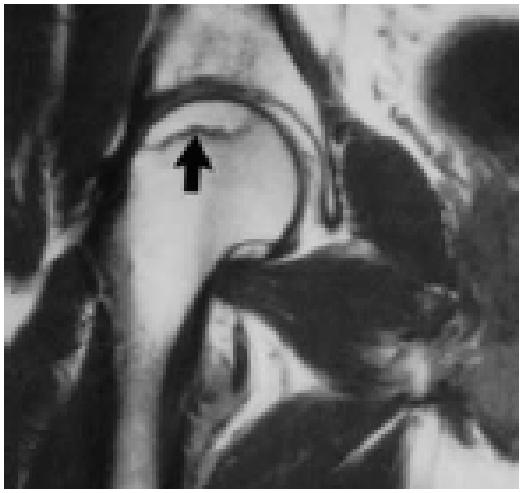
Biopsy

Bone scan features

- Cold area: First 2 wks
- Hot area After 2 wks [repair]

Single Photon Emission Computed Tomography (SPECT) scan : useful

MRI commonly done



MRI

Shows distinctively increased bone density due to reactive new bone formation in the surrounding viable tissue.

Serpiginous line :This line is thought to represent the interface between necrotic and reparative zones. The signal abnormality is usually located between the 10 o'clock and 2 o'clock positions in the anterior aspect of the femoral head.

Blood tests

Bloods

Sickle cell disease, systemic lupus erythematosis, hyperuricemia,

subtle coagulopathies: >70%. with previously diagnosed "idiopathic" AVN. In these cases coagulation profile is normal

A more **complete** coagulation profile: decreased levels of protein C, protein S, and antithrombin III (AT III); increased resistance to activated protein C (RAP-C); elevated antiphospholipid antibodies; plasminogen activator inhibitor activity (PAI-1); Leidon factor

Meta analysis of the literature - 21 studies involving 819 hips Hungerford

Rates of preservation of the femoral head:

- Stage 1 35%
- Stage 2 31%
- Stage 3 13%

Prognosis

1. Stage: I & II better. [Presence of crescent sign = bad]

2. Size of the necrosis: small [<15%] is better than major [>30%]

3. Contour: > 2mm flattening poor prognosis

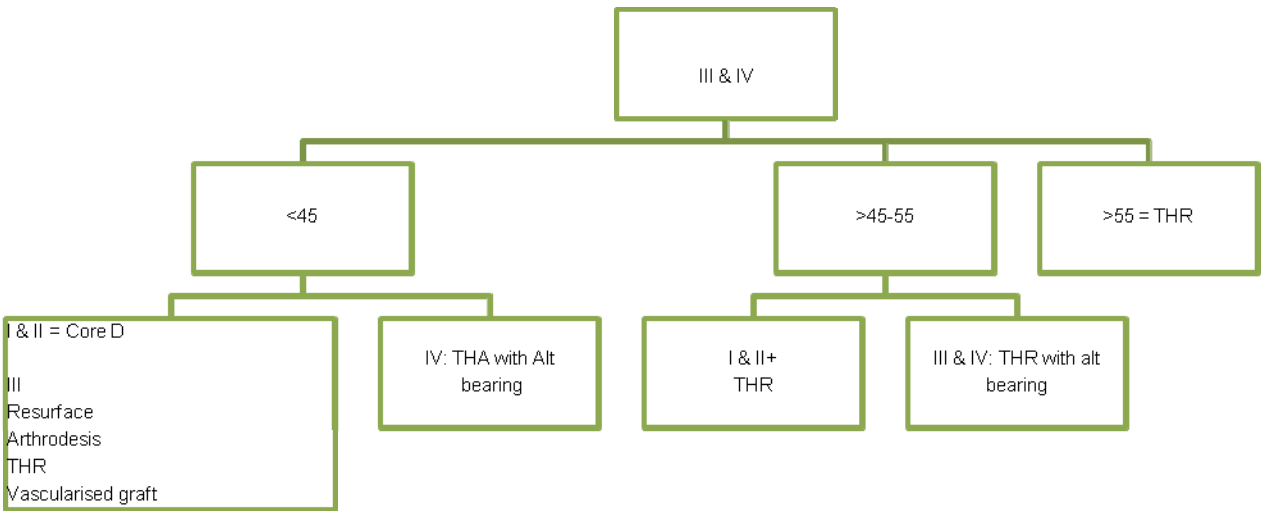
4. Presence or absence of arthritis

5. Steroid AVN has poor prognosis

6. Kerboull Necrotic angle: >200 degrees is poor.

7. Location: when in involvement > 2/3 rd of WB surface

Treatment



In stages 1 and 2
Weight-relief
Surgical decompression of the bone may prevent bone collapse.

In stage 3 and 4
Total Hip arthroplasty
Arthrodesis