Osteonecrosis

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Abstract

Formerly referred to as avascular necrosis, the term osteonecrosis is now preferred. Simply defined, osteonecrosis means 'dead bone'. The 'avascular' state of the necrotic bone is the result of a loss of circulation from numerous potential causes together with multiple risk factors. The resultant state is a sequelae of repair processes leading finally to gross deformation of the bony structural architecture and joint incongruity. Among the commonest sites are the femoral head, talus, lunate, knee and humeral head. In addition to normal radiography, bone scan and MRI have provided early diagnosis of subtle changes in the osteo-histology. Intervention depends upon the phase of disease progression; in the form of a series of preservation and/or salvage procedures.

Keywords avascular necrosis; femoral head; lunate; osteonecrosis; review article; shoulder; talus

Introduction

Formerly referred to as *avascular necrosis*, the term *osteonecrosis* is now preferred. Simply defined, *osteonecrosis* means 'dead bone'. The 'avascular' state of the necrotic bone is the result of a loss of circulation from numerous potential causes. Osteonecrosis describes an end condition that is the result of many possible pathogenic pathways.

The list of risk factors for osteonecrosis is long and includes trauma, corticosteroid use, alcohol abuse, smoking, haemoglobinopathies (e.g. sickle cell anemia), coagulation disorders, myeloproliferative disorders (Gaucher's, leukemia), caisson disease, human immunodeficiency virus infection, and pregnancy. In many cases a cause cannot be identified, and these patients are designated as having idiopathic osteonecrosis.[1]

Aetiology

Several theories on the pathogenesis of osteonecrosis have been proposed. Hypotheses include direct cellular toxicity, coagulopathic states, hyperlipidaemia with fat emboli, vascular interruptions or abnormalities, and elevated bone marrow pressure.

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Factors and conditions associated with osteonecrosis

- Trauma: femoral neck fracture, dislocation
- Corticosteroids
- Alcohol
- Coagulation disorders: thrombophilia, hypofibrinolysis
- Systemic lupus erythematosus and connective tissue diseases
- Hyperlipidemias
- Altered red blood cells: sickle cell anemia, thalassaemia
- Organ transplants: renal, cardiac, liver
- Dysbarism
- Liver dysfunction
- Gastrointestinal disorders
- Myeloproliferative disorders: leukemia, Gaucher's disease
- Radiation
- Pregnancy
- Smoking
- Hyperuricaemia
- Chemotherapeutic agents
- Hypersensitivity reactions
- Idiopathic

Data from reference Steinberg and Mont (2001).[2]

Box 1

None of these theories can fully account for the variety of causes. Most patients with the risk factors just mentioned never develop osteonecrosis, and many patients without identifiable risk factors do acquire the disease. The process is most likely multifactorial.[1] See Box 1.

Pathogenesis

There have been many mechanisms proposed to explain the pathogenesis of osteonecrosis. They can be categorized into six groups: (a) direct cellular toxicity, (b) extraosseous arterial, (c) extraosseous venous, (d) intraosseous extravascular, (e) intraosseous intravascular, and (f) multifactorial (Box 2).

Although several different aetiologic factors can lead to vascular impairment in osteonecrosis, the sequence of events that follow the initial insult or insults is similar. The resultant hypoxia rapidly leads to increased cell membrane permeability, which allows fluid and electrolytes to enter the cell, causing it to swell. Intracellular lysosomal enzymes are released, resulting in autodigestion or coagulation necrosis and cell rupture. Vascular injury leads to tissue oedema and haemorrhage. An inflammatory response ensues, marked by the appearance of neutrophils and macrophages.[2]

In cases of haemoglobinopathies, osteonecrosis is related to the presence of haemoglobin S. The sickling cells cause vessel occlusion leading to local hypoxaemia and cellular death. In homozygotic patients the prevalence is approximately 8% and is even more frequent in double heterozygotes with haemoglobin SC.

Osteonecrosis of the hip

Classification

The following table (Table 1) describes Ficat and Arlet classification. Stage I is the initiation of the disease without significant

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Mechanisms explaining pathogenesis of osteonecrosis

- Direct cellular toxicity
  - Chemotherapeutic agents
  - Radiation
  - Thermal injury
- Corticosteroids
- Alcohol
- Accumulated cell stress
  - Extraosseous arterial fractures
    - Hip dislocation
    - Post-surgery (iatrogenic)
    - Congenital arterial abnormalities
  - Extraosseous venous
    - Venous abnormalities
    - Venous stasis
  - Intraosseous extravascular
    - Haemorrhage
    - Elevated bone marrow pressure
    - Cellular hypertrophy and marrow infiltration
    - Bone marrow oedema
    - Displaced fractures
  - Intraosseous intravascular
    - Coagulation disorders: thrombophilia, hypofibrinolysis
- Sickle cell anemia, thalassaemia
- RBC emboli
- Lipid emboli
- Dysbarism
- Hypersensitivity reactions
- Multifactorial

Data from reference Steinberg and Mont (2001).[2]

Box 2

radiological findings. Stage II shows subchondral cysts or sclerosis. Stage III shows broken contour of the femoral head or sequestrum (Figure 1). Stage IV is the end stage with femoral head collapse, flattening and narrowing of the joint space.[3]

Radiological staging of osteonecrosis of the femoral head

Stage	Description
0	Preclinical disease with no radiological signs
I	Preradiographic disease with no apparent radiological signs
II	Diffuse porosis, sclerosis or cysts
III	Broken contour of the femoral head, or sequestration present; normal joint space
IV	Flattened contour, decreased joint space, head collapse

Data from reference Ficat and Arlet (1980).[3]

Table 1

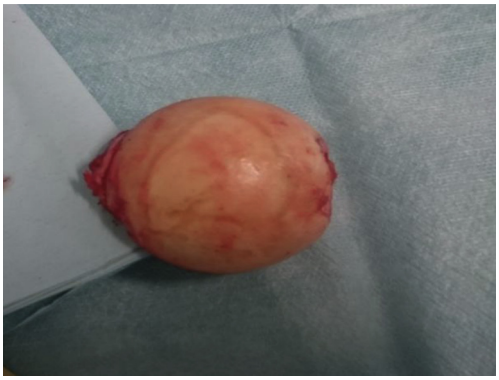

Figure 1 Area of osteonecrosis of the femoral head after collapse.

Steinberg et al subsequently devised the University of Pennsylvania System of Classification and Staging. The advantage of this staging system is that it incorporates MRI to detect a preclinical lesion and determines the size of the lesion (Table 2).[4]

Management

Osteonecrosis of the femoral head (ONFH) is a disease of the young patient with up to 25% of affected patients being less than 25 years in some institutions. Most patients will ultimately need total hip arthroplasty (THA) and it is estimated that ONFH constitutes about 10% of indications of THA procedures performed in the USA.[5]

Many modalities have succeeded in slowing the progress of the disease process or even halt it and prevent the collapse of the femoral head and their use could be promising at the early stages of the disease.[5] See Figures 2 and 3.

Management alternatives for ONFH vary from joint salvaging procedures (including electrical stimulation, proximal femur rotational osteotomy, core decompression sequestrectomy) to replacement with bone cement, non-vascularized cancellous or cortical bone grafting of the lesion, muscle-pedicle bone grafting, and free vascularized fibular grafting. The most commonly used procedures are core decompression, free vascularized fibular grafting and rotational osteotomy. Factors affecting the outcome of these procedures include patient's age, aetiology and stage of osteonecrosis, and size and location of the osteonecrotic lesion. Preservation of the femoral head with osteonecrosis depends on prevention of collapse of the structurally compromised necrotic bone.[6]

Core decompression: core decompression for treatment of ONFH represents a family of procedures that may include vascularized or non-vascularized bone grafts, bone marrow aspirate (BMA), other biologic agents that promote bone repair, or just the core tract alone.[9] In a systematic review of the literature the results of femoral head saving procedures published over the past 10–15 years were evaluated and concluded that core decompression is most effective in the early stages of ONFH and when the lesions are smaller and involve a limited amount of the weight-bearing surface of the femoral head.[10] Core decompression is ineffective when the femoral head has already collapsed.[10]

Ficat described the procedure as introducing a 6- or 8-mm trephine into the lesion from a starting point on the lateral cortex of the greater trochanter. This produced a biopsy of bone for

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