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Case Report
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Massive Hips Osteolysis: A Case Report

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Abstract

The authors present the case of a female patient with chronic alcoholism, necrosis of the humeral heads associated with total lysis of the femoral heads. After a review of the literature concerning osteolysis and osteonecrosis, a discussion the role of alcoholism the development of this pathology is reported.

Introduction

Osteonecrosis can be defined as the cellular death of different components of bone, i.e., bone tissue but also bone marrow. From an etiological point of view, when trauma is excluded, the main etiologies are corticosteroid therapy, sickle cell disease and alcoholism. Alcoholic patients often present lipidic metabolism perturbation with fat embolism and are susceptible to develop diffuse intravascular coagulation in terminal microcirculation of femoral and humeral heads. Alcohol-induced aseptic osteonecrosis is not infrequent but multifocal osseous destruction is very rare. The association of necrosis of the humeral heads associated with total lysis of the femoral heads appeared to us to be a rare entity rarely described in the literature consulted.

Case Presentation

A 73-year-old female was hospitalized in our institution following a trochanteric fracture of the right hip.

The patient had a history of chronic alcoholism. Her hematological values were trivial for age, but the GT gamma was very markedly elevated.

An osteosynthesis by a dynamic screw-plate (DHS) was carried out. In the immediate post-operative period, she developed delirium tremens, with a rapidly satisfactory outcome under treatment.

Physical rehabilitation and walking were proceeding satisfactorily. The patient was seen for control at the outpatient department one month after surgery (Figure 1).

Discussion

Rapidly destructive hip disease is a rare condition, the cause of which is yet to be clarified. Over a period of months, a femoral head can completely disappear, as first reported by Forestier in 1957 [4].

The pathogenesis remains unclear. Possible hypothesis are subchondral bone ischemia and cell necrosis, subchondral insufficiency fracture in patients with or without osteopenia resulting in bone collapse, enzyme disturbance in synovial fluid, and an aberrant bone response to osteoarthritis [9].

Our case refers to a female patient with chronic alcoholism, necrosis of the humeral heads associated with total lysis of the femoral heads.

Similar cases related to osteoarthritis were published [3].

The questions are:

- Is the massive lysis observed in the hips in our case the consequence and the end result of bone necrosis?

- Are the observed humeral osteonecrosis and lysis of the hips two different pathological entities?

The pathophysiology of osteonecrosis of the femoral head has not been completely elucidated. It can be defined as the cell death of different components of bone, i.e., bone tissue but also bone

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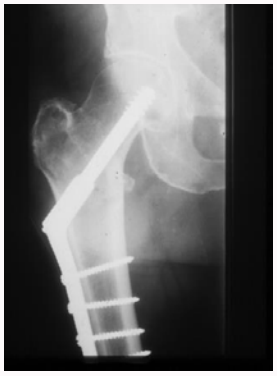

Figure 1: The patient was seen for control at the outpatient department one month after surgery.

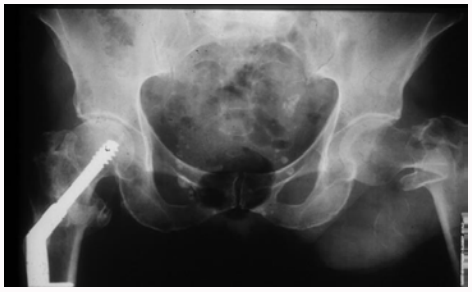

Figure 2: Two years later, she was again hospitalized following a new fall after alcohol consumption with a trochanteric fracture of the left hip.

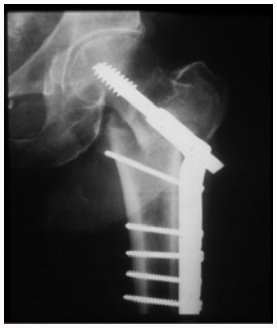

Figure 3: The treatment consisted of dynamic screw-plate osteosynthesis (DHS).
She also presented during this stay a delirium tremens in the immediate postoperative period. X-ray control was satisfactory authorizing rehabilitation and safe return to her home.

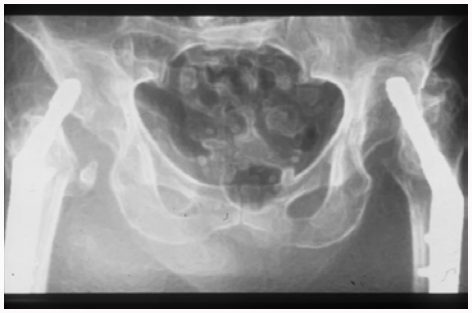

Figure 4: Nineteen months later she was again admitted through the emergency department to the intensive care unit for hypovolemic shock.
She was hypothermic (32°C), with severe anemia (hb at 2.7) and metabolic acidosis (ph 6.9). Clinically, large bruises and hematomas were observed on both thighs. The femoral pulses were absent. Pelvic X-ray showed a total lysis of the left and right femoral heads and necks.

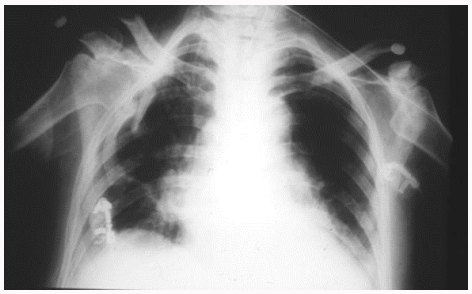

Figure 5: Chest X-ray demonstrated a deformation of the humeral heads suggesting osteonecrosis.

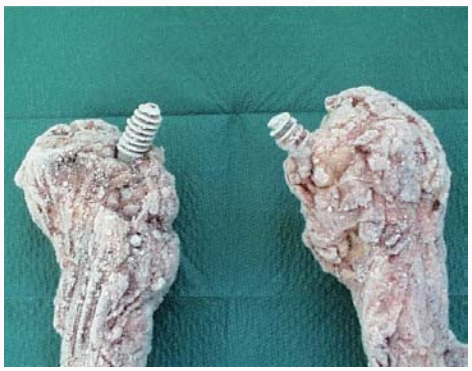

Figure 6: Macroscopic appearance of excisional parts showed screws totally stripped and complete disappearance of the femoral heads and neck.

marrow [13]. For most cases, the pathophysiology is uncertain. Risk factors [8] are listed in Table 1. Multiple investigators have postulated vascular impairment, altered bone-cell physiology, and other theories [7,11].

The role of alcohol is important in the pathogenesis of osteonecrosis [10]. A daily dose of 150 cc of ethanol (1.5 liters of red wine at 10°) is a threshold beyond which the role of ethyl intoxication can be recognized as the etiology of the disease. Several studies have shown alcoholics to have a reduced bone mass compared with nonalcoholic controls with a reduction in trabecular bone [13] seen especially in the vertebrae, ribs, iliac crest, femoral neck and calcaneus [2]. In a more recent study, Yoon et al. studied the effect of alcohol in Japan showing that former alcohol intake increased the risk of Osteonecrosis of the Femoral Head (ONFH) with a marginal significance. Current alcohol intake was associated with an increased risk of ONFH. The dose-response meta-analysis revealed that the risk of ONFH increased by 35.3% for every 100 g/week and by 44.1% for every 500 g drink-years. Current intake and the dose of alcohol were positively associated with an increased risk of ONFH in a non-linear pattern [17].

In its classical form, osteonecrosis of the femoral head is the final common pathway of a series of derangements that result in a decrease in blood flow to the femoral head leading to cellular death, fracture, and collapse of the articular surface [12]. In other words, it is the result of a succession over time of bone resorption and bone formation on the surface of dead trabeculae. Bone building fills in gaps in cancellous tissue, creating patches of true cortical tissue. Cell death preserves the mineral framework, and the dead bone

Table 1: Risk factors for osteonecrosis of the femoral head.

Direct	Indirect
Femoral head/neck fracture	Chronic corticosteroid use
Hip dislocation	Excessive alcohol consumption
Slipped capital femora epiphysis	Coagulation disorders
Radiation	Hemoglobinopathies
Sickle cell disease	Dysbaric phenomena
Caisson disease	Autoinmune diseases
Myeloproliferative disorders	Smoking Hyperlipidemia

initially retains a normal appearance. But massive lysis is not a usual consequence of osteonecrosis.

Osteonecrosis (ON) of the Femoral Head (ONFH) is the final common pathway of a series of derangements that result in a decrease in blood flow to the Femoral Head (FH) leading to cellular death, fracture, and collapse of the articular surface.

Our patient had all the risk factors explaining the osteonecrotic changes observed in in the shoulders.

But how to explain the massive lysis of the hips?

Osteolysis and chondrolysis are found in several clinical situations:

- Infection,
- Metabolic disorders,
- Traumatic conditions,
- Vascular disorders,
- Neuropathic disorders,
- Congenital disorders.

Post Traumatic Osteolysis is known to develop after trauma. Sometimes it would be mistaken for a bone metastasis.

The usual sites of post-traumatic osteolysis are:

- Distal end of the clavicle
- Ischio and iliopubic branches
- Distal end of ulna
- Distal end of radius
- Carpal bones
- Tarsal bones
- The femoral neck

The post-traumatic osteolysis of the distal extremity of the clavicle is a well-known clinical entity already described by Dupas (1936) and Strauch (1970), caused by a simple trauma, the pathogenesis of which is not yet well known. It could also occur in metacarpal bones [1].

Neuropathic arthropathy of the shoulder is a chronic progressive process characterized by joint destruction in the presence of a neurosensory deficit. Causes include syringomyelia, syphilis, diabetes, chronic alcoholism, and leprosy, with syringomyelia accounting for the vast majority of upper-extremity Charcot joints. Early presentation of this rare condition includes nonspecific symptoms such as swelling, erythema, sensory symptoms, and decreased functionality, making diagnosis challenging [15].

Primary idiopathic osteolysis is rare and is characterized by spontaneous resorption of bone with no apparent explanation. The skeleton initially appearing to be normal undergoes total or partial resorption. This process evolves over several years leading to deformation and functional impotence.

Torg et al. [16] described the following four clinical entities:

- Hereditary multicentric osteolysis with dominant transmission.
- Hereditary multicentric osteolysis with recessive transmission.
- Idiopathic nonhereditary multicentric osteolysis with nephropathy.
- Gorham's massive osteolysis.

Gorham [5] reported two patients who presented a curious form of massive osteolysis. The following year Gorham and Stout [6], proposed the term hem angiomatosis and published 24 cases previously described as massive osteolysis, disappearing bone disease, vanishing or phantom bone disease, Gorham disease. There was no sex ratio and no preferential location, however there was greater frequency in the pelvic and shoulder girdles. In all instances there had been a progressive, extensive lysis of one or more bones in a unicentric fashion. Except for pathologic fractures, the process was painless and systemic manifestations were not observed. Generally, the slowly progressive absorption of bone was observed to stabilize; the degree of disability resulting was less than expected considering the amount of bone resorption. Regeneration of bone did not occur.

Conclusion

Rapidly progressive hip disease is a rare syndrome of unknown etiology also known as vanishing hip and rapidly progressive osteoarthritis [3].

Based on this case report we suggest that the association of metabolic disturbances, chronic alcoholism and its consequences (osteoporosis, osteonecrosis), advanced age, can account for an extremely severe osteonecrotic process that can explain this massive lysis.

The differential diagnosis of vanishing hip should include a malignant cause of bone destruction, septic arthritis, avascular necrosis and neuropathic arthropathy. It should also include the Gorham-Stout disease, known as a cause of mysterious bone disappearing.

Interesting about our case is that since both hips were involved and associated to osteonecrosis of the shoulders it may suggest a more systemic process. We presume that alcohol consumption led to osteonecrosis, and collapse of the femoral heads. That both femoral heads collapsed at the same time may be coincidence, but a more systemic process remains unclear.

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