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Original Research

Assessment of cases of femoral head osteonecrosis

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ABSTRACT:
Background: Osteonecrosis (ON) is defined as avascular necrosis or aseptic necrosis, is characterized as bone cell death that follows an impairment of the blood flow to the bone from a traumatic or non-traumatic origin. The present study was conducted to assess cases of femoral head osteonecrosis. **Materials & Methods:** 210 patients of femoral head osteonecrosis of both genders were included. History of alcoholism, steroids, smoking, lifestyle, specific family history, and associated systemic illness was recorded. **Results:** out of 210 patients, males were 110 and females were 100. Common etiology was trauma in 48, alcohol in 50, idiopathic in 5, steroids in 52, drug induced in 20, aplastic anemia in 18 and pregnancy in 17 cases. The difference was significant (P< 0.05). ARCO stage 1 was seen in 24, 2 in 46, 3 in 60 and 4 in 80 cases. The difference was significant (P< 0.05). **Conclusion:** Most common cause of femoral head osteonecrosis was steroid intake, alcoholism and trauma.
Key words: Alcoholism, femoral head, Osteonecrosis.

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INTRODUCTION

Osteonecrosis (ON), also defined as avascular necrosis or aseptic necrosis, is characterized as bone cell death that follows an impairment of the blood flow to the bone from a traumatic or non-traumatic origin. ON most often happens in the hip joint (femoral head) but may also occur in other anatomical locations (e.g. shoulder, knee and ankle). The observed incidence of ON in a study population in the UK between 1989 and 2003 was in the range of 1.4 to 3.0 per 100000. The hip joint was mostly involved, accounting for 75.9% of cases.[1]

Osteonecrosis of the femoral head (ONFH) is a disabling condition of the hip joint that primarily affects the young individuals. The etiology, natural history, and epidemiology of ONFH have not been fully elucidated.[2] There are associations of many diseases and drugs with ONFH. ONFH is characterized by a compromised subchondral microcirculation, especially in the small retinacular vessels, which ultimately leads to necrosis of bone. An accumulation of microfractures is seen and, as there is no bone remodelling, a collapse of the subchondral bone occurs.[3]

Most theories point towards an alteration in the intravascular blood flow as the potential mechanism of ON initiation.[4] These alterations may occur either from a traumatic or a non-traumatic cause or be a consequence of some well-accepted risk factors. Regarding the traumatic cause, it is important to notice that the majority of the blood supplied originates from the retinacula arteries supplying the superolateral weight-bearing portion of the femoral head. These retinacular vessels originate from the lateral epiphyseal artery which is a branch of the medial circumflex arteries.[5] Among traumatic causes, physical trauma, decompression sickness or radiation may be cited. In the non-trauma cases, two theories are disputed: the first concerns the occurrence of an intravascular coagulation and the second one attributes the ischaemia to extravascular compression.[6] The present study was conducted to assess cases of femoral head osteonecrosis.

MATERIALS & METHODS

The present study comprised of 210 patients of femoral head osteonecrosis of both genders. All were

enrolled after they agreed to participate and gave their written consent.
Data such as name, age, gender etc. was recorded. A thorough clinical examination was performed. History of alcoholism, steroids, smoking, lifestyle, specific family history, and associated systemic illness was recorded. In posttraumatic conditions, the details of injury, duration of injury to surgery/intervention, surgical procedure, and modalities of evaluation for ONFH were collected. Routine laboratory investigations such as complete blood count, renal function test, liver function test, lipid profile and coagulation profile were performed for all patients. Results were analyzed statistically. P value less than 0.05 was considered significant.

RESULTS

Table I Distribution of patients

Total- 210		
Gender	**Males**	**Females**
Number	110	100

Table I shows that out of 210 patients, males were 110 and females were 100.

Table II Etiology of cases

Etiology	Number	P value
Trauma	48	0.05
Alcohol	50	
Idiopathic	5	
Steroids	52	
Drug induced	20	
Aplastic anemia	18	
Pregnancy	17	

Table II, graph I shows that common etiology was trauma in 48, alcohol in 50, idiopathic in 5, steroids in 52, drug induced in 20, aplastic anemia in 18 and pregnancy in 17 cases. The difference was significant (P< 0.05).

Graph I Etiology of cases

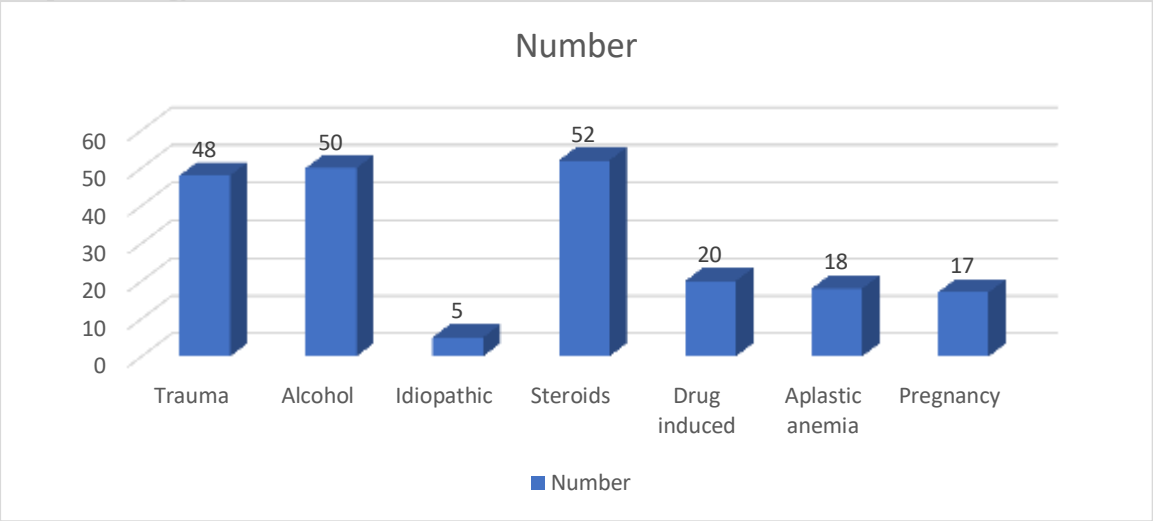

Table III Distribution based on Association Research Circulation Osseous Staging (ARCO)

Stage	Number	P value
1	24	0.02
2	46	
3	60	
4	80	

Table III shows that ARCO stage 1 was seen in 24, 2 in 46, 3 in 60 and 4 in 80 cases. The difference was significant (P< 0.05).

DISCUSSION

In femoral head osteonecrosis, intravascular coagulation can occur as the end result of local vascular impairment; vascular occlusion occurs because of thrombus formation due to abnormally shaped red blood cells as seen in sickle cells anaemia or fat or nitrogen embolism.[7] Extravascular compression may arise secondary to damaged femoral head vessels that permit the accumulation of fat and blood in the extravascular space which leads to alterations in blood flow through local compression.[8] Moreover, it is nowadays admitted that the pathophysiological mechanism arises from an interaction between vascular impairment, altered bone-cell physiology, risk factors as well as genetics.[9] Vascular impairment appears as the end result of coagulation disorders seen in hypercoagulable conditions such as sickle cell anaemia, hereditary thrombophilia, antiphospholipid antibodies, malignancy and inflammatory bowel disease. An altered cell-bone physiology is often proposed as being part of the osteonecrotic process and the hypothesis is that ON appears secondary to impaired mesenchymal differentiation which leads to a damage of the bone structure.[10] Under physiological conditions, it needs about three months to build new bone with effective mechanical properties whereas it needs three weeks for osteoclast to affect mechanical strength of the trabecular bone. So, any dysfunction of the mesenchymal cell that lead to changes in osteogenic differentiation and alterations in blood flow through an increased adipogenic volume would ultimately support the ON of the femoral head.[11] The present study was conducted to assess cases of femoral head osteonecrosis.

In present study, out of 210 patients, males were 110 and females were 100. Vardhan et al[12] in their study 249 patients (382 hips) of osteonecrosis femoral head (ONFH) were evaluated. The mean age was 34.71 years (range 14-70 years) and 70.28% (n=175) patients were between 20 and 40 years. Male to female ratio was 5:1. Bilateral ONFH was observed in 53.41% (n=133) patients. In atraumatic conditions, bilateral involvement was seen in 61.61% (130/211) patients. Steroid administration (37.3%, 93/249) was most commonly observed in the patients followed by idiopathic in 21.3% (53/249) patients, chronic alcohol consumption in 20.1% (50/249) patients, and trauma in 15.3% (38/249) patients. There were 48% (185/382) hips in ARCO Stage 2 followed by 33% (125/382) in Stage 3 and 16% (61/382) in Stage 4. The mean HHS was 80.97 ± 14.35 in unilateral ONFH. The mean HHS was 72.79 ± 14.43 and 80.07 ± 13.52 in more involved hip and in less involved hip, respectively, in bilateral ONFH. The ARCO staging had statistically significant correlation with HHS in unilateral ONFH patients and more severely affected hip in bilateral ONFH, but it did not show any association with less involved hip in bilateral cases.

We found that common etiology was trauma in 48, alcohol in 50, idiopathic in 5, steroids in 52, drug induced in 20, aplastic anemia in 18 and pregnancy in 17 cases. Mont et al[13] found that corticosteroid administration is one of the most common risk factors for ONFH, but the true extent of its use that constitutes a risk factor is still under debate. Although patients receiving corticosteroids have at least one other confounding factor, multivariate analysis has suggested that corticosteroid use, especially in high doses, is an independent variable. Dosages typically considered to be associated with the disease are >2 g of prednisone, or its equivalent, within a period of 2–3 months. The risk period for development of ONFH following corticosteroid therapy has been more exactly defined to be 12 months or less for the majority of patients receiving corticosteroids. Nontraumatic ONFH associated with steroid intake was accounted for 44% patients. The mean daily intake of prednisolone in unilaterally affected patients was 28 mg (70% of patients had consumed >2 g prednisolone intake in 2 months), and with bilateral involvement, the mean intake was 28.8 mg per day (86% of patients had consumed >2 g prednisolone intake in 2 months).

We observed that ARCO stage 1 was seen in 24, 2 in 46, 3 in 60 and 4 in 80 cases. Kang et al[14] found that 14.6% were related to steroid intake. Although these data were comparable to those reported previously in Korea, they are quite different from France where 30% ONFH were secondary to steroid intake.

CONCLUSION

Authors found that most common cause of femoral head osteonecrosis was steroid intake, alcoholism and trauma.

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