RELATIONSHIP OF ALCOHOL AND TOBACCO WITH AVN OF FEMORAL HEAD IN ADULT INDIAN POPULATION

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Abstract:
Background: Avascular necrosis (AVN) or osteonecrosis is the death of bone tissues due to decreased blood flow of femoral head, it is a major orthopaedic disease with many risk factors including the chronic alcohol and tobacco consumption.

Aims and objectives: To study the relationship of alcohol and tobacco with AVN of femoral head in adult population.

Materials and Methods: This study included 72 patients of AVN of femoral head (ONFH) who attending our hospital between Sept 2018 to Sept 2020. History of alcoholism and tobacco consumption, detailed information of the amount and duration of smoking and alcohol intake was recorded.

Results: Majority of the AVN patients were working age groups males (72%) and 28% were females. Out of 72 patient 55% had history of alcohol consumption and 60% were smokers which indicates a positive correlation of cellular toxicity with AVN of femoral head.

Conclusion: Present study conclude that the alcohol ingestion and tobacco smoking are the major cause of cellular toxicity and AVN of femoral heads.

Keywords: alcohol drinking, femur head necrosis, smoking

Introduction:
Osteonecrosis is a degenerative bone condition characterized by the death of cellular components of bone secondary to an interruption of the subchondral blood supply. It is also known as avascular necrosis (AVN), aseptic necrosis, and ischemic bone necrosis. It typically affects the epiphysis of long bones at weight-bearing joints. Severe cases can lead to the destruction of subchondral bone or the collapse of an entire joint.

The most common sites for AVN to occur are the femoral head (FH), knee, talus, and humeral head. The hip is the most common location overall.

There are numerous risk factors and theories on the development of this vascular impairment which can be classified in six categories as Extraosseous arterial fracture, Extraosseous venous, Intraosseous extravascular compression, Intraosseous intravascular occlusion and Direct cellular toxicity which includes the Chemotherapy, Radiotherapy, Thermal injury, Alcohol ingestion and Smoking.

Alcohol use has been associated with approximately 31% of patients who develop hip AVNFH. Excessive alcohol consumption related to AVN of the FH is the result from the decreased bone genesis caused by excess lipid formation and increased intracellular lipid deposits which results in tissue death.

There is a growing body of evidence of deleterious effects of smoking on the musculoskeletal system. Smoking has been shown to interfere with bone metabolism, revascularization and bone formation. Smoking impaired the flow of oxygenated blood to bones and nicotine decreases bone healing strength as well as revascularization of bone grafts, which results in AVN of bones.

This study attempts to reveal the relationship of alcohol and tobacco with AVN of femoral head in patients attending the Index Medical College, Indore, India.
Materials and Methods:

Present observational study enrolled 72 patients who were attending Index Medical College, Indore and diagnosed with AVNFH and between Sept 2018 and Sept 2020.

After receiving subject’s formal consent and ethics committee’s approval we have recorded the medical history of patients, medical history of family and history of injuries. For patients with history of alcoholism and tobacco consumption, detailed information of the amount and duration of smoking and alcohol intake was recorded. Demographic and anthropomorphic details were also recorded.

All the patients were subject to the clinical and radiological examination and those who have features of femoral heads as crescent sign, or band-like sclerosis or collapse were included in the study.

Patients with major traumatic hip injury or history of hip injury, patients with diabetes, cardiovascular disease or on any medication were excluded from this study.

All the subjects were undergone the laboratory tests for complete blood count (CBC), renal function, liver function, lipid profile and coagulation profile.

Microsoft excel and IBM SPSS were used to record and analyse the study data. Frequency distribution and cross tabulation was used to prepare the tables. Quantitative data is expressed as number whereas categorical data is expressed as number and percentage.

Result:

As part of this study we have recorded the demographic parameters and results of various clinical-radiological tests of all 36 subjects.

Mean age of cohort was 45.11 years where youngest patient was 19 years and eldest of 52 years, out of 72 patient’s majority were 72% were males in working-age group, while 28% were females. Mean age of male subjects was lower than females. BMI calculations show majority of the patients were overweight.

Table 1: Demographic details of patients.

| Gender | Count | Mean Age (Years) | BMI |
|--------|-------|------------------|-----|
| Male   | 26 (72%) | 44 | 25 |
| Female | 10 (28%) | 48 | 24.5 |

AVN occurs when blood flow to a bone is interrupted or reduced. It can be caused due to joint or bone trauma as a result of an injury, dislocated joint, damaged blood vessels etc. Other major cause is the fatty(lipid) deposits in blood vessels which blocks the small blood vessels and reduces the blood flow to the bones. Excessive alcohol consumption cause the fatty deposits in the blood vessels. Similarly Smoking restricts the flow of oxygen-rich blood to bones and reduces the body’s ability to absorb calcium which in turn results in AVN.

In present study we have recorded risk factors were Alcohol consumption (55%) was the most common, followed by the tobacco (Smoking) consumption in 60% subjects.

Table 2: Etiology distribution of AVN

| Aetiology       | Number | Percentage |
|-----------------|--------|------------|
| Alcohol         | 40     | 55         |
| Idiopathic      | 7      | 10         |
| Drug induced    | 2      | 3          |
| Smoking         | 43     | 60         |

The mean alcohol consumption in alcoholic patients of this study was 520 ml/week ranging from 250 m to 900 ml/week. The mean duration of alcohol ingestion in former drunker was 72 months, 77 months in the quitters and 84 months in the regular drinkers. AVNFH associated with alcohol administration was mainly seen only in male population.

Average cigarette smoked was 12 per day ranging from 2 to 20, all the smokers were males and average age was 39 years.

Table 3: Distribution of alcoholism and smoking of the patients.

| Parameters                        | Frequency | Count | %  | Mean Consumption | Mean Duration (years) |
|-----------------------------------|-----------|-------|----|------------------|-----------------------|
| Alcohol Consumption               | Never     | 25    | 35 | 0                | 0                     |
|                                   | Former    | 7     | 10 | 520ml/week       | 6                     |
|                                   | Occasional| 18    | 25 | 300ml/week       | 6.5                   |
|                                   | Regular   | 22    | 30 | 650ml/week       | 7                     |
| Cigarette smoking (pack = 20 cigarettes) | Never | 22    | 30 | 0                | 0                     |
|                                   | Former    | 7     | 10 | 3pack/week       | 11                    |
|                                   | Current   | 36    | 50 | 4pack/ks/week    | 13                    |

Discussion:

Alcohol and smoking are the major risk factor of AVN of bones, which majorly affects the long bones at weight-bearing joints and therefore AVN of FH is
commonly associated with the alcoholism and tobacco smoking.

AVN of FH is more prevalent among the of working age population, in our study the average age of AVNFH patients was 45.11 years ranging from 19 to 52 years. Our study witnessed the male preponderance (72%) with mean age of 44 years while females (28%) mean age was 48 years.

Similar retrospective study by Ponzio DY et al (2018) studied 43 patients (62 hips) who underwent primary hip arthroplasty for a diagnosis of AVN of the femoral head recorded the mean age was 51 years, predominantly male (88%), with a high rate of comorbidities which is comparable to our study. 5

Another study by Mayers W, et al (2016) also recorded similar age distribution where the mean patient age of the AVN group was significantly lower (56.9 vs 65.9 years, p < 0.01). Men accounted for 51.9 % of the AVN group and 43.0 % of the OA group (p < 0.01). 6

Overweight and obesity are often associated with hyperlipidemia. This in turn is associated with elevated fasting and postprandial plasma insulin concentrations that can promote adipose synthesis and inhibit the decomposition of adipose tissue. Given that hypercholesterolemia has also been associated with idiopathic avascular necrosis. 7

In our study also shows that the mean BMI of male and subjects was 25 and 24.4kg/m2 respectively. Zhao DW, et al (2015) also observed the positive association between AVN of femoral head and the BMI of the patients. 8

There are multifactorial aetiologies of AVN of femoral head out of which cellular toxicity if the major cause of AVN. Smoking and alcoholism are the major cause of cellular toxicity. In our study 55% patients had alcohol ingestion 55% were smokers. Similar study by Ponzio DY et al (2018) observed the similar results where they recorded the history of cigarette smoking in 65% subject. 5

K. Matsuo, et al (1988) study showed a clear increase in the risk of femoral head osteonecrosis in individuals consuming greater than 400 mL of alcohol per week. 7

Metanalysis by Rico et al. reported that alcohol-induced osteonecrosis FH induced by alcohol stimulate the excessive secretion of adrenal glucocorticoids and found that alcohol and hormones may stimulate FH through a common pathway. 9

Our study is also supported by Sakata R et al (2003) where the multivariate analysis based on a hierarchically well-formulated model strategy for males revealed that cumulative alcohol consumption (OR = 1.016, 95% CI: 1.005, 1.026) remained as the significantly associated risk factors for osteonecrosis after adjusting the remaining confounders. 10

Gan D et al (2013) also recorded that the Alcohol-induced osteonecrosis FH accounts for approximately 1/3 of total ACNFH. Alcohol intake and the incidence of osteonecrosis FH has a significant dose-effect relationship. There are some correlations between alcohol-induced osteonecrosis FH and lipid metabolism, secretion of corticosteroid, and some gene of alcohol or lipid metabolism. 11

Conclusion:

Based on the observations and above discussion we concluded that the alcohol consumption and smoking are the major factors of cellular toxicity which causes the narcosis of bones and majorly affect the big bones joints alike femoral head and hips.

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