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Research article

Unraveling Risk Determinants in Avascular Necrosis: A Cross-Sectional Analysis of Demographic, Behavioral and Hemostatic Profiles

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Abstract: Avascular necrosis (AVN), or osteonecrosis, is a debilitating condition caused by reduced blood flow leading to bone tissue death. While commonly linked to trauma, corticosteroid use, and alcohol abuse, emerging evidence suggests that microvascular thrombosis and hypercoagulability may significantly contribute to its development. Prothrombotic condi-tions can obstruct bone microcirculation, resulting in tissue necrosis without infection or malignancy. This study explores the role of coagulation abnormalities as potential risk factors for AVN through a cross-sectional, observational, and analytical approach. A total of 28 patients with non-traumatic osteonecrosis were recruited from a tertiary care center. Each underwent comprehensive coagulation testing to assess thrombotic risk. The study also analyzed demographic and behavioral factors such as age, sex, alcohol intake, and smoking. Preliminary results revealed a high incidence of procoagulant abnormalities, supporting the theory that impaired hemostasis may underlie ischemic bone damage. These findings highlight the importance of including coagulation profiles in the diagnos-tic and prognostic assessment of AVN, promoting a more integrative approach to manag-ing patients at risk for this condition.

Keywords: osteonecrosis, procoagulant status, thrombotic risk

1. Introduction

Avascular necrosis (AVN), or osteonecrosis, is a progressive pathological entity characterized by ischemic death of bone tissue resulting from impaired blood supply. The condition most commonly affects the femoral head but may also involve other anatomical sites such as the humeral head, distal femur, and proximal tibia. Left unrecognized or untreated, AVN leads to subchondral bone collapse, joint dysfunction, and ultimately degenerative osteoarthritic changes, significantly impacting patients' quality of life and functional independence[1].

While established etiological factors include traumatic injury, prolonged corticosteroid therapy, excessive alcohol intake, and hematological disorders, a substantial proportion of cases remain idiopathic. In recent decades, attention has shifted toward the role of hematological and vascular dysfunctions—particularly hypercoagulable states—as possible contributory mechanisms in non-traumatic AVN. The hypothesis that intravascular thrombosis within the microcirculation of bone may impair perfusion and initiate the necrotic cascade is increasingly supported by emerging data.

Numerous studies have identified a spectrum of prothrombotic and hypofibrinolytic abnormalities in patients with AVN. For instance, Chotanaphuti et al. reported that 32.5% of patients with idiopathic osteonecrosis of the femoral head exhibited thrombophilic disorders, with deficiencies in protein C and protein S being among the most

common findings[2]. This prevalence is notably higher compared to the 1–15% observed in the general population, suggesting a significant association between these coagulation defects and AVN development.

Furthermore, a study by de Oliveira et al. compared patients with idiopathic and secondary osteonecrosis of the femoral head, finding that those with idiopathic AVN had higher odds of presenting with protein S and protein C deficiencies. Specifically, the odds ratio for protein S deficiency was 5, indicating a fivefold increased risk, while the odds ratio for protein C deficiency was 2.14. [3]

Glueck et al. (2003) demonstrated a high prevalence of thrombophilic conditions, including elevated plasminogen activator inhibitor-1 (PAI-1), anticardiolipin antibodies, and resistance to activated protein C in individuals with idiopathic osteonecrosis of the femoral head[4]. Similarly, Korompilias et al. (1997) found a statistically significant association between anticardiolipin antibody positivity and non-traumatic AVN, implicating antiphospholipid syndrome as a possible pathogenic factor[5].

A systematic review by Orth and Anagnostakos (2013) emphasized the role of coagulation factor abnormalities—such as deficiencies in protein C, protein S, antithrombin III, and elevated factor VIII—as potential contributors to impaired osseous perfusion and necrosis[6,7]. Furthermore, inherited mutations such as factor V Leiden and the prothrombin G20210A variant have been implicated in increasing thrombotic risk and AVN susceptibility.

In this context, the present study aims to explore the procoagulant profile as a potential predictive risk factor for aseptic osteonecrosis. We conducted a cross-sectional, observational, descriptive, and analytical investigation involving 28 patients diagnosed with osteonecrosis at the Ilfov County Emergency Hospital. Each patient included in the study underwent a comprehensive thrombophilia screening panel through venous blood sampling. The investigations targeted both inherited and acquired coagulation abnormalities, aiming to elucidate potential correlations with disease severity in aseptic osteonecrosis.

The laboratory assessment encompassed genetic mutations and biochemical markers associated with thrombotic risk. In addition to the genetic panel, functional coagulation parameters were also measured:

Moreover, demographic variables (age and sex) and behavioral risk factors (alcohol consumption and tobacco use) were systematically documented. These parameters were subsequently subjected to statistical analysis to identify significant associations with the clinical severity and extent of osteonecrotic lesions.

Through this multidimensional analysis, our objective is to elucidate whether laboratory indicators of hypercoagulability correlate with osteonecrosis severity and to determine if such parameters may aid in stratifying patient risk and guiding early therapeutic intervention.

2. Methodology

This article presents a cross-sectional, observational, descriptive, and analytical study conducted with the primary aim of evaluating the procoagulant status as a potential predictive risk factor for aseptic osteonecrosis. The study was anchored in the clinical hypothesis that specific coagulation abnormalities—either genetically mediated or functionally acquired—may generate a biological predisposition toward thrombotic events in bone microcirculation, thus contributing to the ischemic cascade that underlies the pathogenesis of osteonecrosis.

The research was carried out between January 2023 and March 2025 within the Ilfov County Clinical Emergency Hospital, located in Bucharest, Romania - a tertiary care institution with multidisciplinary diagnostic capacity. The study was conducted with full respect for ethical standards in medical research. All participating patients provided written informed consent, and the research protocol received formal approval from the hospital's Ethics Committee.

A total of 28 patients diagnosed with aseptic osteonecrosis were included in the study.

Inclusion criteria encompassed patients with a confirmed diagnosis of aseptic osteonecrosis based on clinical and imaging findings, aged 18 years or older, who were able to provide written informed consent and who fully completed the behavioral questionnaire regarding alcohol and tobacco consumption. Additionally, only patients with complete laboratory data for the full coagulation panel - including both genetic and functional parameters - were included to ensure consistency and comparability across the study cohort.

Data were compiled in a dedicated database capturing demographic variables such as age, sex, and place of residence (urban or rural), along with laboratory, clinical, and behavioral metrics.

Venous blood samples were collected from each patient to perform a comprehensive coagulation risk panel, including both genetic mutations and functional coagulation markers known to be associated with thrombotic predisposition. Specifically, the following parameters were analyzed:

- Genetic variants:
  - Factor V Leiden mutation (G1691A)
  - Prothrombin gene mutation (Factor II G20210A)
  - MTHFR gene polymorphisms (C677T and A1298C)
  - Factor XIII polymorphism (G102T)
  - PAI-1 gene polymorphism (675 4G/5G)
  - Endothelial Protein C Receptor (EPCR) gene variants
- Functional coagulation parameters:
  - Antithrombin III (reference range: 83–128%)
  - Protein C activity (70–140%)
  - Protein S activity:
  - Males: 74–146.1%
  - Females: 54.7–123.7%
  - Plasma homocysteine levels (4.3–11.1 μmol/L)

Based on these laboratory results, each patient was categorized into one of three procoagulant status profiles:

- Normal
- Moderate hypercoagulability
- Severe hypercoagulability

This stratification allowed for comparative analysis regarding osteonecrosis severity and clinical correlations.

To complement the biological assessment, a behavioral risk evaluation was conducted using a structured questionnaire. This included 10 items specifically targeting alcohol consumption patterns and 2 additional questions addressing tobacco use for those who self-reported as smokers. Based on cumulative scoring, patients were classified into risk categories using the following scale:

- Score I (0–7 points): Low risk
- Score II (8–15 points): Moderate risk
- Score III (16–19 points): High risk
- Score IV (20–40 points): Probable dependence

This behavioral assessment aimed to capture the synergistic influence of modifiable lifestyle factors in potentiating the thrombotic risk in AVN patients.

Together, the integration of genetic, biochemical, and behavioral data served as the foundation for a multidimensional exploration into the pathophysiological substrate of aseptic osteonecrosis.

The database was compiled and managed using Microsoft Excel (latest version, Microsoft 365, release 2501), where all patient variables - demographic, behavioral, and laboratory - were systematically encoded. Excel was also used for initial descriptive

analysis and graphical representation of the data, including bar charts, scatter plots, box plots, and frequency histograms, to visualize distributions, trends, and outliers across the studied parameters.

For advanced statistical analysis, the dataset was exported to IBM SPSS Statistics (version 30.0.0). Within SPSS, we conducted both descriptive statistics (mean, median, standard deviation, range) and inferential analysis.

This integrated approach - merging classical statistical tools with algorithmic processing - ensured robust, reproducible, and nuanced data interpretation, enabling precise identification of predictive patterns relevant to prothrombotic risk in aseptic osteonecrosis.

3. Results And Discussion:

A total of 28 patients diagnosed with aseptic osteonecrosis were included in the study. The age distribution within each sex category observed in our cohort reflects partially established epidemiological patterns described in the literature. In our study, males accounted for 60.7% (n=17) and females for 39.3% (n=11) of the 28 patients diagnosed with aseptic osteonecrosis.

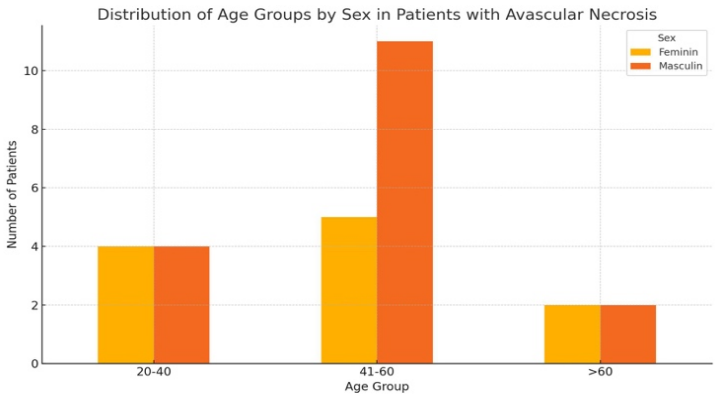

Fig. 1 - Distribution of Age groups by Sex in Patients with AVN

The age distribution within each sex category is illustrated in the chart above (fig. 1), highlighting subgroup sizes across defined age intervals for both male and female patients.

These findings are in line with medical literature data suggesting a higher incidence of osteonecrosis in males during the fourth decade of life and in females during the fifth. For example, a recent population-based study conducted in China reported a mean age of 47.65 ± 12.12 years among patients with osteonecrosis of the femoral head, with peak incidence observed in the 40s for men and in the 50s for women[8]

Among the patients included in the study, 20 individuals (71.4%) were from urban areas, while 8 patients (28.6%) originated from rural settings.

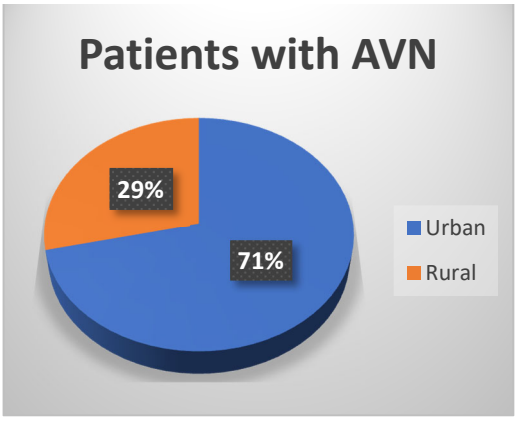

Fig 2. Distribution of patients with AVN by environment

This distribution may reflect disparities in healthcare access, lifestyle behaviors, and occupational exposures - variables known to influence both the incidence and clinical progression of aseptic osteonecrosis. Understanding the environmental context of affected individuals offers valuable insight for risk stratification and helps guide targeted preventive interventions at the population level.

The analysis of genetic thrombophilia markers in the cohort of 28 patients with aseptic osteonecrosis revealed several relevant patterns (figure 3):

1. Factor V Leiden (G1691A):

o Present in 46.4% of patients: 10 heterozygous and 3 homozygous cases.

o This mutation, a well-known contributor to activated protein C resistance, has been significantly associated with microvascular thrombosis and ischemic bone necrosis in prior studies. Its relatively high prevalence in this cohort suggests a possible pathogenic role.

2. Prothrombin G20210A mutation (Factor II):

o Detected in 7 patients (25%) as heterozygous carriers.

o While less frequent than Factor V Leiden, this mutation leads to elevated prothrombin levels, favoring a hypercoagulable state that may impair intraosseous circulation.

3. MTHFR gene mutations:

o C677T variant: Found heterozygously in 6 patients, with no homozygous cases.

o A1298C variant: Present in 5 patients as heterozygotes.

o These polymorphisms are linked to elevated homocysteine levels, an independent risk factor for endothelial dysfunction and thrombosis.

4. Factor XIII G102T:

o This mutation was absent in all patients, suggesting limited relevance in this cohort's thrombotic profile.

5. PAI-1 gene polymorphism (675 4G/5G):

o Notable for 8 patients being heterozygous 4G/5G and 3 being homozygous 4G/4G.

o This polymorphism is associated with reduced fibrinolytic activity, increasing thrombotic potential - particularly relevant in microvascular settings such as subchondral bone.

6. EPCR gene polymorphism:

o No variants were detected in the studied population.

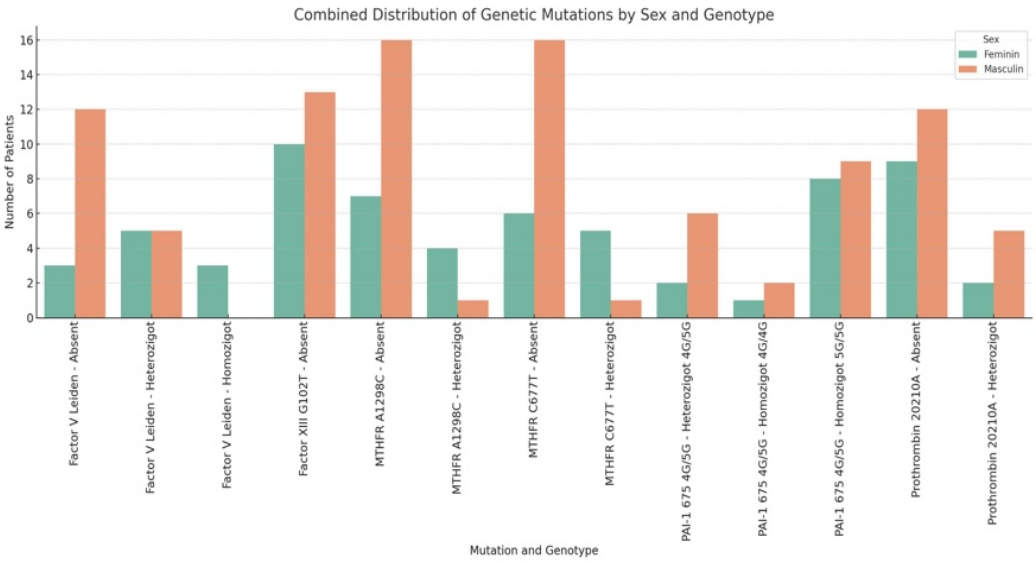

Fig. 3. Distribution of Genetic Mutations by Sex and Genotype

Factor V Leiden mutations were more frequently observed in male patients, with a predominance of heterozygous presentations. This aligns with existing data suggesting a higher prevalence of thrombotic risk mutations in men with idiopathic osteonecrosis.

Prothrombin G20210A mutation also demonstrated a male predominance in heterozygous expression, reinforcing the notion that this prothrombotic variant may be implicated more commonly in males with ischemic bone pathology.

MTHFR polymorphisms (C677T and A1298C) showed a relatively even distribution between sexes, although a slightly higher number of heterozygous variants were observed in females. These mutations are associated with hyperhomocysteinemia, a known endothelial toxin, and may contribute to microvascular compromise regardless of sex.
PAI-1 (675 4G/5G) genotypes were found across both sexes, with the 4G/5G heterozygous variant being the most common. This suggests that impaired fibrinolysis could be a shared mechanism contributing to intravascular clotting and osteonecrosis pathophysiology.
Overall, the chart underscores a multifactorial thrombophilic profile with varying sex-specific trends. Such data support the clinical utility of incorporating sex-stratified genetic screening in the diagnostic work-up of patients with suspected or established osteonecrosis, particularly when traditional risk factors are absent.
These findings support the hypothesis that thrombophilic genetic variants may contribute to the pathogenesis of aseptic osteonecrosis by promoting localized intravascular coagulation and impairing bone perfusion. The predominance of Factor V Leiden and PAI-1 mutations in this cohort aligns with literature emphasizing their role in hypercoagulable states. Such data reinforce the potential clinical utility of genetic screening in young or idiopathic cases of osteonecrosis.
The comparative analysis of functional coagulation markers between male and female patients with aseptic osteonecrosis revealed the following insights:

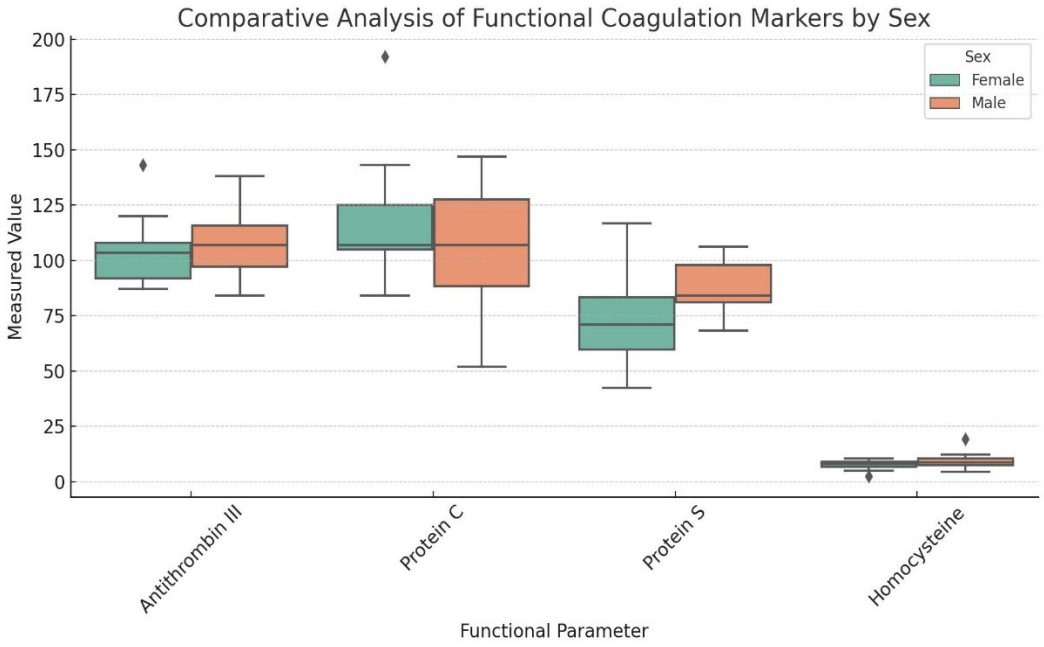

Fig 4. Distribution of functional coagulation tests by sex

1. Antithrombin III levels were slightly lower in males compared to females, though still within the normal reference range. A relative deficiency, even subclinical, may predispose to impaired thrombin neutralization and favor microvascular clot formation. Although both sexes showed values within the reference range, females demonstrated a slightly higher median and narrower interquartile range, suggesting more stable anticoagulant function compared to males.
2. Protein C activity demonstrated a minor sex-based variation, with females showing marginally higher mean values. This anticoagulant plays a central role in downregulating thrombin generation, and reduced function may contribute to thrombotic risk in osteonecrosis. Mean and median levels were relatively similar across sexes. However, slight variations in range may reflect individual variability in protein C synthesis or degradation, particularly under inflammatory or thrombotic conditions.

3. Protein S values showed greater variability, particularly in male patients. Given that free Protein S levels are physiologically higher in males, such fluctuations could reflect underlying prothrombotic predisposition when subnormal. Female patients tended to have slightly lower values, consistent with physiological reference differences. Notably, values closer to the lower limit in some cases may indicate functional impairment, particularly in estrogen-influenced states.
4. Homocysteine levels were elevated in several individuals, particularly among males. Hyperhomocysteinemia is an established vascular risk factor, promoting endothelial dysfunction, oxidative stress, and platelet aggregation - all mechanisms implicated in the pathogenesis of ischemic bone necrosis.
These findings suggest that functional deficiencies or borderline impairments in natural anticoagulants and metabolic markers like homocysteine could exacerbate thrombotic tendencies and contribute to the microcirculatory compromise characteristic of aseptic osteonecrosis. They highlight the relevance of incorporating sex-specific reference thresholds and personalized risk stratification in the clinical management of patients with suspected thrombophilia-associated bone ischemia.
Among the 28 patients analyzed, a subset presented with elevated levels of key functional coagulation parameters - specifically Antithrombin III, Protein C, Protein S, and Homocysteine. These individuals were further assessed to determine whether their hypercoagulability profiles coincided with underlying genetic thrombophilia (Figure 5).

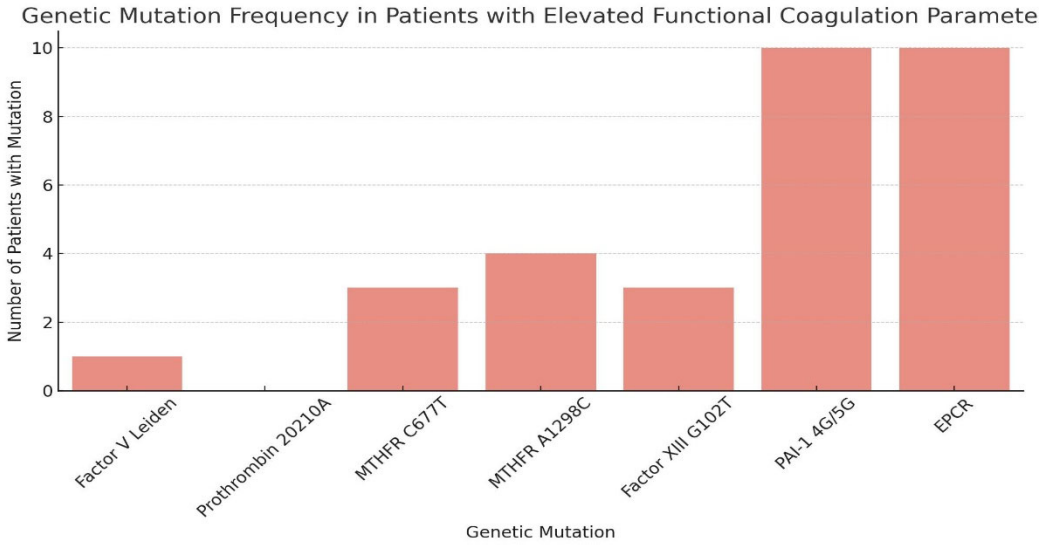

Fig.5. Genetic Mutation frequency in patients with elevated functional coagulation tests

Patients with elevated homocysteine and/or high activity of coagulation inhibitors often harbored one or more thrombophilic mutations, suggesting a compound prothrombotic state.
The most commonly observed mutations in this subgroup were:
o PAI-1 4G/5G polymorphism, detected in the majority of elevated cases.
o Factor V Leiden, also present in several individuals.
o MTHFR variants, particularly C677T, which are mechanistically linked to hyperhomocysteinemia.
Interestingly, even among those with supranormal antithrombin or protein C/S activity, genetic prothrombotic risk persisted, suggesting that functional compensations might coexist with inherited thrombogenic potential.
This analysis supports a model in which elevated coagulation markers are not purely compensatory or reactive, but may in fact coexist with genetic mutations, amplifying thrombotic risk. This reinforces the clinical importance of dual assessment—both genetic and functional—in patients with osteonecrosis or unexplained hypercoagulable states.

In the present cohort of 28 patients diagnosed with aseptic osteonecrosis, we evaluated the distribution of procoagulant status, stratifying individuals into three categories: normal, moderately elevated, and elevated, based on the cumulative findings from both functional and genetic thrombophilia panels (figure 6).

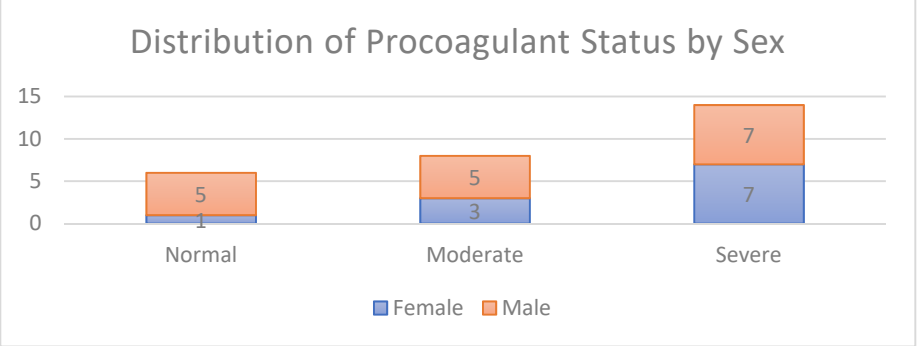

Fig. 6. Distribution of procoagulant status by sex

When stratified by sex, distinct patterns emerged:

Male patients were disproportionately represented in the moderately elevated and elevated procoagulant status categories. This suggests a higher burden of thrombogenic risk factors among men, potentially reflecting a combination of inherited thrombophilic mutations (such as Factor V Leiden or Prothrombin G20210A) and metabolic alterations (e.g., hyperhomocysteinemia). This finding is consistent with literature that associates male sex with more severe or earlier-onset forms of osteonecrosis, possibly due to a greater predisposition to microvascular thrombotic events.

Female patients, showed a more even distribution, with a significant number maintaining a normal or only mildly altered coagulation profile. However, several female patients did present with elevated procoagulant markers, reinforcing the concept that osteonecrosis is a multifactorial disease not exclusively dependent on sex, but rather on the interplay between hormonal, genetic, and behavioral risk factors (e.g., smoking, alcohol).

From a clinical perspective, these findings support the notion that prothrombotic status should be routinely assessed in all patients with osteonecrosis, particularly in male individuals or those with idiopathic or multifocal disease presentations. Early identification of a hypercoagulable profile may inform decisions regarding secondary prevention strategies, including thromboprophylaxis or lifestyle modification.

As part of this observational study, all 28 patients diagnosed with aseptic osteonecrosis completed a structured online questionnaire assessing their consumption of alcohol and tobacco. The instrument included 10 standardized questions related to alcohol use and 2 additional questions on smoking behavior, assigning a score to estimate the level of behavioral risk associated with vascular and bone health.

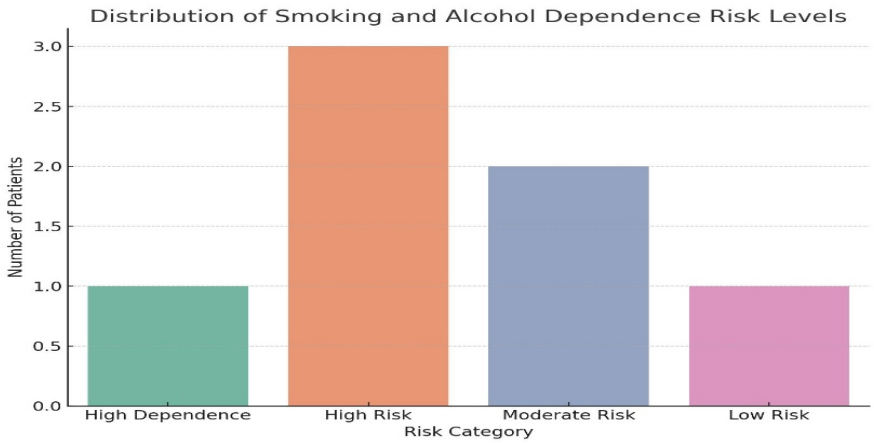

Fig. 7 - Distribution of smoking and alcohol dependence risk levels

Date collected showed the following (figure 7):
1 patient (3.6%) was classified as having high dependence, with a total score of 22 points. This patient was over 60 years old, a chronic smoker for 48 years, and presented the highest risk profile in the cohort.
3 patients (10.7%) fell into the high-risk category:
o One was over 60, smoked two packs per day for 35 years, and scored 16.
o Another, also over 60, had a 35-year smoking history, with a score of 18.
o The third was aged 41–60, had smoked for 16 years, and scored 16.
2 patients were categorized as having moderate risk, both aged 41–60, though specific smoking durations were not reported.
The remaining patients (78.6%) were placed in the low-risk group, indicating minimal or no current behavioral impact from tobacco or alcohol use.
The data show a clear clustering of elevated behavioral risk among older male smokers, supporting the hypothesis that long-term exposure to tobacco and alcohol may act synergistically with prothrombotic mechanisms, contributing to the pathophysiology of osteonecrosis. The high-dependence patient, who had the longest smoking history, aligns with prior research linking chronic endothelial injury and vasoconstriction to impaired subchondral perfusion.
This behavioral risk stratification not only adds depth to the biological profile of each patient but also reinforces the need for integrated interventions targeting modifiable lifestyle factors in the prevention and management of osteonecrosis.

4. Conclusion

This cross-sectional, observational, and analytical study conducted on a cohort of 28 patients with aseptic osteonecrosis has revealed a complex interplay between genetic, functional, and behavioral prothrombotic risk factors. The findings underscore the multifactorial nature of this ischemic bone pathology, in which both intrinsic and extrinsic determinants converge to compromise subchondral vascular integrity.
A substantial proportion of patients exhibited genetic polymorphisms associated with thrombophilia—most notably Factor V Leiden, the PAI-1 4G/5G genotype, and MTHFR mutations - often coexisting with functional impairments in natural anticoagulants such as protein C, protein S, and antithrombin III. Elevated plasma homocysteine levels, observed in a significant subset, further accentuated the prothrombotic profile and correlated modestly with these genetic alterations.
Sex-based stratification demonstrated a predominance of moderately to severely elevated procoagulant status among male patients, suggesting a heightened thrombotic predisposition that may underlie the pathogenesis of osteonecrosis in this subgroup. Additionally, the behavioral risk assessment revealed that high-risk or dependent patterns of alcohol and tobacco use were more frequently identified in older males with prolonged smoking histories, reinforcing the role of modifiable lifestyle factors as aggravating contributors to microvascular compromise.
Collectively, these findings advocate for a comprehensive, multidimensional diagnostic approach in patients with osteonecrosis, integrating genetic screening, coagulation profiling, and behavioral evaluation. Early identification of a hypercoagulable state—whether hereditary, acquired, or behaviorally induced—may allow for tailored preventive strategies and targeted therapeutic interventions aimed at mitigating disease progression and improving patient outcomes.

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References

1. Stanciu L. I., Blendea D. C., Teoibas-Serban D., Cirstoiu C. Where Endocrinology Meets Orthopaedics: A Comprehensive Review of Aseptic Bone Necrosis in Cushing's Syndrome. Balneo and PRM Research Journal. 2025, 16(2):823. doi: https://doi.org/ 10.12680/balneo.2025.823.
2. Chotanaphuti T, Heebthamai D, Chuwong M, Kanchanaroek K. The prevalence of thrombophilia in idiopathic osteonecrosis of the hip. J Med Assoc Thai., 2009,92, Suppl 6, S141-6, PMID: 20120676.
3. Garcia FL, Ramalli EL, Picado CH. Coagulation disorders in patients with femoral head osteonecrosis. Acta Ortop Bras., 2013,21(1), 43-5, doi: 10.1590/S1413-78522013000100009.
4. Jones LC, Mont MA, Le TB, Petri M, Hungerford DS, Wang P, Glueck CJ. Procoagulants and osteonecrosis. J Rheumatol. 2003, 30(4), 783-91, PMID: 12672200.
5. Korompilias AV, Gilkeson GS, Ortel TL, Seaber AV, Urbaniak JR. Anticardiolipin antibodies and osteonecrosis of the femoral head. Clin Orthop Relat Res., 1997, (345), 174-80, PMID: 9418637.
6. Orth P, Anagnostakos K. Coagulation abnormalities in osteonecrosis and bone marrow edema syndrome. Orthopedics. 2013, 36(4), 290-300, doi: 10.3928/01477447-20130327-08.
7. Bernbeck B, Christaras A, Krauth K, Lentrodt S, Strelow H, Schaper J, Janssen G, Mödder U, Göbel U. Bone marrow oedema and aseptic osteonecrosis in children and adolescents with acute lymphoblastic leukaemia or non-Hodgkin-lymphoma treated with hyperbaric-oxygen-therapy (HBO): an approach to cure? -- BME/AON and hyperbaric oxygen therapy as a treatment modality. Klin Padiatr., 2004, 216(6), 370-8, doi: 10.1055/s-2004-832341.
8. Liu, W., Yue, J., Guo, X. et al. Epidemiological investigation and diagnostic analysis of osteonecrosis of the femoral head in three northeastern provinces of China. J. Orthop. Surg. Res., 19, 292 (2024), doi:10.1186/s13018-024-04768-y