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Original Research Article

PREVALENCE OF MULTIVITAMIN DEFICIENCY AND OBESITY IN ASSOCIATION WITH VARICOSE VEINS IN A SOUTH INDIAN POPULATION: A CROSS-SECTIONAL STUDY

Kritika Jakhar[1], Jayanth Lavu[2]

[1]Final Year Student, All India Institute of Medical Sciences (AIIMS), Mangalagiri, Andhra Pradesh, India.
[2]MS Geneal Surgery, Department of General Surgery, All India Institute of Medical Sciences (AIIMS), Mangalagiri, Andhra Pradesh, India.

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Corresponding Author:
Dr. Jayanth Lavu,
MS Geneal Surgery, Department of General Surgery, All India Institute of Medical Sciences (AIIMS), Mangalagiri, Andhra Pradesh, India.
Email: lavujayanth@aiimsmangalagiri.edu.in

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ABSTRACT

Background: Varicose veins are a prevalent chronic venous disorder, particularly among individuals in prolonged-standing occupations. While obesity is a known risk factor, the role of nutritional deficiencies, specifically Vitamin D, in venous wall integrity and disease progression is gaining research interest. The objective is to determine the prevalence of obesity and multivitamin deficiencies (D, B12, and folic acid) and evaluate their correlation with varicose vein severity (CEAP classification) in a South Indian population.

Materials and Methods: A cross-sectional study was conducted at AIIMS Mangalagiri involving 189 adult patients. Clinical evaluation via CEAP classification was paired with laboratory analysis of Vitamin D, B12, folic acid, and coagulation profiles (PT/INR).

Results: The mean age was 48.6 years, with 85% of patients being overweight or obese. Vitamin D deficiency was strikingly prevalent at 84%. Standing occupations (58%) and smoking (32%) were significant lifestyle factors. Advanced disease (CEAP C5–C6) was present in 23% of cases.

Conclusion: Obesity and severe Vitamin D deficiency are major contributors to varicose veins in this population. Integrative management focusing on weight reduction and Vitamin D supplementation is recommended.

Keywords: Varicose Veins, Obesity, Vitamin D Deficiency, CEAP Classification, South India, Phlebesity.

INTRODUCTION

Varicose veins are characterized by the dilatation, tortuosity, and incompetence of superficial veins, primarily in the lower limbs. They represent a significant global health burden, affecting 20–30% of adults. Untreated cases often progress to chronic venous insufficiency (CVI), resulting in edema and venous ulceration. Recent research suggests that nutritional status, particularly Vitamin D levels, may influence endothelial function and venous wall integrity. While obesity and smoking are recognized aggravating factors, there is limited biochemical correlation data specifically for the South Indian population. This study aims to address this gap by exploring the prevalence of obesity and multivitamin deficiencies in these patients.[1]

Review of Literature: The etiopathogenesis of varicose veins involves venous hypertension, valvular incompetence, and structural deterioration of the venous wall.[2]

Obesity and "Phlebesity": Obesity is a consistent risk factor; studies show a significant association between elevated BMI and symptomatic varicose veins (OR 3.28 for obesity). This relationship is likely causal, as genetically predicted higher BMI increases susceptibility to the condition. The term "phlebesity" describes the cluster of obesity and venous disease driven by increased intra-abdominal pressure and chronic inflammation.

The Role of Vitamin D: Vitamin D regulates vascular homeostasis and nitric oxide (NO) pathways. Deficiency may exacerbate venous wall dysfunction by increasing oxidative stress. Evidence

indicates that Vitamin D supplementation can improve endothelial nitric oxide synthase (eNOS) expression. Furthermore, deficiency is linked to higher risks of venous thromboembolism (VTE) and impaired healing of venous ulcers.

Other Factors: While Vitamin B12 and folate regulate homocysteine, their direct association with varicose veins remains weak compared to Vitamin D. Lifestyle factors, such as prolonged standing and smoking, further modulate risk through gravitational pooling and endothelial injury.[3]

Aims & Objectives

The primary objectives of this study are:

- To determine the prevalence of obesity (BMI >25 kg/m²) among South Indian varicose vein patients.
- To assess the prevalence of Vitamin D, Vitamin B12, and folic acid deficiencies.
- To evaluate correlations between BMI, vitamin levels, clotting factors (PT/INR), and CEAP severity.

MATERIALS AND METHODS

Study Design: Cross-sectional study.

Setting: General Surgery OPD & IPD of AIIMS Mangalagiri.

Population: 189 consecutive adult patients diagnosed with varicose veins.

Inclusion/Exclusion

Patients aged ≥18 years were included; those with previous vascular surgery, pregnancy, or severe comorbidities were excluded.

Data Collection

Demographics, BMI measurement, and CEAP clinical classification were recorded.

Laboratory Analysis

Serum levels of Vitamin D, B12, and folic acid were measured alongside PT and INR.

RESULTS

Patient Demographics

The mean age of the participants was 48.6 ± 11.9 years, with the 40–60 age group being most affected. A slight female predominance (52%) was noted.

BMI and Occupation

A total of 85% of cases were either overweight or obese. Occupational analysis revealed that 58% of patients (111/189) held high-risk jobs involving prolonged standing.

Table 1: BMI Distribution

Category	Count	%
Obese	89	47%
Overweight	71	38%
Normal	29	15%

Clinical Severity (CEAP)

Advanced stages (C5–C6), indicating healed or active ulcers, accounted for 23% of the cohort.

Table 2: Distribution accordance to CEAP Classification

CEAP Type	Count	Interpretation
C3EpAsPr	67	Edema
C4EpAsPr	34	Skin changes
C2EpAsPr	33	Varicosities
C5EpAsApPr	32	Healed ulcers
C6EpAsApPr	12	Active ulcers
Advanced stages (C5–C6): 44 cases indicating late presentation.		

Biochemical Profile

Vitamin D deficiency was the most significant finding, affecting 84% of the population.

Table 3: Vitamin and Coagulation Profile

Parameter	Mean	Deficiency	Interpretation
Vitamin D	15 ng/mL	159 (84%)	Severe deficiency
Vitamin B12	283 pg/mL	8 (4%)	Mostly normal
Folic Acid	6.3 ng/mL	4 (2%)	Mostly normal
Prothrombin Time	14.05 sec	—	Normal
Vitamin D deficiency was the most striking finding (84%).			

DISCUSSION

This cross-sectional study highlights a compelling triad in the South Indian population with varicose veins: obesity, severe Vitamin D deficiency, and advanced clinical presentation. The findings reinforce the multifactorial pathophysiology of chronic venous disease (CVD), wherein mechanical, metabolic, and biochemical factors act synergistically.[4]

The observation that 85% of patients were overweight or obese strongly supports the concept of *phlebesity*, a term describing the coexistence of

obesity and venous disease. Elevated BMI contributes to increased intra-abdominal pressure, impaired venous return, and sustained venous hypertension. Chronic venous hypertension promotes valvular incompetence, venous dilation, and inflammatory remodeling of the venous wall.

Beyond mechanical stress, obesity induces a pro-inflammatory milieu characterized by elevated cytokines (TNF-α, IL-6) and oxidative stress. These mediators accelerate endothelial dysfunction and extracellular matrix degradation, worsening venous wall compliance. The high prevalence of advanced CEAP stages (C5–C6, 23%) in our cohort may therefore reflect prolonged exposure to both hemodynamic and inflammatory stressors. The association aligns with previous epidemiological and genetic data demonstrating a causal link between increased BMI and symptomatic varicose veins.[5]

The most striking biochemical abnormality in our study was Vitamin D deficiency in 84% of patients, with a mean serum level of 15 ng/mL, indicating severe deficiency. This prevalence is substantially higher than general community estimates, suggesting a possible disease-specific association.

Vitamin D plays a critical role in vascular homeostasis. It modulates endothelial nitric oxide synthase (eNOS) activity, enhances nitric oxide bioavailability, and reduces oxidative stress. Deficiency may impair endothelial-dependent vasodilatation and promote venous wall stiffness. Experimental studies have shown that Vitamin D supplementation can improve endothelial function and attenuate inflammatory signaling within vascular tissues.[6]

Furthermore, Vitamin D influences matrix metalloproteinases (MMPs), which regulate collagen and elastin turnover in the venous wall. Dysregulation of MMP activity contributes to structural weakening and venous dilation. In advanced CEAP stages (C5–C6), ulcer formation and delayed healing may partly result from impaired angiogenesis and persistent inflammation—both processes modulated by Vitamin D. The high proportion of ulcerative disease in this study strengthens the hypothesis that deficiency may influence disease progression rather than merely coexist.

Additionally, emerging evidence links low Vitamin D levels with increased risk of venous thromboembolism (VTE). Although PT/INR values were normal in our cohort, the prothrombotic risk associated with deficiency may operate via endothelial and inflammatory pathways rather than overt coagulation abnormalities.

In contrast to Vitamin D, deficiencies of Vitamin B12 (4%) and folic acid (2%) were uncommon. These vitamins regulate homocysteine metabolism, and hyperhomocysteinemia has been implicated in vascular dysfunction. However, their minimal deficiency prevalence in this cohort suggests that homocysteine-mediated endothelial injury may not be a dominant mechanism in varicose vein pathogenesis within this population.

Similarly, normal PT and INR values indicate preserved coagulation profiles, reinforcing that structural and inflammatory venous wall changes, rather than systemic coagulopathy, are central to disease expression in these patients.

More than half of the participants (58%) were engaged in prolonged-standing occupations, a recognized risk factor due to gravitational venous pooling and sustained venous hypertension. Smoking (32%) likely compounds endothelial injury through oxidative stress and impaired microcirculation. When combined with obesity and Vitamin D deficiency, these factors may create a "multi-hit" environment accelerating disease progression. The finding that nearly one-quarter of patients presented in advanced stages (C5–C6) underscores delayed healthcare seeking and possible lack of awareness regarding early symptoms. In a resource-limited setting, ulcer care imposes significant socioeconomic burden. Early screening—particularly in high-risk individuals with obesity and occupational exposure—could reduce progression to chronic venous insufficiency and ulceration.[7]

Integrated Pathophysiological Model

The data support a composite model wherein:

- Obesity → Increased venous pressure + chronic inflammation
- Vitamin D deficiency → Endothelial dysfunction + oxidative stress + impaired venous wall remodeling
- Prolonged standing/smoking → Sustained venous hypertension + microvascular injury

Together, these factors drive valvular incompetence and structural venous deterioration, culminating in advanced CEAP stages.

Clinical Implications

Given the high prevalence of modifiable risk factors, management should extend beyond procedural interventions such as endovenous ablation. A comprehensive strategy including:

- Weight reduction programs
- Routine screening for Vitamin D deficiency
- Targeted supplementation
- Occupational counseling
- Smoking cessation

May slow progression and reduce ulcer formation.

CONCLUSION

Obesity, prolonged standing, and Vitamin D deficiency are the primary contributors to varicose veins in South Indian patients. Vitamin D deficiency is exceptionally prevalent and may influence the development of ulcers. Management should include weight reduction, occupational modifications, and targeted Vitamin D supplementation to slow disease progression.

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