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Clinical Profile and Risk Factors of Venous Thrombosis: A Prospective Observational Study from a Tertiary Care Center in South India

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ABSTRACT

Background: Venous thromboembolism (VTE), encompassing deep vein thrombosis (DVT), pulmonary embolism (PE), and cerebral venous thrombosis (CVT), is a major cause of morbidity and mortality. Identifying clinical and laboratory risk factors is crucial for early diagnosis and prevention, particularly in resource-limited settings.

Methods: A prospective descriptive study was conducted over a two-year period (November 2017–November 2019) at a tertiary care teaching hospital in South India. A total of 59 patients with radiologically confirmed venous thrombosis were included. Clinical data, comorbidities, and laboratory parameters, including complete blood count, ESR, CRP, serum homocysteine, and antiphospholipid antibodies, were collected. Statistical analysis was performed using SPSS version 17. Associations were assessed using Chi-square or Fisher's exact test, and p-values <0.05 were considered significant.

Results: Among 59 patients, the majority were male (≈80%), with peak incidence in the 20–30 and 40–50-year age groups. DVT was the most common presentation (≈60%), followed by CVT (≈20%). Alcohol consumption (35.6%), High BMI (45%), and H/o prior thrombosis (20.3%) were frequent risk factors. Comorbidities included hypertension (30.5%), diabetes mellitus (27%), and chronic liver disease (30.5%). Elevated serum homocysteine (>15 µmol/L) was observed in 78%, and elevated CRP in 81% of cases. Polycythemia (20.3%) and H/o recent surgery (6.8%) were also associated with thrombosis. Significant associations were noted between patients with diabetes and chronic liver disease with pulmonary embolism.

Conclusion: Venous thrombosis in this cohort predominantly affected young to middle-aged males and was frequently unprovoked. Modifiable risk factors such as obesity, alcohol use, and metabolic comorbidities were prevalent. Elevated inflammatory markers and homocysteine levels highlight the role of inflammation and metabolic dysregulation in thrombogenesis.

Keywords: *Venous thromboembolism; Deep vein thrombosis; Pulmonary embolism; Cerebral venous thrombosis; Homocysteine; Risk factors.*

INTRODUCTION

Venous thromboembolism (VTE), comprising deep vein thrombosis (DVT), pulmonary embolism (PE), and cerebral venous thrombosis (CVT), represents a major global health burden with significant morbidity and mortality. It is increasingly recognized as a preventable yet underdiagnosed condition that contributes substantially to hospital-related complications and long-term disability. The annual incidence of VTE is estimated at approximately 1–2 per 1000 population, with a rising trend observed across both developed and developing countries, likely reflecting aging populations, increased prevalence of comorbid conditions, and improved diagnostic capabilities. [1,2] Despite advances in clinical awareness and imaging modalities, VTE continues to pose a considerable challenge in terms of timely diagnosis and effective risk stratification.

DVT and PE together contribute substantially to mortality, accounting for nearly 100,000 deaths annually in the United States alone. [3,4] Pulmonary embolism, in particular, is associated with significant early mortality due to hemodynamic compromise and right ventricular failure, with up to 40% of patients dying within three months of diagnosis if not promptly treated. [5,6] Even among survivors, the long-term consequences are considerable, including chronic thromboembolic pulmonary hypertension and post-thrombotic syndrome, which impair quality of life. Furthermore, recurrence remains a major concern, with nearly 30% of patients experiencing recurrent thromboembolic events within 10 years, particularly in cases of unprovoked thrombosis. [7] These findings underscore the importance of early identification of high-risk individuals and implementation of preventive strategies.

Cerebral venous thrombosis, although less common compared to DVT and PE, has gained increasing recognition in recent years due to improved neuroimaging techniques and heightened clinical suspicion. The estimated incidence ranges from 2–5 per million annually; however, recent population-based studies suggest higher incidence rates, especially among younger individuals and women of reproductive age. [8,9] Clinically, CVT presents with a wide spectrum ranging from isolated headache to seizures, focal neurological deficits, and altered consciousness, making diagnosis challenging. Unlike arterial thrombosis, which is predominantly driven by atherosclerosis, venous thrombosis is multifactorial and is classically explained by Virchow's triad: venous stasis, endothelial injury, and hypercoagulability. [10] These interrelated mechanisms provide a framework for understanding the diverse etiological factors involved in thrombus formation.

Risk factors for VTE are heterogeneous and include both acquired and inherited components. Common acquired risk factors include prolonged immobilization, major surgery, trauma, malignancy, obesity, and chronic medical illnesses such as cardiovascular and metabolic disorders. [11–13] In addition, emerging evidence highlights the role of biochemical abnormalities in thrombogenesis. Hyperhomocysteinemia has been associated with endothelial dysfunction and enhanced coagulation activation, while elevated inflammatory markers such as C-reactive protein reflect an underlying proinflammatory state that may predispose to thrombus formation. [14,15] The interplay between inflammation, coagulation, and endothelial injury further supports the concept of thrombosis as a dynamic and systemic process rather than a purely localized event.

In resource-limited settings, comprehensive thrombophilia evaluation, including genetic testing, is often not feasible due to financial and logistical constraints. Consequently, there is a critical need to identify clinically relevant, accessible, and cost-effective predictors of venous thrombosis that can aid in early diagnosis and risk stratification. Understanding the clinical profile and associated risk factors in such settings is essential not only for improving patient outcomes but also for guiding preventive strategies tailored to local populations.

Objectives

1. To study the clinical profile of venous thrombosis
2. To identify associated clinical and laboratory risk factors

METHODS

This Prospective observational analytical study was conducted at a tertiary care hospital in South India over a period of two years, from November 2017 to November 2019. A total of 59 consecutive patients with radiologically confirmed venous thrombosis were included in the study.

Eligible participants were adults aged 18 years or older with a confirmed diagnosis of venous thrombosis based on appropriate imaging modalities. Only patients willing to provide written informed consent were included. Patients who refused to participate or had incomplete or missing clinical or laboratory data were excluded from the study.

Detailed clinical data were systematically collected for all participants. This included demographic information (age and sex), clinical presentation, and site of thrombosis. Personal history variables, including alcohol consumption and body mass index (BMI), were recorded. Information regarding comorbid conditions such as diabetes mellitus, hypertension, chronic kidney disease (CKD), chronic liver disease (CLD), ischemic heart disease (IHD), and cerebrovascular accident (CVA) was obtained. In addition, past history of venous thrombosis and recent history of surgery or immobilization were documented.

All patients underwent a standardized set of laboratory investigations. These included complete hemogram, erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), peripheral blood smear examination, serum homocysteine levels, antiphospholipid antibody (APLA) testing, and lipid profile. These investigations were selected to evaluate both hematological and biochemical factors associated with thrombogenesis.

The primary outcome of interest was the type and distribution of venous thrombosis, categorized as deep vein thrombosis, cerebral venous thrombosis, pulmonary embolism, or visceral thrombosis. Secondary outcomes included associations between various clinical and laboratory risk factors and different types of venous thrombosis.

Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) version 17. Categorical variables were expressed as frequencies and percentages. Associations between variables were assessed using the Chi-square test or Fisher's exact test, as appropriate. A p-value of less than 0.05 was considered statistically significant.

The study was conducted in accordance with ethical principles and was approved by the Institutional Ethics Committee . Written informed consent was obtained from all participants prior to enrollment.

RESULTS

Table 1: Baseline Characteristics, Clinical Profile, and Risk Factors (n=59)

Variable	Category	Frequency (%)
Age (years)	20–30	18 (30.5%)
	40–50	15 (25.4%)
Gender	Male	47 (79.7%)
	Female	12 (20.3%)
Type of Thrombosis	DVT	35 (59.3%)
	CVT	12 (20.3%)
	PE	7 (11.9%)
	Visceral	5 (8.5%)
Risk Factors	BMI >25 kg/m²	27 (45.8%)
	Alcohol use	21 (35.6%)
	Previous thrombosis	12 (20.3%)
	Surgery (<12 weeks)	4 (6.8%)
	Varicose veins	4 (6.8%)
Comorbidities	Hypertension	18 (30.5%)
	Diabetes mellitus	16 (27.1%)
	Chronic liver disease	18 (30.5%)
	CKD	9 (15.3%)
	IHD	5 (8.5%)
	CVA	4 (6.8%)
Laboratory Findings	Homocysteine >15 µmol/L	46 (78.0%)
	Elevated CRP	48 (81.4%)
	Polycythemia	12 (20.3%)
	APLA positive	2 (3.4%)

As shown in Table 1, the study population demonstrated a marked male predominance (79.7%), with peak incidence in the 20–30-year age group (30.5%), followed by the 40–50-year age group (25.4%). Deep vein thrombosis was the most common presentation (59.3%), followed by cerebral venous thrombosis (20.3%) and pulmonary embolism (11.9%). Among risk factors, Higher BMI (>25 kg/m²) was observed in 45.8% and alcohol use in 35.6% of patients. Comorbidities such as Hypertension and chronic liver disease were each present in 30.5% of cases. Laboratory abnormalities were notable, with elevated CRP (81.4%) and hyperhomocysteinemia (78%) seen in the majority of patients, indicating a strong inflammatory and metabolic component.

Table 2: Association of Risk Factors with Type of Venous Thrombosis

Risk Factor	DVT (n=35)	CVT (n=12)	PE (n=7)	p-value
BMI >25 kg/m²	20 (57.1%)	5 (41.7%)	2 (28.6%)	<0.05
Alcohol use	13 (37.1%)	6 (50.0%)	2 (28.6%)	0.08
Previous thrombosis	8 (22.9%)	3 (25.0%)	1 (14.3%)	0.62
Surgery	2 (5.7%)	1 (8.3%)	1 (14.3%)	0.41
Varicose veins	3 (8.6%)	1 (8.3%)	0 (0%)	0.73

As illustrated in Table 2, elevated BMI (>25 kg/m²) was statistically significantly associated with thrombosis subtype ($p<0.05$), with the highest prevalence in DVT (57.1%). Alcohol consumption was more frequently observed among CVT patients (50%) compared to DVT (37.1%) and PE (28.6%), although this did not reach statistical significance ($p=0.08$). Other factors, such as prior thrombosis, recent surgery, and varicose veins, were relatively evenly distributed across subtypes, with no significant associations. These findings suggest that obesity may have a preferential association with DVT, whereas other risk factors contribute more broadly across thrombosis types.

Table 3: Association of Comorbidities with Venous Thrombosis

Comorbidity	DVT	CVT	PE	p-value
Diabetes mellitus	8 (22.9%)	2 (16.7%)	6 (85.7%)	<0.01

Hypertension	10 (28.6%)	3 (25%)	5 (71.4%)	<0.05
Chronic liver disease	9 (25.7%)	3 (25%)	6 (85.7%)	<0.01
CKD	5 (14.3%)	2 (16.7%)	2 (28.6%)	0.21

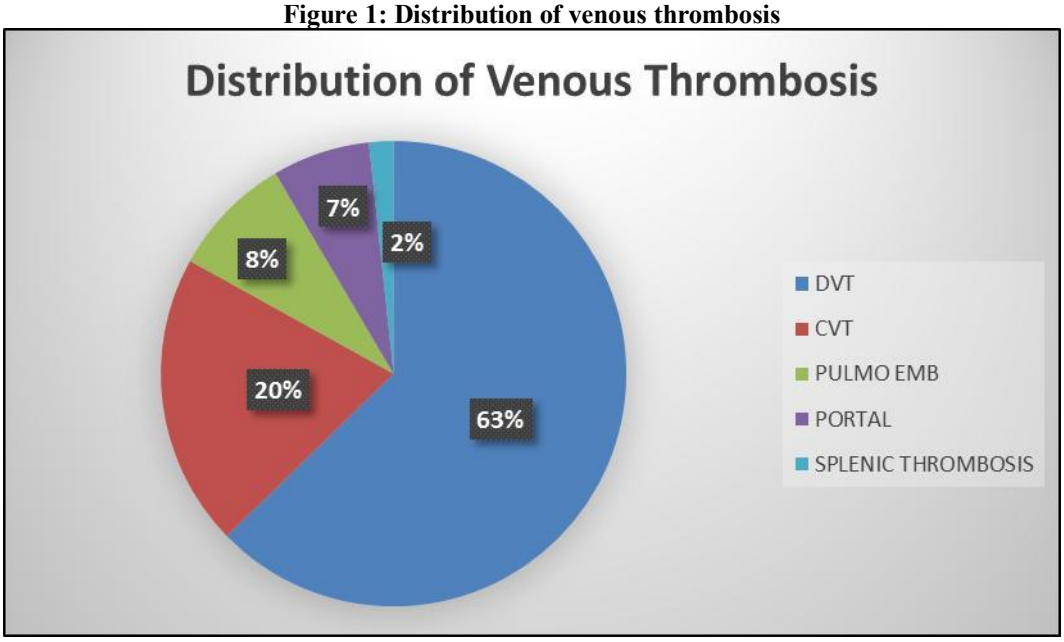
As shown in Table 3, systemic comorbidities were strongly associated with pulmonary embolism. Diabetes mellitus was present in 85.7% of PE cases compared to 22.9% in DVT and 16.7% in CVT ($p<0.01$). Similarly, chronic liver disease was observed in 85.7% of PE patients ($p<0.01$), indicating a significant relationship. Hypertension was also more prevalent in PE (71.4%, $p<0.05$), whereas chronic kidney disease did not differ significantly across groups. These findings highlight the role of metabolic and systemic conditions in influencing thrombus severity and embolic risk.

Table 4: Association of Laboratory Parameters with Thrombosis

Parameter	DVT (n=35)	CVT (n=12)	PE (n=7)	p-value
Homocysteine >15 µmol/L	28 (80.0%)	10 (83.3%)	6 (85.7%)	0.67
Elevated CRP	30 (85.7%)	9 (75.0%)	6 (85.7%)	<0.05
Polycythemia	10 (28.6%)	2 (16.7%)	0 (0%)	<0.05
APLA	1 (2.9%)	1 (8.3%)	0 (0%)	0.48

As shown in Table 4, hyperhomocysteinemia was highly prevalent across all thrombosis subtypes—DVT (80%), CVT (83.3%), and PE (85.7%)—with no significant intergroup variation ($p=0.67$), suggesting a generalized prothrombotic effect. Elevated CRP was observed in the majority of cases, particularly in DVT and PE (85.7%), and was significantly associated with thrombosis subtype ($p<0.05$). Polycythemia was predominantly associated with DVT (28.6%, $p<0.05$), while APLA positivity was infrequent and not statistically significant. Overall, inflammatory and hematological markers appear to play a central role in thrombogenesis.

Figure 1: Distribution of venous thrombosis



Among 59 patients, 37 (62.71%) had deep vein thrombosis of the limbs. 12 patients (20.3%) presented with cortical venous thrombosis; 5 patients (8.47%) had pulmonary embolism; 4 patients (6.7%) presented with portal vein thrombosis; and 1 patient presented with splenic vein thrombosis.

DISCUSSION

Demographic Profile

The present study demonstrated a marked male predominance (79.7%) with peak incidence in young adults (Table 1). Similar findings were reported by Silverstein et al., who observed higher incidence rates in males (130 per 100,000 vs 110 per 100,000). [1] Garg et al. reported male predominance of 72%. [16] Naess et al. documented increased incidence in younger males (5.2 per 1000 person-years). [17] However, Rosendaal et al. reported higher incidence in elderly populations (>60 years), with rates exceeding 5 per 1000 annually, indicating differing demographic patterns. [11]

Distribution of Thrombosis

In the present study, DVT was the predominant subtype (59.3%), as shown in Table 1. This is consistent with Heit et al., who reported DVT in 55–60% of cases. [2] Cushman et al. reported a similar proportion (58%). [18] The proportion of

CVT (20.3%) aligns with Dentali et al. (15–20%). [8] However, Stein et al. reported higher PE prevalence (30–40%), contrasting with the lower rate (11.9%) observed in Table 1. [19]

Obesity and Lifestyle Factors
Elevated BMI (45.8%) was significantly associated with DVT, as demonstrated in Table 2. Ageno et al. reported obesity prevalence of 30–40% with a two-fold increase in VTE risk. [12] Stein et al. reported rates of approximately 33%. [20] Alcohol use (35.6%) was also notable (Table 1). Large studies, however, report lower prevalence (<20%), suggesting regional variation. [13]

Comorbidities
Diabetes mellitus (27.1%), hypertension (30.5%), and chronic liver disease (30.5%) were common in this study (Table 1), with significant associations with PE (Table 3). Tsai et al. reported diabetes prevalence of approximately 25%. [22] Glynn et al. reported hypertension in 28% of cases. [23] Chronic liver disease prevalence (30.5%) was higher than that reported by Northup et al. (20–25%). [24] However, some studies did not find a strong independent association between hypertension and VTE. [13]

Inflammation and Hyperhomocysteinemia
Hyperhomocysteinemia (78%) and elevated CRP (81.4%) were prominent findings (Table 1 and Table 4). Den Heijer et al. reported that homocysteine levels were elevated in approximately 65% of patients. [14] Ridker et al. demonstrated a 2–3 fold increased risk of VTE with elevated CRP. [15] Cushman et al. reported a 70% elevation in CRP. [18] However, Kyrle et al. observed that CRP may not independently predict thrombosis risk after adjustment. [25]

Polycythemia
Polycythemia was present in 20.3% of cases (Table 1) and was significantly associated with DVT (Table 4). Marchioli et al. reported that thrombotic events occurred in 15–20% of patients with polycythemia. [26] Tefferi et al. reported similar rates (~18%). [27] In contrast, the prevalence in the general population is lower (5–10%). [11]

Surgery and Provoking Factors
Recent surgery was observed in 6.8% of patients (Table 1). Heit et al. reported that surgical procedures contribute to 25% of VTE cases. [2] Anderson et al. reported a postoperative risk of 10–20%. [28] The lower prevalence in this study suggests a predominance of unprovoked thrombosis.

Strengths and Limitations
This study has several strengths, including its prospective design, systematic evaluation of a spectrum of venous thrombosis subtypes, and incorporation of clinically relevant and cost-effective laboratory markers such as C-reactive protein and homocysteine, which enhance its applicability in resource-limited settings. Additionally, the study provides an integrated analysis of demographic, clinical, and biochemical risk factors, offering a comprehensive overview of thrombosis patterns in a tertiary care population. However, certain limitations must be acknowledged. The relatively small sample size (n=59) and single-centre design may limit the generalizability of the findings. The absence of extensive inherited thrombophilia testing restricts evaluation of genetic predisposition, and the lack of long-term follow-up precludes assessment of recurrence rates and clinical outcomes. Furthermore, potential referral bias inherent to a tertiary care setting may have influenced the observed distribution of thrombosis subtypes.

CONCLUSION
In this prospective study of patients with venous thrombosis at a tertiary care center, deep vein thrombosis emerged as the predominant presentation, with a notable burden of disease among young and middle-aged males. The findings highlight the significant contribution of modifiable risk factors, particularly obesity and alcohol use, along with a high prevalence of metabolic comorbidities such as diabetes, hypertension, and chronic liver disease. Importantly, elevated inflammatory and biochemical markers—including C-reactive protein and homocysteine—were observed in the majority of patients, underscoring the role of inflammation and metabolic dysregulation in the pathogenesis of venous thrombosis. The strong association of certain comorbidities with pulmonary embolism further suggests heterogeneity in risk profiles across thrombosis subtypes. These results emphasize the need for early risk stratification using readily available clinical and laboratory parameters, especially in resource-limited settings. Larger, multicentric studies with longitudinal follow-up are warranted to validate these findings and to better define targeted preventive and therapeutic strategies.

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