

Prevalence of Factor V Leiden and Other Inherited Thrombophilic Disorders in Patients Presenting with Thrombotic Events

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ABSTRACT

Background: Inherited thrombophilic disorders, particularly Factor V Leiden mutation, are important yet underrecognized contributors to venous and arterial thrombotic events.

Objective: To determine the frequency of different causes of inherited thrombophilia and evaluate clinical presentations in patients presenting with documented venous or arterial thrombosis.

Methodology: One hundred and twelve participants meeting the selection criterion and offering written informed consent were recruited. This is a cross-sectional study and conducted at the Department of Medicine, Northwest School of Medicine, Peshawar, Pakistan from March 2023 to February 2024. Clinical presentations were recorded and patients were assessed under inherited thrombophilia. Factor V Leiden mutation and prothrombin gene mutation were detected by the use of polymerase chain reaction. The anticoagulatory activity of protein C and S had been evaluated by a Pro C global clotting-based screening assay, and quantitative analysis of the functional antithrombin activity through a CS5100 automated coagulation analyzer.

Results: The average age of the patients was 30.78 +/-14.88 Years. Thrombophilia hereditary was detected in 27 (25.0) patients. The results found factor V Leiden mutation in 15 (55.6%), antithrombin deficiency in 6 (22.2%), protein C deficiency in 3 (11.1%), protein S deficiency in 2 (7.4), and prothrombin gene mutation found in 1 (3.7) patient. Deep venous thrombosis was the most frequent clinical manifestation, as it occurred in 11 (40.7) patients, then pulmonary embolism, which occurred in 5 (18.5), portal vein thrombosis, occurring in 3 (11.1), and superficial venous thrombosis in 3 (11.1).

Conclusion: The most frequent inherited cause of thrombophilia was factor V Leiden mutation, and the most common presenting manifestation was deep venous thrombosis.

Keywords: Factor V Leiden, Inherited Thrombophilia, Venous Thromboembolism, Thrombotic Events, Genetic Thrombophilic Disorders

INTRODUCTION

Thrombosis is a leading cause of morbidity and mortality globally, and it plays a substantial role in the burden of cardiovascular disease in the world.^{1,2,3} The third cardiovascular disorder to address is venous thromboembolism (VTE) (deep venous thrombosis (DVT) and pulmonary embolism), the most frequently occurring after ischemic heart disease and stroke.^{4,5,6} The triad of Virchow (endothelial injury, venous stasis, and hypercoagulability) is the classical explanation of the appearance of thrombosis.^{7,8,9} Among them, hypercoagulability can be caused by factors that are either acquired or inherited, and inherited thrombophilia can be considered an essential position of cause within this group, especially in young people and those with repeated or unexplained examples of thrombotic occurrences.^{10,11} Inherited thrombophilia is a collection of hereditary disorders that increase the risk of thrombosis because of the defect in the natural system of anticoagulant or the pathways of coagulation.^{12,13,14} Factor V Leiden (FVL) mutation, a point mutation in the factor V gene (G 1691A), causing resistance to activated protein C (APC) and resulting in excessive production of thrombin and subsequent clotting, is the most prevalent inherited thrombophilic defect across the world.¹⁵ FVL is more prevalent in different populations, and its prevalence is 38 per cent in the general Caucasian population and 20.5 per cent among patients with VTE.¹⁶ Nevertheless, it is less prevalent among the Asian and African populations possibly indicating a high ethnic and geographical diversity.

The prothrombin gene mutation (G20210A), which increases the plasma prothrombin levels and predisposes to venous thrombosis is another significant inherited thrombophilic defect.¹⁷ It is the

second most frequent inherited thrombophilia in Western nations; nevertheless, its ratio is relatively low in Asian communities.¹⁸ Besides these mutations, inherited defects of natural anticoagulants such as antithrombin, protein C, and protein S are also well-known causes of thrombosis.¹⁹ The proteins are essential in the regulation of coagulation because they inhibit the activated factor of clots; hence, their deficiency predisposes thrombotic severity significantly. Specifically, Antithrombin deficiency is linked to an excessively high risk of venous thromboembolism throughout life.²⁰

In genetic thrombophilia, people are mainly at risk of developing venous not arterial thrombosis.²¹ The clinical presentation can be deep venous thrombosis, pulmonary embolism, portal vein thrombosis, superficial venous thrombosis, and repeat pregnancy loss.²² The presence of inherited defects combined with acquired risk factors like surgery, immobilization, pregnancy, oral contraceptives, malignancy, or systemic inflammatory conditions increases the risk of thrombosis.²³ Thus, the clinical significance of inherited thrombophilic condition diagnosis is to reduce the risk, preventing its development, and adjusted duration of anticoagulation therapy.²⁴

Although much is known about this subject among Western populations, little is known about prevalence and pattern of inherited thrombophilia among the South Asian populations and Pakistan is not an exception.²⁵ The difference in access to healthcare and environmental factors as well as genetic variation may affect the prevalence and clinical manifestation of these disorders in various groups. Additionally, regular routine screening of inherited thrombophilia is prevalent in most developing countries because of the prohibitive cost and the paucity of molecular diagnostic centers.²¹ Subsequently, the actual load of inherited thrombophilic diseases among patients with thrombotic events would be overshadowed.

The importance of early identification of inherited thrombophilia, especially young patients, is particularly observed in cases of unprovoked or repeated thrombotic events, as well as in patients with a positive family history.²⁶ The strategy of identifying a background genetic predisposition can inform clinical practice, such as long-term anticoagulation, prophylaxis in high-risk circumstances, and family-member counseling.²⁷ Nevertheless,

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the usefulness of routine thrombophilia testing is controversial, which shows that region-specific epidemiology is the source of informed evidence-based guidelines. Consequently, the research was done to identify the frequency of Factor V Leiden mutation and other inherited thrombophilic conditions among patients who presented with a known venous or arterial thrombosis at a tertiary care hospital in Pakistan. The study further sought to compare clinical presentations of these inherited disorders, and thus add to local data that has the potential of enhancing diagnostic approaches and management guidelines to thrombotic disorders.

METHODOLOGY

The study was a cross-sectional one, which was implemented within the conducted at the Department of Medicine, Northwest School of Medicine, Peshawar, Pakistan from March 2023 to February 2024. The purpose of the study was to establish the prevalence of the Factor V Leiden mutation and other inherited thrombophilic disorders in patients that have been reported to have experienced thrombotic events. Before starting with the study, an institutional review board in the hospital was enlisted to provide ethical approval. All the participants or their legal guardians provided informed consent in writing. Eleven patients that satisfied the pre-determined inclusion criteria were enrolled through the use of non-probability consecutive sampling method. Both male and female patients with objectively confirmed venous or arterial thrombosis, such as a deep venous thrombosis (DVT), pulmonary embolism (PE), portal vein thrombosis, and superficial venous thrombosis were involved. Thrombotic events were diagnosed based on the relevant imaging medium including Doppler ultrasonography of the thrombotic events, computed tomography pulmonary angiography (CTPA), or contrast-enhanced CT scan as per the indication. Acquired etiologies of thrombophilia like malignancy, pregnancy, prolonged immobilization, recent major surgery, liver disease, nephrotic syndrome, patients on anticoagulant therapy over two weeks before assessment were excluded so as to reduce confounding factors. A structured proforma was used to capture detailed demographic and clinical data, such as age, gender, type of thrombotic event, family history of thrombosis and recurrence of thrombotic episodes. Aseptic blood samples were taken using venous blood and tested in the laboratory to evaluate the inherited thrombophilic disorders. Movement Molecular Analysis Data revealed a specific mutation of the Factor V Leiden (G1691A) mutation and the prothrombin gene (G20210A) mutation by the use of polymerase chain reaction (PCR) methods that involve DNA extraction into peripheral blood leukocytes. Coagulation-based assays were used to measure conditions of functional deficiency of natural anticoagulants. Pro C Global clotting-based screening assay was used to analyze the mixture of anticoagulatory action of protein C and protein S. A CS5100 automated coagulation analyzer evaluated the quantitative functional activity of antithrombin as per the manufacturer. Any analysis that was done in the laboratory was conducted in the central diagnostic laboratory of the hospital in a standardized and quality-controlled manner. Statistical Package for Social Sciences version 25.0 was used to enter and analyze data. Continuous variables like age were reported in the form of mean standard deviation whereas categorical variables like type of mutation and clinical presentation were reported by means of frequencies and percentages. Prevalence rates of various inherited thrombophilic states were determined and descriptive statistics were employed to present clinical features of the study group. The p-value of 0.05 or less was regarded as statistically significant where necessary.

RESULTS

The study involved 112 patients who had reported some incidences of venous or arterial thrombosis. The study sample was

composed of a mean of 30.78 years old with a standard deviation of 14.88 years. They greatly represented both sexes, though with a slight predominance of female patients (distribution in Table 1). Twenty-seven (25.0) patients were found to have inherited thrombophilia, and the rest 85 (75.0) patients had no identifiable inherited thrombophilic disorder on screening (Table 2). The commonest abnormality was Factor V Leiden mutation (n = 15), which was seen in 55.6% of the patients (n = 27), with all diagnosed with inherited thrombophilia. This was subsequent to antithrombin deficiency in 6 (22.2), protein C deficiency in 3 (11.1) and protein S deficiency in 2 (7.4), and prothrombin gene mutation in 1 (3.7), patient (Table 3). As far as the clinical presentation of patients with inherited thrombophilia is concerned, the most prevalent appearance was deep venous thrombosis (DVT), detected in 11 (40.7) patients. The other presentations were pulmonary embolism (PE) in 5 (18.5%), portal vein thrombosis in 3 (11.1%), and superficial venous thrombosis in 3 (11.1%) patients (Table 4)

Table 1: Demographic Characteristics of Study Participants (n=112)

Variable	Value
Mean Age (years)	30.78 ± 14.88
Male	52 (46.4%)
Female	60 (53.6%)

Table 2: Frequency of Inherited Thrombophilia (n = 112)

Inherited Thrombophilia	Frequency	Percentage
Present	27	25.0%
Absent	85	75.0%

Figure 1

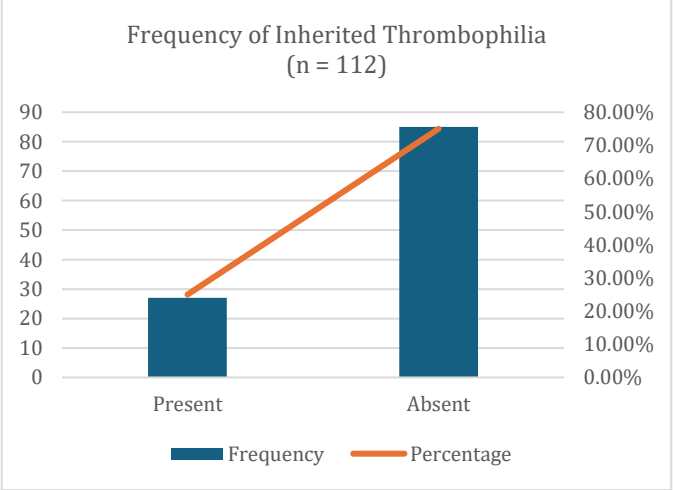


Table 3: Types of Inherited Thrombophilic Disorders (n = 27)

Type of Disorder	Frequency	Percentage
Factor V Leiden mutation	15	55.6%
Antithrombin deficiency	6	22.2%
Protein C deficiency	3	11.1%
Protein S deficiency	2	7.4%
Prothrombin gene mutation	1	3.7%

Table 4: Clinical Presentations in Patients with Inherited Thrombophilia (n = 27)

Clinical Presentation	Frequency	Percentage
Deep Venous Thrombosis (DVT)	11	40.7%
Pulmonary Embolism (PE)	5	18.5%
Portal Vein Thrombosis	3	11.1%
Superficial Venous Thrombosis	3	11.1%
Other thrombotic events	5	18.5%

Figure 2

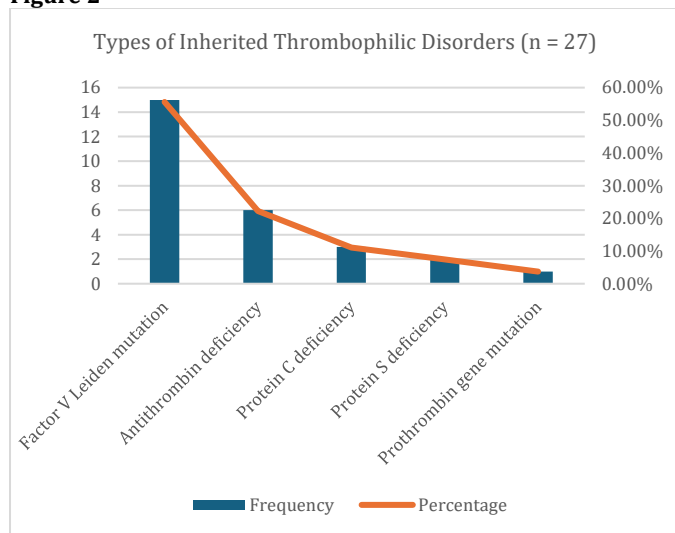
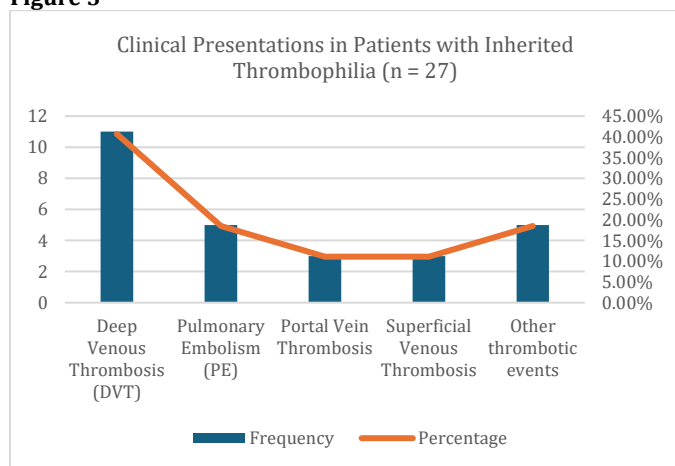


Figure 3



DISCUSSION

This cross-sectional research paper examined the proportion of Factor V Leiden (FVL) mutation, and other hereditary thrombophilic syndromes among patients who showed recorded cases of thrombotic incidence at a tertiary care hospital in Pakistan. The proportion of inherited thrombophilia in our cohort was 25.0, which aligns with a range of 20-40% in previous studies of selected populations with no known cause of thrombosis or a decreasing onset age (Khan, Altaf, Saeed Malik, Abdul Naeem, & Latif, 2021; Baccouche et al., 2022). These results support the clinical significance of inherited thrombophilia screening in young patients with a presentation of thrombotic events.¹²

Factor V Leiden mutation was the most common abnormality reported in our study with 55.6 percent of thrombophilia reported as inherited. This is in line with the world literature cited FVL as the most prevalent hereditary thrombophilic defect, which is especially prominent in Caucasian groups with a prevalence ranging 3-8 percent in the general population and 20-25 in patients with venous thromboembolism (VTE).^{26,27} Even though FVL is reported to be less common among Asian populations than western societies, new regional reports indicate that the prevalence rates are on the rise as a result of more effective molecular diagnostics and clinical awareness (Khan, Altaf, Saeed Malik, Abdul Naeem, and Latif, 2021). In our study, we propose that FVL mutation is still a prominent factor predisposing thrombosis even among the Pakistani population.¹²

The second highest prevalence was observed in antithrombin deficiency (22.2%), protein C deficiency (11.1%), and protein S deficiency (7.4%), respectively.^{4,5,6} These are well-recognized natural anticoagulant deficiencies, which are risk factors of venous thrombosis and are linked to an exceptionally high life-time risk of

re-occurring thromboembolic events (Tormene et al., 2020). Among them, specifically antithrombin deficiency is acknowledged as one of the most thrombogenic hereditary conditions, where severe venous thrombosis rates of 5-50 times higher based on the mutation type and the activity level are proven (Abbattista et al., 2020). Our cohort has a relatively low protein C and protein S deficiency frequency, which is consistent with the global epidemiological patterns.⁴

The mutation at prothrombin gene (G20210A) was only identified in 3.7% of the thrombophilia-positive patients. It is the second most frequent inherited thrombophilic defect after FVL in most Western populations but the prevalence is usually lower in Asian populations.^{25,26,27} Our study has a low rate of detection hence in line with regional measures of genetic distribution.

The most frequent clinical manifestation was deep venous thrombosis (DVT) (40.7% in the group of patients with inherited thrombophilia) and pulmonary embolism (18.5) (Khan, Altaf, Saeed Malik, Abdul Naeem, and Latif, 2021; Nachman, Pollack, and Herzog, 2021). This finding lends support to known evidence to the effect that inherited thrombophilic disorders mainly predispose subjects to venous, as opposed to arterial thrombosis.² Portal vein thrombosis and the superficial venous thrombosis to be observed in a certain group of patients also underscores the diversity of clinical presentations of episodes related to hypercoagulable conditions.^{8,9}

The moderate age of the study population (30.78 ± 14.88 years) highlights the significance of evaluating inherited thrombophilia among young people with a thrombotic event without any substantial acquired risk factors.^{26,27} The timely detection of such conditions has considerable consequences in the long-term management process, such as the length of anticoagulation therapy, thromboprophylaxis in potentially dangerous conditions (e.g., surgery, pregnancy), and the screening of relatives in the appropriate case.^{7,8}

There are some limitations of this research. The study is a cross-sectional, single-centered study; thus, it might not represent the entire population. We did not also consider other less prevalent genetic mutations or acquired thrombophilic conditions (such as antiphospholipid syndrome), which might have led to thrombotic risk in some patients.^{14,15} In spite of these drawbacks, the study offers useful regional information on the trends of inherited thrombophilia in Pakistan.⁴

Finally, we find that Factor V Leiden mutation is the most common inherited thrombophilic disease associated with patients presenting with thrombotic occurrences, and deep venous thrombosis was the most common clinical presentation.^{4,5,6} These findings substantiate the role of targeted thrombophilia screening in the clinically unexplainable or recurrent thrombotic occurrences of young patients to inform specific management approaches.^{15,16}

CONCLUSION

This paper has served to bring out the important role of inherited thrombophilic disorders in thrombotic events in our population. A quarter of the patients who presented with reported venous or arterial thrombosis were identified to be having an identifiable inherited thrombophilic defect. Factor V Leiden mutation was found to be the most common deficiency and was then antithrombin, protein C, and protein S deficiencies and the prothrombin gene mutation was rare. Deep venous thrombosis was the most common clinical presentation, which supports the close correlation between inherited thrombophilia and venous thromboembolism. The issue of inherited causes is highlighted by the comparatively youth of the patients with thrombosis at an early stage or without a clear cause. The early identification of these disorders holds some significance on the personalized approach to anticoagulation control, recurrence prevention, family counseling, and risk evaluation. It is suggested that wider multicenter research is needed to better characterize the national prevalence trends and streamline the thrombophilia screening guidelines.

Authors' Contribution

AK, RUJ: Concept & study design, data collection and drafting of the manuscript.

MDK: Data entry, statistical analysis, interpretation of the results.

AQ: Manuscript editing and critical revision of the manuscript.

All authors approved the final version of the manuscript to be published.

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Conflict of Interest

The authors declared no conflict of interest.

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Data Availability

Data is available with authors and can be provided on request.

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