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ORIGINAL ARTICLE

Etiological spectrum of arterial and venous thrombosis in a tertiary-care hospital

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ABSTRACT

Background: There are heterogeneous pathogenesis of arterial and venous thrombosis, and little information exists locally on the etiological spectrum of these diseases. The objective of this study was to identify the etiological spectrum of arterial and venous thrombosis in patients who came to the tertiary care hematological service.

Methods: This cross-sectional research was done in the Department of Haematology, Shaikh Zayed Hospital, Lahore. A total of 70 patients with objectively recorded arterial or venous thrombosis aged 16-75 were used; it has excluded myocardial infarction and iatrogenic embolization/thrombosis. Clinical, available thrombophilia, demographic and imaging analyzes were checked. Etiological results were classified as APS / APS-compatible antiphospholipid-antibody results, myeloproliferative-neoplasm-associated mutation, Factor V Leiden, protein C deficiency, protein S deficiency or no known etiology. All participants were individually put under one major etiological category. The laboratory abnormalities were taken with caution because documentation of timing and repeat confirmation of certain thrombophilia tests was not always undertaken. Unadjusted comparisons were done using descriptive statistics and unpaired Fisher exact test.

Results: Of 70 patients, 45 (64.3%) had venous and 25 (35.7%) had arterial thrombosis. In 51 (72.9%), a specific category of etiology was determined. The 12 (17.1%), 10 (14.3%), 9 (12.9%), 8 (11.4%), 7 (10.0%), and 5 (7.1%) respectively consisted of APS/APS-compatible findings, myeloproliferative-neoplasm-related mutations, protein S deficiency, Factor V Leiden, protein C deficiency, and antithrombin deficiency respectively; 19 (27.1%) was of undetermined etiology.

Conclusion: This tertiary-care cohort had more venous than arterial thrombosis. The most common acquired etiological categories were APS/APS-compatible laboratory results and myeloproliferative-neoplasm-related mutations. Since not all thrombophilia abnormalities had repeat confirmation means that findings cannot be taken to be population prevalence or even definitive causal diagnoses but must be taken as implied etiological findings of the hospital setting. Selective and clinically whole-hearted testing of thrombophilia is endorsed.

Keywords: Antiphospholipid Syndrome, Arterial Thrombosis, Myeloprolliferative Disorders, Thrombophilia, Venous Thrombosis

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Introduction

Thrombosis refers to the pathologic development of blood clot in the vascular system leading to the partial or total blockage of blood circulation. It can be arterial or

venous and result in ischemia, infarction, congestion of the veins, pulmonary embolization, disease endangering the limbs, stroke, or death. Thrombosis in arteries and veins has some common clinical and biological risk factors even though the mechanisms that are predominant in each are different as age, inflammation, malignancy, metabolic disease, antiphospholipid antibodies, and inherited thrombophilia (1).

The inception of venous thromboembolism, which refers to deep-vein thrombosis and pulmonary embolism, is a widespread yet a recurrent problem. Notable inciting causes consist of advanced age, hospitalization, surgery, immobility, malignancy, pregnancy, hormonal therapy, and chronic inflammatory disease; inherited thrombophilia might be contributive. Venous thrombosis is typically characterized by pain, swelling, warmness, or erythema of the limb of the organism and unusual-site thrombosis may have abdominal or neurological outcomes (2).

Arterial thrombosis is usually mainly platelet-laden and is better related to atherosclerosis, endothelial discharge, high blood pressure, diabetes, dyslipidemia together with smoking and vascular inflammation. The clinical manifestations comprise ischemic stroke, acute limb ischemia, retinal artery occlusion, mesenteric ischemia as well as other organ ischemia. Though it is a significant arterial thrombotic event, myocardial infarction was out of the present study based on the study criteria (3).

The etiological assessment is mostly applicable to younger patients and recurrent, unusual site, and mixed arterial and venous thrombosis that lacks any provocation, particularly when there is a family history. Routine provocative factors ought to be differentiated with the thrombophilia research as the clues to etiological research differ in arterial and venous ailment. Detection of a clinically significant cause can affect the antithrombotic treatment, counselling and risk-control measures (4).

Antiphospholipid syndrome (APS) is an autoimmune thrombophilia of acquired origin and is linked to the presence of next to death anti-phospholipid antibodies plus the presence of qualifying clinical events like arterial or venous thrombosis or pregnancy morbidity. The laboratory testing consists of lupus anticoagulant, anticardiolipin antibodies, and anti- -2-glycoprotein I antibodies; continual retesting is needed to make a definitive classification of APS (5,6,7).

Myeloprolliferative neoplasms are clonal haematopoietic diseases that are linked with thrombosis, caused by abnormal cell counts, platelet and leukocyte activation, endothelial interaction, inflammation, and driver mutations. Mutations in JAK2 V617F are the most common ones; CALR and MPL can also be implicated in proven myeloproliferative neoplasms. Thrombosis can lead to a haematological diagnosis and be located in unusual locations, e.g., in the splanchnic veins (8).

The other factor of chosen etiological evaluation is inherited thrombophilia. Factor V leiden is responsible of activated protein C resistance, and more closely related to venous than arterial thrombosis, but with strikingly different ethnic occurrences (9).

In severe, recurring or early onset venous thrombosis, deficiency of the natural anticoagulants antithrombin, protein C and protein S is less frequent but might have clinical significance. These anomalies disfavor natural anticoagulant mechanisms and augment thrombin creation (10).

The thrombophilia test should be interpreted with care since protein C, protein S and antithrombin levels may be altered due to acute thrombosis, anticoagulant therapy, liver disease, pregnancy and inflammation.

Likewise, the sole positive antiphospholipid-antibody test is not sufficient to establish APS. Confirmation should thus be done in time and in instances where applicable repeated to protect misclassification (6,7).

Local information about the etiological range of arterial and venous thrombosis is minimal and trends of inherited thrombophilia are also not necessarily the same as those in the western population publications. This was therefore a descriptive study that attempted to describe the etiological spectrum of objectively reported arterial and venous thrombosis among patients reporting to a tertiary-care hematology service in Lahore.

Methods

The cross-sectional study took place at the Department of Haematology, Shaikh Zayed Hospital, Lahore between 15 May 2026 and 14 August 2026. All participants enrolled provided written informed consent and confidentiality was assured. Ethical approval reference 02-TERC/NHRC-SZH/INT-SC/941 dated 20-12-25.

The calculator of WHO single-proportion was used to calculate the sample size. The confidence level was 90, precision was 6 percent, and it is expected that the proportion of APS will be 10 percent, which led to the required sample of 70, considering previous evidence. Non-probability consecutive sampling method was utilized which involved enrollment of eligible patients until the target size was attained.

The patients were aged 16 to 75 years and of both sexes in which the arterial and/or venous thrombosis was objectively documented. Thrombotic had to be accepted with imaging or specialist documentation that is or should be at an anatomical location (venous duplex/ compression ultrasonography of lower-limb DVT, cerebral venographic imaging of cerebral venous sinus thrombosis, pulmonary vascular imaging of pulmonary embolism, abdominal vascular imaging of splanchnic venous thrombosis, brain imaging of ischemic stroke, arterial vascular imaging of peripheral arterial thrombosis, and an ophthalmology record of retinal artery occlusion). The research was based on available clinical documentation and not a re-research in imaging. Myocardial infarction and iatrogenic embolization or thrombosis were ruled out.

At the level of the structured proforma, the demographic and clinical data were inputted. Some of the variables were age, gender, body mass index, duration of the symptoms, type and location of thrombosis, clinical manifestation, diabetes mellitus, hypertension, smoking history (>5 pack-years), anemia, family history of thrombosis, and present thrombophilia tests. Manifestations that were recorded included abdominal pain/tenderness, abdominal distension, nausea/vomiting, limb pain/tenderness, limb swelling, limb erythema /warmth, neurological symptoms and dyspnea.

Etiological evaluation was done through documented lab and molecular reports. APS was defined as definite only when a qualifying thrombotic event was in the setting of documented persistent lupus anticoagulant, anticardiolipin and /or anti-' and with persistence testing not documented, the outcome was outlined as an APS-compatible antiphospholipid-antibody profile not definite APS (11,12,13). A case of myeloproliferative-neoplasm-related etiology was captured in cases where a corresponding driver/clonal mutation was reported, such as JAK2, CALR, and MPL on molecular testing (6,14,15). Factor V Leiden was founded on reported molecular testing (7,19). Abnormalities of antithrombin, protein C and protein S were determined using available activity / antigen assays and were viewed

with caution since precise timing with reference to the acute process and exposure to anticoagulants did not always exist in source records (8,9,17,18,20,22). To be analysed, the 7 etiological groups in Table 4 were viewed as mutually exclusive groups and each respondent belonged to one main category.

Data analysis was done using SPSS version 26. The quantitative variables were summarized as the mean, standard deviation and the frequencies and percentages used to summarize the categorical variables. The reported unadjusted 2x2 comparisons were performed using two-sided Fisher exact tests since some of the expected cell counts were small. Analysis was descriptive and unadjusted with no independent-predictor or causal inference. P-value less than0.05 was considered to be statistically significant because the two sides were below.

Results

A total of 70 patients who had arterial and/or venous thrombosis were used. Mean age was 39.8 ± 14.2 years (range 16–75 years); 38 patients (54.3%) were aged 16–40 years and 32 (45.7%) were aged >40 years. There were 41 men (58.6%) and 29 women (41.4%). Mean BMI was 27.1 ± 4.6 kg/m²; 26 patients (37.1%) had BMI <25 kg/m² and 44 (62.9%) had BMI ≥25 kg/m². Poan duration of symptoms was 12.4 ± 9.6 days.

There was more venous thrombosis that arterial thrombosis: 45 (64.3) of the patients had venous and 25 (35.7) had arterial. The most common site of venous thrombosis was lower-limb DVT (28/70, 40.0%), then cerebral venous sinu thrombosis (7/70, 10.0%), pulmonary embolism (5/70, 7.1%), and splanchnic venous thrombosis (5/70, 7.1%). Arterial events- the ischemic stroke (14/70, 20.0%), peripheral arterial thrombosis (7/70, 10.0%), retinal artery thrombosis (4/70, 5.7%). The most common clinical presentation was limb pain/tenderness in 38 (54.3%), limb swelling of 35 (50.0%), limb warmth/erythema of 30 (42.9%), neurological symptoms of 21 (30.0%), abdominal pain/tenderness of 9 (12.9%), nausea/vomiting of 8 (11.4%), dyspnea of 8 (11.4%), abdominal distension of 6 (8.6%). Hypertension was present in 16 patients (22.9%), diabetes mellitus in 12 (17.1%), smoking >5 pack-years in 18 (25.7%), anemia in 15 (21.4%), and a family history of thrombosis in 13 (18.6%).

In 51 patients it was found to belong to a particular etiological category (72.9%), and 19 (27.1 percent) resulted in no etiology. The commonest were APS/APS-compatible (12/70, 17.1%), myeloproliferative-neoplasm-related mutation (10/70, 14.3%), protein S deficiency (9/70, 12.9%), Factor V Leiden (8/70, 11.4%), protein C deficiency (7/70, 10.0%) and antithrombin deficiency (5/70, 7.1%). The primary etiological classification was on these mutually exclusive categories.

Descriptively, the distribution of certain etiological categories spanned by thrombosis type varied. In 6 arterial and 6 venous cases, 6 apoptotic protein syndromes/adenine-purine syndrome-positive syndrome correlations were documented as APS/APS-compatible, myeloprolliferative-neoplasm-correlated mutations were identified in 7 arterial and 3 venous cases and Factor V Leiden in 1 arterial case and 7 venous cases. Only the venous cases had antithrombin deficiency, and there was a higher tendency of protein C and protein S deficiency in the venous thrombosis. The same differences are described (as opposed to being compared statistically as a separate exact multi-category test) since a number of category-specific cells were sparse, and no separate exact multi-category test was prespecified.

In unadjusted Fisher exact comparisons of identified versus unidentified etiology, younger age (16–40 years: 31/38 [81.6%] vs

>40 years: 20/32 [62.5%]; p=0.106), positive family history (12/13 [92.3%] vs 39/57 [68.4%]; p=0.097), and absence of hypertension (42/54 [77.8%] vs 9/16 [56.3%]; p=0.114) showed the largest numerical differences, but none met the conventional p<0.05 threshold. Other comparisons which were shown were not statistically significant as well.

Table 1. Baseline demographic and clinical characteristics, n=70

Variable	Frequency/ Mean	Percentage/ SD
Age, years	39.8	±14.2
Age 16–40 years	38	54.3%
Age >40 years	32	45.7%
Male	41	58.6%
Female	29	41.4%
BMI, kg/m²	27.1	±4.6
BMI <25 kg/m²	26	37.1%
BMI ≥25 kg/m²	44	62.9%
Duration of symptoms, days	12.4	±9.6
Duration ≤7 days	29	41.4%
Duration >7 days	41	58.6%
Family history of thrombosis	13	18.6%

Table 2. Type and anatomical site of thrombosis

Thrombosis variable	Frequency	Percentage
Venous thrombosis	45	64.3%
Arterial thrombosis	25	35.7%
Lower-limb deep vein thrombosis	28	40.0%
Cerebral venous sinus thrombosis	7	10.0%
Pulmonary embolism	5	7.1%
Splanchnic venous thrombosis	5	7.1%
Ischemic stroke	14	20.0%
Peripheral arterial thrombosis	7	10.0%
Retinal artery thrombosis	4	5.7%

Table 3. Clinical presentation and medical history

Variable	Frequency	Percentage
Limb pain / tenderness	38	54.3%
Limb swelling	35	50.0%
Warmth / erythema in limb	30	42.9%
Neurological symptoms	21	30.0%
Abdominal pain / tenderness	9	12.9%
Abdominal distension	6	8.6%
Nausea / vomiting	8	11.4%
Dyspnea	8	11.4%
Hypertension	16	22.9%
Diabetes mellitus	12	17.1%
Smoking >5 pack-years	18	25.7%
Anemia	15	21.4%

Table 4. Etiological spectrum of thrombosis

Etiology	Frequency	Percentage
APS / APS-compatible antiphospholipid-antibody findings	12	17.1%
Myeloproliferative neoplasm-related mutation	10	14.3%
Factor V Leiden	8	11.4%
Antithrombin deficiency	5	7.1%
Protein C deficiency	7	10.0%
Protein S deficiency	9	12.9%
No identified etiology	19	27.1%
Any identified etiology	51	72.9%

Table 5. Unadjusted Fisher exact comparisons for identification of a specific etiology

Variable	Identified etiology n/N (%)	No identified etiology n/N (%)	p-value
Arterial thrombosis	17/25 (68.0%)	8/25 (32.0%)	0.579
Venous thrombosis	34/45 (75.6%)	11/45 (24.4%)	
Age 16–40 years	31/38 (81.6%)	7/38 (18.4%)	0.106
Age >40 years	20/32 (62.5%)	12/32 (37.5%)	
Male	30/41 (73.2%)	11/41 (26.8%)	1.000
Female	21/29 (72.4%)	8/29 (27.6%)	
BMI <25 kg/m²	20/26 (76.9%)	6/26 (23.1%)	0.593
BMI ≥25 kg/m²	31/44 (70.5%)	13/44 (29.5%)	

Variable	Identified etiology n/N (%)	No identified etiology n/N (%)	p-value
Family history present	12/13 (92.3%)	1/13 (7.7%)	0.097
Family history absent	39/57 (68.4%)	18/57 (31.6%)	
Duration ≤7 days	20/29 (69.0%)	9/29 (31.0%)	0.592
Duration >7 days	31/41 (75.6%)	10/41 (24.4%)	
Hypertension present	9/16 (56.3%)	7/16 (43.8%)	0.114
Hypertension absent	42/54 (77.8%)	12/54 (22.2%)	
Smoking present	12/18 (66.7%)	6/18 (33.3%)	0.545
Smoking absent	39/52 (75.0%)	13/52 (25.0%)	

Discussion

This was a cross-sectional study, which outlined the etiological continuum of arterial and venous thrombosis at a tertiary-care haematology unit. Sixty four three per cent of the cohort were attributable to venous events and 72.9 per cent to a given category of etiology. The most common type was the findings in APS/APS compatible followed by myeloproliferative-neoplasm-related mutations and inherited thrombophilia related findings. These frequencies in hospitals should not be construed as prevalence in the population or as a demonstration of causation.

Dominating venous thrombosis is compatible with the clinical setting where thrombophilia is most commonly thought. Khan et al. explain venous thromboembolism as a multifactorial and repeatedly occurring disorder where acquired and inherited factors interact (11). Venous events were 64.3% of the cases in the current cohort, which justifies the selectiveness of etiological assessment especially in younger patients and in thrombosis of unprovable or unusual site.

Findings in 17.1% of patients were noted to be APS/APS compatible. Hubben and McCrae underline that APS may manifest itself with arterial or venous thrombosis and that laboratory testing involves lupus anticoagulant, anticardiolipin antibodies, and anti-2-glycopyrotein I antibodies (10). Due to the fact that repeat persistence testing was never always recorded in the current records, these results cannot be all perceived as newly confirmed APS diagnoses.

There were 50: 50 (APS/APS-compatible) findings in arteries and venous cases. Manning et al. outline the diagnostic and treatment issues of APS in venous, arterial, obstetric, and microvascular presentations (13). The current evidence thus reflects the idea of viewing antiphospholipid-antibody testing in duly chosen, both arterial and venous thrombosis, with established requirements of confirmation.

Mutations that were associated with myeloproliferative-neoplasm were identified in 14.3% of the cases and were numerically higher among arterial cases. According to Chia et al., molecular genetics of thrombotic myeloprolliferative neoplasma and the role of JAK2, CALR, and MPL mutations are important (6). The results provide a rationale to use specific molecular diagnosis in the patients with unexplainable or unusual-site thrombosis and may indicate a reasoned blood-count distortion instead of randomly screening the patients.

The correlation between clonal haematopoiesis, myeloid neoplasms, and thrombo-inflammatory vascular events are discussed by Awada et al. (15). Mutation testing can be used in clinical practice to determine a underlying myeloid disorder in individual patients with thrombosis in presence of unexplained erythrocytosis, thrombocytosis, leukocytosis, or splenic thrombosis.

The most common natural-anticoagulant abnormality in this cohort was protein S deficiency (12.9) and more prevalent in venous episodes. According to Padda et al., protein C and protein S are natural anticoagulants that lack of them can contribute to the higher generation of thrombin and thromboembolic risk (16). Since protein S can be affected by acute disease, pregnancy, hepatic disease, and anticoagulant therapy, a low result must be contextualized in both clinical and laboratory.

Protein C deficiency was documented in 10.0 percent of patients, and was more common with venous than arterial thrombosis. Casini et al. point out that inherited natural-anticoagulant deficiencies are best applicable to those who are selected, such as patients with severe, recurring, or early onset of venous thrombosis (17). The current evidence upholds the use of selective tests as opposed to routine testing, and repeat testing when clinically necessary.

The incidence of antithrombin deficiency was 7.1 percent, and only occurred in venous. Alnor et al. indicate that hereditary antithrombin, protein C, and protein S deficiencies have a higher risk of venous thromboembolism (8). Testing in acute thrombosis or in the presence of anticoagulant can be difficult to interpret just like the other natural anticoagulants.

F V Leiden was detected in 11.4 per cent of patients and mostly in venous thrombosis. Moore also discusses activated protein C resistance and the Factor V Leiden role and mentions that the population and ethnic variation play a significant role (7). The frequency that is observed in the hospital setting of this study cannot be, therefore, extended to the overall Pakistani population.

By and large, the inherited thrombophilia results were localized to only few venous events. According to Stevens et al. hereditary thrombophilia testing is highest in the small groups of patients who have venous thrombophilia whereas the testing is less informative in the everyday assessment of arterial thrombosis (20).

In 27.1% of the patients, no specific etiology was detected. This is medically plausible since thrombosis can be multifactorial and can be associated with temporary inciting factors, obesity, hospitalization, inflammation, malignancy or immeasurable genetic mutations. Arachchillage and colleagues suggest that the application of thrombophilia testing should be placed within clinical context and that testing even with negative results should not negate the possibility of thrombotic in future (9).

The positive family history was found to have a strong numerical correlation with a known etiology (92.3% vs 68.4%), although the two-sided Fisher exact test was not statistically significant (p=0.097). This trend favors cautious family-history evaluation but cannot be construed as a predictive in itself. Tang et al. also encourage selective hereditary thrombophilia testing in case there is evidence of a personal and familial history of hereditary predisposition (22).

A specified etiology was also numerically more common in patients aged 16 40 years old than in patients aged >40 years old (81.6% vs 62.5; Fisher exact p=0.106). This difference is clinically plausible since thrombosis at younger age more frequently leads one to consider APS or inherited thrombophilia, or occult myeloprolliferative disease, but the crude comparison fails to demonstrate any independent age effect.

Other factors, including hypertension, smoking, sex, BMI, symptom duration, and type of thrombosis did not significantly affect identification of a particular etiology in the unadjusted analyses presented. Such null results should be treated with reservations

since the sample size is quite small and a number of subgroups are skewed.

The most frequent arterial presentation was ischemic stroke. Bernardi et al. summarize arterial APS symptoms, such as stroke and cardiac disease (23). APS and myeloproliferative-neoplasm-related abnormalities might, therefore, be considered alongside traditional vascular risk factors in some younger patients with ischemic stroke or otherwise unexplained arterial thrombosis.

The most common venous presentation was lower-limb DVT. Cerebral venous sinus- and splenic venous thrombosis also were present. These rare-location occurrences can be linked to APS, myeloproliferative neoplasma, inherited thrombophilia, pregnancy, infection, inflammation or local and thus tend to warrant focused etiological examination.

The analysis utilized reported imaging and thrombophilia, which is part of the current practice under the conditions of a scarce resource. Nevertheless, the time of the protein C, protein S, and antithrombin testing, in relation to the acute event and anticoagulant treatment was not always reported and the repeat confirmatory testing was not equally provided. These issues may have resulted in laboratory misclassification and this should be taken into account when interpreting the reported etiological categories.

The clinical implication is to adopt a discriminated testing strategy: antiphospholipid-antibody testing in appropriately selected venous or arterial thrombosis; myeloprolliferative-neoplasm mutation testing in unexplained, unusual-site thrombosis, or putative abnormalities of blood counts; and inherited thrombophilia testing in properly selected younger patients with venous, recurrent, unexplained, or familial thrombosis. This can enhance clinical superbility, without exposing people to haphazard testing.

The strong points of the study are that it encompassed both arterial and venous thrombosis, objective clinical records were utilized, both acquired and inherited etiological types were assessed, and local tertiary-care data was generated. Clinical presentation, comorbidities, family history and anatomic sites of thrombosis were also noted in the study.

Limitations: This was small and single-centre cross-sectional study by non-probability consecutive sample. Other thrombophilia tests can even be done in the course of acute thrombosis or when the patient was taking anticoagulants and the time was not always recorded. Confirmation of antiphospholipid antibodies and natural-anticoagulant abnormalities was not consistently available thus posing a risk of misclassification. Some of the subgroup cells were sparse and the analyses were not adjusted. The findings thus reflect hospital etiological descriptions and cannot be used as an indication of population prevalence, causal influences or independent predictors.

The study has local descriptive data on the etiological discovery experienced in a tertiary-care haematology service despite these constraints to offer a selective, clinically assisted method to the evaluation of thrombophilia.

Conclusion

Venous thrombosis was seen more commonly than arterial thrombosis amongst patients attending this tertiary haematology service, and a particular etiological group was identified in about a third of cases. The most common acquired categories were the APS/APS compatible antiphospholipid-antibody finding, as well as mutations causing myeloprolliferative-neoplasm, whereas the most noticeable inherited categories were the protein S deficiency and Factor V Leiden. Due to the lack of consistently reported

confirmatory testing of all abnormalities of thrombophilia and unadjusted analyses, the results ought to be viewed with caution as descriptive hospital-based outcomes.

Recommendations

A structured clinical evaluation of provoking factors and the selected acquired or inherited causes should be performed on patients with confirmed arterial or venous thrombosis, especially when it is young-onset, recurrent, unprovoked, or has a strange location or when there is a strong family history. History of thrombosis in the past, his family history, smoking history, hypertension, diabetes, pregnancy, drug exposure, immobilization, surgery, malignancy and other provocation details should be included.

Antiphospholipid-antibody testing is recommended in selective use in arterial and venous thrombosis, especially in younger people, inexplicable ischemic stroke, recurrence thrombosis, atypical-site thrombosis, and pregnancy morbidity. Diagnosis of APS must be based upon accepted clinical criteria in combination with ongoing laboratory positivity at repeated testing at the advised interval.

Myeloprolliferative-neoplasm-related mutations, especially JAK2 V617F and, in some cases, CALR or MPL should be tested in unexplained or splanchnic thrombosis, recurrent or unusual-location events and thrombosis in association with abnormal blood counts. The positive molecular results are to be properly evaluated with haematologically.

Testing of factor V Leiden, antithrombin, protein C and protein S should be selective and not routine. The most applicable testing is on younger patients who have venous, recurrent, unprovoked or familial thrombosis. Protein C, protein S and antithrombin levels are to be interpreted regarding such conditions as acute thrombosis, using anticoagulant therapy, liver disease, pregnancy, and inflammation, and confirmation should be repeated in case it was necessary in clinical conditions.

Individual counselling to patients with known thrombophilia should be based on the risk of recurrence, anticoagulation, pregnancy, a family assessment where necessary, and prevention or avoidance of high-risk situations are prolonged immobilization and surgery. A negative thrombophilia work up cannot be construed as lack of future risk of thrombosis.

To further characterize the local range of thrombosis and assess recurring thrombosis, bleeding, and treatment outcomes, larger multicenter trials where timing of thrombophilia testing is standardized, any relevant abnormalities are re-tested, and there is prospective follow-up are suggested.

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