STUDY OF FIBRINOLYTIC FACTORS ALONG WITH THROMBOPHILIA SCREENING GIVES A COMPREHENSIVE PICTURE OF THE CAUSE OF VENOUS THROMBOSIS IN INDIAN PATIENTS

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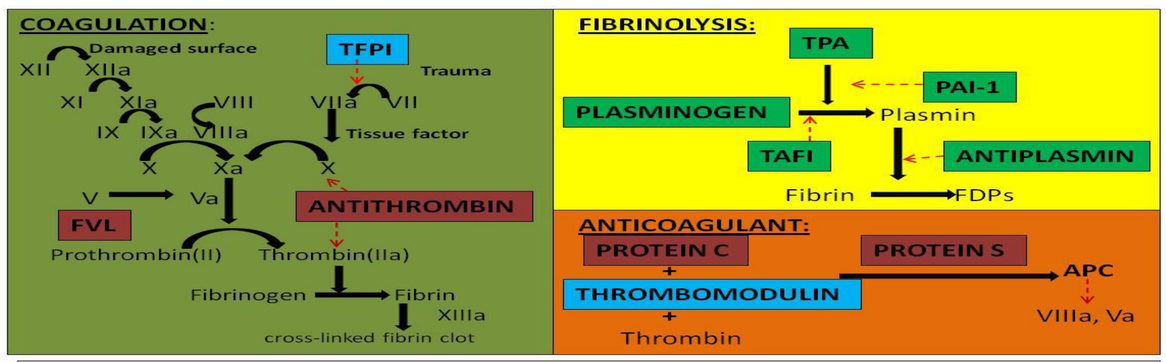
INTRODUCTION

Fibrinolysis serves an important role in the process of coagulation by ensuring clots formed in response to injury resolve after the injured tissue is repaired. About 2/3rd of venous thrombosis patients are negative for the conventional thrombophilia markers even though some of them have strong recurrence history or thrombosis in unusual sites. Defects in the fibrinolytic system, either in factors that accelerate fibrinolysis or those which regulate acceleration, can directly affect the thrombus.

BIOCHEMISTRY OF VENOUS THROMBOSIS

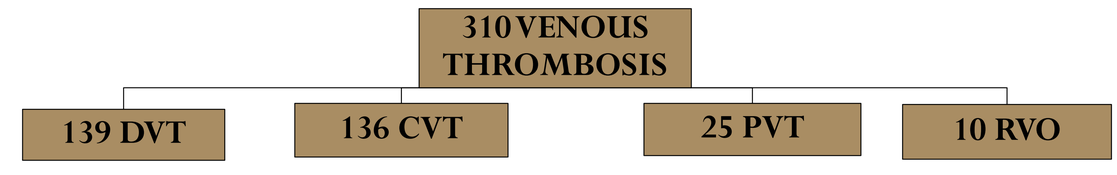


Key: **FVL**: Factor V Leiden; **TFPI**:Tissue factor pathway inhibitor; **TPA**: Tissue Plasminogen activator; **PAI-1**:Plasminogen activator inhibitor-1; **TAFI**:Thrombin activatable fibrinolysis inhibitor

AIM AND OBJECTIVE

To study the fibrinolytic pathway defects along with conventional thrombophilia in venous thrombosis patients and establish a co-relation between impaired fibrinolytic system and venous thrombosis in Indian patients.

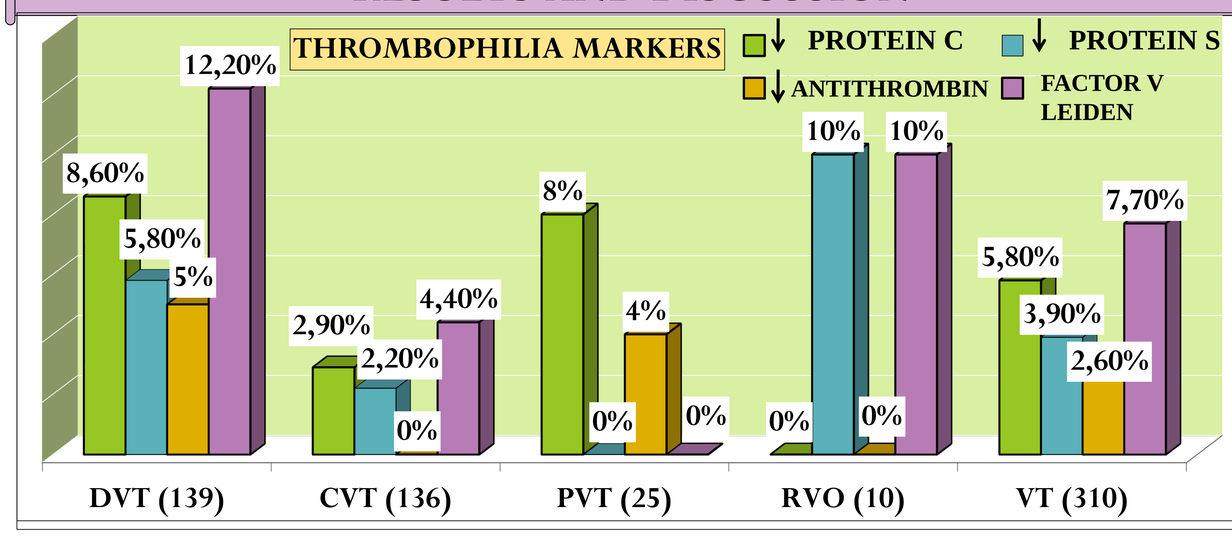
MATERIALS AND METHODS



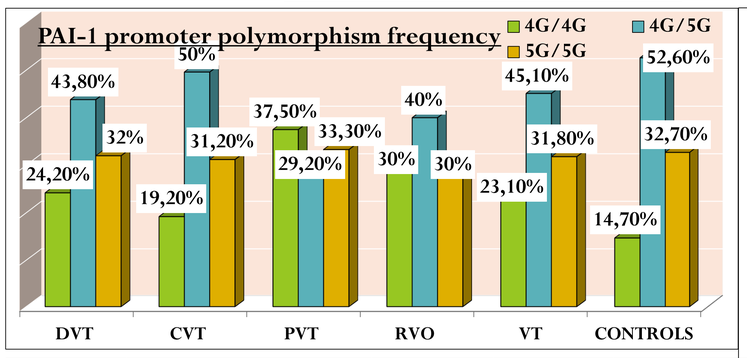
ELISA: Protein C, Protein S, Antithrombin III, Plasminogen, Plasminogen activator inhibitor-1 (PAI-1), Tissue Plasminogen activator (TPA), Antiplasmin Thrombomodulin, Tissue Factor Pathway Inhibitor (TFPI).

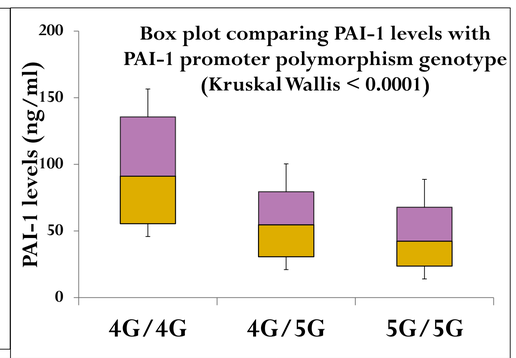
PCR- RFLP: Factor V Leiden, **Allele specific PCR**: PAI-14G/5G polymorphism.

RESULTS AND DISCUSSION



FIBRINOLYTIC MARKERS

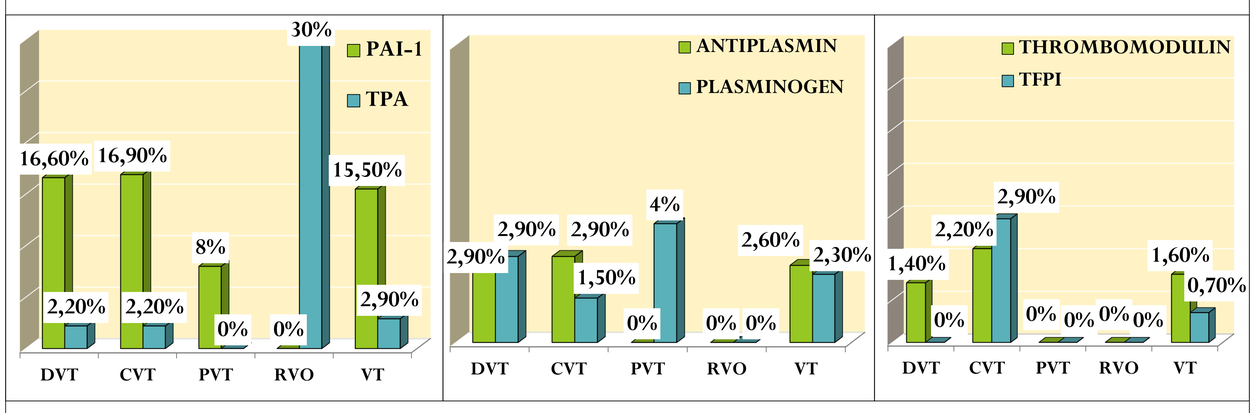




	DVT (n=139)	CVT (n=136)	PVT (n=25)	RVO (n=10)	TOTAL (n=310)
P-value	0.024	0.271	0.007	0.202	0.015
OR	1.85	1.37	3.47	2.48	1.74
95% CI	1.08-3.15	0.78-2.42	1.42-8.51	0.61-10.02	1.11-2.71

PAI-I PROMOTER POLYMORPHISM	PAI- 1 LEVELS (ng/ml)
4G/4G	107.37 ± 75.12
4G/5G	62.14 ± 40.72
5G/5G	51.4 ± 38.36

RESULTS AND DISCUSSION



- 7.7% • Factor V Leiden mutation
- 5.8% • Protein C deficiency
- 3.9% • Protein S deficiency
- 2.6% • Antithrombin III deficiency
- 15.5% • High Plasminogen Activator Inhibitor - 1 (PAI-1)
- 2.9% • Low Tissue Plasminogen Activator (TPA)
- 2.6% • High Antiplasmin
- 2.3% • Low Plasminogen
- 1.6% • Low Thrombomodulin
- 0.7% • Low Tissue Factor Pathway Inhibitor (TFPI)

❖ **35.5%** (110 cases) of venous thrombosis could be accounted.

❖ 3 cases (1%) had at least two abnormal fibrinolytic proteins.

❖ 15 cases (4.8%) had a combined defect i.e. positive for a conventional thrombophilia marker and a defective fibrinolytic parameter.

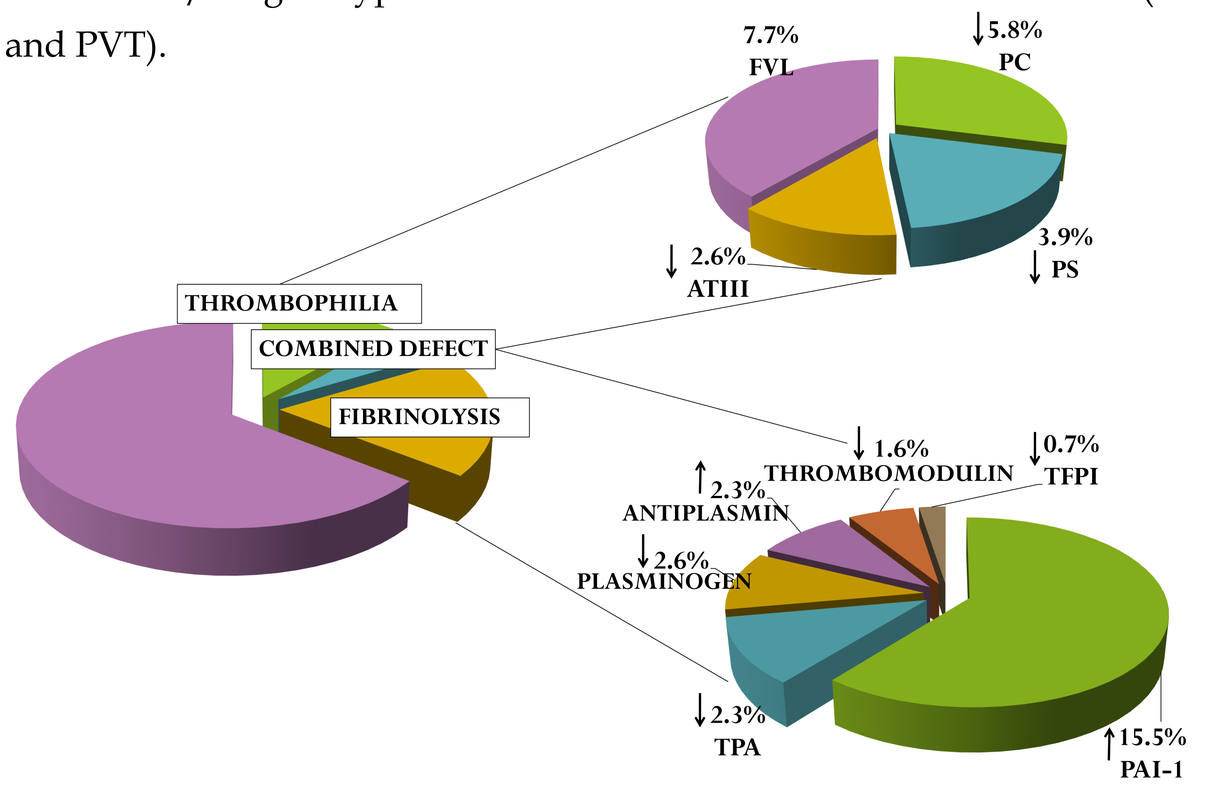
❖ Conventional thrombophilia explained 51 (16.5%) cases alone.

❖ High PAI-1 major contributor of fibrinolytic defect: 48 cases (15.5%).

❖ 14 (4.5%) of these 48 were collected in acute phase of thrombosis however 6 (1.9%) of them had a 4G/4G PAI-1 genotype.

❖ PAI-1 4G/4G genotype was associated with high levels of PAI-1 leading to a hypofibrinolytic state increasing thrombotic risk.

❖PAI-1 4G/4G genotype was associated with venous thrombosis risk (DVT and PVT).



CONCLUSION

Data shows that investigating for a combination of markers both in the anti-coagulant system and in the fibrinolytic pathway will facilitate in a more comprehensive explanation of the cause of venous thrombosis.

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