



HEMOSTASIS IN HYPERTENSION - A PIVOT ROLE BLOOD PLATELETS, PT & APT

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ABSTRACT

INTRODUCTION : Hypertension the most recognized risk factor for coronary artery disease, cerebrovascular diseases & renal diseases, is the root cause of 7.6million premature deaths in people aged above 25years. Atherosclerosis MAY be cause of abnormal cascade in blood coagulation following MI or CVD. Present study determines the role of blood parameters like platelets count, prothrombin time & activated partial thromboplastin time in hypertensive patients as an AID in prevention of coagulopathy & thereby CAD & stroke.

MATERIALS & METHODS:**Inclusion criteria :**

1. Test group-40 males hypertensive patients 38-60yrs males.
2. Control group-40 age & sex-matched normotensive subjects.

Exclusion criteria: TY2DM, TB,CLOTING DISEASES , ANTIPLATELETS MEDICATION, chronic illness & any infections. -BP recording: by manual sphygmomanometer Written consent was taken .-Lab. Analysis Hemostatic XF 2.0 & Horiba pentra XL 80. Statistical analysis: Student t test (unpaired)

Results: Prothrombin time & partial activated thromboplastin provides an effective insight about the Physiology of hemostatic and its pathology (Hypercoagulability). Blood platelets are most compelling parameters in monitoring thrombosis. In present study Platelets count, prothrombin time & activated partial thromboplastin are ELEVATED in hypertensive subject compared with that of normotensive subjects. Conclusion: Hypertension is a risk factor for thrombotic diseases like stroke & CAD due to associated hemostatic dysfunction promoting coagulopathy

KEYWORDS : Hypertension, Hemostatic system, Thrombosis & Coagulation**INTRODUCTION**

Hypertension the most recognized risk factor for coronary artery disease, cerebrovascular diseases & renal diseases, is the root cause of 7.6million premature deaths in people aged above 25years. Contributing factors to the prevalence of hypertension are diets, lifestyles, stress, bodyweight, family history & genetic factors. WHO suggested growth on processed food industry has affected global salt concentration in diets that plays a crucial role in hypertension³

Altered hemostasis aids endothelial cell dysfunction. Atherosclerosis MAY be cause of abnormal cascade in blood coagulation following MI or CVD. Furthermore hemostatic dysfunction are frequently encountered complications with hypertension¹.

Hypertension is the leading cause of cardiovascular diseases in the world and to this atherosclerosis could be the CAUSE for abnormal lopsided coagulation of blood.¹

Present study determines the role of blood parameters like platelets count, prothrombin time & activated partial thromboplastin time in hypertensive patients as an AID in prevention of coagulopathy & thereby CAD & stroke.

Prevalence of hypertension

Hypertension directly responsible 57% all strokes death & 24% CAD in India 13. In an analysis's of worldwide data for Global burden of hypertension, 20.6% Indian men, 20.9% Indian women suffering from hypertension 2005.

Rate of hypertension in percentage are projected to go up to 22.9 & 23.6 for Indian men & women respectively by 2025¹⁴

BURDEN OF Hypertension-India

29.2% overall prevalence, among them 27.6% were urban India & 33.8% were rural India. These data were reported & published by community based studies done between 2011-2013¹⁵ In present scenario, existing interventions should look at incorporating multi-component & multilevel approach's for better managing blood

pressure among Indians.

MATERIALS & METHODS**Inclusion criteria:**

1. Test group-40 males hypertensive patients 38-60yrs.
2. Control group- 40 age & sex-matched normotensive subjects.

Exclusion criteria: Type2 Diabetes Mellitus, TB, CLOTING DISEASES, anti platelets medication, chronic illness & any infections.

BP recording: manual Sphygmomanometer : supine position, after 5min of rest. Average of 3 readings, each at an interval of 2min considered.

-Written consent was taken: sample collection , Lab. Analysis: Hemostatic XF 2.0 & Horiba pentra XL 80

STATISTICAL ANALYSIS: Student t test-unpaired

Results & Discussion**Table 1 .**Effect of Hypertension: Hemostatic parameters

Parameters	Mean \pm Standard error		p-value	t-value
	Study group	Control group		
Prothrombin time (PT) secs	15.44 \pm 0.31	11.85 \pm 0.17	0	11.144
Activated partial thromboplastin time	36.45 \pm 0.48	27.55 \pm 0.61	0	8.036
Platelets count (X 10 ⁹ / L)	190.06 \pm 9.22	270.66 \pm 10.40	0	-4.925

Significant variation (p <0.001) between study & control group of each parameters.

Dysfunction of hemostatic system predisposes the patients to atherosclerosis which is the RISK factor for hypertension along with

endothelial destruction & dysfunction leading to hyper activation of platelets. Present study comparable to finding of Adaaze et al2 reported PT- 14.45sec, aPT- 35.42 sec in hypertensive patients.

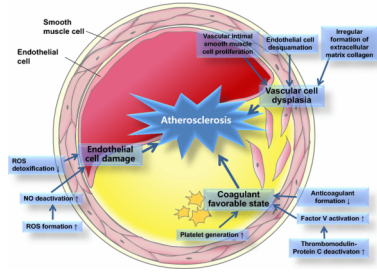


Fig.1 Pathology of atherosclerosis and activation of coagulation cascade.



Fig.2 Activation of Reactive oxygen species & Uric acid free radicals in hypertension leading to atherosclerosis

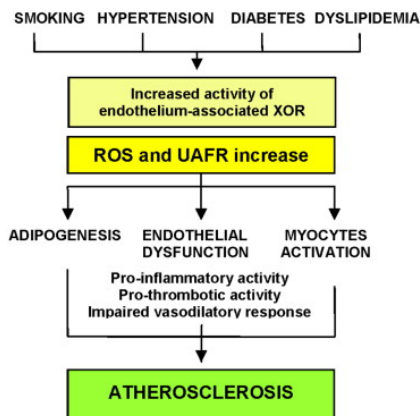


Fig.3 Complex process progression of atherosclerosis11

Molecular mechanisms: Endothelial cells exhibit dynamic state & respond to alteration in level of shear stress and various + by induction of different genes & release of various factors 4-5 In vitro & animal experiments revealed that rise in blood pressure & shear stress induces coagulation activation 6-7 Increases in shear stress, release of angiotensin & stress hormones induces TF-mRNA in vitro 8-10 these factors could contribute to elevated levels of prothrombin fragments TF 1+2.

Wilcox et al .showed the direct synthesis (mRNA expression) of coagulation proteins in the arterial wall by both smooth muscle cells & macrophages12

Limitations of the study:

1. Follow up of patient
2. Bizarre history of Anti-platelets medication.
3. Measurement of other Biomarkers of inflammation would have accurately given the cause effect relationship in hypertension, measures as CRP & P-selectin etc
4. COST EFFECTIVE factor

CONCLUSION: Enhanced inflammation, platelet activation & coagulation activation could potentially contribute TARGET ORGAN DAMAGE

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