Analysis of Prothrombin Time (PT) and Activated Partial Thromboplastin Time (aPTT) in Patients with Acute Myocardial Infarction on Anticoagulation Therapy to Assess the Thrombogenic Potential

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Abstract

Background: Activated partial thromboplastin time and prothrombin time measures the action of the intrinsic and extrinsic pathways of coagulation needed to maintain homeostasis in the body. We studied aPTT and PT levels in acute myocardial infarction (AMI) patients on anticoagulation therapies and normal subjects. The aim of the study is to assess aPTT and PT in patients of acute MI on anticoagulation.

Materials And Method: This study was conducted on 42 patients with chest pain and 84 AMI patients, admitted to Dhiraj hospital, Vadodara. The AMI patients were classified into STEMI (n=28) and NSTEMI (n=56). PT and aPTT were assayed on a fully automatic Stago-coagulometer instrument (STA Compact Max).

Conclusion: Patients with STEMI had mean aPTT of 40.79 ± 1.83 s, NSTEMI had 41.33 ± 2.06 s, aPTT in control subjects was 31.35 ± 0.48 s. Patients with acute coronary syndrome had significantly higher levels of aPTT. Patients with STEMI has PT of 17.42 ± 5 s, NSTEMI had 18.56 ± 5 s. Patients on anticoagulation therapy had higher aPTT and PT values.Both PT and aPTT are high in acute MI patients on anticoagulants. The elevations in PT values were more than 3 fold greater than aPTT suggesting that PT has a higher sensitivity for predicting blood clotting capacity in patients already on anticoagulations.

Keywords: *AMI, STEMI, PT, aPTT, thromogenic potential.*

Introduction

For maintenance of hemostasis, there is a balanced equilibrium needed between thrombus destruction and thrombus formation. This fine balance is maintained by aninteractive foreplay between platelets, coagulation factors, the vascular endothelium and the fibrinolytic system. The coagulation cascade involves an interaction between the contact activation pathway or the intrinsic system and the tissue factor pathway or the extrinsic system. These two independent pathways lead to the conversion of factor X to Xa, which is the beginning of the common pathway. This common pathway converts prothrombin to thrombin, which subsequently catalyzes the formation of fibrin and ultimately leads to the stabilization of aggregated platelets to form a stable clot[1,2]

PT measures the number of seconds it takes to form a clot in our sample of blood after reagents are added. The PT is often performed along with a partial thromboplastin time (PTT) and together they assess

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the amount and function of coagulation factors that are needed for blood clot formation.

- The PT test evaluates coagulation factors in the extrinsic and common pathways of the coagulation cascade work together: factors I (Fibrinogen), II (Prothrombin), V, VII and X.
- The aPTT, which evaluates the clotting factors that are part of the intrinsic and common pathways: XII, XI, IX, VIII, X, V, II (prothrombin) and I (fibrinogen) as well as prekallikrein (PK) and high molecular weight kininogen (HK)[3]

During a laboratory test, there are two "pathways" that can initiate clotting, the extrinsic and intrinsic pathways. Both of these then merge into a common pathway to complete the clotting process[4]. Increase fibrinopeptide A in MI predisposes patients to increased risk for sudden cardiac death[5]. Increase in fibrin turn over can be estimated by plasma concentrations of crosslinked FDP's (fibrin degradation products), proves to be a marker for risk of myocardial infarction[6]. Soluble fibrin was found significantly higher in acute coronary syndrome patients than in controls especially in young myocardial infarction[7]. The PT and PTT evaluate the overall ability to produce a clot in a reasonable amount of time and, if any of these factors are deficient in quantity or not functioning properly, the test results will be prolonged.

Materials and Method

This study was conducted on 84 AMI patients and 42 patients with chest pain, admitted to Dhiraj Hospital coronary care unit, Sumandeep Vidyapeeth deemed university, Vadodara, Gujarat for a period of 1 year from January 2019 to January 2020. The AMI patients were classified into STEMI (n=28) and NSTEMI (n=56). Mean age group of STEMI, NSTEMI patients were 60-70 years and 50-60 years respectively. All the AMI patients received standard IHD treatment according to recent ACC/AHA guidelines.

The diagnosis of myocardial infarction done by any two:

(i) Troponins levels above the 99th percentile of upper limit of normal

(ii) History of prolonged dull aching radiating chest pain or discomfort with perspiration

(iii) Presence of new bundle branch blocks, Q waves or new abnormal ST-T depressions or elevations[8].

Patients with STEMI were classified on the basis of:

(i) ST-segment elevation of ≥0.1-0.2mV in ≥2 contiguous precordial leads or ≥0.2mV in ≥2 contiguous limb leads or development of new left bundle branch block.

(ii) Presentation within the first 24 hours from first episode of chest pain.

(iii) Continuous chest pain not relieved by nitrates or rest and lasting ≥30 min[9].

Patients with NSTEMI were required to have:

(i) ST segment downloping sagging of ≥0.1-0.2 mV in ≥3 contiguous ECG leads.

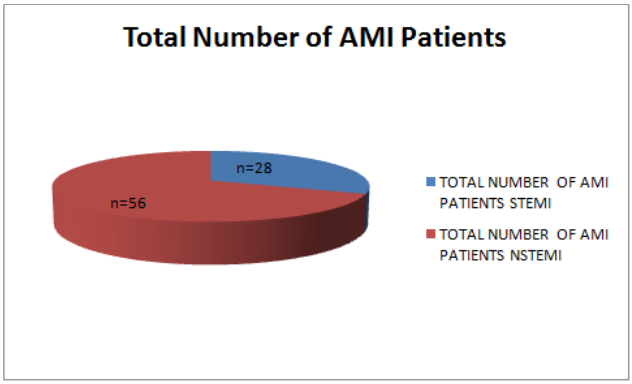
(ii) Typical radiating chest pain even at rest lasting ≥ 15 min[9].

The patient exclusion criteria included chronic inflammatory diseases, active infection, recent surgery, significant renal or hepatic dysfunction and malignancy.

Blood samples for aPTT and PT were collected in 3.2% trisodiumcitrate anticoagulant vaccutainers in the proportion of 1 volume of citrate to 9 volumes of blood is processed by centrifugation at room temperature for 15 minutes at 2000 rpm. Prothrombin time (PT), activated partial thromboplastin time (aPTT) assayed on a fully automatic Stago coagulometer instrument (STA Compact Max) which is based on clotting time or clot-based tests (i.e. chronometric) measurements and photometric assays (at specific wave lengths) on plasma samples.

Results and Discussion

This study was conducted on 84 acute MI patients and 42 patients with chest pain. The AMI patients were classified into STEMI (n=28) and NSTEMI (n=56) (Graph 1). Mean age group of STEMI, NSTEMI and chest pain patients were 65-75 years, 55-65 years and 45-55 years respectively (Table 1). Their were significant increase in PT level in NSTEMI from 13 s to 23 s as compared to control group which ranges from 11 s to 14 s. PT levels in patients with STEMI ranging from 12 s to 22 s. The level of aPTT in control subjects was 30 s to 40 s. Patients with NSTEMI shows 37 s to 47 s and STEMI shows 35 s to 45 s which has significantly higher levels of aPTT. (Table 2).



Graph 1: Total number of AMI patients.

Table 1: Mean Age group of AMI patients

Mean Age Group of AMI Patients	
AMI Patients	**Age Group in years**
Stemi	55-65
Nstemi	45-55

Table 2: Alterations in PT & aPTT

Alterations in PT & aPTT of AMI Patients			
S.No.	**Patients**	**PT**	**aPTT**
1	Stemi	12-22 sec	35-45 sec
2	Nstemi	13-23 sec	37-47 sec
3	Control	11-14 sec	30-40 sec

Patients of acute myocardial infarction are on dual antiplatelets and anticoagulants like unfractionated heparin, low-molecular weight heparin or warfarin, for which PT and aPTT are used.

PT/INR detects disorders of the extrinsic and common coagulation pathways. Abnormal result is usually seen when factor I, II, V, VII, X are deficient [10] while the aPTT looks for abnormalities of the intrinsic and common coagulation pathways. It monitors the activities of FI, II, V, VIII, IX, X, XI, XII[11]. Schwartz et al[12] studied 220 subjects with acute coronary syndrome and found out that 30 (13.6%) and 28 (12.7%) had INR and aPTT values not within the reference range. Salamonson studied that patients reaching the therapeutic aPTT threshold after heparin treatment within 1 day were less than those who did not reach threshold suggesting that a non-weight based heparin regimen is ineffective in the achievement of therapeutic aPTT[9]. Pearson showed positive correlations for myocardial infarction and death in ACS patients treated with unfractionated heparin but negative correlations between aPTT and the day of onset of recurrent angina. aPTT tended to be prolonged in the group with physical training, while it was shortened in the control group[13]. Granger et al[14] showed a positive correlation between the aPTT and the risk of subsequent reinfarction. PT and aPTT may also be used to monitor therapy in acute myocardial infarction with or without venous thromboembolism, atrial fibrillation or LV clot.

Conclusion

Both PT and aPTT are useful to measure the thinning potential of the blood and can be used to guide medical therapy and post PTCA patients also to prevent stentthrombosis. The values of aPTT are higher in acute coronary syndromes due to associated usage of anticoagulation to stop further thrombosis in coronaries. The elevations in PT values were more than 3 times higher than aPTT suggesting a greater responsive potential of PT for predicting blood clotting tendency in patients receiving anticoagulation therapy.

Ethical Clearance: Taken from sumandeep vidyapeeth deemed university ethical committee.

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