Journal of Research in Medical and Dental Science
2021, Volume 9, Issue 12, Page No: 502-504

Available Online at: www.jrmds.in
eISSN No.2347-2367: pISSN No.2347-2545

Assessment of Coagulation Factors in Various Liver Diseases

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ABSTRACT

Background: *Patients with liver disease are at a substantially increased risk of thrombosis and hemorrhage. The present study was conducted to assess coagulation factors in various liver diseases.*
Materials and Methods: 150 patients of liver diseases were divided into 3 groups based on diseases. Group I was of cirrhosis, group II had other liver disease and group III had hepatitis. APTT-was performed by Tulip diagnostic kit and PT-by Agappe diagnostic kit.
Results: *Group I had 25 males and 25 females, group II had 30 males and 20 females and group III had 28 males and 22 females. Increased prothrombin time was seen in 40 in group I, 42 in group II and 45 in group III. APTT was enhanced in 42 in group I, 28 in group II and 30 in group III. The difference was significant (P< 0.05).*
Conclusion: *Prolongation of PT and APTT in patients with liver cirrhosis indicates damage to the liver parenchyma.*

Key words: Coagulation factors, Liver, Prothrombin

HOW TO CITE THIS ARTICLE: Ishani Gupta, Roopali Jandial, Bhavneet kour, Subhash BhardwajAssessment of Coagulation Factors in Various Liver Diseases, J Res Med Dent Sci, 2021, 9(12): 502-504

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Received: 01/12/2021
Accepted: 15/12/2021

INTRODUCTION

The liver plays a crucial role in the clotting process. Acute and chronic liver diseases are invariably associated with coagulation disorders due to multiple causes including: decreased synthesis of clotting and inhibitor factors, decreased clearance of activated factors, quantitative Patients with liver disease are at a markedly increased risk of thrombosis and hemorrhage. Owing to the considerable overlap in the haemostatic oddities detected in the patients with acute infectious or toxic hepatitis, chronic hepatitis, and cirrhosis, the severity of hepatocellular dysfunction is typically more enlightening than the etiology.

Maximum coagulation factors are produced in liver, but the response of each factor to liver disease is variable due to differences in biologic half lives and acute phase reactions. The PT is usually prolonged first, then APTT. Prothrombin time (PT) and APTT correlates well with the intensity of hepatocellular damage as well as with the manifestation of abnormal bleeding and the overall prognosis and qualitative platelet defects, hyperfibrinolysis, and accelerated intravascular coagulation. Factor VII has shortest biologic half life, often affected earliest with largest decrease in plasma level. Factor VII also decreases earliest with warfarin treatment. Factor VIII: may be normal or elevated due to acute phase reactants. Factors XI and XII have long biologic half lives, and may be normal until liver disease is advanced.

Chronic hepatitis, constitutes a major health problem and can be caused by different etiological agents. In chronic liver diseases, the levels of anticoagulant proteins like antithrombin III, protein S, protein C, and alpha-2 macroglobulin are reduced. The present study was conducted to assess coagulation factors in various liver diseases.

MATERIAL AND METHODS

The present study was conducted among 150 patients of liver diseases of both genders between April 2020 to March, 2021. Enrolment of all patients in the study was done after obtaining their written consent.

Demographic data such as name, age, gender etc. was recorded. Patients were divided into 3 groups based on diseases. Group I was of cirrhosis, group II had other liver disease and group III had hepatitis. Blood samples were collected in vaccutainer containing 3.2% sodium citrate as anticoagulant. Blood to anticoagulant ratio was 9:1. Plasma was obtained following centrifugation of the anticoagulated blood at 3000 rpm for 20 minutes. APTT-was performed by Tulip diagnostic kit and PT-by Agappe diagnostic kit. Results thus obtained were subjected to ststistical analysis. P value less than 0.05 was considered significant.

RESULTS

Groups	Group I	Group II	Group III
Diseases	Cirrhosis	Other liver disease	Hepatitis
M:F	25:25	30:20	28:22

Table1: Distribution of patients.

Table 1 shows that group I had 25 males and 25 females, group II had 30 males and 20 females and group III had 28 males and 22 females.

Parameters	Group I	Group II	Group III	P value
Increase PT	40	42	45	0.91
APTT	42	28	30	0.01

Table2: Assessment of parameters.

Table 2, graph I shows that increased prothrombin time was seen in 40 in group I, 42 in group II and 45 in group III. APTT was enhanced in 42 in group I, 28 in group II and 30 in group III. The difference was significant (P< 0.05).

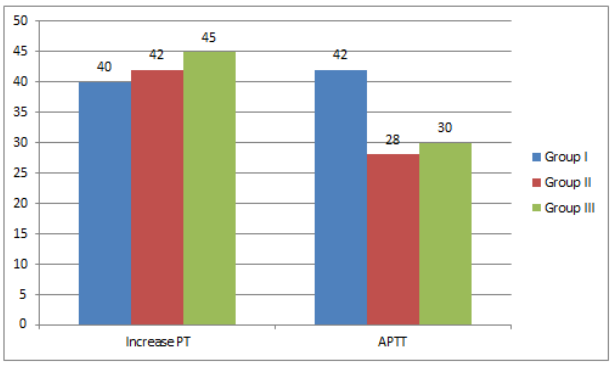

Figure1: Assessment of parameters.

DISCUSSION

The bleeding tendency accounts for high risk of morbidity and mortality in patients with liver disease undergoing diagnostic or therapeutic invasive procedures. Coagulation disorders in liver disease are widespread, complex and expensive to treat. Many mechanisms can cause the coagulation changes, but many can be attributed to cytokine activation. Sepsis further impairs hemostasis in patients with liver cirrhosis bleeding from esophageal Thrombotic events, even if rare in cirrhotic patients, can occur. In liver disease the platelet count is either normal or decreased. The liver is a source of thrombopoietin which stimulates platelet production. Mild splenomegaly may be present in cirrhosis resulting in platelet pooling. Alcohol intake inhibits the production of platelets by megakaryocytes. Folate deficiency, which may accompany cirrhosis may also cause thrombocytopenia. The present study was conducted to assess coagulation factors in various liver diseases.

In present study, group I had 25 males and 25 females, group II had 30 males and 20 females and group III had 28 males and 22 females. It was included 225 patients clinically diagnosed with liver disease who were divided into three categories: 1-cirrhosis, 2-other liver disease and 3- Hepatitis.The coagulation tests PT and APTT were performed and the results were evaluated in groups. 25 normal patients were taken as controls. Result: Out of 250 patients, 190(85%) were males and 60(24%) were females. A total of 13(6%) patients were of cirrhosis, 100 (44%) were of viral hepatitis and jaundice, and 112 (50%) were of other liver diseases and 25 normal patients (10%). Prothrombin time showed marked significant prolongation in all liver diseases. In cirrhosis: 90-100% bleeders showed elevation of Prothrombin time and non bleeders showed elevation in 50-55% cases. In viral hepatitis: 45% cases showed rise in PT. In Alcoholic liver diseases; 38.5% cases showed rise in PT. APTT is quite Significant in cirrhosis. In cirrhosis, Bleeders showed elevation of APTT in100% cases and non bleeders show 50% cases. In viral hepatitis, 25.3% rise in APTT in Alcoholic liver diseases, 25.9%cases showed rise in APTT.

We found that increased prothrombin time was seen in 40 in group I, 42 in group II and 45 in group III. APTT was enhanced in 42 in group I, 28 in group II and 30 in group III. Sale et al12 did evaluation of the frequency of coagulation abnormalities in patients with cirrhosis of liver. 82 patients presenting with cirrhosis of liver were selected and were evaluated for coagulation profile. Out of 82, fifty (60.9%) were males and thirty two (39.1%) were females. According to Child's Pughs classification, 37(45.12%) cirrhotic patients were in class A, 13 (15.85%) in class B and 32 (39.02%) in class C. The PT was prolonged (mean+SD=20.67 ± 4.12 sec) in 44 (53.65%) patients, while38 (46.34%) patients had normal PT which was less than 14 seconds (mean +SD=12.13±1.01sec). Activated partial thromboplastin time was prolonged in47 (57.31%) patients, while 35 (42.68%) patients had normal APTT which was less than 40 seconds (mean + SD=33.05 ± 3.06 sec). PT and APTT were significantly raised in cirrhotic patients. Approximately 39% CLD cases had reduced platelet count. Relative risk of GI bleeding with aberrant clotting tests in CLD cases were weakly positive for PT (RR=1.02; 95%CI, 0.49-2.10), negative for aPTT (RR=0.83; 95% CI,

0.47-1.45), strongly positive for decreased platelet counts.

CONCLUSION

Authors found that prolongation of PT and APTT in patients with liver cirrhosis indicates damage to the liver parenchyma.

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