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Study on Haemostatic Dysfunction in Acute Viral Hepatitis

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ABSTRACT

Background: Viral hepatitis leads to liver failure in only a small number of cases (<1%). The reason for development of acute liver failure in certain patients is not clear. The present study was undertaken to evaluate the platelet quantitative and qualitative dysfunction and disturbances in coagulation function in acute viral hepatitis.

Materials & Methods: The present study included 40 patients of either sex who attended medical outpatient department and got admitted in wards of R.B.M. Government Hospital, Bharatpur, Rajasthan (India) with jaundice with the history and examination suggesting viral hepatitis (with or without hepatic failure). These patients have been confirmed with viral marker for acute infection and were taken into study for haemostatic dysfunction.

Results: In our study showed the out of 40 subjects, 16 were males and 24 were females. Only Hepatitis B infection was observed in the 50-60 year old group, which accounted for 14.28% of cases of Hepatitis B. Various haemostatic factors in patients with viral hepatitis when we compared these values with control was statistical significant, only clot retraction time did not show significant.

Conclusion: We concluded that the platelet counts, clotting time, prothrombin time, activated partial thromboplastin time and fibrinogen levels show significant derangement when compared to controls, the defect being attributed to the deranged liver function.

KEYWORDS: Acute Viral Hepatitis, Platelet Counts, Clotting Time, Prothrombin Time, Activated Partial Thromboplastin Time, Fibrinogen.

INTRODUCTION

Viral hepatitis is widely prevalent in India and is responsible considerable morbidity and mortality. Chronic and acute decompensated liver disease is associated with a specific disorder of coagulation, which both increases the risk of bleeding and serves as a marker for severity of disease.[1]

Liver plays a crucial role in haemostasis. All of the major coagulation factors are synthesized in hepatocytes except factor VIII, which is synthesized in vascular endothelium and reticulothelial cells.

Impaired haemostasis and bleeding tendency may be encountered in patients with acute infection of the liver or toxic drug induced hepatitis. The external of impairment relates directly to the magnitude of liver parenchymal cell damage.[2]

Acute liver failure in most instances is associated with massive necrosis of hepatocytes as seen in viral hepatitis and drug and toxic induced hepatitis. In certain conditions, such as fatty liver of pregnancy and reye's syndrome, liver failure can occur without necrosis.[2]

In most series, the foremost cause of acute liver failure is acute viral hepatitis, accounting for upto 72% of all cases. Viral hepatitis leads to liver failure in only a small number of cases (<1%). The reason for development of acute liver failure in certain patients is not clear. Host factor as well as virulence and quantity of viral inoculation are probably important.[2]

Patients with acute liver failure or fulminant hepatic failure have an increased risk of haemorrhage due to deficiency in coagulation factors and thrombocytopenia. Such critically ill patients thus have a propensity to gastro intestinal stress ulceration and consequent bleeding.[3] Various factors which have been incriminated in the pathogenesis of bleeding are:[1]

a) Thrombocytopenia and impaired platelets aggregation.
b) Decreased synthesis of vit K dependent factors II, VII, IX, X and other factors including AT III, protein C, S.
c) Fibrinolysis

The present study was undertaken to evaluate the platelet quantitative and qualitative dysfunction and disturbances in coagulation function in acute viral hepatitis. An effort was made to correlate the findings with the severity of the liver disease, the incidence of bleeding.

MATERIALS & METHODS

The present study included 40 patients of either sex admitted in R.B.M. Government Hospital, Bharatpur, Rajasthan (India) with jaundice with the history and examination suggesting viral hepatitis (with or without hepatic failure). These patients have been confirmed with viral marker for acute infection and were taken into study for haemostatic dysfunction.

Inclusion Criteria

1. Patients with jaundice within 8 weeks of febrile illness.
2. Jaundiced patient presenting with signs and symptoms of acute liver failure.
3. Viral markers indicative of acute viral infection.

Exclusion Criteria

1. Past history of jaundice
2. History of hepatotoxic drug ingestion
3. Exposure to hepatotoxic toxins
4. Gram negative endotoxemia with shock
5. Gram positive septicemia
6. Malignant disease

Table 1: Sex Incidence of Acute Viral Hepatitis

Type of viral hepatitis	Male	Female
Hepatitis A	2 (12.5%)	4 (16.67%)
Hepatitis B	8 (50%)	6 (25%)
Hepatitis C	2 (12.5%)	2 (8.33%)
Hepatitis E	4 (25%)	12 (50%)
Total	16 (100%)	24 (100%)

Table 2: Incidence of Hepatitis in Various age groups

Age group in years	Hepatitis A Virus	Hepatitis B Virus	Hepatitis C Virus	Hepatitis E Virus
20-39 yrs	4 (66.57%)	4 (28.58%)	2 (50%)	11 (68.75%)
40-50 yrs	2 (33.33%)	8 (57.14%)	2 (50%)	5 (31.25%)
50-60 yrs	0 (0%)	2 (14.28%)	0 (0%)	0 (0%)
Total	6	14	4	16

Table 3: Comparison of haemostatic defects in patients with and without bleeding manifestation

Haemostatic parameters studied	Case with bleeding & deranged test (N=11)		Case without bleeding & deranged test (N=11)		P-value
	No.	Mean±SD	No.	Mean±SD	
Platelet count	7	1.097±1.004	5	1.92±0.98	0.044
Bleeding count	6	5.563±2.03	5	3.14±1.41	0.001
Clot retraction time	0	50.09±3.53	0	50.50±6.04	0.0842
Clotting time	4	10.40±6.90	0	4.85±1.53	0.004
Prothrombin time	10	40.16±30.66	12	25.75±21.78	0.165
APTT	9	55.07±10.22	9	47.60±12.17	0.108
Fibrinogen	7	229.54±62.115	5	266.15±59.51	0.136
Fibrin degradation product	7	-	3	-	

Table 4: Comparison of haemostatic defects in patients with acute viral hepatitis and control group

Haemostatic parameters studied	Case of acute viral hepatitis (N=40)	Control (N=40)	P-value
	Mean±SD	Mean±SD	
Platelet count	1.873±1.536	3.012±0.606	0.000
Bleeding count	3.6105±1.877	2.7388±1.223	0.016
Clot retraction time	50.075±4.643	50.75±3.128	0.448
Clotting time	6.1090±4.576	4.4538±0.957	0.028
Prothrombin time	25.87±23.028	13.35±1.312	0.001
APTT	46.3625±11.98	38.8675±3.182	0.000
Fibrinogen	261.4250±60.099	287.40±20.093	0.011

RESULTS

In our study showed the out of 40 subjects, 16 were males and 24 were females (table 1). Of the 24 female patients, 6 were pregnant and all of them were found to be Hepatitis E positive. It was observed that, Hepatitis B was the most common viral infection among males, accounting for 50% of all the cases and Hepatitis E was the most common viral infection among the female subjects, accounting for 50% of all cases.

It was observed that hepatitis A and E virus infections were commonest among the 20-39 year old group with incidence of 66.67% and 68.75% respectively. Hepatitis B virus infection was found to be more common among the 40-50 year old group, which accounted for 57.14% of cases of Hepatitis B. Hepatitis C infection was observed to be equally common among the 20-39 year and 40-50 year old group of patients. Only Hepatitis B infection was observed in the 50-60 year old group, which accounted for 14.28% of cases of Hepatitis B.

Those with bleeding manifestations showed thrombocytopenia and prolonged bleeding time in 63.7% and 54.3% patients respectively as compared to thrombocytopenia in 29.41% and prolonged bleeding time in 29.4% of cases without clinical bleeding. Platelet count & bleeding time was statistical significant (P=0.44** & P=0.001**). Prothrombin time, Activated partial thromboplastin time & Fibrinogen was not statistical significant.

Various haemostatic factors in patients with viral hepatitis when we compared these values with control was statistical significant, only clot retraction time did not show significant.

DISCUSSION

Haemostatic dysfunction in acute viral hepatitis is well recognized. The pathogenesis of haemostatic defect in acute viral hepatitis is multifactorial. In majority of cases, it is due to the impaired synthesis of a number of coagulation factors by the diseased or damaged liver.

Several workers have extensively studied the haemostatic disturbances in acute viral hepatitis from time to time.

Our study showed thrombocytopenia in 32.5% of patients with acute viral hepatitis (P=0.00***). Roma Issacs et al[4] conducted a study at Christian Medical college and Hospital, Ludhiana and reported thrombocytopenia in 29% of cases (P<0.001***). Singh DS et al[5] observed thrombocytopenia in 86% of cases through a study conducted at the Institute of Medical Sciences, BHU, Varanasi. Singh VP et al[6] also found thrombocytopenia in 50% of cases in his study at the same institute (P<0.001***). The genesis of a low platelet count is reduced production, probably due to reduced bone marrow thrombopoietic reserve, resulting from toxic and metabolic effect due to liver disease. Other factors incriminated may be disseminated intravascular coagulation, agglutinins against platelets and multiple transfusions with platelet poor blood.

Our study showed prolonged BT in 30% of cases (P=0.016). Roma Issacs et al[4] reported prolonged bleeding time in 8.8% of cases (P<0.05), while Singh DS et al[5] observed it in 78% of cases. Prasad HSK et al[7] reported prolonged BT in 78.5% of cases. This prolongation in bleeding time may be due to thrombocytopenia or defective platelet function.

Our study showed prolongation of PT in 57.5% cases (P=0.001***). Prothrombin time was prolonged in 89% of cases in the study conducted by Roma Issacs et al[4] (P<0.001***) while Singh VP et al[6] reported it in 100% cases (P<0.05***). Prolongation of PT may be due to a defect in the synthesis of coagulation factors in the liver.

45% of cases in our study showed prolonged APTT (P=0.000***). Prolonged APTT was observed by Roma Issacs et al[4] in 84% of cases in their study (P<0.001***). This prolongation of APTT indicates poor synthesis of coagulation factors. The rise in fibrinogen levels may be as an acute phase reactant while the decrease in

fibrinogen levels may be due to fibrinolysis or disseminated intravascular coagulation or reduced synthesis or dysfibrinogenemia. Fibrinogen degradation products were observed to be raised in 32% cases in our study. This could be due to fibrinolysis or disseminated intravascular coagulation.

CONCLUSION

We concluded that there is a significant defect in various haemostatic parameters in patients with acute viral hepatitis when compared to controls. The platelet counts, clotting time, prothrombin time, activated partial thromboplastin time and fibrinogen levels show significant derangement when compared to controls, the defect being attributed to the deranged liver function.

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