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Original Research Paper

General Medicine

PREVALENCE OF HEAMATOLOGICAL MANIFESTATIONS IN PATIENTS WITH CIRRHOTIC LIVER DISEASE.

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ABSTRACT This study conducted in Sree Mookambika medical college and hospital is done to assess the hematological abnormalities in cirrhotic liver patients. About 100 patients were admitted and diagnosed to have chronic liver disease. Patients were evaluated with hemoglobin, PT-INR, platelets, albumin and WBC count. According to the study, more prevalent was normocytic normochromic anemia, thrombocytopenia, leukocytosis, hypoalbuminemia and elevated PT-INR.

SUMMARY: This study was done to estimate the prevalence of the hematological manifestations in patients with confirmed alcoholic or non-alcoholic liver cirrhosis. By studying the hematological parameters like hemoglobin, PT-INR, platelets, albumin and WBC count, we would able to estimate both the prognosis of the patient and also helps in detecting the expected complications in an earlier date such that helping in prevention of the complications as far as possible.

KEYWORDS : ANAEMIA, CIRRHOSIS, HEAMATOLOGICAL, LIVER.

INTRODUCTION

One of the largest organs of the body is liver, weighing 1-1.5kg. cirrhosis liver is defined histopathologically and radiologically by blunted margins and micro or macronodularity. (1) Most common cause being alcoholic liver cirrhosis and primary biliary cirrhosis. Although it is considered not reversible, some condition like hepatitis-C and haemochromatosis are reversible. (2)

Recent studies suggest that hematological abnormalities in cirrhotic patients is associated with poor prognosis. Multiple causes contribute to the occurrence of hematological manifestation.(3)

Patients with compensated cirrhosis presents with fatigue, weight loss, anorexia, hypocalcemia and osteoporosis due to vitamin D malabsorption.(4) Decompensated cirrhosis may result in ascites, spontaneous bacterial peritonitis, hepatic encephalopathy and portal hypertension leading to variceal bleeding.(5)

Anemia in liver disease:

Anemia is due to erythropoietin deficiency which although predominantly is produced by kidney, is also produced by liver. (6) Most of the patients with alcoholic liver cirrhosis is postulated to have normocytic normochromic anaemia. Anaemia in alcoholic coagulopathy is universal due to: decreased synthesis of clotting factors and impaired clearance of anticoagulant. (7) Also attributed to thrombocytopenia and neutropenia due to hypersplenism. Leucocytosis is more common due to Spontaneous Bacterial Peritonitis and secondary peritonitis. (8)

Causes of abnormal hematological indices in cirrhosis:

- Portal hypertension-induced splenic sequestration
- Alteration in erythropoietin and thrombopoietin (9)
- Bone marrow suppression mediated by toxins (eg. alcohol, hepatitis B and C)
- Increased blood loss (eg. Hemorrhage, hemolysis)
- Consumptive coagulopathy (eg. Low grade disseminated intravascular coagulation, acquired intravascular coagulation and fibrinolysis)
- Alteration in granulocyte-colony stimulating factor and granulocyte macrophage-colony stimulating factor (10)

Management of recurrent variceal bleeding:

Reduction of portal hypertension can be done by conservative management like propranolol. Variceal bleeding can be managed by banding or stapling procedures. (11) Recurrent acute requires endoscopic therapy with or without pharmacological therapy. They are classified into compensated and decompensated liver disease for further management. Compensated liver disease patients can undergo surgical shunt vs TIPS followed by liver transplantation. (12) For decompensated cirrhosis, evaluation for transplant is done following which endoscopic therapy or beta blockers are required. Then these patients can be considered for TIPS followed by liver transplantation. (13)

Abnormalities in coagulation:

Coagulopathy is almost universal in patients with cirrhosis. There is decreased synthesis of clotting factors and impaired clearance of anticoagulants. In addition, patients may have thrombocytopenia from hypersplenism due to portal hypertension. (14)

Vitamin K requires biliary excretion for its subsequent absorption, thus in patients with chronic cholestatic syndromes, vitamin K absorption is frequently diminished. (15)

MATERIALS AND METHODS

After obtaining clearance from IEC and consent from patients, we included 100 patients of age group 21-65 years, who were admitted in Sree Mookambika Institute of Medical Sciences from September 2021 to November 2021, who had chronic liver disease were included. Inclusion criteria included confirmed cases of chronic liver disease. Relevant data were obtained, hemoglobin, PT-INR, platelets, albumin and WBC count were taken to identify patients with hematological abnormalities. Descriptive and Analytical statistics was performed by SPSS version 16. A p value of less than 0.5 was considered statistically significant.

RESULTS

Table :1 Age, Gender And Hematological Abnormalities In Cirrhotic Liver Disease

VARIABLES		PERCENTAGE
Age, years (mean)		40 years +/- 8.6
Gender	Males Females	80 20
Hematological Abnormalities	Present Absent	75 25

Out of the 100 patients included in our study, 75 percent had haematological abnormalities. Mean age of the study group was 40+/- 8.6 years. 80 percent of the study population were males and 20 percent were females.

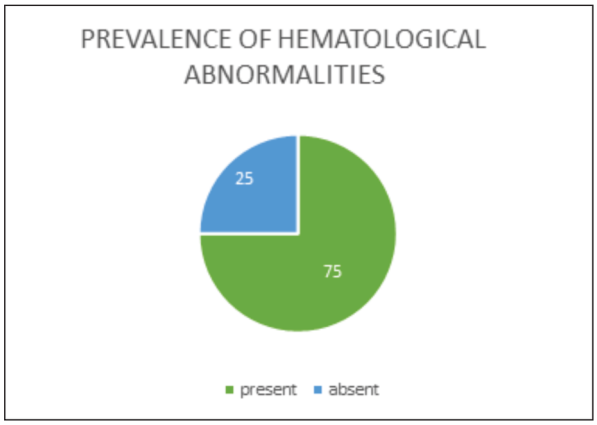

Figure 1 : Prevalence Of Hematological Abnormalities

The mean haemoglobin value calculated was 8g/dl. Among the 75 patients with anaemia, 35 patients had mild anaemia, 25 patients had moderate anaemia and 15 patients had severe anaemia. Most of these patients had normocytic normochromic anaemia in peripheral blood smear, thrombocytopenia and elevated PT-INR. Most of the patients who presented with fever and abdominal pain had leucocytosis suggestive of spontaneous bacterial peritonitis requiring oral or intravenous antibiotics. Most of the patients also had hypoalbuminemia.

DISCUSSION

Chronic liver disease patients are considered as the high-risk group, in whom there is haematological abnormalities which may result in complications like variceal bleeding, spontaneous bacterial peritonitis and bleeding manifestations like malena, haematemesis and bleeding from the injured site or intraabdominal bleeding due to coagulopathies.

In our study, a significant 75 percent of patients had haematological abnormalities among patients with chronic liver disease. According to the severity of the anaemia, among the 75 patients with anaemia, 35 patients had mild anaemia, 25 patients had moderate anaemia and 15 patients had severe anaemia. Most of these patients had normocytic normochromic anaemia in peripheral blood smear followed by macrocytic anaemia the least being dimorphic anaemia. patients with anaemia required packed red blood cells transfusion, oral supplementation with drugs and dietary supplementation based on the severity of anaemia. it was also associated with thrombocytopenia and elevated PT-INR. Thrombocytopenic patients required platelet and fresh frozen plasma transfusion based on the severity of thrombocytopenia and bleeding manifestations. The patients with coagulopathy also required vitamin K supplementation and fresh frozen plasma supplementation based on the elevated PT-INR and bleeding manifestations. Most of the patients who presented with fever and abdominal pain had leucocytosis suggestive of spontaneous bacterial peritonitis requiring oral or intravenous antibiotics. Most of the patients also had hypoalbuminemia, about half of the patients needed intravenous albumin transfusion and others were managed with oral dietary supplementations with egg whites and milk protein.

CONCLUSION

Our study showed significant number of patients with chronic liver disease having haematological abnormalities like anaemia, thrombocytopenia, leucocytosis, hypoalbuminemia and elevated PT-INR suggesting coagulopathy. Thus, it is found that early detection of these haematological abnormalities in chronic liver disease patients helps in appropriate management of anaemia. Thus, better and early management of these haematological manifestations at-least by simple dietary modifications like high protein and normal salt intake helps in preventing further complications. If dietary modifications failed, early intervention of these haematological manifestations like packed red blood cells transfusion, albumin transfusion, vitamin K supplements and fresh frozen plasma supplementation helps in improving the prognosis of the patient.

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