

A Study of Patients with Liver Cirrhosis and Portal Hypertension with **Special Reference to Hematological Profile**

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ABSTRACT

Background: Liver cirrhosis is known to lead portal hypertension and its complications thereby. Bleeding from esophageal varices is a common cause for increased morbidity and mortality in cirrhotic patients. Early detection of esophageal varices by screening endoscopy is recommended. However, this approach is limited by its availability, semi invasive nature and cost.

Methods: We undertook a study of 50 patients with liver cirrhosis, portal hypertension with special reference to their hematological profile.

Results: The incidence anemia was 92%. There was a statistically significant association between the severity of anemia and advanced grade of esophageal varices (P value-0.004).

Conclusion: The Cirrhotic patients with normal hemoglobin or only mild anemia can be closely followed up and wait for endoscopy being at a low risk for variceal bleeding, especially

INTRODUCTION

Portal hypertension is a known complication of liver cirrhosis. Splenomegaly is present in patients having portal hypertension. Bleeding esophageal varices is a life threatening complication for which screening endoscopy is indicated.¹ Both, liver and spleen are important organs of hemopoeitic system. Hematologic abnormalities are commonly seen in liver disease. These include thrombocytopenia, anemia and leucopenia in isolation or in combination. There are multiple mechanisms by which hematological abnormalities occur in liver cirrhosis.² Also liver is a site of synthesis of procoagulant proteins. So coaguloapathies are common in liver disease. The hematological abnormalities tend to increase the morbidity and mortality as well as cost burden of the patients with liver cirrhosis in the form prolonged stay and requirement of transfusion of blood products.

OBJECTIVES

- To study clinical profile of patients admitted with liver cirrhosis
- To study endoscopic findings of patients having portal hypertension and liver cirrhosis

in financially deprived countries and resource limited setting. Patients with moderate and severe anemia are likely to have large varices, requiring urgent endoscopy and possibly ligation.

Keywords: Cirrhosis, Anemia, Varices, Endoscopy. *Correspondence to:

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Received: 11-02-2018, Revised: 07-03-2018, Accepted: 28-03-2018

Access this article online				
Website: www.ijmrp.com	Quick Response code			
DOI: 10.21276/ijmrp.2018.4.2.077				

To study hematological abnormalities associated with liver cirrhosis and its correlation with endoscopic findings

MATERIALS AND METHODS

We conducted a retrospective study at our tertiary care centre over the period of 6 months from July 2017 to December 2017. It included 50 patients with liver cirrhosis and portal hypertension standard definition) who have (as per undergone gastroduodenoscopy (upper GI endoscopy). Detail clinical profile in the form of presenting symptoms, physical examination findings and laboratory reports were studied. Special emphasis was given on hematological findings. Study variable were anemia (Hemoglobin less than 11gm/dl), thrombocytopenia (platelet count less than 1 lac/cmm), leucopenia (total white blood cell count less than 4000/cmm). Further anemia was classified as mild if Hb 11 to 9 gm/dl., moderate if Hb between 7 to 9 gm/dl and severe if less than 7 gm/dl. Upper GI scopy was performed with Olympus video endoscope in the endoscopy unit. Correlation of hematological findings with severity of grades of esophageal varices was studied. Esophageal varices were grades according to Westaby

and colleagues grading system proposed in 1984, based on the luminal occupancy of esophageal varices, which has been endorsed by the British Society of Gastroenterology in its guidelines.³

Grade 1: Varices appearing as slight protrusion above mucosa, which can be depressed with insufflations.

Grade 2: Varices occupying <50% of the lumen.

Grade 3: Varices occupying >50% of the lumen and which are very close to each other with confluent appearance.

Statistical Analysis

The data was processed with statistical analysis by using Chi Square test and P value was calculated to know the statistical significance of the association of clinical and laboratory parameters.

OBSERVATIONS AND RESULTS

Total patients (n) = 50. Males were-41 and females were 9. The age range was from 16 to 80 yrs. with mean age of 45.3 years. Most common finding on clinical examination was pallor (n=48) followed by splenomegaly (n=47) (Table 1) This table shows the most common hematological abnormality was prolongation of prothrombin time followed by anemia. Leucopenia was the least common while thrombocytopenia was present in more than half of the patients. Severity of anemia had a different distribution in males and females. Maximum males (41%) tend to have mild anemia while maximum females (55%) had moderate anemia. 195 of the males and 11% of the females had severe anemia i.e. hemoglobin less than 7gm/ dl and required transfusion of packed cells during hospitalization. P value for above was - 0.004 which was significant with 99% confidence interval of 0.01.

Table 1: Physical Findings				
Findings	N			
Pollar	48 (96%)			
Splenomegaly	47 (94%)			
Icterus	34 (68%)			
Ascites	35 (70%)			
Encephalopathy	15 (30%)			

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Table 2: Hematological abnormalities						
	Anemia	Thrombocytopenia	Leucopenia	Prolonged PT		
Male	38	22	17	38		
Female	8	6	6	10		
Total	46 (92%)	28 (56%)	23 (46%)	48 (96%)		

Table 3: Correlation of anemia with grade of varices on endoscopy						
Anemia	Endoscopic Findings					
	Normal	Grade 1	Grade 2	Grade 3		
Mild	1	8	9	1		
Moderate	0	2	6	9		
Severe	0	0	0	9		

DISCUSSION

The liver plays a key role in both protein biosynthesis and lipid metabolism. As a result, hepatic synthetic dysfunction can have adverse effects on both cellular and soluble components of blood.4 Anemia in liver cirrhosis may occur due to variety of causes.⁵ The common cause being anemia due to blood loss. It Results from bleeding from the upper gastrointestinal tract in the form of variceal bleed. Esophageal and gastric fundic varices bleed and give rise to anemia. Anemia also can result due to bleeding from portal gastropathy. In our study the severity of anemia correlated with grading of esophageal varices. Severe anemia was seen in advanced grades of esophageal varices. This association was statistically significant with P value less than 0.05. Another type of anemia is spur cell anemia which is a basically a hemolytic anemia.6 It occurs due to abnormal lipid composition of the red blood cell membrane. Anemia can result from Hypersplenism as well due to portal hypertension. Nutrional deficiency of vitamins arising out of anorexia and anemia of chronic disease can both add to occurrence of anemia in liver cirrhosis.

Thrombocytopenia may result from several different mechanisms. Hypersplenism and reduced synthesis of thrombpoeitin are being the important mechanisms.^{7,8} In our study, 28 patients (56%) patient had thrombocytopenia.

Leucopenia was the least common hematological abnormality fond in our study. 23 out of 50 patients (46%) had leucopenia. Leucopenia is caused by hypersplenism in liver cirrhosis and it is usually seen with other cytopenias.⁹

Prothrombin time-The liver is the primary site for synthesis of most procoagulant and anticoagulant proteins.¹⁰ The coaguloapathies of liver disease is therefore mixed and complex. Early stage of liver disease, thrombocytopenia is there coaguloapathies associated with a prothrombotic state may be seen, whereas with more advanced disease hemorrhagic coagulopathy becomes manifest.

A study done by Kaji, B.C et al, showed that hematological markers can be non-invasive markers for portal hypertension.¹¹ Thus we can see the correlation of hematological markers with severity of portal hypertension. Thus we could also correlate severity of anemia with degree of esophageal varices.

CONCLUSION

The Cirrhotic patients with normal hemoglobin or only mild anemia can be closely followed up and wait for endoscopy being at a low risk for variceal bleeding, especially in financially deprived countries and resource limited setting. Patients with moderate and severe anemia are likely to have large varices, requiring urgent endoscopy and possibly ligation.

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Source of Support: Nil.

Conflict of Interest: None Declared.

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Cite this article as: Atmaram Chhimpa, Sanjay Sharma, Pawan Saini, Archana Ruhella, Jitendra Acharya. A Study of Patients with Liver Cirrhosis and Portal Hypertension with Special Reference to Hematological Profile. Int J Med Res Prof. 2018 Mar; 4(2):343-45. DOI:10.21276/ijmrp.2018.4.2.077