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Evaluation of platelet count in Chronic liver disease patients to predict the presence of Esophageal varices.

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ABSTRACT: Introduction: About 50-60% of patients with cirrhosis of the liver were found to have esophageal varices.[1] About one-third of patients die of bleeding gastroesophageal varices.[2] The gold standard investigation for diagnosis of esophageal varices is upper GI endoscopy.[3] Upper GI endoscopy being an invasive procedure, expensive and there are limited resources, it may therefore be more cost effective to routinely screen patients at high risk for the presence of varices, this will reduce the increasing burden and procedure costs of endoscopy units.
Aim: To find the correlation of Platelet Count with presence of esophageal varices in chronic liver disease patients.
Materials and Methods: A total of 65 patients were included in the study after applying the inclusion & exclusion criteria. All patients underwent detailed clinical examination, biochemical investigations, and Upper GI endoscopy. The platelet count was compared between the two groups of patients with and without EVs.
Conclusion: In our study we found that mean platelet count in patient with no varices on endoscopy was 171677.78/µL as compared to 91787.23/µL in group of patients with esophageal varices on endoscopy. The study was found to be significant with P value of 0.001.
Keywords: Cirrhosis, Upper GI Endoscopy, Platelet Count, Esophageal Varices.

I. INTRODUCTION

Chronic liver disease (CLD) is defined as a progressive deterioration of liver functions for more than six months. These deteriorations include synthesis of clotting factors, other proteins, detoxification of harmful products of metabolism and excretion of bile. Cirrhosis is a continuous process of inflammation, destruction, and regeneration of liver parenchyma.
Portal hypertension is a serious complication of Chronic liver disease (CLD). Portal hypertension is a result of resistance to portal blood flow because of cirrhotic and non-cirrhotic etiology. A portal venous pressure above 7 mmHg is considered as portal hypertension. Esophageal variceal bleeding is the most common life-threatening complication of CLD.[4] Upper gastrointestinal endoscopy is the gold standard for the detection of esophageal varices. Upper GI endoscopy being an invasive procedure, expensive and there are limited resources it may therefore be more cost effective to routinely screen patients at high risk for the presence of varices, this will reduce the increasing burden and procedure costs of endoscopy units. The most recent Baveno VI guidelines suggest that endoscopy is not necessary in patients with liver stiffness <20 kPa and platelets >150,000/µL, as they exhibit a very low risk of having high-risk varices (HRVs); they can therefore safely avoid screening endoscopy.[5,6]
The 1- year bleeding rate in patients with cirrhosis, without a history of hemorrhage, ranges from 6% to 76% and depends on endoscopic features as well as the degree of hepatic decompensation.[7] The majority of deaths occur within the first 6 weeks after the bleeding episode.[8]

Thrombocytopenia, defined as a platelet count below 150,000/µL, is the most common hematological complication and is often the first abnormality seen in patients with chronic liver disease (CLD).[9] Changes in platelet levels in these patients can be a result of (a) reduced production of platelets (e.g., reduced TPO), (b) splenic sequestration of platelets (e.g., portal hypertension leading to hypersplenism with an inverse relation between the spleen size and platelet count), or (c) increased destruction of platelets/increased platelet consumption (e.g., immune-mediated destruction).[10]
Thrombopoietin (TPO) production and splenic platelet sequestration are the main mechanisms for the development of thrombocytopenia in patients with CLD.[11] Thrombocytopenia affects

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approximately 6% of patients without cirrhosis and 70% of patients with cirrhosis.[12]

Data on the overall significance of platelet counts in patients with cirrhosis are equivocal as several studies indicate severe thrombocytopenia to be a significant predictor of major bleeding and re-bleeding.[13]

II. JUSTIFICATION FOR STUDY

There are huge number of patients with chronic liver disease, being asymptomatic in early stages, most of them present late in course of disease with variceal bleeding.

Therefore it is needed to screen such patients for presence of esophageal varices.

In view of less endoscopic unit and large number of cases of chronic liver disease. There is need for non-invasive predictors that can reduce the morbidity and mortality in such patients.

Hence, predicting the presence of esophageal varices through non-endoscopic and non-invasive markers is important to identify the patients who benefit from endoscopy screening. This may reduce considerable number of avoidable endoscopies.

III. MATERIAL AND METHODS

Methods of collection of data

All the patients admitted in the department of medicine and attending outpatient department of medicine at NSCB hospital, Jabalpur during the period of March 2019 to September 2020, who were fitting into the inclusion criteria, were included.

Inclusion criteria

patients with chronic liver disease within an age group of 25-80 years of age.

diagnosis was done based on a combination of history, clinical findings, impaired liver function tests, and abdominal ultrasound.

Exclusion criteria

patients with present or previous history of variceal bleed.

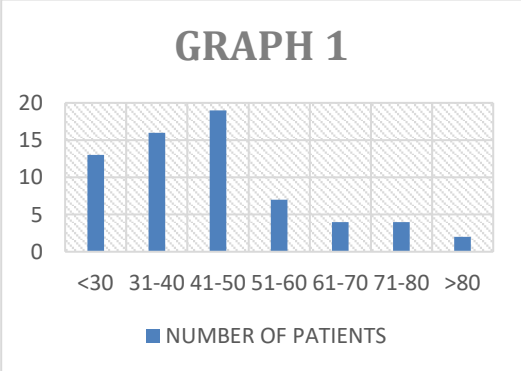
patients who have undergone sclerosis (or) band ligation of esophageal varices, TIPSS (or) surgery for portal hypertension.

patients having fever associated with thrombocytopenia in the past and h/o fever in the past 15 days will excluded.

Procedure

All patients in the study underwent a full clinical evaluation. Clinical history and physical examination findings were recorded with particular attention to present or previous hematemesis, melena, bleeding per rectum, bleeding tendencies, alcoholism, blood transfusion, intake of hepatotoxic drugs, exposure to sexually transmitted diseases, iv drug abuse, jaundice, anemia, edema, stigmata of chronic liver disease, dilated abdominal veins, ascites, splenomegaly and encephalopathy. All patients underwent biochemical tests like liver function tests, complete blood counts, renal function tests, prothrombin time, ultrasonography of the abdomen to confirm the presence of cirrhosis, ascites, and presence of collaterals. Upper GI endoscopy was done in all patients to confirm the presence of varices and to grade them. All endoscopies were performed in a single endoscopy unit using a video endoscope.

IV.OBSERVATION AND RESULT



AGE(YEARS)	NUMBER OF PATIENTS
<30	13
31-40	16
41-50	19
51-60	07
61-70	04
71-80	04
>80	02

Above table and bar diagram shows distribution of cases according to age groups. Incidence of CLD was more in age group of 41-50 years followed by 31-40 years.

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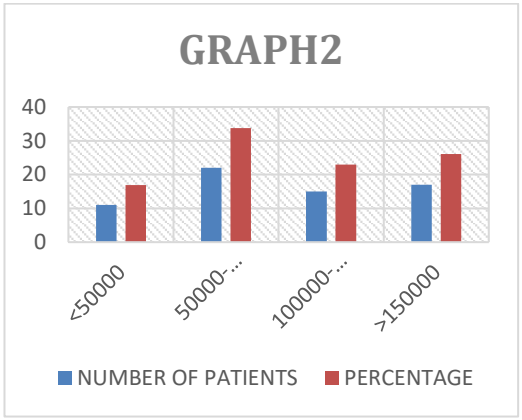
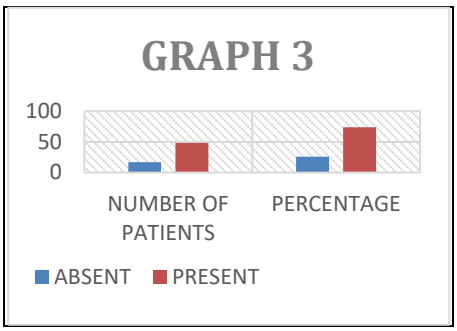
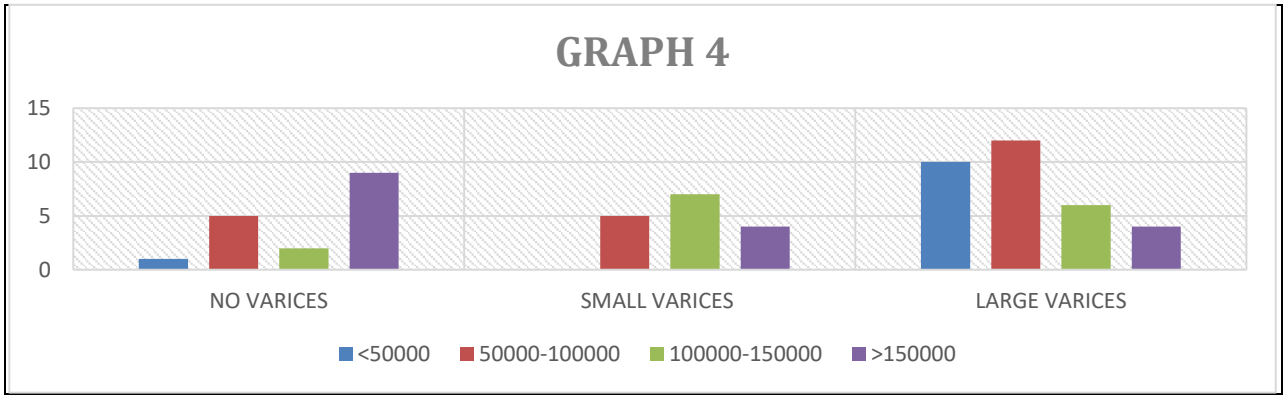


PLATELET COUNT(CELLS/µL)	NUMBER OF PATIENTS
<50000	11
50000-100000	22
100000-150000	15
>150000	17

Above table and bar diagram shows distribution of cases according to Platelet counts. Most patients had platelet count LESS THAN 150000/µL.



VARIBLES	VARICES			NUMBER OF PATIENTS
	NO VARICES	SMALL VARICES	LARGE VAICES	
PLATELET COUNT				
<50000	1	0	10	11
50000-100000	5	5	12	22
100000-150000	2	7	6	15
>150000	9	4	4	17



Above table and bar diagram shows small and large varices were more commonly seen in patients with platelet count less than 1,00,000/µL. As the platelet count decreases, patient with esophageal varices on endoscopy increases.

STATISTICAL ANALYSIS OF DIFFERENT VARIABLES REGARDING THEIR SIGNIFICANCE IN ESTIMATING PRESENCE OF ESOPHAGEAL VARICES IN PATIENT WITH CLD.

VATIABLES	VARICES				t TEST	P VALUE
	NO		YES			
	MEAN	SD	MEAN	SD		
PLATELET COUNTS	171677.78	130547.66	91787.23	47084.08	3.66	0.001

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V. DISCUSSION

The current gold standard techniques to assess portal hypertension includes the invasive evaluation of hepatic venous pressure gradient (HVPG) and endoscopy. But these are invasive procedures and cannot be performed in settings not equipped for these procedures.[14] So in our study we considered only simple, commonly available, reproducible parameters. In the present study, total of 65 patients were studied. Endoscopy was performed to detect the presence or absence of varices.

According to GBD 2017 Cirrhosis Collaborators in almost all regions, the number of deaths from CLD peaked in the middle-aged age groups (approximately 50–74 years).[15] As shown in **Table and Graph no.1**, in our study also, incidence of CLD was maximum in age group of 41-50 years followed by 31-40 years. Our study was also in accordance with the study conducted by Mukherjee PS et al, Sharma B et al and Goyal P et al, in their study the mean age of patients was 54 ± 9.3 years.[16,17,18] As shown in **Table and Graph no.2**, most patients in our study had platelet count less than 150000/µL which was according to Priyadarshi BP et al; in their study 44% of the patients had platelets in the range of 50000 to 99000/µL followed by 21% of the patients having platelets in the range of 100000 to140000/µL.[19] Also Zaman A et al. reported that groups without varices had a higher mean platelet count (mean platelet count 1,28,500) than the group with small varices (mean platelet count, 1,07,800) and platelet count of <90,000 increased the risk of having EV by nearly 2.5 fold.[20]

Varices eventually develop in all patients with liver cirrhosis and they tend to increase in size with time and also have increased chance of bleeding. We also know that the prevalence of varices is higher in decompensated than in compensated cirrhosis. The reported prevalence of EVs is varied. As depicted in **Table and Graph no.3**, upper GI endoscopy showed presence of varices in 48 out of 65 patients, which was same as the study done by Hossain E et al who through endoscopic examination of EV in the study population stated that "majority, i.e., 45 (45.0%) patients, had medium EV followed by 27 (27.0%) had small and 19 (19.0%) patients had large EV. 9 (9.0%) patients had no EV". That means maximum study population showed the presence of esophageal varices.[21]The study conducted by Filippo Schepis et al. and Chalasani et al also found the presence of EVs in maximum study patients in their respective studies.[22,23]

As seen in **Graph no.4**, we found that as platelet count decreases chances of EVs increases. This was also correlated by Priyadarshi B P et al; in their study among 100 patients studied 90% patients were found to have esophageal varices. Based on endoscopic grading, incidence of grade 2 and grade 3 esophageal varices predominated, accounting to 48% and 23 % respectively. On correlation of platelet count with grades of esophageal varices it was evident that 44 patients had their platelet count less than 1 lac out of which 24 patients had grade 2 varices followed by 14 patients with grade 3 varices. Thus they concluded by their study that with decrease in platelets count the chances of formation of higher grades of esophageal varices increases and a positive association exists.[19]

Schepis et al. reported screening by gastroscopy where platelet count <100,000/µL resulted in a significant yield of esophageal varices.[22] Another study of cirrhotic patients without history of variceal bleeding who underwent gastroscopy as part of a liver transplant evaluation, found platelet count of <88,000/µL associated with the presence of large varices. The discriminating threshold for the presence of varices varied widely, ranging between 68,000 and 160,000/µL.[24] The present study was not in accordance with the study done by Qamar AA et al; who found no definite level of platelet count that accurately predicted the presence of esophageal varices (AUROC curve 0.63) and they, therefore, concluded that platelet count is an inadequate noninvasive marker for prediction of the presence of esophageal varices. In an attempt to improve the predictive value of the platelet count, it can combined with other variables.[25]

VI. CONCLUSION

Most current guidelines recommend that all cirrhotic patients be screened by upper gastrointestinal endoscopy for the presence of EVs at the time of diagnosis. As discussed previously, the screening upper gastrointestinal endoscopy is recommended every two to three years in patients without varices, and repeat endoscopy is recommended every one to two years in patients with small varices. Our study was an attempt to evaluate the relation of platelet count in patient of Chronic Liver Disease with presence of esophageal varices. Our study we found that small and large varices were more

commonly seen in patients with platelet count less than 1,00,000/µL.As the platelet count decreases, patient with esophageal varices on endoscopy increases. Should we therefore change our policy and restrict screening endoscopy to those patients with platelet count <1,00,000/µL. We believe that some issues still need to be addressed before we take such a radical step. First the sample size in our study was low and Second our study was a univariable Assessment that included only Platelet count. There may other variables apart from platelet count alone that may have influenced the end result in our study.Hence we believe that more studies may be required in a larger population of Chronic Liver Disease patients for validation and to determine a cut-off value that can be safely recommended for the noninvasive diagnosis of EV using platelet count.

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