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CLINICAL PROFILE OF 100 PATIENTS WITH PORTAL HYPERTENSION IN SIR T. GENERAL HOSPITAL, BHAVNAGAR.

Medicine

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ABSTRACT

Objective: To study clinical & investigative profile of patients with portal hypertension. To discuss etio-pathogenesis of portal hypertension. To study the predictive power of non-invasive investigative parameters (clinical, biochemical, radiological) for detection of esophageal varices in patients with portal hypertension (PHT) as compared to invasive parameters (upper gastrointestinal endoscopy). To study outcome in patients of portal hypertension.

Materials and methods: 100 patients with PHT, from April 2015 to March 2016, were studied. Those who had age <12 years & not giving consent were excluded. Detailed clinical history was taken and physical examination was done. Patients underwent the required hematological, biochemical, radiological and endoscopic investigations.

Results: Alcoholic liver disease is most common cause of portal hypertension with middle aged males being most commonly affected population. Hepatic encephalopathy & hepatorenal syndrome are major causes of mortality in patients with portal hypertension, whereas esophageal varices & ascites are most common specific presenting complications causing large number of morbidity. Platelet count/spleen size ratio showed a significant correlation between presence or absence and grade of esophageal varices ($p < 0.00015$). If a cut-off value of 1,000 is taken, then 85% patients with esophageal varices have ratio <1,000 while 26% of patients with ratio <1,000 did not have any varices. It was also observed that lower the ratio, higher the grade of varices.

KEYWORDS:

Portal hypertension, non-invasive predictors, platelet/spleen size ratio, esophageal varices, ascites, hepatic encephalopathy, hepatorenal syndrome.

INTRODUCTION

Portal hypertension is the most common complication of cirrhosis accounting for significant morbidity and mortality mainly because of variceal haemorrhage, ascites, bacterial infections, hepatic encephalopathy, and hepatorenal syndrome. Alcoholic liver disease is the commonest cause of cirrhosis of liver, culminating in portal hypertension. By detecting portal hypertension early in its course, taking appropriate measures to prevent its complications will help in reducing morbidity and mortality associated with it.

Materials & Methods

The cross Sectional study was conducted on 100 patients from April '15-March '16 in Department of General Medicine, Govt. Medical College, Bhavnagar after taking permission from IRB (HEC).Patient were subjected to following tests: Hemogram with thin peripheral smear and ESR, RBC indices, prothrombin time and INR, serum bilirubin, alkaline phosphate (ALKP), alanine transaminase (ALT), aspartate transaminase (AST), total serum proteins, albumin and globulin levels, serum electrolytes(namely sodium & potassium)and blood urea, serum creatinine, random blood sugar, ascitic fluid analysis, HbsAg, HCV, chest X-ray, abdominal ultrasonography and portal vein Doppler and upper gastrointestinal endoscopic examination.

Table 1 Inclusion, Exclusion & Diagnostic criteria for patients in present study

Inclusion Criteria	Age >12 yrs	
	Patients giving written consent for study	
	Patients fulfilling the criteria for PHT	Radiological: Portal vein diameter on USG (>12mm) or evidence of portal hypertension on portal venous Doppler study Clinical: Ascites, Hematemesis, Splenomegaly, Jaundice Esophageal varices are graded as follows:18 Grade 0: No varices Grade 1 (F1): varices depressed by endoscope. Grade 2 (F2): varices not depressed by endoscope. Grade 3 (F3): varices are confluent around the circumference of the esophagus.
Exclusion Criteria	Patient with age <12yrs	
	Patients not giving consent for participation	
Diagnosis	Radiological+ Any of clinical criteria mentioned above or	
	Endoscopic varices + Any of clinical criteria mentioned above	

Results & Discussion

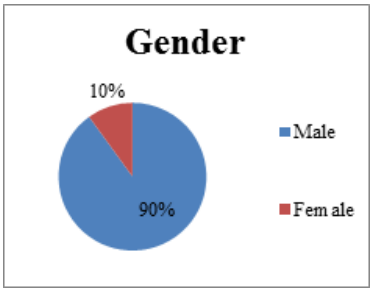


Figure 1 Gender distribution of cases of portal hypertension in present study

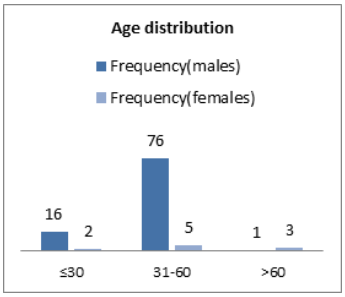


Figure 2 Age distribution of incidences of portal hypertension in present study

Clinical Presentation

Constitutional symptoms like fever, anorexia, malaise & headache (98%) and hematemesis/mucosal bleeding (65%) were most common presenting features, followed by mucosal bleeding, abdominal distention, jaundice and pedal edema. Pallor (65%), ascites (54%) and splenomegaly (70%) were common signs followed by icterus (44%).Anemia with hemoglobin (Hb) <12 g/dl was found in 65% of cases. Elevated prothrombin time (65%), Platelet count <150,000/cu mm (63%), serum albumin <2.9 g/dl (54%) and in descending order of frequency were the common abnormalities.

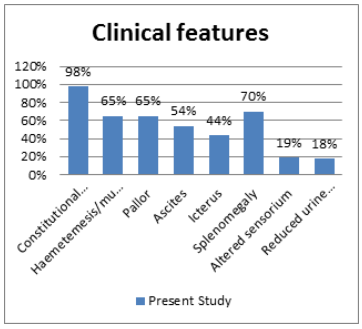


Figure 3 Clinical features in cases of patients in present study

Laboratory parameters

In present study, 18 cases presented with reduced urine output had serum creatinine level > 2 mg%. In present study, out of 54 cases presented with ascites, 14(26%) of them had exudative type of asicitic fluid on laboratory examination. They were found to have spontaneous bacterial peritonitis. Serum bilirubin was raised in 44 cases. ALT was raised in all of these cases, while AST was raised in 41, suggesting that ALT being more specific enzyme for liver. 19(43%) of the 44 cases had hepatic coma.

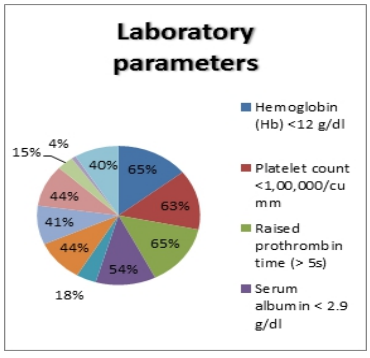


Figure 4 Laboratory parameters of patients in present study

Etiology

The etiology was alcoholic liver disease in 81%, chronic viral etiology in 5% and portal vein thrombosis in 3% of cases.

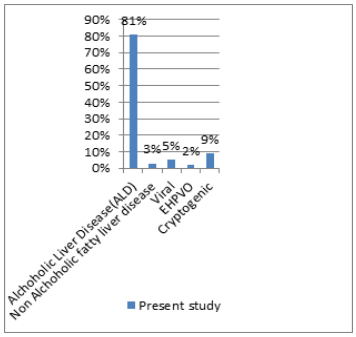


Figure 8 Etiological distribution in present study

Diagnosis

In cases, ultrasonography showed liver parenchymal disease in 98% of the cases, in the form of altered echo texture in 87% of the cases. In 2% of cases extra hepatic portal venous obstruction was present. Ultrasonography showed splenomegaly in 80% of cases and ascites in 88% of cases. Portal vein diameter was >12 mm in 68% of cases; splenic vein diameter was >7 mm in 70%. In 46% of cases portal venous Doppler was done which showed evidence of portal hypertension, irrespective of portal or splenic venous size, based on flow in collaterals. Esophageal varices were found in 60% of cases, 27% had Grade 1, 35% had Grade 2 and 38% had Grade 3 esophageal varices. It was observed that packed cell volume (PCV), mean corpuscular volume (MCV) and WBC count did not show any significant correlation with esophageal varices. Serum bilirubin and liver enzymes like ALT, AST and ALKP failed to show any significant correlation with size and presence or absence of esophageal varices. Similarly, serum albumin had nothing to do with esophageal varices.

Correlation between platelet count/spleen size and esophageal varices

Platelet count/ spleen size ratio showed a significant correlation between presence or absence of esophageal varices ($p < 0.00015$) ( ). If a cut-off value of 1,000 taken, then 85% (51/60) cases with esophageal varices have ratio <1,000 while 26% (6/23) of patient with ratio <1,000 did not have any varices. It was also observed that lower the ratio, higher the grade of varices. The Sensitivity & Specificity were 70% & 80%, respectively, with the Positive predictive value & Negative predictive value being 92.7% & 67.9%, respectively. But no correlation can be made between platelet count/ spleen size ratio & grade of esophageal varices (Figure 7).

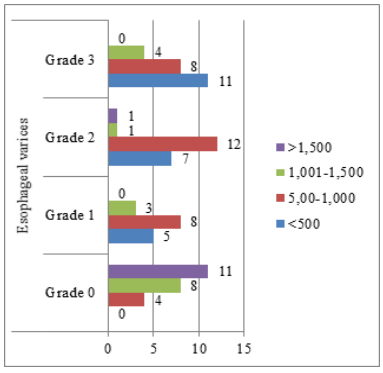


Figure 6 Correlation between platelet count/spleen size and esophageal varices

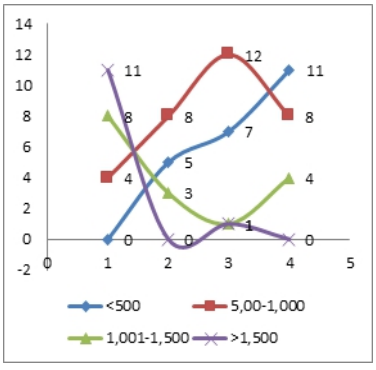


Figure 7 Correlation between platelet count/spleen size and grading esophageal varices

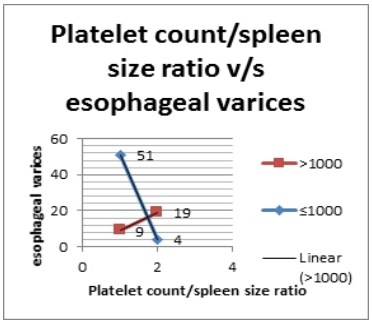


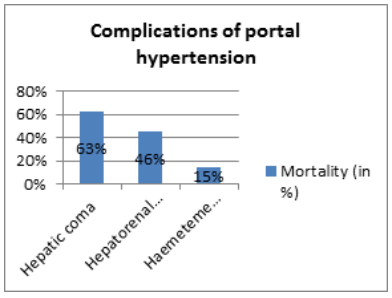
Figure 8 Correlation between platelet count/splenic size and presence esophageal varices

Mortality among patients of portal hypertension

Out of 31 deaths, 12(39%) were due to hepatic coma, 10(32%) were due to hematemesis, 7(22%) were due to hepatorenal syndrome. 2(6%) deaths were due to other complications than mentioned above. 10 out of 65 cases admitted with hematemesis died during the course of treatment. Prognosis was relatively better among this group of cases than other 2 groups.

Hepatic coma, hepatorenal syndrome & hematemesis are leading causes of mortality in cases with portal hypertension (Figure 10).

Out of 100 cases, 18 cases were having hepatorenal syndrome. 11 out of 18 cases received terlipressin injection. The response was dramatic as serum creatinine levels normalize within a week of initiation of treatment (Figure 11).



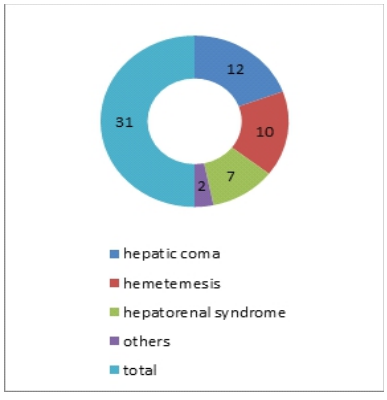


Figure 10 Mortality among patients of portal hypertension

In present study, 19 cases presented with altered sensorium were diagnosed as having hepatic coma. Out of them, 10 had Hypokalemia. 4 of them patient had hematemesis ( ).

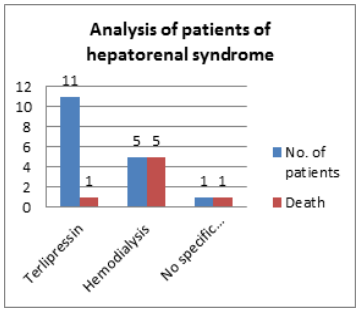


Figure 10 Analysis of patients of hepatorenal syndrome

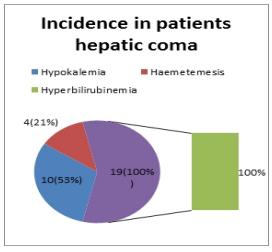


Figure 10 Analysis of patients with hepatic coma

Prognosis: MELD score

In this study, we have used MELD score to determine its relation with mortality.

Equation 1

MELD score = 3.8 x log (e) (bilirubin mg/dL) + 11.2 x log (e) (INR) + 9.6 log (e) (creatinine mg/dL) [INR: international normalized ratio].

Since patients present at different stages in the course of their liver disease, it is difficult to predict the rate of progression to severe decompensation. At the time of the initial evaluation for liver transplantation, the prospective recipient is given a baseline MELD score based on current INR, creatinine and total bilirubin, which is intended to reflect the severity of liver disease. A single MELD score at the time of presentation can accurately reflect mortality risk. Patients with MELD score > 20 have massive rise in in-hospital mortality as per data given below (Figure 13).

SALIENT FEATURES

Most commonly affected patients were middle-aged males coming from lower socioeconomic class. Most common etiology for PHT was alcoholic cirrhosis of liver.

Hematemesis was the most common specific presenting complaint followed by abdominal distention, jaundice and edema over feet. Pallor, splenomegaly, ascites were common signs followed by and icterus. Ascites & hematemesis are being most common complications & major causes of morbidity in patients of portal hypertension. With the help of ultrasonography portal venous diameter >12mm or spleen vein diameter >8mm, diagnosis of portal hypertension can be made (p value<0.005). Ascites can easily be diagnosed by ultrasonography & managed partially with diuretics. Refractory ascites requires specialized surgical procedures.

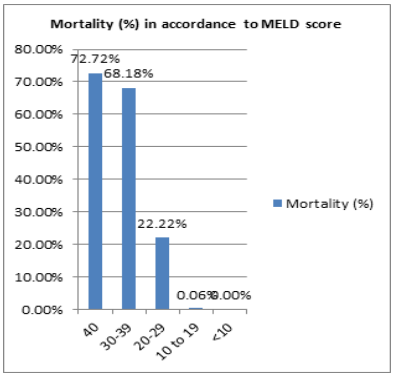


Figure 11 Prognosois in accordance with MELD score

Endoscopy is an invasive diagnostic procedure to diagnose & treat esophageal varices. Therefore, introduction of noninvasive parameters for assessment of presence and size of esophageal varices has been one of the objectives.

A cut-off value of platelet count/spleen diameter <1,000/cu mm. Eighty-five percent of patients with varices had ratio <1,000. The sensitivity was 85% and specificity was 82.6%. Nevertheless, this platelet count and spleen size ratio may serve for selection of patients who need more frequent endoscopies. This ratio will help to identify patients at higher risks for development of esophageal varices.

Hepatic coma, followed by hepatorenal syndrome is most dreadful complication, having got highest mortality among the patients of portal hypertension. MELD score should be calculated in patients with portal hypertension at each clinical visit. Patients with MELD score 20 or above should be referred to a specialist for liver transplant evaluation.

Conclusion

Esophageal varices & ascites are major causes of morbidity in portal hypertensive patients. Hepatic coma & hepatorenal syndrome are leading causes of death in these patients.

In conclusion, the platelet/spleen size ratio cannot substitute for upper gastrointestinal endoscopy in the scrutiny of esophageal varices. However, this ratio can be a useful noninvasive method. Cirrhosis of

liver is largely a preventable condition because the commonest etiology is alcoholic cirrhosis of liver that can be largely prevented by abstinence of alcohol.

The Model for End Stage Liver Disease (MELD) score provides accurate short-term prognostic information and should be calculated on any patient with cirrhosis or advanced liver disease.

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