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A STUDY OF CLINICAL PROFILE OF PORTAL HYPERTENSION PATIENTS & EVALUATION OF NON INVASIVE PREDICTORS OF ESOPHAGEAL VARICES AT TERTIARY CARE CENTRE, KARWAR

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Abstract

Background: Portal hypertension (PH) is defined as when the portal venous system pressure exceeds 10 mm Hg. **Objective:** To study the predictive power of noninvasive investigative parameters (clinical, biochemical, radiological) for detection of esophageal varices in patients with portal hypertension (PHT) as compared to invasive parameters (upper gastrointestinal endoscopy). **Materials and methods**: This was a prospective observational study.Fifty patients with Portal Hypertension were studied at tertiary care hospital , KRIMS, Karwar, during Jan 2022 to June 2022. Those who had decompensated liver diseases, HIV, hepatocellular carcinoma, metastasis in liver, parentral drug addiction, chronic febrile illness, H/O treatment taken for PHT in the form of surgery or endoscopic bending or sclerotherapy were excluded. Detailed clinical history was taken and physical examination was done. All patients underwent the required hematological, biochemical, radiological, endoscopic and histopathological investigations. **Results:** Platelet count/splenic size showed a significant correlation between presence or absence and grade of esophageal varices ($p < 0.00015$). If a cut-off value of 1,000/cu mm is taken, then 87.5% (35/40) patients with esophageal varices have ratio of<1000, while 20% (2/10) of patients with ratio <1,000 did not have any varices. It was also observed that lower the ratio, higher the grade of varices. **Conclusion:** Asymptomatic esophageal varices, which is quite common, can be easily diagnosed with invasive endoscopy or otherwise can be suspected with non invasive predictors like platelet/spleen size ratio in our country, where financial constraint is a major problem for investigations like endoscopy.

Keywords: Portal hypertension, noninvasive predictors, asymptomatic esophageal varices, platelet/spleen size ratio

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Introduction

Portal hypertension (PH) is defined as when the portal venous system pressure exceeds 10 mm Hg.[1] Portal hypertension is characterised by the increase in portosystemic pressure gradient in any portion of the portal venous system . Portal hypertension is the main complication of liver cirrhosis.[2,3] In 2007 in United States, liver cirrhosis accounted for 30,000 deaths; making it the 12th leading cause of death in the US, this was in accordance with the National Institute on Alcohol Abuse and Alcoholism (NIAAA).[4] In western countries, cirrhosis of the liver accounts for more than 90% cases of portal hypertension.5 It is reported that in India, extra hepatic portal venous obstruction (EHPVO) is responsible for about one third cases of adults and more than half of the cases in children as a cause of portal hypertension.[5]

The causes for portal hypertension are

1. Pre hepatic PH [normal wedged hepatic venous pressure (WHVP), free hepatic venous pressure (FHVP) with normal hepatic venous pressure gradient (HVPG)],
2. Hepatic PH (increased WHVP, normal FHVP, and increased HVPG) and
3. Post hepatic PH (increased WHVP and FHVP and normal HVPG)[6]

Portal hypertension (PHT) commonly accompanies cirrhosis of liver. Development of esophageal varices is one of the major complications of PHT. A major cause of PHT-related morbidity and mortality is the development of variceal hemorrhage, which occurs in 25-40% of patients. Esophageal varices are diagnosed by endoscopy. Further follow-up should then relate to the initial size of varices. In case of large varices, endoscopic follow-up is not necessary and primary prophylaxis with a nonselective β-blocker should be started. Endoscopic band ligation is useful in preventing variceal bleeding in patients with medium or large varices.[7]

Accurate identification of patients at the highest risk of bleeding allows stratification in an attempt to avoid unnecessary preventive measures in 60-75% of patients who will never have variceal bleeding in future. In order to reduce the increasing burden that endoscopy units will have to bear, some studies have attempted to identify characteristics that noninvasively predict the presence of large esophageal varices like platelet count and splenomegaly.[8] In this study, we have used the platelet count/spleen diameter ratio as a noninvasive predictive parameter. Apparently, the decrease in platelet count in all these patients was most likely due to hypersplenism because of PHT.

Materials And Methods

This was a prospective observational study.

This study was conducted at tertiary care hospital, KRIMS,Karwar. The study was conducted from Jan 2022 to June 2022 and 50 patients were studied. Adult patients with PHT, which had been diagnosed clinically, biochemically, radiologically and endoscopically were included. Those who had decompensated liver diseases, HIV, hepatocellular carcinoma, metastasis in liver, parentral drug addiction, chronic febrile illness, H/O treatment taken for PHT in form of surgery or endoscopic banding or sclerotherapy were excluded. All 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

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globulin levels, serum electrolytes and blood urea, serum creatinine, random blood sugar, ascitic fluid analysis, HbsAg, chest X-ray and abdominal ultrasonography and portal vein Doppler and barium swallow, upper gastrointestinal endoscopic examination and percutaneous liver biopsy. This study was approved by Institutional ethical committee of KRIMS,Karwar.

Esophageal varices are graded as follows:

Grade 0: No varices

Grade 1: Varices small and straight

Grade 2: Varices obliterating less than one-third of esophageal lumen

Grade 3: Varices obliterating more than one-third of esophageal lumen.

Statistical Analysis: All data were instilled chronologically and were calculated statistically. Subgroup analysis were done for statistical significance. Statistical analysis was carried out by IBM SPSS Statistics for Windows version 25.0. All 'p" values <0.05 were considered significant.

Results

There was a male preponderance (M:F = 2.1:1), with mean age of 41 ± 10.9 years. Most were from lower socioeconomic class. This is due to high prevalence of alcoholism in this group.18 Constitutional symptoms (100%) and abdominal distension (80%) were most common presenting features, followed by jaundice and pedal edema. Pallor (88%), ascites (80%) and splenomegaly (70%) were common signs followed by icterus (52%). Anemia with hemoglobin (Hb) 13 mm in 58% of patients; splenic vein diameter was >7 mm in 70%. Asymptomatic esophageal varices were found in 80% of patients, 20% had Grade 1, 26% had Grade 2 and 34% had Grade 3 esophageal varices.

It was observed that Hb, 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, ALKP failed to show any significant correlation with size and presence or absence of esophageal varices. Similarly, portal vein diameter, splenic vein diameter and serum albumin had nothing to do with esophageal varices. There was significant correlation between platelet count and esophageal varices (Table 1). In the present study, 70% (28/40) patients with esophageal varices had platelet count 120 mm. Forty percent (5/11) with normal longitudinal spleen diameter had no varices. Ninety-two percent (26/28) patients with Grade 2 or 3 varices had spleen diameter >120 mm (Table 2). Eighteen percent (2/11) patients with normal spleen size had Grade 2 or 3 esophageal varices ($p < 0.014$).

Platelet count/ spleen size ratio showed a significant correlation between presence or absence of esophageal varices and grade of esophageal varices ($p < 0.00015$) (Table 3). If a cut-off value of 1,000/cu mm is taken, then 87.5% (35/40) patients with esophageal varices have ratio while 20% (2/10) of patient with ratio <1000 did not have any varices. It was also observed that lower the ratio, higher the grade of varices.

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Table 1: Correlation between Platelet coiunt & esophageal varices

Platelet count (cumm/dl)	EV grade 0	EV grade 11	EV grade 2	EV grade 3
<50,000	0	1	0	5
50,001-1,00,000	2	6	8	8
1,00,001-1,50,000	0	3	3	2
1,50,001- 2,00,000	4	0	1	2
2,00,001- 2,50,000	4	0	1	2
>2,50,000	0	0	0	0

*EV- Esophageal varices

Table 2: Correlation between Spleenic size & esophageal varices.

Spleen size (mm)	EV grade 0	EV grade 1	EV grade 2	EV grade 2
<120	5	4	1	1
>120	5	6	12	14

Table 3: Correlatio between platelet count /speeni size ratio & Esophageal varices.

Platelet count/Splenic size ratio	EV grade 0	EV grade 1	EV grade 2	EV grade 3
<500	0	3	2	10
501-1000	2	6	9	5
1001-1500	4	1	1	2
>1500	4	0	1	0

Discussion

Most commonly affected patients were middle-aged males coming from lower socioeconomic class. Most common etiology for PHT was alcoholic cirrhosis of liver, which is a potentially preventable form. Abdominal distension was the most common specific presenting complaint followed by jaundice and edema over feet. Pallor, ascites were common signs followed by splenomegaly and icterus. Incidence of esophageal varices in patients with PHT is approximately 90-95%, but only 30-50% develop variceal bleeding, which is usually associated mainly with fatal outcome.

Therefore, regular control and evaluation of esophageal varices with timely introduction of nonselective b-blockers and variceal ligation play an important role in prevention of bleeding. Endoscopy is an invasive and costly diagnostic procedure. Therefore, introduction of noninvasive parameters for assessment of presence and size of esophageal varices is a major goal of numerous studies. To date, seven studies have been published concerning the noninvasive diagnosis of the presence of either any esophageal varices or large esophageal varices in patients with PHT.[9-13] The reason for this effort is simple: the number of patients undergoing screening for the presence of esophageal varices is going to increase in the near future as a result of the growing pool of patients with chronic liver disease.[14,15]

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In general, most important noninvasive parameters esophageal varices are decreased platelet count and splenomegaly.[16] A p value of 120 mm then it has specificity of 85% and sensitivity of 50%. We also tried to corroborate other parameters like portal vein diameter, splenic vein diameter and serum albumin with presence or absence of esophageal varices or grade of esophageal varices but specificity was low and $p < 0.5$, which is not significant. Only 12% of patients with esophageal varices (10% had large varices) were missed considering a cut-off value of platelet count/splenic diameter.[17-18]

Conclusion

Portal hypertension is largely a preventable condition because the commonest etiology is alcoholism. Asymptomatic esophageal varices, which is quite common, can be easily diagnosed with invasive endoscopy or otherwise suspected with noninvasive platelet/spleen size ratio in country like ours, where financial constraint is a main problem. It can be very useful and applicable at small centers like community health centers (CHCs) and primary health centers (PHCs) in our country with limited resources.

Conflict of interest: Nil

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