FREQUENCY OF GASTRIC VARICES IN PATIENTS WITH PORTAL HYPERTENSION BASED ON ENDOSCOPIC FINDINGS

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ABSTRACT

Objective: To find out the frequency of gastric varices in patients with portal hypertension based on endoscopic findings.
Study Design: Descriptive Study.
Place and Duration of Study: Department of Gastroenterology, Military Hospital, Rawalpindi from Jan to Jun 2011.
Material and Methods: All patients fulfilling the inclusion criteria were selected through consecutive sampling. The patients presenting with hematemesis, melena or ascites with portal hypertension on ultrasound abdomen were admitted in the hospital. The patients were first stabilized hemodynamically and then kept empty stomach for at least four hours before endoscopy. The patients were sedated with intravenous midazolam and endoscopic findings obtained were entered on the patient proforma.
Results: The overall frequency of gastric varices was 11%, whereas 89% had no gastric varices.
Conclusion: A large number of patients with portal hypertension have gastric varices. It is recommended that endoscopy be carried out in all patients with identified portal hypertension.

Keywords: Esophageal varices, Gastric varices, N-butyl-cyanoacrylate, Portal hypertension.

INTRODUCTION

Portal hypertension is defined as a pressure in the portal veins that is at least 5mm higher than the pressure in the inferior vena cava[1]. The increased pressure is due to a functional obstruction to blood flow from any point in the portal system's origin, through the hepatic veins, into the systemic circulation or from an increase in blood flow in the system. Normal portal pressure is defined between 5 and 10 mm Hg. However, once the portal pressure rises to 12mm Hg or greater, complications can occur, such as varices and ascites. Many conditions are related with portal hypertension, of which cirrhosis is the most common. When portal hypertension is present in lack of cirrhosis, the condition is called "non-cirrhotic portal hypertension". The causes of noncirrhotic portal hypertension can be separated into prehepatic, intrahepatic (presinusoidal, sinusoidal, and post sinusoidal), and post hepatic causes[1].

Portal venous pressure (P) is the product of vascular resistance (R) and blood flow (Q) in the portal bed[2] In cirrhosis, both intrahepatic.

vascular resistance and portal flow are increased[3] Portal hypertension leads to the development of Porto systemic collaterals. However, due to their higher resistance and increased portal venous inflow, these collaterals are unable to diminish the hypertension.

Gastroesophageal variceal hemorrhage is a common and serious complication of portal hypertension and is a leading cause of morbidity and mortality in patients with liver cirrhosis. Advanced liver failure, failure in controlling variceal bleeding, early rebleed, and marked elevations in portal pressure are related with increased mortality[4]. Varices are Porto systemic collaterals created after preexisting vascular channels have been dilated by portal hypertension. The most commonly used

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measurement for portal pressure is the hepatic venous pressure gradient (HVPG), (normal gradient being <5 mm Hg). At a hepatic venous pressure gradient of less than 12 mm Hg, varices do not form. However varices are not invariable in patients with gradients of 12 mm Hg or more; thus, this pressure gradient is obligatory but not sufficient for variceal formation[5]. Varices rupture if the wall tension exceeds a certain point. The probability that a varix will rupture and bleed increases with increasing size/diameter of the varix and with increasing variceal pressure, which is again proportionate to the HVPG[5].

The risk of rebleed decreases significantly with reductions in HVPG greater than 20% from baseline[6]. Patients whose HVPG decreases to <12 mmHg, or at least 20% from baseline levels, have a lower likelihood of developing recurrent variceal hemorrhage[7], and also have a lower risk of ascites, spontaneous bacterial peritonitis, and death.

Gastroesophageal variceal bleeding is responsible for 10 to 20 percent of all cases of bleeding from upper gastrointestinal tract[8]. Frequency of esophageal varices varies from 40% to 60% in patients with cirrhosis[9] and 9-36% of patients have what are identified as "high-risk" varices. Esophageal varices build up in patients with cirrhosis at an annual rate of 5-8%, but the varices are large enough to pose a risk of bleeding in only 1-2% of cases. Approximately 4-30% of patients with small varices will grow large varices each year and will therefore be at risk of bleeding[10].

An international normalized ratio (INR) score >1.5, a portal vein diameter of >13 mm, and thrombocytopenia have been found to be predictive of the possibility of varices being present in cirrhotics. If none, one, two, or all three of these conditions are present, then <10%, 20-50%, 40-60%, and >90% of the patients are likely to have varices, respectively[10]. The existence of one or more of these conditions represents an indication for endoscopy to search for varices and carry out primary prophylaxis against bleeding in cirrhotic patients.

Although gastric variceal bleeding occurs less often than esophageal varix bleeding, whenever it occurs it tends to be more severe and has a higher mortality than esophageal variceal bleeding[11]. In a local study, prevalence of gastric varices in patients with portal hypertension was 15 percent[12]. The management of gastric varices differs from that of esophageal varices in that gastric variceal bleeding are usually much more difficult to control, especially endoscopically[13].

Endoscopic therapy for variceal hemorrhage involves use of band ligation, sclerotherapy and tissue glue. Because gastric varices are located deeper in the submucosa than esophageal varices, sclerotherapy and ligation are usually ineffective in controlling acute bleeding from gastric varices and may be harmful. N-butyl-2-cyanoacrylate (tissue glue) has been proven to be effective for bleeding gastric varices[14].

Present study will assess the frequency of gastric varices in patients with portal hypertension based on endoscopic findings, which would help to sensitize the treating physicians regarding the load of patients with gastric varices. This study will also help to quantify the patients which require alternative approaches like use of tissue glue for control of variceal bleed. Quantifying patients with gastric varices will help in allocation of hospital budgets for tissue glue purchase, since it's the only endoscopic modality available for treatment of gastric varices at present.

MATERIAL AND METHODS

In our study 100 outdoor patients with previously diagnosed portal hypertension, presenting with hematemesis, melena or ascites were selected by non-probability consecutive sampling. Both male and female patients between 15 and 75 years of age and having portal hypertension, either cirrhotic or non-cirrhotic were included in the study. Patients having Shock,atlantoaxial subluxation,possible visceral perforation, severe respiratory disease, recent

myocardial infarction, unstable angina and cardiac arrhythmiaswere not included in the study. One hundred patients were selected, admitted, stabilized hemodynamically and kept empty stomach for at least four hours before endoscopy. Permission from hospital ethical committee was obtained (approval attached). All the patients before being subjected to endoscopy were given complete information about the purpose and nature of the study and their consent was obtained. The patients were sedated with intravenous midazolam and local anesthetic was applied at the back of throat. Patients were made to lie on their left side and endoscopes introduced in the mouth, findings thus obtained wererecorded. Hospital registration number, name, age, gender, address and phone number (optional) were noted.

Control of bias and confounding factors was done by strictly following the exclusion criteria. All the data had been analysed through Statistical Package for Social Sciences (version 14.0). Descriptive statistics were calculated to summarize the data. Mean and standard deviation (±SD) were calculated for the quantitative variables i.e. age.Frequency and percentages were calculated for qualitative variables i.e. gender, gastric varices and cirrhosis.

RESULTS

One hundred patients were recruited for study after careful scrutiny using above mentioned inclusion and exclusion criteria. Mean age was 52.19 years (SD ± 12.744) (table-I). There were 67% males whereas 33% were females. The overall frequency of gastric varices was 11%, whereas 89% had no gastric varices (table-II) (fig). Out of 11 patients positive for gastric varices, 7 were males, and 4 were females (table-III). Among the selected group 1% had non-cirrhotic portal hypertension, whereas 99% had cirrhotic portal hypertension.

Table-I: Mean age ± standard deviation of subjects.

	n	Minimum	Maximum	Mean	Std. Deviation
Age in years	100	15	75	52.19	12.744

Table-II: Frequency of gastric varices.

GASTRIC Varices	Frequency	Percent	Valid Percent	Camulative Percent
Positive	11	11.0	11.0	11.0
Negative	89	89.0	89.0	100.0
Total	100	100.0	100.0	

Table-III: Gender related frequency of gastric varices.

Gender	Varices		Total
	Positive	Negative	
Male	7	60	67
Female	4	29	33
Total	11	89	100

DISCUSSION

Portal hypertension is the rise in Porto-systemic pressure gradient in any portion of the portal venous system. Although portal hypertension can result from pre-hepatic abnormalities (e.g. portal or splenic vein thrombosis), post-hepatic abnormalities (e.g. Budd-Chiari syndrome) or intrahepatic non-cirrhotic causes (e.g. schistosomiasis, sinusoidal obstruction syndrome), cirrhosis is by far the most common etiology of portal hypertension.

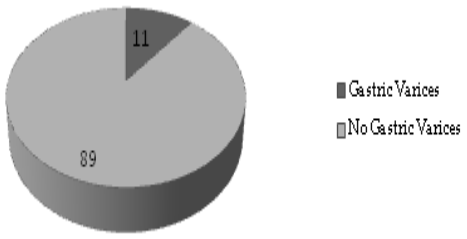

Figure: Frequency of gastric and no gastric varices.

An HVPG above 5 mmHg defines portal hypertension, however an HVPG of 10 mmHg or greater points towards clinically significant portal hypertension as this pressure gradient predicts clinical course in patients with cirrhosis including development of varices[15], clinical decompensation (i.e. development of ascites, variceal hemorrhage and encephalopathy)[16], decompensation or death after liver resection, and hepatocellular carcinoma[17].

The complications that result directly from portal hypertension are the development of varices and variceal hemorrhage. Esophageal varices are the most frequent types of varices in patients with portal hypertension. Gastric varices although uncommon, can result in significant mortality due to complicated control of acute hemorrhage. Compared to endoscopic injection sclerotherapy (EIS) or endoscopic variceal ligation (EVL), endoscopic variceal obturation with a tissue adhesive such as N-butyl-cyanoacrylate, or isobutyl-2- cyanoacrylate is more effective for acute fundic gastric variceal bleeding. The results include a better rate of control of initial hemorrhage as well as lower re-bleeding rate[18,19]. It is therefore important to identify the frequency of patients who have gastric varices due to portal hypertension, since these are the patients who require tissue glue instead of band ligation for prevention as well as control of acute variceal hemorrhage.

Examination of the results of study revealed that the overall frequency of gastric varices was 11%. Interpretation of the results show this much frequency of patients with portal hypertension presenting in outpatient departments have gastric varices.

Variceal hemorrhage is a frequent complication of portal hypertension and chief cause of disability and death in patients with liver cirrhosis. Advanced stage of liver failure, failure to control acute hemorrhage, early rebleeding and markedly elevated portal pressure is associated with poor prognosis[4]. Variceal hemorrhage is seen in 25 to 30% of patients with cirrhosis and is responsible for 80 to 90% of bleeding episodes in these patients. Combined treatment with vasoactive drugs, prophylactic antibiotics, and endoscopic techniques is the suggested standard of care for patients with acute variceal bleeding[20]. However treatment failure occurs in 10 to 15% of patients[21]. These patients need repeat endoscopic therapy and multiple transfusions. The 6-week mortality with each episode of variceal hemorrhage is about 15 to 20%, ranging from 0% among patients with child class A disease to approximately 30% among patients with Child class C disease[22,23].

Gastric varices are less common than esophageal varices and are present in 5%-33% of patients with portal hypertension with a reported incidence of bleeding of about 25% in 2 years, with a higher bleeding incidence for fundal varices[24].

CONCLUSION

A large number of patients with portal hypertension have gastric varices. It is recommended that endoscopy be carried out in all patients with identified portal hypertension.

CONFLICT OF INTEREST

This study has no conflict of interest to declare by any author.

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