The Professional Medical Journal
www.theprofesional.com
DOI: 10.17957/TPMJ/15.3016

ORIGINAL PROF-3016

DECOMPENSATED CIRRHOSIS;
PREVALENCE OF GASTRIC VARICES

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Article received on:
09/07/2015
Accepted for publication:
17/08/2015
Received after proof reading:
13/11/2015

ABSTRACT…: Cirrhosis with subsequent portal hypertension is a major health problem worldwide. Among various etiologies, HCV is the leading cause of chronic hepatocellular injury. Cirrhosis being the commonest cause of portal hypertension results in a spectrum of complications. Approximately 5-15% of cirrhotic develop varices. Gastric varices although present less frequently as compared to esophageal varices but are associated with greater mortality and morbidity. **Objectives:** To determine prevalence of Gastric varices in patients with decompensated cirrhosis. **Study Design:** Cross sectional study. **Place & Duration:** Carried out at two centers, Pakistan Ordinance Factories Hospital Wah Cantt and Shalimar Hospital Lahore from Jan 2014 to Jan 2015. **Subjects**: A total of 421subjects of decompensate cirrhosis were included in the study. **Methods:** Patients with decompensate cirrhosis of any etiology who were visiting the two hospitals as inpatient or outpatient were included in the study. Upper GI endoscopy was done in all these subjects for determination of gastric varices. **Results:** Among 421 patients of decompensate cirrhosis, frequency of gastric varices was 10.9%, GOV1 as the most common variant. Gastric varices contributed to 5.4% of all variceal bleeds. **Conclusion:** Gastric varices are found in significant number of patients of decompensate cirrhosis. Since gastric varices are associated with increased mortality and poor outcomes, these should be carefully looked during upper GI endoscopy. Successful management of gastric variceal hemorrhage necessitates availability of expertise and newer diagnostic modalities.

Key words: Cirrhosis, Portal hypertension, Gastric varices, Variceal hemorrhage.

Article Citation: Iqbal S, Yousaf MH, Yousuf MI, Wasim-Uddin. Decompensated cirrhosis; prevalence of gastric varices. Professional Med J 2015;22(11):1397-1402. **DOI:** 10.17957/TPMJ/15.3016

INTRODUCTION

Chronic liver disease and its complications are a major health problem worldwide and significant cause of morbidity and mortality among patients. It is the commonest cause of portal hypertension and more than 60% of patients with cirrhosis eventually develop portal hypertension, which in turn is responsible for the development of as cites and esophagogastric varices. Approximately 5-15% of patients with cirrhosis per year develop varices.[1]

Varices can be either esophageal or gastric where esophageal ones are much more frequent. Gastric varices are present in 20% of patients with portal hypertension of all etiologies.[2,3] Gastric varices occur in 5-33% of patients with portal hypertension due to underlying cirrhosis[4], whereas isolated gastric varicesare present in 5% of cirrhotics.[5] They represent 10-15% of all variceal bleeds.[6] They may develop either due to generalized or segmental portal hypertension as in splenic vein thrombosis.[2] Gastric varices are classified based on their location in stomach and relation to esophageal varices, described by Sarin and Kumar.[2] GOV (gastro-esophageal varices) when they extend from esophagus to stomach and IGV (isolated gastric varices) in the absence of esophageal varices. GOV are subdivided into GOV1, extending along lesser curvature of stomach and GOV2, extending along greater curvature of stomach. In the same way IGV are subdivided into IGV1 (located in gastric fundus) and IGV2 (located in antrum, corpus, pylorus or upper duodenum).[2,3,7](figure-1)

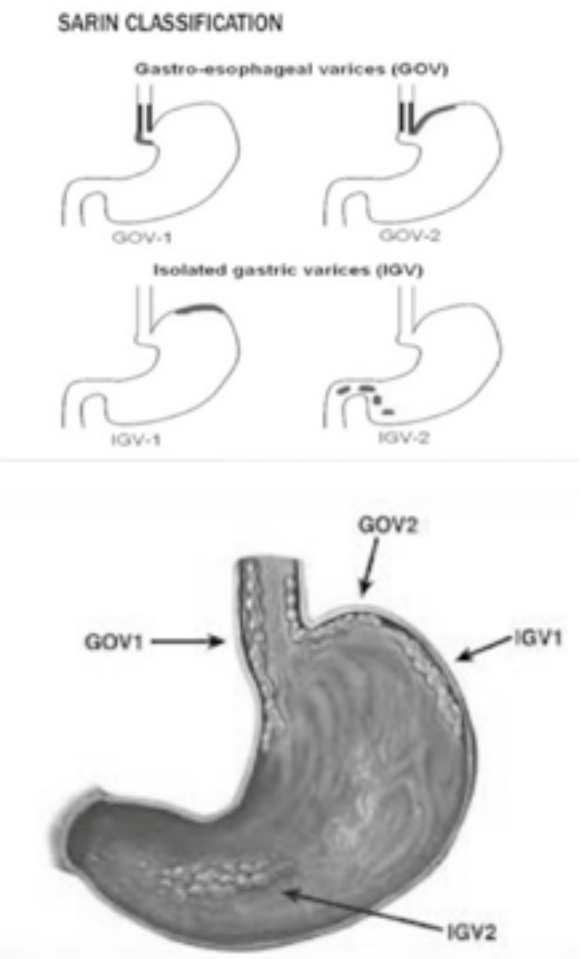

Figure-1. Gastric Varices sub-types; Sarin Classification

Gastric varices although bleed less frequently as compared to esophageal varices but are associated with more severe bleeding and morbidity.[2,8] Bleeding gastric varices can be technically difficult to treat. They rebleed frequently despite initially successful endoscopic therapy. The reason for this disparity has been explained by our knowledge of portal system which revealed that gastric varices have a large larger vascular bed and multiple communications making conventional therapies less successful.

Multiple studies have now concluded that tissue adhesives like cyanoacrylate are now the standard of treatment in acute gastric variceal bleed wherever expertise are available. Cyanoacrylate has been shown to achieve good homeostasis with fewer complications and decreased frequency of rebleeds as compared to variceal band ligation. As far as prevention of recurrent bleeding is concerned, it can be achieved with use of β-blockers in a similar fashion as those for esophageal varices.[9]

Complications of cirrhosis are basically the same irrespective of etiology. Etiology of underlying cirrhosis worldwide as well as in Pakistan is predominantly HCV, HBV or co-infection, majority being HCV related.[1,10]

Since gastric varices are associated with higher morbidity and mortality, these should be carefully looked for during upper GI endoscopy and their presence documented. A similar study conducted at Agha Khan University Hospital provided good statistical evidence about gastric varices[4] and this study conducted in another set of population will further strengthen the existing data. Also it will put an emphasis on necessity and availability of newer therapeutic measures

METHODOLOGY

A total of 421patients with evidence of decompensate cirrhosis presenting to inpatient, Outpatient and Endoscopy department of the two hospitals were subjected to further evaluation. Inclusion criteria were cirrhotic patients with any one of the following; h/o variceal bleed, encephalopathy, splenomegaly, as cites, thrombocytopenia or portal vein diameter of ≥13mm on ultrasonography. Patients with other major organ failure and co-existent HCC were excluded from the study.

Thorough clinical examination was done in all patients to look for ascites, splenomegaly and hepatic encephalopathy. Baseline investigations; blood complete count, liver function tests, prothrombin time, serum albumin concentration were carried out. Abdominal ultrasound was done to ascertain presence of portal hypertension in terms of as cites, splenomegaly and portal vein dilatation and to look for any underlying GI malignancy.

Hepatitis B/C serology was done in those previously not evaluated for viral etiology. Patients were categorized into HBV related, HCV related

and non B/C related cirrhosis on the basis of etiology. Patients were also categorized individually into respective Child Class. Upper GI endoscopy was done to look for gastric varices and their subtype. Co-existent portal gastropathy and esophageal varices were also noted.

All data was entered and analyzed using SPSS version 16.0. For quantitative variable of age mean± standard deviation has been presented. For categorical variables like gender, etiology of cirrhosis, Child class, presence of gastric varices, co-existent esophageal varices/portal gastropathy, Sarin class, H/O of variceal bleed frequencies (percentages) are presented.

RESULTS

Four hundred and twenty one patients with decompensated cirrhosis from the two centers were enrolled in the study (218 males and 203 females). Mean age was 54 ± 11yrs (table-I). Chronic HCV was the main etiological factor. On further evaluation and screening 76% patients had esophageal varices and 76.4% had portal gastropathy. Most of the times both were seen in combination. Gastric varices were found in 10.9% of patients out of which GOV1, GOV2, IGV1 and IGV2 comprised 54.3%, 17.3%, 23.9% and 4.3% respectively (table-II and III). Of 52% of total bleeds 5.4% were attributable to gastric varices. If considered independently 26% of gastric varices ultimately bled necessitating use of tissue adhesives and 49.8% of esophageal varices presented with upper GI bleed requiring band ligation. Apparently seems smaller percentage but gastric varices were associated with higher percentage of rebleeds and mortality recorded during hospital stay and within 1 week of discharge after initial bleeding episode (table-IV). Another difference that was noted is that gastric varices were found more in males (70%) as compared to females (30%).

Minimum	18yrs
Maximum	90yrs
Mean	54yrs
Std deviation	11

Table-I. Age Distribution

Characteristics	Percentage (n=421)
Males	218 (51.7%)
Females	203 (48.2%)
Etiology	
HCV	381 (90.4%)
HBV	10 (2.3%)
Non B/C	30 (7.1%)
Esophageal varices	320 (76%)
Portal gastropathy	322 (76.4%)
Gastric varices	46 (10.9%)
Bleeding history	222 (52.7%)
Child Class	
A	101 (24%)
B	219 (52%)
C	10 (24%)

Table-II. Baseline Characteristics

Gastric Varix subtype	Percentage (n=46)
GOV1	25 (54.3%)
GOV2	8 (17.3%)
IGV1	11 (23.9%)
IGV2	2 (4.3%)

Table-III. Subtypes of Gastric Varices

	Esophageal Varices (n=159)	Gastric Varices (n=12)
Rebleed	11 (7%)	5 (41%)
Mortality	2 (1.2%)	1 (8.3%)

Table-IV. Bleeding Complications

DISCUSSION

Cirrhosis is the ultimate outcome of chronic hepatocellular injury. It is one of the leading cause of morbidity and mortality worldwide. A number of etiologies have been implicated, the commonest being chronic viral hepatitis where chronic HCV takes the lead. Contrary to that chronic ethanol ingestion is sharing a larger percentage in western world. This study showed frequency of HCV and HBV as 90.4% and 2.3% respectively whereas rest of causes accounting for another 7.1% of cases.

Progressive decomposition of Porto-hepatic sys-

tem results in a spectrum of complications serving as determinants of prognosis. Variceal hemorrhage has been recognized since long as one of the life threatening complications and frequent cause of hospitalization. High portal pressure determined in terms of HVPG has direct correlation with variceal formation. However certain relatively noninvasive methods as has been used in this study have reasonable sensitivity with respect to degree of decompensation.[11]

Esophageal varices occur much more frequently as compared to gastric varices, however gastric varices are associated with more serious bleeds and greater mortality.[12] The risk of bleeding is low in patients with small varices (5% per year) whereas large varices have an annual risk of bleeding of 15%.[13] The life time risk of one bleeding episode from varices is 33% with 70% having rebleed.[14]

Frequency of gastric varices in my study turned out to be 10.9% among total of 421 patients with decompensate cirrhosis, which is comparable to international studies showing a frequency of 5-33% in cases with cirrhosis.[2,3,4]

Gender distribution among patients with gastric varices was that, out of total of 46 patients, 32 were males and 14 were females. No obvious explanation to this male predominance could be sorted out but the same has been observed elsewhere also.

Patients who develop one or more signs of decompensated cirrhosis ultimately also develop co-existent esophageal varices and portal gastropathy as well. Esophageal varices were found in 76%. Portal gastropathy was seen in 76.4% of patients. 52.7% of patients had one or more episodes of bleeding in past.

Gastric varices are responsible for 10-15%[4] of all variceal bleeds and rebleeding rate of 34% to 89%.[15] Gasricvariceal hemorrhage has been associated with mortality rate of as high as 45%.[16] When compared esophageal varices are associated with overall mortality of 30%.[59] In this study 52.7% of patients with esophageal andco-existent gastric varices bled and percentage of bleeds attributable only to gastric variceal hemorrhage was 5.4%.

Endoscopic evaluation reveals that gastric varices tend to be larger, more tortuous combined with their anatomic location makes their management challenging. Sarin and Ryan described various types of gastric varices. GOV1 is the commonest variant accounting for 74% followed by GOV2, IGV1 and IGV2 that comprise up to 16%, 8% and 2% respectively.[2] In this study the most frequent variant was GOV1 making up 54.3%% followed by IGV1 of 23.9%.

Understanding portal hemodynamic patterns is of utmost importance in order to design different therapeutic measures for management of gastric varices. Hemodynamic in gastric varices are quite different from those of esophageal varices. In past conventional method of portal vein catheterization was used to view portal system of veins and feeding channels of gastric varices. With the advent of new radiological techniques Multi-detector Computer Tomography Portal Venography (MCTPV) has shown promise with good outcomes.[17]

Afferent venous drainage of gastric varices mainly comes from Left Gastric vein, Short Gastric vein or Posterior Gastric veins and drain into inferior vena cava through Gastro-renal shunt or inferior phrenic vein. Similarly drainage into superior vena cava is via azygous vein. Some of these shunts are not present under normal conditions and arise only when portal pressure rises.[7,17]

Knowledge of this portal system anatomy has now revealed why variceal ligation proved unsuccessful in case of gastric varices due to large area of distribution and multiple communications. Similarly increased incidence of rebleeds and ulcer formation has been noted with above measure.[17]

Taking into account this diversity of gastric varices and large vascular bed, use of tissue adhesives and Cyanoacrylate has been very successful with fewer complications, good homeostasis

and decreased frequency of rebleeds. Similarly Balloon-occluded retrograde obliteration has been tried in various clinical trials.[18-22]

Modified Child Pugh Classification system is a good prognostic indicator that directly or indirectly commensurate with degree of decompensation.[23] Majority of patients in this study belonged to Child Class B followed by Child Class A and C respectively. The smaller percentage of Child Class C patients may be attributed to overall decreased 1year survival rate.

CONCLUSION

Gastric varices are present in significant number of patients with decompensate cirrhosis. Since gastric varices are associated with increased mortality and poor outcomes, these should be carefully looked during upper GI endoscopy. Successful management of gastric variceal hemorrhage necessitates availability of expertise and newer diagnostic modalities.

This study did not reveal any particular risk factor associated with increased frequency of gastric varices however male predominance has been noted.

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"Loyalty is so rare now a days. If someone remains loyal to you, appreciate them, don't take them for granted."

Unknown

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