Original Research Paper

Volume - 7 | Issue - 7 | July - 2017 | ISSN - 2249-555X | IF : 4.894 | IC Value : 79.96

Gastroenterology

A STUDY ON GASTRIC AND GASTROESOPHAGEAL VARICES IN A TERTIARY REFERRAL CENTRE

Dr.B.Sivasubramaniam	Department of Medical Gastroenterology, Govt Mohan Kumaramangalam Medical college, Salem 636030, Tamilnadu, India
Dr.R.Karthikeyan	Department of Medical Gastroenterology, Govt Mohan Kumaramangalam Medical college, Salem 636030, Tamilnadu, India - Corresponding author
Dr.E.Kandasamy alias Kumar	Department of Medical Gastroenterology, Govt Mohan Kumaramangalam Medical college, Salem 636030, Tamilnadu, India

ABSTRACT **Aim:** i.To assess the prevalence of gastroesophageal varices in patients with portal hypertension in a tertiary referral centre ii.Characteristics of the gastric varices iii.Natural history of gastric varices in portal hypertension.

Materials and methods: It is a prospective study conducted in the department of Medical gastroenterology, Stanley Medical College, Chennai between April 2010 and October 2011 where consecutive patients with the diagnosis of cirrhosis, NCPF, EHPVO and BCS undergoing oesophagogastroduodenoscopy (OGD) scopy as a routine evaluation and for upper gastrointestinal bleeding were included. Patients with cirrhosis and NCPH are subjected to endoscopy in which if there are gastric varices, they are included in this study.

Results: 81 patients had gastroesophageal varices and those were taken for analysis. Mean age of the patient is 41.23 ± 15.4 years. The aetiology for the gastroesophageal varices was: Cirrhosis in 49.4%. Extrahepatic portal vein obstruction in 27.2%. Non cirrhotic portal fibrosis in 23.5%. Among the 81 patients, 75 (92.6%) patients had history of upper gastrointestinal bleeding and 6 (7.4%) patients had no history of bleeding. Subsequent bleed was observed in 58% (47) . 4 (4.9%) patients had bleed in the form of melena. All other patients (77) developed hematemesis during the index bleed. Among the bleeders blood transfusion required in 87.7 %. The mean volume of units of blood given to the bleeding patients was 2.28L. The number of patients underwent surgery for the bleeding episodes were 19 (23.5%).Rectal varix was seen in only one patient. GOV 1 was the commonest type which was seen in 40% of patients. Index bleed was present in 92.6% patients and subsequent bleed was seen in 58% of patients

Conclusion: Prevalence of gastroesophageal varices was low but within the range . GOV1 is relatively have a benign course and requires treatment only by sclerotherapy .Gastric varices have the potential to cause severe upper GI bleeding, its recognition is very important to manage appropriately.

KEYWORDS : Gastric varices, Gastro esophageal varices

Back ground: Portal hypertension can occur due to many reasons. One of the commonest causes for portal hypertension is cirrhosis. Other important causes are non cirrhotic portal fibrosis (NCPF) and extra hepatic portal vein obstruction (EHPVO). Portal pressure increases in cirrhosis initially as a result of an increased resistance to portal flow. This mostly results from fibrous tissue and regenerative nodules formation within the hepatic parenchyma which leads to distortion of the architecture of the liver.1Along with this structural resistance to blood flow, there is an intra-hepatic constriction of the vessels that accounts for twenty to thirty percent of the increase in resistance within the liver. This happens because there is decreased synthesis of nitric oxide endogenously. 2-3 The obstruction to the portal flow is at perisinusoidal level in NCPF but in EHPVO the obstruction is extra hepatic, which is commonly due to the formation of thrombosis in the portal vein.Porto-systemic collaterals are formed due to the development of portal hypertension. Although the collaterals are formed to relieve the portal pressure portal hypertension persists due to two causes: (1) an increase in portal venous inflow due to splanchnic arteriolar vasodilatation along with the formation of collaterals4 and (2) inadequate decompression of the portal venous system through the collaterals since they have a higher resistance than the normal liver.5 Therefore, an increased portal pressure gradient results from both an increase in portal blood inflow and increase in resistance to portal flow. Gastroesophageal varices are commonly seen in up to 50% of patients with cirrhosis.6 Gastric varices are seen in 20-25% of patients with portal hypertension. If the patient is not having varices it will develop at the rate of 8% per annum 8-9 and one who have small varices will develop larger varices at 8% per year.. In few subsets of patients such as in primary biliary cirrhosis and hepatitis C with bridging fibrosis, even in the absence of overt cirrhosis they have propensity to develop varices in up to 16 % of the patients.6-7 Irrespective of the aetiology, the important and dreadful complication of varices is upper gastrointestinal bleeding. Prevalence of gastric varices is low when compared to esophageal varices. They are present in 6%-35% of patients with portal hypertension. The incidence of bleeding is about twenty-five percent in 2 years and highest bleeding rate is for fundal varices. Risk factors for gastric variceal haemorrhage include fundal varices size (large varices defined as >10 mm, medium -5-10 mm and small >5 mm), Child-Turcotte-Pugh score, particularly Child C status and endoscopic presence of variceal red spots (defined as localized reddish mucosal area or spots on the mucosal surface of a varix). Gastric varices are classified into four types. The relationship of gastric varices with that of esophageal varices and the position in the stomach decides the gastroesophageal varices classification. Gastroesophageal varices (GOV) are classified into 2 types. Type 1 gastroesophageal varices are called as GOV1 which runs along the lesser curvature of the stomach and this most frequently seen. Since they are similar to esophageal varices, the management is same to that of esophageal varices. If the varices extend along the fundus, it is called as Type 2 gastroesophageal varices and tends to be longer and more tortuous.. In Indian study by Sarin et al, the incidence of gastric varices is just 4% in cirrhotics patient who has not bled. Others have shown that 25% of cirrhotics had gastric varices at screening endoscopy with 18% of patients having both gastric and esophageal varices.10 Gastric varices are also more common in NCPH and EHPVO which is present in 25% and 33% of patients respectively.11 . The risk of bleeding with gastric varices is half that of esophageal varices. The transfusion requirement and mortality are high once the bleeding has occurred particularly for isolated gastric varices (IGV). Large gastric varices patients have a lower portal pressure compared to esophageal varices, which is due to the development of gastrorenal portosysytemic shunts, or large size of the varices resulting in increased wall tension.[12.]

Aim:To assess the prevalence of gastroesophageal varices in patients with portal hypertension in a tertiary referral centre 2.Characteristics of the gastric varices and 3.Natural history of gastric varices in portal hypertension. Materials and methods: It is a prospective study conducted department of Medical gastroenterology Stanley Medical College,Chennai between April 2010 and October 2011 where consecutive patients with the diagnosis of cirrhosis, NCPF, EHPVO and BCS undergoing oesophagogastroduodenoscopy (OGD) scopy as a routine evaluation and for upper gastrointestinal bleeding were included. Patients with cirrhosis and NCPH are subjected to endoscopy in which if there are gastric varices, they are included in this study.Among the 1083 patients who underwent OGD scopy, 81 patients were found to have gastric varices and those were included in

the study. A non bleeder was defined as any patient without a history of hematemesis or melena.A written consent was obtained from all the patients. Institute ethical committee has approved the study. All patients with gastric varices were included in the study. All the patients had baseline investigations.A detailed history about the upper gastrointestinal bleeding was obtained from all the patients such as age at the time of diagnosis, duration of illness, cause of portal hypertension, index bleed , subsequent bleed with dates, volume of blood vomiting, presence of liver cell failure at the time of bleed, use of Sengstaken Blackmore tube to arrest bleeding, use of vasopressors drugs, application of sclerotherapy, variceal ligation and glue, surgery details in case of failure of endotherapy and outcome after the bleed. A detailed clinical examination was also performed in all patients which includes pallor, jaundice, pedal edema, fever, asterixis, clubbing, cyanosis , presence of liver cell failure. Results:A total of 1083 patients underwent upper gastro intestinal endoscopy during the study period. 81 patients had gastroesophageal varices and those were taken for analysis. Mean age of the patient is 41.23 ± 15.4 years. The aetiology for the gastroesophageal varices was: Cirrhosis in 49.4%. Extrahepatic portal vein obstruction in 27.2%. Non cirrhotic portal fibrosis in 23.5%. Among the 81 patients, (Table-1) 75 (92.6%) patients had history of upper gastrointestinal bleeding and 6 (7.4%) patients had no history of bleeding. Subsequent bleed was observed in 58% (47) of the patients who has bled initially. 4 (4.9%) patients had bleed in the form of melena. All other patients (77) developed hematemesis during the index bleed.Among the bleeders (Table-3) blood transfusion required in 87.7 % of the patients. The mean volume of units of blood given to the bleeding patients was 2.28L. The number of patients underwent surgery for the bleeding episodes were 19 (23.5%).Rectal varix was seen in only one patient. GOV 1 was the commonest type which was seen in 40% of patients. Index bleed was present in 92.6% patients and subsequent bleed was seen in 58% ofpatients

Table-1. Demographic characteristics of the study population

Characteristics		n (%)
Total No of cases		81
Male		50(61.8%)
Female		31(38.2%)
Mean age (Years)		41.23± 15.4
Literacy	Yes	68 (84%)
status	No	13 (16%)
Per capita income	≤ 5000	74(91.3%)
	≥5000	7(8.7%)
Aetiology of portal hypertension		
Cirrhosis		40 (49.4%)
EHPVO		22 (27.2%)
NCPF		19 (23.5%)
Bleeder		75(92.6%)
Non bleeder		6(7.4%)
Outcome	Alive	74(91.4%
	Dead	7(8.6%)
HBsAg positive		4(4.9%)
Anti-HCV positive		3(3.75%)
Bleeder and nonbleeders		

Table-2 Index and subsequent bleed

Characteristics	Index bleed (P value)	Subse bleed (P value)
Alcohol	0.423	0.017
Age at diagnosis	0.235	0.002
Quantity of bleed	0.001	0.003
Cause of PHT	0.193	0.005
Duration of illness	0.452	0.001
Follow up	0.023	0.001
No.of TX	0.001	0.001
SBT	0.266	0.006
EVL	0.046	0.001
EST	0.080	0.001
Surgery	0.159	0.008

Table-3. Bleed details

GOV1	40(49.4%)
GOV2	14(17.3%)
IGV1	14(17.3%)
IGV AND ESOPHAGEAL VARICES	12(14.8%)
GOV1&2	1(1.2%)
Index bleed cases	75(92.6%)
Subsequent bleed cases	47(58%)
Transfusions requirement	71(87.7%)
Mean volume of blood given (L)	2.28
Rectal varices	1(1.2%)

Characteristics	Bleeder n (%) n= 75	Non bleeder n = 6	P value*
Age (mean)	40.8	44.8	0.306
Gender			
Male	48	1	0.197
Female	27	5	
Type of gastric varices			
GOV1	37	3	0.701
GOV2	13	1	0.636
IGV1	12	2	0.782
IGV &Esophageal Varices	12	Nil	---
GOV1&2	1	Nil	---

Discussion: The type of varices commonly seen in cirrhosis is GOV1, in EHPVO IGV1 is more frequently seen and GOV2 is mostly in NCPF.The patients with GOV2 had more number of bleed episodes and severe bleed when compared to other types .There is no significant difference between the bleeders and non-bleeders with regards to age (p=0306), gender (p=0.197), type of gastric varices- OGV1 (p=0.701), OGV2 (p=0.636), IGV1 (p=0.782), aetiology of portal hypertension-Cirrhosis (p=0.193), NCPF (p= 0.106) and EHPVO (p= 0.184). The index bleed parameters such as quantity of bleed (p=0.001), follow up in months (p=0.023), no of transfusions (p=0.001), EVL(0.046) had significant p value in index bleed group. Variables such as alcohol consumption (p=0.017), age at diagnosis (p=0.001), quantity of bleed (P=0.003), cause of PHT(P=0.005), duration of illness (p=0.001), follow up in months (p=0.001), No. of transfusions (p=0.001), EVL (p=0.001), EST(p=0.001) and surgery (p=0.008) had significant p value in subsequent bleed group. Gastric varices were approximately five times more common in bleeders than in non bleeders. This indicates that the gastric varices develop at a more advanced stage of portal hypertension. The most common type is GOV1, constitutes 75% of all primary varices. In our study we have encountered of GOV1 varices in 49.4% patients. Other workers have also found GOV1 to be the most common type of gastric varix .12. GOV2 constituted in 14(17.3%) patients. IGV1 was also observed in 14 (17.3%) patients. Watanabe et al found them in 3% of their patients. IGV1 and esophageal varices were simultaneously seen in 12 patients. These varices develop because of dilatation of short gastric and posterior gastric varices in patients with EHPVO or because of direct anastomotic veins between the gastric and retroperitoneal veins in patients with cirrhosis. Only one patient had GO1 and GOV2 in combination. In our study only one patient had colonic varices in the form of rectal varices.

Summary: In the present study, The prevalence of gastric varices is 7.5% among the various aetiologies. The commonest type of gastroesophageal varices is GOV1 which is followed by GOV2 and IGV1 which is well correlated with various studies. Index bleed is seen in 92.6% of patients and subsequent bleed is seen in 58% of the patients. Among the aetiology, cirrhosis is the most common cause and EHPVO is the next common cause for gastroesophageal varices. There is no significant difference between the bleeders and nonbleeders with regards to age, gender, type of gastric varices- GOV1, GOV2, IGV1, aetiology of portal hypertension (Cirrhosis, NCPF and EHPVO), and complication such as hepatic encephalopathy. Nineteen patients (19/81 - IGV1-14, OGV2-5) have undergone surgical treatment to arrest the recurrent bleeding. The index bleed parameters such as quantity of bleed, follow up in months, no of transfusions, EST and EVL had significant p value in index bleed group.

Conclusion: The results of our study confirm that the prevalence of gastroesophageal varices was low but within the range when compared with various studies.The type of the varices in Our study tallies with the international classification and the common type is GOV1 as denoted by many studies.GOV1 is relatively have a benign course and requires treatment only in the form of gastric variceal sclerotherapy if they bleed.For most GOV2 varices, endoscopic variceal obliteration therapy with N-Butyl 2- Cyanoacrylate is quite useful in arresting the bleeding and achieving the variceal obliteration.Although endoscopic therapy was effective in treating some patients with IGV1 varices, surgery was required in significant no. of patients to prevent re bleeding.IGV2 cases were less in Our study and it might require long

term follow up to identify such patients.The subsequent bleed parameters - alcohol consumption, age at diagnosis, quantity of bleed, cause of PHT, duration of illness, follow up in months, no. of transfusions, SBT, EVL, EST and surgery had significant p value in subsequent bleed group. In our study mortality is less (8.6%), when compared to other studies. Because the gastric varices have the potential to cause severe upper GI bleeding, its recognition is very important to manage the cases appropriately.

References:

1. Bhathal PS, Grossman HJ. Reduction of the increased portal vascular resistance of the isolated perfused cirrhotic rat liver by vasodilators. J Hepatol 1985;1:325-337.
2. Gupta TK, Chung MK, Toruner M, Groszmann RJ. Endothelial dysfunction in the intrahepatic microcirculation of the cirrhotic rat. Hepatol 1998;28:926- 931
3. Wiest R, Groszmann RJ. Nitric oxide and portal hypertension: its role in the regulation of intrahepatic and splanchnic vascular resistance. Semin Liver Dis 2000; 19:411-426.
4. Sikuler E, Kravetz D, Groszmann RJ. Evolution of portal hypertension and mechanisms involved in its maintenance in a rat model. Am J Physiol 1985;248(6 Pt 1):G618-G625.
5. Sikuler E, Groszmann RJ. Interaction of flow and resistance in maintenance of portal hypertension in a rat model. Am J Physiol 1986;250(2 Pt 1):G205-G212.
6. Pagliaro L, D'Amico G, Pasta L, Politi F, Vizzini G, Traina M, et al. Portal hypertension in cirrhosis: Natural history. In: Bosch J, Groszmann RJ. Portal Hypertension. Pathophysiology and Treatment. Oxford, UK: Blackwell Scientific, 1994: 72-92
7. Navasa M, Pares A, Bruguera M, Caballeria J, Bosch J, Rodes J. Portal hypertension in primary biliary cirrhosis. Relationship with histological features. J Hepatol 1987;5:292-298.
8. Sanyal AJ, Fontana RJ, DiBisceglie AM, Everhart JE, Doherty MC, Ever- son GT, et al. and the HALT-C trial group. The prevalence and risk factors associated with esophageal varices in subjects with hepatitis C and advanced fibrosis. Gastrointest Endosc 2006;64:855-864.
9. Groszmann RJ, Garcia-Tsao G, Bosch J, Grace ND, Burroughs AK, Planas R, et al. for the Portal Hypertension Collaborative Group. Betablockers to prevent gastroesophageal varices in patients with cirrhosis. N Engl J Med 2005;353:2254-2261.
10. Sarin SK, Lahoti D, Saxena SP, Murthy NS, Makwana UK. Prevalence, classification and natural history of gastric varices: a long-term follow-up study in 568 portal hypertension patients. Hepatology 1992; 16:1343-1349.
11. Sarin SK, kumar A. Gastric varices: profile, classification and management. Am.J gastroenterology 1989 : 1244-1249
12. Watanabe K, Kimura K, Matsutani S, Ohto M, Okuda K. Portal hemodynamics in patients with gastric varices. A study in 230 patients with esophageal and/or gastric varices using portal veincatheterization.Gastroenterology1988;95: 434-440