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Original Article

Development of Fundal Varices in Cirrhotic Patients after Eradication of Esophageal Varices

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Abstract

Background: To analyze the development of fundal varices in cirrhotic patients after eradication of esophageal varices

Methods: In this observational study 150 patients of liver cirrhosis, with the history of upper gastrointestinal tract bleed and esophageal varices of grade F2 and F3 but absence of fundal varices, were included. Patients who had previous history of banding were excluded.

Results: Eighty one (54%) patients were male. Mean age of the patients was 49.34±11.45 years. Twenty two (14.66%) developed fundal varices of which 2(1.33%) patients developed fundal varices at 2 months, 8(5.33%) patients developed fundal varices at 4 months following banding, and 12(8%) patients developed fundal varices at 6 months following banding of esophageal varices. Fourteen (63.63%) patients had GOV2 while 8(36.36%) had IGV1. Conclusion: New fundal varices develop with increasing frequency in patients treated with esophageal variceal band ligation, and the incidence has a time-dependent relationship.

Key Words: Fundal Varices,Cirrhosis, Esophageal Varices

Introduction

Liver cirrhosis and liver related diseases are one of the frequent causes of hospitalization and a major burden on health system. Gastric varices are less prevalent than esophageal varices with a higher bleeding incidence for fundal varices. Endoscopic variceal band ligation is the recommended form of endoscopic therapy for esophageal variceal bleeding. New incidence of fundal varices is found in 16% of patients after eradication of esophageal varices at follow up of 6 months. Liver cirrhosis is the twelfth leading cause of death in USA. Liver cirrhosis and liver related diseases are one of the most frequent cause of hospitalization in Pakistan and a major burden on health system because of its grave complications and expenses.[1] The most common cause of portal hypertension is liver cirrhosis which leads to development of gastroesophageal varices with or without bleeding, ascites, hepatorenal syndrome, and hepatic encephalopathy.[2]

Gastroesophageal varices occur in 50% of cirrhotic patients at a rate of 10% per year. The clinical course of chronic liver disease is complicated by variceal hemorrhage in 30% of cases. With each episode of variceal bleeding the mortality is 20 - 30%. Around 70% of survivors have recurrent bleeding after their first variceal hemorrhage.[3]

Gastric varices are less prevalent than esophageal varices and are present in 25% of patients with portal hypertension with a higher bleeding incidence for fundal varices.[4] Secondary gastric varices have a significant association with Child-Pugh class, presenting grade, increasing number of ligation session and prior existence of gastric varices. [5] The commonly used classification system introduced by Sarin comprises of four types of gastric varices based on gastric location and relationship with esophageal varices. Gastro-esophageal varices (GOV) either are an extension of esophageal varices for 2 to 5 cm along the lesser curve of the stomach (GOV1) or extend along the greater curve into the fundus (GOV2). [6] Isolated gastric varices (IGV) are located either in the fundus (IGV1) or in other parts of the stomach (IGV2). GOV1 account for 74% of all GV, but the incidence of bleeding is highest with fundal varices (IGV1 and GOV2).[4-8]

Endoscopic therapy is recommended in any patient who presents with upper GI bleeding. Endoscopic variceal band ligation (EVBL) is the recommended form of endoscopic therapy for acute esophageal variceal bleeding, although sclerotherapy may be used in the acute setting if ligation is technically difficult. Endoscopic therapy with tissue adhesive (e.g. N-butyl-cyanoacrylate) is recommended for acute bleeding from isolated gastric varices (IGV) and those gastro-esophageal varices, type 2 (GOV2), that extend beyond the cardia. EVBL or tissue adhesive can be used in bleeding from gastro esophageal varices type 1 (GOV1).[8,9]

One local study suggests that new incidence of fundal varices is found in 16% of patients after eradication of esophageal varices at follow up of 6 months while presence of fundal varices at presentation before eradication was 7.4%.[5] Another local study suggested the presence of fundal varices to be in 3.1% patients at presentation for upper GI endoscopy but no follow-up after eradication of esophageal varices was done.[8]

Patients and Methods

This observational study was conducted at Department of Medicine, Aziz Bhatti Shaheed Teaching Hospital Gujrat from 15-06-15 to 15-03-17 . Sample size was calculated to be 150 patients using 95% confidence level with 5% margin of error with an expected percentage of new fundal varices in 16% of patients after band ligation.[5] Non-probability consecutive sampling method was used and patients who had liver cirrhosis with the history of upper GI bleed and esophageal varices of grade F2 and F3 but absence of fundal varices on endoscopy were included in study. Patients who refused to consent and who had previous history of banding were excluded from study. Severity of liver disease was noted and was graded using Child Pugh score. Patients with score of 5-6 were defined as Child class A, 7-9 as Child Class B and 10-15 as Child Class C. Cirrhosis was defined as moderate to severely coarse echotexture of liver on abdominal ultrasound. Spleen size, presence of ascities and liver span were also noted. The dilated veins above the lower esopheageal sphincter found during endoscopy were considered as esophageal varices. Gastric varices were classified according to classification described by Sarin et al.[6] Out of gastric varices gastroesophageal varices type 2 (GOV2) and isolated gastric varices type 1 (IGV1) were considered as fundal varices as they are located in fundus of stomach.After initial resuscitation, the stabilized patients were admitted in the ward. A full history and examination was done and the patients were prepared for an elective endoscopy list. All baseline investigatioons were done and noted and Child Pugh Score was calculated. Consent was taken, upper GI endoscopy and band ligation was done by the consultant gastroenterologist. Performa were filled by concerned doctor, and the patients were examined at regular interval of two months during study duration for a period of 6 months. Those patients who developed fundal varices either at second or fourth month were not further followed and were included in our results. The presence or absence of fundal varices and the minimum time to develop fundal varices were recorded. All data were entered into a predesigned performa.

Results

Out of 150 patients, 81 patients (54%) were male and remaining 69 patients (46%) were female. Mean age of the patients was 49.34±11.45 years. Majority patients (70%) were 41-60 years of age (Table 1) . Only one patient (0.6%) was between 81-100 years of age. 55 patients were Child Class A, 67 Child Class B and 28 were Child Class C. Mean hemoglobin was 7.62 ± 3.12 g/dl, mean platelet count was 79 ± 69 x 10^9/l, mean albumin was 3.2 ± 2.1 g/dl and mean bilirubin was 1.78±1.21 mg/dl. Mean liver size was 12.13 ± 4.34 cm, mean spleen size 11.67 ± 3.11 cm and ascities and esophageal varices were present in all patients. F3 esophageal varices were present in 52.67% patients but none of patients had fundal varices (Table 2).

Table 1: Distribution of Patients in Age Groups

Age (years)	No(%)
20-40	27(18)
41-60	105(70)
61-80	17(11)

Table 2: Patient Characteristics

Total Number of Patients (n)	150 Patients
Gender	-Male: 81 (54%) -Female: 69 (46%)
Mean Age	49.34±11.45 years
Mean Hemoglobin (g/dl)	7.62 ± 3.12
Mean Platelet Count (10^9/l)	79 ± 69
Mean Albumin (g/dl)	3.2 ± 2.1
Mean Bilirubin	1.78 ± 1.21
Mean Liver Size (cm)	12.13 ± 4.34
Mean Spleen Size (cm)	11.67 ± 3.11
Ascites	150 (100%)
Esophageal varices	150 (100%)
Grades of esophageal varices at presentation	F2 = 71 (47.33%) F3 = 79 (52.67%)
Number of sessions required for eradication of esophageal varices	3±2 sessions
Fundal varices at Presentation	None (0%)
Child Pugh Class	-Child Class A= 55 (36.67%) -Child Class B =67 (44.67%) -Child Class C= 28 (18.66%)

Table 3. Frequency of fundal varix formation following esophageal variceal banding

Follow-Up Duration	Number	Percentage	GOV2	IGV1
2 months	2	1.33%	2	0
4 months	8	5.33%	5	3
6 months	12	8%	7	5
Total	22	16%	14	8

Twenty two (14.66%) developed fundal varices. Of these 12 patients (8%) developed fundal varices at 6 months following banding of esophageal varices (Table 3). Out of these 22 patients 14(63.63%) had GOV2 while 8(36.36%) had IGV1.There was no age co-relation found (p-value 6.9) with the development of fundal varices in patients who were banded for esophageal varices.

Discussion

Gastric varices are less prevalent than esophageal varices and are present in approximately 25% of the patients with portal hypertension.[4] Eradication of esophageal varices has been identified as a risk factor for the development of fundal varices. This can be explained by the gastric hemodynamic changes that result from the blockage of shunting in the palisade zone and the formation of "new" or "Secondary" gastric varices.[5]

Yuksel et al found that 37 out of 85 patients had fundal varices before they underwent ligation of esophageal varices, increasing to 46 observed at 3 month follow up endoscopy after the procedure, a statistically significant increment of almost 10.59% after eradication. The severity of portal hypertensive gastropathy also increased.[10] Our study demonstrates a higher incidence at six months duration as to this study which reflects that formation of fundal varices is time dependent.

In a large reterospective study by Mumtaz et al. comprising 1436 patients, gastric varices were present in 220 (15%) patients at presentation. [12] Secondary gastric varices were found in 23% of patients within 6 months after eradication of esophageal varices as compared to 14.66% of fundal varices in our study. This discrepancy may be due to small sample size in this study and that they included all types of gastric varices in their as compared to only fundal varices in this study.

Korula et al. in their 7 years follow up of patients receiving endoscopic sclerotherapy for variceal hemorrhage found fundal varices in 4.1% cases only. [13] Method used for bleeding control in their study was sclerotherapy as compared of EVBL in this study. There is also a difference in sample size and ethnic group.

Available local study has determined that the frequency of occurrence of secondary gastric varices six months after endoscopic variceal band ligation which approximated to 16.04%.[5] Our study demonstrated a similar result of 14.66% overall occurrence of fundal varices following esophageal variceal band ligation but in comparison our study has established that fundal varices develop as early as 2 months after band ligation of esophageal varices.

In another local study by Naseer et al. the frequency of fundal varices was found to be 3.1% in patients presenting for endoscopy. [8]It is much less than our results(14.66%). They did not follow the patients after eradication of esophageal varices to determine the incidence after EVBL which is hallmark of this study.

The frequency of new fundal varix formation at two months was 1.33%, seen in only 2 patients out of a hundred and fifty patients being followed. At four months of follow up 8 other patients developed fundal varices, representing 5.33% of the study population. Six months after esophageal variceal band ligation, another 12 patients (8%) who had been banded for esophageal varices developed fundal varices. Our study thus comes as a first in reporting the development of new fundal varices in relation to time since banding. Therefore, in order to screen for new fundal varices, patients must be subjected to an endoscopic surveillance program.

According to our results, the frequency of occurrence, and the probability of developing a fundal varix was time dependant. However, there was no predilection for the development of fundal varices after esophageal variceal band ligation in any particular age group, nor does the sex of the patient predispose to increased incidence of developing a fundal varix.

Since limited data exists with regard to the development of new fundal varices following esophageal banding, there is plenty of space for future research, and studies are required with a larger patient pool, and with same expert endoscopist for the particular period of research. Studies can also be done to observe the frequency of formation of fundal varices after esophageal banding in comparison with controls, and also in patients treated with beta-blockers.

Conclusion

1. New fundal varices develop with increasing frequency in patients treated with esophageal variceal band ligation, and the incidence has a time-dependent relationship.

References

1. Shah SMA, Mashia SA, Younus MF, Ghauri A, Ejaz R, Alshalabi H et al Hepatic cirrhosis-disease burden. J. Rawalpindi Med. Coll 2015;19:17-20.
2. Al-busafi SA, Ghali P, Wong P, Deschenes M. Endoscopic Management of portal hypertension. Inter J of Hepa. 2012:1-12.
3. Mehta G, Abraldes JG, Bosch J. Developments and controversies in the management of oesophageal and gastric varices. Gut 2010 ; 59(6):701-05.
4. Sarin SK, Mishra SR. Endoscopic therapy for gastric varices. Clin Liver Dis 2010 ;14:263-79.
5. Samiullah S, Memon MS, Memon HG, Ghori A. Secondary gastric varices in hepatic cirrhosis. J Coll Physicians Surg Pak. 2011 Oct;21(10):593-96.
6. Sarin SK. Long-term follow-up of gastric variceal sclerotherapy: an eleven-year experience. Gastrointest Endosc1997;46:8-14.
7. Song WK. Management of gastric varices. Clinical liver disease. 2012 ;1(5):158-62.
8. Naseer M, Khan A, Yasmeen R, Gillani FM, Saeed F. Determination of frequency and treatment outcome in patients of fundal varices presenting with upper gastrointestinal bleeding. Pak Arm Forces Med J. 2012 ;483-86.
9. Franchis R. Revising consensus in portal hypertension: Report of the Baveno V consensus workshop on methodology of diagnosis and therapy in portal hypertension. J of Hepa. 2010;53:762–68.
10. Yuksel O, Koklu S, Arhan M, Yolcu OF, Ertugrul I. Effects of esophageal varices eradication on portal hypertensive gastropathy and fundal varices: a retrospective and comparative study. Dig Dis Sci 2006;51:27-30.
11. Mumtaz K, Majid S, Shah HA, Hameed K, Ahmed A. Prevalence of gastric varices and results of sclerotherapy with N-butyl 2-cyanoacrylate for controlling acute gastric variceal bleeding. World J Gastroenterol 2007; 13:1247-51. 18.
12. Korula J, Chin K, Ko Y, Yamada S. Demonstration of two distinct subsets of gastric varices: observations during a seven-year study of endoscopic sclerotherapy. Dig Dis Sci 1991; 36:303-09.