Original Article

Endoscopic Evaluation of Upper Gastrointestinal Bleeding in Liver Cirrhosis

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Abstract:

Liver cirrhosis is a common, often progressive and usually fatal disorder. Upper gastrointestinal (UGI) bleeding is a leading cause of death in patient with cirrhosis. In this observational study, 50 patients were included according to selection criteria. Maximum participants (40%) were in the age group of 40-50 years of age (Mean \pm SD = 42.94 \pm 9.72, Minimum = 20 and Maximum = 62). Male were 68% and female were 32%. Male were more sufferer than female. Overall male to female ratio were 3:1. Esophageal varies was the commonest cause of haematemesis and melaena followed by portal hypertensive gastropathy, duodenal ulcer, gastric ulcer, erosive gastritis and reflux oesophagitis in patient with liver cirrhosis. Oesophageal varies were found in 60% participants. There were no gastric varices found among the participants. Presence of Portal HTN gastropathy was in 14% patients. Gastric ulcer was found in only 6% patients, 10% patients had duodenal ulcer, 3 (6%) patients had reflux oesophagitis, erosive gastritis was also present in 3(6%) of participants. Haematemesis, melaena and both haematemesis and melaena were noted in 24%, 22% and 54% of the patients respectively. HBsAg was positive in 50% patient. HCV was positive among 18% patients.

Key words: Upper GI bleeding, Cirrhosis of liver, Endoscopic evaluation.

Introduction:

Hepatic cirrhosis is a common disease characterized by diffuse hepatic fibrosis and nodule formation. The cardinal feature of cirrhosis is an increase in fibrous tissue, progressive and wide spread death of liver cells, and inflammation leading to loss of the normal liver architecture¹.

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duodenum at the ligament of Treitz³. Upper gastrointestinal haemorrhage is a common and serious medical as well as surgical emergency. The most common presenting features of upper GI haemorrhage

are haematemesis, melaena and shock⁴. Upper gastrointestinal bleeding results from a variety of lesions. In patients with liver cirrhosis, bleeding occurs from the same lesions seen in the general population in addition to those resulting from portal hypertension, namely gastroesophageal varices and portal hypertensive gastropathy⁵.

In patients with acute upper gastrointestinal bleeding and liver cirrhosis, the most common source of bleeding was varices, in particular oesophageal varices (57.7%), much less common was gastric varices (5.1%). Varicose bleeding represented a total of 62.8% of all bleeding cases. Portal hypertension gastropathy was the cause in another 9.5% of cases. Bleeding due to portal hypertension, from varices plus portal hypertensive gastropathy, comprised 72.3% of all bleeding cases. The second most frequent cause of bleeding was peptic gastric and duodenal ulcers with a share of 18.2%, with the proportion of gastric ulcers and duodenal ulcers being equal. The other proven non-varicose sources included reflux oesophagitis (2.9%); Mallory-Weiss syndrome occurred with the same frequency, while erosions appeared in 1.5% of cases. In 2.2% of cases the source of the bleeding was not discovered during gastroscopy⁶.

Another study shows less frequent causes of acute bleeding are: reflux oesophagitis 1.7%, Mallory-Weiss syndrome 2% and erosive gastritis 3.9%^{7,8}. Acute esophageal variceal bleeding is a catastrophic complication of portal hypertension with a mortality ranging from 30% to 50%⁵. Prompt diagnosis and treatment is, therefore, essential. One-half to two-thirds of patients with cirrhosis who present with upper gastrointestinal bleeding have nonvariceal sources of bleeding, and many of these patients have more than one lesion⁹. The annual risk of development of esophageal varices is approximately 5%. However, only a third of all patients with esophageal varices will bleed¹⁰.

Upper gastrointestinal endoscopy is the most valuable initial procedure of choice for the evaluation of upper GI bleeding. Early endoscopy allows not only the detection of cause and source of bleeding, it also gives estimation of the risk of recurrent bleeding and potentially enables various therapeutic options. Studies have shown that early endoscopy is associated with lower healthcare costs and improved medical outcomes, compared with other procedures. However, upper GI endoscopic findings are not diagnostic in about 10% of cases¹¹⁻¹⁴.

So it is essential to identify the causes of upper gastrointestinal bleeding in liver cirrhosis for proper management of the patient. There are no significant studies in Bangladesh to see aetiology of upper gastrointestinal bleeding in liver cirrhosis, so the findings of this study will be able to strengthen the current knowledge and will be useful for targeting the preventive strategies and proper management.

Materials and Methods:

This observational study was conducted at Medicine & Gastroenterology departments of Faridpur Medical College Hospital, Faridpur from June 2015 to November 2015. Patients of liver cirrhosis present with upper gastrointestinal bleeding were included maintaining inclusion criteria and exclusion criteria with a sample size of 50. After hemodynamic stabilization, patients were evaluated through detailed history, thorough clinical examination and all necessary investigations including upper gastrointestinal endoscopy to find out the cause of bleeding. The sample was selected purposively. All patients / legal guardian were briefed about the study. Informed and written consent obtained from all patients who could give the consent and those who were unable to respond, their appropriate relatives/legal guardian gave the consent. Confidentiality and privacy was maintained throughout the study. Participant refusal and withdrawal from the study at any time was accepted. A semi-structured questionnaire was developed. Then data were entered into computer and analyzed with SPSS version 16.

Results:

In this observational study, among 50 patients maximum participants (40%) were in the age group of 40-50 years of age (Table I). Male were 68% and female were 32%.

Table-I:	Distribution of the patient according to
age (n=5	0)

Age group	Number of Patient (%)
15-20	2 (4)
20-30	3 (6)
30-40	13 (26)
40-50	20 (40)
50-60	10 (20)
> 60	2 (4)
Total	50 (100)

Mean \pm SD = 42.94 \pm 9.72, Minimum = 20 and Maximum = 62

In this series, the maximum number of patients 20 (40%) were between 40-50 years age group, next 13 (26%) were between the age group of 30-40 years.

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Table-II: Distribution of the patient according to	
Pattern of upper GI bleeding (n=50)	

Pattern	Number of Patient (%)	
Presence of both haematemesis and melaena	33 (66)	
Only haematemesis	10(20%)	
Only melaena	7(14%)	
Total	50(100%)	

Both haematemesis and melaena was present in 66.0% patients. Only haematemesis was present among 20.0% participants. Only melaena was present among 14.0% participants.



Figure 1: pie-chart showed oesophageal varices (OV)was present in 60% of patients, Erosive gastritis (6%) was present in 6% of patients, Reflux oesophagitis was present in 4% of patients, duodenal ulcer was present in 10% of patients, gastric ulcer (GU) was present in 6% of patients, Portal hypertensive gastropathy (PHG) in 14% of patients.

Discussion

Maximum participants (40%) were in the age group of 40-50 years of age (Mean \pm SD = 42.94 \pm 9.72, Minimum = 20 and Maximum = 62). This finding can be compared with some other studies. Ahmed et al in there study showed average age of the patients was 39.9 years with the range of 18-90 years & 50% of patients were in the age range 20-39 years¹⁵. Mamun et al in his study showed that the mean age of the patients with upper gastrointestinal haemorrhage was 35.65 years and 49.40 years for duodenal ulcer and gastric ulcer respectively¹⁶.

In this study Male were 68% and female were 32%. Hadayat et al showed in their study 158 (62.7%) were males and 94 (37.3%) were females. Findings are almost similar to our study¹⁷. Regarding sign and symptoms, both haematemesis and melaena present in 66% patients. Only melaena was present among 14 % participants and only haematemesis among 20%. Ahmed et al in their study showed haematemesis or melaena and both haematemesis and melaena were the presenting feature in 16%, 42% and 42% of the patients²⁰. HBsAg was positive in 50%, HCV was positive among 18% patients and it was not done in 12% patients, but it was negative in the rest of the patients. In another study, the etiology of the liver cirrhosis was alcohol 65.7%, alcohol + viral hepatitis B 2.9%, alcohol + viral hepatitis C 2.2%, purely viral hepatitis B 7.3%, only viral hepatitis 9.5%¹⁸. This variation of study may be due to cultural and religious difference. It is dissimilar to our study as alcohol consumption in our country is rare.

Different studies showed that the majority of cases showed multiple findings in the upper digestive tract, each of which was a potential cause of bleeding. Portal hypertension led to bleeding caused by varices and portal hypertension gastropathy in 72.3% of patients, 62.8% of patients suffered from purely variceal bleeding and 37.2% from non-variceal bleeding^{7-9,11,14}.

In our study Oesophageal varices was found in 60% patients. Common source of bleeding was varices, in particular oesophageal varices (60%), no isolated gastric varices. Variceal bleeding presented a total of 60% of all bleeding cases. Portal hypertensive gastropathy was the cause in another 14% of cases. Bleeding due to portal hypertension, from varices + portal hypertensive gastropathy, comprised 74% of all bleeding cases. The second most frequent cause of bleeding was peptic gastric and duodenal ulcers with a share of 16%, with the proportion of gastric ulcers and duodenal ulcers 3:5. The other proven non-variceal sources included reflux oesophagitis (4%); while erosions appeared in 6% of cases. Other study showed similar type endoscopic findings. Svobodaa et al showed in their study the most frequent causes of acute bleeding were: oesophageal varices (57.7%), peptic gastric and duodenal ulcers (18.2%), portal hypertensive gastropathy (9.5%), gastric oesophagitis varices (5.1%),reflux (2.9%),Mallorv-Weiss syndrome (2.9%)and erosive gastropathy (1.5%). A negative diagnosis was made in not more than 2.2% of patients18.

The majority of cases showed multiple findings in the upper digestive tract, each of which was a potential cause of bleeding. There were no gastric varices found among the participants. Presence of portal hypertensive gastropathy was in 14% patients. Gastric ulcer was found in only 6% patients, 10% patients had duodenal ulcer. In

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a recent study conducted in Romania, Romcea et al included 1284 cirrhotic patients, out which 297 had upper GI bleeding. The dominant aetiology was variceal haemorrhage which was present in 217 cases (73%), while 80 patients had non-variceal upper gastrointestinal bleeding (27%). Of the cases with variceal bleeding 199 (91.7%) were from oesophageal¹⁹, these are almost similar to our study.

In our study only (4%) patients had reflux oesophagitis. Erosive gastritis was present in 6 % of participants. No patient had Mallory-Weiss syndrome.

A study showed 30–40% of cirrhotic patients may have non-variceal upper gastrointestinal bleeding (NVUGIB), and it is frequently caused by gastroduodenal ulcers^{20,21}. Svoboba P. et al¹⁸ found that 18.2% of the cirrhotics who presented with upper GI bleed had peptic ulcer on endoscopy, while in the Bilal A. et al study, 34% had peptic ulcer. There is a higher incidence of peptic ulcer in cirrhotics^{22,23} as compared to the "general population" (8.3%), 10.3% of patients had peptic ulcer, which is comparable to the incidence in general population. This study comparable to the study done by D'Amico G. et al,⁷ Kantorova I. et al ²⁴ and Seo YS. et al¹¹ which was 7.5%, 8.5% and 13.3 % respectively.

In some studies, the most frequent causes of acute bleeding were: oesophageal varices (57.7%), peptic gastric and duodenal ulcers (18.2%), portal hypertensive gastropathy (9.5%), gastric varices (5.1%), reflux oesophagitis (2.9%), Mallory-Weiss syndrome (2.9%) and erosive gastropathy $(1.5\%)^{2-7,10-14}$. In one study of 40 patients, (70%) had gastroesophageal varices diagnosed at upper endoscopy, while 50% actually bled from varices⁶.

In our study, endoscopic finding were similar to the above study. Our study showed in 60% of cases, variceal bleeding was happened, whereas in 40 % it was followed by bleeding from a non-variceal source in patients with liver cirrhosis.

Conclusion:

Most common source of bleeding in liver cirrhosis was oesophageal varices. Among non-variceal sources of bleeding peptic ulcer was the leading cause. The findings of this study will be able to strengthen the current knowledge and will be useful for targeting the preventive strategies and proper management. A large scale study is needed to reveal the actual situation regarding aetiology and prompt management to reach a favourable outcome in our country.

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