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Spectrum of upper gastrointestinal bleed in patients with cirrhosis of liver

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ABSTRACT

Background & Objectives: Acute upper gastrointestinal (UGI) bleeding is a serious medical problem in patients with cirrhosis of liver associated with high mortality. Gastro-oesophageal variceal bleed is the most common complication of portal hypertension in patient with liver cirrhosis. This study was undertaken to establish the causes of UGI bleed in cirrhosis, their relative incidences, clinical presentation , endoscopic findings, outcomes during hospitalization including rebleeding and mortality were studied. **Materials & Methods:** One hundred and twenty patients with clinical features, sonological and endoscopic evidence of portal hypertension and cirrhosis of liver who presented with upper gastrointestinal bleed were included in the study. After haemodynamic stability, each patient underwent UGI endoscopy usually within 12 hours and the aetiology with diagnostic findings were documented. **Results:** Ruptured oesophageal varices was the most common cause of UGI bleed in cirrhotic patients. Non variceal causes of UGI bleed accounted for 33.3 % of cases. The majority of non variceal bleed was peptic ulcer disease and accounted for 19.2 % of total UGI bleed in liver cirrhosis. This was followed by portal hypertension gastropathy, erosive gastropathy, mallory-weiss tear and others. **Conclusion:** The most frequent causes of acute gastrointestinal bleeding in cirrhosis was oesophageal varices. Peptic ulcer disease is also a common aetiology of UGI bleed in cirrhosis. Cirrhotic patients with variceal etiology have more chances of rebleeding and have higher mortality than those with non variceal aetiologies.

Key words: Cirrhosis of liver; endoscopy; oesophageal varices; peptic ulcer disease; upper gastrointestinal bleed

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INTRODUCTION

Acute upper gastrointestinal bleeding is a serious medical problem in cirrhotic patients. Acute gastrointestinal (GI) bleeding is a potentially life-threatening emergency that is a common cause of hospitalization with increased morbidity and mortality. Upper gastrointestinal bleeding (UGIB) is defined as bleeding derived from a source proximal to the ligament of Treitz. Bleeding from the upper GI tract is more common than bleeding from the lower GI tract.[1]

Acute upper gastrointestinal bleeding is a serious medical problem in patients with cirrhosis of liver.[2] The most frequent causes of acute bleeding in cirrhosis are from oesophageal varices, gastric varices, peptic ulcer disease, portal hypertension gastropathy, reflux oesophagitis, mallory-weiss tear, erosive gastropathy and others.[3,4] Variceal bleed is the most dangerous complication of portal hypertension in patient with liver cirrhosis.[5]

This study was undertaken to highlight the clinical spectrum and identification of different aetiologies of upper gastrointestinal bleed in patients with liver cirrhosis. This study may prove helpful in establishing clinical and endoscopic correlations and formulating guidelines in management and treatment of patients with cirrhosis of liver presenting with UGI bleed.

MATERIALS AND METHODS

This observational, cross-sectional, prospective hospital based study was carried out in the department of medical gastroenterology at College of Medical Sciences Teaching Hospital, Nepal from January 2015 to December 2016. Liver biopsy is the gold standard for diagnosis of cirrhosis. However, biopsy was not performed in our cases. All subjects hospitalized under the department of medical gastroenterology for acute upper gastrointestinal bleeding with clinical features, laboratory and sonological non invasive and indirect findings suggestive of cirrhosis of liver and assessed according to Child-Turcotte-Pugh (CTP) score along with sonological and/ or endoscopic evidence of portal hypertension were included in the study. Critically ill patients and those who failed to give consent were excluded from the study.

Data regarding demographic variables, clinical features, bleeding characteristics were collected and alongside blood investigations like complete blood count, platelets count, blood grouping, liver function test , prothrombin time / international normalized ratio (PT / INR), coagulation profile and viral serologies were sent. Patients were classified under CTP classes.

After haemodynamic stabilization, usually within 12 hours but sometimes as late as 24 hours, each patient underwent endoscopic investigation by standard flexible gastro-duodenal endoscope (PENTAX EPK 700, PENTAX JAPAN Inc) and diagnostic findings were documented. In cases, when multiple lesions were found in UGI endoscopy, the lesion with active bleeding or recent stigmata of haemorrhage was considered the cause of bleeding. For control of bleeding, endoscopic and pharmacologic treatments were used. Variceal bleeders were treated with injection terlipressin or octeotride and with variceal band ligation. Clinical outcomes during hospitalization including rebleeding and mortality were assessed.

Data were collected on a structured proforma and entry was done in Statistical Packages for the Social Sciences version 20. All categorical data were expressed in percent and absolute number. All numerical continuous data were expressed in mean ±SD. The data analysis was done using SPSS version 20. All tests were analyzed with a 95% confidence interval and a P value of <0.05 was considered significant.

Table 1: Age group and sex of patients

Age group (Years)	Sex	
	Male	Female
<30	11	2
31-50	33	11
51-70	41	13
>71	8	1

Table 2: Classification of cirrhotic patients according to CTP classes

CTP CLASS	VARICEAL GROUP (N=80)	NON VARICEAL GROUP (N=40)
A	0	0
B	12	15
C	68	25

RESULTS

One hundred fifty patients of cirrhosis of liver with UGI bleeding were taken up for the study. But 18 patients were taken away to home or elsewhere by patient relatives against medical advice despite initial management and few days of admission and 12 were excluded because of inadequate data.

Finally 120 patients were enrolled in the study. There were 93 males and 27 females (M:F=7:2). The ratios of male and female were 3:1 in variceal group and 4:1 in non variceal group. Mean age of the patients was 53.4 years (range of 24 to 75 years). The different age groups of male and female patients were classified as in table 1.

Further, these cirrhotic patients were classified into different CTP clinical classes as in table 2. Majority of cases with or without varices were of Class C. Class C individuals were found to present with variceal bleed 5.6 times that in Class B.

Alcoholic cirrhosis accounted for 108 (90 %) of total cases. Six cases (10 %) were diagnosed with chronic hepatitis B. Three cases (2.5%) were of chronic hepatitis C and rest 3 (2.5%) were classified as cryptogenic.

Majority of patients presented with both haematemesis and melaena. Fifty six (46.7 %) patients presented with both haematemesis and melaena , forty (33.3%) presented with only melaena and rest twenty four (20 %) with haematemesis only. The relative presenataion of UGI bleed in variceal and non variceal group was as shown in table 3.

The most common cause of UGI bleed was ruptured oesophageal varices in 80 patients (66.7

Table 3: Patterns of upper gastrointestinal bleed presentation in cirrhosis

PARAMETERS	TOTAL(N=120)	VARICEAL GROUP (N=80)	NON VARICEAL GROUP (N=40)
Haematemesis only	24	12	12
Melaena only	40	30	10
Haematemesis + melaena	56	38	18

%). Non variceal causes of UGI bleed accounted for 33.3 % of cases in the present study. The majority of non variceal bleed was due to peptic ulcer disease and that accounted for 57.5 % of non variceal and 19.2 % of total UGI bleed cases in liver cirrhosis. Out of 40 non variceal bleed cases, 13 had duodenal ulcers and 10 had gastric ulcers. Various aetiologies of non variceal UGI bleed in cirrhotic patients were as depicted in table 4.

UGI endoscopy of all these patients revealed that 90 patients had varices. Eighty five had esophageal varices and five had gastric varices. Out of 90 patients with gastro-oesophageal varices, 80 patients with oesophageal varices (88.9%) presented with UGI bleeding. No patients with gastric varices presented with UGI bleeding.

Cirrhotic patients were classified under three categories according to UGI endoscopy. First category consisted of 60 patients who had only varices and identified as sole bleeding source on UGI endoscopy. Second category included 30 patients who had varices with non variceal mucosal lesions also. Twenty patients out of these 30 patients actually bled from esophageal varices. Ten bled from other lesions , four from duodenal ulcer, three from gastric ulcer and three from erosive gastritis. Third category comprised of last 30 patients who had no varices but had other non variceal mucosal lesions. Nine of these patients bled from duodenal ulcer, seven from gastric ulcer, and seven from portal hypertensive gastropathy, three from erosive gastritis, three from mallory-weiss tear, and a single case from gastric carcinoma.

Mean haemoglobin at presentation was 7.71±1.63 gm % in variceal group and 8.18±1.48 gm% in non variceal group . Mean complete Rockall score after UGI endoscopy was 4.73±1.20 in variceal group and 4.14±0.98 in non variceal group. Thirty patients (37.5%) in variceal group and five patients (12.5%) in non variceal group had Rockall score more than or equal to 5 as depicted in table 5.

Forty four out of 120 (36.7%) patients presented with shock. The ratio of patients with variceal bleed group presenting in shock was double than those with non variceal bleed group. Rebleeding within 14 days was higher with variceal group compared to that with non variceal group (30 % vs. 10 %).

Similarly, mortality was also higher with variceal group compared to that with non variceal group (27.5 % vs. 7.5%). Mortality during hospitalization was higher with variceal bleed group compared to that with non variceal bleed group (27.5 % vs. 7.5%) in the present study as shown in table 5.

DISCUSSION

One hundred twenty patients of cirrhosis of liver presenting with UGI bleeding were enrolled in this study with 93 male (77.5 %) and 27 female (22.5%) ; M:F = 7:2. Studies by Svoboda et al.,[6] Romcea et al.[7], Olajide et al.[8], also have highlighted male predominance of UGI bleed in cirrhosis . Mean age of cirrhotic patients with UGI bleed in this present study was 53.4 years which is almost similar to those reported by Svoboda et al.[6] (mean age of 56.9 yrs) and Romcea et al.[7] (mean age 56.8 years). However, Olajide et al.[8] has reported a lesser mean age of 48.5 years.

In the present series, predominant clinical form of upper gastrointestinal bleed was both haematemesis and melaena in fifty six (46.7 %) , only melaena in twenty four (20 %) and only haematemesis in rest forty (33.3%) patients . Haematemesis and melaena predominated in 84.17% , melaena in 14.81% and hematochezia in 1.01% patients in a series reported by Romcea et al.[7]

Majority of cases with or without varices were of Class C. Class C individuals were found to present with variceal bleed 5.6 times that in Class B. Several studies have have highlighted that variceal rupture risk increases with the increase in severity of the liver disease.[7,9,10]

The most frequent aetiology of upper GI bleed in cirrhosis was rupture of esophageal varices in

Table 4: Aetiologies of non variceal UGI bleed in cirrhotic patients

Causes of non variceal bleed	Number (Total 40)	Percentage
Duodenal ulcer	13	32.5
Gastric ulcer	10	25
Portal hypertensive gastropathy	7	17.5
Erosive gastritis	6	15
Mallory Weiss tear	3	7.5
Gastric carcinoma	1	2.5

in cirrhosis of liver.

Larger percentage of patients in variceal bleed group had Rockall score more than or equal to 5 when compared to non variceal group (37.5% vs. 12.5%) in the present series.

Rebleeding after primary hemostasis within 14 days was higher with variceal group compared to that with non variceal group (30 % vs. 10 %). Rebleeding rate during hospitalization was, however only marginally higher in variceal bleeding group compared to non variceal bleed group (18.9% vs. 17.2%) in the study by Svoboda

Table 5: Comparisions of different parameters between variceal and non variceal groups

PARAMETERS	VARICEAL GROUP (N=80)	NON VARICEAL GROUP (N=40)
Mean Haemoglobin	7.71±1.63 gm %	8.18±1.48 gm%
Shock (SBP< 90 mm Hg)	36 (45 %)	8 (20 %)
Mean Rockall score	4.73±1.20	4.14±0.98
Rockall score > 5	30 (37.5 %)	5 (12.5%)
Rebleeding within 14 days	24 (30 %)	4 (10 %)
Death	22 (27.5 %)	3(7.5 %)

Table 6: Studies showing different aetiologies of non variceal bleed in cirrhosis

Non variceal bleed: Aetiology	Present study	Svoboda et al.[6]	Romcea et al.[7]	Gonzalez et al.[11]
Peptic ulcer disease	57.5 %	43.1%	55%	50.6 %
Portal gastropathy	17.5%	22.4%	17.5%	14.4%
Erosive gastritis	15%	3.5%	11.25%	4.5%
Mallory Weiss tear	7.5%	6.9%	6.25%	11.3%

eighty (66.7 %) patients in our study. Variceal bleed accounted 57.7%, 73% and 50% of acute UGI bleed in cirrhotics in their studies by Svoboda et al.[6] ,Romcea et al.[7] and Olajide et al.[8] respectively.

Non variceal causes of UGI bleed accounted for 33.3 % of total patients in our study. The majority of non variceal bleed was peptic ulcer disease and accounted for 19.2 % of total UGI bleed in liver cirrhosis. Duodenal ulcers were seen more commonly than gastric ulcers. Peptic ulcers with predominance of duodenal ulcers were also noted in studies by Svoboda et al.[6], Romcea et al.[7] and Gonzalez et al.[11] The various aetiologies of non variceal bleed in some series has been highlighted in table 6. All these studies suggest that peptic ulcer is the commonest cause of non variceal UGI bleed et al[6] which is in contrary to the present study.

Mortality during hospitalization was higher with variceal bleed group compared to that with non variceal bleed group (27.5 % vs. 7.5%) in the present study. Similarly, mortality was 20.2% among variceal bleed group and 6.9 % among non variceal bleed group in the study by Svoboda et al.[6]

CONCLUSION

UGI bleed from oesophageal varices is a common manifestation in patients with cirrhosis of liver . The present study showed a high incidence of UGI bleed in cirrhosis patients secondary to ruptured oesophageal varices. However, cirrhotic patients do not always bleed from varices. In our series, we found that one-third patients (33.3%) with liver cirrhosis had UGI bleed of non variceal aetiology,

of which the most common was peptic ulcer; duodenal followed by gastric ulcers. Variceal bleeders present with higher Rockall score compared to those with non variceal bleed. UGI bleed of variceal etiology has more chances of rebleeding and has higher mortality than those with non-variceal aetiologies.

This study highlights the importance of clinical presentation, role of endoscopy in cirrhotic patients with UGI bleed, their outcomes, and may be helpful in formulating guidelines for proper and needful management.

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