Non-variceal Causes and Outcome of Acute Upper Gastro-intestinal Bleeding among Cirrhotic Patients in Tropical Medicine Intensive Care Unit

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Background and aim of the work: non-variceal bleeding is less common than variceal bleeding among cirrhotic patients; hence there are fewer studies that pay attention to its causes and outcomes. The aim of this study is to shed light on the causes of acute non-variceal bleeding in cirrhotic patients and the outcome of acute upper gastrointestinal bleeding in those patients.

Patients and Methods: this cross sectional study included 179 patients, they were allocated into two groups according to the source of bleeding; group I: included 104 patients with variceal bleeding and group II: included 75 patients with non-variceal bleeding.

Results: The frequency of non-variceal bleeding was 41.9%. The mean period of intensive care unit stay was 5.03 ± 2.65 days ranging between 1and 17 days. The overall rate of early rebleeding was 3.7% and the mortality rate in intensive care unit was 4.5%. The most common cause of non variceal bleeding is ulceration (24%), followed by portal hypertensive gastropathy (17.3%), and in 24% of cases the cause of bleeding was obscure. Mortality is significantly correlated to white blood cells count (r=0.2 p=0.002), Child's grade and score (r=0.217 p=0.003 and r= 0.16 p=0.03 successively) as well as Glasgow-Blachford score (r=0.18 p=0.01).

Conclusion: The frequency of non-variceal bleeding among cirrhotic patients admitted with acute upper gastrointestinal bleeding bleeding is 41.9%. The mortality rate in those patients is 4.5%. The degree of deterioration of liver functions and the severity of initial bleeding episode were the most important predictors of mortality.

INTRODUCTION

The most life-threatening complication of liver cirrhosis and portal hypertension is acute variceal bleeding which is associated with increased mortality **[1]**. It accounts for approximately 70% of all cases of upper gastrointestinal bleeding in patients with cirrhosis. The remainder cases bleed from sources other than varices like ulceration or portal hypertensive gastropathy. Hence, whenever a cirrhotic patient is admitted to the emergency room with acute upper GIT bleeding, the first possibility is variceal bleeding, however non variceal bleeding should be put in consideration **[2]**.

Aim of the study:

The aim of this study is to shed light on the different causes of non variceal bleeding and the outcomes of acute upper GIT bleeding among cirrhotic patients.

PATIENTS AND METHODS

This cross sectional analytical study was conducted in Tropical Medicine Department, intensive care and emergency endoscopy units in the period between October 2018 and April 2019. One hundred and seventy nine patients were included in the study. They were randomly selected from patients with liver cirrhosis, diagnosed by combination of clinical, laboratory and radiological evidence,

admitted to the tropical medicine department ICU with acute upper GI bleeding. The exclusion criteria were as follows; patients who did not give informed consent to participate in the study, patients <18 years old, patients who did not undergo endoscopic examination due to severe hemodynamic instability or hepatic coma, or due to Glassgow Blachford score 2 or less.

Patients were allocated into two groups according to the source of upper GI bleeding; group I: included patients with variceal bleeding and group II: included patients with non-variceal bleeding. They were subjected to full history taking, detailed clinical examination, routine laboratory investigations including; complete blood count, liver and kidney function tests, coagulation profile, viral markers and alpha fetoprotein

Patients also performed abdominal ultrasonography with special attention sonographic features suggesting cirrhosis, portal vein, spleen size and ascites. The severity of liver disease was assessed and classified according to Child-Pugh classification **[3]**.

This risk stratification was made using the Glasgow-Blatchford score (GBS) score **[4]**. Initial resuscitation was done after assessing airway, breathing and circulation by fluid replacement using either crystalloid or colloid fluids, supplemental oxygen, Insertion of nasogastric tube for aspiration and lavage procedure, blood transfusion following the restrictive strategy with target Haemoglobin 7-8 g/dl with continuous monitoring of patients' haemodynamics, this was done through observing vital signs and organ-specific perfusion such as capillary refill time and urine output.

Patients were given the following pre-endoscopic medication; proton pump inhibitors (pantoprazole), antibiotic prophylaxis against portal bacteremia and spontaneous bacterial peritonitis (ceftriaxone), prokinetic (metoclopramide), vasoactive agent to reduce portal blood flow (octereotide) and Correction of coagulation abnormalities is done before endoscopy using fresh frozen plasma (FFP) and vitamin K **[5]**.

Patients performed upper GI endoscopy under sedation by midazolam in the first 24 hours of admission. The esophageal varices were graded according to Paquet grading system **[6]**, and portal hypertensive gastropathy was graded according to a three grades grading system **[7]**. The cause of bleeding and the type of the bleeding lesion were recorded for the patients along with any other abnormal findings. The lesion was considered the source of bleeding if it was oozing or spurting blood or has an adherent clot or red spots and if it was the only lesion. Bleeding lesions were managed accordingly **[8]**.

All the patients were followed during their ICU stay to assess: the length of ICU stay, transfusion of blood and blood products (whole blood, packed RBC's, plasma), repeated endoscopy for rebleeding and the mortality rate during ICU stay.

Statistical Analysis

The data were processed using SPSS epi info version 16. The numerical data were presented as mean ± SD and the categorical data were presented as number and percentage. Comparison between the studied groups as regards categorical data was done using chi square and fisher exact. The numerical data were compared using t test for normally distributed and MW was used when data were not normally distributed. The correlation between numerical and categorical dichotomas variables was done using point biserial correlation, whereas the correlation between dichotomas and ordinal variables was done using Spearman rho correlation, and the correlation between two dichotomas variables was done using Phi coefficient.

RESULTS

Among the overall studied population, 73.7% were males and 26.4% were females. Most of the patients had cirrhosis secondary to chronic HCV were (97.8%). Only 3% of patients had history of bilharziasis. The frequency of non-variceal bleeding was 41.9%. The mean period of ICU stay was 5.03 ± 2.65 days ranging between 1and 17. The overall rate of early rebleeding was 3.7% and the in ICU mortality rate was 4.5%.

Table 1, 2 and 3 summarize the demographic, history, clinical and laboratory data. Table 1 represents shows that patients in group I had significantly higher frequency of previous history of upper GI bleeding and upper GI endoscopy (72% vs 53.8% p=0.01 for bleeding and 72% vs 50% p=0.001 for endoscopy.

Table 2 shows that the spleen is significantly larger among patients of group I, the variceal bleeding group. The frequency of normal spleen size was 5.8% in group I vs 21.3% in group II p=0.01. There were no significant differences between the studied groups as regards the frequency of jaundice, lower limb edema, hepatomegaly, ascites, encephalopathy, portal vein patency, presence of focal lesion Child grade and score. Table 2 also shows that group I had significantly higher Glassgow Blatchford score on admission than group II (14.1±2.78 vs 12.7±3.15 points p=0.002).

Table 3 represents the comparison between the studied groups as regards laboratory parameters. It shows that group I patients had significantly lower hemoglobin level than group II. Otherwise, there were no significant differences between the studied groups as regards any of the laboratory parameters.

Table 4 represents a comparison between the studied groups as regards the finding in the initial endoscopic examination of the patients. It shows that the frequency of the non-variceal bleeding is 41.9% in the overall studied population. It also show that the grade of varices is significantly higher in group I than in group II (the frequency of grade III and IV is 41.3% and 25% in group I vs 4% and 1.3% in group II p<0.001). On the contrary, the PHG grad was higher among patients in group II (the frequency of PHG grade 3 was 20% in group II vs 14.4% in group II p=0.02). The frequency of ulcerating lesions in general was significantly higher in group II (24% vs 9.6% in group I p=0.008). The frequency of incompetent cardia was also significantly higher in group II (5.3% vs 0% in group I p=0.02 .(

The percentage of patients who underwent intervention during endoscopy is significantly higher among patients in group I than in group II (90.7% vs 2.9% p<0.001). The most common type of intervention done to group I patients is endoscopic band ligation (79.8%) followed by endoscopic variceal sclerotherapy (17.9%). Argon plasma coagulation was used only on 3 patients in the overall 179 population .

Figure 1 shows that the most common cause of non variceal bleeding is ulcerating lesions in general (24%), followed by PHG (17.3%), then post banding ulcers (14.6%). The frequencies of inflammatory lesions, erosions, polyps and gastric antral vascular ectasia were 5.3%, 6.6%, 5.3% and 4% successively. It also shows that in 24% of cases the cause of bleeding was obscure.

Table 5 represents a comparison between the studied groups as regards the various consequences and outcomes of the bleeding episode. It shows that there were no significant differences between the variceal and non variceal bleeding groups as regards length of ICU stay, plasma transfusion, reendoscopy or in ICU mortality. The variceal bleeding group had significantly higher RBC's transfusion needs (1.8±2 vs 1.1±1.4 in group II p=0.008). The variceal bleeding patients had significantly higher frequency of rebleeding than non variceal bleeding patients (5.8% vs 0% p=0.04).

Table 6 represents the correlation between in-ICU mortality in cirrhotic patients with acute upper GI bleeding with different patients' parameters. It shows that mortality is significantly correlated to WBC's count (r=0.2 p=0.002), Child's grad and score (r=0.217 p=0.003 and r= 0.16 p=0.03 successively). The mortality is also correlated to GBS (r=0.18 p=0.01). Moreover, it was proved to be related to both total and direct bilirubin level (r=0.4 p<0.001 for both). Mortality had no significant correlation to source of bleeding variceal or non variceal, rebleeding, intervention during endoscopy, grade of EV, or PHG, grade of encephalopathy, presence of focal lesion, portal vein patency, or use of NSAID's.

Table 1: Comparison between the studied groups as regards demographic data.

		Group I N=104	Group II N=75	X^2	P
Age (mean±SD)		58.96 ± 8.8	60.7 ± 9.6	T=1.26	0.21(NS)
Gender	Male	78 (75%)	54 (72%)	0.203	0.653 NS
	female	26 (25%)	21 (28%)		
Viral markers	HCV	102 (98.1%)	73 (97.3%)	0.11	0.74 NS
	HBV	1(1%)	3(4%)		
	Negative	2 (1.9%)	2 (2.7%)		
Comorbidities	DM	31 (29.8%)	14 (14.7%)	6.1	0.194 NS
	HTN	4 (3.8%)	6 (8%)		
	Both	5 (4.8%)	2 (2.7%)		
	others	1(1%)	3(4%)		
Bilharziasis		1 (1%)	2 (2.7%)	Fisher	0.381
Past history of upper GI bleeding		56 (53.8%)	54 (72%)	6.06	0.01 (S)
Past history of upper GI Endoscopy		52 (50%)	54 (72%)	8.73	0.003 (S)
Drugs	NSAIDs	3 (2.9%)	5 (6.7%)	Fisher	0.27 (NS)
	anticoagulant	3 (2.9%)	0 (0.0%)	Fisher	0.13(NS)
	OHD	14 (13.5%)	4 (5.3%)	Fisher	0.07(NS)
	Insulin	20 (19.2%)	11 (14.7%)	Fisher	0.23(NS)
	nitrate	2 (1.9%)	0 (0.0%)	Fisher	0.45(NS)
	Psychotherapy	0 (0.0%)	2 (2.7%)	Fisher	0.34(NS)

Table 2: Comparison between the studied groups as regards clinical and sonographic data and Child and Glassgow Blachford scores.

		Group I N=104	**Group II N=75**	**X^2**	**P**
Clinical data					
Jaundice		29 (27.9%)	31 (41.3%)	3.54	0.06 (NS)
LL edema		42 (40.4%0	34 (45.3%)	1.09	0.59(NS)
Encephalopathy	No	97 (93.3%)	71 (94.7%)	0.148	0.7 (NS)
	Grade I-II	7 (6.7%)	4 (5.3%)		
Sonographic data					
Hepatomegaly		28 (26.9%)	20 (26.7%)	0.001	0.97 (NS)
Splenomegally	Normal	6 (5.8%)	16 (21.3%)	10.7	0.01 S
	Mild	30 (28.8%)	20 (26.7%)		
	Moderate	49 (47.1%)	25 (33.3%)		
	Huge	19 (18.3%)	14 (18.7%)		
Ascites	No	48 (46.2%)	42 (56%)	3.41	0.346 NS
	Mild	33 (31.7)	16 (21.3%)		
	Moderate	20 (19.2%)	13 (17.3%)		
	tense	3 (2.9%)	4 (5.3%)		
Portal vein	Patent	20 (19.2%)	17 (22.7%)	0.314	0.57 (NS)
	Thrombosed	84 (80.8%)	58 (77.3%)		
Focal lesion		24 (23.1%)	18 (24%)	0.021	0.88 (NS)
Child and Glassgow Blatchford scores					
Child grade	A	32 (30.8%)	24 (32%)	0.82	0.66(NS)
	B	48 (46.2%)	30 (40%)		
	C	24 (23.1%)	21 (28%)		
Child score (points)		7.94 ± 1.97	7.84 ± 2.27	T=0.321	0.748 (NS)
GBS		14.1 ± 2.78	12.7 ± 3.15	T=3.11	0.002 (S)

Table 3: Comparison between studied groups as regards laboratory data at time of admission

	Group I N=104	Group II N=75	t-test\ MW*	P
Hemoglobin (g/dl)	8.6 ± 2.3	9.5 ± 2.5	2.5	0.02 (S)
WBC's (x10^3cells/µL)	7.7 ± 4.4	8.2 ± 4.4	0.644*	0.522 (NS)
Platelet (x10^3cells/µL)	119.5 ± 72.2	143.2 ± 102.9	1.02*	0.07 (NS)
Serum albumin (g/dl)	2.9 ± 0.64	3 ± 0.78	1.3	0.206 (NS)
Serum total bilirubin (mg/dl)	2.04 3.2	2.04 ± 2.5	0.307*	0.759 (NS)
Serum direct bilirubin (mg/dl)	1.23 ± 2.7	1.22 ± 1.8	1.2*	0.226 (NS)
ALT (IU/L)	59.4 ± 52.4	57.6 ± 98.6	1.7*	0.096 (NS)
AST(IU/L)	38.8 ± 42.6	33.5 ± 39.2	1.1*	0.278 (NS)
INR	1.6 ± 0.51	1.5 ± 0.43	1.7	0.09 (NS)
Serum creatinine (mg/dl)	1.15 ± 0.86	0.98 ± 0.58	1.3*	0.194 (NS)

Table 4: Comparison between the studied groups as regards the findings of initial endoscopy

		Group I N=104	Group II N=75	Test	P
Esophageal varices	Grade 0	10 (9.6%)	34 (45.3%)	97	<0.001 (HS)
	Grade I	4 (3.8%)	31 (41.3%)		
	Grade II	22 (21.1%)	6 (8%)		
	Grade III	42 (40.3%)	3 (4%)		
	Grade IV	26 (25%)	1 (1.3%)		
Fundal varices		19(18.3%)	12(16%)	0.15	0.6(NS)
Post banding ulcer		2(1.9%)	11(14.6%)	10	0.001(S)
Scelrosing ulcer		1(0.96%)	0(0%)	fisher	0.4(NS)
PHG	Grade 0	27 (26%)	33 (44%)	9	0.02(S)
	Grade I	30 (28.8%)	15 (20%)		
	Grade II	31 (29.8%)	13 (17.3%)		
	Grade III	15 (14.4%)	15 (20%)		
DE	Ulceration	10 (9.6%)	18 (24%)	6.8	0.008(S)
	Erosion	2 (1.9%)	5 (6.7%)	2.6	0.1(NS)
	Inflammation	6 (5.8%)	5 (6.7%)	0.06	0.8(NS)
	Polyps	2 (1.9%)	4 (5.3%)	1.5	0.2(NS)
	Hiatus hernia (HH)	1(0.96%)	5 (6.7%)	2.79	0.09(NS)
	Incompetent cardia	0(0%)	4 (5.3%)	fisher	0.02(S)
Intervention	Non	3 (2.9%)	68 (90.7%)	140	<0.001(HS
	EBL	83 (79.8%)	2 (2.7%)	103	<0.001(HS
	EVS (injection)	18 (17.9%)	1 (1.3%)	11.7	0.006(S)
	APC	0	3 (4%)	fisher	0.07(NS)
	Polypectomy	0	1 (1.3%)	fisher	0.4(NS)

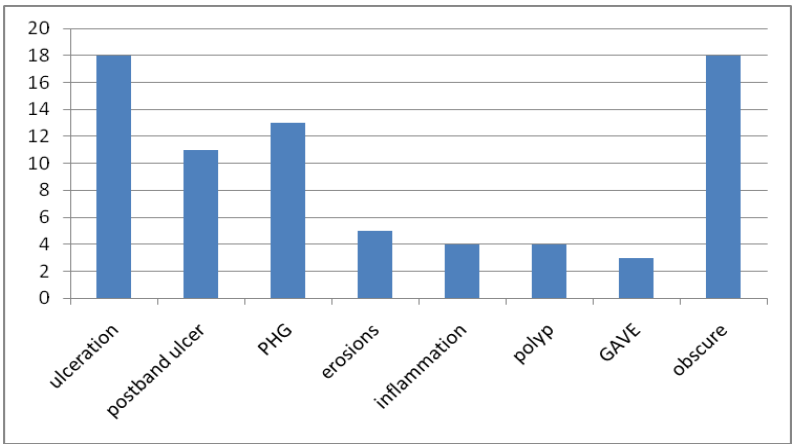

Figure 1: Causes of non-variceal bleeding in cirrhotic patients.

Table 5: Comparison between the studied groups as regards outcomes.

		Group I N=104	Group II N=75	MW	P
Days of ICU stay		5.14 ± 2.6	4.87 ± 2.8	1.17	0.244 (NS)
Transfusion needs	RBCs	1.8 ± 2.02	1.1 ± 1.48	2.64	0.008 (S)
	Plasma	2.8 ± 3.11	2.5 ± 2.6	1.002	0.316 (NS)
		N (%)	N (%)	X^2	P
Rebleeding		6 (5.8%)	0 (0.0%)	Fisher	0.04 (S)
Reendoscopy		4 (3.8%)	0 (0.0%)	Fisher	0.08 (NS)
Mortality		6 (5.8%)	2 (2.7%)	Fisher	0.322 (NS)

Table 6: Correlation between mortality and different variables in the study.

Variable	Mortality outcome	
	r	P
Age	-0.055	0.464(NS)
Encephalopathy grade	0.057	0.447(NS)
Ascites grade	0.072	0.338(NS)
Child score	0.217	0.003*(S)
Child grade	0.162	0.03*(S)
GBS	0.207	0.005*(S)
HB	-0.017	0.820(NS)
WBC	0.234	0.002*(S)
Platelet	0.072	0.882(NS)
Albumin	-0.181	0.01*(S)
Total bilirubin	0.469	<0.001**(HS)
Direct bilirubin	0.485	<0.001**(HS)
INR	0.064	0.353(NS)
Creatinine	0.461	<0.001**(HS)
BUN	0.330	<0.001**(HS)
AST	0.07	0.8(NS)
ALT	0.143	0.06(NS)
Grade of PHG	0.056	0.456(NS)
Grade of OV	0.034	0.655(NS)
Cause of bleeding (variceal/nonvariceal)	Ø=0.074	0.3(NS)
Focal lesion	Ø=0.13	0.06(NS)
Rebleeding	Ø=0.12	0.08(NS)
Intervention during endoscopy	Ø=0.06	0.3(NS)
Portal vein patency	Ø=0.09	0.21(NS)
Use of NSAIDs	Ø=0.04	0.4(NS)

DISCUSSION

AUGIB is either variceal or non-variceal. Many studies were designed to investigate the prediction and management of the variceal bleeding, but in contrast, fewer studies dealt with non-variceal bleeding. This study aims to shed light on the causes, clinical, laboratory and endoscopic findings, of non-variceal bleeding in comparison with variceal bleeding as well as the outcomes and predictors of mortality in AUGIB in cirrhotic patients.

Concerning demographic data analysis of our study, Male cirrhotic patients are more susceptible to UGIB than females (73.7% vs. 26.3%) and this finding comes in agreement with Odelowo et al. **[9]**, Morsy et al.**[10]**, Gabr et al.**[11]**. All these studies say that the males are more at risk of upper GI bleeding than females. However, there were no significant differences between the variceal and non-variceal bleeding as regards gender distribution denoting that the higher risk of bleeding in males is not associated

with higher risk of bleeding from a certain source. This disagrees with Eltoukhy and Issa **[12]** who said that the male patients were at higher risk of variceal bleeding. This disagreement may be because the latter study dealt only with variceal bleeding.

In our study, 97% of patients were HCV positive patients. This is comes in agreement with most of the Egyptian literature that dealt with cirrhosis and its sequelae because HCV is the most common cause of cirrhosis in Egypt **[10,11]**, while alcohol consumption represents the most common cause of cirrhosis in most of the foreign studies **[13,14,15]**.

In our study, we found that most patients with non-variceal bleeding 72% vs 53% in variceal bleeding group had previous history of upper GI bleeding and upper GI endoscopy. This can lead to the assumption that variceal bleeding is more likely to precede bleeding from other sources in patients with portal hypertension. This agrees with Bersci **[16]**, who said that esophageal varices have strong tendency to develop bleeding at HVPG <12 mmHg unlike other portal hypertension associated lesions, and that 30-50% of patients with esophageal varices bleed within one year of diagnosis.

Comparison between the studied groups as regards clinical findings revealed no significant differences as regards any of them except spleen size which was significantly larger among patients with variceal bleeding. This agrees with Umar et al.**[17]** who said that the large spleen size strongly predict the risk of variceal bleeding .

The comparison between the studied groups as regards laboratory parameters at time of admission showed that variceal bleeding group had significantly lower hemoglobin concentration and higher BUN than patients with non-variceal bleeding. Otherwise, there was no significant difference between both groups as regards the rest of laboratory parameters. This indicates that the variceal bleeding tend to be more severe and incapacitating than the non-variceal bleeding that it leads to a serious drop in hemoglobin concentration and more profound hemodynamic instability that can lead to affection of renal blood flow with the subsequent elevation of BUN and creatinine. We also found that the patients with variceal bleeding have significantly higher GBS at time of admission than those with non-variceal bleeding. This latter finding emphasizes that the variceal bleeding is usually more severe than non-variceal bleeding. As a consequence of these findings we also found that the RBC's transfusion needs in the variceal bleeding group was significantly higher than non-variceal bleeding group. This can be explained by the fact that the bleeding varix is a large valveless vein that bleeds seriously when it ruptures, while most of the non-variceal bleeding comes from minute bleeding points and small erosions. This finding comes in agreement with Cremers and Ribeiro **[18]**, who said that the suspicion of variceal bleeding puts the patient at a higher risk category that necessitates immediate intervention.

The frequency of non-variceal bleeding among cirrhotic patients in our study was 41.9%. This is comparable to what was found by Gonzalez-Gonzalez et al.**[19]** who found the frequency to be around 48%. Comparison between the studied groups as regards endoscopic findings revealed that, the patients with variceal bleeding had significantly larger varices size than those with non-variceal bleeding. On the other hand, patients with non-variceal bleeding had significantly higher grade of PHG than those with variceal bleeding. It also shows that the ulcerating lesions and incompetent cardia were significantly more frequent in patient with non-variceal bleeding.

When we compared the studied groups as regards endoscopic intervention and management of the bleeding source, we found that most of the patients with non-variceal bleeding had no intervention at all. The management of non-variceal bleeding depends only on the drug therapy and resuscitation. It also shows that most of the patients with variceal bleeding underwent EBL for their varices. EBL is the most common intervention done during upper GI endoscopy (47.5%), it was even done sometimes in patients with non-variceal bleeding. Although bleeding from PHG represents 17.3% of cases, the argon plasma coagulation was done only in 3 cases (1.7%) from the overall studied population.

The comparison between the studied groups as regards the outcomes of bleeding shows that, this great difference in endoscopic intervention seems to have no effect on the outcomes. Although most of non-variceal bleeding cases undergo no intervention, the frequency of rebleeding after non-variceal bleeding was significantly lower than after variceal bleeding. This means that the conservative measures and

drug therapy alone in most instances can control the non-variceal bleeding effectively and prevent rebleeding and that with the most successful intervention in variceal bleeding, rebleeding is still to be expected. This agrees with Thuluvath and Yoo **[20]** who said that management of PHG with hemospray or APC plays a minor role in the control of bleeding and that most of non-variceal bleeding respond well to vasoactive drugs alone and is less liable to recur.

The most common cause of non-variceal bleeding in our study is bleeding from peptic ulcer; gastric or duodenal (24%). This agrees with Gabr et al. **[11]** who said that peptic ulcer is the most common cause of non variceal bleeding in cirrhotic patients. The PHG comes second with a frequency of 17%. This agrees with Georjievski and Cappell **[21]** who said that the rate of bleeding from PHG ranges between 2 and 20%.

In 24% of cases the source of bleeding was unidentifiable. Koulaouzidis et al. **[22]** stated that when a cirrhotic patient suffers from obscure recurrent bleeding this can be due portal hypertensive enteropathy which is defined as changes in the small bowel due to portal hypertension. They are similar in nature to the PHG and can cause recurrent overt or occult bleeding. The prevalence of such a condition is now known to exceed 50% among cirrhotic patients due to the advances in the diagnostic tools such as enteroscopy and capsule endoscopy. In our study, endoscopic examination was done till the second part of the duodenum so the possibility of having a bleeding lesion lower than that level cannot be excluded.

Comparison between the studied groups as regards mortality revealed that there were no significant differences. This means that the source of bleeding itself had no impact on the mortality. Studying the correlation between mortality and the different patients' parameters revealed that there was significant positive correlation between mortality and Child's grade and score, bilirubin level and INR and a significant negative correlation with albumin level. This means that the deterioration of liver functions is an important predictor of mortality. This agrees with Gonzalez-Gonzalez et al. **[19]** who consider the deterioration I liver functions a strong predictor of mortality after upper GI bleeding. This also agrees with Hassanein et al. **[23]** who said that the in hospital mortality in cirrhotic patients with acute upper GI bleeding was related to MELD score and complications of liver decompensation.

There was also a significant positive correlation with GBS and renal function tests. This also means that the severity of bleeding episode and the resultant hemodynamic changes are very important predictors of mortality. This agrees with Jo et al.**[24]** who said that the severe hemodynamic instability is associated with higher mortality rate in patients with upper GI bleeding. This also agrees with Gonzalez-Gonzalez et al. **[19]** who said that elevated BUN was a predictor of mortality in patient with upper GI bleeding. The WBC's count was also significantly positively correlated with in ICU mortality. The rise of WBC's count in those patients is always associated with hospital acquired infection. This emphasizes the importance of the use of prophylactic antibiotics in cirrhotic patients with acute upper GI bleeding. The occurrence of sepsis can also lead to elevation of creatinine and BUN which were also proved to be correlated to mortality. This agrees with Morsy et al. **[10]** who said that the occurrence of infection is associated with higher mortality rate in patients with upper GI bleeding. This also agrees with Hou et al. **[25]** who said that the use of prophylactic antibiotics help decrease the mortality rates after acute upper GI bleeding especially in patients with severely decompensated liver disease.

The cause of bleeding, the portal vein patency, the presence of focal lesion, NSAIDS use, the size of varices, the grade of PHG as well as the intervention during endoscopy were all proved to have no relation to in-ICU mortality among the studied population.

To sum up, our study found out that the most important predictors of mortality in cirrhotic patients with upper GI bleeding are the severity of the bleeding episode, the severity of deterioration of liver functions and the rise of WBC's count in response to infection.

CONCLUSSION

The frequency of non-variceal bleeding among cirrhotic patients admitted with acute upper GI bleeding is 41.9%. Non-variceal bleeding was proved to have more favorable outcome than variceal bleeding even without endoscopic intervention. The mortality rate in patients with acute

upper GI bleeding is 4.5%. The degree of deterioration of liver functions and the severity of initial bleeding episode were the most important predictors of mortality.

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