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Prospective Analysis of Critically Ill Patients with Gastrointestinal Bleeding: An Observational Study

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ABSTRACT

Objective: The aim of this study was to prospectively evaluate the clinical and laboratory characteristics, as well as the mortality, of patients with gastrointestinal bleeding (GIB) in the intensive care unit (ICU).

Materials and Methods: This prospective study was conducted in the medical ICU. Patients diagnosed with GIB who were older than 18 years and hospitalized for at least 24 hours were included.

Results: A total of 86 patients were enrolled in the study. The mean age was 65±14 years. The Acute Physiology and Chronic Health Evaluation II (APACHE II) score was 13 (range: 2-41). The median Glasgow-Blatchford risk score was 12 (range: 2-18), and the Sequential Organ Failure Assessment (SOFA) score was 3 (range: 0-16). The most common causes of GIB were esophageal variceal bleeding (23%) and duodenal ulcer bleeding (16%). Chronic liver disease (CLD) (22%) and thrombocytopenia (21%) were identified as the most frequent predisposing factors for GIB. There was no significant difference in mortality between variceal bleeding (19%) and non-variceal bleeding (19%) (p=0.952). The APACHE II, SOFA, and Glasgow-Blatchford risk scores of non-surviving patients were statistically significantly higher than those of survivors (p=0.002, p<0.001, p<0.001 respectively). The mean platelet values were significantly higher in survivors (p<0.001). The ICU mortality rate was 19%.

Conclusion: This study demonstrated that the most common cause of GIB in the ICU was esophageal variceal bleeding, and the most frequent predisposing factor for GIB was CLD. The ICU mortality rate was not high in our study.

Keywords: Gastrointestinal bleeding, esophageal variceal bleeding, intensive care unit, mortality, thrombocytopenia.

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INTRODUCTION

Gastrointestinal bleeding (GIB) is a clinical condition affecting the upper esophagus and digestive tract up to the rectum. It is associated with high mortality rates and significant treatment costs. In many cases, it necessitates hospitalization and even follow-up in the intensive care unit (ICU).[1] The incidence of upper GIB is approximately 50–150 per 100,000, while the incidence of lower

GIB is around 20 per 100,000.[2] Although GIB can occur at any age, it is most commonly observed between the ages of 50 and 80 years.[3] In Türkiye, the most common causes of upper GIB are peptic ulcer disease (including duodenal, gastric, and anastomotic ulcers), erosive gastroduodenitis, and esophageal variceal bleeding.[4] Peptic ulcer disease accounts for 60% of all GIB cases in Türkiye.[4] Mortality associated with upper GIB is approximately 14%, whereas it is around 5% for lower GIB.[1]

Although several studies in the literature address the diagnosis, clinical follow-up, treatment, and mortality of patients with severe GIB requiring ICU admission, to the best of our knowledge, comprehensive research evaluating all these aspects is lacking. This study aimed to prospectively evaluate the clinical and laboratory characteristics, as well as the mortality, of patients with GIB admitted to the medical ICU.

KEY MESSAGES

- Esophageal varices and duodenal ulcer bleeding are the most common causes of gastrointestinal bleeding in patients admitted to intensive care.
- Chronic liver disease and thrombocytopenia are the most common predisposing factors.
- A significant proportion of intensive care unit (ICU) patients with gastrointestinal bleeding may face a mortal outcome.

MATERIALS AND METHODS

Patients

This prospective study was conducted in the Medical Intensive Care Unit at Erciyes University, Faculty of Medicine, between October 2018 and March 2020. The study adhered to the principles of the Declaration of Helsinki and received approval from the Erciyes University Clinical Research Ethics Committee (Date: 08.12.2017, Decision no: 2017/563). All participants were provided with both oral and written information about the study, and written informed consent was obtained from each participant or their next of kin in cases where the patient was unconscious.

Patients aged 18 years and older who were diagnosed with GIB and hospitalized for at least 24 hours were included in the study. Patients who developed GIB during their ICU stay were excluded.

Clinical data for all patients were collected from the hospital's electronic health records system and clinical files.

Data Collection

Upon ICU admission, the following information was recorded: gender, age, comorbidities, and reasons for ICU admission. Additionally, predisposing factors such as acute or chronic liver disease, thrombocytopenia (<150,000 µL), use of anti-aggregant or anticoagulant medications, coagulation factor deficiencies, advanced age, and other relevant factors were documented. Symptoms of GIB, the underlying cause, the Forrest classification, and the use of acid-suppressing medications were also recorded. All patients were further stratified using the Acute Physiology and Chronic Health Evaluation II (APACHE II), Glasgow-Blatchford, and Rockall scores at the time of admission.

The following clinical data were recorded daily during the hospitalization of the patients: laboratory values, respiratory support status, Glasgow Coma Scale (GCS) score, Sequential Organ Failure Assessment (SOFA) score, whether GIB was continuous, blood and/or blood product transfusions, presence of sepsis and shock, administered fluids, other medications, deep vein thrombosis (DVT) prophylaxis, nutrition regimen, renal replacement therapy, presence of oliguria, development of acute kidney injury (AKI), and Kidney Disease Improving Global Outcomes (KDIGO) score.[5]

The final status of the patients (death, discharge, or transfer to another service), length of ICU stay, type of intervention for GIB (endoscopy, surgery, or interventional radiology), and the ICU mortality rate were also documented.

Statistical Analyses

All statistical analyses were performed using SPSS for Windows version 22.0 software (SPSS Inc., Chicago, IL). The One-Sample Kolmogorov-Smirnov Test was used to assess the normal distribution of data. Normally distributed data were reported as mean (standard deviation, SD), while non-normally distributed data were reported as median (range: minimum-maximum). Qualitative data were presented as numbers and percentages. Independent qualitative data were analyzed using the Chi-Square test. The means between groups were compared using the independent sample t-test for data conforming to a normal distribution, while the Mann-Whitney U test was used for data not conforming to a normal distribution. A p-value of less than 0.05 was considered statistically significant.

RESULTS

A total of 1,124 patients were admitted to the medical ICU between October 2018 and March 2020. Of these, 86 patients (7.6%) were hospitalized due to GIB, with 60% being male. The mean age of the patients was 65±14 years (range: 27–91). The median APACHE II score was 13 (range: 2–41). The mean GCS score on admission was 13±4, and the median SOFA score was 3 (range: 0–16). Regarding comorbidities, 44% of the patients had hypertension (HT), 27% had diabetes mellitus (DM), 27% had chronic liver disease (CLD), and 23% had coronary

artery disease (CAD). Among the symptoms, 36% of patients presented with hematemesis alone, 24% with hematochezia alone, and 20% with melena alone. Based on endoscopy results, 23% of patients had esophageal variceal bleeding, 16% had duodenal ulcers, 11% had gastric ulcers, 6% had esophageal ulcer, and 20% had no no identified bleeding focus (peptic ulcer disease was identified in 33% of the patients). Additionally, four patients were diagnosed with gastric cancer. Of these, two patients experienced bleeding from the esophagus, one from the anastomosis line, and one from a gastric lesion. In terms of treatment, 91% of patients received omeprazole, and 9% received pantoprazole. Omeprazole and pantoprazole were administered as continuous infusions in 92% of the patients, while 8% received them as intravenous (IV) bolus injections. Upon first admission, the median Glasgow-Blatchford risk score was 12 (range: 2–18), and the median Rockall risk score was 6 (range: 1–10). The demographic and clinical characteristics of the patients are detailed in Table 1.

When the factors triggering GIB were evaluated, CLD was identified in 22% of the patients, thrombocytopenia in 21%, anti-aggregant use in 21%, and anticoagulant use in 11% (Fig. 1).

The esophagus was the source of bleeding in 35% of the patients, while bleeding from the stomach was detected in 24%. In 19% of the patients, no bleeding focus was identified (Fig. 2).

When the 14-day averages of laboratory values were assessed, the mean hemoglobin (Hb) level was 8.3±1.9 g/dL (Table 2).

During the ICU stay, patients received 212 units of erythrocyte suspension (ES) (2.4 units per patient), 70 units of thrombocyte suspension (TS) (0.8 units per patient), 65 units of fresh frozen plasma (FFP) (0.7 units per patient), and four units of cryoprecipitate (0.04 units per patient).

It was observed that 30% of the patients required mechanical ventilation, 19% required renal replacement therapy, and 28% required vasopressors. Additionally, 24% of the patients developed sepsis, 19% experienced septic shock, 9% had hypovolemic shock, and 28% developed AKI (Table 3). Among the 16 patients who required renal replacement therapy, 13 were diagnosed with AKI, and three had chronic kidney disease (CKD).

The median length of ICU stay was 5 days (range: 2–28) (Table 3).

Endoscopy was performed on 91% of the patients for diagnostic purposes. Endoscopic interventions were conducted in 42% of the patients. Esophageal variceal band ligation was performed in 15% of the patients, sclerotherapy in 10%, sclerotherapy combined with bipolar coagulation in 6%, endoclips in 3%, sclerotherapy combined with endoclips in 3%, and sclerotherapy combined with endoclips and bipolar coagulation in 2%. Argon plasma coagulation was performed in 1% of all patients. Additionally, 2% of the patients underwent surgery, and 1% underwent interventional radiology (Table 3). The ICU mortality rate was 19%.

Table 1. Demographic and clinical characteristics of patients with gastrointestinal bleeding (GIB)

Parameter	Value	
	n	%
Age (years), mean±SD	65±14	
GCS, mean±SD	13±3	
APACHE II score, (min-max)	13 (2–41)	
SOFA score, (min-max)	3 (0–16)	
Gender		
Male	52	60
Female	34	40
Comorbid disease		
Hypertension	38	44
Diabetes mellitus	23	27
Chronic liver disease	23	27
Coronary artery disease	20	23
Cancer	18	21
Intensive care arrival location		
Emergency room	47	55
Service	25	29
Other ICU	9	11
Outer center	5	6
Symptoms		
Hematemesis	31	36
Hematochezia	21	24
Melena	17	20
Diagnoses		
Esophageal variceal bleeding	20	23
No focus	17	20
Duodenal ulcer	14	16
Drug administration method		
Infusion	79	92
IV Push	7	8
Glasgow-blatchford risk score (min-max)	12 (2–18)	
Rockall risk score (min-max)	6 (1–10)	

SD: Standard deviation; GCS: Glasgow coma scale; APACHE II: Acute physiology and chronic health evaluation II; SOFA: Sequential organ failure assessment; ICU: Intensive care unit; IV: Intravenous.

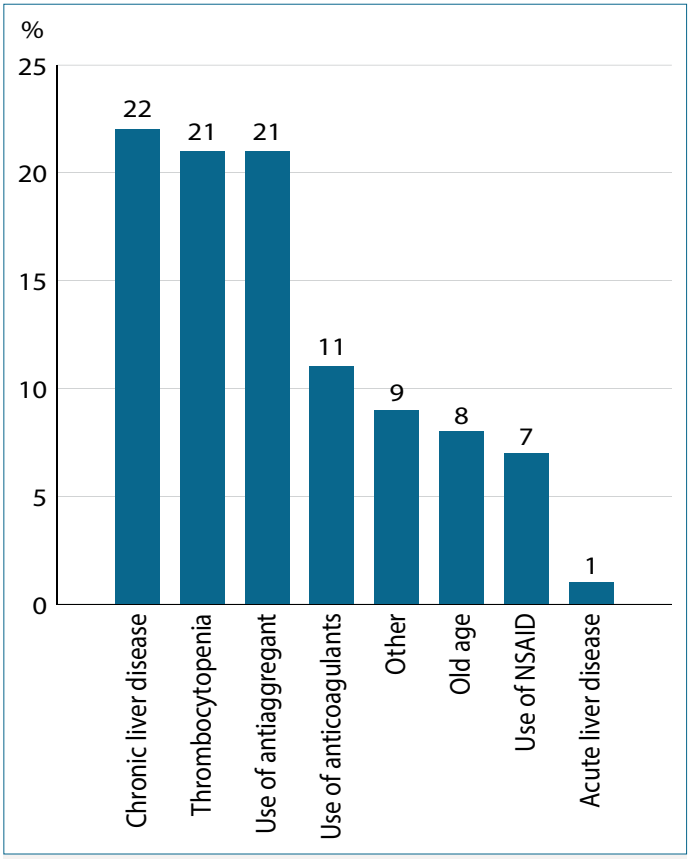

Figure 1. Predisposing factors for inpatients with gastrointestinal bleeding in the intensive care unit.

NSAID: Non-steroidal anti-inflammatory drugs.

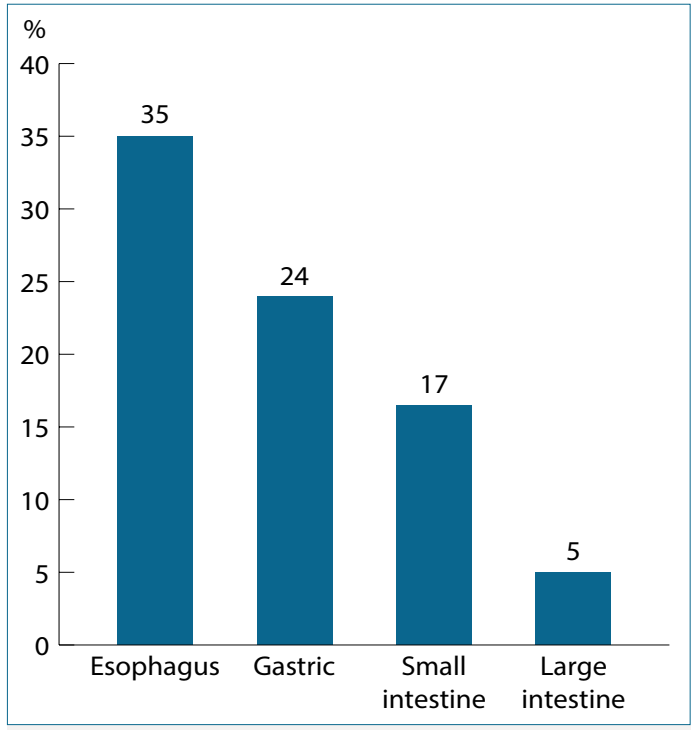

Figure 2. Bleeding foci of the patients: 30 patients had the esophagus as the focus, 21 patients had the stomach as the focus, 15 patients had the small intestine as the focus, four patients had bleeding from the large intestine, and 16 patients had other factors contributing to bleeding.

Outcomes of Patients with Variceal and Non-variceal Bleeding

For subgroup analyses, the patients were divided into two groups based on their diagnosis: variceal bleeding and non-variceal bleeding. Variceal bleeding was present in 21 patients (24%), while non-variceal bleeding was observed in 65 patients (76%). The median SOFA score in patients with variceal bleeding was statistically significantly higher than in those with non-variceal bleeding. The median SOFA liver and coagulation score for patients with variceal bleeding was 4 (range: 2–10) (Table 4).

Patients with variceal bleeding received 37 units of ES (1.7 units per patient), seven units of TS (0.3 units per patient), and 17 units of FFP (0.8 units per patient). In comparison, patients with non-variceal bleeding received 175 units of ES (2.6 units per patient), 63 units of TS (0.9 units per patient), and 48 units of FFP (0.7 units per patient).

The mean platelet (PLT) count during ICU hospitalization was 98×10^3/μL (range: 30–414×10^3/μL) in patients with variceal bleeding and 171×10^3/μL (range: 2–697×10^3/μL) in patients with non-variceal bleeding (p=0.006).

The average ICU length of stay for both groups was five days (p=0.497). The mortality rate was similar in both groups, at 19% (p=0.952) (Table 4).

Outcomes of Survivors and Non-survivors

Sixteen patients died in the ICU (19%). The median APACHE II and SOFA scores of survivors (APACHE II: 13, SOFA: 2) were statistically significantly lower than those of non-survivors (APACHE II: 18, SOFA: 6) (p<0.05).

At the first visit, the median Glasgow-Blatchford risk score was significantly lower in survivors compared to non-survivors (11 vs. 15) (p<0.001).

All patients who died required mechanical ventilation, compared to 14% of survivors (p<0.001). Sepsis developed in 11% of survivors and 81% of non-survivors (p<0.001).

Among survivors, septic shock developed in 4%, hypovolemic shock in 7%, and vasopressor s were required in 13%. In

Table 2. Intensive care laboratory values of patients with gastrointestinal bleeding (GIB)

Parameter	Value
Sodium, mmol/L (min-max)	140 (120–154)
Potassium, mmol/L (min-max)	4.3 (2.8–6.3)
BUN, mg/dL (min-max)	38.4 (8–103)
Creatinine, mg/dL (min-max)	1 (0.1–4.8)
Glucose, mg/dL (min-max)	130 (57–459)
Calcium, mg/dL (min-max)	7.8 (6–9.5)
Total bilirubin, mg/dL (min-max)	0.8 (0.2–8.6)
Direct bilirubin, mg/dL (min-max)	0.3 (0.06–6)
AST, U/L (min-max)	20 (6–540)
ALT, U/L (min-max)	14 (3–322)
Total protein, g/dL (min-max)	5.4 (2.4–8.8)
Albumin, g/dL (min-max)	2.8 (1–4.1)
Lactate (min-max)	1.57 (0.4–14.7)
HB, g/dL 1st day ±SD	8.6±1.6
HB, g/dL 7th day ±SD	8.7±1.1
HB, g/dL 14th day ±SD	8.3±1.9
WBC, 10^3/µL 1st day (min-max)	9.7 (0.2–31)
WBC, 10^3/µL 7th day (min-max)	7.1 (0.01–37.2)
WBC, 10^3/µL 14th day (min-max)	8 (5.3–9.3)
PLT, 10^3/µL 1st day (min-max)	159 (2–697)
PLT, 10^3/µL 7th day (min-max)	106 (18–414)
PLT, 10^3/µL 14th day (min-max)	135 (96–338)
APTT, 1st day ±SD	31.7±14.7
APTT, 7th day ±SD	42.4±23.6
APTT, 14th day ±SD	29.9±2.2
PT, 1st day ±SD	16.1±8.1
PT, 7th day ±SD	14.3±2
PT, 14th day ±SD	13.7±2
INR, 1st day ±SD	1.3±0.7
INR, 7th day ±SD	1.1±0.1
INR, 14th day ±SD	1.1±0.1

BUN: Blood urea nitrogen; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; HB: Hemoglobin; SD: Standard deviation; WBC: White blood cell count; PLT: Platelet; APTT: Activated partial thromboplastin time; PT: Prothrombin time; INR: International normalized ratios.

contrast, 81% of non-survivors developed septic shock, 19% experienced hypovolemic shock, and 94% required vasopressor treatment. The differences in septic shock and vasopressor requirements were statistically significant, with both being significantly higher in non-survivors (p<0.001).

Table 3. Clinical characteristics of patients and interventions applied

Parameter	Value	
	n	%
Deep vein thrombosis prophylaxis	85	99
Mechanical ventilator need	26	30
Vasopressor need	24	28
Acute kidney injury	24	28
Sepsis	21	24
Septic shock	16	19
Renal replacement therapy need	16	19
Hypovolemic shock	8	9
Length of ICU stay	5 (2–28)	
Type of intervention in GIB		
None	47	55
Endoscopy	36	42
Surgery	2	2
Interventional radiology	1	1

ICU: Intensive care unit; GIB: Gastrointestinal bleeding.

AKI developed in 17% of survivors and 75% of non-survivors. Renal replacement therapy was required in 10% of survivors and 56% of non-survivors. Both differences were statistically significant (p<0.001).

The surviving patients received 128 units of ES (1.8 units per patient), 34 units of TS (0.4 units per patient), and 30 units of FFP (0.4 units per patient). In contrast, patients who died received 84 units of ES (5.2 units per patient), 36 units of TS (2.2 units per patient), and 35 units of FFP (2.1 units per patient).

The mean PLT count measured at ICU admission was significantly higher in surviving patients compared to those who died (171×10^3/µL vs. 83×10^3/µL, respectively) (p=0.001).

The details of surviving and deceased patients are presented in Table 5.

DISCUSSION

The etiology, clinical follow-up, and treatment processes of patients hospitalized in the ICU due to GIB were evaluated in this prospective study.

The male sex ratio was higher in this study. Similarly, in a retrospective study by Ozkan Kuscu et al.[6] involving 176 patients with upper GIB, the male sex ratio was higher (66.5%), consistent with our findings.

Table 4. Comparison of demographic and clinical data between variceal and non-variceal gastrointestinal bleeding (GIB)

Variable	Variceal bleeding (n=21)		Non-variceal bleeding (n=65)		p
	n	%	n	%	
Age (years) ±SD	67±12		65±15		0.433
Gender					
Male	10	48	42	65	0.166
Female	11	52	23	35	
APACHE II score (min-max)	15 (2–41)		13 (2–40)		0.328
SOFA score (min-max)	4 (2–10)		2 (0–16)		**0.001**
Glasgow-blatchford risk score (min-max)	13 (7–16)		11 (2–18)		0.192
Rockall risk score (min-max)	7 (4–9)		6 (1–10)		0.067
Mechanical ventilator need	9	43	17	26	0.147
Sepsis	5	24	16	25	0.940
Septic shock	3	14	13	20	0.559
Hypovolemic shock	3	14	5	8	0.366
Vasopressor need	5	24	19	30	0.630
Acute kidney injury	6	29	18	28	0.938
Renal replacement need	2	10	14	22	0.219
Mean PLT (10^3/μL) values measured at admission (min-max)	98 (30–414)		171 (2–697)		**0.006**
Mean HB (g/dL) values measured at admission ±SD	8.29±1.38		8.82±1.77		0.215
Mean APTT values measured at admission ±SD	29.1±7.30		32.5±16.4		0.375
Mean PT values measured at admission ±SD	16.8±3.66		15.8±9.19		0.615
Mean INR values measured at admission ±SD	1.45±0.37		1.34±0.81		0.572
Length of ICU stay (days) (min-max)	5 (2–14)		5 (2–28)		0.497
ICU mortality	4	19	12	19	0.952

SD: Standard deviation; APACHE II: Acute physiology and chronic health evaluation II; SOFA: Sequential organ failure assessment; PLT: Platelet; HB: Hemoglobin; APTT: Activated partial thromboplastin time; PT: Prothrombin time; INR: International normalized ratio; ICU: Intensive care unit.

In this study, the most common etiologies of GIB were esophageal variceal bleeding and duodenal ulcers. A review of the literature shows that the most frequently reported causes are peptic ulcers, esophageal variceal bleeding,[7–9] and bleeding localized in the stomach and esophagus regions. In this study, since all gastric diseases were categorized under separate sub-headings, duodenal ulcers may have been observed at a higher rate compared to other gastric diseases. The most common complaints among patients were hematemesis and hematochezia. In a study conducted by Laine et al.,[7] it was demonstrated that patients with GIB most frequently present with hematemesis and melena. However, the present study revealed that patients presenting with hematochezia and hematemesis, especially hematochezia, were more likely to be admitted with significant bleeding.[7] This may have necessitated the admission of these patients to the ICU. Patients presenting with melena were admitted to the hospital later, and their mortality rate at admission was observed to be lower. In contrast, hematemesis and hematochezia appear to be associated with higher mortality rates.[7]

In this study, the most common comorbid conditions accompanying GIB were HT and DM, followed by CAD and CLD. Additionally, CLD, thrombocytopenia, and anti-aggregant use were identified as the most common triggering factors for GIB. Thrombocytopenia was also commonly observed in patients with CLD. However, the causes of thrombocytopenia in this study were not specifically evaluated. According to a multicenter observational study conducted on 619 patients requiring endoscopic treatment for upper GIB, 44% of the patients were receiving at least one antithrombotic drug

Table 5. Comparison of demographic and clinical data between surviving and non-surviving patients

Variable	Surviving (n=70)		Non-surviving (n=16)		p
	n	%	n	%	
Age (years) ±SD	62±12		66±14		0.276
Gender					
Male	42	60	10	63	0.854
Female	28	40	6	37	
APACHE II score (min-max)	13 (2–39)		18 (9–41)		**0.002**
SOFA score (min-max)	2 (0–9)		6 (2–16)		**<0.001**
Glasgow-Blatchford risk score (min-max)	11 (2–16)		15 (9–18)		**<0.001**
Rockall risk score (min-max)	6 (1–10)		6.5 (2–9)		0.124
Mechanical ventilator need	10	14	16	100	**<0.001**
Sepsis	8	11	13	81	**<0.001**
Septic shock	3	4	13	81	**<0.001**
Hypovolemic shock	5	7	3	19	0.149
Vasopressor need	9	13	15	94	**<0.001**
Acute kidney injury	12	17	12	75	**<0.001**
Renal replacement need	7	10	9	56	**<0.001**
Average PLT (10^3/μL) values (min-max) measured at admission	171 (3–697)		83 (2–414)		**0.001**
Length of ICU stay (days) (min-max)	5 (2–18)		5 (2–28)		0.964

SD: Standard deviation; APACHE II: Acute physiology and chronic health evaluation II; SOFA: Sequential organ failure assessment; PLT: Platelet; ICU: Intensive care unit.

at the time of admission, and 25% were on more than one antithrombotic drug.[8] Although these drugs are recognized as a risk factor for upper GIB, there is no evidence that their use worsens bleeding outcomes.[8–11] CLD triggers GIB by causing esophageal variceal bleeding, disrupting coagulation factors, and impairing thrombocyte shape and function.

In the present study, low Hb levels observed in patients with GIB on the first day indicate significant blood loss due to bleeding. The most commonly used blood products in this study were ES and TS. Surviving patients received 128 units of ES, while patients who died received 84 units of ES. A meta-analysis involving 1,965 patients with upper GIB reported that restrictive transfusion was associated with lower mortality and reduced re-bleeding rates.[12] However, the significantly higher number of surviving patients compared to deceased patients in this study may be related to the larger volume of blood transfusions administered to the surviving patients.

The average length of ICU hospitalization for patients was five days. Compression stockings were applied to all patients, except one, for DVT prophylaxis during their hospitalization. This study did not evaluate the development of thromboembolic events. One study demonstrated that intermittent pneumatic compression is associated with a lower incidence of venous thromboembolism in intensive care patients.[13] Similarly, a study conducted by Geerts et al.[14] recommended mechanical thromboembolism prophylaxis as the sole approach for critically ill intensive care patients in whom anticoagulant use is contraindicated due to a high risk of bleeding.

Cause-oriented treatment is critical for patients with GIB. Almost all patients included in this study underwent diagnostic endoscopy, and nearly half received endoscopic treatment. Esophageal variceal band ligation and sclerotherapy were used as treatment methods. Numerous studies have shown that band ligation, a treatment for esophageal variceal bleeding, has fewer side effects and lower re-bleeding rates compared to sclerotherapy.[15,16]

In this study, nearly all patients were administered proton pump inhibitor (PPI) drugs as an infusion. A review of the literature indicates that a randomized controlled study demonstrated that administering an 80 mg bolus of PPI after successful endoscopy, followed by an 8 mg/hour infusion for three days, reduced both re-bleeding and mortality

rates.[17] The same study also found that intermittent IV or oral PPI administration reduced re-bleeding but did not lower mortality rates.[17] Many guidelines supporting these findings recommend that PPI treatment be administered as an IV bolus followed by an infusion.[18,19]

In our study, the intensive care mortality rate was 19% among 86 patients with GIB. In a retrospective study involving 176 intensive care unit patients with upper GIB conducted by Ozkan Kuscu et al.,[6] the intensive care unit mortality rate was reported as 52.3%. In the same study, the patients' average APACHE II score was 30±9.5. A study by Cook et al.,[20] which examined 1,666 mechanically ventilated patients, reported that GIB developed in 59 patients, with a mortality rate of 45.8%. In that study, the patients' average APACHE II score was 22.9±8.6, and the average ICU hospitalization time was 26 days.[20] In our study, the median APACHE II score of patients was 13. The lower ICU mortality rate in our study may be attributed to the lower APACHE II scores and the reduced need for mechanical ventilation. In another study, Clason et al.[21] prospectively evaluated 326 patients with acute upper gastrointestinal (GI) bleeding to investigate factors predicting mortality. They concluded that advanced age, clinical shock at admission, and re-bleeding episodes after admission appeared to be independently significant predictors of mortality. Similarly, in the present study, the need for vasopressors was 94%, the rate of septic shock development was 81%, and the mean age of the patients was 66±14 years. Furthermore, in our study, the APACHE II, SOFA, and Glasgow-Blatchford scores, which predict mortality, were significantly higher in patients who died. In previous studies that included all types of GIB, the mortality rate has been reported to be approximately 10%.[22–24] As shown in these studies, the mortality rate is lower in patients who do not require intensive care. Additionally, AKI developed in 75% of the patients who died, and renal replacement therapy was required in 56% of these patients. These patients experienced multiple organ failure due to shock.

The length of ICU stay for patients with variceal bleeding was five days, and the mortality rate was 19%. Since patients with variceal bleeding typically have CLD or cirrhosis, the mortality rate may be higher. A study conducted by Majeed et al.[25] reported that the average age of patients with variceal bleeding was 56.4 years, the average ICU hospitalization time was 2.2 days, and the mortality rate was 13%. In that study, the median APACHE II score of patients with variceal bleeding was 15. There are limited studies on this topic in the literature. Majeed et al.[25] also reported a mean APACHE III score of 16.9 (range: 7–41) for patients with variceal bleeding. Compared to their findings, the higher mortality rate observed in our study may be attributed to the advanced age of our patients and their longer ICU stays.

When SOFA scores were compared between patients with variceal and non-variceal bleeding, it was observed that patients with variceal bleeding had higher scores. The SOFA score is evaluated daily in the ICU to monitor organ failure and consists of six components: PaO_2/FiO_2 ratio, hypotension, bilirubin, thrombocyte count, creatinine, and GCS. Two of these parameters, PLT count and bilirubin, are indicative of CLD. In this study, the median "SOFA liver and coagulation score" was 2.5 (range: 1–5), and the median total SOFA score for patients with variceal bleeding was 4 (range: 2–10). This likely explains the higher SOFA scores in patients with variceal bleeding, which are associated with CLD. We may also state that patients with variceal bleeding are more prone to mortality. However, in our study, the mortality rates between the two groups were similar.

Patients with variceal bleeding required mechanical ventilation more frequently than those with non-variceal bleeding. A separate study reported that 43% of patients with variceal bleeding required mechanical ventilation, a result consistent with our findings.[25] This supports the observation that variceal bleeding causes greater clinical deterioration and necessitates intubation to protect the airway in these patients. Sepsis development rates were similar between the two patient groups; however, septic shock was more common in patients with non-variceal bleeding, while hypovolemic shock was more prevalent in patients with variceal bleeding. The severe blood loss experienced by patients with variceal bleeding may have contributed to this situation.

Both patient groups had similar Hb levels at admission, but PLT counts were lower in patients with variceal bleeding. In the study conducted by Majeed et al.,[25] the mean PLT count in patients with variceal bleeding was $95.9\pm70.60\times10^3/\mu L$. Similarly, in our study, the mean PLT count was 98 (range: 30–414)$\times10^3/\mu L$. Since most patients with variceal bleeding also have CLD, their PLT counts may be lower compared to patients with non-variceal bleeding.

CONCLUSION

In conclusion, among patients with GIB admitted to the ICU, the most common symptom was hematemesis, with esophageal variceal bleeding and peptic ulcer being the leading causes. The most common trigger for GIB was CLD, and the most frequent location of GIB was the esophagus. It was observed that patients with variceal bleeding had higher SOFA scores and lower PLT counts compared to those with non-variceal bleeding. The APACHE II, SOFA, and Glasgow-Blatchford risk scores were higher in patients who died, while their PLT counts were lower than those in surviving patients. Patients who died had a greater need for mechanical ventilation, sepsis, septic shock, vasopressors, AKI, and renal replacement therapy compared to those who survived. The main limitations of this study include its single-center design and the absence of a power analysis.

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