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Original Article

Frequency and Etiology of Hematemesis in Patients Presenting to the Emergency Department

Dr. Moazzam Farooq[1], Dr. Farhan Ali[2], Afifa Nighat[3], Dr. Muhammad Haseeb[4], Dr. Leena Siddiqui[5], Arshad Ullah[6], Dr. Wali Ullah[7]

[1] Senior House Officer in Rheumatology, University Hospital Limerick, Limerick, Ireland
[2] Medical Officer, District Headquarters Hospital Jhelum, Jhelum, Pakistan
[3] Specialty Doctor, Independent Researcher, United Kingdom
[4] Medical Officer, Pakistan Aeronautical Complex Hospital, Kamra, Pakistan
[5] Consultant Medicine, Jinnah Postgraduate Medical Centre, Pakistan
[6] Resident Physician Internal Medicine, Ayub Teaching Hospital, Abbottabad, Pakistan
[7] PGR Paeds Medicine, Lady Reading Hospital, Peshawar, Pakistan

Corresponding author: Dr Moazzam Farooq, moazzamfarooq11@gmail.com

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ABSTRACT

Background: Hematemesis is an important manifestation of upper gastrointestinal bleeding requiring prompt stabilization and identification of the bleeding source because management differs substantially between variceal and non-variceal causes. Objective: To determine the frequency and etiological distribution of hematemesis among patients presenting to the emergency department. Methods: This prospective observational study included 100 consecutive patients aged 15–80 years presenting with hematemesis to the Emergency Department of Lady Reading Hospital–Medical Teaching Institution, Peshawar, Pakistan, from 1 January to 30 June 2026. Clinical characteristics and relevant laboratory findings were recorded, and upper gastrointestinal endoscopy was performed after stabilization to establish the bleeding etiology. Data were analysed using IBM SPSS Statistics version 26.0 and summarized descriptively. Results: The mean age was 46.8 ± 16.7 years, with an observed range of 18–80 years; 68 (68.0%) participants were male. Esophageal varices were the most frequent etiology, occurring in 42 (42.0%) patients, followed by peptic ulcer disease in 25 (25.0%), erosive gastritis in 12 (12.0%), gastric varices in 8 (8.0%), Mallory–Weiss tear in 5 (5.0%), esophagitis in 4 (4.0%), and upper gastrointestinal malignancy in 4 (4.0%). Variceal and non-variceal causes each accounted for 50 (50.0%) cases. Chronic liver disease was documented in 55 (55.0%) patients, NSAID use in 28 (28.0%), and previous upper gastrointestinal bleeding in 21 (21.0%). The mean interval from emergency presentation to endoscopy was 18.6 ± 7.4 hours. Conclusion: Variceal bleeding, particularly esophageal varices, and peptic ulcer disease constituted the principal etiologies of hematemesis in this emergency department population. Recognition of the local etiological pattern and timely endoscopic assessment are important components of appropriate clinical management. Keywords: Hematemesis; Upper Gastrointestinal Bleeding; Esophageal Varices; Peptic Ulcer Disease; Gastrointestinal Endoscopy; Emergency Department.

INTRODUCTION

Hematemesis, defined as the vomiting of fresh blood or coffee-ground material originating from the upper gastrointestinal tract, is an important manifestation of acute upper gastrointestinal bleeding (UGIB) and frequently requires emergency assessment and treatment. Acute UGIB remains a substantial cause of hospital admission and is associated with clinically important morbidity and mortality, particularly among older patients, individuals with major comorbidities, and those presenting with hemodynamic instability. Initial management therefore requires prompt assessment of circulatory status, appropriate resuscitation, risk stratification, and timely identification of the bleeding source (1,2).

The etiological spectrum of hematemesis includes both non-variceal and variceal lesions. Peptic ulcer disease remains a major cause of non-variceal UGIB, while erosive gastroduodenal disease, esophagitis, Mallory–Weiss tears, malignant lesions, and vascular abnormalities constitute other recognized sources. Exposure to non-steroidal anti-inflammatory drugs, antiplatelet agents, and anticoagulants may further

increase the risk of gastrointestinal mucosal injury or clinically significant hemorrhage (2–4). In contrast, esophageal and gastric varices arise primarily in the setting of portal hypertension and may produce abrupt and severe bleeding, requiring management pathways that differ substantially from those used for non-variceal hemorrhage (5).

The relative contribution of these causes varies across populations because the underlying burden of chronic liver disease, portal hypertension, peptic ulcer disease, medication exposure, demographic characteristics, and access to gastrointestinal services differs between healthcare settings. Variceal hemorrhage consequently assumes particular clinical importance in populations with a substantial burden of chronic liver disease, whereas peptic ulcer disease continues to account for a major proportion of non-variceal bleeding internationally (3–6). Distinguishing these etiologies is clinically relevant because pharmacological treatment, endoscopic intervention, prevention of recurrent bleeding, and subsequent disease-specific management depend on the underlying source (3,5,6).

Institution-specific information regarding the etiological distribution of hematemesis can support emergency diagnostic pathways, prioritization of endoscopic services, and planning of appropriate management strategies. Such information is particularly relevant where the relative frequencies of variceal and non-variceal hemorrhage may differ from patterns reported in other populations (5,6). The present study therefore aimed to determine the frequency and etiological distribution of hematemesis among patients presenting to the Emergency Department of Lady Reading Hospital–Medical Teaching Institution, Peshawar.

MATERIALS AND METHODS

This prospective observational study was conducted in the Emergency Department of Lady Reading Hospital–Medical Teaching Institution (LRH-MTI), Peshawar, Pakistan, from 1 January 2026 to 30 June 2026. The study population comprised patients presenting to the emergency department with hematemesis during the study period. Ethical approval was obtained from the Institutional Research and Ethics Board of Lady Reading Hospital–MTI, Peshawar (Approval No. 245/LRH/MTI; dated 15 December 2025). Written informed consent was obtained from each participant or, where applicable, a legally authorized attendant before enrollment.

Patients aged 15–80 years presenting with hematemesis were eligible for inclusion. Hematemesis was operationally defined as vomiting of fresh blood or coffee-ground material considered clinically consistent with upper gastrointestinal bleeding. Patients of either sex who fulfilled the eligibility criteria and provided consent were enrolled consecutively using a non-probability consecutive sampling technique until 100 participants had been included. Patients were excluded if they were younger than 15 years or older than 80 years, declined participation or did not have consent provided by an authorized attendant where required, had isolated hemoptysis or epistaxis mistaken for hematemesis, had bleeding attributable to major trauma involving the upper gastrointestinal tract, or had incomplete clinical records or no determinable final diagnosis.

Following presentation, patients underwent initial clinical assessment and stabilization according to the emergency department's routine management procedures. Data were recorded on a structured data-collection form and included age, sex, presenting clinical characteristics, history of chronic liver disease, previous episodes of upper gastrointestinal bleeding, use of non-steroidal anti-inflammatory drugs, antiplatelet or anticoagulant medications, and relevant comorbidities including diabetes mellitus, hypertension, chronic kidney disease, and malignancy.

Laboratory assessment included complete blood count, blood grouping and cross-matching, coagulation profile, liver function tests, renal function tests, and additional investigations when clinically indicated. Upper gastrointestinal endoscopy was undertaken after hemodynamic stabilization. Endoscopy is central to establishing the source of acute UGIB and permits differentiation between variceal and non-variceal

sources that require distinct therapeutic approaches (3,5). The final etiology was determined from endoscopic findings together with relevant clinical, laboratory, and radiological information documented by the treating gastroenterologist. Etiological categories included esophageal varices, gastric varices, peptic ulcer disease involving a gastric or duodenal ulcer, erosive gastritis, esophagitis, Mallory–Weiss tear, upper gastrointestinal malignancy, vascular lesions, and other identified causes.

The primary outcome was the frequency and proportional distribution of the identified etiologies of hematemesis. Secondary descriptive outcomes included the distribution of etiologies according to age and sex. Additional clinical characteristics included history of chronic liver disease, NSAID exposure, previous UGIB, and the interval between emergency department presentation and upper gastrointestinal endoscopy.

Data were entered and analysed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were summarized as mean ± standard deviation when appropriately represented by a normal distribution and as median with interquartile range when appropriate for non-normal distributions. Categorical variables were summarized as frequencies and percentages. Because the principal objective was to describe the frequency and etiological distribution of hematemesis, descriptive estimates constituted the primary analysis. Categorical comparisons were assessed using the Chi-square test where its assumptions were satisfied and Fisher's exact test where sparse cell frequencies made an exact test more appropriate. Statistical significance was defined as a two-sided p-value <0.05.

RESULTS

A total of 100 patients presenting with hematemesis were included during the six-month study period. The mean age was 46.8 ± 16.7 years, with an observed range of 18–80 years. Although participants aged 15–80 years were eligible for enrollment, the youngest enrolled participant was 18 years. Of the total sample, 68 (68.0%) were male and 32 (32.0%) were female. Chronic liver disease was documented in 55 (55.0%) patients, regular or frequent NSAID use in 28 (28.0%), and a previous episode of upper gastrointestinal bleeding in 21 (21.0%) (Table 1).

Table 1. Demographic and Clinical Characteristics of the Study Population (n = 100)

Characteristic	Value
Age, mean ± SD, years	46.8 ± 16.7
Observed age range, years	18–80
Male sex, n (%)	68 (68.0)
Female sex, n (%)	32 (32.0)
Chronic liver disease, n (%)	55 (55.0)
Regular/frequent NSAID use, n (%)	28 (28.0)
Previous upper gastrointestinal bleeding, n (%)	21 (21.0)

Abbreviations: NSAID, non-steroidal anti-inflammatory drug; SD, standard deviation.

Table 2. Etiological Distribution of Hematemesis (n = 100)

Etiology	n (%)
Esophageal varices	42 (42.0)
Peptic ulcer disease	25 (25.0)
Erosive gastritis	12 (12.0)
Gastric varices	8 (8.0)
Mallory–Weiss tear	5 (5.0)
Esophagitis	4 (4.0)
Upper gastrointestinal malignancy	4 (4.0)
Total	100 (100.0)

Esophageal varices were the most frequent identified cause of hematemesis, occurring in 42 (42.0%) patients, followed by peptic ulcer disease in 25 (25.0%). Erosive gastritis accounted for 12 (12.0%) cases, gastric varices for 8 (8.0%), Mallory–Weiss tear for 5 (5.0%), esophagitis for 4 (4.0%), and upper

gastrointestinal malignancy for 4 (4.0%). The seven etiological categories accounted for all 100 patients (Table 2).

When etiologies were grouped according to bleeding source, variceal bleeding accounted for 50 (50.0%) cases, comprising 42 patients with esophageal varices and 8 with gastric varices. Non-variceal causes accounted for the remaining 50 (50.0%) cases. Among patients with non-variceal bleeding, peptic ulcer disease represented 25 of 50 cases (50.0%), erosive gastritis 12 of 50 (24.0%), Mallory–Weiss tear 5 of 50 (10.0%), esophagitis 4 of 50 (8.0%), and upper gastrointestinal malignancy 4 of 50 (8.0%) (Table 3).

Table 3. Variceal and Non-Variceal Distribution of Hematemesis (n = 100)

Bleeding category	Etiology	n	% of total sample	% within category
Variceal	Esophageal varices	42	42.0	84.0
Variceal	Gastric varices	8	8.0	16.0
Variceal subtotal		50	50.0	100.0
Non-variceal	Peptic ulcer disease	25	25.0	50.0
Non-variceal	Erosive gastritis	12	12.0	24.0
Non-variceal	Mallory–Weiss tear	5	5.0	10.0
Non-variceal	Esophagitis	4	4.0	8.0
Non-variceal	Upper gastrointestinal malignancy	4	4.0	8.0
Non-variceal subtotal		50	50.0	100.0
Overall		100	100.0	—

The sex-stratified distribution showed that esophageal varices occurred in 32 of 68 male patients (47.1%) and 10 of 32 female patients (31.3%). Peptic ulcer disease occurred in 18 males (26.5%) and 7 females (21.9%), while erosive gastritis occurred in 8 males (11.8%) and 4 females (12.5%). Gastric varices were identified in 5 males (7.4%) and 3 females (9.4%). The remaining causes—Mallory–Weiss tear, esophagitis, and upper gastrointestinal malignancy—collectively occurred in 5 males (7.4%) and 8 females (25.0%). These values are presented descriptively because a valid inferential comparison cannot be reconstructed from the available aggregate information (Table 4).

Table 4. Distribution of Hematemesis Etiologies According to Sex

Etiological category	Male (n = 68), n (%)	Female (n = 32), n (%)	Total, n (%)
Esophageal varices	32 (47.1)	10 (31.3)	42 (42.0)
Peptic ulcer disease	18 (26.5)	7 (21.9)	25 (25.0)
Erosive gastritis	8 (11.8)	4 (12.5)	12 (12.0)
Gastric varices	5 (7.4)	3 (9.4)	8 (8.0)
Other causes*	5 (7.4)	8 (25.0)	13 (13.0)
Total	68 (100.0)	32 (100.0)	100 (100.0)

Other causes comprise Mallory–Weiss tear (n = 5), esophagitis (n = 4), and upper gastrointestinal malignancy (n = 4). Individual sex-specific counts for these three etiologies were not available.

Table 5. Endoscopic Assessment and Principal Diagnostic Yield (n = 100)

Endoscopic characteristic	Value
Patients undergoing upper gastrointestinal endoscopy, n (%)	100 (100.0)
Presentation-to-endoscopy interval, mean ± SD, hours	18.6 ± 7.4
Etiological source identified, n (%)	100 (100.0)
Esophageal varices or peptic ulcer disease, n (%)	67 (67.0)
Variceal source, n (%)	50 (50.0)
Non-variceal source, n (%)	50 (50.0)

Upper gastrointestinal endoscopy was performed in all 100 patients after initial stabilization. The mean interval from emergency department presentation to endoscopy was 18.6 ± 7.4 hours. Endoscopic assessment identified an etiological source in every enrolled patient, corresponding to a diagnostic classification rate of 100%. The endoscopic distribution was dominated by esophageal varices and peptic ulcer disease, which together accounted for 67 (67.0%) of all identified causes (Table 5).

DISCUSSION

The present study characterized the etiological spectrum of hematemesis among 100 patients presenting to a tertiary-care emergency department in Peshawar. Esophageal varices were the most frequent individual cause, accounting for 42.0% of cases, followed by peptic ulcer disease in 25.0%. When esophageal and gastric varices were combined, variceal and non-variceal causes each accounted for 50.0% of presentations. This pattern emphasizes the clinical importance of distinguishing variceal from non-variceal hemorrhage because their underlying pathophysiology, initial pharmacological treatment, endoscopic management, and secondary-prevention strategies differ substantially (2,7).

The predominance of esophageal varices in the present cohort is clinically consistent with the high proportion of patients with chronic liver disease, which was documented in 55.0% of participants. Portal hypertension is the principal mechanism underlying gastroesophageal variceal formation and rupture, and variceal hemorrhage remains one of the major life-threatening complications of advanced portal hypertensive disease (7,8). Recent hospital-based evidence also demonstrates substantial geographical variation in UGIB etiology. Someili et al. reported esophageal varices in 52.2% of 483 patients with UGIB in a Saudi tertiary-care population, with hematemesis occurring in 67.5% of cases (11). This contemporary finding closely resembles the variceal predominance observed in the present study and supports the interpretation that referral setting and the underlying prevalence of chronic liver disease can substantially influence the etiological profile of UGIB.

The present findings contrast with recent broader epidemiological evidence in which peptic ulcer disease remains the predominant cause of UGIB. A 2025 review of changing UGIB epidemiology reported peptic ulcer disease as the most common etiology, accounting for approximately 43.6% of cases, compared with approximately 8.0% attributable to esophageal variceal bleeding (12). Such differences reinforce the importance of interpreting etiological frequencies within their healthcare and population context rather than assuming that a single distribution applies internationally. Variations in chronic liver disease, Helicobacter pylori infection, medication exposure, population age, referral pathways, and preventive treatment may all contribute to differences between regions and institutions (7,12).

Peptic ulcer disease nevertheless remained an important component of the disease burden in the present study, accounting for one quarter of all hematemesis cases and half of the non-variceal etiologies. Contemporary evidence continues to identify peptic ulcer disease as a major cause of clinically significant UGIB. In a large United States analysis involving 136,425 admissions for ulcer bleeding, Mujadzic et al. demonstrated the continuing clinical and resource burden of gastric and duodenal ulcer hemorrhage and reported high endoscopic hemostatic success when endoscopic treatment was undertaken (13). Together with international recommendations for non-variceal UGIB management, these findings support the continued importance of rapid recognition and endoscopic assessment of ulcer-related hemorrhage (2,5,13).

Medication exposure is also relevant to the interpretation of non-variceal bleeding. In the present cohort, 28.0% of patients reported regular or frequent NSAID use. NSAIDs can impair gastroduodenal mucosal protection and are established contributors to ulcer formation and bleeding, particularly in patients with additional risk factors (3,5). Recent epidemiological analyses continue to emphasize the changing influence of NSAIDs, antiplatelet drugs, and anticoagulants on the contemporary profile of UGIB (12). Nevertheless, because the present study was observational and primarily descriptive, the recorded NSAID exposure should be interpreted as a clinical characteristic of the cohort rather than evidence of a causal association with specific bleeding lesions.

Male patients constituted 68.0% of the study population. A similar male predominance has been reported in contemporary hospital-based UGIB cohorts. Someili et al. reported that 74.1% of their 483 patients were male (11). In the present study, esophageal varices were descriptively more frequent among males than females; however, sex-specific findings should be interpreted cautiously because several etiological

categories contained relatively small numbers and the study was not powered primarily to identify independent demographic predictors of bleeding etiology.

All patients in the present study underwent upper gastrointestinal endoscopy after initial stabilization, with a mean presentation-to-endoscopy interval of 18.6 ± 7.4 hours. Endoscopy remains central to the evaluation of acute UGIB because it permits direct identification of the bleeding lesion and provides an opportunity for endoscopic hemostatic treatment where indicated (2,5,6). Recent clinical data further support the practical importance of timely endoscopy. In the cohort reported by Someili et al., more than half of patients underwent endoscopy within 24 hours, and the investigators demonstrated clinically important associations between presentation characteristics, rebleeding, transfusion requirements, and mortality (11). The complete endoscopic evaluation achieved in the present cohort therefore represents an important methodological strength because etiological classification was based on direct visualization rather than clinical presentation alone.

The observed equal distribution between variceal and non-variceal bleeding also highlights the heterogeneity of patients presenting with hematemesis. Contemporary evidence indicates that UGIB epidemiology continues to evolve in response to improvements in H. pylori eradication, proton-pump inhibitor use, management of portal hypertension, and changing patterns of anticoagulant, antiplatelet, and NSAID exposure (12). Consequently, locally generated etiological data remain clinically valuable even when international guidelines establish broadly applicable principles of stabilization, risk assessment, and endoscopic management (2,7).

The findings have practical implications for emergency and gastroenterology services in settings with a substantial burden of chronic liver disease. The high frequency of esophageal and gastric varices suggests that clinicians evaluating hematemesis in comparable tertiary-care populations should maintain a high index of suspicion for portal hypertensive bleeding while simultaneously considering common non-variceal causes such as peptic ulcer disease. Access to timely endoscopy is especially important because clinical presentation alone cannot reliably establish the lesion responsible for bleeding, and management differs according to the identified source (2,7,11).

Several limitations should be considered. The study was conducted at a single tertiary-care emergency department using consecutive non-probability sampling, and referral patterns may therefore have influenced the high observed proportion of variceal disease. The sample size of 100 participants also limited detailed analysis of relatively uncommon etiologies and subgroup relationships. In addition, the study primarily evaluated etiological frequencies and did not examine important subsequent outcomes such as rebleeding, transfusion requirements, therapeutic endoscopic interventions, duration of hospitalization, or mortality. Contemporary UGIB studies demonstrate that these outcomes provide important additional information regarding disease severity and prognosis (11,13). Future multicenter prospective studies incorporating standardized severity assessment, therapeutic interventions, rebleeding, mortality, and other clinically relevant outcomes would therefore provide a more comprehensive characterization of hematemesis and UGIB in Pakistan.

CONCLUSION

Esophageal varices were the most frequent individual cause of hematemesis in this emergency department population, followed by peptic ulcer disease, while variceal and non-variceal etiologies each accounted for half of all cases. These findings demonstrate the substantial contribution of both portal hypertension-related and non-variceal disease to hematemesis in this setting. Prompt clinical assessment followed by appropriate endoscopic evaluation remains important for identifying the bleeding source and directing etiology-specific management.

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