



**INTERNATIONAL JOURNAL OF
PHARMACEUTICAL SCIENCES**
[ISSN: 0975-4725; CODEN(USA): IJPS00]
Journal Homepage: <https://www.ijpsjournal.com>



Research Article

Clinical Characteristics, Management, and Outcomes of Gastrointestinal Bleeding Among Patients Receiving Antiplatelet Therapy, Anticoagulant Therapy, or Neither: A Retrospective Observational Cohort Study

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ARTICLE INFO

Published: 26 Oct 2026

Keywords:

Gastrointestinal hemorrhage, antiplatelet agents, anticoagulants, rebleeding, mortality, retrospective cohort

DOI:

10.5281/zenodo.23658453

ABSTRACT

Background: Gastrointestinal bleeding (GIB) is a clinically important cause of emergency assessment and hospitalization. Antiplatelet and anticoagulant therapies are widely used for cardiovascular and thromboembolic prevention but may complicate the management of bleeding. Comparing patients receiving these therapies with patients receiving neither can help describe differences in clinical profiles, management, and outcomes. **Objective:** To compare the clinical characteristics, management strategies, and outcomes of patients with GIB according to antiplatelet and anticoagulant medication exposure, and to examine factors associated with 28-day all-cause mortality. **Methods:** A retrospective observational cohort study was conducted at a tertiary care hospital in India from 1 February 2023 through 1 February 2026. The study included 5,000 records: 3,850 patients receiving neither antiplatelet nor anticoagulant therapy, 1,000 receiving antiplatelet therapy, and 150 receiving anticoagulant therapy. Medication exposure and clinical outcomes were abstracted from medical records. **Results:** The mean age was 57.9 ± 16.6 years, and 32.0% of patients were female. Peptic ulcer disease was the most frequently reported diagnosis (52.7%). Hypertension was more frequent in the antiplatelet group than in the reference group (79.4% vs 32.9%). Rebleeding within 28 days occurred in 486 patients (9.7%), and 28-day all-cause mortality occurred in 192 patients (3.8%). The reported multivariable analysis identified antiplatelet exposure, anticoagulant exposure, age, chronic kidney disease, low hemoglobin, low systolic blood pressure, and elevated lactate as factors associated with 28-day mortality. **Conclusion:** In this retrospective cohort, clinical characteristics, management, and outcomes differed across the reported medication-exposure groups.

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Relevant conflicts of interest/financial disclosures: The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.



The findings describe associations and should not be interpreted as evidence of causation.

INTRODUCTION

Gastrointestinal bleeding (GIB) is a common and potentially serious clinical presentation that ranges from self-limited blood loss to life-threatening hemorrhage requiring resuscitation, endoscopic treatment, transfusion, interventional radiology, or surgery. It is commonly classified as upper or lower GIB according to the anatomical source. Hematemesis, coffee-ground emesis, and melena often suggest upper GIB, whereas hematochezia commonly suggests lower GIB, although brisk upper-tract hemorrhage may also present with hematochezia. Initial management requires assessment of hemodynamic status, stabilization when needed, identification of the likely bleeding source, and risk stratification. Contemporary guidelines emphasize early clinical assessment and appropriately timed diagnostic and therapeutic procedures [1–4].

The causes of GIB are heterogeneous and include peptic ulcer disease, variceal hemorrhage, erosive gastroduodenal lesions, malignancy, angiodysplasia, diverticular bleeding, inflammatory bowel disease, and other mucosal or vascular abnormalities. The distribution of these conditions varies with age, comorbidities, medication exposure, and healthcare setting. Peptic ulcer disease is a major cause of upper GIB, while portal hypertension and cirrhosis are important determinants of variceal bleeding. Identifying the cause is essential because treatment differs between ulcer bleeding, portal-hypertensive bleeding, vascular lesions, and lower gastrointestinal sources [1–4,15–17].

Antiplatelet agents are widely prescribed for prevention of atherothrombotic events, including myocardial infarction and ischemic stroke. Common agents include aspirin and P2Y₁₂ inhibitors such as clopidogrel. These agents can impair primary hemostasis when a gastrointestinal lesion bleeds. The clinical consequences depend on the drug, dose, combination therapy, indication, coexisting disease, and bleeding source. Decisions about interruption and resumption require balancing hemorrhagic risk against cardiovascular or cerebrovascular risk [5–7].

Anticoagulants are used for conditions such as atrial fibrillation and venous thromboembolism. These agents include vitamin K antagonists, heparins, and direct oral anticoagulants. Renal function, dose, drug interactions, timing of the last dose, and concomitant antiplatelet or NSAID use may influence clinical risk. Reversal and resumption decisions depend on bleeding severity, the specific agent, the indication for anticoagulation, and thromboembolic risk [5,6,8,9].

Rebleeding and mortality remain important outcomes after GIB. Their occurrence is influenced by the bleeding source, physiological instability, age, comorbidity burden, laboratory abnormalities, treatment response, and the timing of definitive hemostasis. Risk scores such as the Glasgow-Blatchford score, Rockall score, and AIMS65 have been developed to support risk assessment, but their performance varies by outcome and population [10–12]. Clinical characteristics such as low blood pressure, anemia, renal dysfunction, and advanced age may provide prognostic information, although their meaning depends on the broader clinical context.

Comparisons between patients receiving antiplatelet or anticoagulant therapy and those receiving neither are challenging because medication exposure is not randomly assigned. The conditions that prompt antithrombotic prescribing may themselves be associated with worse outcomes. This confounding by indication means observed differences cannot automatically be attributed to medication exposure. Analyses should describe baseline differences transparently and use appropriate statistical adjustment while acknowledging residual confounding and limitations inherent to retrospective observational research [5,6,8,9,18,19].

Although clinical guidelines address the management of upper and lower GIB and antithrombotic therapy, the clinical profile, treatment patterns, and outcomes of patients can vary across hospitals and populations. A structured assessment of demographics, comorbidities, presenting symptoms, vital signs, laboratory findings, diagnoses, medication use, supportive care, procedures, rebleeding, mortality, and length of stay can provide a broad description of care. Such work is most informative when exposure and outcome definitions are explicit and the statistical analyses are reproducible.



Accordingly, this study aimed to compare the clinical characteristics, management strategies, and outcomes of patients with gastrointestinal bleeding according to the medication-exposure groups represented in the available dataset.

MATERIALS AND METHODS

2.1 Study design, setting, and period

A retrospective observational cohort study was conducted at a tertiary care hospital in India using previously documented clinical information from 1 February 2023 through 1 February 2026. The study compared clinical characteristics, management, and outcomes among patients presenting with gastrointestinal bleeding.

2.2 Study population and sample size

The analysis included 5,000 records: 3,850 patients receiving neither antiplatelet nor anticoagulant therapy (reference group; 77.0%), 1,000 receiving antiplatelet therapy (20.0%), and 150 receiving anticoagulant therapy (3.0%). These were the three exposure groups used in the analysis.

2.3 Inclusion criteria

- Patients with a documented diagnosis of gastrointestinal bleeding.
- Patients with symptoms or clinical manifestations consistent with GIB, including hematemesis, melena, coffee-ground emesis, hematochezia, or other documented compatible manifestations.
- Records falling within the defined three-year study period.
- Records with sufficient information to classify medication exposure and assess the relevant outcomes.

2.4 Exclusion criteria

- Records without adequate documentation to establish the GIB diagnosis.
- Duplicate records for the same index episode, unless separately defined episodes were permitted by the protocol.
- Records outside the study period.

- Records with insufficient information for a particular variable were handled using the available observations for that analysis, with denominators based on the available data.
- Records with unresolved inconsistencies in essential variables that prevented assessment of the primary outcomes.

2.5 Medication-exposure classification

Medication exposure was classified from medication histories documented in the medical records for the index bleeding episode. The three analysis groups were: (1) neither antiplatelet nor anticoagulant therapy (reference), (2) antiplatelet therapy, and (3) anticoagulant therapy. Exposure was classified according to the study dataset's recorded categories.

2.6 Data collection and variables

Data were collected using a structured data collection form. Variables included demographics; comorbidities and concomitant medicines; presenting symptoms; vital signs; hematology and coagulation tests; renal and biochemical parameters; bleeding diagnoses; pharmacological treatment; supportive care; procedures; rebleeding; mortality; and length of stay. Blood urea nitrogen (BUN) was recorded in mg/dL.

2.7 Outcomes

The primary outcomes were rebleeding within 28 days and all-cause mortality within 28 days. Secondary outcomes were all-cause mortality within 90 days and length of hospital stay. Outcomes were ascertained from medical records.

2.8 Statistical analysis

Analyses were performed using IBM SPSS Statistics version 32.0. Categorical variables are presented as counts and percentages, and continuous variables as means and standard deviations. Associations between categorical variables were assessed using chi-square tests where appropriate. Continuous variables were compared across groups using the tests specified in the study analysis. Logistic regression was used to evaluate factors associated with 28-day all-cause mortality. Statistical significance was defined as a two-sided p value <0.05 .



2.9 Ethical considerations

Ethical approval was obtained from the Institutional Review Board (IRB), reference NCP/IHEC/-CERTI-0236/2022, dated 12 December 2022. Patient confidentiality was maintained during data collection and analysis.

2.10 Reporting standard

The study was reported with reference to the STROBE recommendations for cohort studies [18].

RESULTS

A total of 5,000 records were included: 3,850 in the reference group, 1,000 in the antiplatelet group, and 150 in the anticoagulant group.

Table 1: Demographic Characteristics

Characteristic	Overall (N=5000)	Reference (n=3850)	Antiplatelet (n=1000)	Anticoagulant (n=150)	p-value
Female sex, n (%)	1598 (32.0)	1248 (32.4)	304 (30.4)	46 (30.7)	0.449
Age, years, mean $\pm$ SD	57.9 $\pm$ 16.6	58.1 $\pm$ 16.6	57.6 $\pm$ 16.5	55.2 $\pm$ 17.9	0.089*

Table 2: Comorbidities and Risk Factors

Characteristic	Overall (N=5000)	Reference (n=3850)	Antiplatelet (n=1000)	Anticoagulant (n=150)	p-value
Hypertension, n (%)	2125 (42.5)	1267 (32.9)	794 (79.4)	64 (42.7)	<0.001
Diabetes mellitus, n (%)	1182 (23.6)	868 (22.5)	274 (27.4)	40 (26.7)	0.004
Previous peptic ulcer disease, n (%)	935 (18.7)	703 (18.3)	197 (19.7)	35 (23.3)	0.195
Previous cerebrovascular accident, n (%)	756 (15.1)	484 (12.6)	253 (25.3)	19 (12.7)	<0.001
Chronic kidney disease, n (%)	399 (8.0)	268 (7.0)	105 (10.5)	26 (17.3)	<0.001
Cirrhosis/liver disease, n (%)	386 (7.7)	317 (8.2)	50 (5.0)	19 (12.7)	<0.001
NSAID use, n (%)	224 (4.5)	145 (3.8)	58 (5.8)	21 (14.0)	<0.001
Corticosteroid use, n (%)	101 (2.0)	51 (1.3)	41 (4.1)	9 (6.0)	<0.001

Table 3: Presenting Symptoms

Characteristic	Overall (N=5000)	Reference (n=3850)	Antiplatelet (n=1000)	Anticoagulant (n=150)	p-value
Melena, n (%)	2783 (55.7)	2122 (55.1)	573 (57.3)	88 (58.7)	0.350
Hematemesis, n (%)	1823 (36.5)	1405 (36.5)	359 (35.9)	59 (39.3)	0.715
Abdominal pain, n (%)	1451 (29.0)	1107 (28.8)	300 (30.0)	44 (29.3)	0.738
Syncope/dizziness, n (%)	1083 (21.7)	838 (21.8)	209 (20.9)	36 (24.0)	0.654
Fatigue/pallor, n (%)	980 (19.6)	749 (19.5)	199 (19.9)	32 (21.3)	0.821
Coffee-ground emesis, n (%)	867 (17.3)	678 (17.6)	164 (16.4)	25 (16.7)	0.650
Hematochezia, n (%)	736 (14.7)	569 (14.8)	147 (14.7)	20 (13.3)	0.887
Diaphoresis, n (%)	578 (11.6)	436 (11.3)	123 (12.3)	19 (12.7)	0.630

Table 4: Vital Signs



Characteristic	Overall (N=5000)	Reference (n=3850)	Antiplatelet (n=1000)	Anticoagulant (n=150)	p-value
Heart rate, beats/min, mean $\pm$ SD	95.7 $\pm$ 18.3	95.8 $\pm$ 18.3	95.2 $\pm$ 18.2	97.0 $\pm$ 19.1	0.446*
Systolic BP, mmHg, mean $\pm$ SD	118.2 $\pm$ 21.2	118.4 $\pm$ 21.1	117.5 $\pm$ 21.6	117.5 $\pm$ 21.5	0.451*
Diastolic BP, mmHg, mean $\pm$ SD	66.5 $\pm$ 13.9	66.4 $\pm$ 13.9	66.8 $\pm$ 14.0	66.9 $\pm$ 15.3	0.677*
Mean BP, mmHg, mean $\pm$ SD	83.7 $\pm$ 11.7	83.7 $\pm$ 11.6	83.7 $\pm$ 11.8	83.7 $\pm$ 11.7	1.000*
Respiratory rate, breaths/min, mean $\pm$ SD	20.3 $\pm$ 4.1	20.3 $\pm$ 4.1	20.4 $\pm$ 4.1	20.5 $\pm$ 4.1	0.687*

Table 5: Hematology and Coagulation Parameters

Characteristic	Overall (N=5000)	Reference (n=3850)	Antiplatelet (n=1000)	Anticoagulant (n=150)	p-value
Hemoglobin, g/L, mean $\pm$ SD	92.1 $\pm$ 27.9	91.9 $\pm$ 27.8	93.4 $\pm$ 28.5	90.0 $\pm$ 28.4	0.203*
Platelets, $\times 10^9$ /L, mean $\pm$ SD	181.2 $\pm$ 85.1	181.7 $\pm$ 85.5	181.9 $\pm$ 83.7	163.9 $\pm$ 83.9	0.041*
INR, mean $\pm$ SD	1.5 $\pm$ 0.4	1.4 $\pm$ 0.4	1.5 $\pm$ 0.4	1.5 $\pm$ 0.5	<0.001*
PT, seconds, mean $\pm$ SD	14.7 $\pm$ 3.0	14.8 $\pm$ 3.0	14.2 $\pm$ 2.9	14.9 $\pm$ 3.2	<0.001*
APTT, seconds, mean $\pm$ SD	32.3 $\pm$ 8.6	32.5 $\pm$ 8.7	31.5 $\pm$ 8.5	33.9 $\pm$ 7.8	<0.001*

Table 6: Renal and Biochemical Parameters

Characteristic	Overall (N=5000)	Reference (n=3850)	Antiplatelet (n=1000)	Anticoagulant (n=150)	p-value
BUN, mg/dL, mean $\pm$ SD	11.9 $\pm$ 8.0	11.9 $\pm$ 8.0	11.9 $\pm$ 8.0	10.3 $\pm$ 7.6	0.054*
Creatinine, μ mol/L, mean $\pm$ SD	106.4 $\pm$ 46.2	104.2 $\pm$ 45.6	112.5 $\pm$ 47.1	120.8 $\pm$ 48.6	<0.001*
Albumin, g/L, mean $\pm$ SD	34.6 $\pm$ 6.3	34.9 $\pm$ 6.3	33.8 $\pm$ 6.4	31.9 $\pm$ 6.1	<0.001*
Total bilirubin, μ mol/L, mean $\pm$ SD	18.6 $\pm$ 11.1	18.7 $\pm$ 11.1	18.3 $\pm$ 11.2	20.4 $\pm$ 11.3	0.093*
Lactate, mmol/L, mean $\pm$ SD	2.4 $\pm$ 1.2	2.3 $\pm$ 1.2	2.4 $\pm$ 1.1	2.7 $\pm$ 1.2	<0.001*

Abbreviations: BUN, blood urea nitrogen; INR, international normalized ratio; PT, prothrombin time; APTT, activated partial thromboplastin time.

Table 7: Pharmacological Management

Characteristic	Overall (N=5000)	Reference (n=3850)	Antiplatelet (n=1000)	Anticoagulant (n=150)	p-value
Proton pump inhibitor, n (%)	4843 (96.9)	3725 (96.8)	971 (97.1)	147 (98.0)	0.614
Somatostatin, n (%)	2504 (50.1)	1932 (50.2)	494 (49.4)	78 (52.0)	0.810
Pituitrin, n (%)	518 (10.4)	386 (10.0)	113 (11.3)	19 (12.7)	0.321



Characteristic	Overall (N=5000)	Reference (n=3850)	Antiplatelet (n=1000)	Anticoagulant (n=150)	p-value
Vasoactive drugs, n (%)	1494 (29.9)	1139 (29.6)	296 (29.6)	59 (39.3)	0.037
Tranexamic acid, n (%)	785 (15.7)	619 (16.1)	135 (13.5)	31 (20.7)	0.032
Oral mucosal protectants, n (%)	1894 (37.9)	1443 (37.5)	393 (39.3)	58 (38.7)	0.561

Table 8: Supportive Care and Transfusion

Characteristic	Overall (N=5000)	Reference (n=3850)	Antiplatelet (n=1000)	Anticoagulant (n=150)	p-value
Stomach tube, n (%)	1131 (22.6)	880 (22.9)	218 (21.8)	33 (22.0)	0.763
Central venous catheter, n (%)	1362 (27.2)	1068 (27.7)	255 (25.5)	39 (26.0)	0.345
Endotracheal intubation, n (%)	708 (14.2)	529 (13.7)	149 (14.9)	30 (20.0)	0.074
ICU admission, n (%)	1189 (23.8)	877 (22.8)	260 (26.0)	52 (34.7)	<0.001
Any blood product transfusion, n (%)	4546 (90.9)	3498 (90.9)	914 (91.4)	134 (89.3)	0.686
Packed RBC transfusion, n (%)	2485 (49.7)	1906 (49.5)	506 (50.6)	73 (48.7)	0.800
Plasma, n (%)	1169 (23.4)	877 (22.8)	248 (24.8)	44 (29.3)	0.088
Platelet transfusion, n (%)	129 (2.6)	102 (2.6)	25 (2.5)	2 (1.3)	0.599†
Cryoprecipitate, n (%)	72 (1.4)	58 (1.5)	13 (1.3)	1 (0.7)	0.641†

Table 9: Procedures and Interventions

Characteristic	Overall (N=5000)	Reference (n=3850)	Antiplatelet (n=1000)	Anticoagulant (n=150)	p-value
Endoscopic treatment, n (%)	1470 (29.4)	1120 (29.1)	308 (30.8)	42 (28.0)	0.532
Surgery, n (%)	486 (9.7)	362 (9.4)	107 (10.7)	17 (11.3)	0.371
Interventional radiology/em-bolization, n (%)	237 (4.7)	188 (4.9)	43 (4.3)	6 (4.0)	0.675
TIPS creation, n (%)	79 (1.6)	61 (1.6)	15 (1.5)	3 (2.0)	0.900†

Table 10: Clinical Outcomes

Characteristic	Overall (N=5000)	Reference (n=3850)	Antiplatelet (n=1000)	Anticoagulant (n=150)	p-value
28-day rebleeding, n (%)	486 (9.7)	347 (9.0)	120 (12.0)	19 (12.7)	0.008
28-day all-cause mortality, n (%)	192 (3.8)	131 (3.4)	49 (4.9)	12 (8.0)	0.002
90-day all-cause mortality, n (%)	351 (7.0)	246 (6.4)	87 (8.7)	18 (12.0)	0.002
Length of stay, days, mean $\pm$ SD	8.6 $\pm$ 3.1	8.6 $\pm$ 3.1	8.5 $\pm$ 3.0	9.7 $\pm$ 3.1	<0.001*

Table 11: Primary Gastrointestinal Bleeding Diagnosis

Diagnosis	Overall (N=5000)	Reference (n=3850)	Antiplatelet (n=1000)	Anticoagulant (n=150)
Peptic ulcer	2636 (52.7)	1932 (50.2)	621 (62.1)	83 (55.3)
Varices	813 (16.3)	760 (19.7)	38 (3.8)	15 (10.0)
GI malignancy	381 (7.6)	313 (8.1)	58 (5.8)	10 (6.7)
Gastritis/duodenitis	363 (7.3)	256 (6.6)	97 (9.7)	10 (6.7)
Erosive/stress lesions	273 (5.5)	209 (5.4)	54 (5.4)	10 (6.7)
Other/undetermined	136 (2.7)	63 (1.6)	65 (6.5)	8 (5.3)
Mallory–Weiss	132 (2.6)	110 (2.9)	20 (2.0)	2 (1.3)
Angiodysplasia	80 (1.6)	56 (1.5)	22 (2.2)	2 (1.3)
Portal hypertensive gastropathy	65 (1.3)	58 (1.5)	6 (0.6)	1 (0.7)
Esophagitis/esophageal ulcer	62 (1.2)	46 (1.2)	10 (1.0)	6 (4.0)
Inflammatory bowel disease	34 (0.7)	26 (0.7)	6 (0.6)	2 (1.3)
Diverticular/lower GIB	16 (0.3)	13 (0.3)	3 (0.3)	0 (0.0)
Dieulafoy lesion	9 (0.2)	8 (0.2)	0 (0.0)	1 (0.7)

The diagnosis-by-group Pearson chi-square test showed an overall difference across groups ($p < 0.001$).

Table 12: Univariate Associations with 28-Day All-Cause Mortality

Variable	Total with variable	Deaths	Crude OR (95% CI)	p-value
Age ≥ 65 years	1726	84	1.50 (1.12–2.01)	0.006
Antiplatelet exposure	1000	49	1.39 (1.00–1.94)	0.051
Anticoagulant exposure	150	12	2.26 (1.23–4.15)	0.007
Heart rate ≥ 101 beats/min	1963	73	0.95 (0.70–1.27)	0.720
SBP ≤ 100 mmHg	1005	55	1.63 (1.18–2.25)	0.003
Respiratory rate ≥ 24 /min	1099	53	1.37 (0.99–1.90)	0.055
Hemoglobin < 80 g/L	1652	82	1.54 (1.15–2.06)	0.004
INR > 1.2	3533	146	1.33 (0.95–1.87)	0.095
Creatinine ≥ 120 μ mol/L	1901	76	1.07 (0.80–1.44)	0.649
Albumin < 30 g/L	1166	51	1.20 (0.86–1.66)	0.279
Lactate ≥ 3 mmol/L	1469	75	1.57 (1.17–2.11)	0.003
Peptic ulcer diagnosis	2636	106	1.11 (0.83–1.48)	0.481
Varices	813	31	0.99 (0.67–1.47)	0.965
GI malignancy	381	12	0.80 (0.44–1.45)	0.466
Female sex	1598	64	1.07 (0.79–1.45)	0.677
Hypertension	2125	79	0.94 (0.70–1.27)	0.699
Diabetes mellitus	1182	48	1.08 (0.77–1.51)	0.651
Previous peptic ulcer disease	935	32	0.86 (0.59–1.27)	0.462
Previous cerebrovascular accident	756	29	1.00 (0.67–1.49)	0.995
Chronic kidney disease	399	29	2.13 (1.42–3.21)	< 0.001
Cirrhosis/liver disease	386	21	1.49 (0.94–2.38)	0.089
NSAID use	224	9	1.05 (0.53–2.08)	0.887
Corticosteroid use	101	5	1.31 (0.53–3.26)	0.557
Syncope/dizziness	1083	56	1.52 (1.10–2.09)	0.010
Diaphoresis	578	34	1.69 (1.15–2.47)	0.007



Table 13: Multivariable Logistic Regression for 28-Day Mortality

Predictor	Adjusted OR (95% CI)	p-value
Anticoagulant vs reference	2.25 (1.23–4.12)	0.009
Antiplatelet vs reference	1.60 (1.08–2.37)	0.018
Age, per year	1.02 (1.01–1.03)	<0.001
Female sex	1.09 (0.83–1.44)	0.534
Hypertension	0.81 (0.59–1.10)	0.175
Diabetes mellitus	1.07 (0.76–1.49)	0.705
Previous peptic ulcer disease	0.81 (0.54–1.20)	0.294
Previous cerebrovascular accident	0.94 (0.61–1.46)	0.787
Chronic kidney disease	1.95 (1.35–2.83)	<0.001
Cirrhosis/liver disease	1.62 (0.97–2.73)	0.068
NSAID use	1.02 (0.50–2.08)	0.962
Corticosteroid use	1.10 (0.42–2.85)	0.851
Hemoglobin <80 g/L	1.57 (1.20–2.06)	0.001
SBP ≤100 mmHg	1.62 (1.11–2.38)	0.013
Lactate ≥3 mmol/L	1.55 (1.16–2.07)	0.003

Table 14: Supplied Subgroup Estimates for 28-Day Mortality

Subgroup comparison	Deaths/total	OR (95% CI)	p-value
Antiplatelet vs reference, male	35/696 vs 85/2602	1.57 (1.05–2.35)	0.027
Antiplatelet vs reference, female	14/304 vs 46/1248	1.26 (0.68–2.33)	0.456
Antiplatelet vs reference, age <65	26/664 vs 74/2507	1.34 (0.85–2.11)	0.206
Antiplatelet vs reference, age ≥65	23/336 vs 57/1343	1.66 (1.01–2.73)	0.045
Anticoagulant vs reference, male	8/104 vs 85/2602	2.47 (1.16–5.24)	0.015
Anticoagulant vs reference, female	4/46 vs 46/1248	2.49 (0.86–7.23)	0.083
Anticoagulant vs reference, age <65	8/103 vs 74/2507	2.77 (1.30–5.91)	0.006
Anticoagulant vs reference, age ≥65	4/47 vs 57/1343	2.10 (0.73–6.05)	0.160

3.1 Summary of results

The mean age was 57.9 ± 16.6 years and 32.0% of patients were female. Hypertension and previous cerebrovascular accident were more frequent in the antiplatelet group, while chronic kidney disease was most frequent in the anticoagulant group. Melena was the most common symptom, and peptic ulcer disease was the most frequently reported diagnosis. Rebleeding within 28 days occurred in 9.7% of patients, and 28-day mortality was 3.8%.

DISCUSSION

The reported results provide a broad comparison of demographic, clinical, laboratory, diagnostic, treatment, and outcome profiles of patients with GIB across the currently available medication-exposure groups. The most prominent reported findings were differences in comorbidity distributions, laboratory parameters, rebleeding, mortality, intensive care admission, and length of stay. These findings are clinically plausible areas of investigation because antithrombotic therapy is often prescribed to patients with cardiovascular or thromboembolic disease, and those underlying conditions may independently affect prognosis [5,6,8,9].



In the study summaries, hypertension and previous cerebrovascular accident were more common in the antiplatelet group, and chronic kidney disease was more frequent in the anticoagulation group. These imbalances are important because medication exposure is determined by clinical indications and is not randomly allocated in observational cohorts. Consequently, unadjusted comparisons can be confounded by the conditions that led to treatment, as well as by frailty, disease severity, and concomitant medicines [8,9,19].

Melena was the most common reported symptom, followed by hematemesis and abdominal pain. Such symptoms are common in GIB but cannot alone establish the bleeding source. Clinical assessment should integrate symptoms with hemodynamic status, laboratory results, and endoscopic or other diagnostic findings. Guidelines emphasize early risk assessment, resuscitation when indicated, and timely diagnostic and therapeutic intervention [1–4].

Peptic ulcer disease was the most frequent diagnosis in the study results. Peptic ulcer bleeding requires attention to hemostasis, acid suppression, risk stratification, and prevention of recurrence. Variceal bleeding requires a different management pathway that may include vasoactive therapy and endoscopic treatment. The distribution of bleeding sources is therefore central to interpreting treatment patterns and outcomes [1,3,4,15–17].

The supplied outcome table reported 28-day rebleeding in 9.7% overall, with higher percentages in the antiplatelet and anticoagulation groups than in the reference group. Reported 28-day and 90-day mortality were also highest in the anticoagulation group. These observations should not be interpreted as proof that antithrombotic medications caused worse outcomes. The severity of the bleeding lesion, indication for therapy, comorbidities, anticoagulant type and timing, renal function, and treatment decisions may all influence the observed association. Evidence on resumption of anticoagulation after GI bleeding illustrates the need to balance recurrent bleeding against thromboembolism and mortality [8,9].

In the supplied regression table, older age, chronic kidney disease, low hemoglobin, low systolic blood pressure, and elevated lactate were reported as

associated with 28-day mortality. These factors may be relevant to clinical severity and deserve further evaluation in verified patient-level data. Risk scores such as Glasgow-Blatchford, Rockall, and AIMS65 have been evaluated for predicting intervention, mortality, and other outcomes, but no score is equally accurate for every endpoint or patient group [10–12].

This retrospective study describes clinical characteristics, management, and outcomes among patients with gastrointestinal bleeding. As medication exposure was not randomized, observed differences may reflect confounding by indication and other measured or unmeasured factors. The findings should therefore be interpreted as associations rather than causal effects.

The retrospective design limits causal interpretation. The study may be affected by selection bias, information bias, exposure misclassification, incomplete documentation, and residual confounding. The findings are drawn from one tertiary care hospital and may not generalize to other settings. Interpretation also depends on the accuracy of exposure classification, outcome ascertainment, and statistical model specification.

5. STRENGTHS AND LIMITATIONS

Strengths include the large overall cohort and assessment of demographic, clinical, laboratory, diagnostic, management, and outcome variables. Outcomes included 28-day rebleeding, 28-day mortality, 90-day mortality, and length of stay.

Limitations include the retrospective single-centre design, dependence on medical-record completeness, possible selection and information bias, and confounding by indication. The anticoagulant group was comparatively small, which may limit precision. The results should not be interpreted causally, and findings may not generalize beyond similar clinical settings.

CONCLUSION

In this retrospective cohort of 5,000 patients with gastrointestinal bleeding, clinical profiles, management, and outcomes were described across three medication-exposure groups. Peptic ulcer disease was the most



frequent diagnosis, 28-day rebleeding occurred in 9.7% of patients, and 28-day mortality occurred in 3.8%. The reported analyses identified several clinical factors associated with mortality. These findings may inform further evaluation but should be interpreted in light of the retrospective design and potential confounding.

7. ETHICS STATEMENT

Ethical approval was obtained from the Institutional Review Board (IRB), reference NCP/IHEC/-CERTI-0236/2022, dated 12 December 2022. Patient confidentiality was maintained during data collection and analysis.

8. DATA AVAILABILITY STATEMENT

The data supporting the findings are held by the study team and are subject to institutional confidentiality and ethical requirements. Access to de-identified data may be considered upon reasonable request, subject to institutional approval.

9. FUNDING

No external funding was received for this study.

10. CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

AUTHOR CONTRIBUTIONS **Riyad Hossain:** Conceptualization, study design, methodology, data curation, formal analysis, interpretation of results, manuscript drafting, critical revision, and project coordination. **Abhisek Chaudhary:** Clinical data review, verification of study variables, and critical revision. **Dhruva Narayan K:** Data organization, validation of extracted records, preparation of tables, and manuscript review. **Debanjan Patra:** Contributions to clinical data review, interpretation of findings, and critical review of the manuscript. **Deep Deb:** Literature review, clinical interpretation, and critical review of the manuscript. **Shyamal Nath:** Literature review, interpretation of findings, and manuscript editing. **Subhrajit Debnath:** Data verification, reference checking, and manuscript review. **Tanmay Paul:** Data organization, table checking, and manuscript review. All authors should review and approve the final manuscript and accept accountability for their contributions.

Riyad Hossain led conceptualization, study methodology, data curation, formal analysis, interpretation of findings, and drafting of the original manuscript. Abhisek Chaudhary, Dhruva Narayan K, Deep Deb, and Shyamal Nath contributed to data interpretation and critical review and revision of the manuscript. All authors are responsible for reviewing and approving the final version.

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HOW TO CITE: Riyad Hossain, Abhisek Chaudhary, Dhruva Narayan K., Debanjan Patra, Deep Deb, Shyamal Nath, Subhrajit Debnath, Tanmay Paul, Clinical Characteristics, Management, and Outcomes of Gastrointestinal Bleeding Among Patients Receiving Antiplatelet Therapy, Anticoagulant Therapy, or Neither: A Retrospective Observational Cohort Study, *Int. J. of Pharm. Sci.*, 2026, Vol 4, Issue 10, 3906-3916. <https://doi.org/10.5281/zenodo.23658453>

