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Management of upper gastrointestinal bleeding due to peptic ulcers in cardiac patients: A retrospective study

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Abstract

Background: Cardiac patients are at increased risk of upper gastrointestinal bleeding (UGIB) due to frequent use of antithrombotic agents. This study examines the characteristics of peptic ulcer bleeding (PUD) in this population.

Methods: A 4-year retrospective study (2020–2024) was conducted at Ibn Sina University Hospital, Rabat. Cardiac patients presenting with UGIB and confirmed PUD on endoscopy were included.

Results: Among 418 UGIB cases, 73 had cardiac disease; 31 had PUD. Most were male (58%) with a mean age of 68.4 years. Antithrombotics were used by 83.9%. Endoscopic hemostasis was needed in 35.5%; rebleeding occurred in 12.9%. Common risk factors included age, prior PUD, NSAID use, and dual therapy.

Conclusion: UGIB in cardiac patients poses management challenges due to bleeding and thrombotic risks. Prevention with PPIs and tailored antithrombotic strategies is essential.

Keywords: PUD; UGIB; APAs; Anticoagulants; PPIs; Cardiac Disease

1. Introduction

Bleeding peptic ulcers (PUD) are the leading cause of upper gastrointestinal bleeding (UGIB), accounting for approximately 36% of cases.[1]

Patients with cardiac conditions are at increased risk of gastrointestinal bleeding, particularly UGIB, due to the frequent use of antiplatelet agents (APAs), vitamin K antagonists (VKAs), and direct oral anticoagulants (DOACs), which are prescribed for ischemic, arrhythmic, or valvular heart disease.

In cardiac patients, UGIB reveals underlying organic lesions in two-thirds of cases, with PUD being the most common.[2] Balancing the risk of worsening bleeding against the risk of thromboembolic events in these patients is a constant clinical challenge, before and after endoscopy, in both short- and long-term management.[3]

This article reports the experience of the Department of Hepato-Gastroenterology and Proctology, Medicine B, at Ibn Sina University Hospital in Rabat, in assessing the epidemiological, clinical, paraclinical, therapeutic, and outcome characteristics of PUD revealed by UGIB in cardiac patients, and the impact of antithrombotic therapy on its occurrence.

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2. Materials and Methods

This was a retrospective descriptive study conducted over 4 years (May 2020 to May 2024) in the Hepato-Gastroenterology and Proctology Department, Medicine B, Ibn Sina Hospital, Rabat. All patients with documented cardiac disease presenting with UGIB and diagnosed with PUD via upper gastrointestinal endoscopy (EGD) were included.

Data were collected from patient medical records and compiled on a pre-established data collection form, following a literature review.

3. Results

Out of 418 patients who underwent EGD for UGIB during the study period, 73 had underlying cardiac disease. Among them, PUD was found in 31 patients.

The mean age of cardiac patients was 68.4 years (range: 24–90 years), with 18 men (58.1%) and 13 women (41.9%), yielding a male-to-female ratio of 1.38.

12 patients (38.7%) had ischemic heart disease, 8 (25.8%) had arrhythmic heart disease, 5 (16.1%) had valvular disease, and 6 (19.4%) had other cardiac etiologies.

13 patients (41.9%) were on anticoagulants alone: 10(32,2%) on VKAs, 3(9,7%) on DOACs). 10 patients (32.2%) were on APAs— 5(16,1%) on aspirin monotherapy, 5(16,1%) on dual antiplatelet therapy (DAPT: aspirin + clopidogrel). 3 patients (9.7%) were on both APAs and anticoagulants. And finally, 5 patients (16.1%) were on no antithrombotic therapy.
A supratherapeutic INR was observed in 8 patients, accounting for 66.7% of those on VKAs.

Relevant medical history elements included the use of nonsteroidal anti-inflammatory drugs in 4 patients (12.9%), a prior history of PUD in 6 patients (19.4%), active smoking in 5 patients (16.1%), dyslipidemia under lipid-lowering treatment in 21 patients (67.7%), and alcohol use in 1 patient (3.2%).

UGIB presentations included melena in 19 patients (61.3%), hematemesis in 3 patients (9.7%), a combination of both in 8 patients (25.8%), and rectal bleeding with hematemesis accompanied by hemodynamic instability in 1 patient (3.2%).

Red blood cell transfusion was required in 17 patients (54.8%).

First EGD was performed within 12 to 24 hours of admission in 21 patients (67.7%).

Duodenal ulcers were identified in 18 patients (58%), and gastric ulcers in 15 patients (48.3%). Regarding Forrest classification, 17 ulcers were classified as Forrest III (54.8%), 5 as Forrest IIc (16.1%), 4 as Forrest IIb (12.9%), 2 as Forrest IIa (6.4%), 3 as Forrest Ib (9.7%), and 2 as Forrest Ia (6.4%).

Endoscopic hemostatic treatment, consisting of adrenaline injection and placement of hemostatic clips, was performed in 11 patients (35.5%).

Proton pump inhibitors (PPIs) were prescribed to all patients.

All antithrombotic therapies were discontinued prior to endoscopic examination except for 4 patients (12.9%) on dual antiplatelet therapy (DAPT), for whom aspirin was continued.

Antithrombotic therapy was resumed in all patients after bleeding cessation and endoscopic evaluation, with dose adjustments in 15 patients (48.4%).

Rebleeding occurred in 4 patients (12.9%).

4. Discussion

Currently, 6% of GI bleeds occur in patients on therapeutic doses of anticoagulants, and 33% occur in those on antiplatelet agents (APAs). [4] Randomized controlled trials, cohort studies, and case-control studies have shown a 2- to 4-fold increased risk of UGIB in patients treated with APAs. [5] Although less well documented, thienopyridines and VKAs also significantly increase the risk of GI bleeding. [6] (Table 1) APAs pose a greater risk compared to clopidogrel (RR: 1.45; 95% CI: 1.0–2.1; CAPRIE study). Among anticoagulants, DOACs increase the incidence of GI bleeding compared to VKAs, particularly in elderly patients. [7] Over the past decade, the role of antithrombotic agents in UGIB has increased, paralleling the rise in prescriptions. In a registry-based study conducted in the United Kingdom, the incidence of UGIB associated with antiplatelet drug (APD) use doubled between 1996 and 2002, while that associated with other antithrombotic agents tripled. During the same period, mortality from myocardial infarction significantly declined [8].

Table 1 Risk of ulcer-related hemorrhagic complications in patients treated with antithrombotic agents alone or in combination: results from the case-control study by Lanas et al. [6]

Treatments	Odds Ratio (95% CI) for Peptic Ulcer Bleeding
APD	3.7 (3.0-4.5)
Thienopyridines (clopidogrel-ticlopidine)	2.8 (1.9-4.2)
VKA	2.8 (2.1-3.7)
APD + non-selective NSAIDs	12.7 (7.0-23.0)
APD + COX-2 inhibitors	14.5 (3.3-63.9)
APD + thienopyridines	16.4 (5.4-49.7)
Thienopyridines + NSAIDs	15.2 (4.1-56.5)
VKA + NSAIDs	19.3 (8.2-45.3)

In our series, 51.6% of our cardiac patients were on anticoagulants, 41.9% on anticoagulants alone, 16.1% on aspirin alone, 16.1% on DAPT, and 9.7% on both APAs and anticoagulants.

The level of gastrointestinal risk associated with APD use is influenced by patient-related factors and concomitant therapies [9]. A history of PUD identifies patients at very high risk of UGIB. Among patients with a previous bleeding episode, the risk of recurrence while on APD therapy can reach up to 15% within one year. The gastrointestinal risk linked to APDs increases with age, reflecting primarily the independent effect of aging, regardless of aspirin use [10].

In the absence of additional risk factors, the risk of severe ulcer bleeding under APD therapy is low in individuals under 50 years of age, estimated at approximately 0.2% per patient-year, but rises to 1.3% in those over 80 years. Several epidemiological studies have shown that Helicobacter pylori infection significantly exacerbates the risk of ulcer complications in patients on APD therapy [11].

The combination of APDs with other antiplatelet agents or with VKAs further increases the risk of gastrointestinal bleeding. In patients receiving APDs, co-prescription of a selective NSAID such as a COX-2 inhibitor (coxib) confers a gastrointestinal risk comparable to that of traditional NSAIDs. Risk factors associated with other antithrombotic agents are less well defined. However, two high-risk situations should be emphasized: the combination of multiple antithrombotic agents and the presence of a history of PUD.[6]

In our study, common risk factors included advanced age, male sex, dyslipidemia, NSAID use, history of PUD, active smoking, and alcohol use.

According to the 2021 guidelines of the European Society of Gastrointestinal Endoscopy (ESGE), the management of antithrombotic therapy in the setting of acute upper gastrointestinal bleeding (UGIB) should be tailored to the indication and type of agent used.

In patients receiving antiplatelet monotherapy (aspirin) for primary prevention, treatment should be temporarily withheld and re-evaluated once hemostasis is achieved. In contrast, aspirin used for secondary prevention should generally be continued; if interruption is deemed necessary, it should be resumed within 3 to 5 days.

In cases of DAPT, aspirin should be maintained, while the second antiplatelet agent may be withheld and reintroduced within 5 days following hemostatic control.

For patients on anticoagulant therapy presenting with UGIB, temporary interruption of the anticoagulant is recommended. In situations of severe bleeding, reversal strategies such as administration of vitamin K, fresh frozen plasma (FFP), prothrombin complex concentrate (PCC), or specific reversal agents for DOACs may be considered. However, these measures should not delay endoscopic evaluation and therapeutic intervention. [12]

In hemodynamically stable patients with acute UGIB and underlying cardiac disease, the target post-transfusion hemoglobin concentration should be ≥ 10 g/dL [12].

PPIs should be initiated as soon as ulcer-related UGIB is suspected, without delaying EGD [13].

Endoscopic therapy is currently indicated for ulcers classified as Forrest Ia to IIb. Dual-modality endoscopic treatment, including diluted epinephrine injection combined with another technique (e.g., thermal coagulation or mechanical clipping), has been shown to be more effective than monotherapy [13].

In patients with persistent bleeding refractory to standard hemostatic techniques, topical hemostatic spray or powder and cap-mounted clips should be considered. If these fail, transarterial embolization (TAE) is recommended. Surgical intervention is indicated in cases of unsuccessful TAE [12].

Medical management involves high-dose intravenous PPI therapy—an 80 mg bolus followed by continuous infusion at 8 mg/h for 72 hours—especially in patients with high-risk ulcers (Forrest I, IIa, and IIb).

For patients with low-risk ulcers (Forrest IIc and III), once-daily PPI therapy (e.g., 40 mg/day) is sufficient [13].

In patients on APT with a history of PUD, eradicating *Helicobacter pylori* infection is recommended, alongside continuous PPI-based gastroprotection throughout the duration of APT. PPIs reduce residual gastrointestinal risk in this population [14,15].

Initiation of PPI-based gastroprotection is warranted in patients at increased risk for gastrointestinal complications, including those with a history of complicated or uncomplicated ulcers, combination therapy with another antithrombotic agent, concurrent corticosteroid or NSAID use (selective or non-selective), and age over 70 years [16].

Currently, no specific European recommendations exist for patients treated with anticoagulants alone [3].

For patients on VKAs, esomeprazole and lansoprazole have been associated with increased international normalized ratio (INR) levels; thus, alternative PPIs should be preferred.[17]

In patients receiving DOACs, PPI co-administration does not appear to alter the pharmacokinetics of these agents, and no restriction on PPI selection is necessary [3]. Based on pharmacological interaction studies, pantoprazole and rabeprazole are considered to have the lowest potential for drug interactions [18].

Following an episode of UGIB, a reduced dose of aspirin (75 mg) may be considered for patients who were previously receiving a higher dose (160 or 325 mg) [10]. Findings from the ADAPTABLE trial, presented at the European Society of Cardiology (ESC) Congress, showed no significant difference in secondary prevention after myocardial infarction between high- and low-dose aspirin regimens [19].

Anticoagulation should be resumed once bleeding has been controlled, ideally within 7 days or shortly after the hemorrhagic event, depending on the patient's thromboembolic risk. The fast onset of action of DOACs, compared to VKAs, should be taken into account in this setting.

The use of validated clinical risk assessment tools—such as the CHA_2DS_2-VASc score to estimate thromboembolic risk and the HAS-BLED score to assess bleeding risk—can assist in guiding clinical decision-making [12].

5. Conclusion

Managing UGIB in cardiac patients remains a challenge due to comorbidities and antithrombotic use. The vascular benefits of these drugs are offset by a significant risk of ulcer-related bleeding, especially in patients with ulcer history, combination therapy, corticosteroid or NSAID use, H. pylori infection, or age >70. Prevention relies on PPIs and H. pylori eradication when appropriate. Multidisciplinary management of APA and AC before and after UGIB must follow expert consensus and available guidelines, though scientific evidence is sometimes limited.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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