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# Upper Gastrointestinal Bleeding in Cardiac Patient: Etiology, Risk Factors and Management

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**Abstract** **Original Research Article**

Upper Gastrointestinal bleeding (UGIB) is a frequent emergency in hepato-gastroenterology, particularly severe in cardiac patients. The aim of this study was to investigate the etiologies, risk factors and management of upper gastrointestinal bleeding in these patients. We conducted a descriptive cross-sectional study in our department, between April 2020 and January 2024, including 56 cardiac patients who presented with UGIB and benefited from endoscopic exploration. The mean age of the patients was 69 years, with a male predominance. Of these, 46,4 % were on anticoagulants alone, 32,1% on antiplatelet agents alone, and 5,3% on a combination of the two. Gastro-duodenal ulcer was the cause of bleeding in 46.4% of patients. Blood transfusion was required in 29 patients (51,8%), and intensive care in 12 patients (21,4%). The evolution was marked by a recurrence during the same hospitalization in 10 patients (17.8%), and 3 patients (5,3%) died during the same hospitalization from hemorrhagic shock. In conclusion, cardiac patients present a higher morbidity and mortality in cases of digestive gastrointestinal bleeding, underlining the importance of rapid and effective management.

**Keywords:** Gastrointestinal bleeding, anticoagulants, antiplatelet agent.



## INTRODUCTION

Cardiac patients are at increased risk of upper Gastrointestinal bleeding (UGIB), due to the use of antiplatelet agents (APAs), antivitamin K (VKAs) and direct oral anticoagulants (DAAs). Managing this risk requires a careful assessment of the benefit-risk ratio between potential worsening of bleeding and thromboembolic risk, especially in these often comorbid patients. A multidisciplinary approach, involving both gastroenterologists and cardiologists, is required, based on the recommendations of learned societies or expert consensus for the management of PAAs and anticoagulants during bleeding.

The aim of our work is to study the etiologies, risk factors and management of upper GI bleeding in these patients.

## MATERIALS AND METHOD

This is a descriptive cross-sectional study, conducted in our department between April 2020 and January 2024.

We included 56 patients with cardiac disease who underwent Oeso-Gastro-Duodenal Endoscopy (EGDF) for HDH.

## RESULTS

The mean age of our patients was 69 years, with extremes of 24 and 90 years. There were 35 men (62,5%) and 21 women (37.5%), with a M/F sex ratio of 1,66.

21 patients (37,5%) had ischemic heart disease, 14 patients (25%) had arrhythmic heart disease, 6 patients (10,7%) had valvular heart disease and 15 (26,8%) had heart disease of other etiologies (3 cases of dilated Cardiomyopathy, 1 patient had a single ventricle and 8 patients followed for hypertensive heart disease and 3 for undocumented heart disease).

29 patients (51, 8%) were on anticoagulants (AC), of whom 26 patients (46,4%) were on AC alone, 21 (37,5 %) on Vitamin K antagonist (VKA) alone and 5 (8.9 %) on Direct Oral Anticoagulants (DOACs) alone.

18 patients (32,1%) were on antiplatelet agent alone, of whom 6 (10.7%) were on Aspirin alone, and 12

(21,4%) patients were on Dual antiplatelet therapy (DAPT).

3 patients (5,3%) were on antiplatelet agent in combination with anticoagulant. (2 VKAs and 1 DOACs). And 9 (16,1 %) were on no treatment at all.

In addition to heart disease, 23 patients (41,1%) had dyslipidemia on lipid-lowering therapy, and 15 (26,8%) were taking NSAIDs, a history of peptic ulcer disease in 11 patients (19,6%), active smoking in 11 patients (19,6 %), previous digestive bleeding in 6 patients (10,7%), cirrhosis in 3 patients (5,3%), and alcoholism in 3 patients (5,3%).

**Table 1: Summary table of risk factors found in our cardiac patients**

| Risk factors | N (%) |
|---|---|
| Dyslipidemia on lipid-lowering therapy | 23 (41,1 %) |
| NSAID use | 15 (26,8 %) |
| History of peptic ulcer disease | 11 (19,6 %) |
| Active smoking | 11 (19,6 %) |
| Previous digestive bleeding | 6 (10,7 %) |
| Cirrhosis | 3 (5,3 %) |
| Alcoholism | 3 (5,3 %) |

The reasons for hospitalization were dominated by melenas alone in 32 patients (57,1%), hematemesis + melenas in 14 patients (25%) and hematemesis alone in 10 patients (17,8%), of whom 12 (21,4%) were admitted with hemorrhagic shock and hemodynamic instability.

All our patients had their anticoagulants stopped as soon as they were admitted to the emergency department, in consultation with the cardiologists. At the same time, they benefited from non-specific resuscitation measures, with correction of the hemodynamic state by filling +/- blood transfusion, and medical treatment based on proton pump inhibitors (PPIs) 80 mg IVD then 8 mg/h SAP and/or octreotide (Sandostatin) in 7 patients (12,5%).

In fact, we had to resort to blood transfusion in 29 patients (51,8%), and to a stay in intensive care in the event of hemorrhagic shock in 12 patients (21,4%).

Oeso-Gastro-Duodenal Endoscopy was performed on all our patients, and the most frequent aetiology was peptic ulcer disease in 26 patients (46.4%), followed by severe oesophagitis in 9 patients (16,1%), erosive gastritis and/or bulbitis in 7 patients (12,5%), esophageal varices or sus cardial varices in 7 patients (12,5%), gastric or bulbar angiodysplasia in 4 patients (7,1%) and 2 cases of gastric tumours (3.6%) (Table 1).

Of the 26 patients in our series with peptic ulcer disease as etiology, 2 patients had Forrest Ib ulcers, 3 patients had Forrest IIb ulcers, 6 patients had Forrest IIc ulcers and 15 patients had ulcers classified as Forrest III.

**Table 2: Endoscopic lesions responsible for digestive hemorrhage in our cardiac patients**

| Endoscopic lesions | N (%) |
|---|---|
| Peptic ulcer disease | 26 (46,4) |
| Severe esophagitis | 9 (16,1) |
| Erosive gastritis/bulbitis | 7 (12,5) |
| VO or VSC | 7 (12,5) |
| Gastric or bulbar angiodysplasia | 4 (7,1) |
| Gastric tumor | 2 (3,6) |

16 patients (28,6 %) received endoscopic treatment: hemostatic clips with adrenaline injection in 5 patients (8,9%), esophageal varices ligation in 5 patients (8.9%), gastro-oesophageal varices (GOV) gluing in two patients (3.6 %) and argon plasma coagulation in 4 patients (7,14%).

The evolution of the bleeding episode was marked by recurrence during the same hospitalization in 10 patients (17.8%). 3 patients (5,3%) died during the same hospitalization due to hemorrhagic shock (including 1 patient with normal Oeso-Gastro-Duodenal Endoscopy who died before completing digestive exploration, and 2 patients with Forrest Ib and IIb bulbar ulcers). The evolution was favorable in 43 patients (76,8%), with cessation of bleeding and resumption of Ac in consultation with cardiologists.

## DISCUSSION

The risk of Gastrointestinal (GI) bleeding is multiplied by 1.8 (95% CI: 1.5-2.1), in a patient treated with low-dose aspirin, and up to 7.4 times (95% CI: 3.5-15) in the case of treatment with a antiplatelet agents [1].

Given the seriousness of GI bleeding in this population, knowledge of the risk factors predisposing to GI bleeding in cardiac subjects is important for the management of patients taking these treatments, and early detection of high-risk patients for preventive treatment, in order to reduce incidence and mortality.

Some studies have shown that male gender is a risk factor for the occurrence of high Non-variceal GI in patients taking aspirin or other non-steroidal anti-inflammatory drugs [2]. Multiple co-morbidities such as cirrhosis, renal failure, previous UGD or HP infection have been shown to be associated with the occurrence of GI in hospitalized elderly patients, as has the combined use of several drugs (NSAIDs, lipid-lowering agents, corticoids).

With regard to the management of anti-thrombotic molecules, and according to the recommendations of ESGE 2021 [3]:

For patients with acute UGIB on low-dose aspirin monotherapy for primary cardiovascular prophylaxis, aspirin should be temporarily discontinued and may be resumed after careful reassessment of its

clinical indication. But aspirin should not be interrupted if taken as monotherapy for secondary cardiovascular prophylaxis, and if for any reason it is interrupted, it should be resumed as soon as possible, preferably within 3 to 5 days.

For UGIB patients on dual antiplatelet therapy (DAPT). for secondary cardiovascular prophylaxis, aspirin should not be discontinued, but the second antiplatelet agent should be stopped and resumed as soon as possible, preferably within 5 days.

In patients with acute UGIB on antivitamin K (VKAs), the latter should be discontinued, concerning direct oral anticoagulants (DAAs): ESGE recommends that the anticoagulant should be withheld and endoscopy should not be delayed [3].

ESGE recommends the use of the Glasgow-Blat hford score (GBS) for risk stratification prior to endoscopy in patients with acute UGIB. Patients with a GBS score ≤ 1 have a very low risk of rebleeding and mortality, and can therefore be managed on an outpatient basis [3].

Immediate assessment of patients' hemodynamic status should be made with rapid intravascular volume replacement in the event of hemodynamic instability, initially using crystalloid solutions, as they reduce both mortality and major adverse renal events compared with saline [4, 5].

In hemodynamically stable patients with acute UGIB and a history of acute or chronic cardiovascular disease, a hemoglobin threshold ≤ 8 g/dl triggers red cell transfusion and a target post-transfusion hemoglobin concentration is ≥ 10 g/dl [3].

Pre-endoscopic treatment with high-dose intravenous PPIs should be considered in patients with acute UGIB, to reduce the stigma of bleeding during the endoscopic procedure and thus reduce the need for endoscopic treatment, but this should not delay early endoscopy.

After hemodynamic resuscitation, early upper GI endoscopy (≤ 24 hours) should be performed, and urgent (≤ 12 hours) emergent (≤ 6 hours) upper GI endoscopy are not recommended, as patient outcomes are no better than those of early endoscopy [6-13].

It is recommended that the Forrest (F) classification be used in all patients with peptic ulcer haemorrhage to differentiate low-risk from high-risk endoscopic stigmata, and does not recommend endoscopic haemostasis in patients with peptic ulcers with a flat pigmented spot (FIIc) or a clean base (FIII), as these stigmata present a low risk of rebleeding [3].

Peptic ulcers with jet or sheet bleeding (FIa and FIb respectively) or with a visible non-bleeding vessel (FIIa) should benefit from endoscopic hemostasis, as these lesions present a high risk of persistent or recurrent bleeding. ESGE suggests that peptic ulcers with an adherent clot (FIIb) should have the clot removed endoscopically. Once the clot has been removed, any identified underlying active bleeding (FIa or FIb) or visible non-bleeding vessel (FIIa) should undergo endoscopic hemostasis [3].

For patients with actively bleeding ulcers (FIa, FIb), combination therapy using epinephrine injection plus a second hemostasis modality (thermal or mechanical contact therapy).

For patients with ulcers with a visible non-bleeding vessel (FIIa), ESGE recommends the use of either thermal or mechanical therapy, as monotherapy or in combination with epinephrine injection. [3]

In patients with persistent bleeding refractory to standard haemostasis modalities, the use of a topical haemostatic spray/powder or cap-mounted clip should be considered; if this fails, trans-arterial embolization (TAE) is recommended. Surgery is indicated after failure of TAE [3].

High-dose PPIs therapy is recommended for patients who have undergone endoscopic haemostasis, and for patients with ulcer who have not been treated endoscopically [14, 15].

In patients suffering from UGIB secondary to peptic ulcer disease, it is recommended to check for Helicobacter pylori infection in the acute phase (during initial endoscopy) and to initiate appropriate antibiotic therapy if Helicobacter pylori is detected [3].

In patients requiring continuous anticoagulation following acute Non-variceal upper gastrointestinal bleeding, anticoagulation should be resumed as soon as bleeding has been controlled, preferably within 7 days of bleeding or shortly thereafter, depending on thromboembolic risk. The rapid onset of action of direct oral anticoagulants compared with vitamin K antivitamin K (VKAs), must be taken into account in this context [3].

The balance between ischemic and hemorrhagic risk is a key issue for these therapies, and a number of scores have been established. For anticoagulants, the HAS-BLED score is used to assess bleeding risk, and the CHA2DS2-Vasc score for ischemic risk [3].

Regarding the indication for preventive PPIs therapy in dual antiplatelet therapy, the latest European Society of Cardiology recommendations [16] recommend the administration of a PPIs in patients at high risk of gastrointestinal bleeding (a history of gastrointestinal ulcer or hemorrhage, with concomitant

anticoagulation, taking non-steroidal anti-inflammatory drugs or corticosteroid therapy or having at least two of the following factors: age > 65, dyspepsia, gastro-oesophageal reflux disease, Helicobacter pylori infection or chronic ethylism).

There is no specific European recommendation for patients treated with anticoagulants alone [17]. Patients with a history of gastrointestinal bleeding or ulcer, a high bleeding risk score or multiple risk factors for gastrointestinal bleeding (Helicobacter pylori infection, age 65, concomitant use of antiplatelet agents or non-steroidal anti-inflammatory drugs or corticosteroids), should benefit from gastroprotective PPI therapy [17].

## CONCLUSION

In cardiac patients, the risk of Upper Gastrointestinal bleeding is accentuated, particularly with the use of antithrombotic treatments such as dual antiplatelet therapy, and antivitamin K. Effective management of these patients requires a multidisciplinary approach, involving gastroenterologists, and cardiologists to balance thrombotic and hemorrhagic risk, and the importance of preventive treatment with proton pump inhibitors (PPIs) for patients at high risk of gastrointestinal bleeding on these therapies.

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