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| RESEARCH ARTICLE

Consequential Catastrophe: A Rare Interaction of Upper Gastrointestinal Haemorrhage and Acute Coronary Syndrome

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| ABSTRACT

Gastrointestinal (GI) bleeding is a common medical condition that results in up to 10% mortality rate. The severity of bleeding ranges from occult blood loss to massive hemorrhage, with the latter often resulting in rapid onset of anemia. Anemia is an independent risk factor for developing acute coronary syndrome (ACS). Here, we present a case of acute upper GI bleeding developing anemia, which led to ACS.

| KEYWORDS

ACS, Cardiology, UGIB

| ARTICLE INFORMATION

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1. Introduction

The gastrointestinal (GI) tract is a common source of acute hemorrhage requiring timely intervention. The upper GI tract is a more frequent source of hemorrhage compared to the lower tract (1,2). In some cases, severe GI hemorrhage can lead to a rapid decrease in the red blood cell count, impairing oxygen delivery and resulting in acute anemia (3). The relationship between anemia and cardiovascular disease is well-established in the literature, with anemia being recognized as an independent risk factor for cardiovascular events in the general population and patients with a previous history of acute coronary syndrome. The estimated prevalence of anaemia upon admission in cases of acute coronary syndrome (ACS) ranges from 10% to 43% among patients. (4).

The development of acute coronary syndrome (ACS) because of acute anaemia involves several interconnected mechanisms, with the reduction in oxygen-carrying capacity being a primary factor. This reduction leads to inadequate oxygen delivery to myocardial tissue. In response, the heart attempts to compensate by increasing its rate and contractility, which further elevates myocardial workload and oxygen demand. This heightened demand creates a significant mismatch between oxygen supply and demand, ultimately resulting in ischemic heart disease (5).

This case report discusses a patient developing acute anemia secondary to GI bleeding, which subsequently precipitated acute coronary syndrome. It highlights the critical need for prompt diagnosis and management of both the primary bleeding source and its systemic effects.

2. Case details

A 66-year-old South Asian male presented to the emergency department as a transfer from a private hospital with complaints of Black, starry stool for three days duration. The patient noted that the color started three days back and was associated with epigastric abdominal pain and nausea. The patient had a history of diabetes mellitus, hypertension, and previous non-ST elevation myocardial infarction (NSTEMI) in 2020. The patient attended a private health center where a complete blood count was done and showed a hemoglobin of 8.7g\dL and, therefore, was referred to a tertiary center for further management. On examination, the patient was pale, with a heart rate of 116 and a blood pressure of 100\88 mmHg. A rectal exam was done and showed black stool with no signs of fissure, hemorrhoids, abscess, or perianal disease.

The patient was started on omeprazole infusion and kept on a cardiac monitor with labs sent urgently. Suddenly, the patient started to experience mild chest discomfort, and an electrocardiogram (ECG) was done. The ECG showed ST Depression in V3 to V6 with reciprocal changes. ACS protocol could not be initiated, as the patient was currently being investigated for bleeding. Therefore, a cardiology consult and a diagnosis of Type 2 Myocardial infarction secondary to acute blood loss, with treatment to be determined after the diagnosis of underlying pathology. Furthermore, repeated investigations showed a hemoglobin of 5.4g\dL and elevated cardiac enzyme and troponin. Moreover, the patient had a urea of 15 mmol\L compared to creatinine of 66 umol\L. The patient had an emergency endoscopy showing multiple bleeding duodenal ulcers, which were coagulated. Blood transfusions were given, and the patient was admitted to the cardiac care unit. Moreover, the patient improved over time and was sent for a coronary angiogram, which showed minor coronary artery disease. Thus, it was determined that the case of NSTEMI is more likely related to acute blood loss rather than coronary occlusion.

3. Discussion

Despite the advancements in endoscopic intervention and the overall declining mortality rate associated with gastrointestinal hemorrhage (currently projected to be 5-10%, remaining stable over the previous decades (6-8)), it continues to pose a management challenge for healthcare systems around the world. A linear regression analysis done using the Centers for Disease Control and Prevention Wide-Ranging Online Data for Epidemiologic Research (CDC WONDER) database to show the projected mortality rates for UGIB in 2021, using the data collected throughout 2012 to 2019 showed an increase in mortality rates about UGIB, rates saw an increase from 3.3 per 100,000 to 4.3 per 100,000, seeing a year on year increase of 0.4-0.9 per 100,000 in mortality rates from 2012 to 2019, although it is important to note that these values may have been influenced by superimposed COVID-19 infections at the time due to the inflammatory process downregulating coagulation processes in the body leading to increased severity of bleeds (7). Non-variceal gastrointestinal bleeding and its associated complications, as outlined in this paper, continue to burden healthcare systems around the world.

UGIB refers to bleeding in the gastrointestinal tract anywhere from the mouth to the ligament of Treitz. Patients with acute upper gastrointestinal bleeding may present with varying symptoms, the most common being melena, which can develop with blood loss as small as 50ml or coffee-ground hematemesis or bright red vomitus in association with upper gastrointestinal hemorrhage, large UGIB can also rarely present with bright red blood in stools more commonly associated with colonic bleeds, however presentations such as dizziness and syncope should not be discounted in the context of UGIBs (9,10).

Peptic ulcers account for the majority cause of non-variceal UGIBs (28-67%), but this has seen a decline due to PPI use and triple therapy for H. pylori eradication, other causes include gastritis, esophagitis, Mallory-Weiss syndrome, NSAID use, and malignancy. Bedside investigations post a thorough history and physical exam for an acute presentation for a UGIB include a full blood count, electrolytes, a coagulation profile, and a group and crossmatch, a delayed drop in hematocrit and an elevated urea should be expected due to increased processing of digested blood proteins in the liver via the urea cycle in such patients. A diagnostic endoscopy should not be delayed for a low hematocrit as it has been shown to not increase the risk of cardiac events. An IV bolus of crystalloid fluid like normal saline can be used to correct hypovolemia and serve as an intravascular fluid replacement in hemodynamically unstable patients; blood transfusion should be considered if the hemoglobin drops below 7g/dl. A CT angiography, although not a first-line investigation for UGIBs, can be used as a non-invasive method to detect GI hemorrhage at a sensitivity of 89% and can detect bleeding at rates of 0.5ml per minute (10-12).

Target hemoglobin should be kept between 7 and 9 g/dl with a higher threshold for patients with ischemic heart disease. A high dose IV PPI should be used at presentation and continued till 72 hours post endoscopy to minimize rebleeding risk (initially 80 mg IV bolus and then an 8mg/hour continuous infusion). The Glasgow Blatchford score (shown as superior to the Rockall score (8) should be used for pre-endoscopy risk stratification for UGIB to help determine ICU admission and transfusion need; the Rockall score can be used as a predictor of one-month mortality (endoscopy is the gold standard for diagnosing UGIBs with a sensitivity

of 92-98%). Endoscopy should be offered within 24 hours for hemodynamically unstable patients. The European Society of Gastrointestinal Endoscopy (ESGE) also recommends using a pre-endoscopy single dose of IV erythromycin in patients with ongoing severe bleeds. It has been shown to improve visualization during the scope and decrease the number of days spent in the hospital. The use of the Forrest classification during the endoscopy can be used to guide management; the ESGE recommends that peptic ulcers with Forrest classification Ia/b or IIa, i.e., those actively bleeding or have a non-bleeding visible vessel should be managed with endoscopic hemostasis (not using epinephrine injection as a monotherapy). Ulcers with associated clots (IIb) should be considered for clot removal, and any subsequent bleeding can be treated with hemostasis. Ulcers classed as III/I have a low risk of rebleeding, and thus, patients can be managed with oral PPIs at home. In a non-acute setting, patients should be investigated for H. pylori. With patients on aspirin therapy, aspirin can be restarted after the endoscopy for patients with a low risk of re-bleeding and at day 3 for high-risk patients. (10-12)

Acute Coronary Syndrome (ACS) is a possible complication of UGIB, ACS leads to myocardial ischemia as a result of an ST elevation Myocardial infarction, non-ST-elevation Myocardial infarction, or Unstable angina. ACS can contribute significantly to mortality and morbidity on its own, much more when associated with a UGIB. One of the potential theories as to how a UGIB can lead to ACS is that during an acute GI bleed, there is a sharp drop in circulating blood volume due to bleeding in the gastrointestinal tract, which leads to a drop in cardiac output and subsequent reduction in the perfusion of the coronary arteries. Ischemia also leads to activation of the coagulation factors, which can lead to thrombus formation. These changes are likely to affect the stability of local pre-existing atherosclerotic plaques, which can then cause coronary ischemia and thus cause ACS. Some of the risk factors of an ACS episode during a UGIB include increased fibrinogen and an increased red blood cell volume distribution width (RDW), whilst hemoglobin plays a protective role (13,14). Fibrinogen is associated with an increased plaque burden and is seen elevated in patients with ACS post-UGIB, whilst low hemoglobin has been shown to worsen myocardial ischemia (8,14).

RDW elevation acts as an independent risk factor for mortality in ACS (8). In patients with ACS, a compensatory neurohumoral mechanism is triggered, which leads to an increase in angiotensin, norepinephrine, and vasopressin, as well as the production of erythropoietin, thus accelerating the production of erythropoiesis and increasing the number of immature red blood cells, increasing cell heterogeneity. ACS patients are frequently accompanied by partial or systemic inflammatory responses, which may cause iron utilization disorders and a decrease in bone marrow responsiveness to erythropoietin, suppress antiapoptotic effects, and promote cell maturation, resulting in an increase in the number of immature cells released into the peripheral circulation, thereby increasing red blood cell heterogeneity. Abnormal red blood cells may assist in the formation of myocardial fibrosis by inflammatory amplification, resulting in reduced cardiac oxygenation. (8)

There lies a large contraindication in the treatment of UGIBs complicated by ACS, as UGIB requires hemostatic treatment, whereas ACS requires antithrombotic therapy. ACS inhibits the use of endoscopy, even though endoscopic hemostasis is a quick and effective way to manage UGIB. Meanwhile, percutaneous coronary intervention (PCI) and antithrombotic therapy are critical therapies for ACS. Deaths in cases such as these can be chalked up to refractory heart failure or multi-organ failure in most cases, with a study quoting mortality as high as 20.78% (14).

A rise in troponin-I and white cell count has also been observed in patients who suffered mortality due to ACS as a result of a UGIB, an elevated white cell count, Troponin-I, and the usage of mechanical ventilation act as independent factors contributing to a poor prognosis, although it is important to consider that troponin-I (trop level 0.5ug/L) release has only been observed in select cases post UGIB as a marker of myocardial injury and ischemia, usually in patients over the age of 65 or presenting with a hemodynamically unstable bleed with quoted values as low as only 19% in a sample of 156 UGIB patients (15). Blood transfusion following a UGIB may cause systemic inflammation in the prothrombotic condition, increasing oxidative stress and paradoxically lowering oxygen supply, all of which might contribute to a poor prognosis. Furthermore, due to the danger of significant bleeding, ACS post-UGIB patients are often unable to undergo antithrombotic treatment and PCI on time. Furthermore, patients with ACS following GI hemorrhage tend to have poor baseline features, such as advanced age, which may be related to further poor outcomes. However, it is recommended that patients undergo an endoscopy before PCI, as this approach has been shown to have lower mortality. (13,14)

It is vital to identify risk factors such as older age, prior history of coronary heart disease or a prior ACS episode, preexisting anaemia (less than 8g/dl, for every 10 g/L decrease in haemoglobin, the mortality rate with cardiovascular events within 30 days increases by 21% (8,11)), and history of hypertension (puts them at a 4 times higher risk for inpatient myocardial injury than a non-hypersensitive patient due to the plaque creating abilities of hypertension due to vessel wall injury) (OR: 4.252, 95% CI: 1.149–15.730, P = 0.030), or a left ventricular ejection fraction (LVEF) of less than 68% (OR: 3.667, 95% CI: 1.085–12.398, P = 0.037) in patients with a UGIB to aid in decreasing the incidence of ACS in these patients as these can be considered as independent risk factors for myocardial injury in patients with UGIB, the majority are shown to not present with angina symptoms, with some centres reporting little to non in terms of ECG changes, further decreasing the likelihood of early diagnosis, making troponin-I and risk factor identification a vital part of the diagnosis and management of an ACS episode post UGIB (13,17,18) .

4. Conclusion

Upper Gastrointestinal bleeding is a significant medical emergency that requires timely intervention. Severe GI bleed can lead to acute anemia, which drastically impairs sufficient oxygen supply to myocardial tissue. This mismatch of oxygen supply and demand can precipitate ischemic heart disease. The presented case report focuses on a patient who developed acute anemia secondary to GI bleed, leading to acute coronary syndrome. Emphasizing the need for prompt management for both conditions. Management strategies for GI bleeding include timely endoscopy, fluid resuscitation, blood transfusions, and Proton Pump Inhibitors (PPIs). Despite the advancement in GI bleeding management, including endoscopic interventions, the morbidity and mortality rates remain a concern. Especially if it is accompanied by cardiovascular manifestation, the need to balance hemostatic management for UGIBs and antithrombotic interventions for ACS is crucial in this case. In clinical practice, early identification of the risk factors is critical in improving patient outcomes. Recognizing the potential of ACS in patients with anemia, prior cardiovascular events, advanced age, and hemodynamic instability should guide early intervention and close monitoring. Further research is needed to optimize treatment protocols and have a deeper understanding of the pathogenesis and associated risk factors. Guiding early recognition and timely management.

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