

Duodenal Ulcer Complicated with Massive Upper Gastrointestinal Bleeding: Case Report

Emanuel da Cruz ^{1*}, Indira Eugénia Eduardo ¹, Francisco Panzo ¹, André Domingos Fuacatino ¹, Stephany Lukeni e Silva ¹

¹ Intensive Care Unit, Clínica Multiperfil, Luanda, Angola.

* Correspondence: dacruzmanuel640@gmail.com.

Citation: Cruz E, Eduardo IE, Panzo F, Fuacatino AD, Lukeni e Silva S. Duodenal Ulcer Complicated with Massive Upper Gastrointestinal Bleeding: Case Report and Literature Review. Brazilian Journal of Case Reports. 2025 Jan-Dec;05(1):bjcr87.

<https://doi.org/10.52600/2763-583X.bjcr.2025.5.1.bjcr87>

Received: 20 April 2025

Accepted: 17 May 2025

Published: 20 May 2025

Abstract: This case report aims to present a severe manifestation of duodenal ulcer complicated by massive upper gastrointestinal bleeding and critically low hemoglobin levels, highlighting the importance of early recognition, immediate intervention, and a multidisciplinary approach in managing patients at imminent risk of hemodynamic instability. A 64-year-old male patient with a recent history of orthopedic surgery and exploratory laparotomy was admitted to the emergency department in a coma, presenting signs of hemodynamic instability, severe anemia (hemoglobin 2.8 g/dL), metabolic acidosis, elevated lactate levels, and clinical features of septic shock originating from a surgical wound infection. The patient was intubated, received fluid resuscitation and blood component transfusion, and was admitted to the intensive care unit (ICU). Emergency upper gastrointestinal endoscopy revealed an actively bleeding duodenal ulcer classified as Forrest IB, with abundant clots. Despite endoscopic treatment with adrenaline injection, hemodynamic instability persisted, necessitating surgical intervention. Exploratory laparotomy confirmed a 30 mm bleeding ulcer, which was treated with duodenorrhaphy and omentoplasty. The patient showed clinical improvement postoperatively and was transferred from the ICU to the surgical ward. This case underscores the importance of prompt diagnosis and timely intervention in patients with severe upper gastrointestinal bleeding due to duodenal ulcer.

Keywords: Duodenal ulcer; Hemorrhage; Transfusion.



Copyright: This work is licensed under a Creative Commons Attribution 4.0 International License (CC BY 4.0).

1. Introduction

Duodenal ulcer is a form of peptic ulcer that occurs in the first portion of the small intestine. It results from erosion of the duodenal mucosa due to the action of gastric acid and pepsin. The primary causes include *Helicobacter pylori* infection and prolonged use of non-steroidal anti-inflammatory drugs. Duodenal ulcer is one of the leading causes of upper gastrointestinal bleeding, and in severe cases, it can result in significant blood loss and hemodynamic instability [1]. The incidence of upper gastrointestinal bleeding is estimated to be 100 to 150 cases per 100,000 inhabitants per year, with peptic ulcers accounting for approximately 50% of these cases [1].

Clinically, patients with upper gastrointestinal bleeding may present with hematemesis, melena, and hematochezia. Additional symptoms include dizziness, syncope, and signs of hypovolemic shock in severe cases [1, 2]. Initial diagnosis is based on clinical and hemodynamic assessment, followed by laboratory tests including hemoglobin and hematocrit levels. Upper gastrointestinal endoscopy is the diagnostic method of choice to identify the bleeding source and allow endoscopic hemostatic therapies. Treatment of upper gastrointestinal bleeding caused by duodenal ulcer includes hemodynamic stabilization with fluid resuscitation and blood transfusions, administration of proton pump inhibitors,

and eradication of *H. pylori* when present. In refractory cases or those with recurrent bleeding, surgical intervention may be necessary [3, 4].

Prognosis depends on the severity of bleeding, response to treatment, and patient comorbidities. Mortality associated with upper gastrointestinal bleeding ranges from 2% to 14%, being higher in elderly patients or those with persistent hemodynamic instability [1]. This case report aims to present a severe manifestation of duodenal ulcer with massive upper gastrointestinal bleeding and critically low hemoglobin levels, emphasizing the importance of early recognition, immediate intervention, and a multidisciplinary approach in the management of patients at imminent risk of hemodynamic instability.

2. Case Report

A 64-year-old male patient presented with a history of orthopedic surgery approximately one month prior, involving open reduction and internal fixation of the right leg with the placement of an external fixator due to tibia and fibula fractures, as well as exploratory laparotomy for hemoperitoneum. He also reported self-medication with non-steroidal anti-inflammatory drugs (NSAIDs).

His symptoms began 24 hours prior to hospital admission and were characterized by decreased level of consciousness and marked mucocutaneous pallor. According to family members, he had passed dark-colored stools on multiple occasions, which had not initially been given due attention. Due to persistent clinical deterioration, he was brought to the emergency department by his relatives. On examination, the patient was comatose, with a Glasgow Coma Scale score of 6. Pupils were isochoric and reactive to light, with no signs of focal neurological deficits. He was bradypneic and hypoxemic on room air (SpO₂ 60%), presenting with sinus tachycardia, arterial hypotension (BP: 80/46 mmHg; MAP: 57 mmHg), heart rate of 130 bpm, capillary refill time >3 seconds, dry and pale mucosa and skin, and hypothermia (body temperature: 34°C). The surgical wound on the midline supra- and infraumbilical region was in the healing phase, dry, with well-approximated edges and no discharge. The right lower limb showed an external fixator, local edema, and purulent discharge with foul odor from the pin sites.

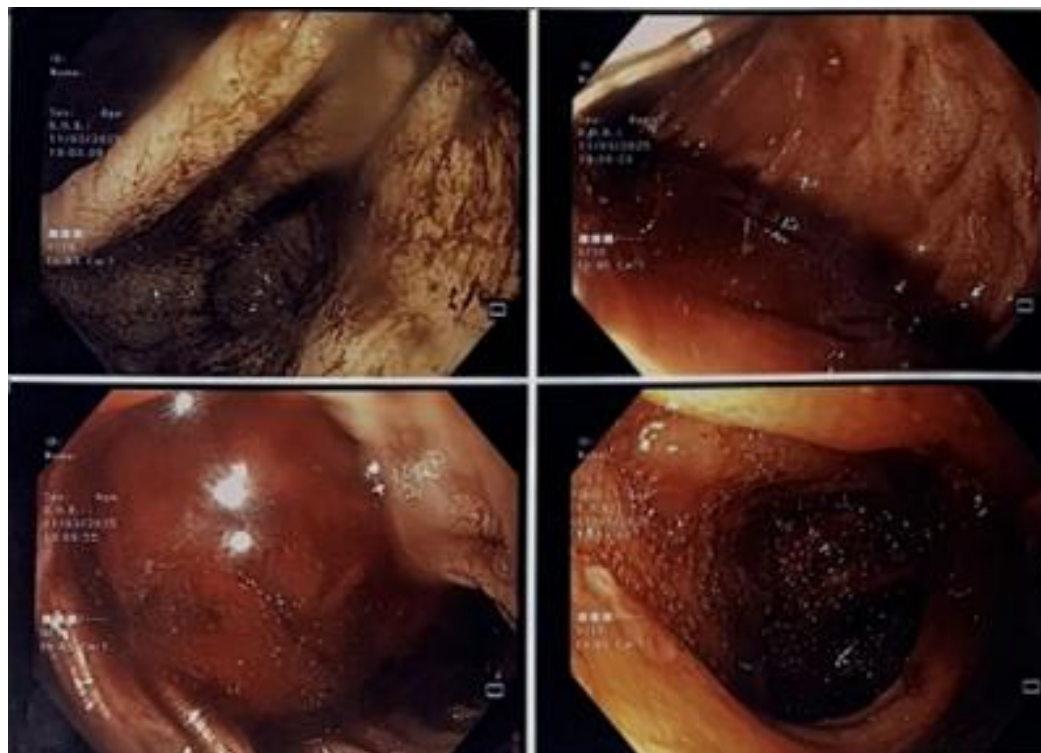
The laboratory evaluation revealed severe anemia, with a hemoglobin level of 2.8 g/dL and hematocrit of 7.7%. Leukocytosis was present, with a total leukocyte count of 86,030/mm³, accompanied by a balanced distribution of neutrophils and lymphocytes (49.3% each). Platelet count was markedly elevated at 479,000/mm³. Arterial blood gas analysis indicated significant metabolic acidosis, with a pH of 7.12, low bicarbonate (HCO₃⁻) at 7.6 mmol/L, reduced total CO₂ (7.0 mmol/L), and a base excess of -22.9. Compensatory respiratory alkalosis was observed, with a pCO₂ of 19.3 mmHg. Despite the acid-base imbalance, oxygenation parameters were preserved (pO₂: 101.0 mmHg; O₂ saturation: 96%). Lactate levels were markedly elevated at 11.7 mmol/L, indicating tissue hypoperfusion and possible septic shock. Microbiological culture of the leg wound secretion isolated *Acinetobacter* spp., which was sensitive to meropenem and amikacin.

In the emergency department, the working diagnosis was shock of undetermined etiology. Rapid sequence orotracheal intubation was performed, and the patient was resuscitated with fluid therapy and a 1:1 transfusion ratio of packed red blood cells and fresh frozen plasma. Empirical antibiotic therapy with vancomycin, piperacillin, and tazobactam was initiated following the collection of specimens for culture. Complementary laboratory and clinical findings confirmed the diagnosis of mixed shock (hemorrhagic and septic) associated with multiorgan dysfunction.

The patient was transferred to the intensive care unit (ICU), where he remained intubated and under mechanical ventilation with continuous analgo-sedation using ketamine and propofol. Hemodynamic instability persisted, requiring vasopressor support with norepinephrine at 0.23 mcg/kg/min, and further transfusions of blood products were administered. An urgent upper gastrointestinal endoscopy was performed 12 hours after ICU admission, revealing a distensible but deformed duodenal bulb with a deep ulcer exhibiting active oozing (Forrest IB). The lesion was well-defined, with a smooth surface

and a large volume of adherent clots, extending beyond the duodenal sweep and measuring approximately 30 mm. Endoscopic treatment with epinephrine injection was performed (Figure 1).

Figure 1. Distensible, deformed duodenal bulb with a deep, oozing ulcer classified as Forrest IB, well-defined, smooth surface with a large amount of coagulum, extending past the duodenal knee, measuring approximately 30 mm.



During the clinical course, the patient exhibited worsening hemodynamic instability due to a significant drop in hemoglobin levels despite transfusion of blood products. Culture of purulent discharge from the right leg revealed growth of *Acinetobacter* spp., sensitive to meropenem and amikacin. Antibiotic therapy was subsequently adjusted according to the susceptibility profile, with meropenem initiated. Following a discussion with the surgical team, the patient was taken to the operating room, where an exploratory laparotomy was performed with the following relevant findings: a well-defined, oozing duodenal ulcer, with a large amount of coagulum, measuring 30 mm, and melena within the intestinal lumen. Duodenorrhaphy and omentoplasty were performed without complications (Figure 2).

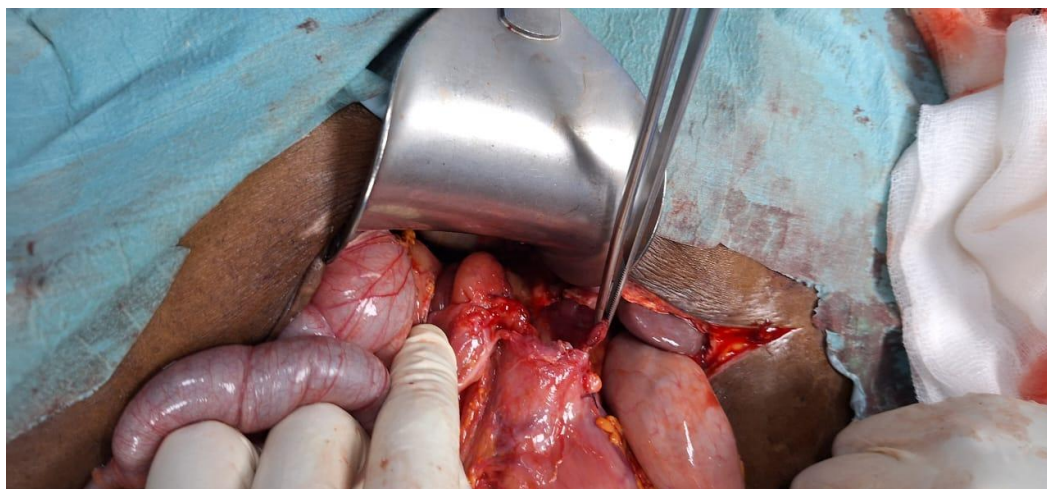
The patient showed a favorable clinical evolution following surgical intervention, with progressive hemodynamic stabilization and resolution of the acute condition. Post-operative recovery was uneventful. He remained under close supervision by both the intensive care and surgical teams, who oversaw rehabilitation and monitoring for potential complications. Improvement of the soft tissue infection was observed after adjustment of the antibiotic regimen. The patient remained clinically stable, without significant deficits, and was subsequently transferred to the surgical ward.

3. Discussion and Conclusion

This case describes a 64-year-old male patient with a history of polytrauma treated with orthopedic surgery and a recent exploratory laparotomy, who developed massive upper gastrointestinal bleeding (UGIB) secondary to a deep duodenal ulcer (Forrest IB classification), associated with severe hemodynamic instability, multiorgan dysfunction,

and mixed (septic and hemorrhagic) shock. The etiology of the ulcer in this context is multifactorial. The patient had significant risk factors, including recent use of nonsteroidal anti-inflammatory drugs (NSAIDs) at home and physiological stress conditions following major surgical interventions. From a clinical standpoint, the presentation was severe and consistent with high-volume gastrointestinal bleeding, as evidenced by melena, hypotension, altered level of consciousness, bradypnea, shock, and a critically low hemoglobin level (2.8 g/dL). These findings were accompanied by severe metabolic acidosis (pH 7.12), marked elevation of lactate (11.7 mmol/L), and signs of tissue hypoperfusion, compatible with profound shock and multiorgan failure.

Figure 2. Well-defined duodenal ulcer with a large amount of coagulum, measuring 30mm.



The concomitant presence of soft tissue infection in the right lower limb (external fixator with purulent discharge and foul odor) and marked leukocytosis suggest that the septic shock was associated with an orthopedic source of infection, potentially exacerbating the hemodynamic instability and contributing to multisystem failure. An emergency upper gastrointestinal endoscopy, performed after initial stabilization in the intensive care unit (ICU), confirmed a bleeding duodenal ulcer (Forrest IB), initially treated with epinephrine injection. Endoscopic therapy failure was evidenced by persistent hemodynamic instability and progressive decline in hemoglobin levels, warranting surgical intervention. Laparotomy revealed a 30 mm actively bleeding duodenal ulcer, which was managed with duodenorrhaphy and omental patch repair. The subsequent clinical course was favorable, with hemodynamic stabilization, resolution of organ dysfunction, and improvement of the soft tissue infection following empirical antibiotic adjustment (vancomycin, piperacillin and tazobactam).

Several strategies could have minimized the risk of hemorrhagic complications in this patient: closer monitoring for signs of occult bleeding (such as early melena and gradual hemoglobin drop); early assessment of gastrointestinal bleeding risk, particularly in patients with multiple risk factors (NSAIDs, trauma, abdominal surgery, mechanical ventilation); and patient and caregiver education on the dangers of indiscriminate NSAID use at home after major surgery.

The literature indicates that complicated peptic ulcers are responsible for up to 50% of UGIB cases, with NSAID use being one of the leading precipitating factors, especially in elderly and hospitalized patients. Moreover, failure of initial endoscopic therapy occurs in up to 20% of cases, with surgical intervention indicated in cases of recurrent or refractory bleeding, as illustrated in this report. The presence of concomitant septic shock further compromises prognosis, and successful treatment depends on early recognition,

timely intervention, and coordinated multidisciplinary critical care management, as demonstrated in this case [5].

Funding: None.

Research Ethics Committee Approval: We declare that the patient provided informed consent and that the study complied with the ethical guidelines established by the Declaration of Helsinki.

Acknowledgments: None.

Conflicts of Interest: The authors declare no conflicts of interest.

References

1. Ket SN, Sparrow RL, McQuilten ZK, Gibson PR, Brown GJ, Wood EM. Critical peptic ulcer bleeding requiring massive blood transfusion: outcomes of 270 cases. *Intern Med J*. 2021;51(12):2042–50. doi:10.1111/imj.15009.
2. Segovia Lohse HA, Segovia Lohse HR. Upper gastrointestinal bleeding. In: Coccolini F, Catena F, editors. *Manual of Emergency General Surgery*. Cham: Springer; 2023. p. 1–7. doi:10.1007/978-3-031-22599-4_69.
3. Laine L, Jensen DM. Management of patients with ulcer bleeding. *Am J Gastroenterol*. 2012;107(3):345–60. doi:10.1038/ajg.2011.480.
4. Kurokawa T, Arikawa T, Sano T, Nonami T. Surgical Treatment: Sugiura Procedure and Hassab Operation. In: Obara K, editor. *Clinical Investigation of Portal Hypertension*. Singapore: Springer; 2019. p. 433–42. doi:10.1007/978-981-10-7425-7_43.
5. MacLaren R, Dionne JC, Granholm A, et al. Society of Critical Care Medicine and American Society of Health-System Pharmacists Guideline for the Prevention of Stress-Related Gastrointestinal Bleeding in Critically Ill Adults. *Crit Care Med*. 2024;52(8):e421-e430. doi:10.1097/CCM.0000000000006330.