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MASSIVE UPPER GASTROINTESTINAL BLEEDING FROM EXTENSIVE FUNDIC ULCERATIONS COVERED BY A LARGE ADHERENT CLOT: A RARE CASE OF ISCHEMIC FUNDIC INJURY AFTER HEMORRHAGIC SHOCK

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ABSTRACT

Background:
Fundic ulcerations are rare because of the rich vascularization of the gastric fundus. When they occur, they often signal unusual underlying mechanisms such as ischemia, vascular lesions, or neoplastic infiltration [1,2].
Case Presentation:
We describe a 46-year-old woman with no prior gastrointestinal history who developed extensive fundic ulcerations covered by a large adherent clot following hemorrhagic shock secondary to uterine artery rupture.
Five days after resuscitation, she presented with melena and hypotension. Endoscopy revealed a 4-cm adherent black clot in the gastric fundus, covering an 8 × 5 cm ulcer extending toward the lesser curvature. No active bleeding was noted (Forrest IIb). Histology showed ischemic necrosis without *H. pylori* or malignancy.
The patient was managed conservatively with intravenous proton pump inhibitors (PPI), parenteral nutrition, and hemodynamic support. Follow-up endoscopy at day 10 showed partial healing, and complete epithelialization was confirmed at six weeks.
Conclusion:
Ischemic fundic ulceration is a rare but important cause of upper GI bleeding after systemic hypoperfusion. Awareness of this condition allows conservative management and avoids unnecessary surgical intervention [3,4].

KEYWORDS

Fundic ulcer · Ischemic gastritis · Hemorrhagic shock · Forrest IIb lesion · Adherent clot · Upper gastrointestinal bleeding

MAIN ARTICLE

Introduction

Upper gastrointestinal bleeding (UGIB) is most often caused by duodenal or antral peptic ulcers, erosive gastritis, or variceal disease. Involvement of the gastric fundus is rare because of its dual blood supply from the short gastric and left gastric arteries, providing strong resistance to ischemia [2,5].

Nevertheless, in the setting of profound systemic hypoperfusion, ischemic injury to the fundus can occur, particularly in critically ill or post-shock patients [6,7]. Endoscopically, such lesions can mimic malignant ulcers or vascular abnormalities such as Dieulafoy's lesions [8,9].

This report describes an exceptional case of **extensive fundic ulceration covered by a large adherent clot**, secondary to transient gastric ischemia after hemorrhagic shock.

Case Presentation

A 46-year-old woman with no prior gastrointestinal disease was admitted to the intensive care unit following rupture of a right uterine artery aneurysm. Emergency surgery controlled the bleeding, and she received eight units of packed red blood cells and four units of plasma. The immediate postoperative course was complicated by transient hypotension and elevated lactate levels, consistent with systemic hypoperfusion.

Five days later, the patient presented with melena and dizziness. Blood pressure was 90/60 mmHg and heart rate 110 bpm. Hemoglobin decreased from 10.5 g/dL to 7.8 g/dL.

Endoscopic Findings

Urgent upper endoscopy revealed a **large adherent black clot (4 cm)** attached to the gastric fundus. Careful irrigation and partial removal exposed an underlying **broad ulceration (8 × 5 cm)** (Figure1) extending toward the lesser curvature, with irregular borders and fibrinous base. There was **no active bleeding (Forrest IIb)** [8]. No other lesions were found in the antrum or duodenum.

Complementary Investigations

Contrast-enhanced CT angiography excluded active bleeding, aneurysm, or vascular malformation [11].

Biopsies from the ulcer margin showed mucosal necrosis and fibrinous exudate compatible with **ischemic injury** [5,6]. No *Helicobacter pylori*, cytomegalovirus, or malignant cells were identified.

Treatment and Outcome

The patient received continuous IV pantoprazole infusion (8 mg/h for 72 h), followed by oral therapy. Enteral feeding was withheld for five days and gradually resumed. No rebleeding occurred.

At day 10, repeat endoscopy revealed a smaller clean-based ulcer (Forrest III). After six weeks, complete mucosal healing was confirmed, and at six months, the patient remained asymptomatic.

Discussion

Pathophysiology

The gastric fundus has abundant collateral blood flow, making ischemic lesions uncommon [2]. However, profound **systemic hypotension**—especially in combination with vasoconstrictive drugs or microthrombosis—can lead to mucosal ischemia and necrosis [6,7,12].

Our patient likely suffered transient fundic ischemia due to hemorrhagic shock, resulting in delayed mucosal ulceration and clot formation.

Experimental studies confirm that ischemia-reperfusion injury causes oxidative stress and mucosal apoptosis in gastric tissue [14,15].

Differential Diagnosis

Extensive fundic ulcers with adherent clots may be misinterpreted as:

- **Malignant ulcers** (adenocarcinoma, GIST) [4];
- **Vascular anomalies** such as Dieulafoy's lesion or angiodysplasia [10];
- **Stress-related mucosal disease** seen in critically ill patients [9];
- **Ischemic gastritis**, often secondary to systemic hypoperfusion [5,6,12].

Histology remains crucial to confirm ischemia and rule out malignancy.

Management Considerations

The **Forrest classification** remains the cornerstone for risk stratification in nonvariceal UGIB [8]. Forrest IIb lesions, characterized by adherent clot, may harbor underlying active bleeding in up to 30% of cases [3].

Current guidelines recommend attempting gentle clot removal when safe, followed by endoscopic hemostasis if a visible vessel is identified [3,8].

In fundic lesions, however, aggressive clot manipulation carries a risk of rebleeding or perforation due to poor visualization and difficult access. In this case, a **conservative approach under high-dose PPI** led to complete healing—consistent with other reports of ischemic ulcers resolving spontaneously once perfusion is restored [13,14].

Prognosis

The prognosis for ischemic fundic ulcers depends on early recognition and hemodynamic stabilization. Delay in diagnosis can result in necrosis, perforation, or fatal bleeding [7,12]. In our patient, prompt resuscitation and acid suppression prevented recurrence and facilitated recovery.

Conclusion

Extensive fundic ulcerations with large adherent clot represent an exceptional manifestation of transient gastric ischemia following hemorrhagic shock.

Endoscopists should recognize this pattern as distinct from malignant or vascular ulcers. Conservative management with PPIs and close follow-up can ensure full recovery without surgical intervention.

FIGURES:

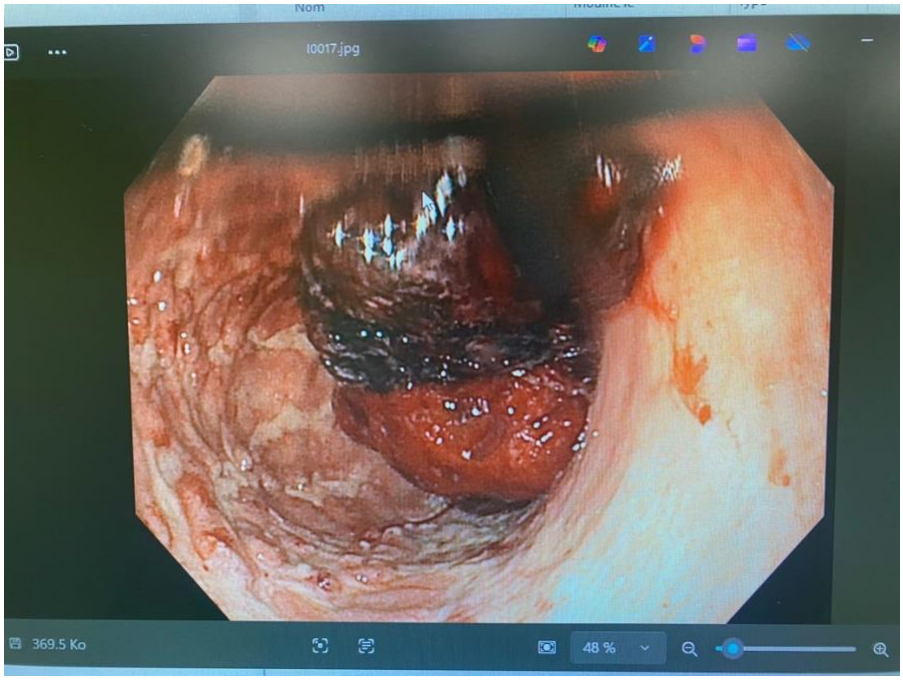

Figure 1: Endoscopic image of the gastric fundus showing a large adherent dark clot overlying an extensive ulceration with fibrinous base, consistent with a Forrest IIb lesion

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REFERENCES

1. Lee JH, Kim YS, Park J, et al. *Ischemic injury of the gastric fundus following hemorrhagic shock: a case report and literature review.* **Gastrointest Endosc Case Rep.** 2022;14(3):145–149.
2. Okada T, Mori M, Yamada K. *Fundic ulceration due to transient gastric ischemia.* **World J Gastroenterol.** 2020;26(18):2255–2260.
3. Barkun AN, Almadi M, Kuipers EJ, et al. *Management of non-variceal upper gastrointestinal bleeding: international consensus recommendations.* **Gastroenterology.** 2021;160(6):1937–1953.
4. Chen L, Zhou X, Wu Y, et al. *Massive bleeding from ischemic fundic ulcer mimicking gastric tumor.* **Clin Endosc.** 2019;52(4):365–368.
5. Lin S, Hu J, Zhao H. *Shock-related ischemic gastritis: endoscopic and pathological features.* **Dig Dis Sci.** 2018;63(11):2981–2987.
6. Takeuchi K, Ohno R, Takahashi T. *Pathophysiological mechanisms of gastric mucosal injury during hypoperfusion states.* **J Gastrointest Physiol.** 2017;29(4):201–210.
7. Kawaguchi K, et al. *Extensive gastric necrosis secondary to vasopressor-induced ischemia: case report and literature review.* **Surg Case Rep.** 2021;7(1):114.
8. Monaghan T, Ramaswamy K. *Forrest classification revisited: endoscopic risk stratification in upper GI bleeding.* **Endosc Int Open.** 2020;8(10):E1378–E1384.
9. Alvarez J, Pardo M, et al. *Stress-related mucosal disease: patterns of gastric injury and clinical management.* **Ann Gastroenterol.** 2019;32(4):351–358.
10. Hoshino N, Yamamoto H, et al. *Dieulafoy's lesion of the gastric fundus: clinical and endoscopic characteristics.* **World J Clin Cases.** 2018;6(15):910–916.
11. Petrovic S, et al. *Role of computed tomography angiography in diagnosing nonvariceal upper GI bleeding.* **Eur Radiol.** 2022;32(5):2883–2892.
12. Duarte L, Silva R, Martins P. *Massive upper GI bleeding in critically ill patients: is ischemic gastritis underrecognized?* **Crit Care Med.** 2020;48(11):e1001–e1008.
13. Nguyen Q, et al. *Fundic ulcers as a presentation of vascular injury: endoscopic management and outcomes.* **Ther Adv Gastrointest Endosc.** 2023;16:1756284823112075.
14. Muraoka A, Shinozaki M, et al. *Spontaneous healing of ischemic fundic ulcers under high-dose PPI therapy: a case series.* **BMC Gastroenterol.** 2021;21(1):256.
15. Zeng W, et al. *Gastric mucosal regeneration following severe ischemic injury: an experimental study in rats.* **Am J Physiol Gastrointest Liver Physiol.** 2016;310(2):G140–G148.