

Enema-induced rectal injury presenting as upper gastrointestinal bleeding: a case report

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Case Report

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Additional Declarations: No competing interests reported.

Abstract

A 61-year-old man developed coffee-ground emesis and melena on postoperative day 10 after undergoing craniotomy for intracerebral hemorrhage. Initial evaluation suggested upper gastrointestinal bleeding, and gastroscopy was performed at the referring hospital, but no definite bleeding source was identified. The patient was then transferred to our hospital. CT angiography performed at our institution demonstrated active rectal bleeding. Subsequent bedside sigmoidoscopy revealed a superficial rectal lesion located 4–6 cm from the anal verge, and hemostasis was achieved by endoscopic clip application. Further history-taking disclosed recent glycerin enema administration by family members. Rectal mucosal injury related to improper enema use was considered the most likely cause of the severe bleeding. The patient recovered after conservative management without recurrent bleeding.

Background

In emergency practice, coffee-ground emesis and melena usually direct early clinical suspicion toward upper gastrointestinal bleeding. In postoperative neurosurgical patients, gastrointestinal bleeding is commonly attributed to stress-related mucosal disease or coagulation abnormalities[1], whereas anorectal procedure-related injury is less frequently considered [2]. Enema therapy is commonly used in patients who are confined to bed for prolonged periods and is generally regarded as a safe treatment for constipation. However, rare but serious complications may still occur [3], including rectal injury and massive bleeding[4], may occur. We report a postoperative neurosurgical patient who developed severe gastrointestinal bleeding secondary to rectal mucosal injury following glycerin enema administration. The initial manifestations were coffee-ground emesis and melena, leading to an initial misdiagnosis of upper gastrointestinal bleeding.

Case

A 61-year-old man was transferred to our emergency department because of suspected gastrointestinal bleeding 10 days after craniotomy for intracerebral hemorrhage. According to the referral history, he had developed coffee-ground emesis and melena. Upper gastrointestinal bleeding was initially suspected, and gastroscopy had been performed at the referring hospital, but no definite bleeding source was identified.

On arrival at our emergency resuscitation room, the patient was critically ill and unable to provide history because of impaired consciousness. Given the discrepancy between the clinical presentation and the negative gastroscopy findings, further evaluation was undertaken. Contrast-enhanced CT angiography demonstrated active rectal bleeding(Fig. 1). Urgent bedside sigmoidoscopy was then performed. Visualization was initially obscured by a large amount of blood clots(Fig. 2). After digital evacuation of the clots, a superficial oozing rectal lesion was identified approximately 4–6 cm from the anal verge(Fig. 3). Hemostasis was successfully achieved by endoscopic clip application.

After bleeding control, further questioning of the family revealed that a glycerin enema had been administered shortly before the onset of symptoms. Enema-related rectal mucosal injury was therefore considered the most likely cause of the bleeding episode. The patient was treated conservatively after endoscopic hemostasis and recovered without recurrent bleeding.

Discussion

This case is noteworthy not simply because the patient had rectal bleeding after enema administration, but because the initial clinical picture strongly suggested an upper gastrointestinal source[5]. In previously reported cases of enema-related rectal injury or routine anorectal instrumentation, patients typically present with immediate painless hematochezia[6], often with large-volume fresh blood per rectum[7]. By contrast, our patient first manifested coffee-ground emesis and melena, and the relevant enema history was disclosed only after endoscopic hemostasis. This combination created substantial diagnostic bias toward upper gastrointestinal bleeding and delayed recognition of a distal rectal source.

In retrospect, the early diagnostic orientation toward upper gastrointestinal bleeding was clinically understandable[8]. Neurocritical care patients are recognized to be at increased risk of stress-related mucosal disease and overt upper gastrointestinal bleeding[9], which is why coffee-ground emesis in this setting is usually interpreted as evidence of an upper gastrointestinal lesion[10]. Neurocritical care literature also supports the use of stress ulcer prophylaxis precisely because this population is considered vulnerable to stress-related upper gastrointestinal complications.

However, in the present case, subsequent localization by contrast-enhanced CT and bedside sigmoidoscopy supported a distal rectal bleeding source as the major lesion. The upper gastrointestinal–like manifestations should therefore be interpreted cautiously. Rather than proving that lower gastrointestinal bleeding routinely presents as upper gastrointestinal bleeding in neurologically impaired patients, this case suggests that symptom-based localization may become unreliable when severe neurological dysfunction coexists with major gastrointestinal dysmotility and limited clinical history.

A plausible pathophysiological explanation begins with the neurocritical state itself. Following intracerebral hemorrhage, craniotomy, prolonged coma, and immobilization, the patient likely developed profound autonomic and gastrointestinal motility disturbances[6]. Reduced vagal activity, sympathetic overactivation, delayed gastric emptying, diminished intestinal propulsion, and even paralytic ileus are well-recognized components of gastrointestinal dysfunction in severe neurological illness. Under these conditions, intraluminal blood may not follow the expected aboral transit pattern. Instead, prolonged retention within the bowel, altered pressure gradients, and impaired gastrointestinal transport may distort the temporal sequence and external appearance of bleeding.

The distal rectal lesion in this patient appeared superficial and oozing rather than spurting, making a relatively slow but persistent bleeding pattern plausible. Slow bleeding is important because it allows sufficient time for blood to remain within the gastrointestinal tract and undergo chemical modification.

Blood retained in the stomach is exposed to hydrochloric acid, resulting in hemoglobin denaturation and the formation of hematin, which produces the characteristic coffee-ground appearance. Blood retained within the intestine for a sufficient duration may also lead to melena. From this perspective, the coexistence of coffee-ground emesis and melena does not necessarily prove a primary upper gastrointestinal lesion; instead, it indicates that blood remained within the gastrointestinal tract long enough to be transformed before evacuation.

That said, the mechanism of coffee-ground emesis in this case should not be overstated. Several possibilities must be acknowledged. First, profound gastrointestinal dysmotility may have promoted abnormal retention and retrograde redistribution of intraluminal blood. Second, impaired swallowing and reduced consciousness may have altered the way blood was retained or observed. Third, although the initial gastroscopy reportedly failed to identify a definite bleeding source, a transient or self-limited upper gastrointestinal lesion cannot be completely excluded. A similar principle has been described in patients with severe autonomic dysfunction after spinal cord injury, in whom gastrointestinal bleeding may be clinically delayed, muted, or otherwise atypical. Accordingly, the mechanism proposed here should be regarded as a pathophysiological inference rather than a proven explanation.

The case also highlights the practical importance of procedural history. The turning point was not the initial symptom pattern, but the delayed disclosure of caregiver-administered glycerin enema after endoscopy. This temporal association does not prove causality in a histopathological sense, but it substantially strengthens the inference that the distal rectal lesion was procedure-related. In the existing literature, enema-associated rectal injury has been linked to mucosal tears, ulceration, perforation, sepsis, and occasionally massive bleeding, particularly in debilitated or neurologically impaired patients[11]. The present case broadens that spectrum by suggesting that delayed and misleading upper gastrointestinal–like manifestations may occur even in the absence of immediate post-enema fresh bleeding.

From a clinical standpoint, the most important lesson is not that lower gastrointestinal bleeding “mimics” upper gastrointestinal bleeding as a general rule, but that, in selected neurocritical patients, the outward pattern of bleeding may be insufficient for source localization. In such circumstances, clinicians should resist premature anchoring to a presumed upper gastrointestinal source, remain alert to iatrogenic anorectal injury, and repeatedly revisit caregiver-provided history when the initial account is incomplete or inconsistent with subsequent findings.

In summary, this case expands the recognized presentation spectrum of enema-related rectal injury. It suggests that, in the setting of severe neurological impairment, delayed gastrointestinal transit, altered consciousness, and incomplete procedural history may collectively produce a misleading bleeding phenotype. The key implication is therefore diagnostic humility: when symptom pattern and objective localization diverge, the latter should guide interpretation, while any mechanistic explanation should be presented as biologically plausible rather than conclusively established.

Conclusion

This case demonstrates that, in a neurocritical patient, enema-related distal rectal injury may be associated with a misleading upper gastrointestinal bleeding–like presentation. It also underscores that symptom pattern alone may be insufficient for source localization when severe neurological impairment, gastrointestinal dysmotility, and incomplete procedural history coexist. Careful reassessment of caregiver-performed interventions is therefore essential in complex bleeding presentations.

Declarations

Authors' contributions

The authors confirm contribution to the paper as follows: Study conception and design: YW W and S W; data collection: DP Y; draft manuscript preparation:

ML H. All authors reviewed and approved the final version of the manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Ethics approval and consent to participate

This case report was approved by the Ethics Committee of the Second Affiliated Hospital, Zhejiang University School of Medicine (2026-0328), and was conducted in accordance with the Declaration of Helsinki. Written informed consent for publication was obtained from the patient's guardian.

Consent for publication

Written informed consent for publication of their clinical details and clinical images was obtained from the patient's guardian. A copy of the consent form is available for review by the Editor of this journal.

Competing interests

The authors declare no competing interests.

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Figures

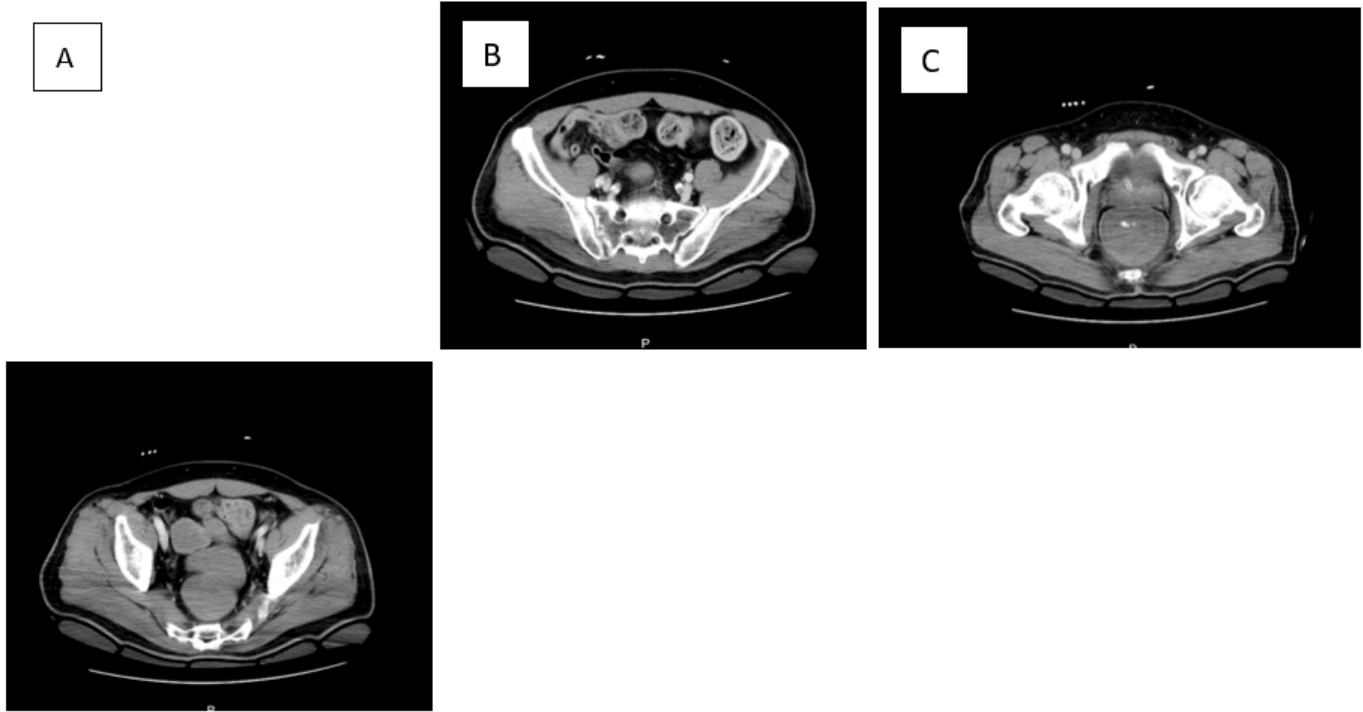


Figure 1

Contrast-enhanced CT angiography finding. **A** Colonic blood accumulation. **B** A large amount of blood accumulation in the rectum. **C** The extravasation of the contrast agent at the end of the rectum.

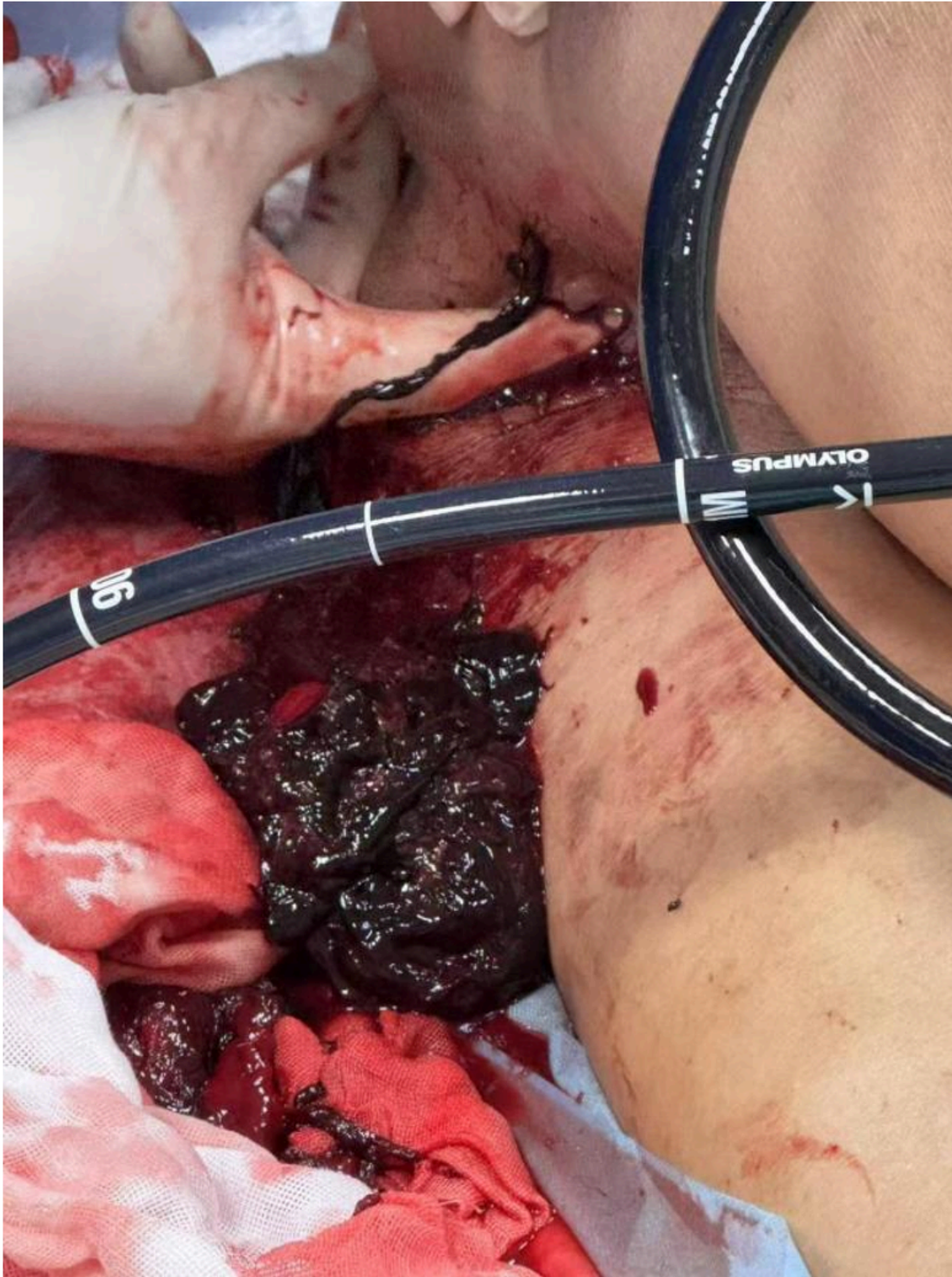


Figure 2

Large amount of blood clots manually evacuated from the rectum prior to endoscopic visualization.

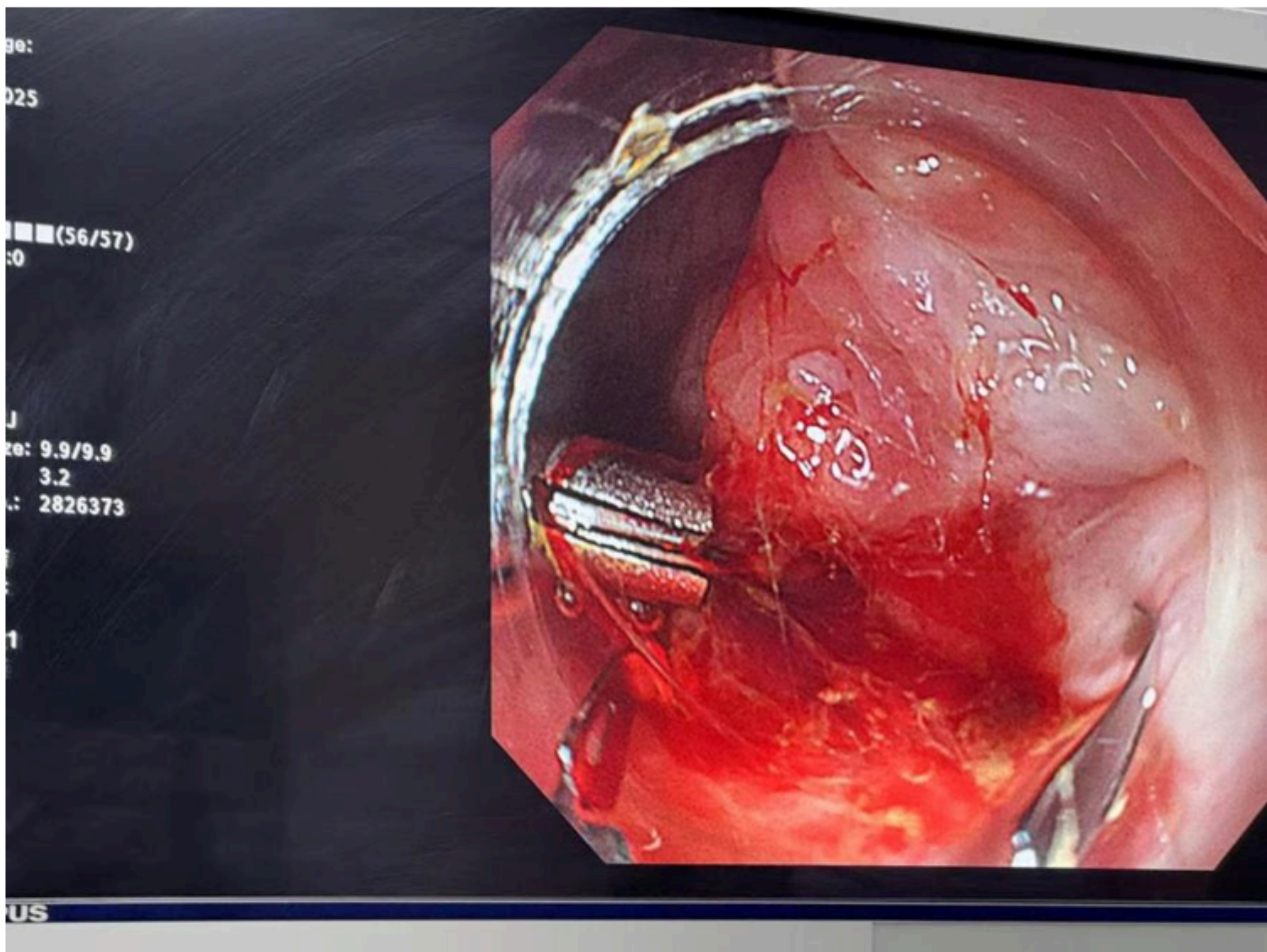


Figure 3

Endoscopic view showing a superficial bleeding lesion in the distal rectum and attempted hemostasis using endoscopic clips.