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Case Report

Duodenal ulcer double jeopardy: a case report of sequential bleeding and perforation

Azra Tabassum*, Summiaya Ali, Selwyn Selvendran

Department of General Surgery, Campbelltown Hospital, New South Wales, Australia

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***Correspondence:**
Azra Tabassum,
E-mail: azraalam2001@hotmail.com

ABSTRACT

We reported a rare case of a 78-year-old woman who presented with acute duodenal ulcer bleeding requiring emergency surgical intervention. Despite initial control of the bleeding, she subsequently developed a new duodenal ulcer perforation, which necessitated prompt reoperation. This case highlights the importance of timely surgical intervention and meticulous clinical decision-making in managing complex gastrointestinal emergencies in an acute surgical setting.

Keywords: Bleeding duodenal ulcer, Duodenal ulcer perforation, Emergency surgical intervention

INTRODUCTION

Peptic ulcer disease (PUD) affects millions globally and commonly presents with bleeding or perforation, two major life-threatening complications.[1,2] Traditionally, these events are viewed as distinct entities; classical surgical teaching suggests that bleeding ulcers rarely perforate and perforated ulcers rarely bleed, furthermore, Blackford and his associates in 1940's concluded as well "bleeding ulcers don't perforate; and ulcer perforations don't bleed.[3,4] However, recent literature identifies occasional overlap, including simultaneous anterior perforation and posterior bleeding (such as kissing ulcers), sealed perforations, and giant duodenal ulcers with dual complications highlight the potential severity of duodenal ulcer disease.[5-7] This reflecting distinct underlying pathophysiology and the typically divergent clinical trajectories of these complications. Nevertheless, a small but important subset of patients develop progressive ulcer deterioration, in which initial haemorrhage is followed by subsequent structural failure of the duodenal wall, culminating in perforation. This sequential progression, where a duodenal ulcer first bleeds and later perforates, remains distinctly uncommon and sparsely reported.

Acute duodenal ulcer bleeding is a life-threatening emergency requiring rapid resuscitation and intervention.[8] Although the incidence of peptic ulcer disease has declined.[9] Endoscopic therapy is first line; however, surgery is indicated for persistent bleeding or complication such as perforation.[10] Here, we presented a case of sequential bleeding followed by duodenal perforation and discuss its pathophysiological implications, diagnostic challenges, and management considerations. The present case adds to the limited literature describing this sequential progression.

CASE REPORT

A 78-year-old woman presented to Campbelltown Hospital, a 306-bed non-tertiary hospital serving a population of 176,519 in Southwestern Sydney, with acute duodenal ulcer bleeding. Her medical history was significant for a recent hysterectomy for endometrial cancer, chemotherapy, and radiotherapy. On admission, on arrival, she was hemodynamically unstable (systolic BP 80 mmHg, HR 120 bpm) and required urgent resuscitation with intravenous fluids and blood transfusions.

Initial management

The patient was managed according to the hospital's upper GI bleed protocol, which include the following.

Resuscitation

IV fluids and blood transfusions were given to stabilize vital signs.

Risk assessment

Evaluation of bleeding risk factors, including age, comorbidities, and medications was done.

Endoscopy

Urgent endoscopy to identify and treat the bleeding lesion was performed.

Surgical consultation

Early involvement of the surgical team to discuss potential operative intervention was provided.

Surgical intervention

Despite initial endoscopic attempts, bleeding persisted, and the patient underwent emergency midline laparotomy. A duodenostomy was performed to control the hemorrhage. The bleeding gastroduodenal artery was under-run, and the duodenum was closed with 3.0 polydioxanone (PDS) sutures.

Postoperative course

On postoperative day 4, bile drainage suggested duodenal leak. Relaparotomy revealed a new duodenal ulcer perforation lateral and inferior to the duodenostomy site. This was managed with patch repair, pyloric exclusion, and gastrojejunostomy. The patient recovered steadily in the ICU and ward, despite developing a VRE wound infection, which was appropriately treated.

DISCUSSION

Sequential duodenal ulcer bleeding followed by early perforation within a short time frame is an uncommon but clinically significant pattern. Although isolated reports exist, the phenomenon remains poorly characterised.[11] Our case adds to a small but growing body of international literature describing patients who achieve initial hemostasis, whether endoscopically or surgically and yet develop a new perforation within days. This accumulation of cases underscores that although rare, the phenomenon is repeatedly observed across diverse.

In a brief literature review, five comparable cases were identified in which perforation followed initial control of duodenal ulcer bleeding within a short interval.[12-16] In these reports, involved patients aged 55-75 years who underwent endoscopic or surgical hemostasis and subsequently developed duodenal perforation between postoperative days 1 and 7, with all patients ultimately requiring operative intervention. Notably, the new perforation occurred separate from the duodenostomy site, suggesting a multifocal pathophysiology rather than breakdown of the operative repair. Similar findings were observed in published cases, where perforation developed at new ulcer sites after both endoscopic and surgical hemostasis. This pattern suggests that the risk of delayed perforation is not mitigated simply by achieving hemostasis, and that factors intrinsic to the patient or underlying ulcer pathology may drive continued tissue breakdown

The underlying mechanisms are likely multifactorial: persistent mucosal ischemia, may compromise mucosal defence and contribute to tissue necrosis, as described in classical models of peptic ulcer pathophysiology.[17] The same review emphasizes that impairments in mucosal defence (e.g., from ischemia) may be more critical than aggressive factors like acid in ulcer development. A recent surgical study identified that low albumin and tissue oedema (fluid extravasation) are associated with worse healing after duodenal perforation repair, suggesting that mucosal injury and microvascular compromise contribute to tissue weakening.[18] Ongoing exposure to gastric acid, bile, and pepsin can exacerbate this process, particularly in regions with impaired perfusion (e.g., after surgical stress).[19]

Hemodynamic instability, ongoing mucosal ischemia, high acid load, and stress-related mucosal injury, NSAID-related mitochondrial injury, *H. pylori* inflammation, or immunosuppression can predispose an already vulnerable duodenum to delayed perforation despite initial hemostasis.[20-22] Complex comorbidities, delayed presentation, and prior abdominal surgeries further compound the risk and challenge timely diagnosis.

Several factors distinguished our case from earlier reports. First, our patient had recent pelvic radiotherapy and chemotherapy, both of which impair mucosal regeneration and microvascular integrity. Radiation-induced endarteritis, fibrosis, and local ischemia may predispose to delayed ulcer extension and perforation. Second, she presented in significant hemorrhagic shock, and profound early mucosal hypoperfusion may have contributed to subsequent tissue breakdown. Third, she required operative management for the initial bleed, reflecting both the severity of hemorrhage and the limited response to endoscopy.

Our case also underscores several important principles relevant to clinical practice.

First, early postoperative deterioration may be subtle, and vigilance is essential even after apparently successful hemostasis. Regular clinical assessment, close biochemical monitoring, and a low threshold for early cross-sectional imaging are fundamental to detecting evolving perforation. Especially those with large ulcer craters, hemodynamic instability at presentation, or persistent epigastric pain may warrant closer post-bleed monitoring for signs of evolving perforation. Second, coordinated management between gastroenterology and surgical teams plays a critical role in optimizing outcomes, particularly when patients deviate from expected recovery trajectories. Finally, the integration of standardized postoperative protocols and clear escalation pathways may support earlier recognition of complications and facilitate prompt intervention.

Across reported cases, the time frame for subsequent perforation overlaps closely with the early postoperative period when patients may appear clinically stable. This highlights the need for heightened vigilance during the first week following treatment of significant duodenal ulcer bleeding, especially in high-risk groups such as older patients, those with recent chemoradiation, or those who require operative control of hemorrhage. Low thresholds for repeat imaging, serial abdominal examinations, and early surgical reassessment may facilitate timely identification of evolving perforation.

Despite her high-risk profile, our patient ultimately recovered after reoperation, although her postoperative course was complicated by a VRE wound infection. Her outcome reinforces that prompt recognition and aggressive surgical management remain critical for favourable results. While sequential bleeding and perforation remains an uncommon pattern, recognizing this possibility facilitates earlier diagnosis and can significantly improve outcomes in complex gastrointestinal emergencies, particularly in elderly or medically fragile patients.

CONCLUSION

The sequential occurrence of bleeding followed by perforation suggests that duodenal ulcer complications may lie on a continuum in select patients, driven by persistent local ischemia and failure of mucosal restitution. This case therefore serves as a reminder that a seemingly stabilized ulcer may still progress to catastrophic complications. It also emphasizes the need for vigilant clinical assessment, timely imaging, and multidisciplinary care. Reporting such cases contributes to the growing understanding of this uncommon but clinically significant pattern and may assist clinicians in early recognition of similar presentations in the future.

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