

Iliac Arterioenteric Fistula Presenting as Ileus: A Catastrophic Late Complication of Pelvic Surgery and Radiotherapy

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ABSTRACT

Background: Arterioenteric fistulas are rare but life-threatening causes of gastrointestinal hemorrhage, most commonly occurring after aortic or aortoiliac reconstruction. Iliac artery–enteric fistulas without prior vascular surgery are exceptionally uncommon and may be difficult to recognize, particularly in a radiation-altered pelvis.

Case Report: A 54-year-old male with prior low anterior resection and adjuvant chemoradiotherapy for rectal cancer presented with ileus-like symptoms two weeks after inguinal hernia repair. After initial improvement with conservative management, he developed sudden abdominal pain, hemodynamic collapse, and massive hematochezia. During colonoscopy, diffuse intraluminal hematoma and hemorrhagic content were observed throughout the lumen; however, no definite active bleeding source was identified. After the procedure, the patient developed hemodynamic instability, which progressed to hemodynamic collapse. Emergency laparotomy revealed massive hemorrhage from an arterial defect at the iliac bifurcation adherent to small bowel within a radiation-fibrotic pelvis, consistent with an iliac arterioenteric fistula/arterial erosion. Proximal vascular control and definitive repair were performed, along with extensive small-bowel resection and stoma formation.

Conclusions: Iliac arterioenteric fistulas can occur without prior vascular reconstruction and may initially mimic ileus, even in the absence of herald bleeding. Non-localizing endoscopy should not exclude the diagnosis in patients with abrupt deterioration. While endovascular techniques aid stabilization, open surgical repair remains essential when bowel compromise or contamination is present.

Keywords: Iliac arterioenteric fistula, Gastrointestinal hemorrhage, Pelvic radiotherapy, Ileus

Introduction

Arterioenteric fistulas represent a rare but devastating cause of gastrointestinal hemorrhage, typically presenting with abrupt hemodynamic collapse and carrying high mortality if diagnosis and definitive management are delayed.¹⁻³ In most contemporary series, secondary arterioenteric fistulas are encountered in the setting of prior aortic or aortoiliac reconstruction, where graft infection, anastomotic degeneration, or pseudoaneurysm formation predispose

to erosion into adjacent bowel.⁴⁻⁵ In contrast, iliac artery–enteric fistulas without preceding vascular reconstruction are distinctly uncommon and therefore may not be immediately suspected during initial assessment.^{3,6}

Pelvic surgery and radiotherapy can create a “hostile pelvis” characterized by obliterated tissue planes, chronic inflammation, and dense fibrosis.⁶ These late radiation-related changes may increase vulnerability to vascular injury and facilitate pathologic adher-

ence between bowel and major pelvic vessels, predisposing to arterial erosion and fistulization.⁶⁻⁸ Clinically, this mechanism can obscure the diagnosis, particularly when early symptoms mimic benign postoperative conditions such as ileus or adhesive small-bowel obstruction, and when endoscopic evaluation fails to localize a discrete bleeding point.^{1,3,9}

Herein, we report a case of massive intraluminal hemorrhage arising from findings consistent with an iliac arterioenteric fistula/arterial erosion into an adherent small-bowel loop in a radiation-fibrotic pelvis, presenting initially as ileus in a patient with prior low anterior resection and adjuvant chemoradiotherapy. This case underscores the need for heightened suspicion and rapid multidisciplinary intervention when patients with prior pelvic oncologic therapy deteriorate abruptly, even in the absence of a herald bleed.

Case Presentation

A 54-year-old male presented to the emergency department with progressive abdominal pain, distension, nausea, and absence of flatus and stool for the preceding 3–4 days. His symptoms had begun approximately four days after undergoing an elective, open extraperitoneal left inguinal hernia repair two weeks prior to admission, performed without entry into the peritoneal cavity.

His medical history was significant for hypertension and diabetes mellitus. Of note, in 2019, he had undergone low anterior resection with protective loop ileostomy for rectal adenocarcinoma, followed by neoadjuvant chemoradiotherapy. The ileostomy had been closed later the same year. There was no history of prior vascular surgery.

On examination, the abdomen was distended with tenderness predominantly in the hypogastric region and left upper quadrant. Bowel sounds were hypoactive. Nasogastric decompression yielded approximately 700 mL of gastric content. Laboratory evaluation revealed a leukocyte count of 8,600/mm³, a hemoglobin level of 11.6 g/dL and C-reactive protein level of 60.8 mg/L. Upright abdominal radiography demonstrated multiple air–fluid levels consistent with ileus. Contrast-enhanced computed tomography (CT) of the abdomen and pelvis performed at presentation likewise demonstrated findings consistent with small-bowel ileus, without evidence of bowel wall ischemia, active extravasation, or vascular abnormality.

The patient was admitted with a diagnosis of postoperative adhesive ileus and managed conservatively for six days with bowel rest, intravenous fluids, and nasogastric decompression. Serial abdominal radiographs showed partial regression of air–fluid levels, and oral intake was gradually resumed.

However, shortly before planned discharge, the patient developed sudden severe abdominal pain, loss of consciousness, and hypotension. He was immediately transferred to the intensive care unit. Rectal examination revealed massive hematochezia. Emergency colonoscopy demonstrated extensive intraluminal hematoma throughout the colon and terminal ileum without

identification of a discrete bleeding focus.

Immediately following colonoscopy, the patient developed hemodynamic collapse with clinical signs of shock. Given the rapid deterioration and suspicion of hemorrhagic shock, the patient was promptly intubated in the intensive care unit and transferred emergently to the operating room for exploratory laparotomy. In view of the presumed ongoing hemorrhage, a decision for surgical intervention was made without delay, and conservative measures were not awaited for clinical response.

Upon midline laparotomy, markedly distended small bowel loops were encountered. Approximately 100 cm proximal to the ileocecal valve, a small bowel segment was found densely adherent within the pelvis. Enterotomy at this level revealed massive fresh arterial bleeding within the lumen. Further dissection along the adhesion plane demonstrated an actively bleeding arterial defect at the level of the iliac bifurcation adjacent to the aortic bifurcation. The affected arterial segment was in close contact with the adherent small bowel loop. Temporary hemorrhage control was achieved by digital compression. Given the dense radiation-induced fibrosis and limited local tissue planes, direct primary repair at the bleeding site was not feasible.

The cardiovascular surgery team was urgently involved. Due to dense fibrosis and adhesions at the infrarenal level, the abdominal aorta could not be identified and accessed at this site. Therefore, following Kocherization of the duodenum, proximal exposure of the aorta was achieved, and the aorta was clamped at a more proximal level. Primary repair of the iliac bifurcation injury was performed without the need for graft interposition, reflecting an adequate caliber of healthy residual vessel wall. After clamp release, no further active bleeding was observed.

Inspection of the small bowel revealed transmural ischemia and perforation extending from approximately 150 cm distal to the ligament of Treitz to 30 cm proximal to the ileocecal valve. The involved segment was resected. A proximal end ileostomy was created in the left lower quadrant, and the distal bowel was exteriorized as a mucous fistula in the right lower quadrant. Two intra-abdominal drains were placed in the pelvis and Morrison pouch, and the abdomen was closed in anatomical layers. Intraoperatively, the patient received approximately 3–4 units of packed red blood cells. Postoperative hemoglobin was 6.6 g/dL.

The patient was transferred intubated to the intensive care unit for postoperative management; however, despite aggressive resuscitative efforts, the patient died 7 hours after surgery.

Discussion

Massive gastrointestinal hemorrhage originating from arterial–enteric communication is an uncommon but frequently fatal clinical event. Arterioenteric fistulas account for a small proportion of such cases and are most often encountered as late complications of open or endovascular aortic reconstruc-

tion.^{2,4,5,11} In this context, pathological processes such as prosthetic graft infection, anastomotic degeneration, pseudoaneurysm formation, or progressive mechanical erosion may ultimately create a communication between the vascular lumen and adjacent bowel.^{4,5} Consequently, much of the available literature has focused on graft-related fistulas involving the aorta—most commonly the duodenum—where prompt recognition and multidisciplinary management remain critical for survival.^{1-3,10,12}

The present case is important because it diverges from that common paradigm. Iliac artery–enteric fistulas are far less frequently reported than classic aortoenteric fistulas, and the absence of prior vascular reconstruction can markedly reduce clinical suspicion.^{3,14} Literature also highlights that iliac–enteric variants are often recognized only after catastrophic bleeding or intraoperative discovery.^{3,5,14} Accordingly, the educational value of this case lies not only in the diagnosis itself but also in demonstrating how an iliac arterioenteric communication can arise outside the typical post-graft setting.

A first striking feature is the atypical initial presentation. While “herald bleeding” is frequently described in AEF literature, it is not universal, and the absence of a herald bleed should not falsely reassure clinicians.^{1,9} In our patient, symptoms were initially consistent with ileus, followed by abrupt hemodynamic collapse and massive hematochezia. Similar diagnostic pitfalls are repeatedly stressed in the literature: endoscopic evaluation may be non-localizing, and clinical deterioration can be sudden, leaving limited time for stepwise diagnostic pathways.^{1,3,9} This reinforces a central principle from earlier series; AEF is a diagnosis of suspicion, not a diagnosis of confirmatory testing alone.^{2,3,11} As in our case, radiation-induced adhesions and dense fibrosis not only predispose to fistula formation but also create substantial technical challenges in exposing vascular anatomy and performing definitive vascular repair.

The second major point concerns pathogenesis in a hostile pelvis. Unlike graft-related fistulas, our case is best interpreted through the lens of pelvic oncologic therapy: prior pelvic surgery and radiotherapy can result in obliterated tissue planes, chronic inflammation, and dense fibrosis that predispose to intimate adherence between bowel and major pelvic vessels.^{6,7} In addition, these post-radiation changes may significantly hinder identification of normal anatomic landmarks, complicate vascular control, and increase the risk of intraoperative morbidity. Importantly, prior case experiences support this mechanism: iliac–enteric fistulation has been described after pelvic irradiation in oncologic settings, and anti-angiogenic therapy (e.g., bevacizumab) has also been associated with iliac artery–enteric fistula formation in patients treated for pelvic malignancies.^{6,8} Collectively, these reports strengthen the plausibility of radiation- and therapy-related vascular injury as a non-graft pathway to fistulization.

A third point is the diagnostic pattern observed in this case. Endoscopy revealed a diffuse intraluminal hematoma extending to the terminal ileum without a discrete bleeding focus, a finding that can occur when hemorrhage is brisk, intermittent,

or originates from a proximal arterial communication.^{1,3,9} Prior publications on AEF consistently emphasize false-negative or non-definitive endoscopy and the potential for delayed diagnosis; some reports describe the need for repeated evaluations or alternative modalities when conventional tools fail to localize bleeding.^{9,13} In practice, the combination of abrupt shock, massive GI bleeding with non-localizing endoscopy, and relevant risk factors (hostile pelvis, prior radiotherapy, malignancy therapies) should prompt early escalation to cross-sectional imaging when feasible and, in unstable patients, a low threshold for operative exploration.^{3,6,9,14}

Management experience in literature also contextualizes why this case is notable. Multiple reports and small series describe endovascular hemorrhage control covered stents, plugs, and embolic agents as a valuable emergency strategy, often serving as a bridge to definitive surgery.^{6,10,15} However, longer-term outcomes can be limited by rebleeding, persistent contamination, or graft infection, and several authors advocate staged or hybrid approaches depending on physiology and contamination burden.^{5,10,12} In our patient, the presence of bowel ischemia/perforation and the need for definitive abdominal intervention made emergency laparotomy unavoidable; thus, open vascular control and repair, coordinated with vascular surgery, was the most appropriate route. This aligns with the broader lesson from the endovascular series: minimally invasive control is powerful for stabilization, but definitive source control is essential when bowel compromise or contamination is present.^{10,12,15}

Prior case reports of iliac arterioenteric fistulas underscore the same themes: rarity, diagnostic delay, and the need for rapid multidisciplinary intervention.^{3,14} Reports involving iliac or aortoiliac graft–enteric fistulas illustrate how massive lower GI bleeding can be the first major manifestation and how staged endovascular-to-open strategies can be lifesaving.¹² Meanwhile, oncologic and irradiation-associated cases highlight that the initiating insult may be radiation vasculopathy, tumor-related erosion, or therapy-related tissue fragility rather than prosthetic infection.^{6,7,8} By adding a presentation dominated by ileus physiology and a radiation-fibrotic pelvis, our case expands the clinical spectrum in which iliac arterioenteric fistula should be considered.

A recent case report by Isaac et al. described a similar radiation-induced iliac arterioenteric fistula in a patient with prior pelvic radiotherapy for cervical carcinoma, in whom massive hemorrhage was successfully controlled with an endovascular covered stent as a bridge to definitive management.¹⁶ Our case differs from this report in that the dominant initial presentation was ileus rather than overt bleeding, and definitive management required emergency open repair with extensive bowel resection rather than an endovascular-first strategy.

In conclusion, when interpreted in the context of the existing literature, this case underscores that iliac arterioenteric fistulas, may develop in the absence of prior vascular reconstruction, particularly in patients with a hostile, radiation-altered pelvis. The clinical presentation can be misleading, initially

mimicking ileus and occurring without a herald bleed, thereby delaying suspicion. Endoscopic evaluation may fail to localize the source despite extensive intraluminal hematoma, making diagnosis heavily dependent on clinical vigilance and recognition of risk factors. Ultimately, management must be individualized; while endovascular strategies offer valuable rapid hemorrhage control in unstable patients, open surgical intervention remains essential when bowel compromise or intra-abdominal contamination necessitates definitive source control. This case reinforces that arterioenteric fistula should be considered in any patient with a history of pelvic surgery and radiotherapy who develops sudden, unexplained hemodynamic instability, even without a preceding herald bleed.

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