

A Case Report of Delayed Patchy Bowel Necrosis Due to Nonocclusive Mesenteric Ischemia Despite a Functioning Stoma

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Case Report

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Abstract

Background

Small bowel obstruction in the elderly carries high morbidity, and postoperative stoma complications and non-occlusive mesenteric ischemia pose difficult diagnostic challenges. This case is novel because it reports a sequential and paradoxical scenario: superficial stomal mucosal necrosis that resolved without surgery, immediately followed by silent non-occlusive mesenteric ischemia that developed proximal to a healthy, functioning stoma while therapeutic anticoagulation was being administered, exposing a critical clinical pitfall.

Case presentation:

An 80-year-old woman with hypertension and ischemic heart disease presented with severe abdominal pain, distension, and vomiting. Examination revealed tachycardia, hypotension, and abdominal tenderness. Laboratory tests showed a white blood cell count of 10,500/ μ L, elevated lactate, and elevated C-reactive protein. Computed tomography demonstrated small bowel obstruction with signs of ischemia. Emergency laparotomy identified 180 cm of gangrenous ileum caused by an adhesion band. Because of intraoperative hemodynamic instability, a double-barrel ileostomy was performed. On postoperative day 2, superficial mucosal necrosis appeared above the stoma fascia; the stoma remained functional and lactate normalized, so conservative management was pursued. On postoperative day 4, the patient developed an abrupt decline in consciousness without abdominal signs or change in stoma output. A second laparotomy revealed 50 cm of patchy gangrenous bowel 20 cm proximal to the patent stoma, consistent with non-occlusive mesenteric ischemia. The necrotic segment was resected and the stoma revised. The patient stabilized and was discharged on day 12.

Conclusions

A well-functioning stoma does not rule out ongoing proximal bowel ischemia from non-occlusive mesenteric ischemia. In elderly surgical patients, unexplained neurological deterioration should prompt immediate suspicion of mesenteric ischemia, even in the absence of abdominal tenderness or stoma changes, and despite therapeutic anticoagulation. Superficial stomal mucosal necrosis in a viable stoma can be managed conservatively. High vigilance, early imaging, and readiness for re-exploration are essential.

Background

Because advanced age, frailty, and treatment delays amplify the risks of complications and death, experts advocate for an integrated geriatric care model in small bowel obstruction (SBO) (1). Most cases stem from adhesions, yet it remains difficult to foresee which patients will progress from a simple

obstruction to irreversible ischemia. A recent investigation identified the neutrophil-to-lymphocyte ratio as a promising predictor of bowel ischemia in non-strangulated adhesive SBO, potentially supporting earlier surgical intervention (2).

The Bologna guidelines advise that patients with an uncomplicated adhesive small bowel obstruction can safely undergo initial non-operative treatment. Immediate surgery becomes necessary if there is evidence of strangulation, peritonitis, or progressive clinical deterioration. For those who are hemodynamically unstable with extensive peritoneal soiling, the recommended approach is damage-control surgery with stoma creation rather than attempting a primary anastomosis (3). However, creating a stoma introduces its own complications. Problems arising from a stoma are encountered in 20–70% of patients (4–7). When necrosis involves the entire bowel wall, surgical correction is usually unavoidable; in contrast, when only the mucosal surface is affected, a watch-and-wait strategy often succeeds because the intestinal epithelium can regenerate (8).

The postoperative phase is further jeopardized by non-occlusive mesenteric ischemia (NOMI), an insidious condition frequently under-recognized in critically ill older patients and therefore described as the wolf in sheep's clothing (9). On computed tomography (CT), findings associated with severe disease and prognosis include absent bowel-wall enhancement and gas in the portal or mesenteric venous system (10).

We present the case of an 80-year-old woman who, following emergency double-barrel ileostomy for gangrenous adhesive SBO, initially developed superficial stomal mucosal necrosis that resolved with conservative management, and subsequently suffered new patchy bowel necrosis proximal to a functioning stoma while on therapeutic heparin. This sequence of events illustrates the diagnostic challenges inherent in assessing evolving stoma viability, the silent advancement of postoperative NOMI, and the delicate balance between thromboembolic risk and bleeding posed by anticoagulation in this vulnerable population. This case report was prepared in accordance with the CARE guidelines.

Case Presentation

Patient Information

An 80-year-old Iranian woman with a history of hypertension and ischemic heart disease presented to the emergency department with a 2-day history of severe abdominal pain, distension, and vomiting. Family history was non-contributory, and no relevant psychosocial or genetic history was identified. She had no history of previous abdominal surgery or other relevant abdominal intervention. Her regular cardiovascular medications, treatment adherence, allergy history, and outcomes of prior cardiovascular treatment were not documented in the available clinical records.

Clinical Findings

Vital signs revealed tachycardia (heart rate 110 bpm), hypotension (blood pressure 100/60 mmHg), and tachypnea (respiratory rate 20/min). Physical examination showed a distended, tender abdomen with absent bowel sounds. Laboratory findings included a white blood cell count of 10,500/ μ L (normal: 4,000–11,000/ μ L), lactate of 3.2 mmol/L (normal: 0.5–2.2 mmol/L), and C-reactive protein of 120 mg/L (normal: <10 mg/L).

Diagnostic Assessment

A contrast-enhanced abdominopelvic CT scan demonstrated small bowel obstruction with air-fluid levels, bowel-wall thickening, and signs of ischemia (Fig. 1). The working diagnosis was strangulated adhesive small bowel obstruction with bowel ischemia. The extent of gangrenous bowel (180 cm) and the patient's hemodynamic instability were adverse prognostic indicators that prompted a damage-control strategy with stoma formation rather than primary anastomosis. When the patient's consciousness deteriorated on postoperative day 4, the differential diagnosis included sepsis, a cerebrovascular event, and recurrent mesenteric ischemia; the normal abdominal examination and patent stoma initially made mesenteric ischemia appear less likely. The main diagnostic challenge was distinguishing NOMI from other causes of acute neurological decline, because the stoma appeared well perfused and the abdomen remained non-tender. The final diagnosis of NOMI was based on the patchy intraoperative distribution of necrosis and the perioperative low-flow context. No repeat CT, mesenteric angiography, or histopathologic confirmation was available.

Therapeutic Interventions

Emergency laparotomy revealed 180 cm of gangrenous ileum up to the ileocecal valve, caused by an adhesion band (Fig. 2). Given the patient's intraoperative hemodynamic instability (systolic blood pressure < 100 mmHg) and risk of poor perfusion, a double-barrel ileostomy was performed instead of a primary anastomosis, despite the healthy appearance of the remaining bowel. The patient received crystalloid resuscitation guided by central venous pressure (CVP) monitoring (target 8–12 mmHg) and therapeutic heparin (80 units/kg bolus followed by 18 units/kg/hour). The indication, planned duration, and total duration of heparin therapy were not documented in the available clinical records. No other specific pharmacologic agents, including antibiotics, were documented.

Follow-up and Outcomes

On postoperative day 2, superficial mucosal necrosis was observed above the stoma fascia. Because the stoma remained functional with normal output and lactate normalized to 1.8 mmol/L, conservative management was pursued. On postoperative day 4, the patient developed sudden reduced consciousness (Glasgow Coma Scale score, 12/15). Despite the stoma's normal appearance and function, a second laparotomy was performed, revealing 50 cm of patchy gangrenous bowel 20 cm proximal to the stoma. The affected segment was resected and the stoma was revised. Tolerability was

assessed clinically: the heparin infusion was administered without documented bleeding or other adverse effects, and the revised stoma functioned well. The patient stabilized and was discharged on postoperative day 12. No postoperative laboratory or imaging studies, patient-reported outcome measures, or post-discharge follow-up data were available; clinical recovery was assessed by the uneventful hospital course and discharge.

Timeline

A summary of the clinical course is presented in Table 1.

Table 1
Clinical Timeline of the Case

Time Point	Event	Details
Day 0 (Presentation)	Admission to Emergency Department	80-year-old woman with 2-day history of severe abdominal pain, distension, and vomiting. Vital signs: HR 110 bpm, BP 100/60 mmHg, RR 20/min. Exam: distended, tender abdomen, absent bowel sounds. Labs: WBC 10,500/ μ L, lactate 3.2 mmol/L, CRP 120 mg/L. CT: small bowel obstruction with air-fluid levels, wall thickening, and ischemia.
Day 0 (Emergency Surgery)	First Laparotomy	Revealed 180 cm gangrenous ileum up to ileocecal valve due to an adhesion band. Double-barrel ileostomy performed (instead of primary anastomosis) due to intraoperative hemodynamic instability (SBP < 100 mmHg) and risk of poor perfusion, despite healthy remaining bowel. Resuscitation with crystalloids (CVP guided, target 8–12 mmHg) and therapeutic heparin (80 units/kg bolus, then 18 units/kg/hour) initiated.
Postoperative Day 2	Complication & Conservative Management	Superficial mucosal necrosis observed above the stoma fascia. Stoma remained functional with normal output; lactate normalized to 1.8 mmol/L. Conservative management pursued.
Postoperative Day 4	Second Complication & Relaparotomy	Sudden reduced consciousness (GCS 12/15). Despite normal stoma appearance and function, second laparotomy performed, revealing 50 cm of patchy gangrenous bowel 20 cm proximal to the stoma. Affected segment resected and stoma revised.
Postoperative Day 12	Discharge	Patient stabilized and was discharged.

Footnote: HR: Heart Rate; BP: Blood Pressure; RR: Respiratory Rate; WBC: White Blood Cell count; CRP: C-Reactive Protein; CT: Computed Tomography; CVP: Central Venous Pressure; SBP: Systolic Blood Pressure; GCS: Glasgow Coma Scale. "Day 0" denotes the day of emergency admission and first laparotomy.

Discussion

This case merits reporting because it illustrates two sequential and uncommon postoperative complications that pose a substantial diagnostic challenge: superficial stomal mucosal necrosis that was successfully managed without surgery, immediately followed by the subtle development of NOMI in bowel located proximal to a well-functioning stoma, all while therapeutic anticoagulation was being administered. The juxtaposition of a healthy, patent stoma with upstream gangrene is rare and exposes a critical clinical pitfall.

Each significant treatment decision and complication in this case has meaningful parallels and distinctions in the existing literature. In agreement with the Bologna guidelines, constructing a stoma as part of a damage-control approach is advised when patients are hemodynamically unstable and there is gross peritoneal contamination, because the likelihood of anastomotic breakdown is unacceptably high (3). Studies focusing on SBO in older patients stress that physiological frailty demands a comprehensive, multidisciplinary approach to improve outcomes (1).

Superficial ischemia of the stomal mucosa is not an uncommon occurrence. Complications related to stomas arise in 20–70% of patients (4–7), and while transmural necrosis generally makes revision unavoidable, superficial involvement can often be observed expectantly due to the mucosa's inherent regenerative ability (8). Our report extends the literature by showing that when systemic perfusion is maintained, conservative management of superficial necrosis in an otherwise functioning stoma can succeed, supporting the existing evidence for non-operative care in selected patients.

The most instructive comparison concerns the emergence of NOMI. Käser et al. have characterized NOMI as *the wolf in sheep's clothing* because it commonly evolves without typical abdominal symptoms and remains under-diagnosed in the critically ill elderly (9). In the patient described, the sole presenting clue was an abrupt decline in consciousness; there was no abdominal tenderness, distension, or change in stoma effluent. Unlike many NOMI case series where necrosis occurs in the absence of a recent bowel anastomosis or stoma, our patient developed segmental, patchy gangrene situated far proximal to a completely patent stoma. This finding contrasts with ischemia caused by a mechanical obstruction and emphasizes that NOMI must be entertained as a diagnosis even when a recently created stoma appears entirely normal.

The discovery of patchy gangrenous bowel proximal to a functioning stoma, despite therapeutic heparin, can be explained by the underlying pathophysiology of NOMI. In contrast to thromboembolic acute mesenteric ischemia, NOMI is driven by a low-flow state within the splanchnic circulation, often triggered by a low-flow state from systemic hypotension or microvascular dysregulation (9, 10). In this instance, borderline perfusion, superimposed on the physiological insult of major abdominal surgery, likely precipitated mesenteric vasoconstriction and capillary stasis. Therapeutic heparin offers protection against macrovascular thrombosis but does not reliably counteract the microvascular hypoperfusion central to NOMI. The segmental pattern of necrosis is also consistent with recognized CT characteristics of NOMI, where scattered areas of compromised tissue may be interspersed among well-perfused loops

(10). The stoma itself remained viable because its vascular pedicle was independent of the compromised vascular arcades.

A strength of this report is the detailed chronological correlation between stoma appearance, neurological deterioration, and operative findings. However, no repeat CT imaging or mesenteric angiography was performed at the time of neurological deterioration; therefore, NOMI was diagnosed intraoperatively and retrospectively, without the prognostic imaging signs that might have prompted earlier intervention (10). The neutrophil-to-lymphocyte ratio, a marker proposed for predicting bowel ischemia in adhesive small bowel obstruction (2), was not tracked serially after surgery. Other limitations include incomplete medication and treatment records, absence of histopathologic confirmation, and lack of post-discharge follow-up. These limitations reflect the realities of an emergency clinical setting and identify areas in which prospective protocols could improve care.

Conclusion

A patent, normally functioning intestinal stoma does not exclude the presence of ongoing ischemia in the proximal bowel. When an elderly surgical patient experiences unexplained clinical deterioration, *especially a decline in consciousness*, NOMI should be actively considered, even when the abdomen remains non-tender and stoma output is normal. A high degree of suspicion, timely advanced imaging, and readiness for re-exploration are crucial. Additionally, superficial stomal mucosal necrosis that occurs in a viable stoma can be safely managed without early surgical revision. This case underscores the importance of sustained vigilance for NOMI in postoperative elderly patients, irrespective of anticoagulation status.

Patient and Family Perspective

A direct patient perspective was not documented in the available clinical records; therefore, the family's perspective is presented. The patient's family shared that the rapid succession of events after the first surgery, particularly the sudden decline in consciousness while the stoma looked healthy, was extremely frightening. They were relieved when the surgical team acted promptly and the second operation resolved the problem. They expressed gratitude for the close monitoring and for the decision to re-explore despite the absence of abdominal signs, and they were thankful that the patient could return home in good condition.

Abbreviations

CVP
Central venous pressure
CT
Computed tomography
NOMI

Non-occlusive mesenteric ischemia
SBO
Small bowel obstruction

Declarations

Ethics approval and consent to participate: This case report was reviewed and approved by the Ethics Committee of North Khorasan University of Medical Sciences (Ethics approval No. **IR.NKUMS.REC.1405.084**). The report was prepared and conducted in accordance with the ethical principles and regulations of the approving ethics committee.

Consent to participate: Informed consent to participate in this case report was obtained from the patient's family because the patient was unable to provide consent during the acute episode of reduced consciousness.

Consent for publication: Informed consent for publication of this case report was obtained from the patient's family because the patient was unable to provide consent during the acute episode of reduced consciousness.

Availability of data and materials: The de-identified data supporting the findings of this case report are available from the corresponding author upon reasonable request, subject to patient confidentiality and institutional requirements.

Competing interests: The authors declare that they have no competing interests.

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Figures

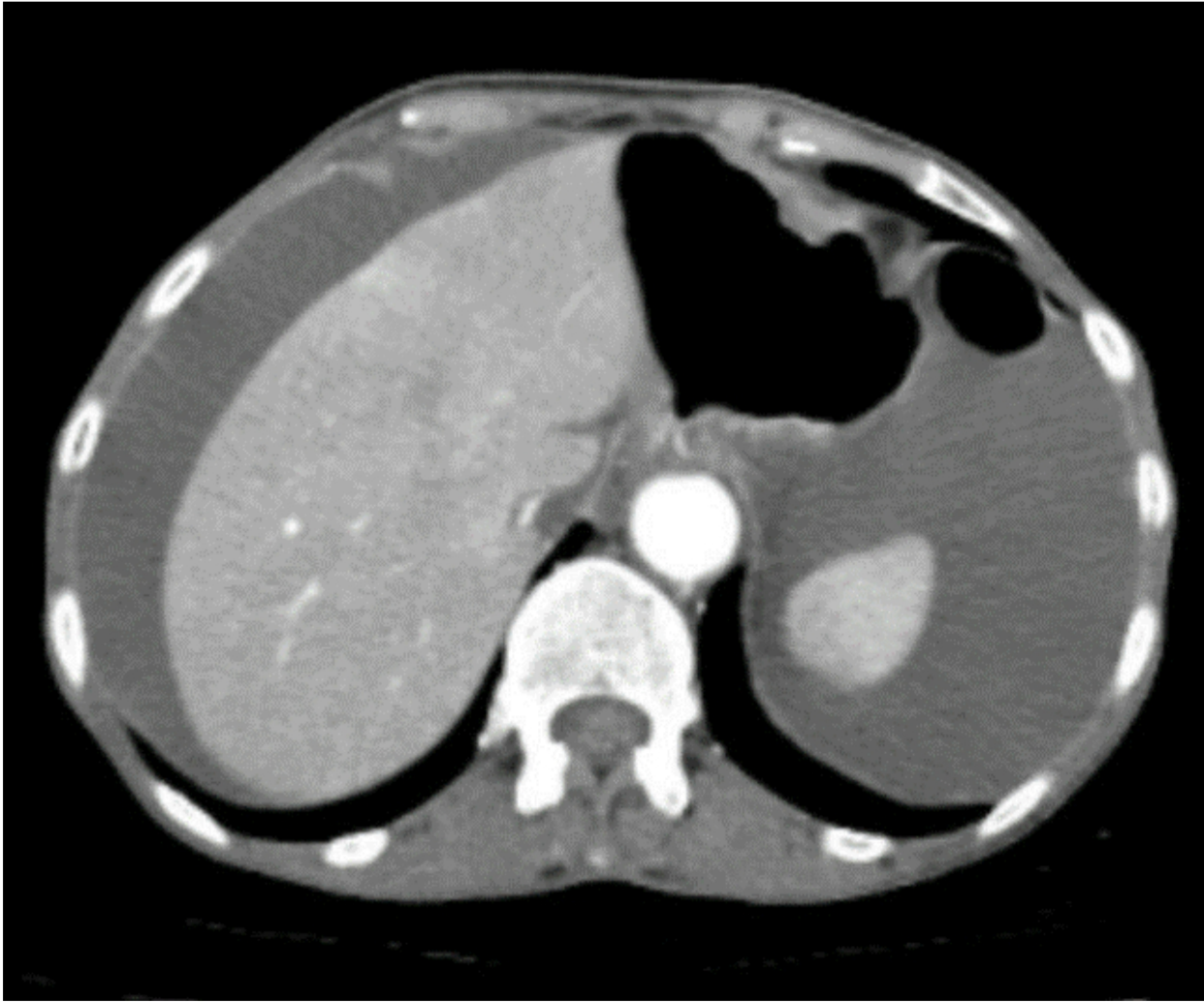


Figure 1

Axial contrast-enhanced abdominopelvic CT image obtained at presentation. The complete CT examination demonstrated small bowel obstruction with air-fluid levels, bowel-wall thickening, and signs of ischemia.



Figure 2

Intraoperative photograph showing 180 cm of gangrenous ileum up to the ileocecal valve, caused by an adhesion band, during the initial laparotomy.