

Case Report

Spontaneous resolution of non-occlusive mesenteric ischemia in the setting of hemodialysis: a case report

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ABSTRACT

Non-occlusive mesenteric ischemia (NOMI) is a rare yet serious cause of acute abdominal pain, particularly affecting critically ill and dialysis-dependent patients. Unlike occlusive mesenteric ischemia, NOMI results from mesenteric hypoperfusion without vascular obstruction, making early diagnosis challenging. We report a case of a 46-year-old female with end-stage renal disease (ESRD) on hemodialysis who presented with severe abdominal pain and hypertensive emergency following dialysis. Initial imaging revealed portal venous gas (PVG) without evidence of bowel obstruction or bowel ischemia. Despite intractable pain, her condition improved with supportive care, including blood pressure management, without the need for invasive intervention. A repeat CT scan showed resolution of PVG, correlating with clinical improvement. Hemodialysis-associated hypotension is a known risk factor for NOMI due to transient mesenteric hypoperfusion. This case highlights the role of blood pressure fluctuations in NOMI's pathophysiology, with post-dialysis hypertension potentially aiding in recovery. Additionally, the transient presence of PVG on imaging, which resolved alongside symptom improvement, suggests its utility in diagnosing and monitoring NOMI. This case underscores the importance of recognizing NOMI in dialysis patients with abdominal pain, close blood pressure monitoring, and utilizing serial CT imaging for non-invasive disease management. Our findings suggest that in select patients with rapid clinical recovery, PVG disappearance may serve as a marker for NOMI resolution, potentially avoiding unnecessary laparotomy.

Keywords: Mesenteric ischemia, Hemodialysis, Nonoperative management

INTRODUCTION

Non-occlusive mesenteric ischemia (NOMI) is a significant but rare and often underrecognized cause of acute, intractable abdominal pain, primarily affecting elderly patients with multiple conditions, particularly those in intensive care units.¹ Unlike occlusive acute mesenteric ischemia (AMI), which is caused by a direct blockage of the mesenteric arteries, NOMI results from a reduction in mesenteric blood flow without any actual obstruction. A Swedish population-based study conducted between 1970 and 1982 suggested an incidence of fatal NOMI at 2 per 100,000 person-years.²

The pathophysiology of NOMI is complex and incompletely understood, involving vasoconstriction, reduced cardiac output, impaired autoregulatory response of the mesenteric vessels, and ischemia-reperfusion injury. Conditions associated with NOMI include systemic hypotension, heart failure, sepsis, and vasopressors—all impair mucosal perfusion. Additionally, hypotensive episodes during hemodialysis have been linked to an increased risk of NOMI.³

In a retrospective study between 2007 and 2008, it found the incidence of NOMI was 1.04% per patient-year for all dialysis patients.⁴

CASE REPORT

A 46-year-old female with a complex medical history including ESRD on hemodialysis (Mon/Wed/Fri), and a history of a transient ischemic attack (TIA) in 2007, presented to the emergency department with severe abdominal pain and in hypertensive emergency following her recent hemodialysis session. She described her abdomen as distended and pain rated 10 out of 10, with associated bowel movements but no nausea or vomiting. Patient denied abdominal surgeries. However, the patient has a history of failed peritoneal dialysis. Her last dialysis session was at her Rehab center the day of presentation through her left femoral ash catheter.

Upon arrival, she was hypertensive to 279/179 and was started on an Esmolol drip, which was later changed to a nicardipine drip due to inadequate response. Blood pressure was monitored hourly. Her lab work showed non-elevated lactic acid level of 0.7, non-elevated white blood cell counts 9.5 with elevated Neutrophil percent of 80.3 and an elevated creatinine level of 5.27. Electrocardiography showed sinus rhythm. A CT scan of the abdomen and pelvis demonstrated hepatomegaly, polycystic renal disease, and abundant ascites in the peritoneal cavity, PVG, and no evidence of bowel obstruction. Morphine was given every four hours and one stat dose of hydromorphone was administered with minimal response.

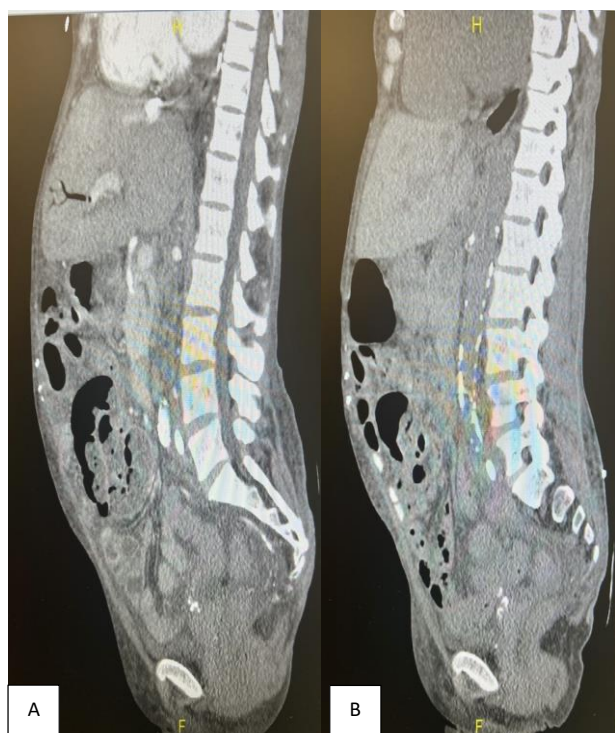


Figure 1: A) demonstrates initial CT scan and B) demonstrates repeat CT.

Gastroenterology and surgery were consulted due to concern for NOMI. During surgical assessment, multiple

attempts were needed to obtain a complete history from the patient, who denied any prior abdominal surgeries. Her abdomen was soft and mildly tender to palpation over epigastric region. On gastrointestinal evaluation the following day, there was no tenderness or signs of acute abdomen. At this point, repeat CT abdomen pelvis was obtained and did not reveal PVG.

Nephrology was consulted regarding her ESRD management. She underwent a 3-hour dialysis session with a potassium bath of 2K, blood flow of 400, and ultrafiltration of 2.5L.

By day 3, she tolerated oral intake, and her hypertensive emergency resolved. She was discharged home with her regular antihypertensive medications.

DISCUSSION

Early diagnosis of NOMI is crucial yet challenging due to its nonspecific clinical presentation and subtle radiological findings. Patients often present with intractable abdominal pain, vomiting, diarrhea, and gastrointestinal bleeding, with deterioration following an episode of acute circulatory collapse and profound hypotension. As Bourcier et al discuss, these nonspecific symptoms often overlap with other gastrointestinal disorders, making it difficult to promptly diagnose NOMI.¹ Furthermore, the clinical manifestation of NOMI can be easily mistaken for other conditions, contributing to delayed recognition and treatment.⁵

Hemodialysis and NOMI

This patient's hemodialysis regimen played a significant role in her clinical presentation, as it caused the inciting hypotensive episode. Hypotension has been linked to an increased risk of NOMI due to a reduction in mesenteric blood flow. Studies have shown that dialysis patients are particularly vulnerable to NOMI due to multiple risk factors, including volume depletion and the effects of vasopressor use.^{3,4,10} Acosta et al also highlighted that hypotension during hemodialysis is a well-established risk factor for fatal NOMI.² This case report emphasizes the importance of close blood pressure monitoring in patients undergoing dialysis, and though NOMI's presentation is very vague, considering NOMI as a potential diagnosis in patients with abdominal pain following hypotension during dialysis is crucial. As our patient's symptoms resolved quickly following immediate post-dialysis hypertension (blood pressure as elevated as 279/179), it raises the question of whether this rapid increase in blood pressure may have contributed to the resolution of NOMI.

The association between post-dialysis hypertension and improvement in NOMI has been noted in previous studies, which suggest that prompt blood pressure correction may help reverse ischemia in some cases.¹

Diagnostic utility of CT imaging and monitoring PVG

CT imaging was vital in assessing and monitoring PVG in our patient. Initial abdominal CT provided evidence of PVG, suggesting NOMI. A follow-up CT scan performed 16 hours later showed complete resolution of PVG, coinciding with the patient's rapid clinical improvement. Literature suggests that transient PVG, which disappears within hours, may indicate the resolution of NOMI.⁸ Our case corroborates this finding, indicating that PVG may appear in imaging prior to irreversible gut ischemia. As pointed out by Versyck et al the transient nature of PVG in some patients can provide critical insight into the resolution of NOMI, reducing the need for aggressive surgical interventions.⁹

The rapid correction of the patient's condition with medical therapy suggests that, in certain cases where clinical status improves quickly, monitoring PVG for disappearance could help avoid unnecessary laparotomy. The resolution of PVG on follow-up imaging supports CT as a valuable tool in identifying NOMI before the onset of transmural necrosis and monitoring the condition's progression and resolution. The potential for CT to distinguish between reversible and irreversible ischemia underscores its importance as a non-invasive diagnostic tool in the management of NOMI.^{6,7}

CONCLUSION

This case report details a unique clinical presentation of NOMI in a patient undergoing dialysis, emphasizing diagnostic challenges and therapeutic considerations. Unlike typical cases requiring papaverine infusion into the superior mesenteric artery or surgical intervention to address necrotic tissue, this patient's symptoms spontaneously resolved with supportive care, including blood pressure management. This case provides two significant insights into the management of NOMI: 1) It suggests that post-NOMI hypertension may aid in resolving NOMI and preventing necrosis. 2) It implies that in patients with improving clinical status, monitoring PVG for disappearance using CT can help avoid unnecessary laparotomy.

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