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Enteral Feeding Induced Nonocclusive Mesenteric Ischemia

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Background	Nonocclusive mesenteric ischemia (NOMI) is caused by inadequate perfusion of the intestinal mucosa secondary to a nonocclusive reduction in arterial blood flow. Herein, we present a case of NOMI triggered by enteral feeds in a patient who had just undergone extensive esophageal surgery for hypopharyngeal squamous cell carcinoma.
Summary	A 58-year-old male with no known past medical history presented to the emergency department (ED) with a history of unintended weight loss and a large infrahyoid neck mass suggestive of a malignancy. Immediate surgery was performed by the otolaryngology team, and a gastrostomy tube was concomitantly placed. Four hours after the surgery, he was started on continuous enteral feeds with a dietary solution containing 1.5 calories/mL at 10ml/hr and was advanced to 60 ml/hr within six hours. The following day, continuous feeds were discontinued, and he was started on bolus feeds. He received a total of three bolus feeds before they were discontinued due to severe abdominal pain and distension. His electrolytes were within normal limits, indicating the absence of refeeding syndrome. A computed tomography (CT) scan of the abdomen showed diffuse pneumatosis, portal venous gas, and abdominal free air. He was emergently taken to the OR for an exploratory laparotomy due to suspected NOMI and admitted to the intensive care unit. Interventional radiology (IR) performed superior mesentery angiography, which showed gracile vessels, and a Papaverine infusion was started. Two days later, he developed an ileus with fever, tachycardia, and agitation. He was subsequently transitioned to comfort care by his family and died 48 hours later.
Conclusion	We postulate that this patient's continuous feeds were advanced too quickly in the setting of malnutrition (24% loss in body weight over two to three months) and recent surgery. To prevent future cases of NOMI, we recommend nutritional evaluation before initiation of enteral feeds and increased education of hospital staff regarding clinical symptoms of NOMI. Currently, the main concern with enteral feeding is the development of refeeding syndrome, a potentially fatal condition resulting from rapid fluid and electrolyte shifts in malnourished patients receiving artificial refeeding. However, we encourage monitoring for NOMI as well. By identifying high-risk patients and raising awareness of the importance of pertinent clinical symptoms, it should be possible to reduce the risk of a fatal outcome in malnourished patients with underlying vascular risk factors for NOMI.

Key Words	nonocclusive mesenteric ischemia; enteral feeds; postoperative complications

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Case Description

A 58-year-old male with no known past medical history presented to the ED with three weeks of dysphagia. He had a 40-pack-year smoking history and an alcohol misuse disorder. He also reported an unintentional 20 to 30 lbs weight loss (24% decrease in body weight) over the previous two to three months and had a BMI of 18 kg/m2. A CT scan of the neck revealed a large infrahyoid neck mass of the hypopharynx extending to the supraglottic larynx with multiple bilateral cervical lymph nodes. The patient was taken to the operating room (OR) by the otolaryngology team for direct laryngoscopy and tissue biopsy. The emergency general surgery (EGS) team was consulted for the placement of a simultaneous Stamm gastrostomy tube (G-tube) for enteral feeding access.

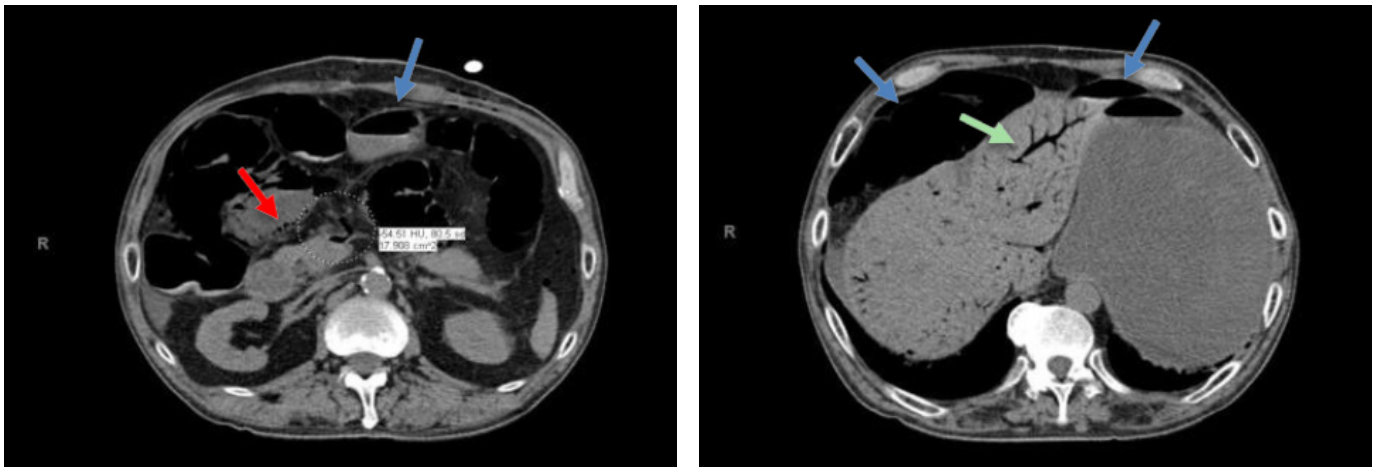
Four hours after surgery, the patient was started on continuous enteral feeds with a dietary solution containing 1.5 calories/mL at 10 ml/hr and was advanced by an additional 10 ml/hr until reaching a final 60 ml/hr within a six-hour duration. After tolerating continuous feeds for 24 hours, he was transitioned to enteral bolus feeds at a rate of 237 ml every four hours. He received a total of three bolus feeds before they were discontinued due to gastrointestinal (GI) intolerance, which consisted of worsening abdominal pain and distension.

On postoperative day (POD) 3, he developed tachycardia with continued abdominal pain and distension, a normal white blood count, elevated lactate (8.2 mmol/L), and normal levels of phosphorous, magnesium, and potassium, which ruled out a diagnosis of refeeding syndrome, which is a potentially fatal condition that results from rapid fluid and electrolyte shifts that occur in malnourished patients receiving artificial nutrients.[1] A computed tomography (CT) scan of the abdomen showed diffuse pneumatosis along with portal venous gas with intraabdominal free air concerning for intestinal ischemia and perforation (Figure 1).

He was emergently taken to the OR by EGS for an exploratory laparotomy. There was segmental ischemia of the mucosa of the small and large intestine, but the serosa was intact, and there were no areas of full-thickness necrosis. There was a biphasic doppler signal of the SMA identified by intraoperative doppler. An Abthera device was placed for temporary abdominal closure, followed by admission to the intensive care unit for postoperative resuscitation. He remained on a ventilator with vasopressor support (e.g., vasopressin and norepinephrine). The following day (POD 4), a follow-up CT scan of the abdomen showed resolved pneumatosis and no evidence of mesenteric vascular occlusion. This finding was suggestive of NOMI (Figure 2).

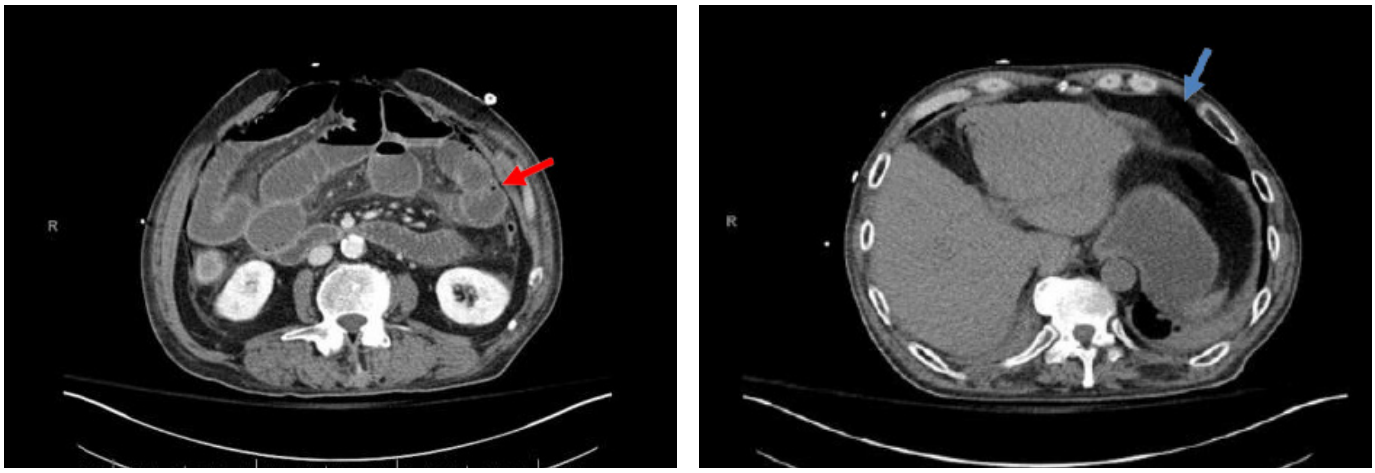
IR was consulted, and a superior mesenteric angiography showed patency of the SMA and its branches without the presence of occlusion, stenosis, or microemboli; however, the peripheral vessels appeared gracile (i.e., slender). A catheter was inserted into the superior mesenteric artery (SMA), and a Papaverine infusion was begun (Figure 3). On POD 5, he was returned to the OR for exploratory laparotomy. No evidence of intestinal necrosis was identified, and the abdomen was closed. On POD 6, the IR catheter was removed after a follow-up angiogram showed improved blood flow through the mesenteric vasculature.

Figure 1. CT Abdomen/Pelvis Without Contrast. Published with Permission



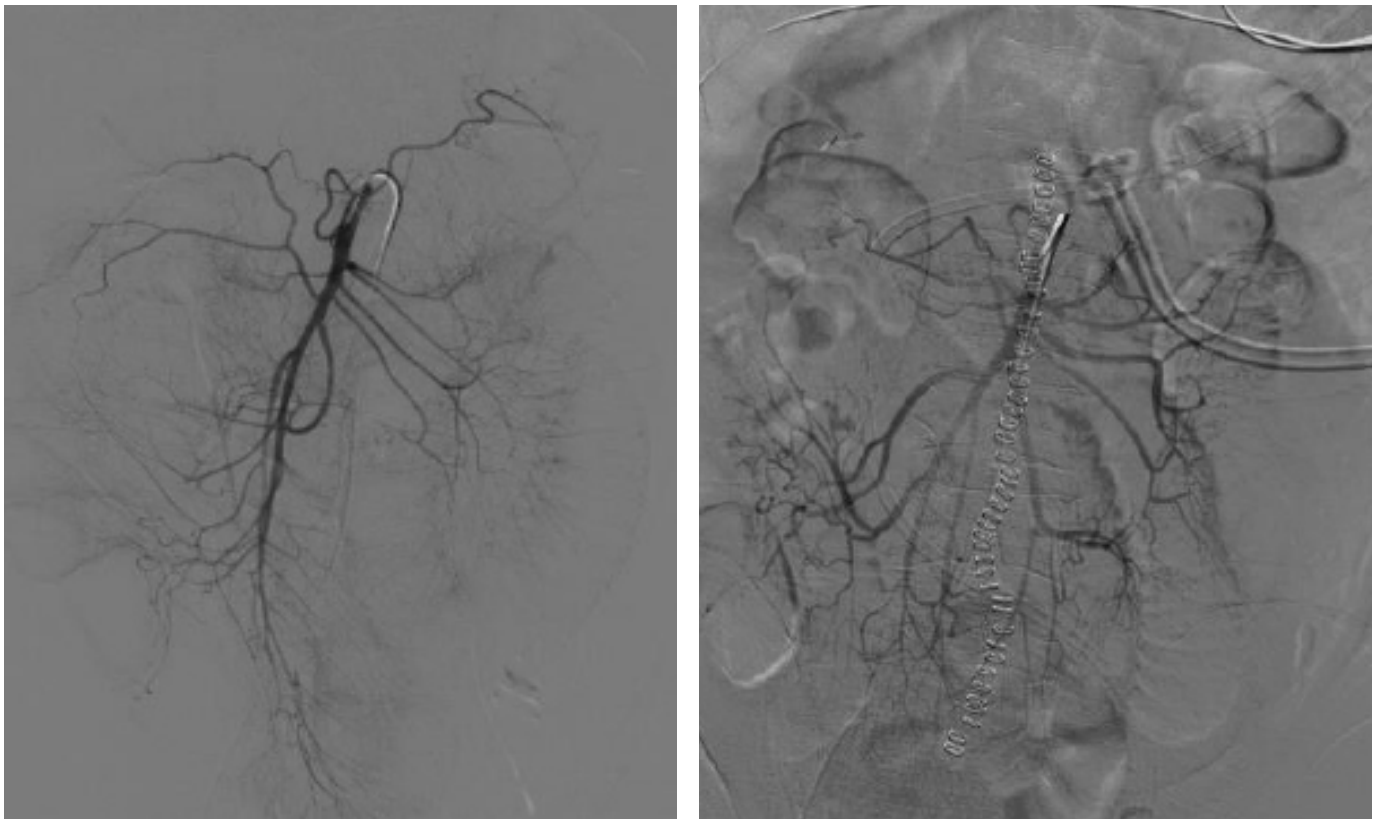
Images show pneumatosis intestinalis (red arrow), free air (blue arrows), and portal venous gas (green arrow).

Figure 2. CT Abdomen/Pelvic Without Contrast. Published with Permission



Images show improved pneumatosis intestinalis (red arrow), free air (blue arrows), and portal venous gas.

Figure 3. Superior Mesenteric Angiography Before (left) and After (right) Papaverine Infusion. Published with Permission



Images show gracile (i.e., slender) peripheral vessels with improvement after IR procedure.

Nevertheless, his clinical condition continued to deteriorate, and he became agitated, tachycardic, hypotensive, hypophosphatemic, hypokalemic, and developed an ileus. Two days later, the patient's family met with the palliative care team, and the patient was transitioned to comfort care. He died 48 hours later due to complications of advanced-stage malignancy and NOMI.

Discussion

NOMI is a rare and potentially life-threatening condition that has been linked to patients receiving early enteral feeding following surgery, particularly in patients with oncologic indications (incidence, 1.5-1.7%).[2,3] NOMI encompasses all forms of mesenteric ischemia with patent mesenteric arteries.[4] This condition is most commonly seen in patients older than 50. It is caused by decreased blood flow to mesenteric vessels secondary to impediments to blood flow, such as cardiovascular or renal disease, hypovolemia, sepsis, dialysis, and vasoconstricting medications.[3–5] Roughly 20-30% of all cases of mesenteric ischemia are caused by NOMI, with a mortality rate ranging from 50% to 80%.[4–6] Several underlying pathological processes may contribute to the development of NOMI. First, the absorption of nutrients by metabolically stressed enterocytes increases intraluminal energy demands, which, in turn, can decrease the threshold for bowel ischemia in patients with concomitant systemic hypoperfusion.[3,6] Secondly, enteral formulas are high-osmolarity fluids that can cause a rapid fluid shift from splanchnic vessels into the intestinal lumen.[3] This creates abdominal distension, which compromises intestinal perfusion.[3] Finally, enteral nutrition supports bacterial overgrowth with concomitant intraluminal gas production, distension, and microbial toxin accumulation.[3,6]

Diagnosing NOMI can be challenging because the symptoms are nonspecific. Due to the rarity of NOMI, current literature on the prevalence of individual symptoms is limited. However, it has been reported that clinical manifestations of NOMI generally include abdominal pain and distension, hypotension, fever, nausea, and vomiting.[4] Laboratory evaluations of patients experiencing NOMI may show an elevated WBC, elevated serum lactate, and metabolic acidosis.[4] The gold standard for diagnosing NOMI is selective angiography, which will show narrowing or spasm of mesenteric arcades, a reduced number of mesenteric vessels, and irregularity of the arterial branches of the mesenteric vasculature on vascular imaging.[4] Intestinal wall thickening with pneumatosis intestinalis are also characteristic findings.[4]

The goal of treatment in patients with NOMI is promptly restoring adequate perfusion to the intestines. This can be accomplished by withdrawing inciting factors (such as vasoconstricting medications), treating the underlying sepsis and congestive heart failure, providing hemodynamic support, and, less commonly, administrating vasodilating medications.[4] Direct infusion of vasodilators, such as the opium alkaloid antispasmodic medication Papaverine [SJR5] or prostaglandin E1, into the mesenteric artery increases small bowel perfusion.[7,8] Several retrospective cohort studies conducted in 2020 showed that vasodilator therapy significantly decreased rates of both hospital mortality and abdominal surgery in patients experiencing NOMI.[8,9] However, laparotomy is the preferred treatment for NOMI in patients with severe, unresolving symptoms that are concerning for intestinal infarction or perforation.[4]

For patients receiving continuous enteral nutrition, the recommendation of the American Society for Parenteral and Enteral Nutrition (ASPEN) is to initiate feeding at 10-40 ml/hr and progress by 10-20 ml/hr every 8-12 hours as tolerated, until the patient's caloric target is met.[10] Patients are transitioned to a bolus feeding regimen once they can tolerate continuous infusions for a full 24 hours.[10] We conclude that our patient's continuous feeds were advanced too quickly in the setting of multiple risk factors for NOMI. He was malnourished, had adverse cardiovascular risk factors (e.g., 40-pack-year smoking history and alcohol misuse disorder), and was placed on vasoconstricting medications.

Conclusion

The standard of care for postoperative management of enteral tube feeding in malnourished surgical patients should be clarified, knowing that NOMI is a rare but possible complication. To minimize the risk of NOMI, we recommend that each patient undergo a nutritional evaluation before initiation of enteral feeds. If patients have risk factors for NOMI, then enteral feeding should be advanced at a slower rate. Additionally, NOMI may present similar to refeeding syndrome, so electrolytes must be closely monitored to distinguish between the two conditions. Finally, we urge clinicians to consider using direct vasodilator therapy in the setting of NOMI because it is a relatively safe and minimally invasive treatment option with the potential to reverse mesenteric vasoconstriction.[9]

Lessons Learned

Early detection of NOMI and cessation of enteral feeds in response are critical in preventing intestinal ischemia. However, the nonspecific presentation of NOMI makes diagnosis challenging, especially in the presence of severe, comorbid illness. This case highlights the critical need for careful progression of enteral feeding and surveillance for this potentially fatal condition.

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