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Case Report

Non-occlusive Mesenteric Ischemia – A Rare but Deadly Condition

Carla Cadet B.S.[1], Farzana Hoque[2]

Abstract

Non-occlusive mesenteric ischemia is a rare but often fatal condition that occurs due to spasms in the splanchnic arteries leading to hypoperfusion, cellular death, bowel ischemia, and eventually perforation. Having a high clinical suspicion in the correct setting is crucial to identify and treating the medical condition quickly. This is a unique case of an 82-year-old Caucasian male who presented with peritonitis secondary to acute mesenteric ischemia caused by hypotension leading to the eventual finding of bowel ischemia and perforation.

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Introduction

Mesenteric ischemia is a serious clinical condition that occurs when there is a reduction in blood supply to the small intestines, leading to cellular death. The small intestine receives blood flow primarily from the superior and inferior mesenteric arteries with an extensive network of collateral circulation intended to maintain the viability of the tissue in the context of inadequate blood flow from the main arteries.[1] The two main forms of mesenteric ischemia include acute and chronic ischemia, with acute ischemia being a surgical emergency requiring prompt recognition and intervention. Many factors can contribute to acute mesenteric ischemia, including arterial embolism, arterial thrombosis, and non-occlusive mesenteric ischemia (NOMI).[1] It is theorized that it is due to spam of the superior mesenteric artery (SMA) causing hypoperfusion to the small intestinal tissue. NOMI has been noted in the literature to occur in critically ill individuals with severe cardiovascular disease, in those receiving vasoconstrictive medications, sepsis, renal failure, recent cardiopulmonary bypass, or profound and prolonged hypotension.[2,7] It is often exceedingly difficult to diagnose due to nonspecific symptoms including mild abdominal pain, nausea, and vomiting. The condition can also be overshadowed by precipitating factors including hypotension, and hypovolemia.[2]

Case report

We report a case of an 82-year-old Caucasian male with a history of COPD, aortic ectasia, hypertension, chemotherapy-induced cytopenia, and invasive bladder cancer status post neoadjuvant chemotherapy followed by open radical cystectomy with pelvic lymph node dissection and ileal conduit who presented on the internal medicine service for additional management of bilateral lower extremity edema and pain worse on the left. Of note, surgical findings during open radical cystectomy included significant adhesions, as well as a small enterotomy primarily repaired during that same procedure. The patient had been complaining of ongoing mildly hindering left inner thigh pain since the day after surgery that was treated with Tylenol, lidocaine patch, and subsequently Ketorolac. The patient was set to be discharged on postoperative day 7 to an skilled nursing facility (SNF) but was unable to leave due to inadequate left inner thigh pain control. On postoperative day 8, he had new chest pain, shortness of breath, and worsening left inner thigh pain with right lateral thigh pain. The EKG performed showed a new right bundle branch block, troponin level within normal range, and a chest X-ray with mild atelectasis as well as concern for intraperitoneal free air that was thought to be related to the recent surgery.

1. Saint Louis University School of Medicine, St. Louis, MO, USA
2. Associate Professor of Medicine, Saint Louis University School of Medicine, St. Louis, MO, USA

Corresponding Author: Farzana Hoque, Associate Professor of Medicine, Saint Louis University School of Medicine, St. Louis, MO, USA. E-mail: farzanahoquemd @gmail.com

Bilateral venous dopplers were also performed with the right showing no acute deep vein thrombosis and the left showing left incomplete peroneal deep vein thrombosis. The patient was then started on Apixaban 10 mg BID for the acute DVT. On postoperative day 9, the patient had worsening bilateral thigh pain, which led to the primary team ordering an MRI (Magnetic Resonance Imaging) of the thoracic and lumbar as well as creatinine phosphokinase (CPK). The MRI was negative for epidural hematoma, and the creatine kinase was within normal limits ruling out myopathy. The internal medicine team was also consulted that same day for additional management of the worsening bilateral lower extremity edema and pain. During the evaluation by the internal medicine team, significant abdominal pain to light palpation was noted. A stat CT was recommended to evaluate the patient for possible post-surgical complications including PE. The CT was delayed until the next day when a rapid response was called on to the patient due to dyspnea, acute hypoxia, and diffuse abdominal pain. CT PE protocol, Abdomen, and Pelvis were conducted for evaluation of PE and pneumoperitoneum. CT revealed no PE but was significant for the thickening of multiple small bowel loops in the lower abdomen with decreased enhancement with areas of pneumatosis as well as extensive mesenteric and portal venous gas which were findings concerning bowel necrosis (Figure 1).

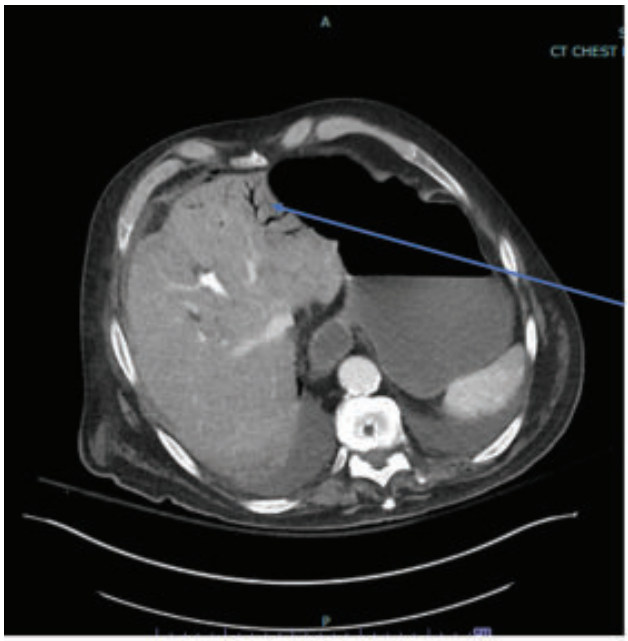

Figure 1: *A blue arrow pointing to portal venous gas in the left hemi-liver noted as peripherally located branching gaseous foci.*

The patient was then transferred to the anesthesiology critical care service for a higher level of care. There the wound drainage was evaluated for concern that output was from an intrabdominal source. A nasogastric tube was inserted and placed on low intermittent wall suction with drainage of over 700 ml of feculent/bilious drainage. The patient was then taken for an urgent laparotomy where it was discovered that the patient had a perforated small intestine just distal to a previous intestinal anastomosis. An ischemic appearing 10cm bowel segment distal to anastomosis was observed. The ischemic portion was resected, and the bilateral ureteral intestinal anastomoses were broken down and repaired. Post-laparotomy imaging showed the interval resolution of the previous portal venous gas that indicated ischemia (Figure 2). The patient was left in discontinuity with two further attempts at abdominal closure. The patient was in the intensive care unit (ICU) for 15 days with worsening medical status including maximum ventilatory support, bilateral nephrostomy catheter placement due to a persistent urinary leak, and bridging vicryl mesh placement as temporary abdominal closure. The patient eventually passed away after the family decided to opt for comfort care after discussing the patient's wishes.

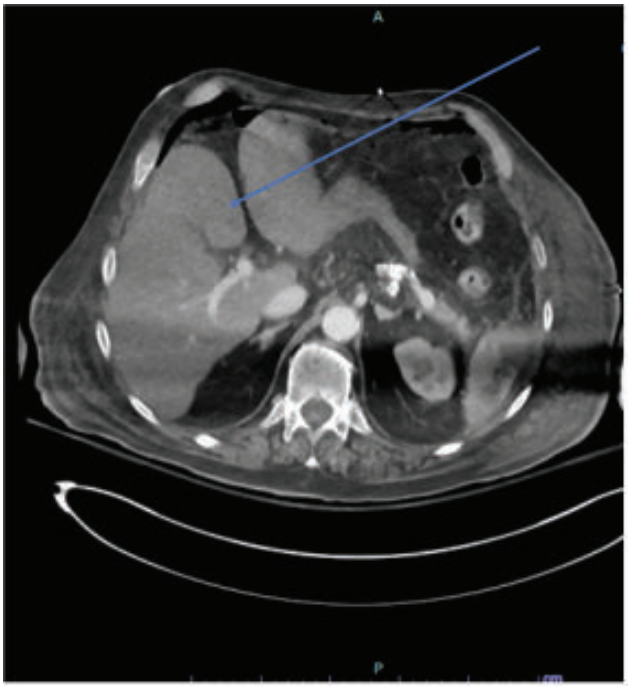

Figure 2: Post exploratory laparotomy: blue arrow showing interval resolution of previously seen portal venous gas in the left hemi-liver.

Discussion

Non-occlusive mesenteric ischemia (NOMI) is a rare but potentially fatal medical condition. It is often caused by

spasms of the mesenteric arteries leading to hypoperfusion of the tissue and cellular death with prolonged and inadequate blood flow.[1,3,5,6] The symptoms of NOMI are often nonspecific and include mild abdominal pain accompanied by bloating sensation, nausea, and vomiting. The literature has shown that up to 1/3 of patients do not have abdominal pain and that the peritoneal signs of rebound tenderness and guarding often are not present on clinical presentation with just the ischemia. Unfortunately, in NOMI, transmural infarction often already occurs at the time peritonitis presents itself, rendering only salvageable interventions.[3,7] For this patient, extensive adhesions were present on the initial open radical cystectomy which led to the primary repair of an enterotomy of the small bowel. Since the patient had an estimated blood loss of 500 ml intraoperatively and multiple episodes of hypotension the night after surgery, reduced blood flow to the repaired enterotomy may have led to its breakdown. This breakdown may have led to small but persistent spillage of bowel content into the abdominal cavity. The patient reported indigestion four days before the peritonitis presentation, which is the only nonspecific sign of NOMI that was present after a careful chart review.[7] The patient had persistent left inner thigh pain since the day after surgery and an incomplete peroneal DVT developed from that same leg 8 days after surgery. The patient was started on subcutaneous heparin on postoperative day 4, which could have dissolved a small embolic clot or a small direct thrombosis at the ischemia area. The CTA did show patent superior mesenteric artery, inferior mesenteric artery, and celiac vessels, however, it cannot be ruled out that embolic or thrombotic causes did not contribute.[4,8] This patient did not have most of the overt risk factors that increased the risk of NOMI such as CHF, history of pancreatitis, use of vasospastic medications, cardiotonic medications, hemodialysis, and cardiac surgery.[4,9] The NOMI risks for that patient were fluctuated intraoperative hypotension, post-operative overnight hypotension, older age, and major abdominal surgery.[4] The literature reports that mesenteric ischemia has a reported prevalence of 0.09% to 0.2% of all medical admissions in the United States. Nonocclusive mesenteric ischemia due to intestinal hypoperfusion accounts for about 20% of those admissions.[3,9] Individuals who do not have the over-risk factors but have the risk of bowel injury during surgery should be carefully monitored and a low threshold for imaging should be in place to promptly identify and intervene to minimize morbidity and mortality. Overall, it is important to consider the diagnosis in the setting of all open abdominal surgeries.

Conclusion

Nonocclusive mesenteric ischemia is a rare yet frequently lethal condition that leads to intestinal necrosis and perforation of the intestinal wall. Swift identification of symptoms and timely interventions are crucial, emphasizing the importance of maintaining a heightened level of suspicion in individuals at risk of developing this condition.

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