

Hypercoagulable Factors Attributing to Superior Mesenteric Vein Thrombosis and Acute Mesenteric Ischemia – A Case Report

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Introduction

- Acute mesenteric ischemia (AMI) is rare and can be difficult to diagnose due to vague symptoms.
- It can be fatal if not discovered in time, as it can lead to bowel ischemia, sepsis, and ultimately death.
- We present a case of a 23-year-old female with hepatic steatosis, obesity, and five-year history of birth control use who developed acute mesenteric ischemia secondary to superior mesenteric venous (SMV) thrombosis, requiring small bowel resection of 238 cm out of 480 cm (49.5%) after delay in diagnosis.
- Hypercoagulable and genetic workup during admission later revealed heterozygous factor V Leiden (FVL) mutation.

thrombosis

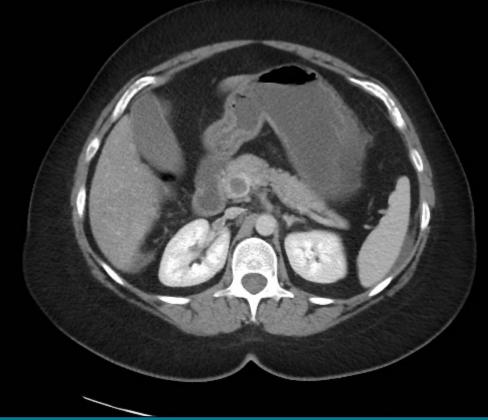


Figure 2: CT imaging of portal vein thrombosis

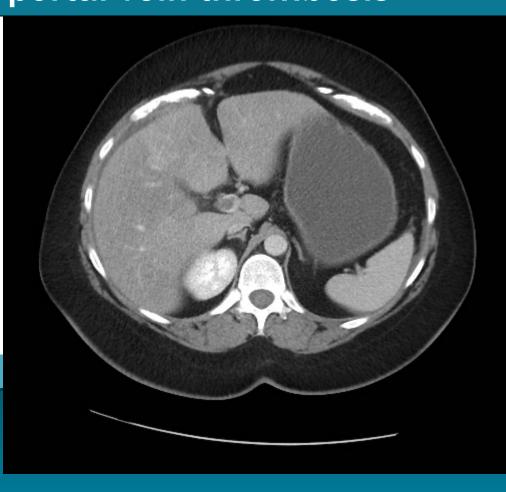
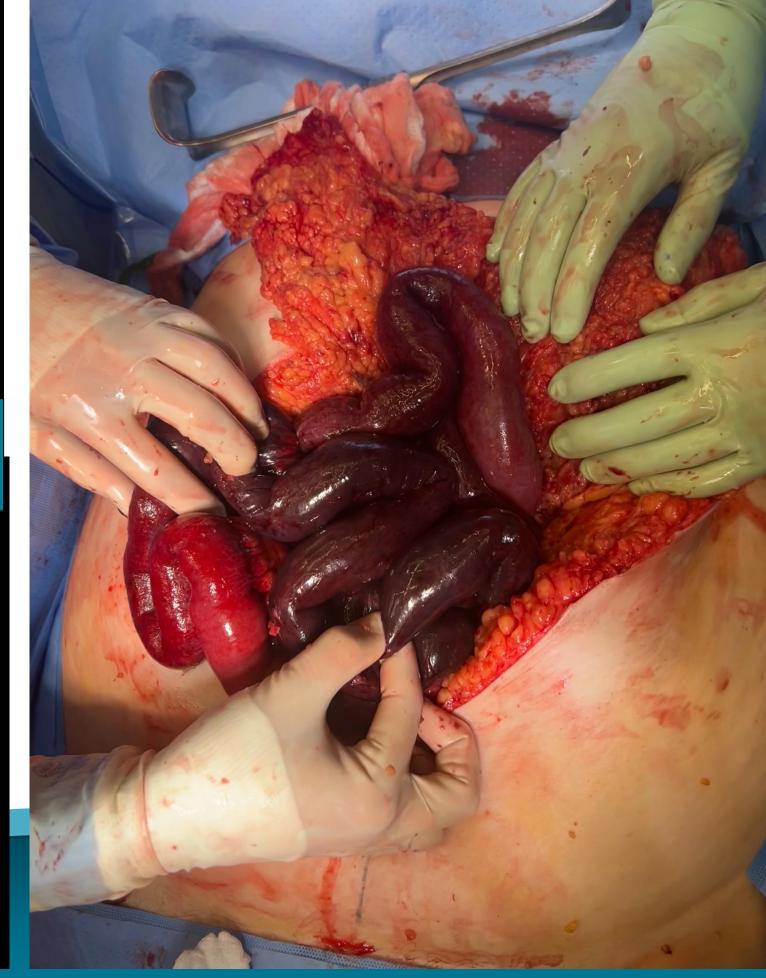


Figure 1: CT imaging of SMV Figure 3: Intra-operative small bowel necrosis



Case Report

- 23-year-old female
- PMHx: migraines, hepatic steatosis, BMI 42, and currently using birth control (estradiol-norgestimate) for ~5 years
- FHx: blood clots in maternal grandmother, no known genetic predisposition
- Presented to an outside hospital multiple times over 5 days with abdominal pain, worse with oral intake, nausea, vomiting, and eventually bloody stools
- Exam: abdomen soft, with mild generalized tenderness, progressed to distension with increasing tenderness
- Repeat CT scans: SMV and portal vein thrombosis (figures 1 & 2)
- with diffuse small bowel wall thickening, free fluid, and mesenteric venous congestion
- Transferred on heparin infusion and exam now with diffuse peritonitis
- Taken emergently to OR, immediately encountering dead bowel
- Resected 225 cm of frankly necrotic small bowel (figure 3), leaving 255 cm of healthy appearing small bowel with intact ileocecal valve
- Temporary abdominal closure and returned to ICU
- Return to OR approximately 24 hours later, additional 13 cm of small bowel was resected, leaving 242 cm of healthy small bowel, which was anastomosed and her abdomen closed with retention sutures
- Complications: re-intubated multiple times due to acute hypoxic respiratory failure; pneumonia, treated with 7-day antibiotic course; acute kidney failure, placed on CRRT; and gastrointestinal bleed with negative EGD though requiring pause in anticoagulation and transfusions
- Hematology/Oncology consult for hypercoagulable workup eventually revealed a heterozygous FVL mutation
- Discharged on hospital day 29 to inpatient rehabilitation
- Discharged from inpatient rehab after an additional 8 days
- On coumadin as an outpatient managed by Hematology

Discussion

- AMI can be attributed to arterial or venous pathologies, with SMV thrombosis only in 6-9% of all mesenteric ischemia cases [1].
- It can be challenging to diagnose, as symptoms described by patients are often non-specific; however, time is tissue, such that swift diagnosis is essential to saving small bowel for maximal functional outcomes.
- Fortunately, our patient has approximately 242 cm of healthy small bowel remaining including her ileocecal valve, and short bowel syndrome typically occurs in patients who have less than 200 cm of small bowel [2].
- At the time of presentation, this patient may have been at higher risk for thrombus due to her hypercoagulable state from both her longterm birth control use and her morbid obesity (with hepatic steatosis)
- In actuality, she was at greater risk due to her FVL mutation, another prothrombotic factor known to play a role in thrombotic events [4], and unfortunately had the perfect storm for SMV and portal vein thrombosis.

Conclusion

- AMI can quickly become a life-threatening disease if not recognized and treated expeditiously.
- It should be high on the differential in patients with risk of hypercoagulable states who present with abdominal pain.
- Although this patient was not diagnosed with FVL until after her acute illness was treated, she had other risk factors including obesity (with hepatic steatosis) and five-year history of birth control use, as well as a family history, which all support a predisposition for hypercoagulability and thrombosis.

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