

## CASE REPORT

# Mesenteric venous thrombosis precipitated by foodborne gastrointestinal illness

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## Abstract

Foodborne illnesses are common and are usually considered as part of the differential diagnosis when a patient presents with gastrointestinal symptoms including nausea, vomiting, abdominal pain, diarrhea and fever. The majority of foodborne illness is transient and self-limited, while life threatening complications are rare. Here, we describe a case of a patient presenting with inflammatory diarrhea after consumption of undercooked seafood. She developed mesenteric and portal venous thrombosis and small bowel infarction requiring surgical intervention and resection of gangrenous small bowel. This is a rare presentation and outcome of common food poisoning. The case report is followed by a brief discussion of common foodborne illnesses and mesenteric venous thrombosis.

## INTRODUCTION

Foodborne illnesses are common and are usually considered as part of the differential diagnosis when a patient presents with gastrointestinal symptoms including nausea, vomiting, abdominal pain, diarrhea and fever. The majority of foodborne illness is transient and self-limited, while life threatening complications are rare. Here, we describe a case of a patient presenting with inflammatory diarrhea after consumption of undercooked seafood. She developed mesenteric and portal venous thrombosis and small bowel infarction requiring surgical intervention and resection of gangrenous small bowel. This is a rare presentation and outcome of common food poisoning. The case report is followed by a brief discussion of common foodborne illnesses and mesenteric venous thrombosis (MVT).

## CASE REPORT

A 65-year-old female with a history of hypothyroidism and essential thrombocythosis (on hydroxyurea) presented with

complaints of acute-onset nausea, vomiting and diarrhea 2 days prior to admission. She had eaten seafood at a local restaurant the night before her symptoms started. Symptoms started within hours of eating that food. She complained of abdominal pain which was centered in the right lower quadrant and was colicky in nature. The intensity of pain varied from 6 to 7 on scale out of 10. On admission, the patient was found to be hypotensive. Abdominal exam revealed distended belly with hypoactive bowel sounds. A computed tomography (CT) scan of the abdomen done on admission did not show any acute findings. Mesenteric and portal veins were well opacified without any thrombus. The patient was given intravenous fluids and started on ciprofloxacin along with metronidazole. Despite this initial treatment, the patient's condition continued to worsen, with new-onset low grade fevers and persistent vomiting. Her abdominal tenderness worsened and bowel sounds remained hypoactive. A repeat CT scan on Day 3 of admission showed evidence of high-grade small bowel obstruction along with mucosal edema. It also showed thrombus in superior mesenteric vein (Fig. 1) and portal vein with early

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Figure 1: Thrombus in superior mesenteric vein and dilated small bowel loops.



Figure 2: Pneumatosis intestinalis.

gangrenous changes of small bowel. Based on these radiographic findings, she was taken for urgent laparotomy. Intraoperative findings included gangrene of the small bowel starting from 35 cm distal to the duodenojejunal flexure to the mid-transverse colon with no perforation. The superior mesenteric vein was thrombosed with cord like appearance. Based on these findings, the patient underwent a near-total small bowel resection. Postoperatively, the patient was started on enoxaparin for anticoagulation due to the findings of thrombosis in the mesenteric vein. Intravenous fluids and antibiotics were continued after the surgery. The patient initially responded favorably and was started on liquid diet. Improvement continued and antibiotics were discontinued on postoperative Day 6. Fourteen days after surgery, however, the patient developed acute abdominal pain. CT at this time showed pneumato-intestinalis (Fig. 2) of remaining part of proximal small gut along with pneumobilia and air in portal vein (Fig. 3), along with massive splenic and liver infarct. At this point, the family refused further intervention and the patient was transitioned to hospice care.

## DISCUSSION

Before we embark on a discussion of MVT, we will briefly discuss common foodborne illnesses. As per recent estimates,

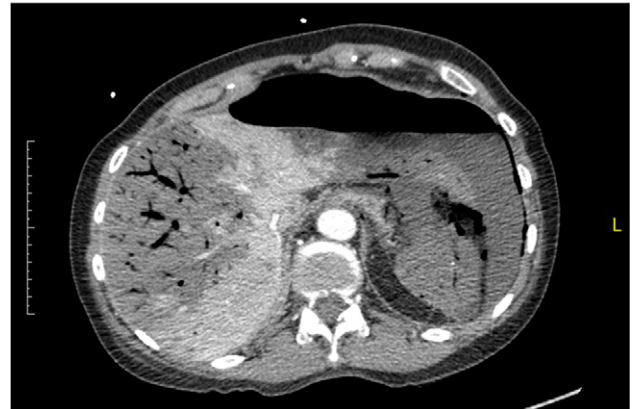


Figure 3: Air in portal vein branches.

there are 9.4 million foodborne illnesses in USA, requiring hospitalization in 55 961 cases and leading to 1351 deaths annually [1]. In the majority of cases, a diagnosis is made based on clinical presentation and generally does not require confirmatory laboratory tests. A typical case will present with nausea, vomiting, abdominal pain, diarrhea and fever. Some infections, however, will have additional features which can help distinguish the microbiologic cause of the illness. For example, jaundice and right upper quadrant pain are associated with Hepatitis A and E, paralysis is associated with botulism, and headaches and paresthesias are associated with Ciguatera fish poisoning or Scombroid. Even among the common symptoms of foodborne illness, some features tend to be associated more with certain pathogens than others:

- (1) Vomiting as the major presenting symptom is seen in food poisoning cases with *Staphylococcus aureus*, *Bacillus cereus*, noroviruses and anisakiasis.
- (2) Watery diarrhea is the most common presentation seen with *Vibrio cholera*, *Clostridium perfringens*, enterotoxigenic *Escherichia coli*, rotavirus, *Cryptosporidium parvum* and *Cyclospora*.
- (3) Inflammatory diarrhea, which is diagnosed by the presence of inflammatory cells and blood in stools, severe abdominal pain, along with fever is usually seen with *Salmonella*, *Campylobacter*, Shiga toxin producing *E. coli*, *Shigella*, non-cholera *Vibrio* and *Yersinia* [2].

The majority of these conditions are self-limited and resolve without any specific intervention. Volume repletion is the mainstay of management in such cases. Antibiotics are not routinely indicated and should generally be avoided except in patients with severe illness, those at high risk of complications including elderly or immunosuppressed patients, or when symptom persist for >1 week despite conservative management [3]. Some patients at high risk for more severe complications may experience hypotension, dehydration and occasionally peritonitis. MVT is a rare complication of foodborne illness. Given the low frequency of occurrence, the diagnosis may be delayed, as patients with inflammatory diarrhea have symptoms that overlap with symptoms of MVT. Therefore, a high degree of suspicion for MVT is critical to diagnosing the condition early enough to successfully intervene, especially in patients who may be predisposed to thromboses, such as those with polycythemia or essential thrombocythosis.

Acute MVT is an uncommon disorder with non-specific signs and symptoms. It was first described over 100 years ago by Elliot, who in 1895 published two cases of small bowel

infarction caused by thrombosis of mesenteric veins [4]. Among all mesenteric ischemic events, venous thrombus is estimated to account for only 5–15% cases [5]. MVT has been estimated to account for one in 5000–15 000 inpatient admissions and one in 1000 emergency surgical laparotomies for acute abdomen [6]. The superior mesenteric vein is more commonly involved than the inferior mesenteric vein. Venous thrombosis is predominantly a result of stagnation of blood flow, vascular injury and hypercoagulability. MVT is classified as either primary (idiopathic) or secondary to known etiologic factors. Underlying causes for MVT are found in ~75% of patients [7]. Risk factors for MVT include:

- (A) Heritable thrombophilias—antithrombin III deficiency, Factor V Leiden mutation, hereditary hemorrhagic telangiectasia, hyperfibrinogenemia, plasminogen deficiency, protein C and S deficiency, prothrombin gene mutation and Sickle cell disease.
- (B) Acquired thrombophilias and systemic hypercoagulable states—antiphospholipid antibodies, polycythemia vera, essential thrombocythosis, paroxysmal nocturnal hemoglobinuria, heparin-induced thrombocytopenia, myeloproliferative disease, malignancy, pregnancy, decompression sickness and disseminated intravascular coagulation.
- (C) Intra-abdominal causes—cirrhosis, inflammatory bowel disease, intestinal volvulus, intra-abdominal infection, pancreatitis and postoperative state.

Presentation of MVT can be acute, subacute or chronic [8]. Acute MVT presents with sudden onset of abdominal pain progressing to signs and symptoms of bowel infarction and peritonitis. Subacute cases present with abdominal pain for days to weeks without gut infarction, while chronic cases generally present with complications of portal vein or splenic vein thrombosis like esophageal variceal bleeding.

A high degree of suspicion is required to make timely diagnosis in acute cases, due to both the rarity of the disease as well as the severe clinical consequences of delayed intervention. Lactate is generally elevated due to gut ischemia, but may be normal upon initial presentation. Other common signs include elevated white blood cell count and stool positive for occult blood, which is present in over 80% of cases [6]. However, no laboratory test is specific for MVT, so diagnosis generally requires abdominal imaging. Plain film X-ray, while not particularly sensitive or specific, may reveal mucosal edema, pneumatosis intestinalis, or air in the portal vein or peritoneal cavity due to infarcted bowel [9]. Ultrasound with Doppler can often detect thrombosis in mesenteric veins; however, technical difficulties such as air in gut loops obscuring proper evaluation of blood vessels can limit the utility of this test. Magnetic resonance venography is quite sensitive; however, it may not be feasible in acute cases as patients are usually significant pain and may not be clinically stable enough to undergo a prolonged test. CT remains the imaging modality of choice to diagnose MVT, reliably identifying the disease in over 90% of cases. CT findings include central lucency in the mesenteric vein (filling defect), enlargement of the involved vein and a sharply defined wall of vein with a rim of increased density. All of these findings are evident even in the early stages of the MVT. In later stages after infarction has occurred, CT may show pneumatosis intestinalis and portal venous gas [8]. Other diagnostic techniques include abdominal paracentesis and selective mesenteric venous angiography; however, with the wide availability of CT, these methods are now rarely used to diagnose MVT.

The goal of initial treatment for acute MVT is to prevent intestinal infarction by reperfusion of the affected bowel. Initial treatment for all patients should include bowel rest,

nasogastric suction, intravenous fluids, prophylactic antibiotics and parenteral anticoagulation. Surgery is indicated in patients who show signs and symptoms of peritonitis, hemodynamic instability or bowel infarction [10]. The mortality rate associated with acute MVT ranges from 20 to 50% [11]. In patients who survive an initial episode, recurrence may present within 1 month of initial presentation. The recurrence rate is lower in patients who receive both anticoagulation and surgery compared to those treated with anticoagulation alone [11].

In conclusion, we present a case of patient who developed mesenteric and portal venous thrombosis after a foodborne illness. She was predisposed to MVT given her underlying hypercoagulability as well as the dehydration and inflammation associated with the diarrhea from her acute gastrointestinal illness. As a result of the MVT, she subsequently developed infarcted small bowel and splenic and hepatic infarcts. She underwent laparotomy where infarcted small bowel was resected and then was started on systemic anticoagulation. Despite all these efforts she was not able to fully recover and eventually expired. This case is unique as she developed this life threatening condition after a minor bout of food poisoning, which in most cases would not prompt further diagnostic workup. The case highlights the importance of maintaining a high degree of suspicion for MVT in patients who present with severe acute gastrointestinal illness that may be predisposed to thrombosis, allowing for prompt identification and treatment of the condition.

## CONFLICT OF INTEREST STATEMENT

None declared.

## REFERENCES

- Scallan E, Hoekstra RM, Angulo FJ, Tauxe RV, Widdowson M-A, Roy SL, et al. Foodborne illness acquired in the United States—major pathogens. *Emerg Infect Dis* 2011;17:7–15.
- American Medical Association, Centers for Disease Control and Prevention, Center for Food Safety and Applied Nutrition, Food and Drug Administration & Food Safety and Inspection Service, US Department of Agriculture. Diagnosis and management of foodborne illnesses: a primer for physicians. *MMWR Recomm Rep Morb Mortal Wkly Rep Recomm Rep* 2001;50:1–69.
- Dryden MS, Gabb RJ, Wright SK. Empirical treatment of severe acute community-acquired gastroenteritis with ciprofloxacin. *Clin Infect Dis Off Publ Infect Dis Soc Am* 1996;22: 1019–25.
- Elliot JW II. The operative relief of gangrene of intestine due to occlusion of the mesenteric vessels. *Ann Surg* 1895;21:9–23.
- Grendell JH, Ockner RK. Mesenteric venous thrombosis. *Gastroenterology* 1982;82:358–72.
- Harnik IG, Brandt LJ. Mesenteric venous thrombosis. *Vasc Med Lond Engl* 2010;15:407–18.
- Kumar S, Sarr MG, Kamath PS. Mesenteric venous thrombosis. *N Engl J Med* 2001;345:1683–8.
- Boley SJ, Kaleya RN, Brandt LJ. Mesenteric venous thrombosis. *Surg Clin North Am* 1992;72:183–201.
- Tomchik FS, Wittenberg J, Ottinger LW. The roentgenographic spectrum of bowel infarction. *Radiology* 1970;96:249–60.
- Chen C. Direct thrombolytic therapy in portal and mesenteric vein thrombosis. *J Vasc Surg* 2012;56:1124–6.
- Rhee RY, Gloviczki P, Mendonca CT, Petterson TM, Serry RD, Sarr MG, et al. Mesenteric venous thrombosis: still a lethal disease in the 1990 s. *J Vasc Surg* 1994;20:688–97.