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Superior mesenteric vein thrombosis as a complication of cecal diverticulitis: A case report

Soniya Pinto[a,*], Terrence Lerner[b], Gowtham Lingamaneni[a], Ken Richards[c]

[a] University of Illinois Chicago (Metropolitan Group Hospitals), Department of General Surgery, United States
[b] Advocate Illinois Masonic Medical Center, Department of General Surgery, United States
[c] Mercy Hospital and Medical Center, Department of General Surgery, United States

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ABSTRACT

Pylephlebitis is an uncommon complication of uncontrolled intra-abdominal infection that is associated with high morbidity and mortality. We present our experience with a unique case of cecal diverticulitis and septic thrombophlebitis of the superior mesenteric vein that was promptly diagnosed with high-resolution imaging and blood cultures. Antibiotic and anticoagulation therapy was instituted on confirming the diagnosis with magnetic resonance imaging (MRI) to control the infection and prevent propagation of the thrombus. Our case report raises awareness about a rare and potentially fatal condition and provides appropriate imaging supplementation to aid in timely diagnosis.

1. Introduction

Pylephlebitis or suppurative thrombosis of the portal mesenteric venous system is a rare complication of intra-abdominal inflammatory processes such as diverticulitis, appendicitis, pancreatitis and inflammatory bowel disease [1–5]. Clinical symptoms are non-specific and include fevers, abdominal pain and nausea [6]. Diagnosis requires imaging findings of thrombosis in the portal venous system on computed tomography (CT) or MRI in conjunction with clinical symptoms [7,8]. If untreated, the condition is potentially fatal. Current treatment regimens are largely based on anecdotal experience with this condition. While antibiotic therapy is a universal recommendation, the role of anticoagulation is controversial with most studies failing to demonstrate a statistically significant benefit [9–11]. We present a case of suppurative thrombophlebitis of the superior mesenteric vein secondary to cecal diverticulitis. We have highlighted pertinent imaging findings and modalities to treat this condition.

2. Presentation of case

A 57 year-old African-American male with a past medical history of diabetes, hypertension, benign prostatic hyperplasia and asthma presented to the emergency room with three days of epigastric pain, nausea, vomiting and an inability to tolerate solid food. He reported regular bowel movements and was passing flatus on presentation. He had no prior abdominal surgeries and no family history of crohn's disease, ulcerative colitis or gastrointestinal malignancies. His medications included terazosin, lisinopril, amlodipine and metformin. He reported smoking half a pack of cigarettes/day for the last 40 years and using heroin intermittently over the last few months.

On Physical exam, he was morbidly obese with a BMI of 36.32. He was febrile to 38.4, tachycardic to 102/minute and saturating within normal limits on room air. His abdomen was soft, non-distended without tenderness, guarding or rebound. He had normoactive bowel sounds and his rectal exam was unremarkable with hemoccult negative stool in the rectal vault.

There were no acute changes in electrocardiography or troponin level. Hematology showed leukocytosis with neutophilia and bandemia. His basic metabolic panel was remarkable for hyponatremia, hypochloremia and hyperglycemia. The most concerning laboratory finding was his elevated total bilirubin of 7.2 mg/dl with a direct bilirubin of 5.1 mg/dl. His alkaline phosphatase, serum transaminases and lipase were within normal limits. His procalcitonin level was elevated to 58.2 ng/ml (normal < 0.1 ng/ml). Blood cultures were collected and sent for microbiological analysis.

Abdominal contrast-enhanced CT revealed scattered colonic diverticulosis with abnormal stranding of the mesenteric vessels in the posterior midline, concerning for mesenteric thrombophlebitis. There was associated thickening of the distal ileum and cecum along the inferior boundary of the process, likely due to cecal diverticulitis (Fig. 1A). In addition, there was a questionable thrombus at the confluence of the superior mesenteric vein and portal vein

* Corresponding author.
E-mail addresses: soniapinto173@gmail.com (S. Pinto), tlernermd@gmail.com (T. Lerner), drroy11@gmail.com (G. Lingamaneni), kenrichards.md@gmail.com (K. Richards).

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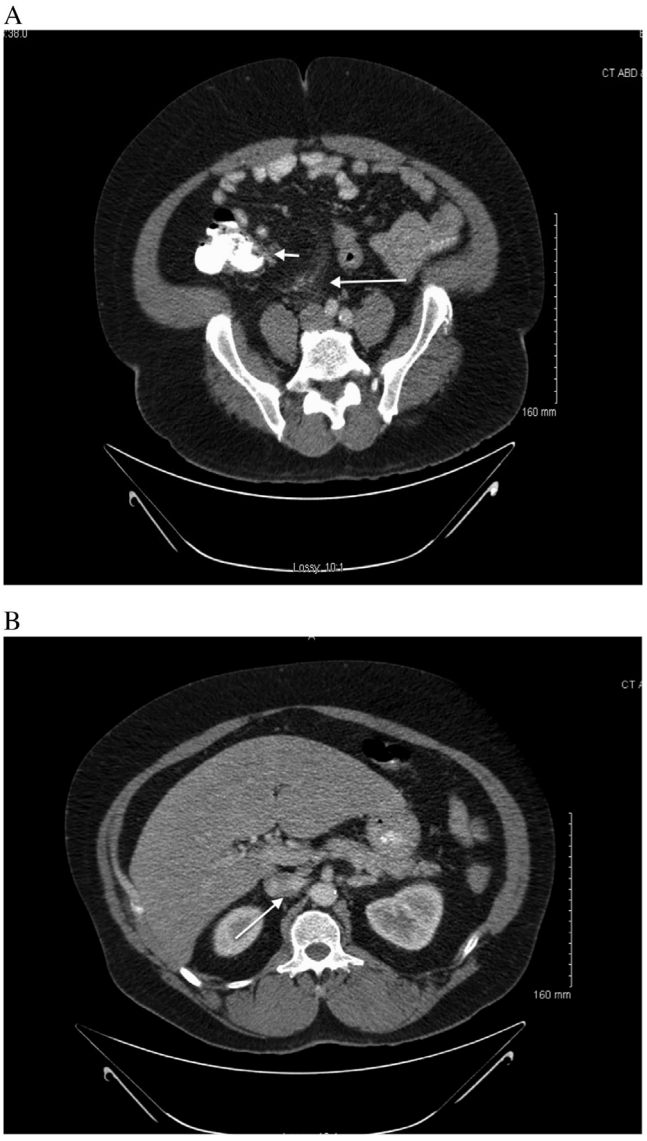

Fig. 1. (A) CT abdomen and pelvis with oral and intravenous contrast. Abnormal fat stranding of the mesentery (long arrow) and thickening of the terminal ileum (short arrow). (B) Suspected thrombus at the confluence of the superior mesenteric and portal veins (arrow).

(Fig. 1B). The biliary system was unremarkable on CT. An MRI was recommended for further evaluation.

The patient was admitted and bowel rest along with aggressive fluid resuscitation and broad-spectrum antibiotics were initiated. MRI confirmed the presence of a thrombus in the distal superior mesenteric vein just below the confluence of the portal vein (Fig. 2). In conjunction with the CT findings earlier, this was thought to be due to the propagation of mesenteric thrombophlebitis, secondary to cecal diverticulitis. An unfractionated heparin drip was initiated.

Over the next two days, he was afebrile, his leukocyte count trended downward and his symptoms improved significantly. His bilirubin continued to trend downward, ultimately reaching a level of 2.6. A hepatitis panel was non reactive. His procalcitonin decreased from an original 58–14.5 ng/ml.

His blood cultures confirmed the presence of beta lactamase producing Bacteroides fragilis, sensitive to ampicillin-sulbactam and his antibiotic regimen was accordingly modified. He was started on a liquid diet, which was gradually advanced to solid foods as tolerated. He was switched to oral amoxicillin-clavulanate and

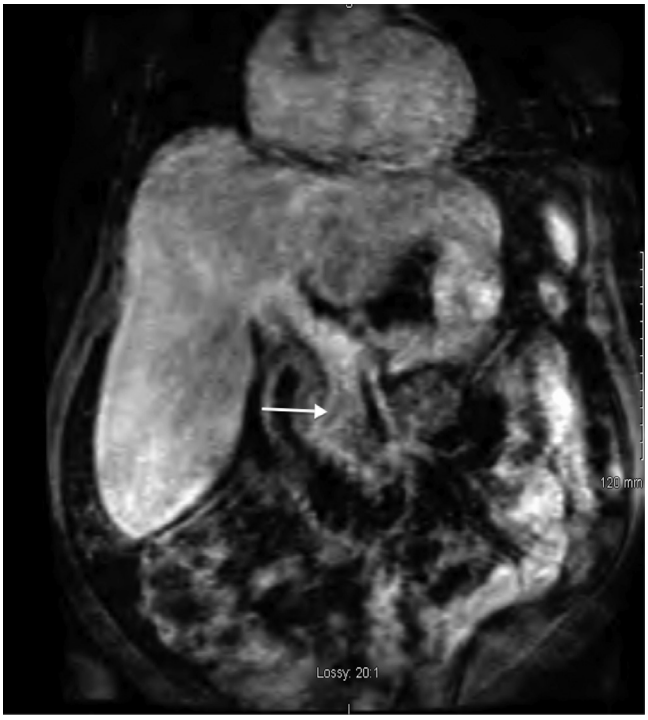

Fig. 2. T1- weighted coronal magnetic resonance imaging of the abdomen showing thrombus within the distal superior mesenteric vein just below the confluence of the portal vein (arrow).

bridged to coumadin. His anticoagulation regimen was later modified to rivaroxaban. He was discharged home after eight days on an additional 10 days of amoxicillin-clavulanate and 6 months of rivaroxaban with close follow up with his hematologist.

3. Discussion

The above case illustrates the vague clinical presentation of pylephlebitis in conjunction with an intra-abdominal inflammatory process. The elevated bilirubin raised clinical suspicion for portal pyemia and further workup was tailored toward detecting the disease. Although CT findings were equivocal, testing was pursued with MRI. Antibiotic and anticoagulation therapy was initiated immediately on diagnosis.

Our patient presented with gastrointestinal symptoms, fevers and leukocytosis with a left shift, suggestive of an intra-abdominal infection. Belhassen-García et al. in their review of 7796 patients from 1999 to 2006 noted that 13 (0.6%) patients with intra-abdominal infections developed pylephlebitis [3]. The incidence of this condition has been estimated at about 2.7 per 100,000 patient years with diverticulitis being the most common inciting event [1–7]. Other common pathologies include appendicitis, pancreatitis and inflammatory bowel disease [4–7]. Smoking and obesity, as in the case presented above were likely contributing factors to the development of superior mesenteric venous thrombus. A high index of suspicion must be maintained in patients with uncontrolled abdominal infections and risk factors for hypercoagulability [8]. Our ultimate diagnosis of pylephlebitis was based on MRI findings of superior mesenteric venous thrombosis and the growth of Bacteroides fragilis from peripheral blood cultures. Pylephlebitis is definitively diagnosed when venous thrombosis is demonstrated in conjunction with culture positive fluid from the portal venous system. However, portal vein aspiration is seldom performed, given its invasiveness. A review of 95 cases of pylephlebitis associated with intra-abdominal infection by Choudhry