



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



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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.

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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 epi-

gastric 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 neutrophilia 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

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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

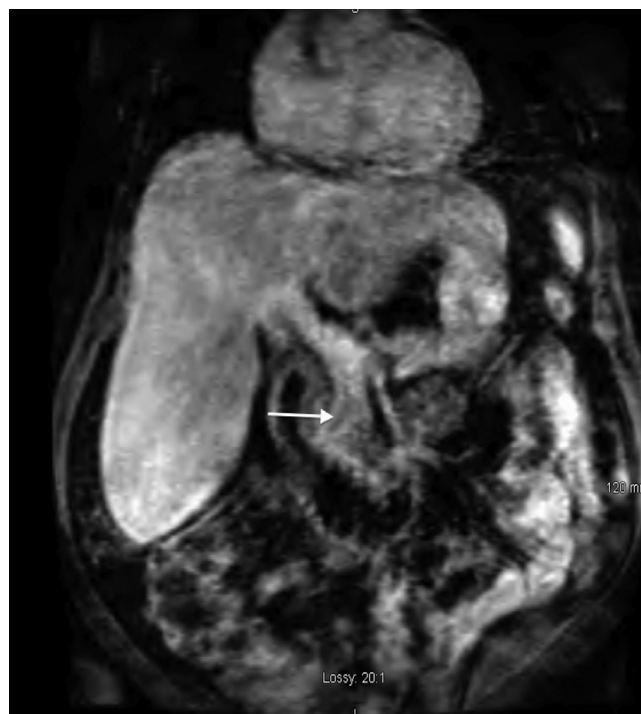


Fig. 2. T1-weighted coronal magnetic resonance imaging of the abdomen showing thrombus within the distal superior mesenteric vein just below the confluence of the portal vein (arrow).

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

et al. defined this condition as portal mesenteric venous thrombosis with or without bacteremia within 30 days of an intra-abdominal inflammatory process [9]. The review included patients with diverticulitis, pancreatitis, inflammatory bowel disease and appendicitis who consequentially developed suppurative thrombophlebitis of the right portal (31 patients), left portal (23 patients), main portal (30 patients), superior mesenteric (29 patients), inferior mesenteric (8 patients) or splenic veins (17 patients). There were patients who had two or more sites of involvement in each category. The right and the main portal veins were the most common sites of involvement.

The imaging findings in the case above were suggestive of superior mesenteric venous thrombosis on CT (Fig. 1B). The portal venous phase has the highest diagnostic value for pylephlebitis and can detect sequelae such as hepatic abscesses and intestinal infarction. Characteristic imaging findings on CT include a well-defined intraluminal-filling defect with central low density surrounded by rim-enhancing venous walls [12]. MRI demonstrated a distinctive central thrombus in the distal superior mesenteric vein just below the confluence of the portal vein (Fig. 1B). Fresh thrombi less than five weeks old are usually hyperintense on T1 and T2 weighted sequences compared to the liver parenchyma and the surrounding muscle tissue [12]. Other useful imaging modalities include duplex ultrasound, which typically demonstrates a flow defect with proximal dilation or absent compressibility of the affected vein [12]. Baril et al. and Plemmons et al. recommended that blood cultures should be drawn in cases of suspected pylephlebitis; however, the incidence of bacteremia varies widely between 23–88% [10,11]. *Bacteroides fragilis* is the most commonly isolated organism in monomicrobial infections of the portal mesenteric system [6]. *Bacteroides* is known to activate the coagulation cascade via the production of anticardiolipin antibodies, which break down heparin. The capsular components of the organism accelerate fibrin cross-linking and thereby, promote thrombosis. Other frequent isolates include *Streptococcus viridans*, *Escherichia coli* and polymicrobial sources.

Our case reported an unusual site of venous thrombosis in the distal superior mesenteric vein. Baril et al. reported the main portal venous system is a far more common site of involvement compared to the mesenteric veins [10].

Once a diagnosis was established initial broad-spectrum antibiotic therapy was instituted, which was later modified based on blood culture results. A dramatic improvement in clinical and laboratory parameters was achieved. The largest institutional retrospective review by Choudhry et al. recommended empiric antibiotic therapy in 86 patients with pylephlebitis [9]. Plemmons et al. have recommended intravenous antibiotic therapy until a significant response occurs followed by oral antibiotic therapy. A total duration of 4 weeks of antibiotic therapy was recommended by this study [11]. There are currently no societal or organization guidelines on the duration of antibiotic therapy given the rarity of the disease. In the above case, a total of 18 days of antibiotic therapy was administered based on the recommendations of a team of infectious disease specialists. Delays in management of pylephlebitis have been associated with mortality rates as high as 25% [9]. Early broad-spectrum antibiotic therapy, even in the absence of positive bacteremia has proved to decrease the incidence of potentially fatal conditions such as bowel ischemia and hepatic abscesses [9].

In the case presented above, there were no contra-indications to the use of long-term anticoagulation and the decision was made to proceed with therapeutic anticoagulation. Baril et al. studied 44 patients with septic thrombophlebitis of the portal vein and reported 5 deaths in the group of 32 patients who did not receive anticoagulation vs. none in the 12 that did receive anticoagulation [10]. Duffy et al. demonstrated recanalization of the

portal vein after anticoagulation in a patient with pylephlebitis secondary to diverticulitis [13]. Kanellopoulou et al. examined 100 cases of pylephlebitis, all of whom received antibiotic therapy and 35 of who received anticoagulation in addition to antibiotic therapy. Patients who received anticoagulation had a favorable outcome compared to those who received antibiotics alone (complete recanalization 25.7% vs. 14.8% ($p > 0.05$), no recanalization 5.7% vs. 22.2% ($p < 0.05$) and death 5.7% vs. 22.2% ($p < 0.01$)) [6]. On the other hand, a retrospective review by Choudhry et al. which included 95 patients with portal, mesenteric and splenic pylephlebitis showed similar mortality rates when antibiotics were used with or without anticoagulation (10 vs. 20%, $p = 0.265$) [11]. Given our limited experience with the disease, our recommendation is that anticoagulation therapy should be individualized, carefully weighing the risks with the benefits. In the absence of an underlying coagulopathy, Allaix et al. recommended 3–6 months of anticoagulation treatment [14]. As yet, the end point of anticoagulation has not been well defined [14,15].

Our study did present certain limitations in terms of diagnostic workup and follow up. Although cecal diverticulitis was our primary diagnosis on imaging, alternative considerations such as Crohn's disease and septic thromboemboli from infective endocarditis were not pursued. Furthermore, our patient did not receive a follow up MRI after completion of antibiotic and anticoagulation therapy.

4. Conclusion

Our case report summarizes the clinical features, imaging and therapeutic modalities of an unusual entity. To date, ours is the only case report on pylephlebitis secondary to cecal diverticulitis that illustrates the utility of MRI when CT findings are equivocal [4,16]. Our results are a valuable addition to surgical literature in terms of guiding clinical judgment and improving outcomes.

Disclaimer

We hereby certify that the views expressed in this paper are our own, and not an official declaration of the institutions.

Conflict of interest

None.

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Ethical approval

Not indicated.

Consent

Obtained from patient.

Author contribution

Soniya Pinto: data collection, drafting, submission, correspondence.

Terrence Lerner: conceptualization, reviewer.

Gowtham Lingamaneni: data collection.

Ken Richards: Conceptualization, reviewer.

Guarantor

Soniya Pinto, MD.

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