case report

Portal-mesenteric vein thrombosis as an unusual presentation of Meckel's diverticulum complication

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Portal-mesenteric vein thrombosis (PMVT) is an uncommon cause of bowel ischemia in adults.¹ It was first recognized more than 100 years ago by Eliot,² who described intestinal gangrene resulting from mesenteric venous occlusion. Most patients with PMVT have an underlying pathology related to Virchow's triad of hypercoagulopathy, stasis and endothelial injury. A primary hypercoagulable state contributes to 42% of PMVT cases.³ However, an intraabdominal pathology secondary to inflammatory,⁴ neoplastic, and infectious processes⁵ should not be overlooked as a potential etiology. To our knowledge, this is the first reported case of PMVT as an initial presentation of Meckel's diverticulum complication. The clinical presentation of PMVT and diagnosis as well as the main therapeutic approaches suggested in the literature are presented.

CASE

A 58-year-old Saudi woman with a history of hypertension presented to the emergency room with abdominal pain of 4-week duration. The pain was periumbilical and crampy in nature. No history of change of bowel habits, bleeding per rectum, or previous surgery was reported. No history of similar attacks or a change of color of urine or stool was reported. The patient denied a history of smoking or drinking. She had no known drug allergies and was on amlodipine for hypertension. She denied personal or family history of coagulopathy or cancer.

On examination, the patient appeared well and was in mild distress. Her blood pressure was 150/90, with a pulse of 90 and a temperature of 99.0°F. The cardiopulmonary examination was normal. The abdomen was mildly distended. Bowel sounds were diminished. There was mild tenderness to palpation in the lower quadrants but no rebound or guarding. Rectal exam revealed no masses and the stool was heme negative. Abnormal laboratory results included an elevated white blood

cell count of $31.0 \times 10^3/\mu\text{L}$. Electrolytes, lipase, amylase, liver enzymes, and the remainder of the complete blood count were normal. An abdominal series did not demonstrate gas-fluid levels or dilated loops of bowel. A computed tomography (CT) scan of the abdomen and pelvis with contrast revealed PMVT, free fluid in the peritoneal cavity and thickened small bowel (Figure 1). A paracentesis, performed to rule out catastrophe, yielded serosanguinous fluid. The patient was admitted for observation, heparinization, intravenous hydration and broad-spectrum antibiotics.

Her symptoms failed to improve upon conservative treatment and she developed small bowel obstruction postadmission. An exploratory laparotomy was performed. The intestine was viable and normal, but there was an inflamed perforated Meckel's diverticulum with an intraabdominal abscess. A 30-cm segment of congested maroon-colored small bowel was resected, including the perforated Meckel's diverticulum (Figure 2), and a primary anastomosis was performed. Sectioning through the mesentery revealed a number of intravascular thrombi. Microscopic examination of the bowel showed infarction involving all layers, with congestion, severe edema, and hemorrhage. The veins within the mesentery were filled with thrombi that had undergone early organization. The Meckel's diverticulum had an ectopic pancreatic tissue. Operative culture failed to grow an organism.

The patient did well following surgery and was restarted on intravenous heparin, and subsequently placed on warfarin prior to discharge. Laboratory results including amylase, lipase, and a hypercoagulable profile were normal. The patient was discharged 10 days after surgery on warfarin therapy and remained well at a 6-month follow-up.

DISCUSSION

In autopsy studies, PMVT is found in approximately

Figure 1. (A) CT scan of abdomen showing portal vein/ superior mesenteric vein filling defect (arrow) with splenic vein thrombosis. Note the mild splenomegaly and area of splenic infarct. (B) Coronal cut of a CT scan of the abdomen showing extensive thrombosis of the portal vein with intrahepatic extension (arrow).

1.5% of the population.¹ Mesenteric thrombosis may be idiopathic or secondary to an associated medical condition. The proportion of patients with primary, or idiopathic, mesenteric venous thrombosis continues to decline as our diagnostic and laboratory power improves. Currently, an etiologic risk factor (Table 1) can be identified in about 75% to 84% of patients.⁶

Our patient presented with PMVT in association with an inflamed Meckel's diverticulum with perforation and intraabdominal abscess. We propose that the inflammatory process of Meckel's diverticulum and stasis secondary to dehydration could have initiated the acute thrombosis of the superior mesenteric vein (SMV). In addition, infection can cause thrombosis and ascending phlebitis. Anaerobic infections,¹⁷ cytomegalovirus, *Yersinia*, and *E. coli* have all been implicated in PMVT. A similar scenario was reported in cases with appendicitis and sigmoid diverticulitis.⁵

The clinical presentation of mesenteric venous thrombosis can be acute, with a sudden onset of symptoms, subacute or chronic, with portal or splenic venous thrombosis and variceal bleeding. The hallmark of PMVT is a generalized crampy abdominal pain that is out of proportion to physical findings. Most patients with PMVT had pain for 2 to 30 days before seeking medical attention.^{7,8} Historically, a previous occurrence of spontaneous venous thrombosis should raise suspicion. Nausea, vomiting, and diarrhea are inconsistent findings. Hematemesis and hematochezia have been found with PMVT, but these are rare and represent

advanced ischemia and necrosis. Fever is usually absent or low grade. Bloody ascites and large fluid losses with third-spacing can lead to dehydration, causing further propagation of the venous thrombosis and worsening of the ischemia.

Serum laboratory test findings, such as leukocytosis, and metabolic acidosis with elevated serum levels of lactate, anion gap and amylase, are not specific for the diagnosis of acute PMVT. The diagnostic value of D-dimer, which is used extensively for the diagnosis of venous thromboembolism of the extremities, remains controversial.⁹ Hyperphosphatemia and hyperkalemia are associated with bowel infarction and are usually late signs.¹⁰ Patients with PMVT should be screened for a hypercoagulable state. This includes tests of antithrombin III, protein C, protein S, and fibrinogen levels. In general, it is best to wait 1 to 2 weeks after warfarin has been discontinued before protein C level measurement.

Because of nonspecific symptoms, the diagnosis and initial treatment are often delayed. Plain radiographs and barium exams are generally nonspecific. If PMVT is suspected on the basis of clinical presentation, a CT scan with intravenous contrast should be performed, which has a sensitivity as high as 90% to 100%.¹¹ Typical findings include central lucency filling defect representing thrombus formation (Figure 1) with a high-density venous wall. Persistent enhancement of the bowel wall, pneumatosis intestinalis, and portal-vein gas are late findings.

Other imaging modalities useful in PMVT diag-

Figure 2. Gross picture of the specimen (arrow pointing at Meckel's diverticulum).

nosis include ultrasound duplex,¹² magnetic resonance imaging¹³ and angiography.¹¹ Angiographic findings in PMVT include demonstration of a thrombus in the SMV with partial or complete occlusion, failure to visualize the SMV, slow or absent filling of the mesenteric veins, arterial spasm, prolongation of the arterial phase and failure of arterial arcades to empty, reflux of contrast into the artery, and a prolonged blush in the involved segment of the bowel. In symptomatic patients with acute PMVT, the choice of treatment is determined by the severity of peritoneal signs. In the absence of peritoneal signs, anticoagulant therapy should be started immediately. Patients are first treated with heparin for 7 to 10 days, and then oral warfarin once there is no evidence of ongoing ischemia. The duration of treatment varies from 6 months to lifelong, depending on the risk of coagulopathy or recurrence.⁷ Other supportive measures include nasogastric suction, fluid resuscitation, broad-spectrum antibiotics and bowel rest.

Immediate exploratory laparotomy is needed in cases with signs of transmural bowel infarction and peritonitis, or hemodynamic instability.¹⁵ The aim of resection is to conserve as much bowel as possible to decrease the risk of short-gut syndrome. Second-look laparotomy, 24 hours later, is preferable to an extensive resection of a borderline viable bowel. Heparinization in the post-operative period has been shown to reduce the recurrence of thrombosis from 26% to 14% and mortality from 59% to 22%.^{7,16} On rare occasions, thrombectomy can be accomplished successfully when the thrombus is recent and is restricted to the SMV. However, surgical thrombectomy has a relatively limited role because of numerous inaccessible clots within the mesenteric venules, and the potential of intimal damage from balloon

Table 1. Conditions associated with mesenteric vein thrombosis.

Direct endothelial injury
Abdominal trauma
Postoperation (postsplenectomy, laparoscopy)
Intra-abdominal inflammatory state (pancreatitis, inflammatory bowel disease)
Peritonitis and abdominal abscess
Diverticulitis
Stasis
Portal hypertension/ cirrhosis
Congestive heart failure
Hypersplenism
Hypercoagulable state
Protein C and protein S deficiency
Antithrombin III deficiency
Activated protein C resistance (factor V leiden gene mutation)
Presence of the 20210 A allele of the prothrombin gene
Methylenetetrahydrofolate reductase mutations
Homocysteinemia
Neoplasms (particularly pancreatic and colonic)
Oral contraceptive use
Pregnancy
Nephrotic syndrome
Polycythemia vera/essential thrombocythosis/paroxysmal nocturnal hemoglobinuria
Heparin-induced thrombocytopenia
Lupus anticoagulant/antiphospholipid syndrome

embolectomy. The more diffuse venous thrombosis seen in the acute form of the condition precludes thrombectomy.

Other proposed methods of treatment include thrombolysis. Thrombolysis is suggested in cases of thrombosis without or prior to the development of bowel ischemia in an attempt to limit or prevent bowel infarction. The thrombolytic agents can be infused using several approaches, such as via the SMV, or internal jugular vein, or transhepatically via the portal vein.¹⁴

The prognosis of patients with PMVT is less ominous compared to arterial thrombosis. The mortality rate among patients with PMVT ranges from 20 to 50 percent.^{7,3} Survival depends on multiple factors,

including age, coexisting underlying conditions, and the timing of the diagnosis and surgical intervention. Postoperative complications include sepsis, wound infection, stricture and short-bowel syndrome.

There are no previous reports of venous thrombosis complicating Meckel's diverticulum as far as we know. With the increased use of CT scanning in the emergency room, PMVT will be seen more frequently in association with various abdominal pathologies. We emphasize that PMVT should be managed with a high index of suspicion, early diagnosis, and prompt anticoagulation. Surgery can be reserved for bowel infarction or treatable inciting intraabdominal pathology. This seems to improve survival and reduce recurrence.

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