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Surgical Management of Chronic Mesenteric Venous Thrombosis

A Case Report

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Although mesenteric venous thrombosis is an uncommon disease, it is a diagnostic dilemma and if left untreated results in significant morbidity and mortality. The clinical presentation of mesenteric venous thrombosis (MVT) is varied and depends on the etiology. Prompt recognition and treatment is important as this may limit the progression of thrombosis. Even though medical management is the current mainstay of therapy, there have been reported cases of clinical improvement with operative management. The authors describe an improved outcome following mesenteric-systemic shunting in a symptomatic patient with acute-on-chronic MVT affecting the proximal superior mesenteric vein.

Introduction

Mesenteric venous thrombosis (MVT) was first described as a discrete clinical entity in 1935.¹ MVT is an uncommon but potentially fatal form of mesenteric ischemia. MVT accounts for 5% to 15% of clinically significant cases of mesenteric ischemia² and is reported to have a mortality rate of 30% to 80%.³

Several published reports have described the difficulty in diagnosing and managing mesenteric venous thrombosis and coexisting/related pathologic entities. Currently, there is no consensus on the initial treatment for MVT, probably because treatment varies with the reported etiology. Acute MVT describes patients with acute abdominal symptoms (ie, anorexia, diffuse abdominal pain, abdominal distension, diarrhea) with a history of previous thrombotic episode. On the other hand patients with chronic MVT present with late complications of portal or splenic vein thrombosis such as variceal bleeding and ascites. The patient described in this case report with acute-on-chronic episodes of MVT (secondary to chronic superior mesenteric vein occlusion) was initially unresponsive to medical management but was amenable to surgical therapy. The exact duration of venous occlusive disease is unknown.

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Case Report

A 56-year-old woman had had multiple admissions for ongoing abdominal pain. Her complex surgical history is significant for a number of abdominal procedures including a cholecystectomy, splenectomy, and a small bowel resection for ischemia. Her medical history suggested the presence of a hypercoagulable state, with her first deep vein thrombosis (DVT) occurring at age 13 years. She had been on anticoagulation for about 6 months but stopped because of rectal bleeding, as well as nasal and auricular bleeding. An inferior vena cava (IVC) filter was placed. She did well for 6 years but over the last year required multiple hospital admissions for nonspecific abdominal pain and distention for problems suggestive of small bowel obstruction. Computed tomography (CT) scans demonstrated inflammation of the right colon with a fluid collection near the right gutter and the pericecal area. The most recent CT scan revealed absence of the superior mesenteric vein (SMV) (Figure 1). Multiple endoscopies revealed engorged veins in the colonic wall at the level of the hepatic flexure and ischemic changes of the colonic mucosa. Visceral angiography with venous phase study showed occlusion of the proximal 4 centimeters of the superior mesenteric vein. There was evidence of extensive venous collateralization in the region of the hepatic flexure, and poor opacification of the distal arterial small vessels to the left colon was also noted.

Her abdominal angiogram with venous phase study had also demonstrated opacification of the inferior mesenteric vein (IMV) as well as a patent portal and splenic vein. Large venous collaterals in the mesentery of the small bowel were also noted. These collaterals were thought to have developed secondary to the central venous occlusion in the SMV responsible for the severe SMV hypertension. Our clinical impression was that central superior mesenteric venous occlusion and venous hypertension were responsible for her signs and symptoms of pain, weight loss, and failure to thrive.

Therefore, she was brought to the operating room with the intention of trying to decompress the obstructed mesenteric venous circulation by providing a bypass into the systemic venous circulation. Our plan was to try and use the inferior mesenteric vein to establish the bypass into the systemic venous side (particularly into the left renal vein). This decision was based on the premise that anatomically there exists a communication between the SMV and the IMV, from the left



Figure 1. Reconstructed computed tomography scan of the abdomen with arrow pointing to a proximal occlusion of the superior mesenteric vein.

branch of the middle colic vein to the left colic vein (a tributary of the IMV). Therefore, high pressure in the obstructed SMV would be transmitted to the IMV, and by connecting the IMV to the left renal vein the obstructed mesenteric venous outflow could be decompressed.

On entering the abdomen the inferior mesenteric vein was identified and found to be distended and clearly under tense pressure. The proximal end of the inferior mesenteric vein was dissected free up to the first large major collateral vein at the level of the inferior border of the pancreas, where it was ligated proximally and divided.

The left renal vein was next identified crossing over the aorta and controlled with vessel loops. Following systemic heparinization, an end-to-side anastomosis was performed between the distal segment of the inferior mesenteric vein and the renal vein. On completion, flow was established through this bypass with good decompression of the mesenteric vessels.

The patient's postoperative course was benign and a follow-up CT scan showed patency of the IMV-to-left renal vein bypass with improvement

in the inflammatory changes that had been earlier seen around the right colon (Figure 2). In addition, decreased edema in the wall and reduced fluid around the colon were also noted. The patient resumed normal dietary intake, was placed on a regimen of aspirin and clopidogrel, and is being followed up as an outpatient.

Discussion

Ischemia of the intestine is a potentially fatal disease if left untreated, occurring as a result of a diverse range of pathological conditions. MVT is a poorly understood condition that leads to intestinal ischemia. Several conditions have been reported as being associated with MVT and have been classified into 4 main categories: traumatic, inflammatory, hematologic, and mechanical obstruction/low-flow states.⁴ Some of these conditions include direct venous injury following abdominal trauma or splenectomy, pancreatitis, inflammatory bowel disease, portal hypertension/

liver cirrhosis, protein C and protein S deficiency, antithrombin III deficiency, neoplasms, polycythemia vera, and lupus anticoagulant/antiphospholipid syndrome. When MVT occurs in the absence of these conditions it is referred to as idiopathic or primary MVT.

Although the symptoms and signs of MVT are widely variable, the time course of these clinical manifestations is characteristically insidious when compared with the rapid fulminant course associated with mesenteric arterial occlusion. Symptoms generally involve a long prodrome of nonspecific abdominal discomfort for days to weeks.⁵ In addition, despite progressive pain and associated nausea and vomiting, patients often continue to eat and therefore may not present early for medical evaluation. When these patients are examined the pain reported is usually out of proportion to the physical findings elicited. The pain is usually diffuse, constant, with a dull tightening quality, and is often associated with a decreased intensity when oral intake is reduced. Stools usually contain traces of blood, and bloody diarrhea is not uncommon.

Laboratory findings are mostly nonspecific and therefore nondiagnostic. Blood tests usually reveal leukocytosis and hemoconcentration, while electrolyte studies usually remain within normal limits except for a mild metabolic acidosis and elevated lactic acid dehydrogenase (LDH).^{4,6} Coagulation studies may identify increased prothrombin, increased partial thromboplastin time, or thrombocytosis.

Plain radiography or contrast studies are also nonspecific and nondiagnostic. These studies may reveal subtle evidence of intestinal obstruction. Oral contrast studies may, on the other hand, occasionally identify more specific evidence of MVT by demonstrating thickened intestinal wall and valvulae conniventes, separation of intestinal loops secondary to mesenteric thickening, and thumbprinting.⁴

More sensitive imaging studies include CT scan of the abdomen and pelvis with IV contrast. This study is more specific when it demonstrates an increase in size of the mesenteric vein, a low-density intraluminal lesion, or a mesenteric vein with a hyperdense peripheral ring after IV contrast. Angiography has the best diagnostic potential. Typical findings on angiograms include a border blush, superior mesenteric artery spasm, a prolonged arterial phase, and a delayed or absent venous phase.

In most cases medical management is the first line of treatment. This involves fluid resuscita-

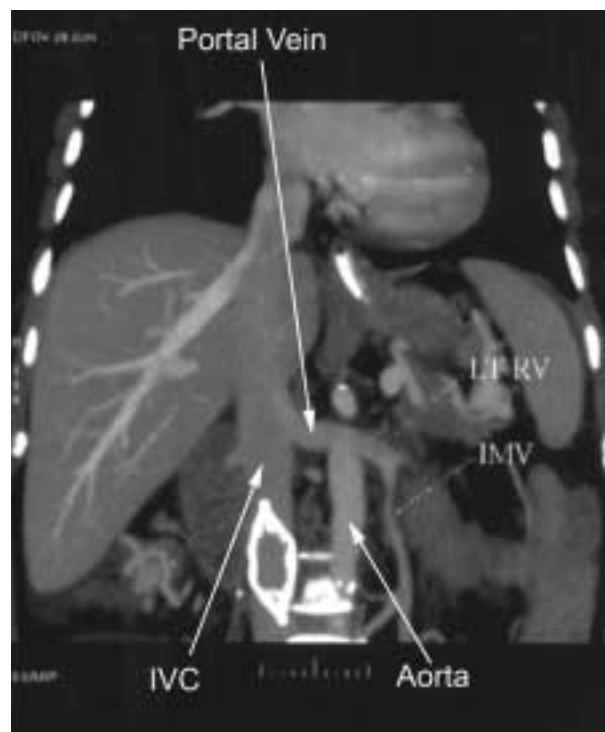


Figure 2. Reconstructed computed tomography scan of abdomen with labeled inferior mesenteric vein (IMV)-to-left renal vein (LT RV) anastomosis.

tion, anticoagulation, and thrombolysis. In addition, prophylactic antibiotics are indicated. Nonoperative management is feasible in established MVT when bowel infarction does not involve transmural necrosis and bowel perforation. Abdu et al,⁷ in a review of the literature from 1911 to 1984, revealed that the use of anticoagulants as an adjunct to bowel resection significantly decreased the mortality rate of MVT (22% vs 59%) and improved the survival of patients (77% vs 65%). In general, the presenting clinical features reported in the literature since 1990 that allow for nonoperative management have been found to represent approximately 36% of patients.⁸⁻¹⁰ However, surgery is indicated if patients demonstrate signs of peritonitis, deteriorate on medical management, or undergo lack of diagnosis with worsening abdominal signs and symptoms. Most studies recommend that all grossly ischemic or questionable viable bowel be resected with primary anastomosis followed by immediate postoperative anticoagulation. Postoperative anticoagulation is recommended because it reduces the incidence of recurrent MVT from 30–40% to 3–5%.¹¹ Recurrent MVT usually occurs 7–10 days postoperatively, although it has been known to also occur months later. Therefore, the optimal duration of anticoagulation therapy is not well defined (ranges from 3 months to indefinitely).

In our patient bowel resection was not a viable option as there was no gross evidence of bowel infarction. Overall, the literature for SMV thrombosis has shown poor results with either operative or nonoperative management, but in this instance, with failure of medical management the patient underwent mesenteric-to-systemic venous bypass, in order to decompress the IMV and mesenteric venous collateral outflow tracts. The IMV-to-left renal vein bypass was accomplished with a single anastomosis and minimal dissection. It is suggested that these operative features are important in maintaining the patency of venous reconstruction. The combination of antiplatelet agents is likely to be of additional benefit. This procedure provided the patient with symptomatic relief in the postoperative period, allowing her to progressively resume a normal dietary intake.

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