SPONTANEOUS PORTOMESENTERIC VENOUS THROMBOSIS WITH BOWEL ISCHEMIA: A RARE CASE REPORT

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ABSTRACT

Although uncommon and often asymptomatic, portal venous thrombosis can have catastrophic consequences for the individual it affects, particularly when the process propagates to involve the superior mesenteric vein. Familiarity with the condition's pathogenesis and presentation however permits early diagnosis and allows aggressive conservative management to achieve a successful outcome. Here we describe the successful outcome of such management for a 45-year-old male patient who developed this condition spontaneously.

Key words - portomesenteric vein thrombosis, bowel ischemia

Case Presentation

A 45-year-old male patient presented as an emergency with severe generalized abdominal pain of sudden onset that radiated straight through to his back with diffuse abdominal tenderness on examination. He also reported several episodes of vomiting but no particular aggravating or relieving factors. He had history of acute coronary syndrome 2 years back for which he is on irregular medication. He admitted moderate alcohol consumption since
7 years. Hematological and biochemical profiling revealed neutrophilic leukocytosis with total wbc count of 15700 with 88% neutrophils but normal amylase and troponin levels. USG abdomen was suggestive of mild hepatomegaly with grade I fatty changes with hypoechoic lesions. A computerized tomogram of his abdomen demonstrated thrombosis in the superior mesenteric vein and portal vein with extension into the splenic vein. Non-enhancing long segment wall thickening involving the distal ileal loops with adjacent mesenteric fat stranding and minimal fluid collection, likely to represent small bowel ischemia. Mild ascitis. Multiple collaterals in the region of portahepatis, pancreatic and perisplenic regions likely to suggest early changes of cavernous transformation of portal vein. Diffuse fatty changes in the liver with caudate lobe hypertrophy. The patient was immediately commenced on full therapeutic anticoagulation (intravenous unfractionated heparin) and emergency bowel resection has been done patient recovered well after the surgery.

**Computerized tomographic imaging of patient's abdomen at presentation.**

![Figure 1 and 2 Thrombosis in the superior mesenteric vein and portal vein with extension into the splenic vein.](image1)

![Figure 3 and 4. Non-enhancing long segment wall thickening involving the distal ileal loops with adjacent mesenteric fat stranding and minimal fluid collection, likely to represent small bowel ischemia](image2)
Figure 5
Multiple collaterals in the region of portahepatis, pancreatic and perisplenic regions likely to suggest early changes of cavernous transformation of portal vein

Discussion

Portomesenteric ischemia accounts for approximately 5–15% of all cases of mesenteric ischemia and has been associated with mortality rates of 20–50% [1, 2]. Recent thrombosis of the portal vein may be asymptomatic or else may be associated with a systemic inflammatory syndrome with or without signs of intestinal ischemia. Old thrombosis of the portal vein is usually only recognizable on imaging by the demonstration of its cavernous transformation. Such a "portal cavernoma" refers to venous collateralization around the portal vein which develops in response to occlusion of the extrahepatic portal system and which partially maintains hepatopedal blood flow [3]. It has been previously shown that the interval between obstruction of the portal vein and the cavernous transformation is approximately 5 weeks [4]. These multiple, millimetric veins tends to occur predominantly around the suprapancreatic part of the common bile duct and may result in cholestasis due to the resulting angulation and even stenosis of the duct [5]. The main complication however of chronic portal vein thrombosis is gastrointestinal bleeding due to rupture of esophageal varices or portal hypertensive gastropathy. Although less frequent, intestinal necrosis may occur due to thrombotic extension that can result in obstruction of the superior mesenteric vein. The cause of thrombosis may be either a general prothrombotic state (e.g. myeloproliferative syndrome, antiphospholipid syndrome, antithrombin deficiency, protein C or S deficiencies, or factor gene mutations) or intraabdominal inflammation (including pancreatitis and inflammatory bowel disease). Furthermore, portal vein occlusion has been reported to occur after abdominal surgery (in particular splenectomy)[6].

Although surgery may be required when venous gangrenes of the intestine occurs, early diagnosis may allow successful conservative management with anticoagulation. Although thrombolysis has been recently proposed [7], heparinization remains the first-line treatment. For this, unfractionated heparin infusion is preferable to fractionated subtypes because of its shorter half-life and ease of reversibility. Upper gastrointestinal bleeding risk can be prevented by beta-adrenergic blockade, endoscopic ligation, or endoscopic sclerosis of varices. Because the risk of disease progression persists early after initiation of therapy, a low threshold for operative exploration is required during conservative management. In the long-term, permanent anticoagulant treatment is recommended when a permanent prothrombotic state exists, even in patients who have a history of gastrointestinal bleeding.
References