Acute Ischemic Bowel Disease due to Portomesenteric Venous Thrombosis: One Case Report

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Diffuse portomesenteric venous thrombosis (PMVT) is an unusual form of deep vein thrombosis. Intestinal gangrenous change induced by PMVT is an extremely rare entity but potentially lethal condition. We reported a 56-year-old male without known risk factors who had diffuse intestinal ischemia secondary to right hepatic vein thrombosis (RHVT), portal vein thrombosis (PVT), and superior mesenteric vein thrombosis (SMVT). The initial biochemistry studies were within normal limits. Contrast enhanced computed tomography confirmed the impression. The patient was successful treated by surgical intervention and postoperative anticoagulation therapy. Diagnosis for PMVT is often delayed and not made until diffuse peritonitis present. Emergent physician should keep alertness in daily working.

Key words: ischemic bowel disease, venous thrombosis

Introduction

Ischemic bowel disease occurs due to insufficient supply of oxygenated blood flow to the intestines. It is an uncommon but complicated disorder which can range from mild symptoms to severe morbidities or even mortality if not diagnose early and treat adequately. There are several possible etiologies of ischemic bowel disease, but secondary to diffuse portomesenteric venous thrombosis (PMVT) is extremely rare.

Portomesenteric venous thrombosis is a very unusual form of deep vein thrombosis, which occurs mainly in patients with predisposing factors or occasionally after varied laparoscopic procedures¹. This probable life-threatening event has a wide variable clinical manifestations, presenting with acute, subacute or chronic course, and diffuse abdominal pain was reported as the most common symptoms (83%-90%)²,³. Delay diagnosis always occurred because the symptoms are usually nonspecific until peritonitis happened, or in part, low degree of suspicion for clinicians. We reported a patient without previous known risk factors, who had diffuse intestinal ischemia induced by right hepatic vein thrombosis (RHVT), portal vein thrombosis (PVT), and superior mesenteric vein thrombosis (SMVT).

Case Report

A 56-year-old previous healthy male visited our emergency department for epigastralgia without radiation pain since 2 days ago, and vomiting several times accompanied with cold sweating before seeking medical advice. His vital signs were stable and physical examination revealed clear breath sounds, regular heart beats without
obvious murmurs, mild abdominal distension with hypoactive bowel sounds and no peritoneal signs. There were neither signs of deep vein thrombosis (extremities pain, redness and swelling) nor recent abdominal blunt traumatic injuries. EKG and KUB showed normal sinus rhythm and bowel gases with distended loops, respectively. Initially white blood cell counts and serum lipase level were within the normal limits. Panendoscopy disclosed superficial gastritis. However, epigastralgia with left upper quadrant abdominal muscle guarding happened to him several hours later. Immediate contrast enhanced computed tomography showed diffuse thrombosis of right hepatic vein, portal vein and superior mesenteric vein (Fig. 1a, 1b, 1c) with thickening small bowel loops in left abdomen. Laparoscopy was performed and we found a congestive intestinal segment 60cm below Treitz ligament with gangrenous change. The procedure was then converted to laparotomy with segmental small intestine resection and end-to-end anastomosis. Pathology reported ischemic hemorrhagic necrosis accompanied with the development of mural thrombus. The patient was successful treated by surgical intervention and postoperative anticoagulation therapy (Heparin, Warfarin).

Fig. 1 Contrast enhanced CT scan of the abdomen shows the thrombus (circle) over right hepatic vein (Fig. 1a), portal vein (Fig. 1b), and superior mesenteric vein (Fig. 1c)
Discussion

Ischemic bowel disease caused by venous thrombosis instead of arterial origin is rather uncommon, and moreover, intestinal gangrenous change secondary to portomesenteric venous thrombosis accounts for less then 5% to 15% of all mesenteric ischemic events.(4) The most common clinical presentation was diffuse abdominal pain out of proportion to physical examination (83%-90%), followed by nausea (54%) and vomiting (42%-77%), with the signs of peritonitis (32%), leukocytosis (49%), elevated serum lactate (28%), fever (25%), and sometimes upper (28%) or lower gastrointestinal bleeding (23%).(2,3) Immediate treatment of anticoagulation for at least six months, or even lifetime therapy are necessary in those with thrombotic disorders or undefined sources. In the retrospective study of Condat B et al, anticoagulant therapy decreased recurrent rate or extension of thrombi by two-thirds, and complete or partial recanalization in 90% of patients in acute PVT under nonsurgical management.(5) However, surgical intervention is usually mandatory to patients with peritonitis or intestinal perforation.

Portal vein thrombosis is theoretically not quite unique in Chinese because of hepatocellular carcinoma and liver cirrhosis. Amitrano L and Janssen HLA et al. found the underlying liver diseases account for 11.2%-21.5% of mesenteric venous thrombosis(6,7), but they infrequently induced intestinal ischemia or infarction (12.6% of PVT patients) (6). In Chinese, diffuse portomesenteric venous thrombosis that do not related to liver diseases is a more rare entity. However, there was no past history of liver diseases or other evidence of liver function impairment in our case.

Delay diagnosis for venous thrombosis took place frequently, especially when there are no known predisposing factors such as hypercoagulable state, portal hypertension, malignancy, pancreatitis, splenectomy and other abdominal inflammation or infectious processes. Among these factors, hypercoagulable disorders had been reported as high as approximately 70% of patients(8), and antithrombin III deficiency, protein C and protein S deficiencies, factor V Leiden deficiency, prothrombin mutations, and hyperhomocysteinemia had also been explored respectively. In our case, there were no existing risk factors or specific underlying diseases can be identified before this episode. Further survey for coagulation status of the patient displayed normal serum level of anti-phospholipid antibody (0.36 RU/ml, negative) and factor V (77%) but decreased serum level of protein C (58%), protein S (19.2%) and anti-thrombin III (76%). Thus, the protein C, protein S and anti-thrombin III deficiency implied that the hypercoagulable condition might be responsible for constructing his diffuse portomesenteric venous thrombosis.

In addition, the general clinical presentation in venous thrombosis is insidious symptoms for weeks to months, or so called intestinal angina that do not conform to the victim. Only a computed tomography abdominal scan permitted the diagnosis of right hepatic vein, portal vein, and superior mesenteric venous thrombosis. In short, early diagnosis, immediate and long term anticoagulation therapy or even adequate surgical intervention are key points to improve the clinical prognosis(9-11). Otherwise, an investigation of intrinsic anticoagulant deficiencies is also warranted since these deficiencies are mostly inherited in an autosomal dominant fashion(12). Emergent physicians should always keep alertness and awareness in daily working.

References

廣泛性肝門腸靜脈栓塞導致腸梗塞性壞死之病例報告

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廣泛性的肝門靜脈與腸靜脈靜脈栓塞是一種罕見的深部靜脈栓塞性疾病。而由此病因所導致的腸梗塞性壞死更是罕見。本文報告一位56歲台灣男性，無特殊過去病史與已知的危險因子，遭遇廣泛性的右肝靜脈、肝門靜脈，與上腸靜脈靜脈之栓塞而導致之缺血性腸梗塞性壞死。病患初期身體理學檢查與生化檢查並無特殊異常。最後經由電腦診斷確認，並以手術切開壞死之腸子以及術後的抗凝藥物成功治療。此類靜脈栓塞性腸梗塞性壞死症狀常不典型且不易診斷，臨床醫師必須有高度的警覺心。

關鍵詞：缺血性腸梗塞性，肝門靜脈栓塞性