Splenic vein thrombosis in cirrhosis of the liver: A rare case

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ABSTRACT

Splenic Vein Thrombosis (SpVT) in a young patient with non-hepatitis B and C liver cirrhosis is an infrequent case generating hemorrhagic manifestations. Herein we report a 28-year-old man presenting with hematemesis, melena, and features of liver cirrhosis. Hematemesis, melena, and ascites resolve following a conservative treatment. Abdominal ultrasound confirmed portal hypertension. Serial endoscopy on day 14, 17 and 1-month evaluation showed grade II-III esophageal varices and severe hypertensive portal gastropathy. Abdominal CT scan with contrast within 1 week after discharge revealed thrombus along ± 5.8 cm, splenomegaly with dilated splenic vein, dilatation and tortuosity of the left gastric vein and visualized distal esophageal vein. Liver biopsy 2 months after hospitalization showed hepatocytes with extensive hydropic degeneration with fibrosis (F3).

1. Introduction

Splenic Vein Thrombosis (SpVT) is a prevalent case in male patients during their fifth decade of life. Patients commonly complain of abdominal pain, gastrointestinal bleeding, and spleen enlargement [1]. Blood analysis may show thrombocytopenia or panacytopenia [2]. SpVT commonly does not coincide liver cirrhosis and requires advanced imaging modalities, such as venous-phase celiac angiography [3,4].

SpVT results in an elevated localized sinistral portal pressure, also known as left portal hypertension. Most patients present with left portal hypertension with no significant symptoms and normal liver function, but still, gastrointestinal bleeding secondary to esophageal or gastric varices commonly arises. Nonetheless, many patients with peripheral artery SpVT other than gastric varices rarely bleed. Because patients without esophageal varices are asymptomatic, treatment is considered not obligatory along with tight monitoring [3]. Referring to SCARE 2020 Guidelines [5], we report a rare case of a non-hepatitis B and C liver cirrhosis patient developing SpVT.

2. Case illustration

A male patient, aged 28, was admitted to of Dr. Soetomo General Hospital with hematemesis and melena. The patient had ascites within the last 6 months accompanied by intermittent abdominal pain. He ever experienced such symptoms 6 years ago. The patient underwent treatment for a week at Sakinah Hospital once and received 3 bags of packed red cell (PRCs), then he was discharged. Three years later, patient was hospitalized twice at Dian Husada Hospital for 6 bags of PRCs transfusion. Abdominal ultrasound revealed liver disease. Patient underwent conservative treatment with low-salt H2 diet 2100 kcal/day, intravenous furosemide 20 mg BID, lansoprazole pump infusion 6 mg/hour, intravenous cefotaxime 1g TID, Lactulose syrup 30 ml QID, Sucralfate syrup 30 ml per oral TID, Spironolactone 100 mg per oral BID, intravenous tranexamic 500 mg TID, intravenous phenytoxamide 10 mg every TID, intravenous octreotide 50 g/hour, and PRC transfusion of PRC 1 bag/day until Hb > 8gr/dL. Continuous laboratory marker monitoring was done on day 3, 8 and 12 of hospitalization (See Table 1).

Abdominal ultrasound on day 7 detected liver cirrhosis with portal hypertension (See Fig. 1A). Esophagogastroduodenoscopy (EGD)
Portal hypertension due to splenic vein thrombosis (SpVT) can induce massive gastrointestinal bleeding from the esophagus or gastric varices and develop hypertensive gastropathy. Acute and chronic pancreatitis, pancreatic pseudocyst, and pancreatic adenocarcinoma accompanied 7%–20% of patients with SpVT [1]. Pancreatitis and perivenuous inflammation are the most common causes of SpVT [3]. Although more than 45% of SpVT patients with chronic pancreatitis have been reported, many of them are asymptomatic [2].

SpVT can induce local hypertension of the splenic vein and create collateral from the spleen to the fundus. The blood therefore returns to the main portal system via the coronary veins. In some cases, gastric varices are often not associated with esophageal varices except for collateral at the gastroesophageal junction, which is the most common site of bleeding. In other cases, spontaneous bleeding is uncommon. Patients with history of previous pancreatitis are suspicious for having SpVT due to enlarged retroperitoneal lymph nodes located near the splenic artery, above the splenic vein. Other risk factors are history of gastrointestinal bleeding, splenomegaly without portal venous hypertension, cirrhosis or haematological disease and gastric varices [3].

Clinicians shall examine the primary cause of hypercoagulation in any SpVT patient with splenomegaly. Hypercoagulation, both hereditary and acquired, predisposes the patient to arterial or venous thrombosis in brain, extremity, and intra-abdomen, with venous thromboembolism (VTE) as the most common manifestation. Other disorders caused by hypercoagulation include myeloproliferative syndrome, hyperhomosystememia syndrome, and antiphospholipid antibodies (APAs).

Splenic vein obstruction can cause retroperitoneal, pancreatic, and perisplenic lymphadenopathy that leads to vein compression, obstruction, and thrombosis [6].

Ascites is a pathological condition due to accumulation of fluid in the intraperitoneal cavity. Ascites is still the leading complication of cirrhosis within first 10 years, detected in roughly 60% of patients with compensated cirrhosis [7]. Portal hypertension still highly underlies ascites in 75% of the patients despite other pathogenesis explained in established literature [8]. Portal hypertension is induced by increased resistance in the liver, connective tissue, regenerative nodules, vasoconstriction, and thrombus. This condition results in the formation of collateral vein around the portal vein, in the skin, esophagus and stomach. In addition, portal hypertension will also cause splanchnic vein vasodilatation which manifesting splenomegaly. Moreover, in the central circulation system, portal hypertension causes systemic vasodilatation resulting in effective hypovolemia and activates renin-angiotensin-aldosterone system (RAAS) and vasopressin. This leads to renal vasoconstriction and sodium and water retention. Retention of sodium and water results in the development of refractory ascites. Additionally, this retention also results in increased cardiac output (CO), thereby elevating flow to the portal and exacerbate the existing portal hypertension [9,10]. SpVT causes localized left venous hypertension returning splenic venous flow to low-pressure collateral vein whereby preventing blood circulating from the spleen. Flow through the short gastric and/or gastro-epiploic vein dilates the sub-mucosal venous system of the stomach and esophagus. Both will form a thin gastric wall and esophageal varices [6]. Since coronary vein supplies the portal system, gastric varices without esophageal varices highly suggests splenic vein occlusion.

SpVT is diagnosed based on abdominal CT, abdominal angiography, MRI, or ultrasound to distinguish with the differential diagnoses, which are Budd-Chiari and Banti syndrome [11]. Furthermore, anticoagulation issue in isolated SpVT is still an unsolved problem. In acute or subacute mesenteric venous thrombosis, heparinization should be done to increase survival and prevent recurrent thrombosis. Anticoagulants can still be administered for gastrointestinal bleeding as long as the benefit of preventing infarction outweighs the risk of bleeding [1]. Immediate splenectomy is strongly recommended in patients with bleeding esophageal varices to prevent esophageal varices from bleeding massively. Besides, no other available treatment is able to control bleeding [12]. Ultimately, splenectomy is the treatment of choice and able to effectively remove collateral outflow [13].
Fig. 1. A. Abdominal ultrasound showing liver cirrhosis with hypertension portal; B: serial esofagastroduodenoscopy (EGD) on day 14, day 17, and one month after hospitalization; C: Abdominal CT with contrast showed thrombus in splenic vein along with splenomegaly.
Ethical approval

The approval has been given by the patient.

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Author contributions

Arina Mana Sikana: case illustration, interpretation, manuscript arrangement, final editing.
Husin Thamrin: case illustration, supervision, final editing.

Trial registry number

1. Name of the registry:
2. Unique Identifying number or registration ID:
3. Hyperlink to your specific registration (must be publicly accessible and will be checked):

Guarantor

Both authors are the guarantor of this work.

Consent

The patient has signed an informed consent.

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Declaration of competing interest

All authors declare no conflict of interest.

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Appendix A. Supplementary data

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