Portal and Mesenteric Venous Calcification in Patients with Advanced Cirrhosis: Two Case Reports and Literature

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Case report

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Abstract

The incidence of portal and mesenteric venous calcifications in patients with cirrhosis has rarely been reported. It is also very difficult to determine the vascular lesions in preoperative imaging examination. The liver cirrhosis patients associated with portal venous calcification have high postoperative complications and mortality, but poor prognosis. We present the case of two patients (45-year-old male, case 1; 50-year-old male, case 2). Both patients were admitted to the hospital with liver cirrhosis and portal hypertension associated symptoms. The cases and review of published reports suggest that, calcification in the portal vein system is extremely rare, and always occurs in patients with long-standing liver cirrhosis with portal hypertension gastroesophageal varices and splenomegaly. The presence of portal vein calcification on CT may be a sign of portal vein thrombosis, which may result in a difficult transplantation, and poor prognosis.

Introduction

The incidence of portal and mesenteric venous calcifications in patients with cirrhosis has rarely been reported. It is also very difficult to determine the vascular lesions in preoperative imaging examination. The liver cirrhosis patients associated with portal venous calcification have high postoperative complications and mortality, but poor prognosis. Here, we reported two recent cases with evidence of calcifications in the portal venous system confirmed by computer tomography.

Case Report

Case 1

A 45-years-old man was admitted for fever of 3 days duration and was diagnosed with acute cholangitis, biliary cirrhosis. He has congenital cystic dilatation of the intrahepatic bile duct but has not been examined and treated. 30 years ago, he underwent splenectomy and venous devascularization due to upper gastrointestinal bleeding caused by portal hypertension-related liver cirrhosis. In the past 20 years, he had repeated gastrointestinal bleedings and underwent endoscopic varicose ligation combined with embolization for hemostasis. In the past 1 year, the patient has repeatedly had fever with chills, abdominal pain, diarrhea, jaundice, and other symptoms. He denied smoking and alcohol abuse. Physical examination was essentially negative except for body temperature as high as 38.7°C. Lab-examination showed: (white blood cell [WBC] = 15.7×10^9 /L, NE% = 77.8% hemoglobin [HB] = 122g/L, platelet [PLT] = 207×10^6 /L; albumin [ALB] = 27.4g/L, alanine aminotransferase [ALT] = 112U/L, aspartate aminotransferase [AST] = 80U/L, total bilirubin [TB] 30.3umol/L, direct bilirubin [DB] = 10.2umol/L; blood ammonia = 106umol/L; prothrombin time [PT] = 17.4s, prothrombin time activity [PTA] = 76.2%, international normalized ratio [INR] = 1.16, activated partial thromboplastin time [APTT] = 30.0s; CA19-9 150.20U/L, CA12-5 174.50U/L, hepatitis related tests are negative. Abdominal enhanced CT indicated multiple dilation of intrahepatic bile duct, which is consistent with the manifestations of Caroli disease (Fig. 1A, B), and calcifications in the portal and superior mesenteric were detected (Fig. 1C, D), liver

cirrhosis with portal hypertension. His liver function was Child-Pugh A grade and MELD 6 points. Based on comprehensive considerations, the patient met the indications for liver transplantation, and was reviewed and approved by the hospital ethics committee to undergo the allogeneic modified piggyback liver transplantation on January 23, 2021. The surgical procedure was complicated and took 25 hours, including 7 hours in the anhepatic phase. The cold ischemia-time of the donor liver was 16.5 hours. After a difficult hepatectomy, the portal vein was found to be calcified and thrombosed. Attempted at direct intraluminal dissection and thrombectomy failed to reestablished patency. Finally, the portal vein stent was placed, and portal flow was reestablished. Intraoperative blood loss is estimated to be 16000ml, 6500ml autologous blood transfusion, 3600ml suspended red blood cells, and 2400ml fresh frozen plasma. Postoperative ultrasound of the transplanted liver vessels showed that the inner diameter of the portal vein was 1.0cm and the flow rate was 18.0cm/s. With long operation time, excessive bleeding, long cold ischemia time of the donor liver, and poor portal vein condition, the patient died of transplanted liver failure, kidney failure, and heart failure 2 days after surgery.

Case 2

A 50-years-old man was diagnosed as having liver cirrhosis twenty years prior to his present admission. In the past 10 years, he had repeated gastrointestinal bleedings and underwent endoscopic varicose ligation. The patient developed refractory ascites 2 years ago and hepatic encephalopathy occurred 1 year ago. On admission, the patient was complained of refractory ascites, and a physical examination revealed remarkable abdominal distension and positive shifting dullness. He had a long-term history of heavy alcohol abuse. Lab-examination showed abnormal liver function and pertinent data were as follows: WBC $3.05 \times 10^9/L$, HB 113g/L(mild anemia), PLT $139 \times 10^9/L$; ALB 28g/L(hypoproteinemia), AST 137U/L, ALT 56U/L, TB 68.8umol/L, DB 43.7umol/L; PT 14.6s, INR 1.26, APTT 25.4s; blood ammonia: 112umol/L (hyperammonemia); Tumor marker: CA19-9 248.78U/L, CA12-5 412.50U/L; Abdominal imaging examination revealed liver cirrhosis with portal hypertension (Fig. 2A), the presence of calcifications of full-length in the portal vein and superior mesenteric vein (Fig. 2B, C, D). His liver function was Child-Pugh C grade and MELD 24 points, was in end-stage liver disease. Based on comprehensive considerations, the patient met the indications for liver transplantation, and was reviewed and approved by the hospital ethics committee to undergo the allogeneic modified piggyback liver transplantation, but the patient and his family refused to be treated by surgery and decided to be discharged.

Discussion

Calcification in the portal vein system is extremely rare, and always occurs in patients with long-standing liver cirrhosis with portal hypertension gastroesophageal varices and splenomegaly¹. For revealing portal vein and its tributaries, abdominal enhanced CT could improve the positive rate, and is the most sensitive examination, and showed the location and direction of portal venous calcification^{2,3}. The distinctive radiographic feature of portal venous calcification is the presence of radiodensity which correspond to the course of the vein⁴. Minimal calcification may be frequently neglected on plain film radiography and pathological examination.

Calcium could be deposited either in thrombus or as in the vessel wall. The mechanical stress may result in sclerosis and calcification with the thickened and media of the vein. Since 1943, Moberg⁵ reported the first case of portal vein calcification, less than 50 documented cases have been described in the English-language literature. The calcified lesions occurred in the portal vein in 100% of patients, the splenic vein in 62%, the superior mesenteric vein in 33%, and the inferior mesenteric vein in 0⁶. Repeated thrombus formation and recanalization may be the main etiologic factor in the formation of calcification. The predisposing factors for the deposition of calcified thrombus in the portal vein well, included visceral infections affected by pancreatitis and cholangitis, history of abdominal surgery, malignant diseases, and hematological abnormalities⁷. It was found by Verma⁴ a high operative mortality associated with calcifications in portal venous system in patients during liver transplantation because of preoperatively undiagnosed thrombosis of the portal venous system. And the presence of portal vein calcification on CT may be a sign of portal vein thrombosis, which may result in a difficult transplantation. The calcification of portal venous system with associated thrombosis is a significant finding and more attention should be devoted to detecting in patients undergoing liver transplantation. Identification of patients at high risk may provide information for prospective planning, rational distribution of organs, and a safer operation.

Declarations

The experimental protocol was established, according to the ethical guidelines of the Helsinki Declaration and was approved by the Human Ethics Committee of Beijing Chaoyang Hospital. The authors do not have any possible conflicts of interest.

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B Pan: literature review, drafting, and editing. SC Lyu: literature review, critical revision of the manuscript. Q He: critical revision of the manuscript.

Author contributions

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Figures

Figure 1

The contrast-enhanced CT of the abdomen of patient 1. A and B, the enhanced CT of the abdomen suggests multiple dilation of the intrahepatic bile ducts because of Caroli's Disease, and liver cirrhosis and portal hypertension. C and D showed multiple calcifications in the running area of the portal vein and superior mesenteric vein.

Figure 2

The Abdominal vascular CT and magnetic resonance cholangiopancreatography (MRCP) of patient 2. A, the abdominal vascular CT showed liver cirrhosis, ascites, portal hypertension with formation of collateral circulation. B, C and D, the magnetic resonance cholangiopancreatography showed full length of portal vein and superior mesenteric vein calcification.