

Duodenal Erosion and Obstruction Caused by a Mesocaval Prosthesis

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ABSTRACT

In the past, vascular prostheses have been developed for mesocaval shunt surgeries in the treatment of portal hypertension. Penetration of prosthesis into the duodenum is a rare complication. We report the case of a 65-year-old man who presented with duodenal erosion and obstruction caused by the prosthesis, 22 years after its insertion. This is the ninth reported case of such events, but it demonstrates the longest interval to date between insertion and complication.

INTRODUCTION

In the past, vascular prostheses were used for mesocaval shunts in patients with portal hypertension. This relatively complicated and invasive surgery has high incidence of chylous ascites. With the development of interventional radiology, it has been gradually replaced by transjugular intrahepatic portosystemic shunt (TIPS) since the 1990s. There were quite a few patients who underwent surgery for mesocaval prostheses.¹ Some medical centers still perform mesocaval shunt surgeries with vascular prosthesis, although literature has rarely reported any in the past 2 decades. Penetration of prosthesis into the duodenum is a rare but serious complication.

CASE REPORT

A 65-year-old man was diagnosed as having alcoholic cirrhosis 22 years ago. He survived 3 prior variceal hemorrhages, which were treated with hemostatic drugs, variceal sclerotherapy, and ligation, respectively. However, during the fourth variceal hemorrhage, he had to receive splenectomy, periesophagogastric devascularization, and mesocaval shunt surgery with a C-shaped vascular prosthesis, which was anastomosed with superior mesenteric vein and inferior vena cava.

The patient remained stable until 1 year ago when he vomited gastric contents and had mild abdominal pain without hematemesis. Endoscopy showed no signs of variceal bleeding and no lesions in the duodenum. Abdominal computed tomography (CT) showed the C-shaped prosthesis of mesocaval shunt with mild thrombosis, but the blood flow was preserved (Figure 1). After antiemetic treatment and anticoagulant therapy, the clinical symptoms improved.

At the current instance of illness, he was hospitalized after a 6-month history of abdominal distension and pain, occurring mostly after eating. Overall laboratory testing showed that his liver function score was Child-Pugh class A. He had no ascites or hepatic encephalopathy. Admission laboratory values were significant with a hemoglobin value of 116 g/L, platelet count of $224.7 \times 10^9/L$, white blood cells of $6.048 \times 10^9/L$, albumin of 29.46 g/L, total bilirubin of 40.33 $\mu\text{mol/L}$, and prothrombin time of 14.38 seconds. The above values had not changed much when compared with those seen 1 year ago, except for albumin which changed from 33.32 to 29.46 g/L. Endoscopy showed that the vascular prosthesis had eroded into the descending portion of the duodenum and below the duodenal papillae (Figure 2). Abdominal CT was consistent with cirrhosis, portal hypertension, and comprehensive thrombosis of

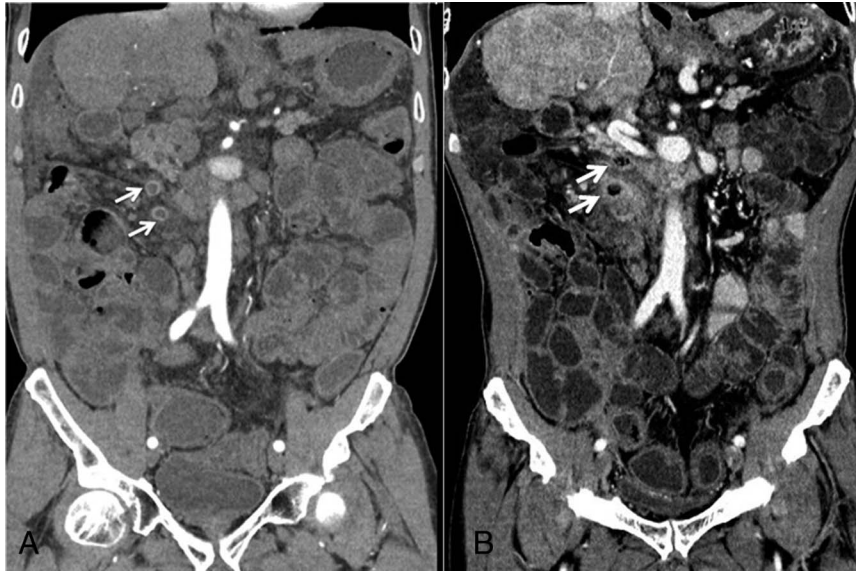


Figure 1. (A) Abdominal computed tomography (CT) 1 year prior showing the prosthesis with blood flow (arrows). (B) Abdominal CT at presentation showing the occluded prosthesis with thrombosis (arrows), and the inflammatory mass around the prosthesis.

the portal vein. A portion of the prosthesis was located within duodenum (between the descending and horizontal portion of the duodenum). It also showed an inflammatory mass with air around the prosthesis, and no blood flow in the thrombotic prosthesis, which presented a sharp contrast with the CT image taken 1 year ago (Figure 1). Gastrointestinal radiography showed that the obstructive site was located between the descending and horizontal portion of the duodenum. The duodenal obstruction was not complete because the contrast solution could reach the distal intestines. The patient could tolerate only a liquid diet and asked for surgical intervention. Considering that the removal of the prosthesis would result in

a high risk of hemorrhage, we performed a subtotal gastrectomy with a Billroth-II anastomosis to relieve the obstruction. The patient slowly recovered and successfully tolerated general food diet without abdominal distension or pain. He was discharged 11 days after surgery. He then received conservative therapy for the thrombosis in the portal veins. Regular follow-up revealed no adverse events.

DISCUSSION

Surgical interventions such as devascularization, shunting, and transplantation have been used as salvage therapies when endoscopic interventions failed to control variceal bleeding. Surgical shunts with vascular prostheses had been widely used before 1990s. After the emergence of TIPS, this method of shunting is no longer routinely used. However, surgical shunts still have a role for selected patients with refractory variceal bleeding in some medical centers and there are still a number of patients who have undergone interposition mesocaval shunts.¹⁻³ Chylous ascites is considered the most common complication after mesocaval shunt. Even though duodenal erosion and obstruction caused by the prosthesis is very rare, it is important to recognize this complication early and design appropriate therapy.

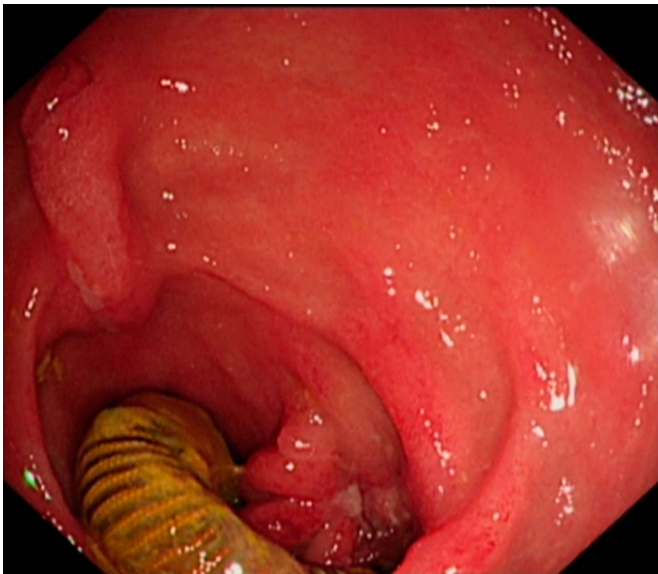


Figure 2. Endoscopy showing the prosthesis in the descending portion of duodenum.

The prosthesis in the mesocaval shunt was mainly composed of Dacron or expanded polytetrafluoroethylene. There are 2 types of mesocaval prostheses: H-shaped and C-shaped.^{4,5} The C-shaped mesocaval prosthesis, first described by Cameron et al., was used in the treatment of portal hypertension secondary to liver cirrhosis or Budd Chiari Syndrome.^{5,6} It has a higher patency than the H-shaped because a more cephalad portion of superior mesenteric vein is anastomosed. However, the complication of eroding into the duodenum may be more

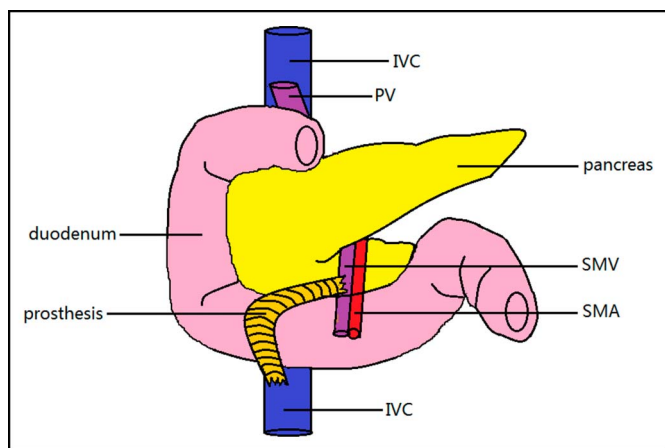


Figure 3. Diagram showing the C-shaped prosthesis of mesocaval shunt between IVC and SMA. IVC, inferior vena cava; PV, portal vein; SMA, superior mesenteric artery; SMV, superior mesenteric vein.

common in patients with C-shaped prostheses, which swung tightly over the descending portion or horizontal portion of the duodenum (Figure 3). The chronic erosion by the prosthesis may eventually result in perforation of the duodenum which can be detected by endoscopy and CT. In addition, the old thrombus of the prosthesis may exacerbate the duodenal erosion. Our patient had no severe symptoms of duodenal perforation, probably because the surrounding inflammatory mass was tightly wrapped around the leakage.

To our knowledge, a total of 8 patients with duodenal erosion caused by mesocaval prostheses have been reported.⁷⁻¹³ The 22-year time frame in the current case is the longest interval after prosthetic insertion to be reported to date. The reason why duodenal erosion occurred after so many years is not clear; it is probably because of the gradual thrombosis and eventual occlusion of the prosthesis. Endoscopic findings usually provide direct evidence for diagnosis. Most of the previous cases resulted in sepsis because of an infected prosthesis. In such cases, prosthesis removal was recommended, but the repair of the veins and duodenum was highly difficult and risky. In the current case, the patient did not have sepsis. Partial duodenal obstruction led to the initial symptoms of the current patient, so we performed a subtotal gastrectomy with gastrojejunostomy without duodenal resection or removal of the prosthesis. In addition, the bypass of food can avoid enlargement of the duodenal erosion. Excellent short-term outcomes were obtained after operation. However, strict follow-up is still needed.

This case showed that the prosthesis of mesocaval shunt for portal hypertension may erode into the duodenum and cause obstruction, even after many years. Endoscopy examination and CT scan are useful approaches to detect this complication.

Subtotal gastrectomy with gastrojejunostomy can be safely performed in such cases.

DISCLOSURES

Author contributions: All authors contributed equally to the manuscript. J. Liu wrote the manuscript and designed tables and figures. S. Chen wrote and edited the manuscript. J. Xiang revised the manuscript. J. Peng wrote and revised the manuscript and is the article guarantor.

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Informed consent was obtained for this case report.

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