

Two cases of hemosuccus pancreaticus in which hemostasis was achieved by transcatheter arterial embolization

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Abstract

Hemosuccus pancreaticus is a rare complication of chronic pancreatitis. We report two cases of hemosuccus pancreaticus in which hemostasis was achieved by transcatheter arterial embolization (TAE). The first patient was a 47-year-old man with alcoholic chronic pancreatitis. He presented with upper abdominal pain and hematemesis. Upper GI endoscopy failed to detect the source of bleeding, but computed tomography (CT) showed a hypervascular area about 3cm in diameter in a pseudocyst at the pancreatic tail. Angiography revealed a pseudoaneurysm in the caudal pancreatic artery. Hematemesis was considered to be due to rupture of the pseudoaneurysm. TAE of the splenic artery was performed selectively, and this successfully stopped the bleeding. The second patient was a 52-year-old man with alcoholic chronic pancreatitis. He presented with hematemesis. Upper GI endoscopy detected bleeding from the papilla of Vater. CT showed hemorrhage in a pseudocyst at the pancreatic body. Angiography revealed angiogenesis around the pseudocyst. Hematemesis was considered to result from rupture of the pseudoaneurysm. TAE of the dorsal pancreatic artery and posterior superior pancreaticoduodenal artery was performed and hemostasis was achieved. We conclude that TAE is a minimally invasive and highly effective treatment for hemosuccus pancreaticus.

Key words Hemosuccus pancreaticus · Transcatheter arterial embolization (TAE) · Gastrointestinal bleeding

Introduction

Hemosuccus pancreaticus is gastrointestinal (GI) bleeding from the duodenal papilla through the pancreatic duct. It is usually complicated by chronic pancreatitis, but it is not easy to diagnose because of intermittent

gastrointestinal bleeding.¹⁻⁵ We report our recent experience with two cases of hemosuccus pancreaticus, one due to a pseudoaneurysm and the other due to remarkable angiogenesis around a pseudocyst, both with alcoholic chronic pancreatitis. In both cases transcatheter arterial embolization (TAE) was successful in achieving hemostasis without any major complications.

Cases

The first patient was a 47-year-old man with alcoholic chronic pancreatitis. He had consumed one-half bottle of whisky per day for the past 25 years. He had a past history of polymyositis, and he had been treated with steroids for 5 years (prednisolone sodium succinate, 12.5mg/day). He presented at a local hospital with epigastralgia, tarry stool, and hyperamylasemia. Computed tomography (CT) revealed peripancreatic fluid collection, and acute pancreatitis was diagnosed, but the source of bleeding was unclear. Six weeks later he complained of hematemesis, but upper GI endoscopy failed to detect the source of bleeding. However, CT revealed a large pseudoaneurysm at the pancreatic tail, and he was referred to our hospital. On admission, he had no abdominal symptoms, and there were no remarkable physical findings. Laboratory data were within normal limits. CT revealed a pseudoaneurysm about 3cm in diameter in a pseudocyst at the pancreatic tail and a pseudocyst without aneurysm at the pancreatic head (Fig. 1a). Angiography revealed a pseudoaneurysm at the caudal pancreatic artery (Fig. 1b). Hematemesis was considered to be due to rupture of the pseudoaneurysm, and TAE of the splenic artery was performed selectively with metallic coils (Fig. 1c). The only complication associated with TAE of the splenic artery was partial splenic infarction, and the gastrointestinal hemorrhage was successfully stopped. However, the patient complained of severe epigastralgia after meals and

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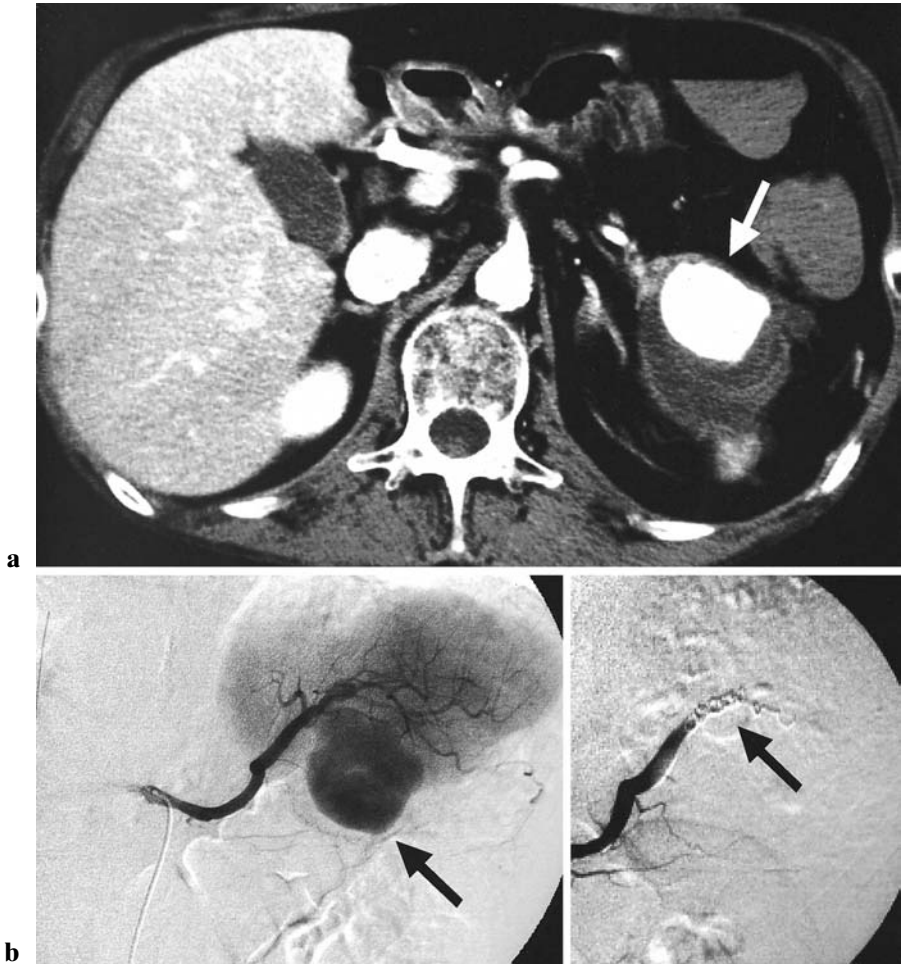


Fig. 1. **a** Computed tomography (CT) revealed a pseudoaneurysm 3cm in diameter in a pseudocyst at the pancreatic tail (*arrow*). **b** Angiography revealed a pseudoaneurysm at the caudal pancreatic artery (*arrow*). **c** Transcatheter arterial embolization (TAE) of the splenic artery was performed with metallic coils (*arrow*)



Fig. 2. **a** CT revealed that the pseudocyst at the pancreatic head had increased in size (*arrow*). **b** Endoscopic retrograde cholangiopancreatography (ERCP) revealed no communication between the pseudocyst and the main pancreatic duct, and external drainage of the pseudocyst (*arrow*) was performed



Fig. 3. **a** Upper gastrointestinal (GI) endoscopy revealed hemorrhage from the papilla of Vater. **b** CT revealed a pseudocyst 4cm in diameter at the pancreatic body (*arrow*). **c** ERCP revealed dilatation of the main pancreatic duct communicating with the pseudocyst and stenosis of the lower common bile duct

showed hyperamylasemia. CT revealed that the pseudocyst at the pancreatic head had increased in size (Fig. 2a). Endoscopic retrograde cholangiopancreatography (ERCP) revealed no communication between the pseudocyst and the main pancreatic duct, and external drainage of the pseudocyst was performed (Fig. 2b). After drainage, the patient was able to eat without abdominal pain or hyperamylasemia, and 2 weeks later the drainage tube was drawn out without any complications.

The second patient was a 52-year-old man with alcoholic chronic pancreatitis. He had consumed one-third bottle of Japanese Shochu per day for the past 30 years. He had a past history of 7 years of alcoholic chronic pancreatitis. He presented at a local hospital with hematemesis 8 months ago, but upper GI endoscopy failed to detect the source of bleeding. He recently presented with hematemesis again, and emergent upper GI endoscopy revealed hemorrhage from the papilla of Vater (Fig. 3a). CT detected hemorrhage in a pseudocyst at the pancreatic body, but he underwent only blood transfusion without treatment for the pseudocyst. Two weeks later, he suffered from repeated hema-

temesis, and he was referred to our hospital. CT revealed a pseudocyst 4cm in diameter at the pancreatic body (Fig. 3b). Angiography revealed remarkable angiogenesis around the pseudocyst with hemorrhage inside (Fig. 4a). Hematemesis was considered to be due to rupture of the pseudoaneurysm, and TAE of the main feeder arteries, the dorsal pancreatic artery, and the posterior superior pancreaticoduodenal artery was performed with metallic coils and successfully stopped bleeding from the papilla of Vater (Fig. 4b). However, this patient also complained of severe epigastralgia after meals, and the pseudocyst of the pancreatic body had increased in size. ERCP revealed dilatation of the main pancreatic duct communicating with the pseudocyst, but the distal pancreatic duct was not demonstrated. ERCP also showed stenosis of the lower common bile duct (Fig. 3c). Drainage of the pancreatic ductal systems was considered necessary to treat the chronic pancreatitis. Side-to-side pancreaticojejunostomy, including the pseudocyst, and transduodenal papilloplasty were performed 1 month after TAE. The patient was discharged on postoperative day 16 and has been followed up without signs of rebleeding. Both pa-

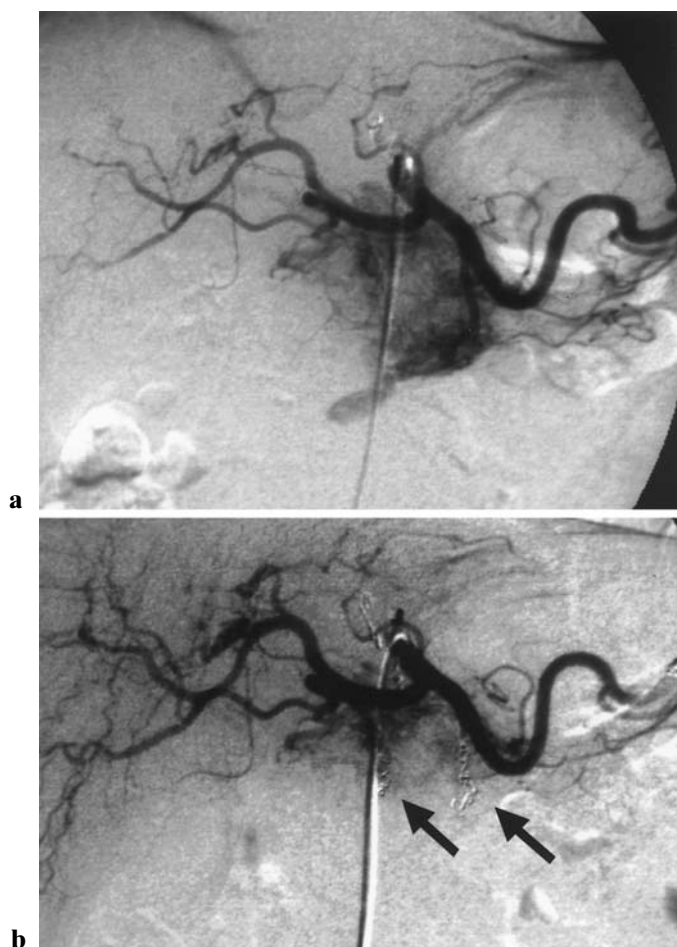


Fig. 4. **a** Angiography revealed angiogenesis around the pseudocyst and hemorrhage inside. **b** TAE of the dorsal pancreatic artery and the posterior superior pancreaticoduodenal artery was performed with metallic coils (*arrows*)

tients have been followed up without any abdominal pain or bleeding up to the present time.

Discussion

Hemosuccus pancreaticus is gastrointestinal bleeding from the duodenal papilla through the pancreatic duct. Originally, succus pancreaticus meant pancreatic juice, and hemosuccus pancreaticus is differentiated from hemobilia. In 1931 Lower and Farrell¹ reported the first case of hemorrhage through the pancreatic duct due to rupture of a pseudoaneurysm of the splenic artery. In 1970, Sandblom² first used the term hemosuccus pancreaticus for two cases of ruptured aneurysms of the common hepatic artery and splenic artery. Other terms used to describe this entity include hemoductal pancreatitis and wirsungorrhagia.³ Hemosuccus pancreaticus is usually caused by rupture of a pseudoaneurysm of a

peripancreatic artery to the pancreatic duct, or hemorrhage of a peripancreatic artery into the pseudocyst communicating with the pancreatic duct, both in chronic pancreatitis. Reportedly, chronic pancreatitis causes a pseudoaneurysm of adjacent arteries in 10% of cases, with rupture in 2%–10%.⁴ The splenic artery and its branches are the most common causes of bleeding (45%), followed by the gastroduodenal artery (17%) and the pancreaticoduodenal artery (16%).⁵

Pathogenesis of the pseudoaneurysm and angiogenesis of the peripancreatic arteries is associated with digestion of the arterial wall by pancreatic enzymes. The pathogenesis of intrapseudocystic hemorrhage is believed to involve the following steps: (1) pancreatic enzymes activated by reflux of enterokinase in the duodenum digest the small vessels in the wall of the pseudocyst; (2) the elevation of intrapseudocystic pressure causes injury of the small vessels in the wall; and (3) a peripancreatic pseudoaneurysm ruptures into the pseudocyst.⁶ Clinical symptoms and signs include upper gastrointestinal bleeding and epigastralgia resulting from the elevation of pressure in the pancreatic duct by blood clots. Jaundice is sometimes seen because of the reflux of blood to the bile duct. It is difficult to diagnose hemosuccus pancreaticus, because the bleeding is usually intermittent, as in our first case. If upper GI endoscopy reveals active bleeding from the duodenal papilla, hemosuccus pancreaticus can be diagnosed easily, as in our second case. However, even when none of the typical findings are seen on upper GI endoscopy, the detection of a blood clot in the duodenum indicates the possibility of hemosuccus pancreaticus. Hemosuccus pancreaticus should be suspected in any case of obscure gastrointestinal bleeding, especially in patients with chronic pancreatitis.

Diagnosis of hemosuccus pancreaticus is made on the basis of ultrasonography or CT, which are performed to detect pancreatic pseudocysts or aneurysm of the peripancreatic arteries. Bleeding into a pseudocyst or a pseudoaneurysm can be detected on dynamic CT, and if hemorrhage is found, abdominal angiography is useful not only to locate the source of bleeding but also to stop the bleeding with TAE. Bivins reported that angiography detected extravasation from the peripancreatic aneurysm to the pancreatic duct in three of eight cases of hemosuccus pancreaticus.⁷

Treatments for hemosuccus pancreaticus include surgical resection or ligation of bleeding vessels and radiologic interventional procedures. In most cases, TAE by metallic coils or gel foams should be attempted before resorting to surgical procedures. Mandel et al.⁴ reported that embolization of peripancreatic aneurysms was performed with a 79% success rate (15 of 19 patients). As in our first case, embolization of the splenic artery causes partial splenic infarction. However, reduced

blood flow to the spleen is usually compensated by sufficient collateralization. In our first case, transient pyrexia occurred after TAE, but no other complications were detected. Benz et al.⁸ reported a case of successful implantation of a metal Palmaz stent in the splenic artery for a short-stemmed aneurysm.

In conclusion, TAE is a minimally invasive and effective treatment for controlling hemorrhage in hemosuccus pancreaticus. However, the achievement of transient hemostasis by TAE does not obviate the necessity of radical treatment of chronic pancreatitis, such as internal drainage, external drainage, or surgical resection. In the present cases, we selected the treatment individually according to the findings of ERCP, such as stenosis or dilatation of the main pancreatic duct and communication between the main pancreatic duct and the pseudocysts. Chronic pancreatitis should be treated with the patient in stable condition after TAE, and careful follow-up is necessary.

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