Multiple pancreaticoduodenal artery aneurysms with a primary duodenal fistula

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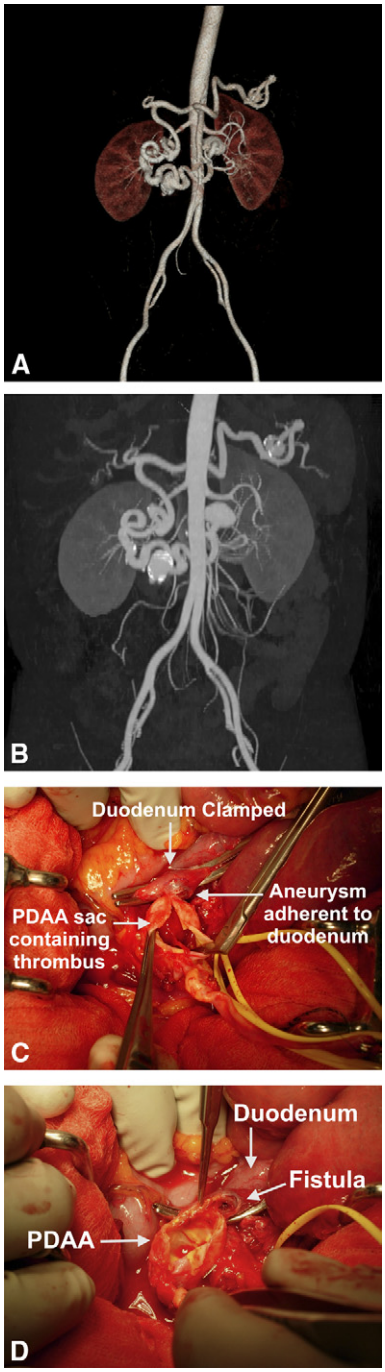
A 48-year-old woman presented with hematemesis and melena. She was a nonsmoker, with a history of hypercholesterolemia. There was no history of alcohol abuse or pancreatitis. She was investigated with a gastroscopy, capsule endoscopy, and push enteroscopy, all of which failed to demonstrate a source of bleeding. She had an episode of syncope associated with a drop in hemoglobin to 47g/L. A computed tomography angiogram demonstrated two saccular aneurysms arising from the inferior pancreaticoduodenal artery measuring 2.5 × 1.9 cm and 2.0 × 2.6 cm, with the larger aneurysm closely abutting the third part of the duodenum. A 1.0-cm aneurysm in the distal splenic artery and a 90% celiac trunk stenosis was also identified (*A*/Cover and *B*).

The patient had ongoing hematemesis requiring an urgent laparotomy. The larger aneurysm was found to be adherent to the third part of the duodenum with a communicating fistula (*C* and *D*). The aneurysms were resected and the duodenum was oversewn. Postoperatively, she developed worsening upper abdominal pain and sinus tachycardia; therefore, on day 2 an exploratory laparotomy was performed. The bowel appeared viable and there was no evidence of leak from the duodenum. Her postoperative course was complicated by a retroperitoneal collection, which was managed with percutaneous drainage and antibiotics. She was discharged home after 4 weeks and has since made a good recovery.

Pancreaticoduodenal artery aneurysms (PDAAs) are estimated to comprise 2% of all visceral artery aneurysms. False PDAAs occur secondary to pancreatitis, septic emboli, or abdominal trauma. True PDAAs are thought to arise due to increased flow through the pancreatic arcade, commonly secondary to celiac trunk stenosis/occlusion. PDAAs may be an incidental finding or present with abdominal pain. In patients with a ruptured PDAA, shock and gastrointestinal bleeding has been described with erosion into the bowel. There is no correlation between the size of the true PDAA and rupture risk, therefore, it has been suggested that all true PDAAs should be treated.[1]

The treatment options include endovascular embolization and open surgical ligation. Some authors have recommended revascularization of celiac occlusive disease in all cases, however, others consider it unnecessary if the risk of ischemia to the liver or duodenum is not considered high.[2]



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