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Gastroduodenal Artery Aneurysm Presenting as Chronic Gastrointestinal Blood Loss

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INTRODUCTION

Gastrointestinal hemorrhage is common in patients with chronic pancreatitis. The bleeding source is usually the mucosa of the stomach and duodenum. Gastroduodenal artery aneurysms in patients with pancreatitis are rare, but frequently present with massive upper intestinal bleeding. We present a case of recurrent occult and overt gastrointestinal bleeding over a 1-year period in a patient with a clinically silent pancreatic pseudocyst. The final diagnosis was established by endoscopic visualization of bleeding from the ampulla of Vater. The natural history and the approach to diagnosis and treatment of peripancreatic pseudoaneurysms is reviewed.

CASE REPORT

A 48-year-old man was in his usual state of health until 11/83 when he was found to be anemic (Hb 6 g/100 ml, hematocrit 18%, mean corpuscular volume-72) after admission for toe surgery. He was given 2 U of packed red blood cells preoperatively and underwent no further evaluation.

During March 1984, he was hospitalized elsewhere twice for melena, hemotochezia, and fatigue. Barium studies of the upper gastrointestinal tract, small bowel, and colon were negative twice. Upper endoscopy revealed only mild gastritis, and colonoscopy a tiny polyp. He continued to be anemic after discharge.

In April 1984, he first developed midepigastric and right upper quadrant abdominal aching, unaffected by meals, position, or bowel movements. Progressing in severity over 2 wk, the pain would briefly remit only if the patient had hematemesis. Upper endoscopy after hematemesis revealed erosive duodenitis with visible blood in the first and second portions of the duodenum. He was later discharged on cimetidine.

His pain became intermittent, lasting 6 to 12 h and occurring every 2 to 3 wk. In May 1984, he was referred to University of Michigan Medical Center for evaluation. After repeated questioning, the patient admitted to heavy ethanol intake and having been hospitalized with pancreatitis twice in 1982. His hematocrit was 27%. Stools were positive for blood. Upper and lower gastrointestinal endoscopy again revealed mild gastritis and small polyps, respectively. Neither of these findings was sufficient to explain the anemia. Small bowel enteroclysis and a technicium-labeled red blood cell scan were negative. Ultrasound of the abdomen demonstrated pancreatic calcifications and a 6 cm fluid filled mass in the head of the pancreas. The patient's abdominal pain spontaneously resolved, and his stools became negative for blood. He was again discharged on antacids, cimetidine, and ferrous sulfate for close follow-up.

On returning to the clinic in June 1984, the patient was free of abdominal pain and had a good appetite. He denied any episodes of overt blood loss. On physical examination, his vital signs were normal and there were no orthostatic changes. There were no stigmata of chronic liver disease. The rest of the physical examination was normal except for mild midepigastric and right upper quadrant fullness with associated tenderness. Blood counts revealed a hematocrit of 24% and a reticulocyte count of 14%. The patient was believed to have chronic ongoing upper gastrointestinal bleeding, and underwent upper endoscopy. The mucosa of the esophagus, stomach, and duodenum was normal. There was fresh blood in the first and second portions of the duodenum. After washing and aspirating the duodenum, blood was seen oozing from the ampulla of Vater. The patient was then admitted with a presumptive diagnosis of bleeding pancreatic pseudocyst. Repeat ultrasound demonstrated no change in the pancreatic pseudocyst. Celiac angiography demonstrated a gastroduodenal artery pseudoaneurysm with accumulation of contrast in a $3 \times 5 \times 5$ cm clot filled cyst (Figs. 1 and 2). Subsequently, ligation of the gastroduodenal artery and Roux-en-Y psuedocyst jejunostomy were performed. The patient was discharged with a hematocrit of 42%, and has had no evidence of recurrent bleeding.

DISCUSSION

The incidence of gastrointestinal bleeding in association with pancreatitis is variable, ranging from 1.4–41% (1–4). The causes of gastrointestinal bleeding in the majority of patients with pancreatitis include coexistant peptic ulcer disease, stress gastritis, variceal bleed-

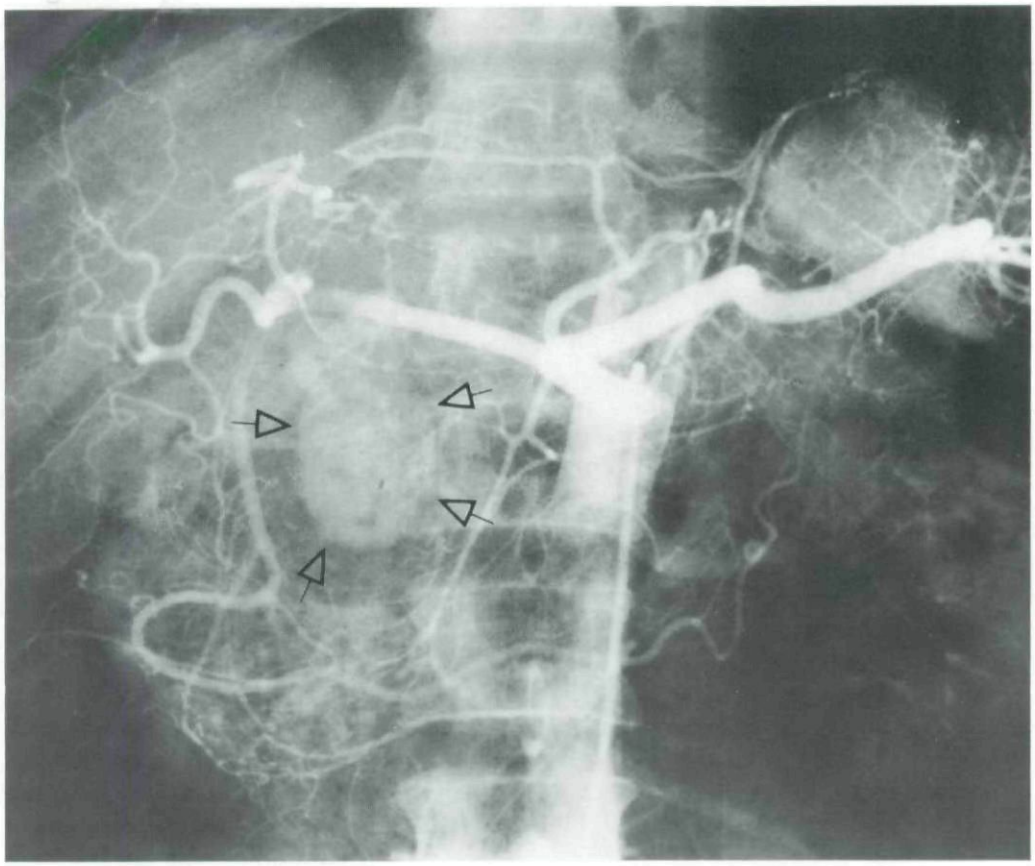

FIG. 1. Celiac angiogram demonstrating blood filled pseudocyst in head of the pancreas (*arrows*).

ing, and intestinal ischemia secondary to enzymatic mesenteric vessel digestion. Gastrointestinal bleeding is usually self-limited and resolves with the acute inflammation.

A small subset of gastrointestinal bleeding associated with pancreatitis is due to pseudoaneurysms of the peripancreatic vasculature. The pseudoaneurysm results from weakening of the arterial wall caused by partial enzymatic digestion and pressure necrosis by the pseudocyst (5, 6). The incidence of pseudoaneurysms diagnosed by angiography in an unselected population with pancreatitis is approximately 10%, although no patient progressed to rupture or bleeding (7). The incidence of psuedoaneurysmal hemorrhage, however, in patients with pseudocysts is 6–8% (8, 9). Chronic exposure of the arterial wall to digestive enzymes and traction on it resulting from scarring and granulation in the wall of the pseudocyst may be important factors in initiating gastrointestinal hemorrhage.

The arteries most commonly involved in pseudoaneurysm formation are: the splenic artery (45–50%), the gastroduodenal artery (15%), and the pancreaticoduodenal arteries (15%) because of their close association with the head of the pancreas (10, 11). Aneurysms of the hepatic, celiac, superior mesenteric, and gastric arteries have been reported, although they rarely bleed (10, 11). Gastrointestinal bleeding is most common from the gastroduodenal and pancreaticoduodenal arteries because of their close association with the pancreatic and biliary ducts (10, 11).

A history of ethanol abuse and episodes of pancreatitis are common in these patients, and was discovered after close questioning in this patient. Hematemesis and epigastric pain were the presenting symptoms in 71% while melena occurred in 43% (12). The relative absence of abdominal pain and the initial lack of overt bleeding delayed the diagnosis in this case.

Pseudoaneurysmal bleeding can present in one of two ways. The first is occult, with chronic blood loss marked by anemia and only intermittently melanotic stools (1). This was the presentation in this case, although the length of time before discovery (7 months), and the relative absence of melena and abdominal pain, makes this case unusual. Marks *et al.* (13) estimate that one-third of patients with gastrointestinal bleeding in association with pancreatitis who have a negative radiological and endoscopic evaluation have peripancreatic vessel bleeding. Bivens *et al.* (14) hypothesized that blood from the leaking pseudoaneurysm traverses the pseudocyst and enters the gastrointestinal tract via the pancreatic duct, biliary tree, or a fistulous connection directly into the gastrointestinal lumen. Cessation

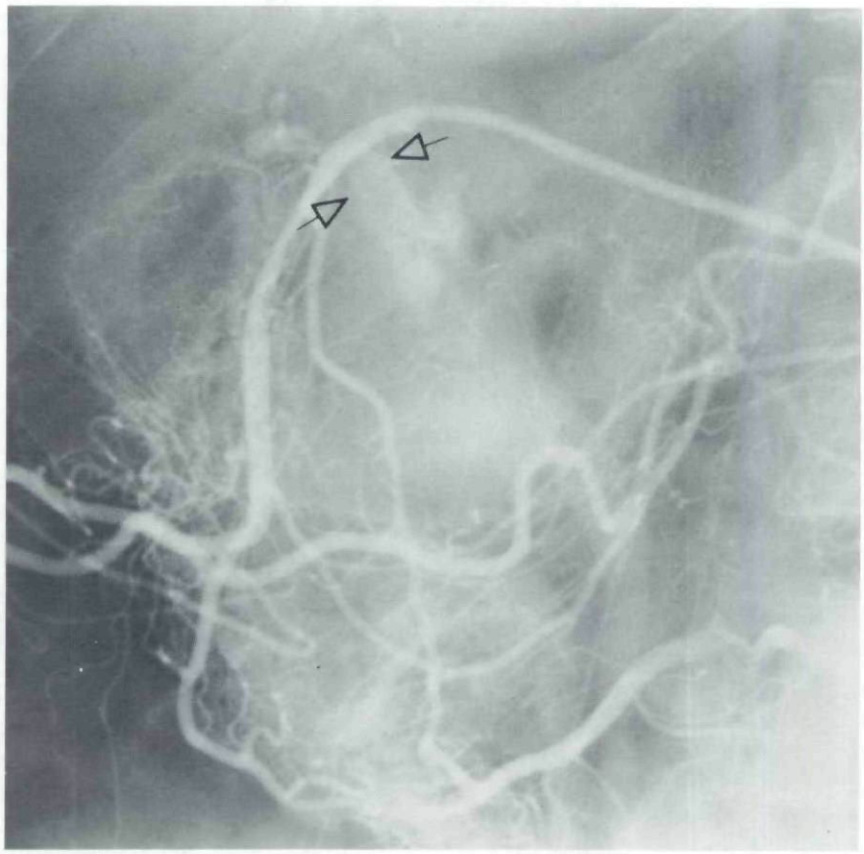

FIG. 2. Selective gastroduodenal artery injection demonstrating pseudoaneurysm and bleeding source (*arrows*).

of bleeding could occur after clot tamponade in the cyst or by obstruction of the outflow tract to the gastrointestinal lumen. Recurrent cycles of self-limited melena could be produced by thrombolysis, duct recannulation, and hemorrhage followed again by fresh thrombus formation and local hemostasis (14). The relief of pain reported by this patient after hematemesis may represent decompression of the blood filled pseudocyst.

The second presentation of pseudoaneurysmal bleeding is massive upper gastrointestinal hemorrhage. This most commonly occurs after several episodes of minor gastrointestinal bleeding, but can be the initial manifestation (10, 11). Bleeding is frequently life threatening with average losses of 2500 ml (3). The mortality rate of untreated cases is 90% (11), while treated cases have a mortality of 25–37% (8, 10, 15, 16). These figures underscore the need to aggressively pursue and define the bleeding source in patients with pancreatitis, especially if pseudocysts are present.

The diagnosis of peripancreatic pseudoaneurysmal bleeding may be difficult, since bleeding can be intermittent and can occur into areas such as the fourth portion of the duodenum and small bowel, which are endoscopically inaccessible (11, 17–21). Direct visualization of bleeding through the ampulla of Vater is uncommon (14, 15), but may be seen more frequently if endoscopy is done during an episode of hematemesis. If upper and lower gastrointestinal endoscopy do not reveal a bleeding source, prompt celiac axis and superior mesenteric arteriography provides the best diagnostic yield, with a positive predictive value of 93% in one series (16). Stabile *et al.* (11) demonstrated that emergent laparotomy without prior arteriography failed to demonstrate a bleeding source in four of four patients, who all required reoperation, while three of four patients with preoperative arteriography had definitive control of their bleeding at the initial operation. Knowledge of the location of a pseudoaneurysm shortens the operative procedure and minimizes vascular damage incurred during an intraoperative search for a bleeding site (10, 11).

If arteriography is not diagnostic, endoscopic retrograde cholangiopancreatography may demonstrate ductal occlusion by thrombus (22, 23). Computerized tomography can demonstrate pancreatic pseudocysts and occasionally the pseudoaneurysm directly, if contrast is used (3, 24).

The definitive treatment for peripancreatic pseudoaneurysmal bleeding has changed over the last 20 years. Conservative, nonsurgical therapy was recommended in the past because of a high operative mortality. Currently, definitive treatment of both the bleeding vessel

and associated pseudocyst has been favored by most authors (10, 11, 14, 25). Pancreatic resection to excise the pseudocyst, coupled by debridément of the pancreatic bed and ligation of bleeding vessels is believed to decrease the incidence of postoperative infection and rebleeding (10). Others believe that direct bipolar ligation of the involved vessel with repair of the pseudocyst wall at the site of vessel erosion would lessen operative mortality (14, 25). Stabile *et al.* (11), in their review of 123 cases, found no important differences in outcome between these two operations. However, they did demonstrate that bleeding pseudocysts in the head of the pancreas carried a higher operative mortality rate (43%) than lesions in the body and tail (16%), irrespective of the mode of repair (11).

Therapeutic angiography is now widely used for many forms of gastrointestinal bleeding. Selective intraarterial vasopressin infusion or embolization techniques may permit the stabilization of the patient before definitive surgery. Recently, embolization therapy with autologous clot, gelatin sponge, and wire coils, as well as electrical coagulation have been successfully used instead of surgical treatment in selected patients with pancreatic pseudoaneurysmal bleeding (11, 24, 26–28). However, the injection of autologous clot and absorbable gelatin sponge has been reported to produce aneurysmal rupture because of the high pressures required to inject these materials (29). The role of therapeutic angiography *versus* surgery for definitive treatment of peripancreatic pseudoaneurysmal bleeding requires further study.

In summary, celiac arteriography demonstrated a large pseudoaneurysm of the gastroduodenal artery in a man with a long history of anemia and recurrent hematemesis. Cyst drainage and gastroduodenal artery ligation resulted in complete recovery.

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