

Spontaneous Regression of Pancreatic Pseudocyst Mimicking a Submucosal Tumor of the Stomach with Upper Gastrointestinal Bleeding : Report of a Case

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Abstract

Hemorrhage of a pancreatic pseudocyst is one of the severe complications of pancreatitis. Erosion into adjacent organs, especially into the upper gastrointestinal tract, by pancreatic pseudocysts has been reported. The endoscopic imaging of involvement of the stomach by pancreatic pseudocyst can mimic a submucosal tumor of the stomach, which may rupture into the stomach and manifest as upper gastrointestinal bleeding. We report a 66-year-old male with alcoholism who presented with tarry stool passage for 2 to 3 days. Panendoscopy revealed a protruding mass, measuring about 6 cm in diameter, with central ulceration and adherent blood clot located over the posterior wall of the cardia. Biopsy showed a focal ulcer with infiltration of inflammatory cells, but without evidence of malignancy. Computed tomography of the abdomen showed a cystic lesion extending from the pancreatic tail to the cardia of the stomach in favor of a pancreatic pseudocyst. He refused either radiologic or surgical treatment and was treated in a conservative way only. Follow-up panendoscopy showed a complete regression of the pseudocyst 6 month later. (J Intern Med Taiwan 2006; 17: 128-132)

Key Words : Pancreatic pseudocyst, Submucosal tumor, Gastrointestinal bleeding, Endoscopy

Introduction

Pancreatic pseudocysts develop in 5% to 15% of cases of acute pancreatitis and in 20% to 40% of cases of chronic pancreatitis¹. Hemorrhage is the single most important cause of death in patients with pancreatic pseudocyst. Erosion into adjacent organs by a pancreatic pseudocyst has been reported in 22% of patients with a pseudocyst². The gastrointestinal tract is most frequently affected³. Hemorrhagic pseudocyst due to involvement of the splenic artery, gastroduodenal artery and pancreaticoduodenal artery has also been commonly reported⁴. The treatment recommendations for hemorrhagic pseudocysts are various. However, most of them will be treated by either radiologic arterial embolization or surgical resection of pseudocyst⁵. We report a case in which a patient presenting with upper gastrointestinal bleeding caused by a hemorrhagic pancreatic pseudocyst directly rupturing into the stomach was successfully treated conservatively.

Case Report

A 66-year-old man presented with tarry stool passage for 2 to 3 days. He had alcoholism with several episodes of alcoholic hepatitis in the past. However, no known history of pancreatitis and cirrhosis was noted before. Except tarry stool passage, neither abdominal pain nor vomiting can be noted during this episode. On admission, physical examination revealed a normal appearance without stigmata of cirrhosis. Panendoscopy showed a protruding mass, measuring about 6 cm in diameter, with central ulceration and adherent blood clot located over the posterior wall of the cardiac area (Fig. 1A). Biopsy of it showed a focal ulcer with infiltration of acute and chronic inflammatory cells, but without evidence of malignancy. Laboratory studies yielded the following data: hemoglobin (Hb) 9.8 g/dL (normal: 13-18 g/dL); aspartate aminotransferase (AST) 355 IU/L (normal: 10-35 IU/L); alanine aminotransferase

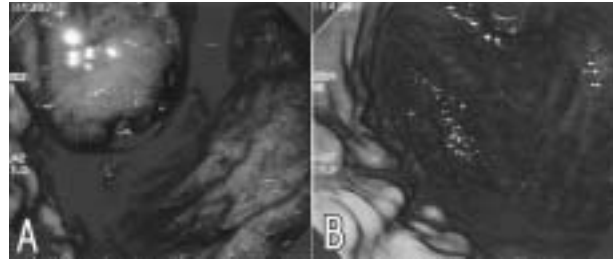


Fig. 1. A. Panendoscopy showed a broad-based protruding tumor mass, measuring about 6 cm in diameter, with central ulceration and adherent blood clot, implying recent hemorrhage. B. Six months later, panendoscopy revealed a complete regression of the tumor mass which was proven to be a pancreatic pseudocyst.



Fig. 2. A. Computed tomography of the abdomen showed a hypodense mass lesion extending from the tail of the pancreas to the posterior wall of the stomach, which favored a diagnosis of pseudocyst. It was noted that the point ruptured into the stomach with ulceration (arrow). B. Sagittal reconstruction of computed tomography showed a clear relationship between the stomach (S), the pancreatic pseudocyst (PP) and the tail of the pancreas (PT, arrow).

(ALT) 161 IU/L (normal: 3-30 IU/L); alkaline phosphatase (Alk-P) 429 IU/L (normal: 65-275 IU/L); gamma-glutamyl-transferase (γ GT) 117 IU/L (normal: 5-40 IU/L); amylase 40 IU/L (normal: 30-200 IU/L); lipase 43.7 IU/L (normal: 10-60 IU/L); carcinoembryonic antigen (CEA) 0.27 ng/mL (normal: 0-7 ng/mL); and CA19-9 < 2 U/mL (normal: 0-37 U/mL). The computed tomography of the abdomen showed a hypodense mass lesion extending from the pancreatic tail to the cardia of the stomach (Fig. 2A, 2B). Pancreatic pseudocyst was diagnosed. The patient refused either radiologic or surgical treatment because of personal reasons. Alcohol abstinence was recommended. Panendoscopy showed dramatic re-

gression of the submucosal lesion with a scarring ulcer and a normal appearing gastric mucosa observed respectively, 1- and 6-month after the episode (Fig. 1B).

Discussion

A pancreatic pseudocyst is a cystic lesion that lacks of epithelial lining which occurs from fluid collection arising from within or around the pancreas⁶. It develops in 5% to 15% of cases of acute pancreatitis and 20% to 40% of chronic pancreatitis¹. It usually develops over a period of 1 to 4 weeks after onset of acute pancreatitis. Abdominal pain with or without radiation to back is the usual presenting symptom. Physical examination may find a palpable and tender mass in the middle or left upper abdomen. The X-ray examination shows the displacement of the gastrointestinal tract. Sonography and computed tomography are two reliable diagnostic tools for the diagnosis of the pancreatic pseudocyst. The complications of pseudocyst include pain, obstruction of the gastrointestinal tract or common bile duct, infection and hemorrhage. However, prolonged studies on the natural history of pseudocysts are a rarity. Vitas and Sarr published a series in which 68 patients with pancreatic pseudocysts were followed⁷. After 51 months, 63% of the patients were asymptomatic. Only 9% of the patients developed any of the complications described above.

Hemorrhage is the most important cause of death in patients with complicated pancreatic pseudocyst. Severe bleeding is seen if the hemorrhagic pseudocyst ruptures into the gastrointestinal tract, peritoneal cavity, retroperitoneum or pancreatic duct⁸. Of them, the gastrointestinal tract is the most frequently affected³. The erosion of vessels by pseudocyst is the main cause of hemorrhagic pseudocyst. The splenic artery is affected in half of the cases, whereas the gastroduodenal and pancreaticoduodenal arteries are less common sources of bleeding^{4,8-10}.

A pancreatic pseudocyst may also involve adja-

cent organs such as the duodenum and colon by contiguity and may extend as far as the groin and mediastinum^{2,11}. Gastric wall involvement has also been reported. Zahlan et al. reported a case in which a pseudocyst of the pancreatic tail that ruptured into the wall of the stomach and mimicked a non-bleeding gastric tumor¹². Urakami et al. also reported a case of alcoholic pancreatitis in which the pseudocyst had ruptured into the stomach and manifested as hematemesis. In this case report, the upper gastrointestinal bleeding was found to be derived from the splenic artery by angiography and was managed by surgery¹³. Radke and Bell reported a case of pancreatic pseudocyst with intramural involvement of gastric wall, but without rupture into the gastric lumen¹⁴. They suggested that it represented an incomplete phase of perforation of the gastric wall, resulting in the formation of submucosal or subserosal gastric masses.

The optimal timing of treatment of pancreatic pseudocyst is occurrence of complications, including uncontrolled pain, infection and hemorrhage, obstruction of the gastrointestinal tract or common bile duct. Percutaneous drainage, endoscopic drainage and surgery are the major methods for the treatment of pancreatic pseudocyst. Focusing on the treatment of hemorrhagic pancreatic pseudocyst, Sand et al. proposed a treatment protocol⁵. In brief, patients with clinical ongoing bleeding and with hemorrhagic pancreatic pseudocyst in sonography and computed tomography, urgent angiography were conducted. When angiography showed a pseudoaneurysm, the patients were treated with immediate embolization and delayed elective surgery whenever candidates for surgery. In contrast, if angiography showed no evidence of a pseudoaneurysm, conservative treatment was recommended.

In our case, despite of long-term drinking by the patient, there are no manifestations of either acute or chronic pancreatitis ever noted. However, the patient developed an upper gastrointestinal bleeding as the

initial manifestation of the pancreatic pseudocyst. Computed tomography of the abdomen did not show any evidence of pseudoaneurysm that usually presents with brisk bleeding and needs to be managed by angiography or surgery. In contrast, in our case, the bleeding was subtle and could be successfully managed with medical therapy. It was regarded as the direct involvement of the gastric wall with minor vessel bleeding, rather than direct erosion of a major vessel. The patient did not re-bleed even though he did not treated surgically or by transcatheter arterial embolization which supports the conclusion of Sand and his colleagues that an angiography negative hemorrhagic pseudocyst can be treated conservatively with low mortality and re-bleeding rate⁵.

Bradley et al reported that spontaneous resolution occurred in 42% of patients who developed pseudocyst for less than 6 weeks. However, only 8% of patients whose pseudocyst persisted for 7 to 12 weeks had spontaneous resolution¹⁵. Although a large proportion of pseudocysts resolve spontaneously in patients with acute pancreatitis, spontaneous regression of a hemorrhagic pancreatic pseudocyst with gastric involvement like the case reported here has been rarely reported.

In summary, this is a case of pancreatic pseudocyst with gastric wall involvement which mimicked a submucosal tumor and ruptured into the gastric lumen with upper gastrointestinal bleeding. It is worthy to note that this pancreatic pseudocyst with bleeding into stomach regressed spontaneously with conservative treatment only.

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以擬胃黏膜下腫瘤併上消化道出血表現之胰臟偽 囊腫其自發性消失：一病例報告

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摘 要

胰臟偽囊腫出血是胰臟炎一嚴重之併發症，主要由胰臟偽囊腫侵犯至鄰近器官，特別是上消化道器官。其對胃部侵犯之內視鏡影像擬胃黏膜下腫瘤，並可往胃內部破裂形成上消化道出血。本病例報告為一66歲酗酒男性以解黑便2至3天表現。胃鏡檢查顯示在賁門處有一6公分之黏膜下腫瘤伴隨有潰瘍出血之情形。病理切片為潰瘍但並無惡性變化。腹部電腦斷層顯示從胰臟尾部至賁門處為一胰臟偽囊腫。此病人拒絕包括放射線科及外科之治療而採保守療法。6個月後胃鏡追蹤檢查顯示此胰臟偽囊腫已完全消失。