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Case Report

Hemosuccus pancreaticus as a complication of acute necrotizing pancreatitis: A case report

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A B S T R A C T

Hemosuccus pancreaticus (HP) is characterized by hemorrhage from the pancreatic duct through the major duodenal papilla. It represents the least common cause of upper gastrointestinal bleeding (UGIB), occurring in approximately 1 in 1,500 cases and predominantly affecting men. A 58-year-old woman with a history of recurrent pancreatitis presented with severe epigastric pain, vomiting, and diarrhea. Laboratory tests revealed hyperamylasemia. Imaging indicated acute necrohemorrhagic pancreatitis with peripancreatic fluid collections. Despite interventions including drainage and antibiotic therapy, she developed massive hematemesis and hemodynamic instability. Computed tomography indicated active bleeding in the peripancreatic region, and angiography confirmed a diagnosis of HP. Embolization of the gastroduodenal artery was performed; however, the patient died of multiorgan failure. HP, although rare, should be considered in patients with chronic pancreatitis and intermittent UGIB. Diagnosing HP can be challenging, requiring collaboration between gastroenterologists and interventional radiologists. Early intervention is crucial due to the high mortality rate associated with severe cases.

Keywords: Embolization, therapeutic; Gastrointestinal hemorrhage; Pancreatitis, acute Hemorrhagic

Introduction

Hemosuccus pancreaticus (HP) is a rare cause of gastrointestinal bleeding, characterized by hemorrhage from the pancreatic duct through the major duodenal papilla.[1] It represents the least common etiology of upper gastrointestinal bleeding (UGIB), occurring in approximately 1 in 1,500 cases of UGIB.[1,2]

In up to 80% of cases, HP originates from the pancreas. It typically results from the rupture of aneurysms due to fibroinflammatory changes associated with chronic pancreatitis. However, it can also occur in the context of acute necrotizing pancreatitis, where released pancreatic enzymes cause damage to the vascular wall, leading to vessel rupture. Other causes include fistulized pancreatic pseudocysts or pancreatic tumors.[1,3] The vessels most commonly involved are the splenic artery, gastroduodenal artery, and mesenteric artery, although intrapancreatic vessels may also be impacted.[3,4]

HP predominantly affects men, with a male-to-female ratio of 7:1, and is associated with alcohol consumption.[2,3] Clinically, it presents as epigastric abdominal pain, hematemesis, and melena. Patients may also exhibit a pulsatile abdominal mass and anemic syndrome, along with elevated amylase levels due to increased intraductal pressure.[2] While the bleeding is typically intermittent and not serious enough to cause hemodynamic instability, it can lead to hemorrhagic shock in severe cases, necessitating immediate intervention.[1,4]

The gold-standard diagnostic tool for this condition is angiography, which additionally offers the opportunity for therapeutic intervention.[1,5] However, diagnosis is most often supported by radiographic findings, including contrast extravasation or the presence of adjacent pseudoaneurysms.[2,3]

We present the case of a patient who experienced a complex episode of acute pancreatitis. During hospitalization, the condition progressed to HP, resulting in multiorgan failure and subsequent death.

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Case Report

A 58-year-old woman presented with a medical history that included cholecystectomy for cholecystitis, splenectomy and hepatic cystectomy due to hydatid cyst, and three episodes of mild acute pancreatitis. The patient had no history of drug or alcohol use. After her most recent episode of pancreatitis, she underwent investigation for autoimmune, lithiasic, alcoholic, and pharmacological causes, all of which yielded negative results. Abdominal magnetic resonance imaging (MRI) revealed a slightly atrophic pancreas but no other abnormalities. Treatment with ursodeoxycholic acid and pancreatic enzymes was initiated.

Three months following her last episode of acute pancreatitis, the patient presented to the emergency department with intense epigastric abdominal pain that radiated in a belt-like pattern, accompanied by vomiting and diarrhea. Upon admission, she was in poor general condition and obtunded, with a blood pressure of 170/90 mmHg and a heart rate of 60 beats per minute, which necessitated her transfer to the intensive care unit. Physical examination revealed epigastric tenderness and delayed capillary refill. The patient exhibited no jaundice, choluria, acholia, or other significant findings.

Laboratory tests upon admission revealed a hemoglobin level of 11 g/dL, leukocytosis with a white blood cell count of 19,500 × 10^3/μL, a C-reactive protein level of 0.04 mg/dL, hyperamylasemia with an amylase level of 2,415 IU/L, total bilirubin of 0.168 mg/dL, and a creatinine level of 0.63 mg/dL. Initial management consisted of fluid therapy, non-steroidal anti-inflammatory drugs, and antiemetics.

Given the patient's unfavorable progression, an abdominal MRI was conducted, which revealed evidence of acute necrotizing pancreatitis involving 60% of the gland. Subsequently, contrast-enhanced abdominal computed tomography (CT) demonstrated acute necrohemorrhagic pancreatitis involving the body and tail, accompanied by multiple peripancreatic collections that exhibited signs of infection (Fig. 1).

The gastroenterology and interventional radiology teams decided to perform ultrasound-guided percutaneous drainage of the collection. They obtained a sample of purulent fluid, which was sent to the laboratory, and initiated empirical antibiotic therapy. Five days after the procedure, a follow-up contrast-enhanced CT scan of the abdomen revealed adequate drainage of the peripancreatic collections and no evidence of new collections.

During the patient's second week of hospitalization, control tests indicated a hemoglobin level of 7 g/dL. Upper gastrointestinal endoscopy (UGE) revealed no abnormalities. A subsequent abdominal CT scan demonstrated well-positioned percutaneous drainage; however, new fluid collections were identified in the left flank, left iliac fossa, and rectouterine pouch (Fig. 2), suggesting the need for a second pelvic drainage placement.

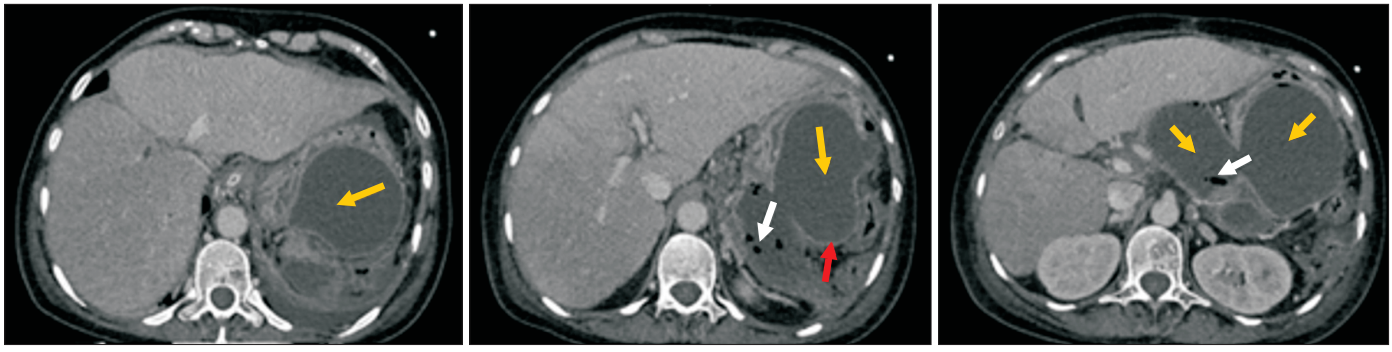

Fig. 1. Contrast-enhanced computed tomography of the abdomen reveals extensive inflammatory changes in the pancreas, affecting the body and tail, with necrosis exceeding 50% (Balthazar E). Orange arrows indicate large multiloculated retroperitoneal, peripancreatic, and retrogastric collections, extending to the left anterior pararenal space. Red arrow denotes a thick peripheral capsule with contrast enhancement, and the presence of gas within these areas (white arrows) suggests signs of an infected collection.

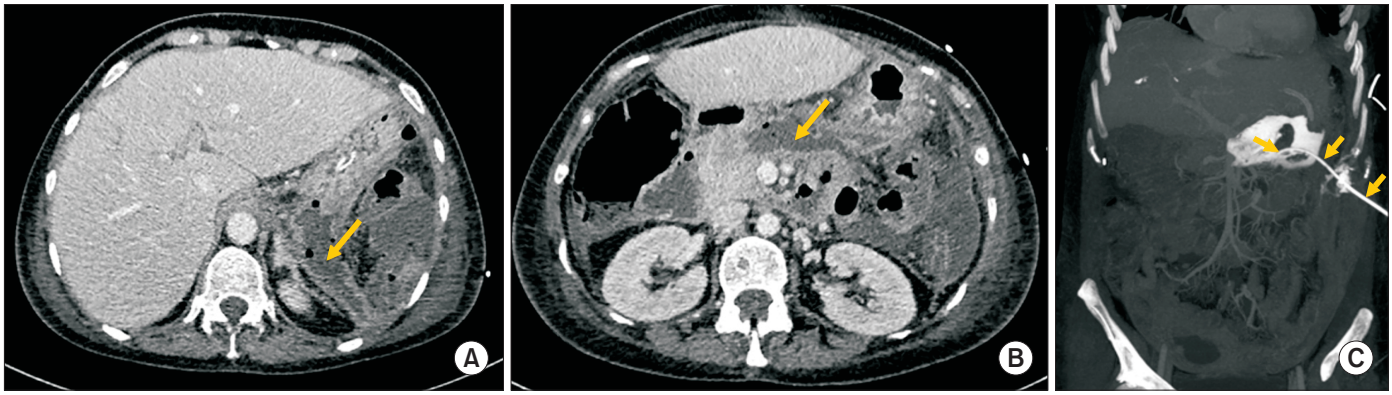

Fig. 2. Follow-up computed tomography (CT) of the abdomen after placement of a percutaneous pigtail catheter. The percutaneous drainage was inserted through the left hypochondrium and flank, with the distal tip positioned within the retroperitoneal peripancreatic collection. (A, B) Axial CT images in the portal phase. Orange arrows indicate the residual collection following placement of the pigtail drainage catheter. (C) Coronal maximum intensity projection reconstruction in the portal phase. Orange arrows show the position of the drainage catheter within the collection.

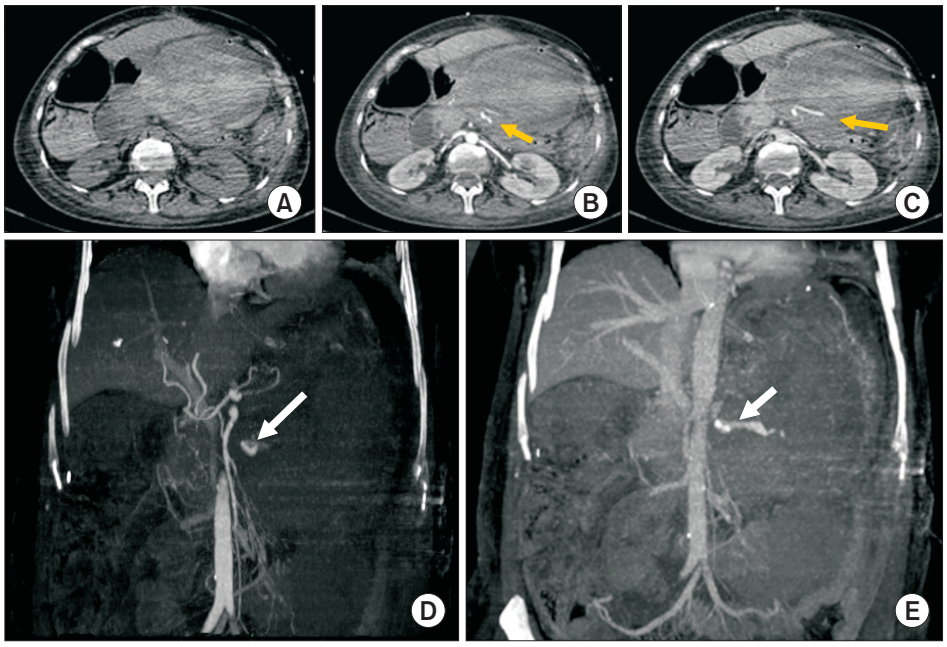

Fig. 3. Computed tomography (CT) scan of the abdomen and pelvis. (A) Non-contrast. (B) Arterial phase. (C) Portal phase. A large acute retroperitoneal peripancreatic hematoma is observed, with contrast extravasation appearing as a jet at the body of the pancreas during the arterial phase and increasing during the venous phase (orange arrows). (D, E) Coronal maximum intensity projection reconstruction in the portal phase. White arrows indicate jet-like bleeding within the peripancreatic region.

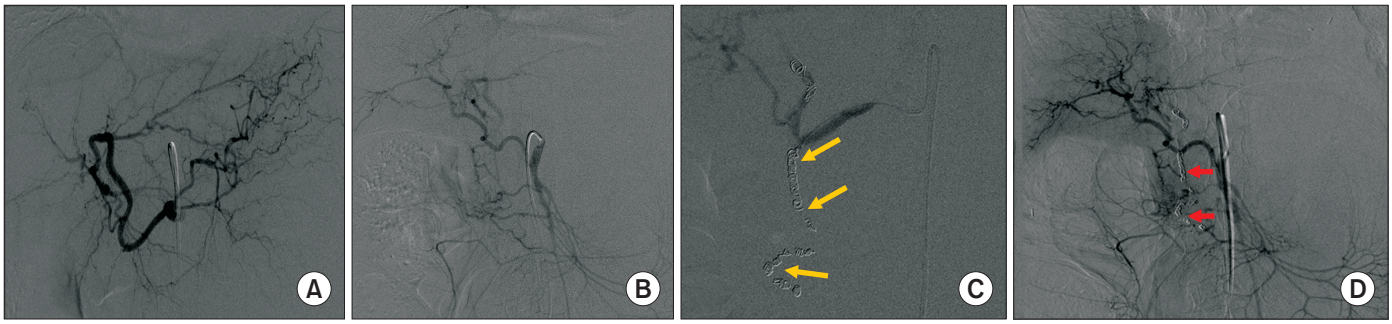

Fig. 4. Selective digital subtraction angiography. (A) The celiac trunk, with the splenic artery absent due to previous splenectomy. The gastroduodenal artery is not filled, likely due to competitive flow through inferior pancreaticoduodenal branches anastomosed with the mesenteric artery. (B) The superior mesenteric artery, shown giving rise to the right hepatic artery. (C) Post-embolization angiographic control of the gastroduodenal artery using coils (orange arrows) placed via the celiac trunk and the superior mesenteric artery. (D) Successful embolization was confirmed by the absence of contrast filling in the gastroduodenal artery and pancreaticoduodenal branches (red arrows).

The following day, the patient experienced massive hematemesis. She also developed hemodynamic compromise and cardiorespiratory arrest, which was reversed within minutes. A hemoglobin check revealed a level of 5.5 g/dL, necessitating the transfusion of two units of red blood cells. Subsequently, the drain in the patient's left flank displayed serohematic content. A contrast-enhanced CT scan of the chest, abdomen, and pelvis revealed contrast extravasation at the pancreatic body during the arterioportal phase, active peripancreatic bleeding, and a 13.4 × 9.3 cm hematoma consistent with a complicated aneurysm (Fig. 3). These findings led to a diagnosis of HP.

Given the high morbimortality associated with surgical intervention, angiography and embolization were chosen as the preferred treatment approach. The patient, exhibiting hemodynamic instability, was admitted to the angiography suite. There, arterial embolization of the gastroduodenal artery was performed distal to the proximal third of the right gastroepiploic artery, successfully halting the extravasation of the contrast medium (Fig. 4).

After 28 days of hospitalization, complicated by multiple interventions and concomitant infections, the patient developed multiorgan failure and died.

Written informed consent was obtained from the patient's next of kin for the publication of this case report and any accompanying images.

Discussion

HP is an uncommon cause of gastrointestinal bleeding and is rarely considered in the differential diagnosis of UGIB. In the present case, suspicion arose from the presence of serohematic content in the drainage, the occurrence of hematemesis, and a decrease in hemoglobin levels.

Diagnosing HP is challenging due to the intermittent nature of the associated bleeding, which can be easily overlooked during examinations.[6] Blood clots that form within the pancreatic duct can obstruct the flow of blood to the duodenum, contributing to the low sensitivity of UGE in detecting HP compared to other UGIB etiologies.[4,5] A recent review reported evidence of bleeding in only 46% of examined cases,[1] while another study indicated that UGE results were normal in nearly half of patients.[7] Thus, the

most reliable diagnostic methods for HP are contrast-enhanced CT and MRI, which can reveal pseudocysts, pseudoaneurysms, or the sentinel clot sign within the pancreatic duct,[6] and angiography, which directly visualizes the source of bleeding and facilitates immediate intervention with embolization.[1]

The reported mortality rate of HP is 9.6%, which increases to 90% in severe cases.[1] Treatment options include interventional radiology or surgery for major vessel hemorrhage. Chemoembolization has a reported success rate of between 80% and 100%[3] and is associated with lower mortality and fewer complications compared to surgery.[6] The most frequent complications include distal ischemia, occurring in 7% to 16% of cases, and rebleeding, reported in 17% to 37% of instances.[3,6]

HP is a relatively understudied condition. As of 2022, only 153 cases had been documented in the English-language medical literature since it was first described by Lower and Farrel in 1931.[1,7,8] A recent search of both Spanish and English sources revealed fewer than 200 reported cases to date. The etiology of HP is diverse and includes acute and chronic pancreatitis, fistulized pseudocysts, pseudoaneurysm rupture, and benign and malignant pancreatic tumors.[8,9] A meta-analysis conducted by Ru et al[9] determined that 83% of HP cases are secondary to pancreatitis, while 16% are attributable to tumors, such as intraductal papillary mucinous neoplasms.

HP is a rare cause of gastrointestinal bleeding that should be considered in patients presenting with chronic pancreatitis, abdominal pain, and intermittent UGIB, as well as in those with acute pancreatitis complicated by bleeding. The initial diagnosis can be challenging; however, once HP is suspected, it is crucial to coordinate care between gastroenterologists and interventional radiologists or surgeons due to the high mortality associated with severe cases of HP.

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Data Sharing Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

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