

Case Report

Stone-induced spontaneous pancreatico-gastric fistula in chronic calcific pancreatitis presenting with upper gastrointestinal bleeding: a rare case report

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ABSTRACT

Pancreatico-gastric fistula is a rare sequela of chronic calcific pancreatitis and only infrequently presents with upper gastrointestinal bleeding, often posing a diagnostic challenge. We describe a 63-year-old male with alcohol-related chronic calcific pancreatitis who presented with hematemesis and melena. Cross-sectional imaging demonstrated a subepithelial gastric lesion closely resembling a gastrointestinal stromal tumor, along with obliteration of the fat planes between the stomach and pancreas. A large calcified calculus was identified within the main pancreatic duct, lying in close proximity to the posterior gastric wall. Endoscopic ultrasound findings further favored a neoplastic etiology. However, surgical exploration revealed a spontaneous pancreatico-gastric fistula resulting from direct erosion of the impacted ductal stone into the stomach. The patient underwent wide local excision of the involved gastric segment along with a Frey's procedure. Postoperatively, he had a biochemical leak, formerly classified as grade A postoperative pancreatic fistula, and had an uneventful recovery. This case illustrates an unusual pathogenic mechanism of fistula formation, where pressure-induced erosion by a pancreatic duct calculus mimicked a gastric neoplasm and presented with upper gastrointestinal bleeding.

Keywords: Chronic calcific pancreatitis, Pancreatico-gastric fistula, Pancreatic duct stone, Upper gastrointestinal bleeding, Frey's procedure

INTRODUCTION

Pancreatic fistula represents a pathological communication between the pancreatic ductal system and adjacent or remote anatomical structures, arising from disruption of ductal integrity with subsequent leakage of enzyme-rich pancreatic secretions.

In benign settings, such fistulas most commonly occur secondary to complications of pancreatitis, including

pseudocyst formation, pancreatic necrosis, or sustained ductal hypertension associated with chronic inflammatory changes.^{1,2}

Malignant etiologies include pancreatic adenocarcinoma and intraductal papillary mucinous neoplasms, where progressive tumor infiltration into adjacent structures can lead to fistula formation.³

Unlike acute pancreatitis, where fistulas often arise due to spontaneous rupture of a pseudocyst into the stomach,

fistula formation in chronic pancreatitis is uncommon owing to dense fibrosis and scarring of surrounding tissues. Nevertheless, when pancreatogastric fistulas develop in this setting, they can result in serious complications, including massive upper gastrointestinal bleeding due to erosion into adjacent vascular structures.^{4,5}

We present a rare case of a spontaneous pancreatico-gastric fistula caused by an impacted calculus in the main pancreatic duct in a patient with chronic calcific pancreatitis, presenting unusually with upper gastrointestinal bleeding.

CASE REPORT

A 63-year-old man presented with a four-day history of hematemesis and melena. He was a known case of alcohol-related chronic calcific pancreatitis since 2018, without evidence of endocrine or exocrine insufficiency.

On admission, he was afebrile, non-pale, and anicteric. His vital signs were stable, with a blood pressure of 110/70 mmHg and a pulse rate of 86/min. Abdominal examination revealed a soft, non-tender abdomen with no palpable masses or organomegaly; the liver and spleen were not enlarged.

Laboratory evaluation revealed hemoglobin 9.9 g/dl and total leukocyte count 5,380/ μ l. Liver function tests showed total bilirubin 0.45 mg/dl, AST 61.9 IU/l, ALT 59.0 IU/l, and alkaline phosphatase 165 IU/l. Renal and metabolic parameters were normal (urea 29 mg/dl, creatinine 1.02 mg/dl, INR 0.97, and HbA1c 5.5%).

Contrast enhanced computed tomography (CECT) of the abdomen revealed a distended stomach with focal bulging and thickening of the posterior gastric wall near the greater curvature. On the upper axial cuts, this produced the appearance of a well-defined, rounded, hypodense lesion projecting into the gastric lumen, closely mimicking a gastric gastrointestinal stromal tumor (GIST) (Figures 1a and b).

However, the lesion showed indistinct margins and complete loss of fat planes with the underlying pancreatic head and body, raising suspicion for an inflammatory mass.

The pancreas was atrophic with heterogeneous attenuation and multiple coarse calcifications consistent with chronic calcific pancreatitis. A large, densely calcified stone was seen impacted in the main pancreatic duct at the level of the pancreatic head, lying directly against the posterior gastric wall (Figures 1c and d).

Upper gastrointestinal endoscopy revealed Barrett's esophagus. A 5×4 cm subepithelial mass was visualized arising from the posterior gastric wall along the greater curvature of the fundus/proximal body, with a 0.5 cm focal mucosal ulceration on its surface. The duodenal bulb (D1)

and the second part of the duodenum (D2) contained altered blood, while the remaining gastrointestinal mucosa appeared normal.

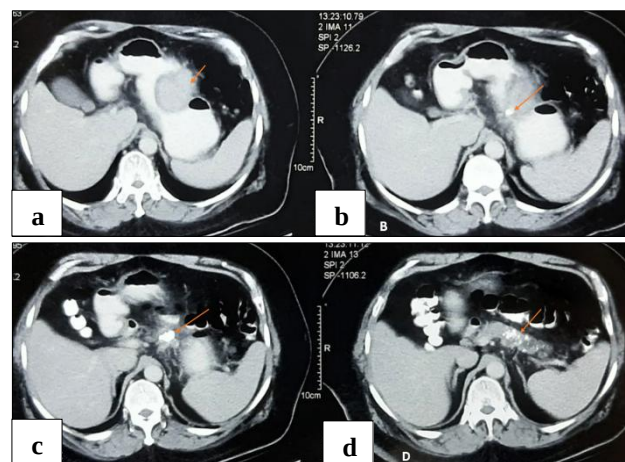


Figure 1: CECT abdomen (axial images) (a and b) distended stomach with posterior wall bulge along the greater curvature, producing a hypodense mass-like appearance mimicking a gastric GIST (arrow), the perigastric complex shows internal air foci and loss of fat planes with the pancreatic head (arrow); and (c and d) atrophic, heterogeneous pancreas with multiple coarse calcifications. A large calcified stone is impacted in the main pancreatic duct at the pancreatic head, abutting the posterior gastric wall, with associated MPD dilatation in the tail (arrow).

Endoscopic ultrasound revealed a well-defined hypoechoic mass measuring 5.4 cm in maximum dimension, located approximately 4 cm distal to the gastroesophageal (GE) junction. The lesion showed a central hypoechoic area and appeared to arise from the muscularis propria, findings highly suggestive of a gastrointestinal stromal tumor (GIST).

Exploratory laparotomy revealed a pancreatico-gastric fistula, formed secondary to a large 2.5×2 cm impacted main pancreatic duct (MPD) stone that had eroded directly into the posterior gastric wall (Figure 2a).

The fistulous tract was carefully dismantled, and a wide local excision of the unhealthy posterior gastric wall was performed. Given the underlying chronic calcific pancreatitis with an obstructed MPD, a Frey's procedure (local pancreatic head coring with longitudinal MPD drainage) was carried out in the same sitting (Figure 2b).

Postoperatively, the patient had a biochemical leak, formerly classified as grade A postoperative pancreatic fistula, with drain fluid amylase measuring 6085 U/l on postoperative day 3. He remained clinically stable and was discharged with the abdominal drain in situ.

Histopathological examination revealed serositis with organizing inflammation in the resected gastric wall, while

the pancreatic tissue showed features consistent with chronic pancreatitis.

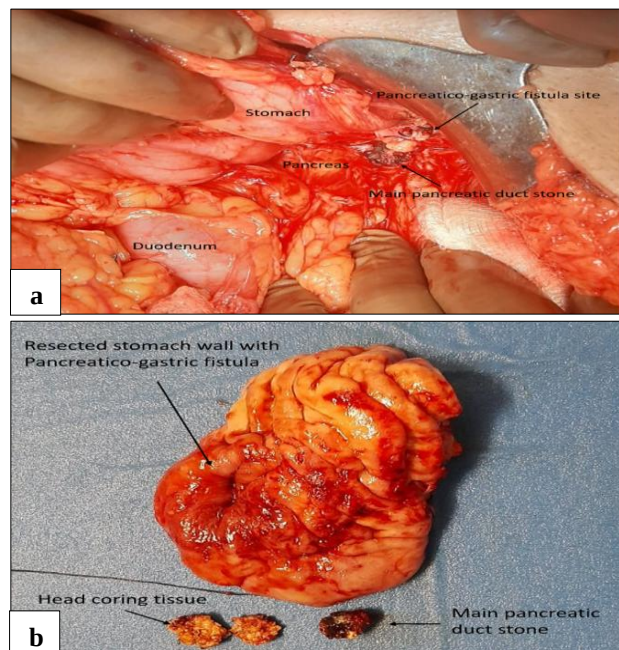


Figure 2: Intraoperative findings and resected specimen (a) intraoperative view showing the pancreatico-gastric fistula along with the impacted main pancreatic duct stone eroding into the posterior gastric wall; and (b) resected posterior gastric wall specimen demonstrating the fistulous opening, along with the extracted pancreatic duct stones and pancreatic head-coring tissue.

DISCUSSION

Pancreatic inflammation may give rise to both internal and external fistulas. External fistulas are most often postoperative in origin, whereas internal fistulas can develop spontaneously in the context of pancreatitis. These may involve adjacent structures such as the gastrointestinal tract or, less commonly, extend into the mediastinum, and can present with severe complications including massive hematemesis or sepsis.⁶

The present patient, a known case of chronic calcific pancreatitis, presented with a four-day history of hematemesis and melena. The pancreatico-gastric fistula in this case likely resulted from prolonged mechanical compression of the posterior gastric wall by the inflamed pancreas and an impacted pancreatic duct calculus, leading to pressure-induced erosion akin to a decubitus ulcer mechanism.

In pancreatitis, fistulous involvement most frequently affects the transverse colon and the splenic flexure, followed by involvement of the duodenum, whereas fistulization into the small intestine, stomach, or esophagus is relatively uncommon.⁷⁻⁹

Our case shares similarities with the recently published report by Wijerathne et al, where chronic pancreatitis led to a pancreatico-gastric fistula presenting with recurrent gastrointestinal bleeding. However, unlike their case, in which the fistula was associated with splenic vein erosion and dense inflammatory adhesions, our patient demonstrated direct erosion of a large pancreatic ductal calculus into the gastric wall—an even rarer mechanism of fistula formation.⁵

A thorough pre-intervention evaluation is essential prior to endoscopic or surgical drainage of pancreatic fluid collections to exclude underlying cystic neoplasms, particularly in patients with recurrent episodes of pancreatitis.

Notably, approximately 50% of internal and 70-90% of external pancreatic fistulas may resolve with nonoperative management.¹⁰

Early surgical intervention is recommended for colonic fistulas, which have a poorer prognosis. In cases of severe hemorrhage, as observed in our patient, surgical intervention remains the only life-saving option.

CONCLUSION

Pancreatico-gastric fistula represents an uncommon complication of chronic calcific pancreatitis and may present with upper gastrointestinal bleeding, posing a diagnostic challenge in the preoperative setting. Surgical management remains the definitive treatment, with the operative approach guided by the extent of fistula involvement and underlying pancreatic ductal pathology. This case underscores a rare pathogenic mechanism, wherein a large impacted pancreatic duct calculus resulted in pressure-induced erosion into the gastric wall, manifesting as upper gastrointestinal bleeding.

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REFERENCES

1. Tsiotos GG, Smith CD, Sarr MG. Incidence and management of pancreatic and enteric fistulas after surgical management of severe necrotizing pancreatitis. *Arch Surg.* 1995;130(1):48-52.
2. Fielding GA, McLatchie GR, Wilson C, Imrie CW, Carter DC. Acute pancreatitis and pancreatic fistula formation. *Br J Surg.* 1989;76(11):1126-8.
3. Kochhar R, Masoodi I, Singhal M, Dutta U, Nagi B, Wig JD, et al. Pancreatogastric fistula after severe acute pancreatitis: a case report. *Gastrointest Endosc.* 2009;69(4):969-71.
4. Al-Tamimi HA, Hancock J, Palacio C, Yao X. Pancreatogastric fistulas associated with chronic pancreatitis: a case report and review of the literature. *Gastrointest Endosc.* 2007;65(6):942-5.

5. Wijerathne S, Bandaranayake G, Abeyasinghe A, Sivaganesh S, Fernandopulle N, Subasinghe D. Pancreatogastric fistula in chronic pancreatitis: a rare and life-threatening cause of recurrent massive upper gastrointestinal bleeding. *J Surg Case Rep.* 2025;2025(9):rjaf619.
6. Kochhar R, Jain K, Gupta V, Singhal M, Kochhar S, Poornachandra KS, et al. Fistulization in the GI tract in acute pancreatitis. *Gastrointest Endosc.* 2012;75(2):436-40.
7. Levy I, Ariche A. Complete recovery after spontaneous drainage of pancreatic abscess into the stomach. *Scand J Gastroenterol.* 1999;34(9):939-41.
8. Aldridge MC, Francis ND, Glazer G, Dudley HA. Colonic complications of severe acute pancreatitis. *Br J Surg.* 1989;76(4):362-7.
9. Doberneck RC. Intestinal fistula complicating necrotizing pancreatitis. *Am J Surg.* 1989;158(6):581-3.
10. Voss M, Pappas T. Pancreatic Fistula. *Curr Treat Options Gastroenterol.* 2002;5(5):345-53.

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