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CASE REPORT | PANCREAS

Transhiatal Herniation of the Pancreas: A Rare Cause of Acute Pancreatitis

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ABSTRACT

Transhiatal herniation of the pancreas is rare. Acute pancreatitis secondary to this phenomenon is particularly unusual. A 102-year-old woman presented with 1 day of severe chest pain, vomiting, dyspnea, and diaphoresis. Serum lipase was elevated, and computed tomography angiogram of the chest and magnetic resonance cholangiopancreatography revealed a hiatal hernia containing the pancreas, with associated findings of pancreatitis. Pancreatitis in this setting may be due to repetitive trauma or ischemia from sliding, intermittent folding of the pancreatic duct, or pancreatic incarceration. Mild cases can be managed supportively, with surgery being reserved for severe cases or for younger patients with low surgical risk.

INTRODUCTION

Hiatal hernia refers to the migration of the stomach through the diaphragm and into the chest cavity. It is more common in women, and the risk increases with age. Type IV hiatal hernia involves large defects in the phrenoesophageal membrane, which allows other intraabdominal organs, most commonly the colon, small bowel, and omentum, to herniate into the chest. Transhiatal herniation of the pancreas in type IV hiatal hernia is rare, with only 16 cases previously described in the literature. Acute pancreatitis secondary to this phenomenon is particularly unusual, as only 7 such cases have been reported.

CASE REPORT

A 102-year-old woman with a history of aortic stenosis, hiatal hernia, and an episode of pancreatitis believed to be from pancreatic herniation 2 years prior presented with 1 day of severe chest pain radiating to the back with nausea, vomiting, dyspnea, and diaphoresis. She denied other abdominal complaints or alcohol use. Her medication list included aspirin, amlodipine, losartan, and pantoprazole. Vital signs were within normal limits. The patient did not exhibit any changes in mental status, and physical exam was notable for mild epigastric tenderness. Laboratory data revealed whole blood count 6.68 x 10^3/μL, hematocrit 34.7%, blood urea nitrogen 40 mg/dL, creatinine 1.2 mg/dL, and calcium 9.7 mg/dL. Other labs included normal liver function tests and triglycerides, and an elevated serum lipase 1,719 U/L. Abdominal ultrasound 3 years prior revealed no evidence of cholelithiasis. Acute coronary syndrome was ruled out. Computed tomography (CT) angiogram of the chest with contrast was negative for pulmonary embolism and aortic dissection. However, a large intrathoracic hiatal hernia containing the stomach, proximal duodenum, and the body and tail of the pancreas was noted, with perigastric fluid in the posterior mediastinum and mesenteric stranding thought to be secondary to pancreatitis (Figure 1).

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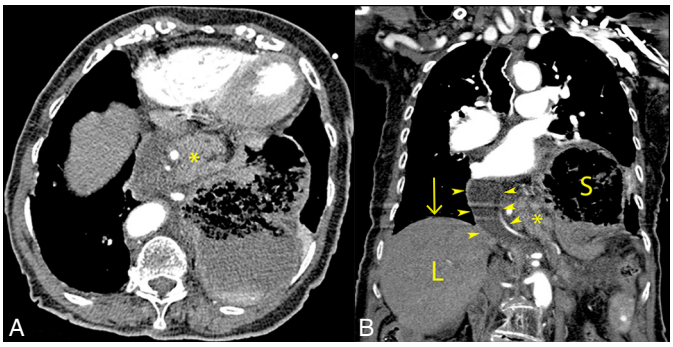

Figure 1. (A) Axial section of CT chest angiogram illustrating herniation of the pancreas (star) into the mediastinum. (B) Coronal section of CT chest angiogram illustrating herniation of the pancreas (star) above the level of the diaphragm (arrow) into the mediastinum, with associated free fluid (arrow heads) secondary to pancreatitis. Other notable structures: liver (L) and stomach (S).

Subsequent magnetic resonance cholangiopancreatography (MRCP) revealed a normal gallbladder without cholelithiasis and the transhiatally herniated pancreas with associated stranding and trace fluid, but no evidence of necrosis or fluid collections (Figure 2). Because her previous episode of pancreatitis resolved with conservative management, the patient was again managed supportively with intravenous fluids. On hospital day 3 she tolerated slow reintroduction of an oral diet. She declined surgical intervention and was discharged home with physical therapy on hospital day 4. The patient felt well at 1 week follow-up and has not had any recurrent episodes 6 months post-discharge.

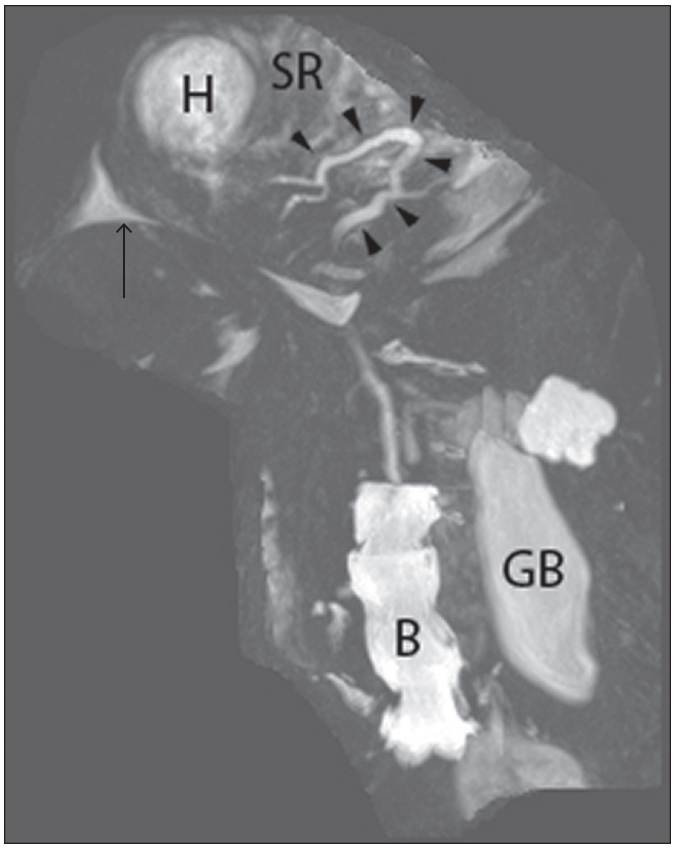

Figure 2. Coronal section of MRCP rotated 180°, confirming herniation of the pancreatic duct (arrow heads) above the level of the diaphragm (arrow). Other notable structures: heart (H), stomach ruggae (SR), gallbladder (GB), and bowel (B).

DISCUSSION

Transhiatal herniation of the pancreas is rare due to its retroperitoneal location and fixation by the ligament of Treitz.[1] Stretching of the transverse mesocolon is thought to increase the laxity of the posterior adhering fascia, thereby permitting mobilization of the pancreas. A review of 16 cases reported an equal proportion of men and women, with 12 cases occurring in or after the sixth decade of life.[2] Fifteen of the patients were symptomatic, and the diagnosis was confirmed with cross-sectional imaging. The one asymptomatic patient was diagnosed by thoracic CT performed to investigate an intractable cough.[3]

Acute pancreatitis as a complication of this phenomenon is even rarer, and has been previously described in 7 patients. While it is difficult to definitively establish that pancreatic herniation was the sole etiology of pancreatitis in these cases, other common causes were excluded. Symptoms primarily consist of pain localized to the chest and epigastrium. Other manifestations include dyspepsia, diaphoresis, nausea, vomiting, and dyspnea. The diagnosis is confirmed by significant serum lipase elevation and imaging evidence of pancreatic herniation with inflammatory changes suggestive of pancreatitis (peripancreatic fluid, mesenteric stranding).

A variety of mechanisms have been proposed to explain hernia-associated pancreatitis. The parenchymal trauma from repetitive transhiatal sliding of the organ may itself induce pancreatitis, or it may cause intermittent stretching and traction of blood vessels such as the vascular pedicle, resulting in ischemic insults. Other possibilities include volvulus formation or intermittent folding of the pancreatic duct leading to pancreatic secretion against a fixed obstruction, intraductal hypertension, and ensuing inflammation. Incarceration of the pancreas resulting in anoxic injury may also be another mechanism.[4-8]

Given the limited number of reported cases, the optimal management of acute pancreatitis secondary to pancreatic herniation is not well established. Historically, patients were managed with immediate hiatal hernia repair.[1] In contrast, more recent cases, including ours, have been successfully managed with supportive care (e.g., intravenous fluid supplementation, analgesia, early feeding) due to perceived elevated surgical risk or patient refusal.[8,9] However, severe cases involving incarceration, perforation, or unresponsiveness to medical therapy should be managed surgically.

The rate of recurrence appears to be low, so the role of elective surgical reduction is unclear. Moreover, most affected patients are of advanced age and often have significant comorbidities that render them poor operative candidates.[10,11] Thus, the decision to pursue surgical repair should be considered on a case by case basis. Elective repair may be considered in younger patients with low perioperative risk to prevent the development of serious complications in the future.

Our patient's presentation was notable for the thoracic localization of her symptoms and the absence of traditional risk factors for pancreatitis, highlighting the importance of appropriate clinical suspicion. It is also the first reported case of recurrent pancreatitis from a hiatal hernia. Clinicians should consider the rare diagnosis of pancreatitis secondary to hiatal herniation in the differential diagnosis of patients of advanced age presenting with chest pain and a negative cardiopulmonary evaluation.

DISCLOSURES

Author contributions: All authors wrote the manuscript. J. Wang is the article guarantor.

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