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Zackria, Rasiq Popa, Alina

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CASE REPORT | PANCREAS

Transhiatal Herniation as the Cause of Acute Pancreatitis After Toupet Fundoplication

Rasiq Zackria, DO¹, and Alina Popa, MD¹

¹Internal Medicine Residency, Graduate Medical Education, Riverside Community Hospital/University of California, Riverside School of Medicine, Riverside, CA

ABSTRACT

Hiatal translocation of the pancreas is rare because of its retroperitoneal location. Acute pancreatitis as a complication of hiatal hernia is uncommon. A 33-year-old man presented for 2 days of worsening epigastric abdominal pain and substernal chest pain. Laboratory studies were essentially unremarkable; however, computed tomography demonstrated a large right-sided hiatal hernia containing the entire stomach and the body of the pancreas, with peripancreatic edema consistent with pancreatitis. Most cases can be managed conservatively; however, elective surgical repair is suggested in severe cases or patients with low surgical risk.

INTRODUCTION

Hiatal hernia refers to the translocation of the stomach through the diaphragmatic esophageal hiatus into the thoracic cavity. The risk of developing a hiatal hernia is higher in women and increases with age, with few cases reported in patients younger than 40 years old. Acute pancreatitis as a complication of hiatal hernia is unusual, and there have been a limited number of case reports published in the literature.¹⁻⁶

CASE REPORT

A 33-year-old man with a medical history of gastroesophageal reflux disease, hiatal hernia status post-Toupet fundoplication 5 years ago, irritable bowel syndrome, and depression presented for worsening intermittent epigastric abdominal pain and substernal chest pain of 2-day duration. He reported associated dyspnea, nausea, and nonbloody nonbilious vomiting. He denied any other symptoms and alcohol abuse. He denied any other abdominal surgeries apart from the Toupet fundoplication. He stated that the current symptoms are not similar to what he had experienced with his gastroesophageal reflux disease or irritable bowel syndrome in the past. Vital signs were within normal limits.

The patient was in moderate distress due to dyspnea and pain, and physical examination was pertinent for epigastric tenderness on deep palpation. Laboratory data revealed a mild leukocytosis of 12.5×10^3 cells/mm³, hematocrit of 37%, blood urea nitrogen of 6 mg/dL, creatinine of 0.72 mg/dL, calcium (corrected for albumin) of 9.6 mg/dL, and negative troponin level. Other laboratory studies included liver function tests, lipid panel, serum lipase, and blood alcohol level that were all within normal limits.

Abdominal ultrasound showed no evidence of cholelithiasis or common bile duct dilation. Computed tomography chest and abdomen with intravenous contrast demonstrated a large right-sided hiatal hernia with the entire stomach above the diaphragm and the body of the pancreas within the hiatal hernia with peripancreatic edema adjacent to the pancreatic body and neck (Figure 1).

The patient was treated with conservative management for his pancreatitis with resolution of symptoms on hospital day 2. Given his history of previous Toupet fundoplication, surgery was consulted for surgical intervention. On hospital day 4, the patient underwent elective repair of hiatal hernia with mesh, anterior gastropexy, and gastrotomy after a failed attempt at laparoscopic repair because of extensive adhesions. The patient had an esophogram that showed no evidence of leak and tolerated oral diet. He was subsequently discharged on hospital day 8 in good condition.

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Correspondence: Rasiq Zackria, DO, Internal Medicine Residency, Graduate Medical Education, Riverside Community Hospital/University of California, Riverside School of Medicine, 4445 Magnolia Ave, Riverside, CA 92501 (rasiqzackria@gmail.com).

DISCUSSION

Hiatal hernia refers to the translocation of the stomach either transiently or permanently through the diaphragm into the thoracic cavity, and this may involve other abdominal organs, primarily the colon, small bowel, and spleen. Pancreatic herniation through the hiatus is extremely rare because of its retroperitoneal location. The movement of the pancreas is limited because of its fixation by the ligament of Trietz; however, loosening of the transverse mesocolon that potentially leads to an increase in the laxity of the posterior fascia causing increased mobility of the pancreas has been proposed.¹

A previous literature review found only 12 cases of pancreatic herniation causing acute pancreatitis from 1958 to 2011; however, more case reports have been published since.^{2,7} Most reports are in the pediatric literature and involve congenital diaphragmatic hernias. In addition, congenital weakness of the hiatus or any condition that causes increased intraabdominal cavity pressure such as pregnancy, ascites, obesity, or trauma may support development of this condition.⁵ Although the etiology for hiatal hernia is unknown in most patients, positive intraabdominal and negative intrathoracic pressures exacerbate the hernia itself.^{8,9} Hiatal hernias are more common in women than in men, and the frequency increases with age, with patients younger than 40 years old comprising less than 10%.

Hiatal hernias are classified based on the position of the gastroesophageal junction and extent of herniation into 4 types: sliding hernia (Type I), paraesophageal hernia (Type II), mixed (Type III), and herniation of additional organs (Type IV).¹⁰ Type II to Type IV comprise less than 15% of the cases, with



Figure 1. Coronal view of computed tomography abdomen showing a large right-sided Type IV hiatal hernia containing the stomach and body of the pancreas with surrounding peripancreatic edema.

Type IV hiatal hernia being the most rare.⁵ Although most cases included the body and tail of the pancreas within the hernia sac, our case is unusual because only the pancreatic body was herniated. Acute pancreatitis as a complication of hiatal herniation is uncommon as previously described. All previous cases from the published literature were diagnosed based on exclusion of other common causes. The rarity of acute pancreatitis in hiatal herniation could be attributed to the anatomical location of the pancreas. This rarity has led to multiple proposed mechanisms to elucidate herniation-associated pancreatitis including repetitive parenchymal trauma from sliding of the organ or ischemia induced by traction and stretching of the blood vessels.^{6,11} Other mechanisms that have been suggested include incarceration of the pancreas causing anoxic injury and intermittent folding of the pancreatic duct causing pancreatic secretion in the setting of fixed obstruction, leading to inflammation.1,3,4,6,11-13

Given the scarcity of reported cases, there is no established approach to the management of acute pancreatitis secondary to pancreatic herniation. Although immediate hiatal hernia repair was suggested in the past, recent cases have demonstrated successful management with a conservative approach.^{1,5,6,13} The rate of recurrence has not been well documented, and therefore, the role of elective surgical repair is unclear. Although routine elective repair is not indicated in this subset of patients, it is strongly advised in patients who are not considered a high surgical risk because a previous study demonstrated improved overall mortality in Type III or IV hernias compared with conservative management.¹⁴ Pancreatitis in our patient was treated conservatively with fluid resuscitation, analgesic, antiemetic, and early enteral feeding as tolerated by the patient. Because of the risk of Type IV hiatal hernias becoming incarcerated, leading to strangulation or perforation, our patient was offered elective surgical intervention. A laparoscopic repair was attempted but aborted because of significant adhesions, and the patient underwent open transabdominal repair.

Although there has been no literature published to suggest that antireflux surgery predisposes a patient to herniation of the pancreas, the ligation of the ligaments supporting the intraabdominal structures could potentially predispose a patient to this situation.¹⁵ The absence of traditional risk factors for pancreatitis in our patient with a history of fundoplication accentuates the importance of high clinical suspicion for pancreatitis due to hiatal herniation; earlier cross-sectional imaging may be warranted in this subset of patients. This is the first reported case of pancreatitis due to transhiatal herniation of the pancreas in a patient with previous surgical repair of the hiatal hernia.

DISCLOSURES

Author contributions: R. Zackria wrote the manuscript. A. Popa edited the manuscript and is the article guarantor.

Informed consent was obtained for this case report.

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