Case Report

Journal of Emergency Medicine Case Reports

A Rare Emergency Diagnosis: Pancreatic Ductal Rupture

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Abstract

Chronic pancreatitis is a condition that results from episodes of acute pancreatitis. It is difficult to diagnose chronic pancreatitis in the early stages and to recognize complications. Recognizing duct rupture, one of the complications of chronic pancreatitis, is important for initiating treatment. A 75-year-old wo man presented to the emergency department with abdominal pain. Amylase and lipase levels were 65 u/L and 75 u/L, respectively. Computed tomography performed in the emergency department revealed ductal rupture and the patient was hospitalized in the gastroenterology service. Treatment included endoscopic examination followed by pancreatic rest, hydration and proton pump inhibitors. Pancreatic duct rupture, which is rarely encountered in the emergency department, should be considered as an alternative diagnosis in patients presenting with abdominal pain and a previous pancreatitis attack.

Keywords: Chronic pancreatitis, complication, ductal rupture

Introduction

Chronic pancreatitis is an inflammatory disease that causes permanent structural changes in the pancreas and can lead to impaired exocrine and endocrine functions. Chronic pancreatitis can result from attacks of acute pancreatitis due to any reason (1). Pseudocysts, biliary obstruction, pancreatic diabetes, vascular complications may occur as a result of chronic pancreatitis. Pseudocysts, one of the complications of chronic pancreatitis develop in about 10 percent of patients with chronic pancreatitis. They may be triggered by an acute exacerbation of pancreatitis or occur as a result of ductal rupture.

Pancreatic duct (DP) rupture and leakage of pancreatic fluid into the abdomen can occur as a complication of acute or chronic pancreatitis (2). The other causes are pancreatic malignancy, abdominal surgery or abdominal trauma (3). DP rupture may occur in the main pancreatic duct or in one of its small branches. The clinical consequences of a DP rupture may depend on the etiology, location, and extent of the rupture. Clinical results occur depending on the rate of pancreatic fluid secretion, the location of the leak relative to anatomical tissue regions, the systemic inflammatory response ability to control the leakage, and the obstruction of the DP with stones or strictures. A small leak of pancreatic

fluid from one of the lateral branches may resolve on its own. A persistent leakage from the main DP rupture may present with pseudocyst formation or be complicated by external pancreatic fistulas or internal fistula formation causing pleural effusion or ascites(4). Early recognition of complications is important for early initiation of treatment.

The diagnosis of ductal rupture is rarely made in the emergency department because pancreatic enzymes are not severely elevated or do not present with severe clinical signs. therefore, suspicion will help in the path to diagnosis. Here, we aimed to discuss our patient who was diagnosed with pancreatic ductal rupture, a rare complication of chronic pancreatitis, in the emergency department.

Case Report

A 75-year-old woman presented to the emergency department with nausea, vomiting and abdominal pain. Her medical history included abdominal pain in the epigastric region for the past 15 days, aggravated after meals, and accompanied by occasional nausea and vomiting. Her medical history included hypertension, diabetes mellitus, nasal squamous cell carcinoma, and previous ischemic stroke. She was taking metformin, ramipril, clopidogrel, and escitalopram. On physical examination, general condition

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Received: 24.03.2024 • **Revision:** 22.07.2024 • **Accepted:** 27.09.2024

DOI: 10.33706/jemcr.1457994

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Cite this article as: Saral Öztürk Z. Akkan S. A Rare Emergency Diagnosis: Pancreatic Ductal Rupture. Journal of Emergency Medicine Case Reports. 2024;15(4): 93-95

was good, consciousness was clear, oriented and coherent. Vital signs were arterial blood pressure: 130/80 mm/hg, pulse rate: 98 beats/minute, fever was 36.7°C. There was no incisional scar on abdominal examination. Bowel sounds were normoactive, tenderness and rebound were present in the epigastric region on palpation. When the patient's previous admissions were analyzed, it was found that she had pancreatitis approximately 1 month and 6 months ago. The patient had no subsequent hospital admissions. Biochemical tests showed amylase 65 u/L and lipase 75 u/L. C-reactive protein was 226 mg/L, white blood cell count was 12100 μL, and other parameters including liver function tests were within normal limits. Contrast-enhanced abdominal computed tomography (CCT) was performed to rule out other causes of acute abdomen due to severe pain, epigastric rebound tenderness, borderline elevated pancreatic enzymes, and elevated acute phase reactants. On CCT, the gallbladder was within normal limits, the pancreatic duct was 5 mm in size, a focal area of defect in the anterior part of the pancreatic corpus and a diffuse heterogeneous inflammatory appearance in the mesenteric adipose tissue in the adjacent peripancreatic area were observed. All these findings were consistent with acute pancreatitis and ductal rupture in the background of chronic pancreatitis (Figure-1). The patient was consulted to the gastroenterology and general surgery clinics. Urgent surgical intervention was not considered and the patient was admitted to the gastroenterology clinic for furthermore evaluation. Intravenous (IV) fluid therapy, proton pump inhibitor, and ceftriaxone were administered during hospitalization. Magnetic resonance cholangiopancreatography (MRCP) was planned in the gastroenterology service but could not be performed because the patient had claustrophobia. The patient followed a stable clinical course and was discharged at the end of treatment with a follow-up visit after 3 months. CCT and Endoscopic retrograde cholangiopancreatography (ERCP) were planned after 3 months for control. The control CCT scan showed a 2.5 cm diameter lobule contoured cystic formation at the level of the pancreatic head (Figure-2). In ERCP, the



Figure 1. Anteriorly ruptured pancreatic duct



Figure 2. Cystic formation in the head of the pancreas

papillary head was not observed in normal localization and the duct could not be entered. The patient was referred to an advanced center for further investigation. The patient died pending further investigation.

Discussion

Chronic pancreatitis is one of the causes of abdominal pain diagnosed in the emergency department (5). Chronic pancreatitis is most commonly seen in patients with frequent pancreatitis attacks. Acute pancreatitis can be considered as a status, chronic pancreatitis is the pathological ongoing response process to pancreatic damage (6,7). Acute and chronic pancreatitis are not two completely separate conditions. Can be thought of as a continuum and two parts of the same disease spectrum. Our patient also had chronic pancreatitis due to an acute pancreatitis attack.

Pancreatic fluid from a DP rupture may spread to the retroperitoneum, mediastinum, or if confined to the small sac, it is then surrounded by a well-formatted, nonepithelialized wall and becomes a pseudocyst over a period of 4 to 6 weeks. Pancreatic pseudocysts are the maturation of a phlegmonous inflammatory event and consist of an accumulation of pancreatic secretions and secondary inflammatory products resulting from pancreatic duct rupture (7). Pancreatic pseudocysts are seen in 10-20% of patients with acute pancreatitis and 20-60% of patients with chronic pancreatitis (8). Many of the clinical features of chronic pancreatitis may take time to develop and may not be present in the early stages of the disease. Chronic pancreatitis may be easier to diagnose when end-stage features develop. The clinical challenge is to make the correct diagnosis in the early cases of chronic pancreatitis, when interventions aimed at preventing disease progression may be effective (8). In our patient with a diagnosis of ductal rupture, no pseudocyst was detected at this stage. However later cystic formation developed at the site of rupture.

Internal pancreatic fistulas formed as a result of DP rupture may occasionally fistulate with other areas such as the pericardium or organs such as the stomach, bronchus and large or small intestine. Pancreatic fluid may also fistulate outward toward the skin surface, creating an external pancreatic fistula. Pancreatic ascites may develop due to anterior DP rupture and leakage of pancreatic fluid into the peritoneal cavity. Posterior DP rupture may cause pancreatic fluid to leak into the pleural space through the esophageal or aortic hiatus and cause pleural effusion. (4). In our patient, the fluid spread anteriorly and peritoneal contamination occurred due to fluid leakage around the pancreas.

ERCP has the highest accuracy in diagnosing pancreatic duct rupture because it provides detailed images of the pancreatic duct. It is also useful in determining the exact location (head, neck, trunk, or tail) and size of the rupture. However, ERCP is no longer used in the diagnosis of chronic pancreatitis due to the availability of alternative imaging methods and the risk of complications. In early disease, changes in the duct may be minimal and the diagnosis may not be obvious. Recent advances in imaging, such as multislice spiral contrast-enhanced computed tomography (CCT) and magnetic resonance imaging (MRI), may aid in the noninvasive detection of ductal rupture. CCT has been shown to be a useful technique for visualizing the pancreatic parenchyma and identifying and localizing pancreatic fluid collections (4). The site of fluid collections seen on CCT may be indicative of the location of DP rupture. In our patient, CCT performed in the emergency department helped us to diagnose the rupture.

Initial management of DP rupture or fistula complications (pleural effusions or pancreatic ascites) includes prolonged pancreatic rest and endoscopic placement of stents in the pancreatic duct (9). Conservative treatment includes recommendations such as adequate drainage of pancreatic secretions and optimal nutritional support during the recovery period to prevent or control sepsis. If the patient becomes septic, aggressive treatment and correction of electrolyte disturbances are required. Total parenteral nutrition therapy can be applied to rest the pancreas and reduce exocrine pancreatic secretions. Treatment may include total parenteral nutrition, jejunal feeding, or medications such as anticholinergics, carbonic anhydrase inhibitors, calcitonin, glucagon, octreotide or somatostatin(4). Our patient was treated conservatively with proton pump inhibitors, antibiotherapy, and intravenous fluid therapy.

Conclusion

The diagnosis of ruptured ductus pancreaticus is a rare entity in the emergency department. Early diagnosis may allow treatment before the patient's clinical condition becomes complicated. Epigastric pain and low level of enzyme elevation should be a warning in this regard. We thought that our case may be noteworthy for clinicians working in the emergency department because it allows early diagnosis and treatment.

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