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A Rare Case of Pancreatic Ascites Secondary to Chronic Pancreatitis

Fisayo Kayode-Ajala, MD/MPH[1], Shruti Jesani, MD[1], Chidinma Ejikeme, MD[1], Henna Asrar, MD[2], and Muniba Naqi, MD, MBA, CHCQM, CPE, FACP[1]

Abstract

Pancreatic ascites refer to continuous leakage of pancreatic secretions in the peritoneum leading to accumulation of pancreatic fluid in the peritoneal cavity. Although literature on the incidence of pancreatic ascites and presenting signs and symptoms is scarce, it may be seen in patients with chronic alcoholic pancreatitis. Patients typically present with acute chronic pancreatitis and new-onset ascites, with or without abdominal pain. A diagnostic paracentesis is usually the first step to determine the etiology of the ascites. Mild cases may resolve with conservative management. Optimization of nutrition status is an important factor to reduce morbidity and mortality. More severe cases or cases refractory to conservative management may require endoscopic or surgical intervention. This case report describes a rare presentation of pancreatic ascites in a 35-year-old female.

Keywords

ascites, chronic pancreatitis, paracentesis, alcohol abuse, pancreatic pseudocyst

Introduction

Pancreatic ascites is a rare disease that results from damage to the pancreatic duct. Literature to date is limited but suggests that the prevalence of pancreatic ascites is approximately 3.5% in patients with chronic pancreatitis.[1] Other etiologies include pancreatic pseudocyst, acute traumatic pancreatitis, and endoscopic ultrasound with fine-needle aspiration of the pancreas. An estimated 10% remain idiopathic. Damage to the pancreatic duct leads to continuous leakage of pancreatic fluid into the peritoneal cavity. While mild cases of pancreatic ascites resolve spontaneously, severe cases of persistent pancreatic ascites accumulation result in a higher rate of complications leading to increased morbidity and mortality in affected patients. Here, we describe such a case.

Case Presentation

Our patient is a 35-year-old Hispanic female with a past medical history of chronic pancreatitis, alcoholic liver disease, and alcohol abuse who presented to the emergency department with a 3-day history of 10/10 diffuse abdominal pain radiating to the thorax. One week prior to presentation, she was admitted and discharged for similar complaints. Investigations during the previous admission revealed acute chronic pancreatitis with pancreatic pseudocyst and necrosis. Of note, she is a current smoker with a 16-pack year history and admits to consuming 1 to 2 glasses of alcohol daily for several years but stated she has been abstinent from alcohol ingestion for 1 month prior to presentation. She denied any history of illicit drug use.

Vital signs on presentation included blood pressure (BP) 91/55, pulse 114 bpm, respiratory rate (RR) 18, T 98.1°F and 99% saturation on room air. On physical examination, the abdomen was markedly distended and diffusely tender to palpation in all quadrants. A positive fluid wave and voluntary guarding was noted. Pertinent lab findings include leukocytosis with white blood cell (WBC) 16.9 (4.8-10.8 K/μL), hemoglobin 10.3 (12.0-16.0 g/dL), sodium 130 (136-144 mmol/L), lipase 149 (22-51 μ/L), and liver function tests within normal limits.

[1]Robert Wood Johnson Barnabas Health/Trinitas Regional Medical Center, Elizabeth, NJ, USA
[2]St. George's University, Grenada, West Indies

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Corresponding Author:
Fisayo Kayode-Ajala, MD/MPH, Internal Medicine Residency Program, Robert Wood Johnson Barnabas Health/Trinitas Regional Medical Center, 225 Williamson St, Elizabeth, NJ 07202, USA.
Email: fisayo.kay@gmail.com

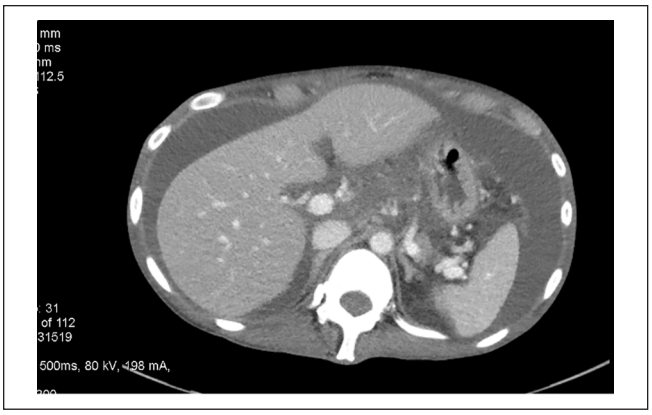

Image 1. CT abdomen demonstrating abdominal ascites.

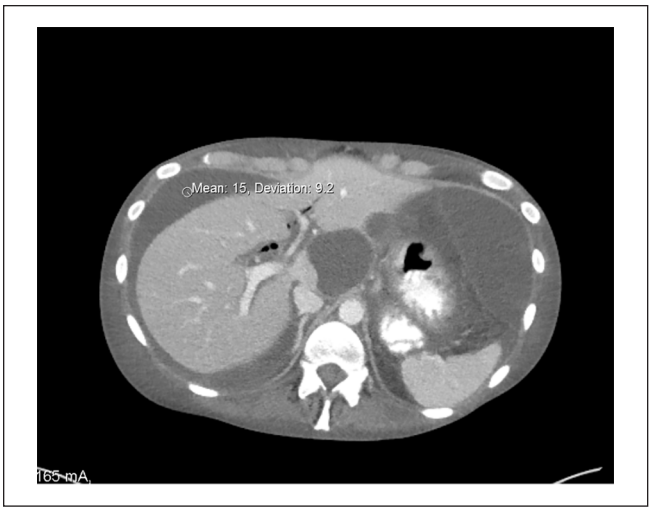

Image 2. CT abdomen showing pancreatic pseudocyst.

Repeat computed tomographic (CT) abdomen and pelvis with contrast compared with admission 1 week prior revealed a significant increase in ascites along with multiple pancreatic cysts, and a 10 mm fluid density lesion at the neck of the pancreas and enlarged peripancreatic fluid.

Approximately 2 L of ascitic fluid was removed via paracentesis. Fluid analysis demonstrated the following: WBC 8423, red blood cell (RBC) 1570, polymorphonuclear (PMN) 82%, protein 3.2, amylase 5507, lactate dehydrogenase (LDH) 478, serum-ascites albumin gradient (SAAG) 0.1. An endoscopic retrograde cholangiopancreatography (ERCP) was done, and a 5 Fr by 9 cm plastic stent was placed 7 cm into the ventral pancreatic duct.

Post ERCP, the patient's clinical condition deteriorated. She was transferred to the intensive care unit (ICU) due to clinical evidence consistent with acute respiratory distress syndrome. Upon being transferred to the ICU, her arterial blood gas (ABG) showed pH: 7.36, PCO_2: 30, PO_2: 50, HCO_3: 18.8 on 100% FiO_2 on a non-rebreathing mask. In the ICU, she required vasopressor support intermittently to maintain a mean arterial pressure (MAP) of 65. Her hospital course was further complicated with progression to septic shock. Laboratory data demonstrated leukocytosis ranging from 16.9 to 69.2K/μL during her stay. Despite multiple attempts to isolate a microbial organism as a source for sepsis, all infectious workup including ascitic fluid culture, blood urine, and endotracheal tube (ET Tube) culture results demonstrated no growth. She was initially placed on ceftriaxone 2000 mg for spontaneous bacterial peritonitis (SBP) treatment but was switched to IV meropenem (1000 mg every 8 hours) for 24 days due to persistent leukocytosis. Serial CT scans of the abdomen with contrast were performed to monitor progression of the disease. Post ERCP imaging did not demonstrate postprocedure worsening of pancreatitis. She was intubated for acute respiratory failure, and eventually needed a tracheostomy due to prolonged need for mechanical ventilation. Subsequently, a percutaneous endoscopic gastrostomy (PEG) feeding tube was inserted.

She received 3 additional paracentesis procedures for recurrent ascites in which 4, 3, and 2 L was removed consecutively, all of which were consistent with pancreatic ascites. She eventually required placement of 2 Jackson-Pratt drains. An interval CT of the abdomen showed an increase in size of the pancreatic pseudocyst, from 2.7 to 5.8 cm, as well as loculated mesenteric collections which raised the concern for serosal implants. Surgical consultation was requested, but due to the patient's fragile clinical status, surgical intervention was not advised. Hyperdense material suggestive of blood products was also seen in the dependent portions of the pseudocyst. This radiographic finding in conjunction with a consistent decline in hemoglobin levels raised concern for hemorrhagic pancreatitis.

Interventional radiology drained the pseudocysts which yielded 1.6 L of dark purulent fluid without growth of any microorganisms. The patient's hemoglobin stabilized to 8.5 g/dL from 6.0 g/dL, and subsequent CT abdomen scans showed resolving pseudocyst collections and pancreatic edema.

The patient subsequently developed bilateral pleural effusions. A thoracentesis was performed with approximately 600 mL of fluid removed. Fluid analysis revealed a transudative pattern demonstrated by WBC 664/mm^3, RBC 1580/mm^3, PMN 62%, glucose 81 mg/dL, protein 2.2 g/dL, LDH 232 μ/L. Her course was further complicated by *Pseudomonas aeruginosa* health care–associated pneumonia for which she completed a course of IV meropenem. *Clostridium difficile*–associated diarrhea was treated with oral vancomycin (500 mg every 6 hours) and IV metronidazole (500 mg every 8 hours). Once clinically stabilized, the patient was transferred to the general medical ward for continued treatment.

On day 51, she was successfully weaned off the ventilator and transitioned to a trach collar. Prior to discharge, she was noted to have significant improvement in her symptoms and

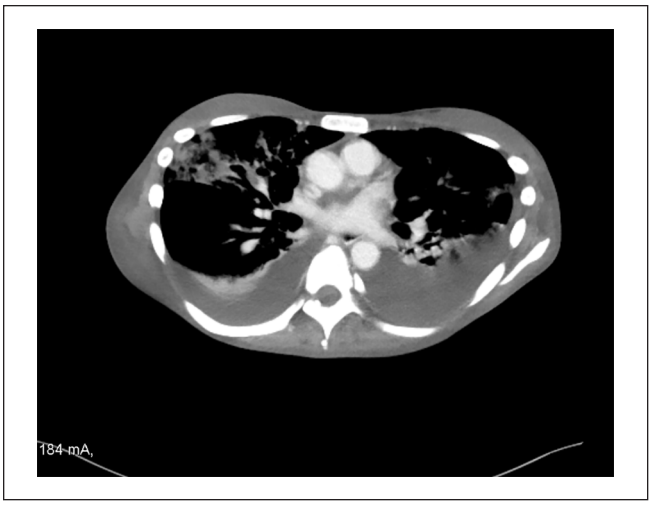

Image 3. CT abdomen showing bilateral pleural effusions.

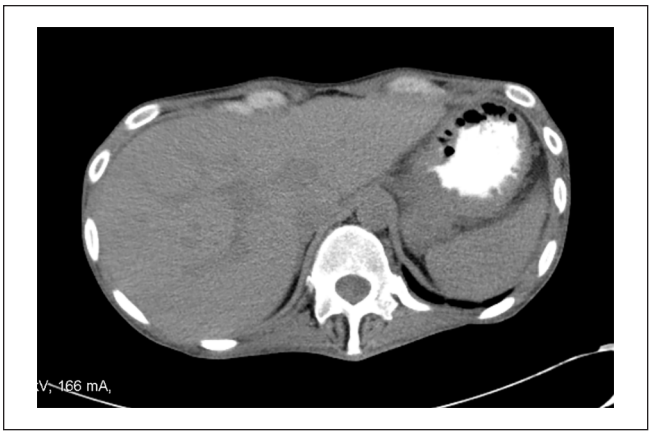

Image 4. CT abdomen showing improvement and resolution of symptoms prior to discharging patient to subacute rehab.

was tolerating oral feeds. She underwent a second ERCP for the common bile duct (CBD) stent removal and PEG tube removal. The patient spent a total of 102 days in the hospital and was eventually discharged to a subacute rehabilitation facility on 1.5 LPM supplemental oxygen via nasal cannula.

Discussion

Pancreatic ascites may occur as a rare sequela of chronic pancreatitis secondary to alcohol consumption. Ascites results from portal hypertension due to liver disease or due to biliary etiology.[1] Roughly 4% of ascites is due to pancreatic origin.[2] Patients generally present with vague symptoms such as abdominal pain, sense of abdominal fullness, and increased abdominal girth. In most cases, patients do not present with inflammatory signs.[3] It is important to obtain a comprehensive history and perform a thorough physical examination, to avoid a delayed or missed diagnosis.

Although a diagnostic challenge, a paracentesis can assist with identification of pancreatic ascites. Often, the fluid analysis will show an amylase greater than 1000 IU/L, total protein greater than 3 g/dL, and SAAG less than 1.1 g/dL.[2,3] In this case, our patient had an amylase of 5570 IU/L, total protein of 3.2 g/dL, and a SAAG of 0.1 g/dL. The SAAG allows for differentiation between ascites due to portal hypertension versus other etiologies such as tuberculosis and malignancy. If clinically indicated, an ERCP can be performed to exclude obstructive pathology.

Pancreatic pseudocysts are a complication of chronic pancreatitis. They can result in fistula formation, which can lead to ascites. In addition, the fibrinous wall of the pseudocyst can obstruct the pancreatic duct leading to fistula tract formation.[3,4]

The management of pancreatic ascites may be conservative or invasive depending on the individual clinical scenario. The conservative approach includes making the patient nil-per-os and managing with total parenteral nutrition and electrolyte replenishment as needed. Somatostatin analogues and diuretics may be added to the management to impede pancreatic exocrine function.[2] This approach requires repeat paracentesis on an as-needed basis over a period of 3 to 4 weeks. If there is no clinical improvement after 4 weeks, an interventional approach may be used.[2]

For refractory or more severe cases, endoscopic or surgical intervention may be considered. Trans-papillary endoscopic drainage and stent placement is an effective approach in relieving ductal obstruction. One study demonstrated that endoscopic drainage is a safe and effective management in treating pancreatic ascites, especially in the setting of chronic pancreatitis.[4] This method allows for drainage through the pancreatic duct sphincter and decreases ductal pressure.[3,5] The result is healing of ductal obstruction and resolution of the ascites. However, if there is a complete ductal obstruction, this method is not sufficient.[3,5] In this case, a surgical approach may be preferred based on the location of the lesion. For distal duct obstructions, pancreatectomy may be a suitable option. At the same time, pancreatojejunostomy is implemented for the treatment of proximal duct obstruction.[3] Any surgical intervention carries risks which need to be weighed against the benefits and communicated to patients so they may make an informed decision. The least invasive approach to optimize outcomes with the highest benefit to risk ratio is preferred and merits consideration.

While it may be a diagnostic challenge, pancreatic ascites has a good prognosis when diagnosed early, especially when treated with an endoscopic approach.[3] Conservative management has a high failure rate of between 40% and 60% and associated mortality rate of about 17%, whereas endoscopic drainage has shown successful recovery in up to 82% of cases with significantly lower mortality rates.[6] It should be noted that the incidence of post ERCP pancreatitis ranges from 3% to 14%.[7] When using a conservative approach, it is imperative to manage electrolyte imbalances and avoid exacerbation of the disease and further complications. Surgical intervention should only be considered in severe cases in

which there is complete ductal obstruction, or when other treatment modalities have failed.

Conclusion

Pancreatic ascites is a very serious and rare complication of chronically recurring alcohol-related pancreatitis. Appropriate management is essential to decrease fatal complications associated with pancreatic ascites.

Declaration of Conflicting Interests

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Ethics Approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed Consent

Verbal informed consent was obtained from the patient for their anonymized information to be published in this article

ORCID iD

Chidinma Ejikeme https://orcid.org/0000-0002-1989-9779

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