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Not Your Typical Case Of Ascites: Pancreatic Ascites In A Patient With Cirrhosis And Pancreatic Duct Leak

Philip Montemuro, MD
Thomas Jefferson University

Abhik Roy, MD
Thomas Jefferson University

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Philip Montemuro, MD and Abhik Roy, MD

Case

A 55-year-old male with a history of hepatic cirrhosis secondary to Hepatitis C and alcohol abuse presented to an outside hospital with progressive abdominal pain and distension. The patient initially complained of "punching" right upper quadrant and epigastric abdominal pain that was 10/10 in intensity and non-radiating. Although the pain had started one to two days prior to presentation, the patient had been experiencing several weeks of increasing abdominal distension. He reported drinking eight 40oz beers daily for over 40 years, and he felt that the abdominal pain improved slightly with cessation of alcohol use. The patient complained of nausea and non-bloody, non-bilious vomiting, progressive dyspnea on exertion, and worsening lower extremity edema. He denied fevers or chills.

Limited outside hospital records indicated that the patient had been stable on diuretics for one month prior to admission. He required three large volume paracenteses during his six days at the outside facility, and he was transferred to Thomas Jefferson University Hospital for further management of his refractory ascites. Medications on transfer included lactulose, furosemide, spironolactone, and octreotide. The patient was also receiving total parenteral nutrition (TPN) via a Peripherally Inserted Central Catheter (PICC).

At the time of transfer, the patient was afebrile with stable vital signs. On physical exam, he appeared cachectic with temporal wasting. The abdominal exam revealed a soft abdomen with moderate distension, diffuse tenderness to palpation without rebound or guarding, positive fluid wave and shifting dullness, and no organomegaly or collateral circulation. Spider angiomata were present on the chest. Lower extremity edema, palmar erythema, jaundice, and gynecomastia were not appreciable.

Complete blood count on transfer showed white count 10,900/L, hemoglobin 9.1 g/dL, and platelets 110/L. The chemistry 7 was remarkable for sodium 133 mmol/L and creatinine 0.7 mg/dL. INR was 1.4, and liver function tests showed total protein 5.0 g/dL, albumin 1.9 g/dL, total bilirubin 0.7 mg/dL, direct bilirubin 0.3 mg/dL, AST and ALT of 17 IU/L, and alkaline phosphatase 38 IU/L. Serum lipase and amylase were 645 U/L and 330 U/L respectively. Hepatitis C antibody was positive with a quantitative polymerase chain reaction (PCR) of 5130 IU/mL.

On the day of transfer, a bedside paracentesis had been performed at the outside hospital and showed clear yellow fluid with 94 red blood cells, 69 white blood cells (15% PMN, 50% lymphocytes, and 35% monocytes), and a negative gram stain and culture. Ascites albumin was 0.7 g/dL, yielding a serum-ascites albumin gradient (SAAG) of 1.2. Of note, the ascites total protein was 2.1 and ascites amylase was significantly elevated at 4000. An

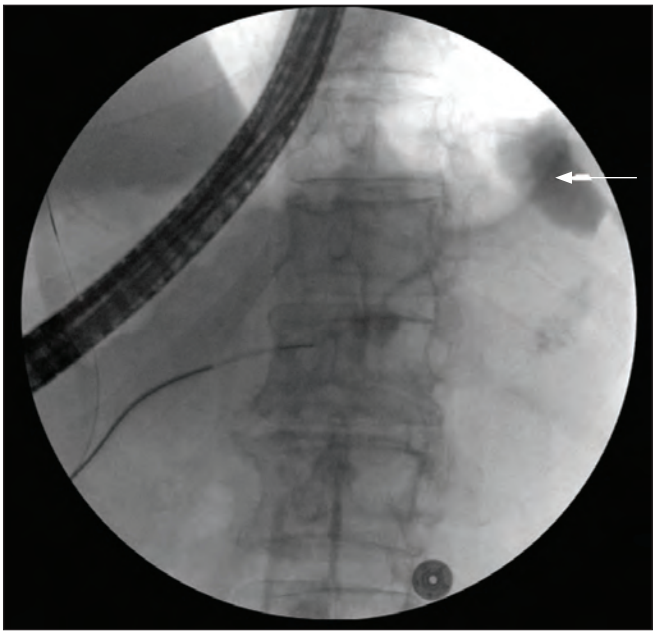

Figure 1. *Pancreatic duct leak with contrast extravasation into the retroperitoneum (white arrow)*

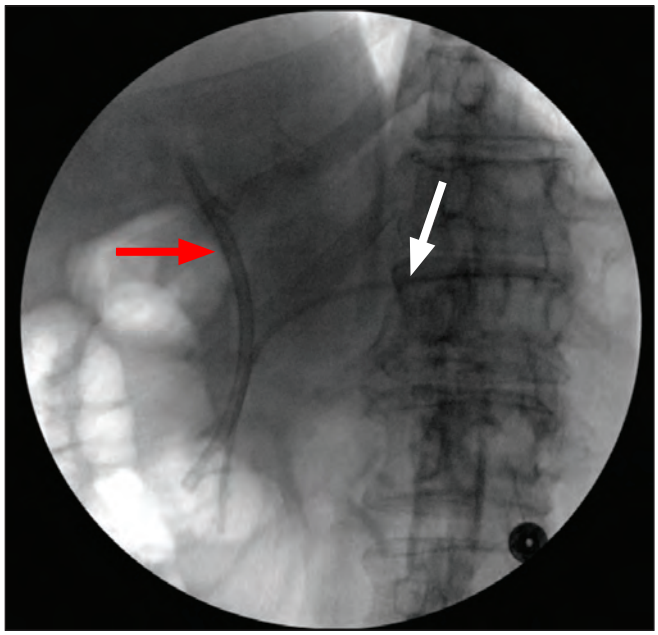

Figure 2. *Two stents were placed to fix the pancreatic duct leak and impaired biliary drainage (the white arrow shows the pancreatic duct stent; the red arrow shows the biliary duct stent)*

Table 1		
SAAG ≥ 1.1, Ascites protein < 2.5 g/dL	**SAAG ≥ 1.1, Ascites protein ≥ 2.5 g/dL**	**SAAG < 1.1**
Cirrhosis	Congestive heart failure Constrictive pericarditis	Biliary leak Nephrotic syndrome
Late Budd-Chiari syndrome	Early Budd-Chiari syndrome IVC obstruction	Pancreatitis Peritoneal carcinomatosis
Massive liver metastasis	Sinusoidal obstruction syndrome	Tuberculosis

abdominal magnetic resonance imaging (MRI)/magnetic resonance cholangiopancreatography (MRCP) had also been performed prior to transfer, and this study demonstrated a cirrhotic appearing liver, moderate to severe ascites with loculated appearance, and mild stenosis of the common hepatic duct with no intra- or extra-hepatic biliary ductal dilation or stones and no evidence of pancreatic pseudocyst.

Given the rapid re-accumulation of ascites requiring multiple paracenteses at the outside hospital, the differential diagnosis at the time of transfer to Jefferson included progression of cirrhosis due to alcohol abuse or Hepatitis C, pancreatic ascites, hepatocellular carcinoma or metastatic liver disease, peritoneal carcinomatosis, Budd-Chiari syndrome, and auto-immune pancreatitis. Shortly after transfer, an abdominal Doppler ultrasound ruled out Budd-Chiari syndrome and detailed serum and ascitic fluid analysis (including cytology) led to the conclusion that most malignant and auto-immune etiologies were less likely. Given the SAAG ≥1.1 and high ascites amylase level, progressive cirrhosis (secondary to alcohol abuse and Hepatitis C) as well as a pancreatic etiology for ascites were believed to play a key role.

An endoscopic retrograde cholangiopancreatography (ERCP) was performed to evaluate the pancreas and pancreatic duct architecture. ERCP revealed a large area of disruption of the major pancreatic duct (MPD) in the mid-body of the pancreas with extravasation of contrast into the retroperitoneum **(Figure 1),** white arrow illustrates contrast extravasation). This finding could certainly have explained the high amylase content in the ascitic fluid. In order to repair the pancreatic duct leak, a 5 French 12 cm Geenen stent was placed in the MPD across the area of mid-body pancreatic duct disruption **(Figure 2, white arrow)**. Of note, a 10 French 5 cm plastic stent was also placed in the biliary duct because there appeared to be impaired drainage of contrast from the biliary tree **(Figure 2, red arrow)**.

Following the ERCP, the patient unfortunately developed chemical peritonitis (secondary to contrast injection during the ERCP procedure), which required prolonged antibiotic therapy. The patient was placed on a low sodium diet as well as a stable oral diuretic regimen. The combination of endoscopic, pharmacologic, and dietary interventions ultimately decreased the rate of re-accumulation of ascites. A repeat paracentesis performed prior to discharge showed a dramatic decrease in the ascites amylase level, demonstrating resolution of the pancreatic duct leakage which had contributed to the patient's ascites.

Discussion

In a patient with ascites of unclear etiology, several key points must be remembered. The most common causes of ascites are cirrhosis (81%), malignancy (10%), and heart failure (3%).[4] The work-up should include history and physical exam, ultrasound, and paracentesis.[5]

On physical exam, the most useful findings to suggest ascites include positive fluid wave and shifting dullness, while the absence of bulging flanks, flank dullness, or shifting dullness are the strongest findings arguing against ascites.[3] Paracentesis and ascitic fluid analysis are essential for establishing a diagnosis and to rule out an infection. Routine ascitic fluid studies should include gross appearance of the fluid, total protein, albumin, cell count and differential, as well as gram stain and culture.[1,4] Other tests to consider include fluid amylase, cytology, and many other less common studies that can be used when clinically indicated.[1] Calculating the serum-ascites albumin gradient (SAAG score) is key for establishing a diagnosis (see Table 1).[1,4,5]

Pancreatic ascites causes approximately 1% of all cases of ascites.[4] The etiology is due to disruption of the main pancreatic duct causing pancreatic fluid accumulation in the peritoneal cavity.[2] It is most commonly associated with chronic pancreatitis secondary to alcoholism, although 67% of patients have no recent pancreatitis flare.[6,7] It can also be caused by an internal fistula between the pancreatic duct and peritoneal cavity, a leaking pseudocyst, or a ruptured duct.[2] Pancreatic ascites is seen in 4% of chronic pancreatitis cases and 6-14% of pseudocyst cases.[8,9] Clinically, it can present in patients with a history of chronic pancreatitis, recent acute pancreatitis, or new onset ascites.

The diagnosis of pancreatic ascites is made by an elevated serum amylase along with ascitic fluid amylase > 1000 IU/L, SAAG <1.1, and total protein >3.[1,2] In our patient, the SAAG and total protein were not consistent with a purely pancreatic cause given the known cirrhosis that was also contributing to his ascites. Imaging that can be utilized for diagnostic purposes include computed tomography (CT) scan to evaluate for a pseudocyst or evidence of pancreatitis as well as MRCP or ERCP. MRCP is utilized if the patient is not an ERCP candidate to examine the

main pancreatic duct. ERCP is favored as it is both diagnostic and therapeutic.[2,10] As was the case for our patient, ERCP can show passage of contrast from either the MPD or pseudocyst into the peritoneal cavity. If this finding is present, a stent can be placed to occlude the site of disruption. Treatment includes both temporary and permanent options.[2] The temporary measures are targeted at reducing pancreatic activity. This involves avoiding enteral feedings and utilizing naso-gastric suctioning and TPN when indicated. Octreotide is also effective in reducing pancreatic secretions. Permanent measures include ERCP with stent placement or surgical interventions if this fails.[2]

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"Monet-Dusk in Venice"
Painting by Nick Zaorsky, MD