Acute Obstructive Suppuration of the Pancreatic Duct Causing Sepsis

Stanley Yakubov, MD,[1] Jack Braha, DO,[2] Joel Albert, MD,[2] Rabin Rahmani, MD,[2] Ira Mayer, MD,[2] and Yuriy Tsirlin, MD[2]

[1] Department of Medicine, Maimonides Medical Center, Brooklyn, NY.

[2] Division of Gastroenterology, Department of Medicine, Maimonides Medical Center, Brooklyn, NY.

Objective: We report a rare case of acute obstructive suppuration of the pancreatic duct causing sepsis, which was successfully treated with emergent endoscopic retrograde cholangiopancreatography (ERCP).

Methods: We describe the patient's clinical presentation, laboratory test results, and imaging used for diagnosis and treatment.

Results: A 33-year-old female with a history of recurrent acute pancreatitis was admitted during an episode of acute pancreatitis. Computed tomography (CT) scan of the abdomen revealed acute pancreatitis, diffuse pancreatic atrophy and pancreatic ductal dilatation with obstruction due to a soft tissue lesion within the distal duct. Shortly after admission she developed symptoms and signs of sepsis. Urgent ERCP was performed to further assess the suspected cholangitis. "Clean" bile emanated from the common bile duct, while copious purulent fluid was detected at the dilated pancreatic duct orifice, confirming suppuration of the pancreatic duct. A plastic single pigtail stent was placed traversing the ampulla and pancreatic duct stones that were causing the obstruction, which were later removed. After endoscopic decompression, the patient rapidly improved over the following 24 hours and had no subsequent admissions for pancreatitis.

Conclusion: Acute suppuration of the pancreatic duct (ASPD) is a rare and potentially fatal infectious complication of pancreatic ductal obstruction with few cases reported in the English literature. It would be of interest to further investigate the exact pathophysiology leading to development of ASPD. The endoscopic methods of urgent ERCP and pancreatic duct decompression utilized in our case proved effective in successfully treating ASPD. This unusual condition should be considered in patients with acute pancreatitis who develop early clinical decompensation.

INTRODUCTION

Pancreatic abscess, necrosis, and pseudocyst are well-known entities that can complicate pancreatitis and lead to pancreatic sepsis (Sarr et al., 2013; Deeb et al., 2008). Acute obstructive suppurative cholangitis is a common clinical entity. However, pancreatic duct (PD) obstruction and acute suppuration of the pancreatic duct (ASPD) leading to sepsis, without a concurrent pancreatic abscess or infected pseudocyst, is an extremely rare complication with few cases reported in the English literature (Fujimori et al., 2011; Tajima et al., 2006). In ASPD, pancreatic obstruction results in the development of infection and pus within the PD, leading to sepsis. Here, we report a case of ASPD successfully treated with emergent endoscopic retrograde cholangiopancreatography (ERCP).

CASE

A 33-year-old female with a past medical history of recurrent acute pancreatitis, likely secondary to tropical pancreatitis, presented with a one-day history of severe epigastric pain radiating to the back. Pain was sharp, 7/10 in intensity, and associated with nausea. No fever or chills were reported. She denied alcohol consumption, smoking, illicit, herbal or prescription drug use, or any other significant medical history. She reported multiple episodes of pancreatitis in the past and had previously undergone ERCP and cholecystectomy at an outside hospital.

On presentation, the patient was afebrile, blood pressure (BP) 137/90 mm Hg, heart rate 70 beats per min, respiration rate 18 breaths per min, and body mass index of 19.8 kg/m^2. On physical examination her abdomen was soft, non-distended, tender in the epigastric area, with guarding but no rebound tenderness. Normal bowel sounds were present. The remainder of the physical examination was unremarkable. Laboratory testing revealed a white blood cell (WBC) count of 8.3 K/μL and normal pancreatic enzymes with an indirect hyperbilirubinemia (1.6/0.2 mg/dL). Ultrasound of the abdomen demonstrated common bile duct (CBD) dilatation of 7 mm, consistent with a post-cholecystectomy state. Computed tomography (CT) scan of the abdomen, with per os and intravenous (IV) contrast, revealed acute pancreatitis (AP), diffuse pancreatic atrophy and pancreatic ductal dilatation (Figure 1) with obstruction due to a soft tissue lesion within the distal duct (Figure 2). The patient was clinically stable upon admission and was treated with bowel rest, pain management and aggressive IV hydration.

Within the next 24 hours, the patient developed leukocytosis (WBC 15.5 K/μL, neutrophil count 92.4%), hypotension (BP 90/51 mm Hg), fever (maximum temperature 101.2° F) and chills. Obtained blood cultures showed no growth after five days. Emergent ERCP was performed. The major ampulla appeared edematous and a previous sphincterotomy was noted. The CBD was cannulated, nonpurulent bile ema-

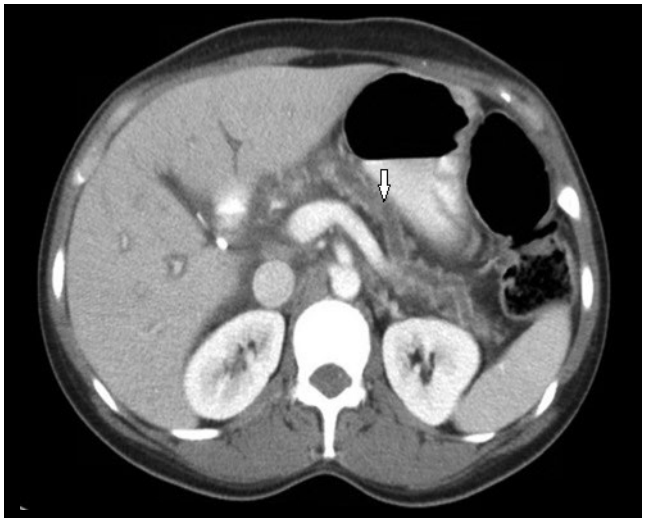

Figure 1 | Diffuse pancreatic ductal dilatation (arrow).

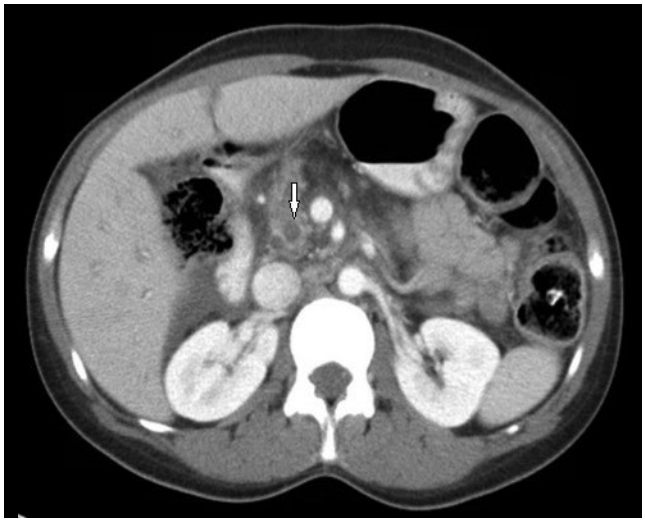

Figure 2 | Soft tissue lesion (arrow) within the distal duct.

nated and cholangiogram did not reveal biliary obstruction. After pancreatic ductal cannulation, copious purulent fluid emanated from the orifice (Figure 3); however, due to the viscosity of the purulent fluid it could not be aspirated or cultured. A pancreaticogram revealed diffuse PD dilatation and multiple filling defects, consistent with stones. A plastic single pigtail stent was placed traversing the ampulla and PD filling defects. Purulent fluid drained from the stent, and was followed by clear pancreatic fluid. The patient rapidly improved over the following 24 hours. Prior to discharge, she underwent ERCP with pancreatic ductal sphincterotomy, multiple stone extraction (Figure 4), and pancreaticogram, which revealed no further filling defects. The remainder of the patient's hospital course was uneventful and she was discharged home. She followed up with the outpatient clinic and had no subsequent re-admissions over the next twelve months.

DISCUSSION

Complications of acute pancreatitis are well known and include pseudocyst formation, pancreatic necrosis, and multisystem organ failure (Sarr et al., 2013; Deeb et al., 2008). Acute pancreatitis is also a risk factor for the development of chronic pancreatitis. Patients with chronic pancreatitis may suffer from chronic pain, exocrine deficiency and diabetes (Forsmark, 2013). Acute suppuration of the pancreatic duct is a rare but significant complication of chronic pancreatitis. ASPD develops due to obstruction of the pancreatic duct resulting in fluid and bacterial stasis (Deeb et al., 2008). Similar to ascending cholangitis, the pancreatic duct fills with pus and the patient quickly decompensates. The treatment of choice is resuscitation, broad-spectrum antibiotics and emergent endoscopic decompression (Fujimori et al., 2011; Tajima et al., 2006).

Risk factors for the development of ASPD have been suggested in the literature. A single culprit microorganism was identified in each of the three previously reported cases. The microorganisms were *Klebsiella ornithinolytica* (Deeb et al., 2008), *Stenotrophomonas maltophilia* (Fujimori et al., 2011), and *Escherichia coli* (Tajima et al., 2006). While Weinman reported a case of ASPD, the culprit microorganism was not identified (Weinman, 1995). Risk factors thought to contribute to the development of ASPD in these previously reported cases included diabetes, post-transplant immunosuppression, and malignancy. Although conclusive evidence of bacterial infection via positive blood cultures is not available in this case, we strongly believe the patient's clinical presentation of fever, hypotension, leukocytosis, and purulent discharge from the pancreatic duct, and her improvement after intravenous antibiotics and emergent ERCP, suggest a bacterial etiology that contributed to sepsis.

Marotta et al. noted that patients with chronic pancreatitis were more susceptible to infections, likely due to impaired antibacterial activity of the pancreatic secretions, as compared to those with a normal pancreas (Marotta et al, 1997).

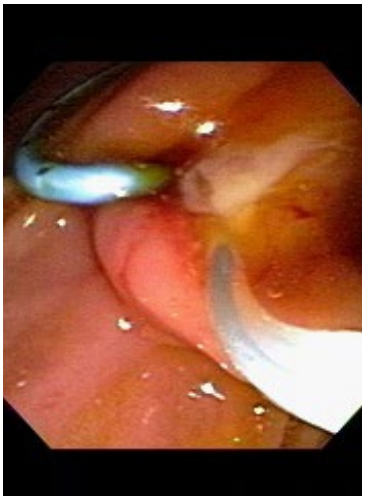

Figure 3 | Pus traversing the pancreatic duct orifice.

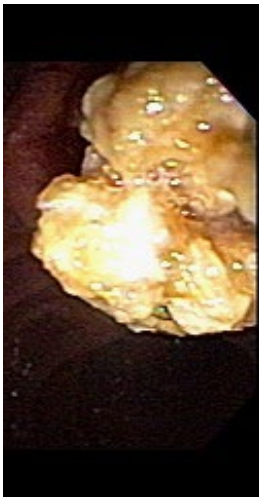

Figure 4 | Large stones from the pancreatic duct.

In 1968, Williams and Byrne demonstrated that injection of bacteria into the pancreatic duct was not sufficient by itself to cause pancreatitis (Williams & Byrne, 1968). Tropical pancreatitis (a type of chronic pancreatitis), history of prior sphincterotomy, and intraductal pancreatic stones (especially if they are also obstructing the outflow of the duct), contribute to ductal infection. Duodenal contents may reflux into the biliary tree or pancreatic duct after sphincterotomy, particularly if the patient has a common biliary and pancreatic sphincter (Weinman, 1995). In the case presented here, risk factors that likely contributed to the development of ASPD included history of prior sphincterotomy, chronic pancreatitis, and pancreatic ductal obstruction by a calculus.

ASPD is a rare and potentially fatal infectious complication of pancreatic ductal obstruction. This unusual condition should be considered in patients with acute pancreatitis who develop early clinical decompensation. It would be of interest to further investigate the exact pathophysiology leading to development of ASPD. As in this case, emergent ERCP and PD decompression are essential in the successful treatment of ASPD.

Corresponding Author: Stanley Yakubov, MD (syakubov@maimonidesmed.org).

Author Contributions: SY and JB researched data and wrote the manuscript. JA, RR, IM and YT contributed to the discussion and reviewed/edited the manuscript.

Acknowledgements: The authors disclose that this case was presented as a poster at ACG 2013 Annual Scientific Meeting and Postgraduate Course. Funding and financial support was not applicable in the writing of this article.

Disclosure: The authors have completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. No conflicts were noted.

References

Deeb, L.S., Bajaj, J., Bhargava, S., Alcid, D., & Pitchumoni, C.S. (2008). Acute Suppuration of the Pancreatic Duct in a Patient with Tropical Pancreatitis. *Case Rep Gastroenterol, 2*(1), 27-32.

Forsmark, C.E. (2013). Management of chronic pancreatitis. *Gastroenterology, 144*(6), 1282-1291.

Fujimori, N., Igarashi, H., & Ito, T. (2011). Acute obstructive suppurative pancreatic ductitis. *Clin Gastroenterol Hepatol, 9*(8), A28.

Marotta, F., Tajiri, H., Li, Z.L., Barreto, R., Bellini, O., & Barbi, G. (1997). Pure pancreatic juice from patients with chronic pancreatitis has an impaired antibacterial activity. *Int J Pancreatol, 22*(3), 215-220.

Sarr, M.G., Banks, P.A., Bollen, T.L., Dervenis, C, Gooszen, H.G., Johnson, C.D., Tsiotos, G.C., & Vege, S.S. (2013). The new revised classification of acute pancreatitis. *Surg Clin North Am, 93*(3), 549-562.

Tajima, Y., Kukori, T., Susumu, S., Tsutsumi, R., Kitasato, A., Adachi, T., Mishima, T., & Kanematsu, T. (2006). Acute suppuration of the pancreatic duct associated with pancreatic ductal obstruction due to pancreas carcinoma. *Pancreas, 33*(2), 195-197.

Weinman, D.S. (1995). Acute Suppuration of the Pancreatic Duct. *Gastrointest Endosc, 41*(3), 268-270.

Williams, L.F., Jr, & Byrne, J.J. (1968). The role of bacteria in hemorrhagic pancreatitis. *Surgery, 64*, 967-972.