

Gangrenous cholecystitis complicating pancreatic head adenocarcinoma: a case report

Ammar Kayyali¹, Anis Toumeh¹, Yuying Jiang², Wael Youssef¹

¹Department of Internal Medicine, the University of Toledo Medical Center, Toledo, OH, USA; ²Department of Pathology, the University of Toledo Medical Center, Toledo, OH, USA

Corresponding to: Anis Toumeh, M.D. Internal Medicine, University of Toledo Medical Center, 3000 Arlington Ave, Toledo, OH 43614, USA. Email: anistoumeh@gmail.com.

Abstract: Gangrenous cholecystitis (GC) is a serious complication of acute cholecystitis. Preoperative diagnosis of this condition maybe difficult and often entertained after deterioration of a patient suspected of having simple or uncomplicated cholecystitis. It was previously demonstrated that 39.8% of patients presenting with acute cholecystitis were found to have gangrenous transformation. To our knowledge, this condition has never been reported in the English literature as a complication of pancreatic adenocarcinoma. This report demonstrates the importance of considering gangrenous cholecystitis as a potential cause of acute illness in the setting of pancreatic adenocarcinoma.

Key Words: Gangrenous cholecystitis; pancreatic head adenocarcinoma



Submitted Jan 25, 2013. Accepted for publication Feb 18, 2013.

doi: 10.3978/j.issn.2224-4778.2013.02.01

Scan to your mobile device or view this article at: <http://www.amepc.org/tgc/article/view/1513/2841>

A 57-year old chronic smoker, chronic alcoholic male presented with a one week history of worsening right upper quadrant sharp abdominal pain, vomiting, dark urine, pale stools and yellowish discoloration of the skin. On physical examination he was hemodynamically stable, jaundiced and icteric sclera was noted. Abdominal exam was significant for diffuse tenderness and moderate distention without peritoneal signs. Laboratory investigations showed a white blood cell (WBC) count of 31 Thou/mm³, total bilirubin of 15 mg/dL, direct bilirubin of 10 mg/dL, alkaline phosphatase of 302 IU/L, ALT of 142 IU/L, AST of 104 IU/L and Albumin of 3 g/dL. Abdominal CT showed dilated intra and extra hepatic biliary ducts (up to 13 mm) with no obstructing lesions. He was started on antibiotics for presumed ascending cholangitis. Urgent diagnostic endoscopic ultrasound with FNA was performed and demonstrated a 15.0 mm × 11.7 mm hypoechoic poorly defined pancreatic head mass infiltrating the distal common bile duct (CBD) and involving the duodenum. Final pathology report confirmed the diagnosis of pancreatic ductal adenocarcinoma (*Figures 1,2*). ERCP was

attempted but cannulation of CBD was unsuccessful. On day 2 of admission percutaneous drainage by interventional radiology was achieved. Despite good drainage, and broad antibiotic coverage, patient's condition deteriorated. He became hypotensive with worsening leukocytosis and developed acute kidney injury, later necessitating hemodialysis. For this he was taken to the operating room for surgical decompression which revealed a distended necrotic gallbladder (GB). A T-tube was then placed in the CBD with good bile drainage. Pathology later demonstrated extensive gangrenous cholecystitis with full thickness necrosis of the wall (*Figures 3,4*). Rendezvous procedure with ERCP was done and CBD metallic stent was placed. Although patient had a protracted stay at the hospital, he had significant improvement and was discharged in a stable condition.

Discussion

GC is an advanced stage of gallbladder inflammation. Incidence ranges from 2-29% in all patients with acute

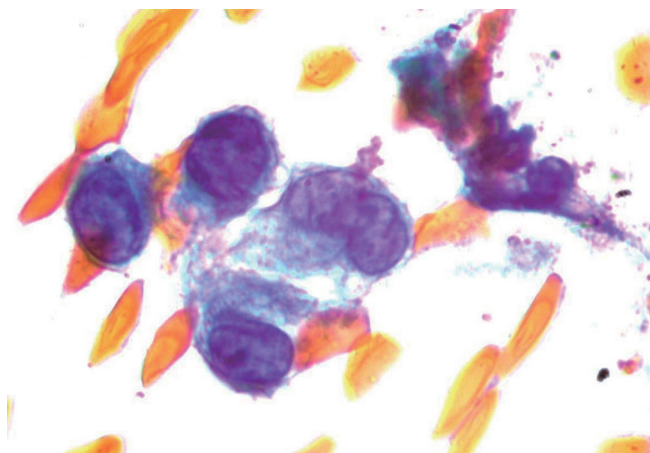


Figure 1 Ductal carcinoma of the pancreas. Endoscopic ultrasound guided fine needle aspiration biopsy. High power magnification. Single malignant tumor cells displaying features of malignancy such as markedly enlarged nuclei with irregular nuclear profiles and unevenly distributed granular chromatin. Papanicolaou stain. Original magnification $\times 1,000$

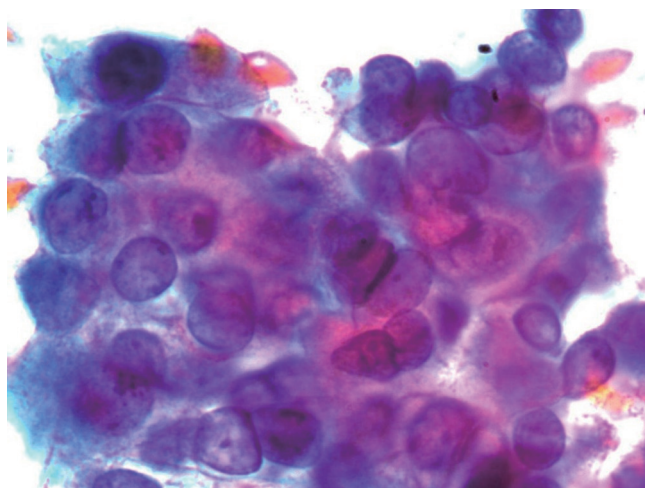


Figure 2 Ductal carcinoma of the pancreas. Endoscopic ultrasound guided fine needle aspiration biopsy. High power magnification. Cluster of tumor cells displaying lack of polarity and malignant nuclear features

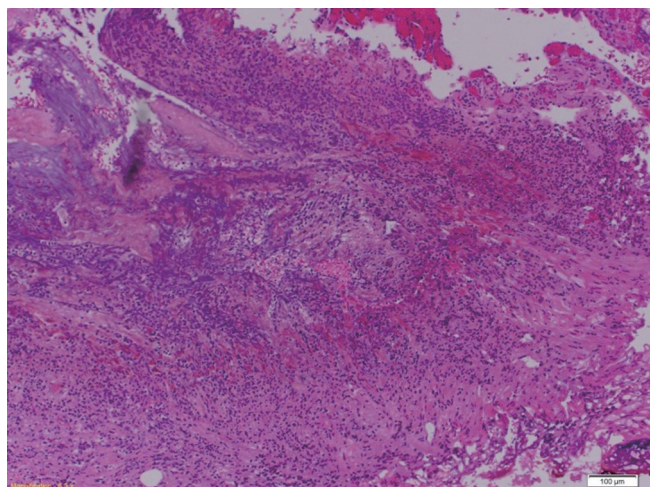


Figure 3 Gallbladder, tissue section. Gangrenous necrosis, low power view. Gallbladder wall display microabscesses and cellular debris amid in fibrinoid necrosis. Hematoxilin and eosin; Original magnification $\times 100$

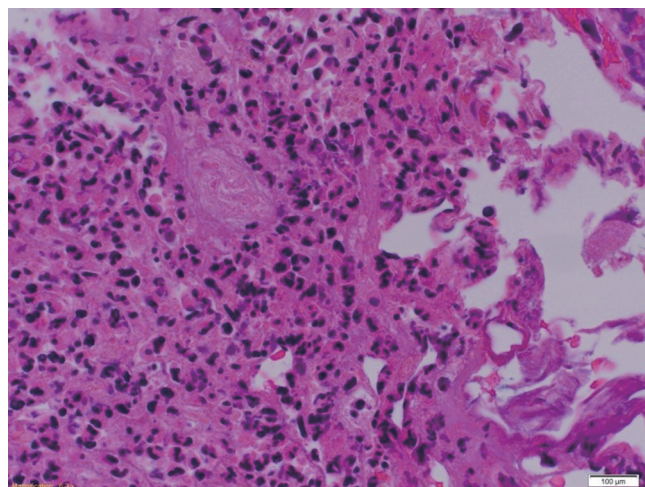


Figure 4 Gallbladder, tissue section. Gangrenous necrosis, high power view. Microabscesses, neutrophils, and necrotic cellular debris. Hematoxilin and eosin; Original magnification $\times 400$

cholecystitis but occurs more often in older male patients (1). The etiology of GC is believed to be primarily due to vascular compromise secondary to sustained obstruction of the cystic duct. It is the combination of GB wall tension and the intense inflammatory reaction that results in either local or global GB wall ischemia (2). The risk of GC increases in patients with coexisting cardiovascular disease, critical

illness and diabetes (1,3). The clinical diagnosis can be extremely challenging as the preoperative differentiation between GC and uncomplicated acute cholecystitis may be very difficult and laboratory and imaging results may be non specific (1,4). Habib *et al.* (5) demonstrated that only 7.5% of patients with GC were able to be identified preoperatively despite the use of clinical, laboratory and

image findings.

Although the patient presented with a picture of ascending cholangitis, the lack of CBD stones or GB stones as a cause of cholangitis combined with the lack of improvement on conventional therapy ought to make one think of alternative diagnoses. There have been no previous reports of pancreatic adenocarcinoma presenting with GC. Given the high morbidity and mortality associated with GC, an early diagnosis is a key especially in the setting of an uncommonly associated disease like pancreatic adenocarcinoma and a prompt treatment approach is lifesaving. In conclusion, GC should be considered in critically ill patients presenting with malignant obstructive jaundice.

Acknowledgements

Disclosure: The authors declare no conflict of interest.

Cite this article as: Kayyali A, Toumeh A, Jiang Y, Youssef W. Gangrenous cholecystitis complicating pancreatic head adenocarcinoma: a case report. *Transl Gastrointest Cancer* 2013;2(3):167-169. doi: 10.3978/j.issn.2224-4778.2013.02.01

References

1. Bennett GL, Rusinek H, Lisi V, et al. CT findings in acute gangrenous cholecystitis. *AJR Am J Roentgenol* 2002;178:275-81.
2. Teefey SA, Baron RL, Radke HM, et al. Gangrenous cholecystitis: new observations on sonography. *J Ultrasound Med* 1991;10:603-6.
3. Contini S, Corradi D, Busi N, et al. Can gangrenous cholecystitis be prevented?: a plea against a “wait and see” attitude. *J Clin Gastroenterol* 2004;38:710-6.
4. Fagan SP, Awad SS, Rahwan K, et al. Prognostic factors for the development of gangrenous cholecystitis. *Am J Surg* 2003;186:481-5.
5. Habib FA, Kolachalam RB, Khilnani R, et al. Role of laparoscopic cholecystectomy in the management of gangrenous cholecystitis. *Am J Surg* 2001;181:71-5.