CASE REPORT JSLS

Minimally Invasive Treatment of Acute Intrahepatic Fluid Collections With Acute Biliary Pancreatitis

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ABSTRACT

Background: Peripancreatic fluid collection suggests the anatomical-clinical scenario of necrotizing acute pancreatitis. However, intrahepatic fluid collection is a rare occurrence with fewer than 30 cases being reported in the medical literature. We describe 2 cases of intrahepatic fluid collection in 2 patients with acute biliary pancreatitis and discuss the therapeutic possibilities.

Case Reports: The first case report is that of a 68-year-old female with a diagnosis of acute biliary pancreatitis with several necrotizing fluid collections and a large infected intrahepatic collection in the left lobe. The patient was successfully treated by percutaneous US/CT guided drainage. The second case report is that of a 72-year-old female with a diagnosis of acute biliary pancreatitis with several peripancreatic fluid collections and a voluminous intrahepatic fluid collection in the left lobe that caused epigastric pain. This patient was also successfully treated with percutaneous US/CT guided drainage.

Conclusion: Intrahepatic fluid collection in the course of acute biliary pancreatitis is a rare occurrence. The therapeutic approach is the same as that for pancreatic and peripancreatic fluid collections. In case of infection, the patient undergoes percutaneous US/CT guided drainage. This therapeutic procedure can be added to the therapeutic program for necrotizing acute biliary pancreatitis together with ERCP/ES and videolaparocholecystectomy (VLC).

Key Words: ERCP, Intrahepatic fluid collection, Biliary pancreatitis, Videolaparocholecystectomy.

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INTRODUCTION

Acute biliary pancreatitis in our area accounts for most pancreatitis. Acute fluid collections arise precociously in acute biliary pancreatitis and are peripancreatic or localized in the pancreas.[1–3] Fluid collection has no surgical indication if it is not complicated (symptomatic or infected), and in the majority of cases it resolves spontaneously. The intrahepatic localization of fluid collection is very rare with fewer than 30 cases being described in literature.[4,5]

We present 2 cases of rare voluminous intrahepatic fluid collections in 2 patients affected by acute biliary pancreatitis. The intrahepatic fluid collections had the same evolution as peripancreatic fluid collection. Both patients were successfully treated with percutaneous US/CT guided drainage applied because of complications.

CASE REPORT ONE

The first case is that of a 68-year-old female with morbid obesity (BMI=47.5), arterial hypertension, and type 2 diabetes, with no alcohol problems. Ten days before admission to our institution, the patient was admitted to another hospital and diagnosed with acute biliary pancreatitis. On admission to our hospital, the patient was in fairly good general condition (ABP 150/80 mm Hg, HR 110 (BPM), spO_2 65 mm Hg, sO_2 90%, total bilirubin 3.12 mg/dL, direct bilirubin 2.65 mg/dL, GOT 87 U/L, GPT 90 U/L, alkaline phosphatase 548 U/L, gamma-GT 943U/L, lypasemia 1288U/L, pancreatic amylasemia 840 U/L, Hb 9.1g/dL, HCT 27%, MCV 62fL, WBC 12000 mm^3, PLT 165000mm^3). The other biohumoral tests were normal. The standard chest x-ray showed left basal pleural effusion. The abdominal examination revealed right hypochondrium pain with negative Blumberg and Murphy signs. The cardiology examination was normal. Twenty-four hours after admission, the patient underwent an ultrasound that revealed abdominal gallbladder microlithiasis with mild dilation of the intra- and extrahepatic biliary ducts without images of intraluminal lithiasis. This was within a scenario of necrotizing pancreatitis with disruption of the entire gland except a small part of the head, and there were several peripancreatic, perihepatic, perisplenic, and pelvic fluid collections (Balthazar E grad-

ing). The patient continued NPO and the appropriate medical therapy. About 72 hours after admission, she was in good general condition and was scheduled to undergo endoscopic retrograde cholangiopancreatography (ERCP) because of the biliary cause of the pancreatitis and the persistence of an increase in the cholestasis tests. However, ERCP was not performed because Vater's papilla could not be cannulated. Seven days after admission, the patient had fever that reached 40.5°C. She therefore underwent a chest-abdominal CT-scan that showed an intrahepatic subcapsular fluid collection in the left lobe about 5cm in diameter with air inside **(Figure 1)**. The intrahepatic collection was successfully treated with percutaneous US/CT guided drainage with a pigtail tube. The chemical-physical and bacteriological examination revealed very high levels of lypase and pancreatic amylase, necrotic material, and *Candida Albicans*. Intravenous B Amphotericin and intradrainage washing of the collection with fluconazole were started. Forty-eight hours after positioning the drain, the patient's fever broke, and 10 days later the drain was removed after CT-scan control because the intrahepatic collections had disappeared **(Figure 2)**. Twenty-five days after admission, the patient was discharged with no major morbidity after the percutaneous treatment, and a cholecystectomy was planned for 30 days later.

CASE REPORT TWO

A 72-year-old female with arterial hypertension, type 2 diabetes, previous acute myocardial infarction, chronic renal failure, with no alcohol problems, had undergone an aorto-bis-femoral by-pass 3 months earlier. She had experienced at least 2 episodes of acute biliary pancreatitis in the previous 6 months and was treated with medical therapy only. On admission, the patient had epigastric pain and vomiting but was in fairly good general condition (ABP 140/80 mm Hg, HR 90/m', spO_2 72 mm Hg, sO_2 95%, total bilirubin 1.15 mg/dL, direct bilirubin 0.6 mg/dL, GOT 45U/L, GPT 40 U/L, alkaline phosphatase 224 U/L, gamma-GT 94 U/L, lypasemia 320 U/L, pancreatic amylasemia 124 U/L, Hb 10.2 g/dL, HCT 31%, MCV 83 fL, BUN 70 U/L, creatinine 1.7 mg/dL, WBC 8000 mm^3, PLT 221000 mm^3. The other biohumoral tests were normal, and the standard chest x-ray showed COPD. The abdominal examination revealed epigastric pain with negative Blumberg and Murphy signs. The cardiology examination was normal. Twenty hours after admission, the patient underwent an abdominal ultrasound that revealed a gallbladder microlithiasis without dilation of the intra- and extrahepatic biliary ducts with lithiasic material inside. Several pancreatic necrotic collections were also reported within the head, body, and tail of the pancreas, as was a voluminous intrahepatic subcapsular fluid collection in the left lobe about 10cm in diameter causing epigastric pain **(Figure 3)**. The patient underwent an ERCP/ES to remove the stone from the main biliary duct, with no major morbidity. Three days later, she successfully underwent percutaneous US/CT guided drainage of the intrahepatic collection with a pigtail tube. The chemical-physical and bacteriological examination revealed very high levels of lypase and pancreatic amylase with a negative microbiological examination. After 12 hours, the pain was gone. The drain

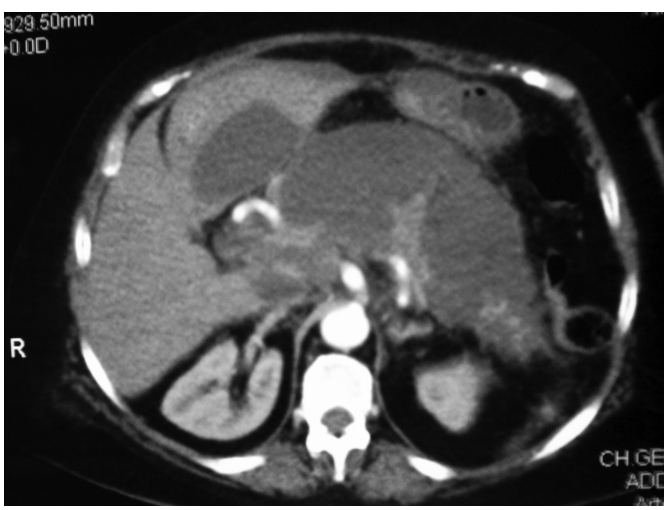

Figure 1. Computed tomographic scan showing an intrahepatic subcapsular fluid collection in the left lobe about 5 cm in diameter with air inside.

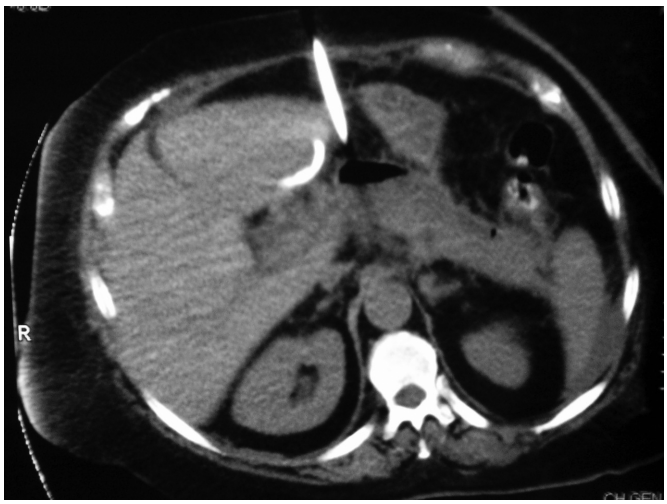

Figure 2. Computed tomographic scan control performed 10 days after drain: disappearance of intrahepatic fluid collection.

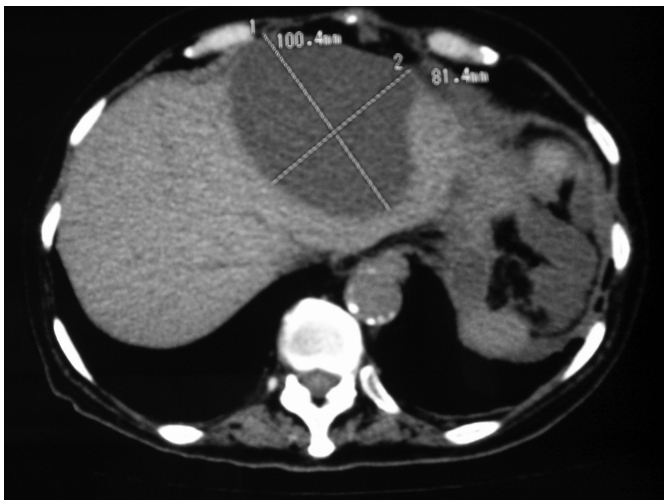

Figure 3. Computed tomographic scan showing a voluminous intrahepatic subcapsular fluid collection in the left lobe about 10cm in diameter.

was removed 7 days later during a control CT-scan that showed that the intrahepatic fluid collections had disappeared **(Figure 4)**. The patient was discharged in good general condition with no major morbidity after the percutaneous treatment, and a cholecystectomy was scheduled for 15 days later.

DISCUSSION

These 2 case reports describe 2 severe acute cases of pancreatitis with a biliary cause.[6] The clinic and laboratory study on admission showed gallbladder lithiasis and principal biliary duct dilation and a severe alteration in the patients' general condition. Thus, the usual therapeutic program for severe acute pancreatitis was started. The morphologic evaluation (US/CT after 48 hours) showed the anatomo-pathologic alterations of the pancreatic and peripancreatic area (necrotizing fluid collections). In both cases, intrahepatic fluid collections were present also. Intrahepatic fluid collections are rare entities in the course of necrotizing acute biliary pancreatitis. To date, only about 30 cases have been reported in the medical literature. However, with the routine use of imaging techniques more will be recognized and references in the medical literature should increase.

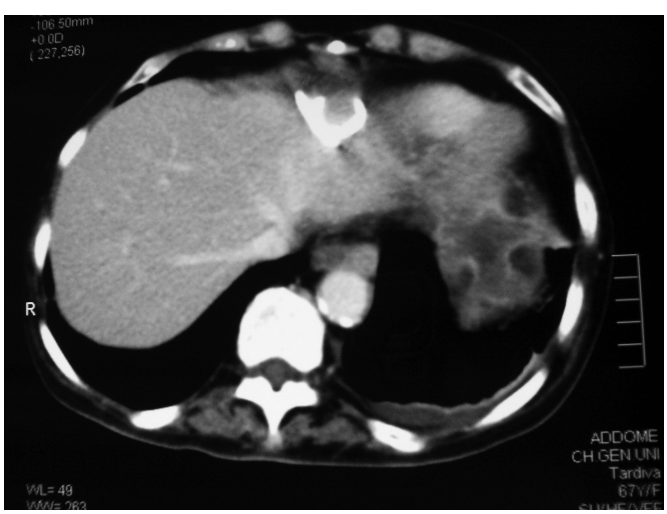

Figure 4. Computed tomographic scan control 7 days later: the intrahepatic fluid collections had disappeared.

Two etiopathogenetic methods for the development of the intrahepatic fluid collections have been proposed.[7–10] The first mechanism consists of pancreatic juice accumulation in the prerenal space and thereafter of eroding through the posterior layer of the parietal peritoneum and into the lesser sac. Then the lesser sac collection tracks along the lesser omentum or gastrohepatic ligament toward the liver leading to the formation of left lobe subcapsular collections. The second mechanism consists of tracking the pancreatic juice along the hepatoduodenal ligament from the head of the pancreas to the porta hepatis, resulting in the formation of intraparenchymal fluid collection. In our patients, it seems that the intrahepatic collections developed by means of the first mechanism because of the left lobe localization. The complete therapeutic program in the 2 reported cases has followed the usual approach for severe acute biliary pancreatitis: control and sustainment of the patient's general clinical condition, early removal (within 72 hours) of the papillary obstacle (in case of the presence of signs of cholestasis and principal biliary duct dilation) by means of ERCP/ES. Following the stabilization of the patient's general condition, VLC was programmed for the treatment of the gallbladder lithiasis. The treatment rationale based on acute pancreatic and peripancreatic fluid collection observation and control was derived from the most frequent evolution. Fluid collections are bound to disappear spontaneously. The therapeutic indication, therefore, is only in case of complications (infection or pain). The intrahepatic localizations, in our experience and in literature reports, had an overlapping evolution like the most frequent peripancreatic collections: therefore, they need the same therapeutic approach. So, in the first phase (collections without a real neo-formed wall), only the infection or painful symptoms derived from compression of the most voluminous collections can justify therapeutic intervention. Moreover, only percutaneous external drainage can be

successfully proposed[11–21] in this phase where there is no stable fibrotic wall. So, only if necessary, the peripancreatic or intrahepatic collections, as shown in this case report, undergo percutaneous US/CT guided drainage if they are infected or if pain is present due to hepatic capsula compression. The percutaneous drainage of the fluid collections in our patients was a safe and efficacious procedure, according to the conclusions derived from the literature.[11–21] Furthermore, today there is a general trend toward reducing all invasive treatment of necrotizing acute biliary pancreatitis. It is a serious disease, dominated by systemic inflammatory response syndrome, where the main therapy (medical, or surgical, or both) is to support and check organic disorders until anatomical and functional recovery of the organism's compromised systems is achieved.

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