Rare Cystic Mass Lesion in the Liver in Alcohol-Associated Cirrhosis

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Abstract

New liver lesions seen in cirrhosis raise strong suspicion for hepatocellular carcinoma. Some of the liver lesions in cirrhosis can have an atypical appearance on standard imaging tests. Alcohol-associated liver cirrhosis can occasionally be accompanied by acute or chronic alcohol-associated pancreatitis. Pancreatic fluid collection is a common complication noted with both acute and chronic pancreatitis. Most acute pancreatitis-related fluid collections are seen in the pancreas and peripancreatic space. Pancreatic pseudocysts are very rarely seen inside the liver and cause significant diagnostic and therapeutic dilemmas. We report a rare case of cystic liver mass in a patient with alcohol-associated cirrhosis and acute pancreatitis.

Keywords: Alcohol-associated cirrhosis, liver lesion, liver lesion biopsy

Introduction

Alcohol-associated cirrhosis can lead to hepatocellular carcinoma (HCC). New onset liver lesions need to be evaluated carefully to rule out HCC. Alcohol-associated liver disease can occasionally be associated with acute pancreatitis. Acute pancreatitis can be complicated with peripancreatic fluid collections. These collections are often seen in the pancreas or near the pancreas. However, they have been described in different sites in the abdomen and pelvis and rarely in the mediastinum.[1] In the absence of ongoing symptoms of acute pancreatitis, incidental fluid collections noted in imaging can cause a diagnostic dilemma. We report a rare occurrence of intrahepatic pancreatic pseudocyst (IHPP) in a patient with alcohol-associated cirrhosis.

Case Report

A 54-year-old man with a history of alcohol-associated compensated cirrhosis presented with dull aching epigastric abdominal pain for one week. He reported no other significant complaints. He has a significant history of alcohol use and was already established in the clinic for follow-up of alcohol-associated cirrhosis. He reported alcohol use 2 days before the hospital visit. Physical examination was largely unremarkable except for mild tenderness in the epigastric area. His liver cancer screening was up to date and the most recent ultrasound of the liver 2 months ago did not reveal any liver mass. His laboratory tests were all at baseline and alfa-feto protein levels were normal. With new onset abdominal pain, we obtained a computed tomography (CT) scan with contrast that revealed an ill defined hypodense lesion in the left lobe of the liver. There was another ill-defined lesion adjacent to head of pancreas that measured 3cm. For further evaluation, a magnetic resonance imaging (MRI) of the abdomen with contrast was obtained and it revealed a 4.5 cm complex cystic mass lesion in the left hepatic lobe [Figure 1]. The images were discussed in a multidisciplinary tumor board, the lesions raised concerns for atypical hepatocellular carcinoma. The liver lesion was

deemed a Liver Imaging Reporting and Data System (LI-RADS)-4 lesion, and percutaneous biopsy was obtained. This revealed normal hepatocytes with some prominent fibroblastic tissue and no malignant cells.

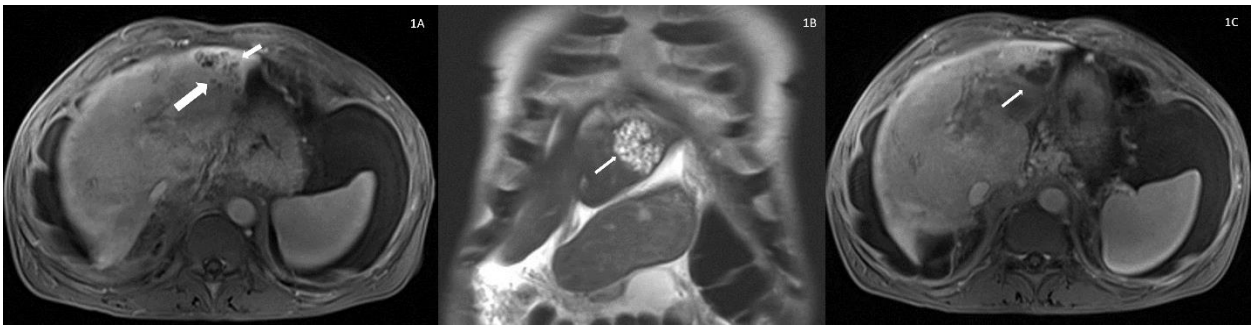

Figure 1 A: T1 weighted, post-contrast (2minute phase) axial image demonstrating broad enhancement of lateral aspect of segment 2 (small white arrow) with "infiltrative masses", some ring enhancement (large white arrow) of individual elements. 1B T2 weighted coronal image through anterior aspect of segments 2-3, high T2 signal indicates high fluid content within mass. 1C T1weighted, post-contrast axial image 1.5 caudal to figure 1 A, revealing cystic component (white arrow) of lesion.

Abdominal pain resolved with symptomatic treatment, and he was discharged after biopsy. He was readmitted with pain in right upper abdomen after 2 days. Repeat CT of the abdomen showed a localized bleed in the perihepatic space adjacent to the biopsy site. Symptoms improved with conservative management. The serum amylase level was normal. His initial CT and MRI were again reviewed after biopsy results. Correlating with the biopsy findings, it was clear that the cyst in the peripancreatic area was suggestive of a pancreatic pseudocyst [Figure 2], and there were subtle imaging changes suggestive of early chronic pancreatitis. This left lobe liver lesion was thereby confirmed as an IHPP.

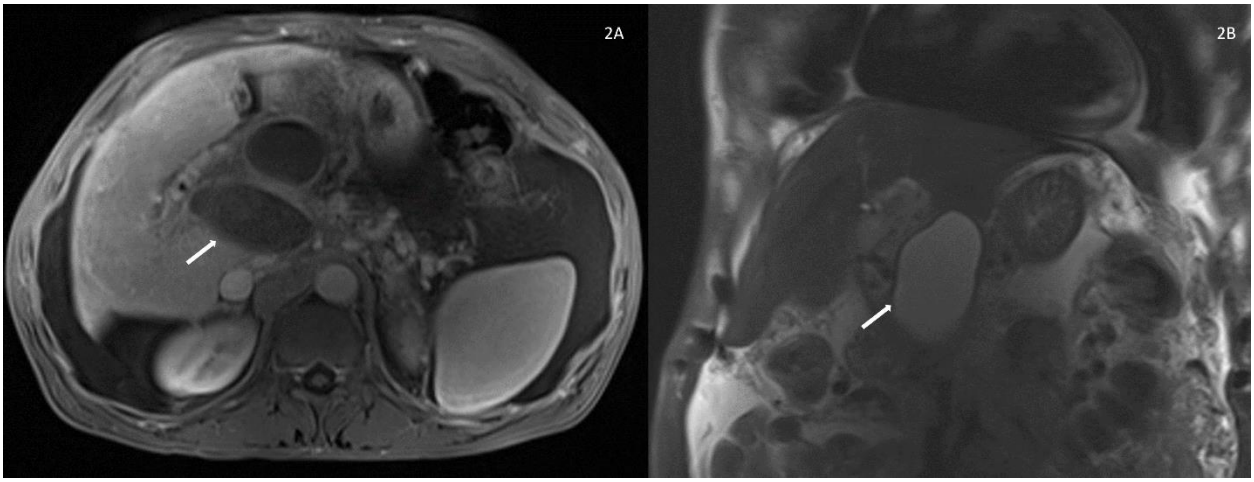

Figure 2 A: T1 weighted, post-contrast axial image 3.5 cm caudal to Figure 1A, indicating a dual rounded fluid collection, part of a rounded U-shaped peripancreatic pseudocyst. 2B T2 weighted coronal image 3.5 cm posterior to figure 1B demonstrating anterior limb (white arrows) of U-shaped abdominal pseudocyst extending cephalad toward undersurface of liver.

He remained abstinent from alcohol and was asymptomatic on follow-up after 6 months, and a repeat CT revealed resolution of intrahepatic and peripancreatic pseudocysts.

Discussion

Pancreatic pseudocysts develop after four weeks of onset of acute pancreatitis, and they do not contain any solid material. Solid contents are usually seen in acute necrotic collections following acute necrotizing pancreatitis.[1] Pseudocysts are often seen in the pancreas and peripancreatic area, but they have been described in different sites in the abdomen and rarely in the mediastinum.[1,2] The liver is an uncommon site for pancreatic pseudocysts. In rare cases reported, they are usually seen in the left lobe of the liver.[3,4] It is believed that pancreatic enzymes released during an episode of acute pancreatitis gravitate towards the lesser sac and then towards the left lobe of the liver along the lesser omentum or gastrohepatic ligament, leading to the involvement of the left lobe of the liver more often.[3] About 40% of reported cases of IHPPs were confined to the liver, and others had cysts in other extrahepatic sites.[3]

Clinical presentation of IHPP is usually nonspecific; abdominal pain is the most common symptom. In a case series, pain was the presenting symptom in 91% of cases.[3] Physical examination mainly was nonspecific, and only 17% of patients in this series had a palpable abdominal mass.[3] The diagnosis of an IHPP is often delayed when they present with nonspecific symptoms. Liver lesions noted on imaging were often mistaken for intrahepatic biliary duct dilatation, hepatic cyst, pyogenic liver abscess, intrahepatic mucinous neoplasm, echinococcal cyst, or cystic malignancy,[3,4] table 1. When imaging is inconclusive, a biopsy of the lesion is considered, which clinches the diagnosis. Biopsy of IHPP is associated with an increased risk of bleeding.[2-4] When fluid can be aspirated from the lesion, this can be diagnostic when it demonstrates high amylase content.[17]

Table 1: The usual differential diagnosis for intrahepatic cystic lesions (References [5-16]).

	Comments	US findings	CT findings	MRI findings
Hepatic cyst[5-7]	Benign, prevalence of 2.5 to 18%, may increase in size with age, usually asymptomatic*[8]	Unilocular and anechoic, thin walls Can rarely be multilocular	Hypodense (attenuation of contents comparable to water)	Hypointense in T1 and hyperintense in T2 images
Polycystic liver disease	Multiple simple cysts (arbitrarily defined as more than 20) *[9] Usually diagnosed in 4th and 5th decades**	Multiple anechoic cysts, variable size	Multiple hypodense lesions	Hypointense in T1 and hyperintense in T2 images
Bile duct hamartoma (von Meyenburg complex)	Asymptomatic Prevalence of about 5.6% in adults (autopsy series), Very rare reports of transformation into cholangiocarcinoma[10]	Small, numerous, usually <1.5cm in size Comet tail artifacts	Hypodense, irregular size	Hypointense in T1 and hyperintense in T2 images No communication with bile ducts
Caroli disease	Both Caroli disease and Caroli syndrome are congenital ductal plate development abnormalities[11]	Fusiform dilation of bile ducts diffusely in liver "Central dot" sign[11] Larger central bile ducts affected	"Central dot" sign more obvious	Communicated with bile ducts (MRCP is helpful)
Caroli syndrome	Symptoms from coexisting hepatic fibrosis Can cause complications of portal hypertension 7% risk of transformation into cholangiocarcinoma[12]	Fusiform dilation of bile ducts, diffusely in liver Liver can be dysmorphic***[13] "Central dot" sign Cysts tend to be smaller than Caroli disease, both central and peripheral bile ducts affected	"Central dot" sign more obvious	Communicated with bile ducts (MRCP is helpful)
Mucinous cystic neoplasm of liver	3-5% of all intrahepatic cysts[14] Predominant in women Surgical resection recommended #	Solitary, large, anechoic Multilocular/septated Thick wall with intracystic nodules	Hypodense contents Hyperdense septae Mural nodule and calcification favor malignancy	Varying signal on T1 and T2 images

Intraductal papillary neoplasm of the bile ducts	10% of all bile duct tumors Symptomatic in about 40% cases (pain, jaundice) Malignant in 40% cases, should be resected if possible[5]	Intraluminal hypo or hyperechoic mass with upstream duct dilatation Variable in size (1.5–35 cm) Mostly seen hear hilum in segment 4, no peripheral edema (compared with abscess)	Intraductal heterogeneous mass- hyperdense at arterial phase, isodense on portal and delayed phase	Hypointense in T1 and hyperintense in T2 images Can have diffusion restriction Connected with bile ducts
Cystic metastasis (secondary to intralesional necrosis or rarely post locoregional therapy)	Very rare, seen with neuroendocrine tumors, malignant melanomas, sarcomas, gastrointestinal stromal tumors[15]	Heteroechoic Thick walled, septated, mural nodules	Variable enhancement Hyperenhancing thick wall, mural nodule and septae	Hyperintense on T2, variable appearance
Pyogenic liver abscess	Abdominal pain, fever Can have associated intraabdominal infection	Variable Anechoic/hypoechoic/hyperechoic. Can be associated with hepatic or portal vein thrombosis	Usually hypodense Variable size “Honeycomb” pattern and “Cluster” sign	Hypointense in T1 and hyperintense in T2 images Central diffusion restriction (large abscess)
Hydatid cyst[16]	More common in certain geographic areas Usually asymptomatic ##	Variable, appearance depends on stage of disease ###	Hypodense, irregular margins, wall calcification, multiloculated, thick walls	Hypointense in T1 and hyperintense in T2 images
Intrahepatic pancreatic pseudocyst	Usually asymptomatic History of pancreatitis and extrahepatic pseudocysts assist in diagnosis	Usually single anechoic cyst Can be multiple (upto 10 cysts reported)	Hypodense on most cases, but can rarely have complex enhancement	Hypointense in T1 and hyperintense in T2 images

* *Complications like, rupture, superimposed infection, intracystic bleed and compression of adjacent structures can lead to pain, fever and jaundice*

** *Multiple pregnancy, increasing age, coexisting kidney disease are risk factors for increase in size of liver cysts*

Spontaneous resolution occurs less commonly in IHPP compared to peripancreatic pseudocysts.[17] Persistent pain or complications such as infection, rupture, biliary obstruction, erosion into adjacent vessels, and bleeding are indications for intervention.[3] Treatment depends on the location, size, and effects of IHPP. Other factors that are considered are the patient's overall clinical stability and the communication of IHPP with the pancreas.[4] Image-guided drainage is successful in some cases. Complex lesions require additional procedures, and if the IHPP communicates with the pancreatic duct, endoscopic pancreatic duct placement is often indicated for complete drainage.[3,4] Large, complicated cysts may require endoscopic ultrasound guidance or adjunctive procedures like naso-pancreatic drain placement. Image-guided and/or endoscopic treatment failure usually necessitates surgical drainage.[2] Recurrences are not uncommon, especially in the setting of pancreatic fistulae or pancreatic duct stricture, and therefore need longitudinal follow-up.[2-4]

In the index case that we reported, the patient had preexisting cirrhosis and did not have florid symptoms of acute or chronic pancreatitis, which led to a delayed diagnosis of IHPP. Concomitant occurrence of alcohol-associated cirrhosis and alcohol-associated chronic pancreatitis is rare. Many postulates attempt to explain this rare occurrence. Cirrhosis leads to decreased synthesis of proteins (lithostatins) that eventually form protein adducts in pancreatic calculi. Alcohol-associated cirrhosis leads to a hypersecretory state within the pancreas. This results from impaired neurohormonal balance at the central nervous system and pancreatic acinar levels. This leads to a washout phenomenon in pancreatic ducts with a decreased tendency of concentration of lithostatins and calcium and, thereby, stone precipitation.[18] The alcohol use patterns are different in those with alcohol-associated chronic pancreatitis and alcohol-associated cirrhosis, this also explains rare concurrence.

The learning points from this rare case report are: 1) Complex lesions in the liver in cirrhosis can be benign, 2) while evaluating hepatic lesions with cystic components, look for similar extrahepatic lesions, 3) synchronous peripancreatic pseudocysts or acute or chronic pancreatitis should raise suspicion of IHPP, 4) if IHPP is suspected, a biopsy should be done with extreme care due to the concomitant risk of bleed.

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