



## Multiple Intrahepatic Pseudocysts: An Unusual Location Following Acute Pancreatitis

### KEYWORDS

Pancreatic pseudocyst, intrahepatic pseudocyst, acute pancreatitis

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### ABSTRACT

*Presence of an intrahepatic pseudocyst following an acute attack of pancreatitis is rare complication with less than 30 cases reported in literature. Lack of experience with this location of pseudocyst makes it a diagnostic challenge. Herein we report a case of a patient who developed multiple intrahepatic pseudocysts following an attack of acute pancreatitis. Though challenging, the diagnosis was made with help of imaging and was confirmed by cytological and biochemical analysis thereby making it an important differential for intrahepatic cysts.*

**INTRODUCTION:** Pseudocyst is defined as a collection of pancreatic juice enclosed by a wall of nonepithelialised granulation tissue or fibrotic capsule. Formation of pseudocyst is a common complication following acute pancreatitis. It is usually formed following 6 weeks of acute attack in about 6% of patients, 40% of these show spontaneous resolution on CECT.(1) But these pseudocyst can occur anywhere in abdomen, depending on where activated pancreatic enzymes are released and what path enzymatic digestion takes.(2) i.e. anywhere from mediastinum to pelvis.(3) An intrahepatic pancreatic pseudocyst is a very rare complication of pancreatitis with less than 30 cases described in the published literature.(3,5) Lack of experience with pseudocyst located in liver makes it a diagnostic as well as therapeutic challenge.

Herein we report a case of a patient who developed multiple intrahepatic pseudocyst following an attack of acute pancreatitis. Though challenging, the diagnosis was made with help of imaging and was confirmed by cytological and biochemical evaluation of aspirated fluid, which was diagnostic as well therapeutic.

**CASE REPORT:** A 40 yrs old chronic alcoholic and diabetic male was admitted to the hospital with complaints of diffuse pain in upper abdomen, malena and blood in vomitus since 5 days. The pain was not radiating to back. Patient gave a past history of an episode of acute attack of pancreatitis 2 months back for which he was admitted in some local hospital and treatment in form of I/V antibiotics and analgesics was given. On examination there was an ill defined swelling in epigastrium and liver was palpable 4cm below the costal margin and was non tender. Blood pressure was 100/70 right arm supine, pulse was 88/min, regular, low in volume with normal rhythm. There was mild anemia and sclera was slightly yellow. PBF showed dimorphic anemia with neutrophilic leucocytosis. Coagulation profile was normal. Liver function test showed 3390 IU/L serum amylase (normal value <115 IU/L), 4200 IU/L serum lipase (normal value <160 IU/L), 681 IU/L AST (normal val-

ue <37), 689 IU/L (normal value <41IU/L) and 258 U/L alkaline phosphate (normal value <130). The APACHE score at admission was 5.

Abdominal USG demonstrated hepatomegaly with evidence of multiple cystic SOL'S of varying sizes in right lobe of liver which were communicating with pancreatic pseudocyst located in the lesser sac. A provisional diagnosis of pancreatic pseudocyst with intrahepatic extension was made. To further confirm the diagnosis contrast enhanced CT scan was done. Axial CECT with oral and I/V contrast at the level of pancreas showed an enlarged and heterogeneous pancreas with poorly delineated borders with walled off hypodense areas in side s/o pseudocyst in the pancreas. Similar walled off hypodense areas, a few being ill defined were seen in segment 6 and 7 of liver s/o intrahepatic pseudocyst. Also there was evidence of marked infiltration of peripancreatic adipose tissue. These cysts were aspirated under CT guidance and 150 ml of straw coloured fluid was recovered. To know the exact nature the aspirated fluid it was sent for cytological and biochemical analysis. Cytology showed that the smears were of low cellularity chiefly comprising of inflammatory cells, a few hepatocytes and macrophages in a proteinaceous background. No neoplastic cells were seen. Amylase was increased (1500 IU/L) along with increased lipase.

Patient was kept NPO and intravenous fluids were given. It was decided to treat the patient conservatively. Enteral nutrition via naso-jejunal tube was started. After 2 weeks a repeat abdominal CECT scan was done, which showed a subtotal resolution of cysts in the liver and total resolution of pancreatic pseudocyst. Enteral nutrition was interrupted and oral ingestion was tolerated by the patient and he was discharged after 18 days stay in the hospital with no residual symptoms.

**DISCUSSION:** Pancreatic pseudocysts can be present virtually in any organ depending on where the activated pancreatic enzymes are released and what path enzymatic

digestion takes place. But intra-hepatic location of pseudocyst following attack of acute pancreatitis is very rare and less than 30 cases are described in literature.(3,5,6,8). Two pathophysiological mechanisms have been proposed trying to explain the location of pseudocyst in liver(3,5). The first suggest a release of pancreatic juice that track through lesser sac towards left lobe of liver along lesser omentum or gastrohepatic ligament, due to proteolytic effect of pancreatic juice, it may dissect the liver capsule leading to formation of subcapsular collections. The second mechanism is that the propagation of pancreatic juice from head of pancreas to portahepatis along the hepatoduodenal ligament resulting in formation of intraparenchymal collection forming intraparenchymal pseudocyst.(2,3,5,7).

Presence of intrahepatic pseudocyst post attack of pancreatitis must be included in the differential diagnosis of cystic lesions in liver.(4, 6). It causes no specific symptoms and can be only an incidental finding. LFT can be normal in some cases.(4, 6). The content of intrahepatic pseudocyst is homogenous with low echoic findings on US and low density on CECT, whereas in liver abscess, the content is denser and contours less demarcated.(6). If it appears after long, pancreas may appear normal. (5)

Amylase levels are increased in the fluid and this is quite diagnostic.(6,5). Rarely intrahepatic pseudocyst can mimic biliary dilatation when pancreatic fluid spreads into hepatoduodenal ligament. In these cases, the differential diagnosis comprises biliary obstruction, both malignant and benign including benign strictures secondary to pancreatic malignancies like pancreatic carcinoma, cholangiocarcinoma.(2) Intra cystic hemorrhage which can occur in 10% of all pancreatic pseudocysts. (6)

Almost every pancreatic pseudocyst improves spontaneously and needs no specific treatment.(3,4) Draining is done when symptoms secondary to compression are found including percutaneous, endoscopic or surgical drainage. (4) Criteria to drain a pancreatic pseudocyst have not been established. Percutaneous drainage is promoted because it allows diagnostic confirmation and treatment for symptomatic and complicated cases only.(4,5,6,7)

**CONCLUSION:** Intra hepatic pseudocyst is to be included in differential diagnosis of cystic lesions in the liver after an attack of acute pancreatitis. It is important due to its rarity and is a diagnostic challenge for a radiologist. As its treatment is drainage or it may show spontaneous regression various differentials are to be negated before one reaches on a definite diagnosis. Role of pathologist cannot be overlooked as cytological and biochemical confirmation of the cystic fluid is also important.

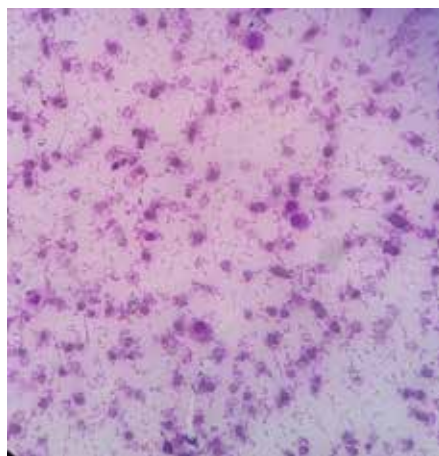


**Fig 1** CECT (axial view), at the level of pancreas showing

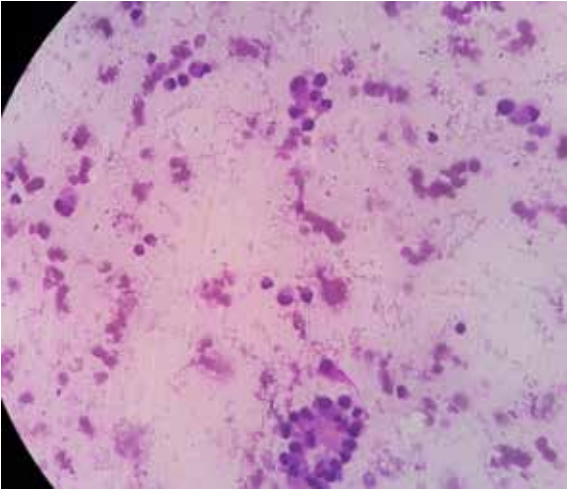
enlarged heterogenous pancreas with poorly delineated borders.



**Fig 2** CECT (axial view), at the level of pancreas showing multiple hypodense lesions in pancreas and liver, S/O pancreatic and intrahepatic pseudocyst.



**Fig 3** Direct smears of the aspirated fluid shows low cellularity with inflammatory cells, occasional hepatocytes and macrophages in a proteinaceous background admixed with RBC's.



**Fig 4** centrifuged deposits of the fluid shows hepatocytes in small clusters, inflammatory cells and macrophages in a proteinaceous background.

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