

taken to embolize it with doxorubicin. The feeding artery was completely embolized using doxorubicin with lipiodol followed by gelfoam slurry (**Figure 3C**). The patient was under constant monitoring in the ICU for 2 days when she left against medical advice. However, she was haemodynamically stable at this time.

Discussion

Complications that may be encountered during or after an RFA procedure include bleeding, peritonitis and injury to the adjacent vessel or the large bowel; these demand immediate attention.² Mechanical damage caused by the placement of electrodes appears to be the likely reason for bleeding,³ especially if the needle is placed directly in a superficially located lesion without traversing significant normal hepatic parenchyma. Coagulopathy is a major risk factor for haemorrhage after RFA. Thus, patients with cirrhosis having a deranged coagulogram are at a higher risk than those without cirrhosis.² Other risk factors include attempt at multiple punctures or the use of multiple electrodes and the location of the targeted lesion behind a major blood vessel. Cauterization of the electrode tract during withdrawal may be an effective method for avoiding or reducing haemorrhage.⁴

Traditionally, the risk of bleeding in subcapsular lesions is considered to be greater in comparison to that due to deep seated lesions. Some authors⁵ have considered subcapsular location as a contraindication for percutaneous therapy of such lesions due to increased haemorrhage and recurrence rates. However, recent results^{6,7} have shown otherwise. Logic tells us that the presence of concomitant ascites would increase the risk of haemoperitoneum after RFA. The hypothesis put forward is that the ascitic fluid would wash away the thrombogenic material at the puncture site and reduce the “tamponade effect” from the opposing parietal peritoneum against the liver.⁸ This would thus increase the risk of sustained intraperitoneal haemorrhage from the hepatic surface in patients who have massive ascites as the tamponade effect would not occur. Fortunately, this potential complication related to free fluid in the peritoneal cavity is quite rare. For deep-seated or “invisible” hepatic tumours that require ablation creating artificial ascites is an established modality for easy visualization of these sonographic “occult” lesions before inserting the needle electrode.^{8,9} Also, artificial ascites helps in insulation and thus prevents the abdominal organs such as diaphragm and bowel loops from the heating effects of RF.

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Calcified pancreatic pseudocyst

Introduction

Pancreatic calcification commonly involves the pancreatic duct. Parenchymal calcification and calcification of pseudocysts are uncommon.

Case report

A 35-year-old man was admitted to our hospital with abdominal pain and intermittent vomiting for 15 days. The patient had been consuming 50–100 mL of alcohol for the past 12 years. He was investigated for alcoholic pancreatitis. The serum amylase was 218 IU/mL and lipase 2530 IU/mL. Plain X-ray abdomen showed pancreatic ductal calcification (stars) and arc-like calcification (arrows) in the pancreatic head region (**Figure 1**). Ultrasound of the abdomen revealed pancreatic ductal calcification and two pseudocysts—one $4.1 \times 3.2 \times 3.7$ cm in the head of the pancreas¹ and the other $8.9 \times 7.2 \times 7.7$ cm in the

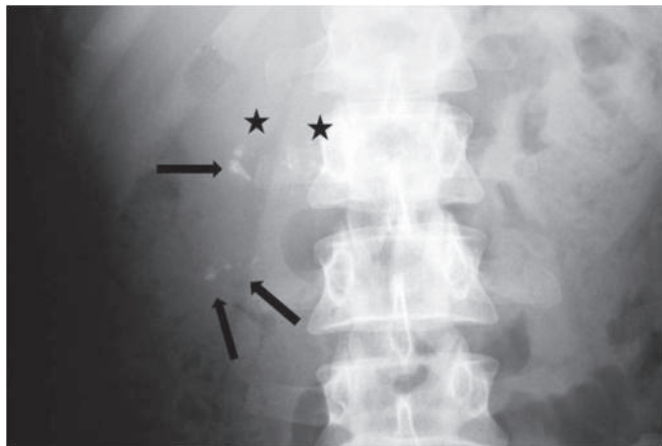


Figure 1: Plain X-ray abdomen showing pancreatic ductal calcification (stars) and arc-like calcification (arrows) in the pancreatic head region

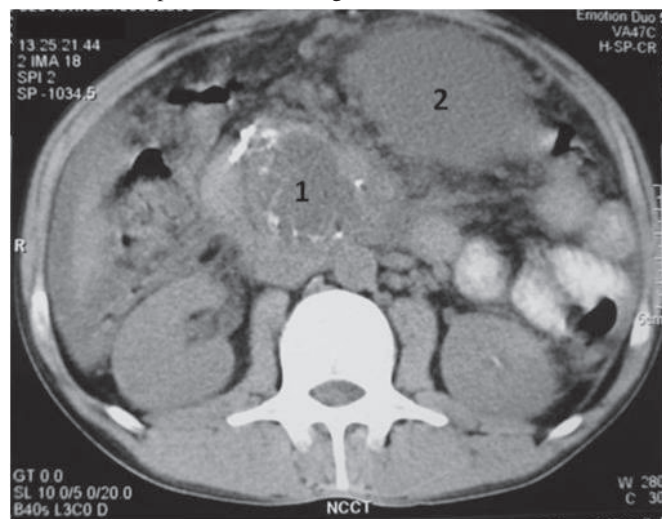


Figure 2: Computed tomography scan showing two pseudocysts—one in the head region (1) with calcification of its wall and the other in the lesser sac (2).

lesser sac.² The pseudocyst in the pancreatic head showed calcification (arrows) along its wall. Plain and contrast-enhanced computed tomography (CECT) showed a pseudocyst in the head region with calcification of its wall and the other in the lesser sac (**Figure 2**). Calcification of the pancreatic duct was

seen separately. The patient was managed conservatively with enzyme supplements, proton pump inhibitors and anti-oxidants; and his symptoms showed improvement. The lesser sac collection resolved on follow-up but the calcified smaller collection has persisted, though he has been pain-free for over 9 months.

Discussion

Calcification of pancreatic pseudocysts is uncommon. It usually occurs when a pseudocyst escapes the initial diagnosis and calcification ensues with the passage of time.¹ Calcification in a pseudocyst has an egg shell-like appearance and must be differentiated from the nodular and granular calcification of chronic pancreatitis. Alcoholic and idiopathic pancreatitis are the two common causes of the chronic pancreatitis. Most often, such calcification is within the pancreatic duct. Sometimes the pancreatic duct can get calcified and pancreatic trauma or infarction with subsequent haemorrhage can lead to parenchymal calcification.¹ Our patient had both, i.e. ductal and pseudocyst calcification. Rowland et al. have also reported the simultaneous occurrence of ductal and pseudocyst calcification.²

Other causes of pancreatic calcification are cystic fibrosis, hyperparathyroidism, mucinous cystadenoma, adenocarcinoma and echinococcal cyst of the pancreas.³ The last three causes can have curvilinear calcification. At times, curvilinear calcification seen on a plain X-ray or CT scan can be the first clue to the underlying chronic pancreatitis. As reported in a case by Munn et al., calcification may be picked up as late as 40 years after the pancreatic trauma.¹

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