

ORIGINAL ARTICLE

PANCREATIC PSEUDOCYSTS AND THEIR UNUSUAL LOCATIONS

Yash Jardosh¹, Hinal Bhagat², Rahul Sharma¹, Dhvani Upadhya¹

Author's Affiliations: ¹Resident; ²Assistant Professor, Dept of Radiodiagnosis & Imaging, New civil hospital, Surat, Gujarat

Correspondence: Dr Yash Jardosh Email: dryashjardosh89@gmail.com

ABSTRACT

Introduction: The aim of our study was to detect the various sites of fluid collections in acute and chronic pancreatitis. Patients presenting with pancreatitis were further evaluated for pseudopancreatic cyst. Characteristics along with location of cysts were compared and unusual location for pseudopancreatic cyst studied.

Methodology: A prospective study of 60 adult patients, (39 male patients and 21 female patients) with Acute and Chronic pancreatitis was undertaken from August 2015 to July 2016. CT scans of 60 patients with acute and chronic pancreatitis were reviewed. Of these in the patients with pseudocyst formation (28 of 60 patients), the location of intrapancreatic, peripancreatic and distal fluid collections in the setting of acute and chronic pancreatitis was studied.

Results: Fluid collections developed in 28 of 60 patients (21 male patients and 7 female patients) for an incidence of 46.6%. Incidence of pseudocyst formation was high in age group of 50-60 yrs. (32.1%), retroperitoneal space according to location (39.2%) and alcohol was noted to be the most common aetiology (60.8%).

Conclusion: Most common locations are the omental bursa and the retroperitoneal space. Pseudocysts in rare sites notably splenic parenchyma, mediastinum and left perinephric space were noted in four patients.

Keywords: Pancreatic Pseudocyst, Mediastinum, Spleen, Perinephric Space

INTRODUCTION

Pancreatic pseudocyst formation is a common complication of pancreatitis. Commonly, it is seen in pancreatic and peripancreatic areas. Pseudocyst may be asymptomatic or may present with variety of symptoms like abdominal pain, nausea, vomiting, upper GIT bleeding.

Diagnosis of complications of pseudocyst like infection, hemorrhage and cyst rupture can be diagnosed at earliest possible stage and reduce morbidity of patient. Mediastinal pancreatic pseudocyst is a rare complication of acute or chronic pancreatitis.¹ Since its first description in 1951, approximately 50 cases have been reported in the world literature. Splenic parenchymal involvement occurs rarely. Heider presented a review of 238 pseudocysts and found only fourteen of them affecting the spleen.^{2,3,4} A dissecting pancreatic pseudocyst can also rarely simulate primary renal disease or perinephric abscess. Our study shows four patients of pancreatitis with pseudocyst formation in spleen, mediastinum and left perinephric space. Study of location of pseudocyst helps in further management of patient and guides exact surgical plane for

better outcome of patient.² Evaluation of location of pseudocyst improves surgical drainage site. With the surgical techniques like internal drainage by cystojejunostomy, permanent resolution of cyst can be achieved. Most cases can be managed conservatively; however, surgical interventions such as splenectomy, distal pancreatectomy and percutaneous drainage are indicated in severe cases. Preserved pancreatic tissue can be exactly determined and salvageable tissue can be useful in future.

METHODOLOGY

The study was conducted in the Department of Radiodiagnosis, New civil hospital, Surat from August 2015 to July 2016. Permission for this study has been obtained from ethical committee of our institute. Patients with clinical history, laboratory findings and/or USG findings suggestive of pancreatitis were prospectively evaluated by supine position in a SIEMENS SOMATOM EMOTION 6 Slice CT machine in suspended inspiration using a kVp of 130 and mAs of 100.

Contrast enhanced CT scan was performed using 1.5 mg/kg of non-ionic iodinated contrast media administered at a flow rate of 2 mL/sec. Approval was obtained by institutional review board, patient's informed consent was waived. Data collection was prospective. The CT scans obtained in patients with acute and chronic pancreatitis presenting to our department from August 2015 to July 2016 were reviewed. We identified 60 adult patients (39 male patients and 21 female patients) with acute and chronic pancreatitis in our radiology database. The patients with concomitant tumour, complications from prior episodes of pancreatitis, motion or streak artefact on CT scan that limited evaluation, unenhanced scans were excluded.

CT Technique: CT examinations were performed on supine position in a SIEMENS SOMATOM EMOTION 6 Slice CT machine in suspended inspiration using a kVp of 130 and mAs of 100. The patients were scanned using our institution's two-phase acquisition pancreatic protocol. The patients were instructed to drink 500 mL of water for negative opacification of the gastrointestinal tract immediately before imaging. The initial pancreatic phase (Late Arterial Dominant Phase) of the examination was performed over the upper abdomen from T11 to L3 vertebral body levels with a scanning delay of 40 seconds after the start of IV administration of 1.5 mL/kg of contrast material at an injection rate of 2 mL/sec.

RESULTS

In the study, 60 patients (39 male patients and 21 female patients) were identified with acute and chronic pancreatitis, of which 28 patients developed pseudocysts with incidence of 46.6%.

Table 1: Distribution of pseudo Cyst among Age Groups

Age (In Years)	No. (%)
20 – 30	3 (11)
30 – 40	5 (18)
40 – 50	7 (25)
50 – 60	9 (32)
60 – 70	4 (14)

Table 2: Distribution of Pseudo cyst in various locations

Location	No. (%)
Retro peritoneal space	11 (39)
Omental bursa	9(32)
Splenic hilum	4(12)
Spleen	1 (3)
Mediastinum	1 (3)
Peri nephric space	2 (6)

Table 3: Distribution of Pseudocyst according to Aetiology

Aetiology	No. (%)
Alcoholic	17 (61)
GB Calculus	5 (18)
Common Bill Duct Calculus	3 (11)
Post-Traumatic	1 (3)
Idiopathic	2 (7)

These 28 patients who developed pseudocysts consisted 21 male patients and 7 female patients. Incidence of pseudocyst among various age group (Table. 1), location of pseudocyst (Table. 2) and aetiology (Table. 3) was described. Incidence of pseudocyst formation was high in age group of 50-60 yrs. (32.1%), Retro- peritoneal space according to location (39.2%) and alcohol was noted to be the most common aetiology (60.8%).

Most common locations are the omental bursa and the retroperitoneal space (anterior and posterior pararenal space). Our study shows four patients of pancreatitis with pseudocyst formation in spleen, mediastinum and left perinephric space.



Figure 1: Well defined collection at splenic hilum and lower pole of spleen



Figure 2: CT Thorax Revealed another Collection Tracking Along the Aortic Recess of Diaphragm into abdomen.

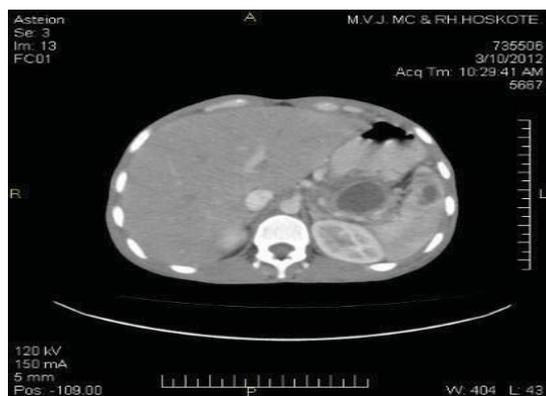


Figure 3: Spleen normal in size with intra parenchymal non enhancing hypodense lesion in lower pole of spleen communicating with hilar cyst. Another collection noted in tail of spleen

DISCUSSION

Conventional radiography and upper gastrointestinal series no longer play an important role in the diagnosis of acute pancreatitis. Radiographic signs of acute pancreatitis include the sentinel loop sign (dilated air-filled duodenum or jejunum), the colon cut-off sign (dilated large bowel to the level of the splenic flexure), loss of the left psoas shadow, ascites, or a gasless abdomen. Pleural effusions, atelectasis, or an elevated hemidiaphragm are suggestive of severe acute pancreatitis.² Thickened rugal and duodenal folds, indentation of the stomach, and enlargement of the C loop of the duodenum are signs of acute pancreatitis on barium meal and follow-through studies. Sonography of patients with acute pancreatitis is often negatively impacted by difficulty visualising the pancreas because of ileus and overlying bowel gas. Abnormal ultrasound findings are seen in 33-90% of patients with acute pancreatitis. Interstitial oedema in acute pancreatitis is depicted on ultrasound as an enlarged hypoechoic gland.³ Although ultrasound may be used to identify peripancreatic acute fluid collections, it is not useful for the detection of necrosis, and therefore its main role in the imaging of acute pancreatitis is limited to the detection of cholelithiasis and choledocholithiasis and identification of fluid collections in the peritoneum, retroperitoneum, and pleural spaces. Contrast-enhanced CT is the imaging modality of choice for the diagnosis and staging of acute pancreatitis. The pancreas enhances uniformly in mild acute pancreatitis and may be normal or enlarged with a variable amount of increased attenuation in the adjacent fat, termed "Stranding".⁴ Local oedema is common and may extend along the mesentery, mesocolon, and hepatoduodenal ligament and into peritoneal spaces.

Extension of oedematous fluid into the anterior perirenal space may create a mass effect and a halo sign with sparing of the perinephric fat. Peri-pancreatic fluid collections consist of exudate, peripancreatic

fat tissue necrosis, or haemorrhage. An organised peripancreatic fluid collection with a fibrous wall occurring greater than 4 weeks after the onset of symptoms is termed a "Pseudocyst". Oedema is differentiated from fluid collections by the identification of fat islands of normal tissue within oedematous fluid. It is usually possible to differentiate acute collections from necrosis. In cases in which CT is unable to accurately differentiate peripancreatic fluid collections from extrapancreatic fat tissue necrosis, it is thought to be safer to consider heterogeneous pancreatic collections as necrotic until proven otherwise. Non-enhancement of all or part of the gland is termed "Necrosis".⁵ CT is 100% specific for necrosis if greater than 30% of the gland is nonenhancing. Necrosis develops between 24 and 48 hours after the onset of acute pancreatitis, and therefore CT within the first 12 hours may be falsely reassuring. Pancreatic abscess formation is usually observed 4-6 weeks after the onset of acute pancreatitis as an area of low attenuation containing pus and a thick wall that may enhance after IV contrast administration. Necrosis and abscess are considered among the most important imaging features of acute pancreatitis because they have prognostic relevance and may precipitate intervention by either interventional radiology or by the surgeons. The MRI imaging of acute pancreatitis is comparable with that of CT, and the same descriptive terminology is used. MRI may be performed using unenhanced and contrast-enhanced T1-weighted and fat-suppressed T2-weighted gradient-echo sequences.

Typically, an enlarged oedematous gland that is low signal on T1-weighted and high signal on T2-weighted MRI is observed.⁶ Acute pancreatitis is sometimes associated with pancreatic ductal dilatation, which can be clearly identified and examined on T2-weighted images. T2-weighted images are also useful for the detection of acute pancreatic collections, pseudocysts, and haemorrhage. The pancreatic duct should be carefully reviewed on T2-weighted images for the presence of disconnection, which can be easily overlooked. Disconnection occurs when necrosis affects the ductal epithelium and an isolated segment of viable pancreatic tissue is disconnected from the duodenum. This creates persistent fistulation and inflammation with an increased incidence of infection. Diagnosis of disconnection of the main pancreatic duct requires visualisation of a necrotic region of at least 2 cm in size, viable pancreatic tissue proximal to the necrosis, and extravasation at pancreatography. The main pancreatic duct usually enters the necrotic material at a 90° angle.

Evaluation for a disconnected pancreatic duct may be performed with CT or MRI. Although early MRCP is generally of limited value for identifying the cause of acute pancreatitis because collections may compress the pancreatic and biliary ducts obscuring gallstones,

MRCP may be of benefit when iodinated contrast administration is contraindicated or if disconnection of the main pancreatic duct is suspected. Secretin-enhanced MRCP may be used for assessment of the pancreatic duct, although concerns regarding the risk of increasing pancreatic inflammation exist. Because ductal pressures approaching those at ERCP cannot be achieved, a normal MRCP is insufficient for exclusion of a disconnected duct in the presence of suspicious features.⁷ An organised peripancreatic fluid collection with a fibrous wall occurring greater than 4 weeks after the onset of symptoms is termed a "Pseudocyst." Intraabdominal fluid collections and collections of necrotic tissue are common in acute pancreatitis. These collections develop early in the course of acute pancreatitis. In the early stage, such a collection does not have a wall or capsule. Most common locations are the omental bursa and the retroperitoneal space (anterior and posterior pararenal space). These collections are the result of the release of activated pancreatic enzymes (namely lipase, trypsin and amylase) which also causes necrosis of the surrounding tissues. This explains why a lot of these collections contain solid debris.

50% of these collections show spontaneous regression. The other 50% either remain stable or increase and undergo organisation and demarcation with liquefaction. They may remain sterile or develop infection. Based on imaging alone, it is often not possible to determine whether these collections contain fluid or necrotic tissue and whether they are infected or not. Consequently, instead of naming them as 'Pseudocysts', 'Abscesses' or 'Necrosis', it is better to describe them as 'peripancreatic collections'. Pancreatic pseudocysts can dissect across tissue planes and boundaries and can envelop, encase and invade adjacent vascular structures and organs resulting in inflammation of peripancreatic tissues. The distal portion of pancreatic tail extends along the splenic vessels to enter splenic hilum located in splenorenal ligament. Because of this close anatomic relationship, splenic vascular involvement in pancreatitis is common. Splenic parenchymal involvement occurs rarely. Dissection of pseudocyst along the course of splenic vessels can cause formation of intrasplenic pseudocysts.

Mediastinal extension of pancreatic pseudocysts can lead to pleural or pericardial effusion, cardiac compression due to mass effect and dysphagia. The ultrasound findings of perinephric involvement of pancreatic pseudocyst can be indistinguishable from a perinephric abscess, but preserved renal function and elevated amylase can be definitive in diagnosis of the pseudocyst. Pseudocysts are due to leakage of pancreatic secretions into surrounding tissues, the rest have occasionally been found involving stomach wall, liver, spleen, mediastinum, neck and pelvis. Since its first

description in 1951, approximately 50 cases have been reported in the world literature. Splenic parenchymal involvement occurs rarely. Heider presented a review of 238 pseudocysts and found only fourteen of them affecting the spleen. As the pancreas and the spleen are located in close proximity, splenic complications may occur in the course of acute and chronic pancreatitis. Splenic involvement in pancreatitis occurs by one of the three aetiologies. One, vascular thrombosis of splenic vein which may lead to chronic pancreatitis. Two, mechanical theory believes that perisplenic adhesions make spleen more vulnerable to injuries. Three, enzymatic theory states that splenic involvement occurs by direct action of pancreatic enzymes on splenic parenchyma or by invasion along the splenic vessels through hilum. In acute pancreatitis, the enzymes released may cause lysis of tissues, may erode splenic capsule resulting in localisation of collection within the parenchyma. Small intrasplenic vessels may be eroded resulting in haematoma which can be contained within a pseudocyst or dissect subcapsularly or cause rupture of spleen. Although very rare (only two percent), splenic involvement of the pancreatitis includes splenic vascular injury, intrasplenic pseudocysts, abscess, haematoma, infarction, necrosis, and rupture. Among these, intrasplenic pseudocysts are most frequent. The most consistent symptom is abdominal pain in the upper left quadrant. Amylase levels may be normal in some cases. In conclusion, patients of acute and chronic pancreatitis should be monitored for these complications.

CONCLUSION

Out of 60 patients with Acute and Chronic pancreatitis, 28 patients had pancreatic pseudocyst formation and most common locations are the omental bursa and the retroperitoneal space. Pseudocysts in rare sites notably splenic parenchyma, mediastinum and left perinephric space were noted in four patients.

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