

PANCREATIC PSEUDOCYSTS AND THEIR UNUSUAL LOCATIONS: A PROSPECTIVE STUDY

Dayananda Kumar ^{R1}, Amith R², Anilesh Pratap Singh³, N. Kudva⁴, Vivek Singh⁵

¹Associate Professor, Department of Radiodiagnosis & Imaging, M. V. J. Medical College & Research Hospital, Bangalore.

²Post Graduate, Department of Radiodiagnosis & Imaging, M. V. J. Medical College & Research Hospital, Bangalore.

³Post Graduate, Department of Radiodiagnosis & Imaging, M. V. J. Medical College & Research Hospital, Bangalore.

⁴Professor, Department of Radiodiagnosis & Imaging, M. V. J. Medical College & Research Hospital, Bangalore.

⁵Post Graduate, Department of Radiodiagnosis & Imaging, M. V. J. Medical College & Research Hospital, Bangalore.

ABSTRACT

OBJECTIVE

The aim of our study was to detect the various sites of fluid collections in acute and chronic pancreatitis.

MATERIAL AND METHODS

A prospective study of 72 adult patients, (45 male patients and 27 female patients) with Acute and Chronic pancreatitis was undertaken from January 2011 to December 2015. CT scans of 72 patients with acute and chronic pancreatitis were reviewed. Of these in the patients with pseudocyst formation (28 of 72 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 72 patients (21 male patients and 7 female patients) for an incidence of 38.9%. 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.

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INTRODUCTION: Pancreatic pseudocyst formation is a common complication of pancreatitis. Commonly, it is seen in pancreatic and peripancreatic areas. Mediastinal pancreatic pseudocyst is a rare complication of acute or chronic pancreatitis. 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. 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.

MATERIALS AND METHODS: The study was conducted in the Department of Radio-diagnosis, MVJ Medical College and Research Hospital, Bangalore, from January 2011 to December 2015. Patients with clinical history, laboratory findings and/or USG findings suggestive of pancreatitis were prospectively evaluated by Toshiba Asteion single slice scanner and GE Brivo 385 16 slice MDCT.

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Corresponding Author:

Dr. Dayananda Kumar,

No. 134, Hosahalli, Hunasamaranahalli (P. O),

Bangalore North-562157, Karnataka.

E-mail: drkrdayanand@gmail.com, dranileshmdrd@yahoo.com

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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 Jan 2011 to December 2015 were reviewed. We identified 72 adult patients (45 male patients and 27 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 Toshiba Asteion single slice scanner and GE Brivo 385 16 slice MDCT scanner. 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 (300 mg I/mL, Ultravist [iopromide, Bayer Health Care] or Omnipaque [Iohexol, GE Healthcare]) at an injection rate of 4 mL/s.

On the 16 - MDCT scanner, the images were acquired at 120 kVp with a detector row configuration of 16×0.75 mm and a table speed of 9.0 mm per rotation with a reconstructed slice thickness of 3 mm. On the 4-MDCT scanner, the images were acquired at 120 kVp with a detector row configuration of 4×1.25 mm and table speed of 7.5 mm per rotation with a reconstructed slice thickness of 2.5 mm. The second portal-dominant phase of the examination was performed from the diaphragm to the symphysis pubis at an 80-second scanning delay. On the 16-MDCT scanner, the images were acquired at 120 kVp with a detector row configuration of 16×1.5 mm, table speed of 18.0 mm per rotation, and reconstructed slice thickness of 4 mm.

RESULTS: In the study, 72 patients (45 male patients and 27 female patients) were identified with acute and chronic pancreatitis, of which 28 patients developed pseudocysts with incidence of 38.9%. 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.



Fig. 1

Fig. 1: CECT Abdomen and Pelvis Revealed Multiple Pockets of Thick Walled Fluid Collections in Lesser Sac and Splenic Hilum.



Fig. 2

Fig. 2: Spleen was Normal in Size with an Intraparenchymal, Nonenhancing Hypodense Lesion in Lower Pole of Spleen Communicating with Hilar Pseudocyst.



Fig. 3

Fig. 3: Ultrasound of Abdomen and Pelvis Revealed a Diffuse Pancreatic Atrophy with Multiple Calcified Foci in Pancreatic Parenchyma.



Fig. 4

Fig. 4: Cystic Lesion with Internal Echoes Was Noted Within the Splenic Parenchyma.

**Fig. 5**

Fig. 5: Spleen was Normal in Size with an Intraparenchymal, Nonenhancing Hypodense Lesion in Lower Pole of Spleen Communicating with Hilar Pseudocyst. There is Irregular Hypodense Collection Noted in Tail of Pancreas.

**Fig. 6**

Fig. 6: The Chest Radiograph Showed a Dense Homogenous Opacity in the Left Midzone and Lower Zone Obliterating the Left CP Angle Causing Mediastinal Shift to the Right Side Suggestive of Gross Pleural Effusion.

**Fig. 7**

Fig. 7: Lower Sections of the CT Thorax Revealed another Collection Tracking Along the Aortic Recess of Diaphragm into the Abdomen.

**Fig. 8**

Fig. 8: CT Scan Showed a Calcified Foci within the Body of Pancreas and a Well-defined Collection near the Head of the Pancreas.

**Fig. 9**

Fig. 9: CT Scan Showed a Well-defined Collection at Splenic Hilum and Lower Pole of Spleen.

**Fig. 10**

Fig. 10: Ultrasound of Abdomen and Pelvis in a 40-year-old Male Patient Revealed a Diffusely Bulky Pancreas with Altered Echotexture. Main Pancreatic duct was Dilated.

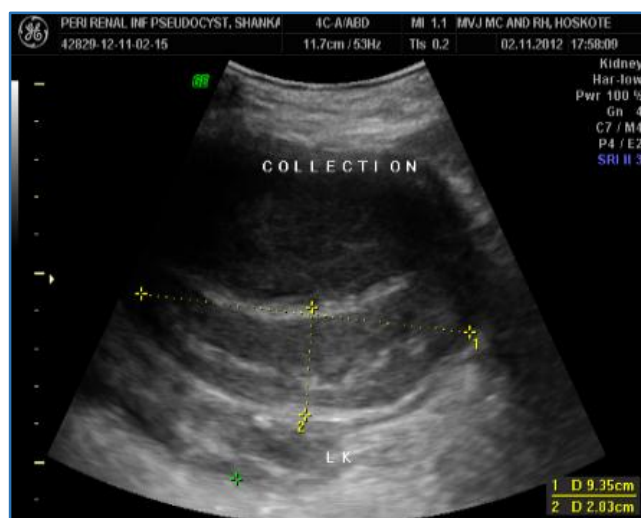


Fig. 11

Fig. 11: A Well-defined Cystic Collection with Internal Echoes in Close Proximity to the Pancreatic Tail Extending along the Posterior Aspect of Left Kidney and Displacing it Anteriorly.

Age Group	No. of Patients with Pseudo Cyst
20 yrs. – 30 yrs.	3
30 yrs. – 40 yrs.	5
40 yrs. – 50 yrs.	7
50 yrs. – 60 yrs.	9
60 yrs. – 70 yrs.	4

Table 1: Showing Incidence among Age Groups

Location of pseudocyst	Number of cases
Retro peritoneal space	11
Omental bursa	9
Splenic hilum	4
Spleen	1
Mediastinum	1
Peri nephric space	2

Table 2: Showing incidence of pseudocyst in various locations

Aetiology	No. of Cases
Alcoholic	17
GB Calculus	5
Common Bill Duct Calculus	3
Post-Traumatic	1
Idiopathic	2

Table 3: Showing Incidence According to Aetiology

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. 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. 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". Local oedema is a common finding 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. Peripancreatic 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". 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 imaging of acute pancreatitis using MRI 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. 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.

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.^[1] 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.^[2]

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.^[3] Since its first description in 1951, approximately 50 cases have been reported in the world literature.^[2] Splenic parenchymal involvement occurs rarely. Heider presented a review of 238 pseudocysts and found only fourteen of them affecting the spleen.^[4] 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.^[5,6] 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.^[1] Although very rare (only two percent), splenic involvement of the pancreatitis includes splenic vascular injury, intrasplenic pseudocysts, abscess, haematoma, infarction, necrosis, and rupture.

Among the above, intrasplenic pseudocysts are the most frequent.^[7] The most consistent symptom is abdominal pain in the upper left quadrant. Amylase levels may be normal in some cases. Most cases can be managed conservatively; however, surgical interventions such as splenectomy, distal pancreatectomy and percutaneous drainage are indicated in severe cases. In conclusion, patients of acute and chronic pancreatitis should be monitored for these unusual complications.

CONCLUSION: Out of 72 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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