

Complications of Acute Pancreatitis: Imaging Revisited

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Abstract

Acute severe pancreatitis is associated with high morbidity and mortality and is frequently complicated by infection, hemorrhage and bowel related complications. The aim of this article is to present an overview of the complications of acute pancreatitis and describe the role of imaging. The findings have been presented in the form of a pictorial essay.

Keywords: Acute Pancreatitis; Hemorrhage; Bowel Related Complications

Introduction

Acute pancreatitis is acute inflammation of the pancreas. The severity of pancreatitis has been graded according to the Atlanta classification proposed in 1992. The Atlanta classification has been revised in 2012 and major changes in the nomenclature of acute and chronic pancreatic fluid collections have been made.

The complications have been divided into local, remote abdominal and systemic. Contrast enhanced Computed Tomography (CECT) scan is the primary imaging modality of choice for grading of severity of pancreatitis and evaluation of complications like necrosis, infection, pseudo-aneurysms, venous thrombosis and bowel related complications.

Magnetic resonance imaging is further helpful for detection of choledocholithiasis, evaluation of ductal anatomy, variations, duct disruption and possible communication with a pseudo-cyst, internal content of the collections and presence of hemorrhage.

The role of ultrasound is limited and is mostly used as a screening tool or for follow up.

The complications of acute severe pancreatitis have been reviewed in the article with emphasis on role of imaging in diagnosis.

Infective complications

Secondary infection in walled-off necrosis

Pancreatic necrosis occurs secondary to thrombosis of microcirculation. It is usually seen 24 - 48 hours after the onset of pancreatitis and a CECT scan should therefore be performed after 3 days of symptom onset [1]. On contrast enhanced CT scan it is seen as non-enhancing parenchyma, which appears more homogenous in the initial disease course and more heterogeneous consequently.

Infection in pancreatic necrosis is the most feared local complication of acute necrotizing pancreatitis and occurs due to contamination with gram negative enteric bacilli [2].

Infection should be suspected clinically in worsening of symptoms along with septic features on examination and supported by laboratory findings.

Air-foci within the necrotic tissue are signs of secondary infection and associated with poorer prognosis. Air foci can also be seen if there is a communication with a hollow viscus or due to post intervention changes, hence clinical correlation with radiology is essential. Fine needle aspiration of necrosis is reserved for patients not responding to empirically started antibiotics and a lumbar retroperitoneal approach is preferred [3] (Figure 1).

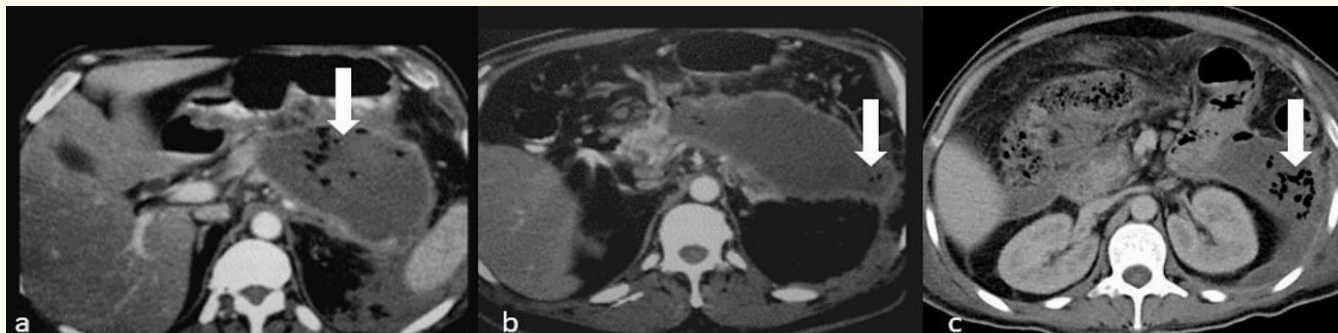


Figure 1: CECT abdomen axial images obtained in venous phase: Intra-pancreatic and extra-pancreatic walled off necrosis with secondary infection seen as non-enhancing necrotic parenchyma with peripheral enhancement replacing the parenchyma of the body and tail of pancreas consistent with walled off necrosis. Internal air foci are noted reflecting secondary infection (white arrows). Necrosis also is seen in the extra-pancreatic retroperitoneal fat.

Vascular complications

Hemorrhage is one of the most life threatening complications of pancreatitis and is multi-factorial. Rupture of pseudo-aneurysm, bleeding gastro-esophageal varices due to portal hypertension, stress gastritis, peptic ulcers and Mallory weiss tear being the causes.

Arterial Pseudo-aneurysms

Rupture of pseudo-aneurysm is the most common cause of hemorrhage in pancreatitis.

Splenic artery is most frequently involved (40%), followed in order of frequency by gastro-duodenal, pancreatico-duodenal, gastric, hepatic and others (superior mesenteric, jejuna, ileo-caecal and aorta) [4].

Formation of pseudo-aneurysms in pancreatitis is multi-factorial and causes are depicted in figure 2.

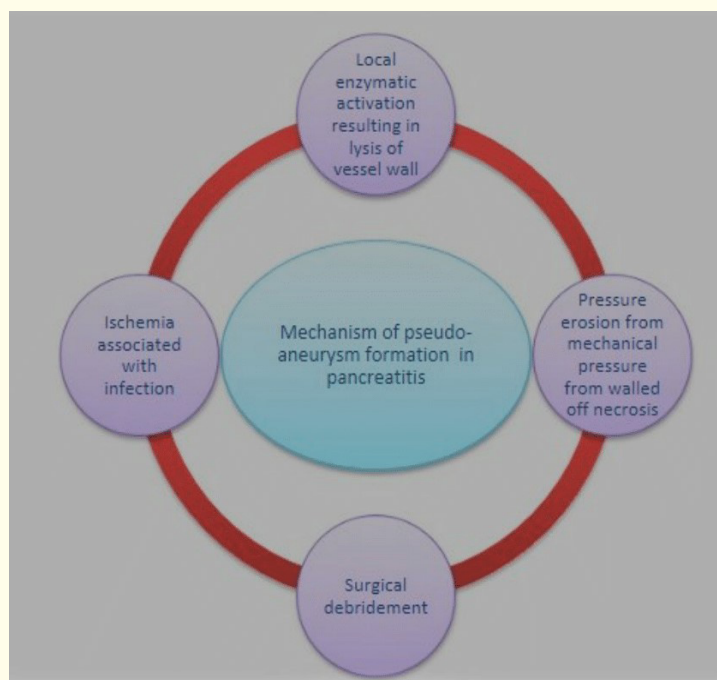


Figure 2: Mechanism of formation of pseudo-aneurysms in pancreatitis.

The presentation may be dramatic in the form of hypotension from sudden bleeding or more subtle in the form of melena due to slow intermittent oozing.

CT abdomen with angiography protocol is the first investigation for evaluation of an arterial pseudo-aneurysm. Arterial pseudo-aneurysms are seen as collections on non-enhanced scan and enhancement with-in the cavity on contrast images with progressive increase in enhancement on dynamic study [4]. CT angiography provides a road map for surgical planning. It is highly accurate in diagnosing arterial pseudo-aneurysms and multi-planar reconstructions with volume rendering and maximum intensity projection allows better understanding of the relationship with surrounding viscera. Partially thrombosed pseudo-aneurysms and surrounding hematomas can also be evaluated [5].

If the infrastructure and expertise is available endovascular treatment is the first line option with coil embolisation and stenting. Re-bleeding rates and overall mortality related to failed angiographic embolisation still remain high [5]. Surgical treatment is indicated if the patient is unstable, expertise is not available or in cases of angiographic failure.



Figure 3: Axial CT images show a well defined heterogenous lesion with peripheral hyper-density on the plain scan and central hypodensity. Contrast opacification is seen in the arterial and venous phases suggestive of splenic artery pseudo-aneurysm (white arrow).

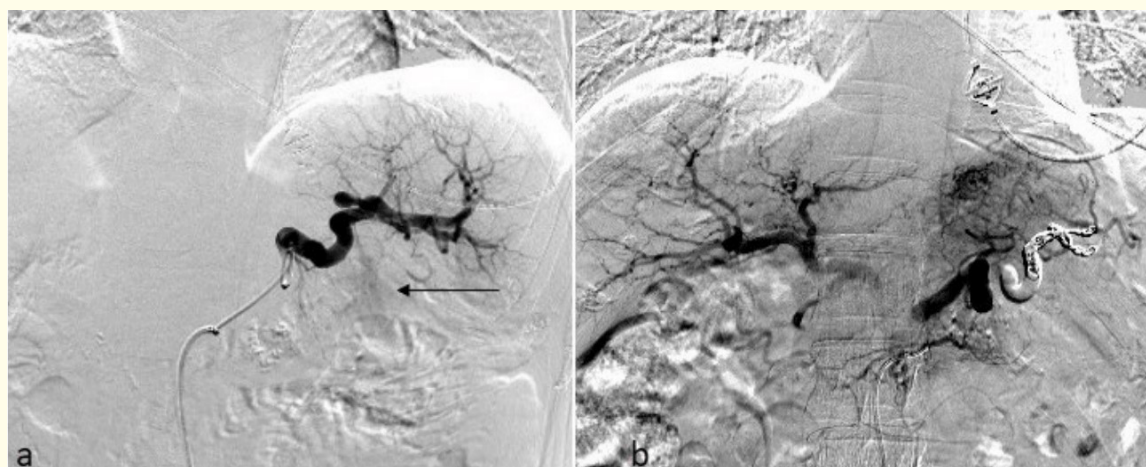


Figure 4: Angiography images showing small rent in the splenic artery with contrast extravasation (black arrow). The aneurysm was managed by coiling and embolization. Post embolization images show embolization of the splenic artery, with preserved splenic perfusion through collaterals.

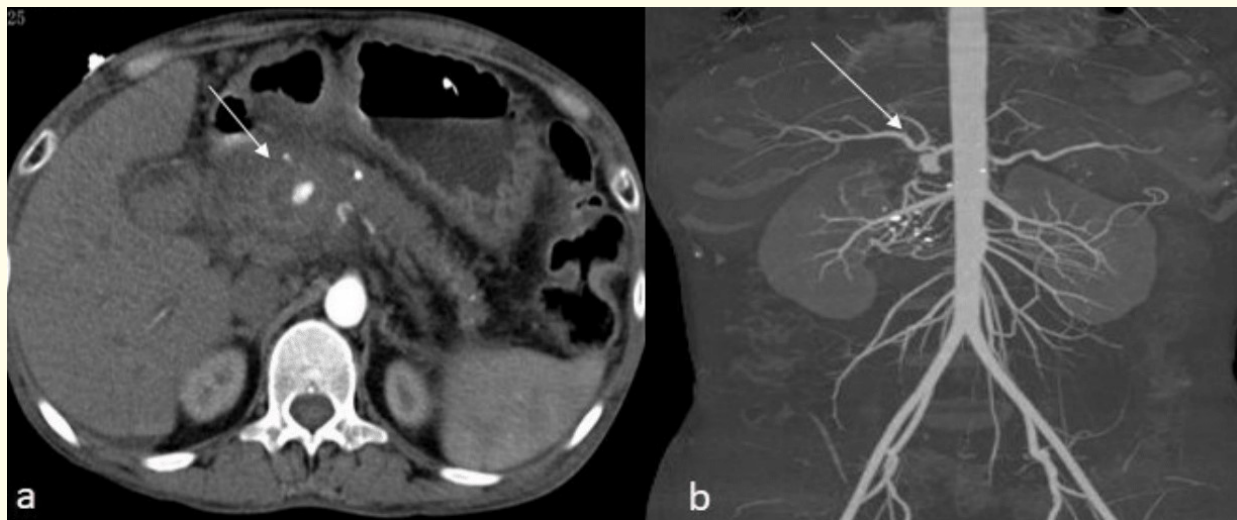


Figure 5: CECT abdomen axial images obtained in the arterial phase reveal, a well-defined collection in the region of head of pancreas, with central contrast opacification (white arrow) Reconstructed MIP CT angiography images reveal, a saccular pseudo-aneurysm from the common hepatic artery.

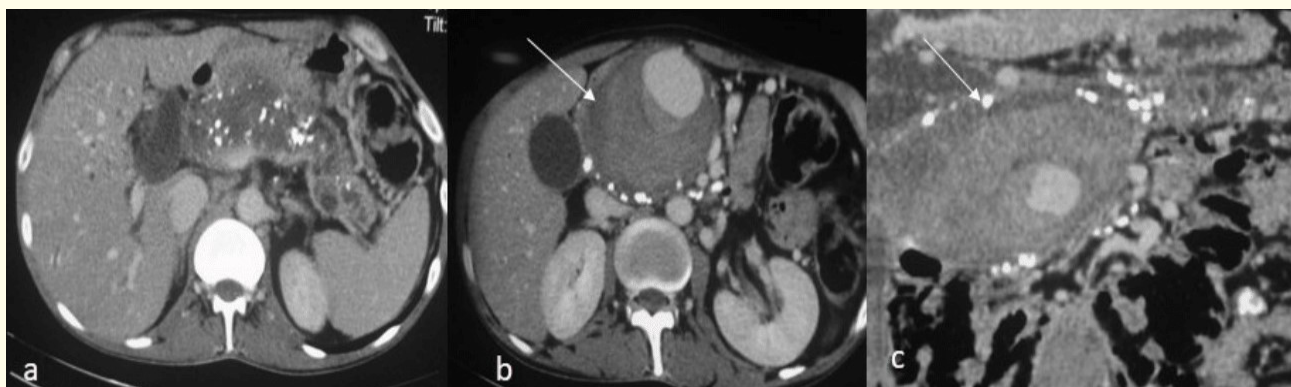


Figure 6: CECT abdomen axial and curved oblique reconstructed image obtained in venous phase showing chronic calcific pancreatitis with features of acute pancreatitis, bulky head and body of pancreas with necrotic parenchyma and mildly dilated main pancreatic duct. Foci of coarse calcification are seen. A well defined collection is seen in relation to the head and body of pancreas with internal enhancing component suggestive of pseudoaneurysm of gastro-duodenal artery and surrounding hyperdensity representing hematoma (white arrow).

Venous thrombosis

The incidence of this complication in severe acute pancreatitis is 1 - 2%.

Splenic vein is most commonly involved (in approximately 70% cases) due to anatomical contiguity. Thrombosis occurs due to stasis of blood flow secondary to mechanical compression by necrosis or extension of inflammation into the intima [6]. Thrombosis can involve the portal and superior mesenteric vein.

Venous thrombosis is seen in contrast enhanced CT imaging as filling defect with non-opacification.

Recanalization of thrombus was observed in approximately one third of patients by Gonzelez HJ, *et al.* irrespective of them receiving or not receiving anti-coagulation. Chronic thrombosis is complicated by varices and left sided portal hypertension. Splenic infraction can occur in long term [2].

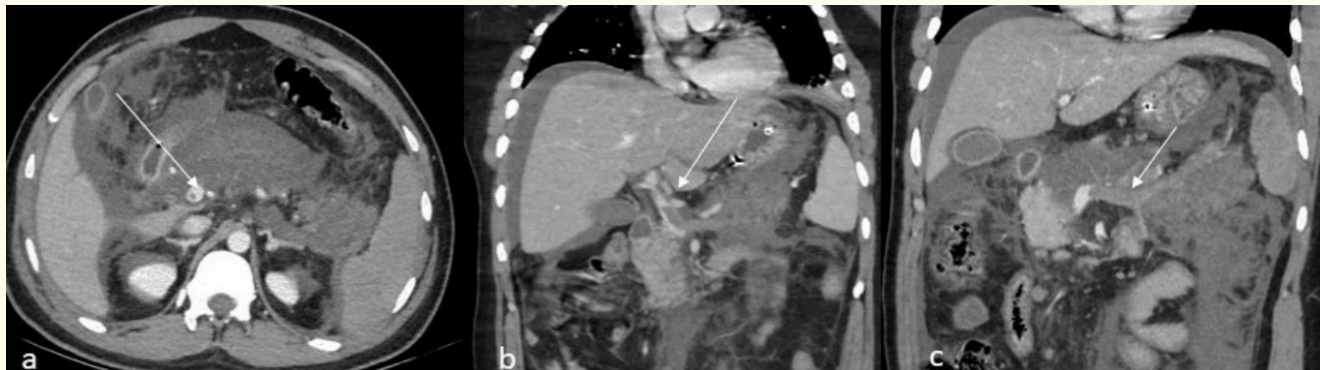


Figure 7: CECT abdomen, axial, coronal and coronal oblique reconstructed images: Acute pancreatitis with pancreatic and peri-pancreatic necrosis. A focal filling defect is seen in the portal vein with complete non-enhancement of the splenic vein suggestive of thrombosis (white arrow).

Bowel related complications

Bowel related complications of pancreatitis are paralytic ileus, mechanical obstruction, ischaemic necrosis, hemorrhage, fistula and perforation [7].

Bowel obstruction

Systemic inflammatory response syndrome mediated paralytic ileus is more common than mechanical obstruction. Mechanical obstruction in pancreatitis can result from extrinsic compression from a pseudo-cyst or secondary to inflammatory stricture due to pancreatic enzymatic extravasation. Transverse colon and splenic flexure are commonly affected segments due to close anatomic relationship. Ischemic strictures may be seen in water shed area like the splenic flexure of colon, secondary to systemic hypotension [8]. Small bowel may be affected due to contiguity of the small bowel mesentery with peritoneal reflection from the anterior surface of pancreas.

Pancreatitis is a pro-coagulant state and thrombosis of mesenteric vessels may occur with consequent ischemia.

Bowel strictures may require operative resection with anastomosis. However surgeries are technically difficult owing to extensive inflammation.



Figure 8: Colonic obstruction secondary to acute pancreatitis Axial and coronal CECT images obtained in the venous phase reveal features of acute pancreatitis with pancreatic necrosis in the region of body and tail and peri-pancreatic necrosis more anteriorly. Concentric thickening of hepatic flexure of colon is seen with non-passage of orally administered contrast beyond and proximal bowel dilatation (white arrow).

Bowel perforation

Perforation of colon and posterior wall of stomach has been described secondary to pancreatitis due to anatomical contiguity. The clinical presentation is variable and symptoms are often masked by the primary disease. Diagnosis is based on high index of suspicion and early imaging in the form of CECT abdomen or endoscopy and colonoscopy.

CECT abdomen is usually the first investigation and bowel perforation is seen as extra-luminal air foci. Contrast extra-vasation could be demonstrated if positive oral and rectal contrast has been administered.

Aghenta AA, *et al.* [9] have reviewed the cases of spontaneous bowel perforation as a complication of severe pancreatitis and suggest individualization of treatment protocol based on the site and severity of involvement. Conservative management with spontaneous drainage may be sufficient in some cases [10], however in ischemic gangrenous colon surgery remains the treatment of choice. The surgery is technically difficult owing to inflammation.



Figure 9: CECT abdomen coronal images showing air containing intra-hepatic collection secondary to acute pancreatitis (bold white arrow). A communication is seen with the mid transverse colon consistent with contained perforation (thin white arrow).

Cystic duodenal dystrophy

Cystic duodenal dystrophy of pancreas is characterized by formation of intramural cysts in the duodenum in the sub-mucosal and muscular layer and surrounding para-duodenal region [11]. Mechanism of pathogenesis and imaging features are similar to groove pancreatitis and may be a result of inflammation and fibrosis of ectopic pancreatic tissue within the duodenal wall. The cystic variant is easily seen on imaging as intramural duodenal cysts. A solid variant is uncommonly seen and shows micro cysts within the duodenal walls which are difficult to recognize on imaging. Duodenal wall thickening is seen in most of the cases and differentiation from entities like a duodenal malignancy or pancreatic carcinoma might not be possible on imaging alone [11]. The diagnosis can be confirmed on endoscopic ultrasound guided aspiration with demonstration of sub-mucosal cysts bulging into the duodenal wall and demonstration of chronic inflammatory infiltrate admixed with fibrosis and heterotopic pancreatic tissue.

Alcohol abstinence and oral octreotide may be useful for reducing the size of cysts. Endoscopic decompression may be used for relieving obstruction, however is associated with relapse. Surgical intervention in the form of a bypass procedure or a more radical surgery like Whipple's pancreatico-duodenectomy offers long term cure [12].

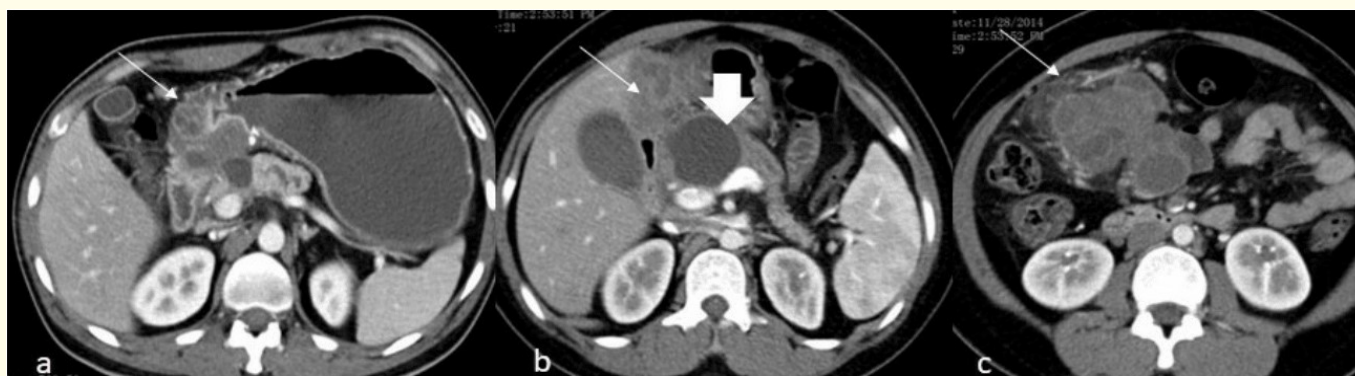


Figure 10: CECT abdomen axial images in venous phase Pylorus and the first part of duodenum show multiple intramural cysts, with significant exophytic involvement in the peri-duodenal region and consequent duodenal narrowing (thin white arrow). A well-defined cystic lesion is seen within the pancreatic body suggestive of intra-pancreatic walled off necrosis (bold white arrow). The body and tail of pancreas is atrophic. Main pancreatic duct is dilated.

Internal pancreatic fistulas

Pleuro-pancreatic fistulas

Pleuro-pancreatic fistula is a rare complication of pancreatitis. Presentation is with recurrent non-resolving pleural effusions which recur rapidly after drainage. Left sided effusions are more common than right sided effusions. The pathogenesis is usually secondary to enzymatic leak from a pseudo cyst or secondary to duct disruption with further extension of the tract into the thorax through the esophageal or aortic hiatus or directly trans-diaphragmatically [13]. Significantly raised amylase in pleural fluid is diagnostic and when complemented with imaging modalities like CECT and Magnetic Resonance Cholangio Pancreaticography (MRCP). MRCP is particularly useful for evaluating the pancreatic duct in its entire extent as well as for identifying developmental anomalies like pancreas divisum. Endoscopic Retrograde Cholangio Pancreaticography (ERCP) may complement the above mentioned modalities and in cases with failure to visualize the tract on cross-sectional imaging.

Thoracocentesis coupled with exocrine suppression using octreotide and ERCP guided stenting is the traditional medical management. It entails bridging the site of duct disruption and allows duct decompression. Operative management is necessary if conservative and endoscopic treatment fails and severe obstruction of pancreatic duct that cannot be managed endoscopically [13].

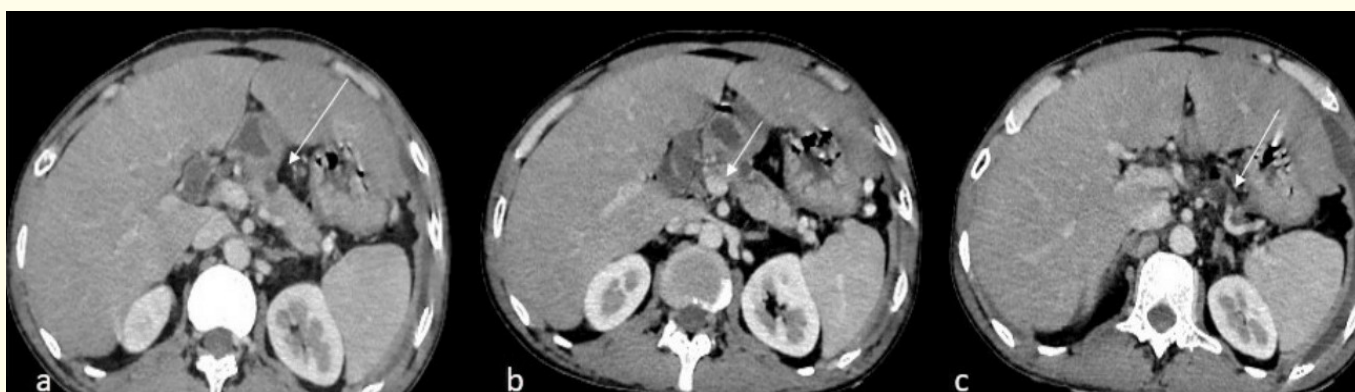


Figure 11: CECT abdomen axial images showing mildly dilated main pancreatic duct in the mid body. A well-defined fluid density tract is seen coursing superiorly from the region of pancreatic body upto the diaphragmatic crura on the left side consistent with pancreatico-pleural fistula (white arrow). The patient had amylase rich pleural effusion refractory to drainage.

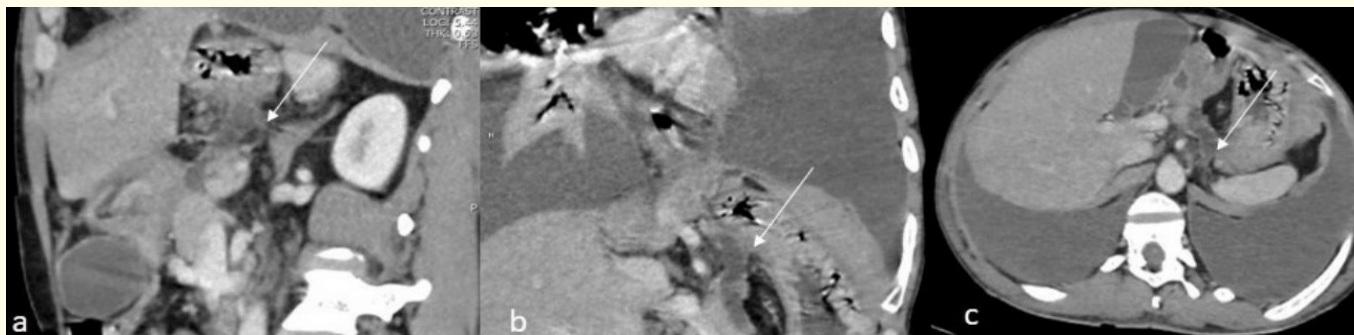


Figure 12: Reconstructed coronal oblique and axial oblique images in the same patient showing a curved fluid filled tract extending from the pancreatic body to the diaphragmatic area (white arrow). Moderate bilateral pleural effusion was seen, with significantly raised amylase on the left side.

Conclusion

Pancreatitis is associated with complications affecting the pancreas itself, surrounding vasculature and bowel. The radiologist and clinician need to be aware of the possible complications and imaging appearance, so that timely diagnosis can be made and further management decisions taken in time to reduce the morbidity and mortality.

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