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Radio diagnosis

Role of Ultrasound and Computed Tomography Scan in Diagnosis of Acute Pancreatitis and Its Complications

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Abstract: The study includes 50 patients of acute pancreatitis, of them all has underwent ultrasound and only 35 patients has undergone CT scan in whom it was indicated. Out of 50 patients 40 were male and 10 were females. Majority of them were in age group of 30 to 50 years and alcoholism was the most common cause. The pancreas was visualized in only 35 patients out *of 50* on ultrasound. In the remaining 15 patients, the pancreas was obscured by bowel gas. Ascites was the most common extra pancreatic finding, being seen in 21 patients.
Keywords: Pancreas, Acute Pancreatitis, ultrasound (US), Computed Tomography (CT) scan.

INTRODUCTION

Pancreas is a retroperitoneal organ with both endocrine and exocrine functions. Acute Pancreatitis is very common disorder of pancreas and have broad spectrum of clinical presentation. Abdominal pain being the most common. Imaging modalities plays an important role in making diagnosis of the diseases and its complications. Various imaging modalities available to diagnose acute pancreatitis includes - x-ray, Ultrasonography (USG), endoscopic ultrasound, Computed Tomography (CT), Magnetic Resonance Imaging (MRI), Magnetic Resonance Cholangiopancreatography (MRCP), Endoscopic Retrograde Cholangiopancreatography (ERCP). Ultrasound is the key and usually first modality to diagnose the acute pancreatitis. However non visualization of pancreas due to bowel gases and normal appearing pancreas in cases of mild pancreatitis warrants use of other imaging modalities in suspected cases.

Excellent resolution of CT scan, its easy availability makes it imaging modality of choice for the diagnosis of disease, assessment of severity & its complications. Use of contrast in CT imaging helps in better delineation of pancreatic and per pancreatic necrosis which is not possible with ultrasound. It is also helpful in looking for morphology of fluid collection, its accessibility for drainage & guiding for interventional based drainage of collections. It is also helpful in monitoring the treatment response in follow-up scans.

MATERIALS AND METHODS

This study included 50 cases of acute pancreatitis that were diagnosed on imaging studies (Ultrasound or CT) or those whose signs, symptoms and laboratory data were indicative of pancreatitis. Of these 50 patients all have underwent ultrasonography of whom CT was done in 35 indicated cases only.

Ultrasound technique used for visualizing pancreas in cases of pancreatitis

- Compression scanning technique with curved linear transducer to displace gas and fluid from the overlying stomach and duodenum which causes obscuration to visualize the body of the pancreas.
- Localizing the vascular landmarks for visualizing the body of the pancreas which includes splenic vein (SV), its confluence with the superior mesenteric vein (SMV), and the superior mesenteric artery (SMA)

- The left lateral decubitus (LLD) position was used to best see the pancreas adjacent to the duodenum [1].

CT technique used for visualizing pancreas in cases of pancreatitis

Both IV and oral contrast should be given to patient for optimum imaging of pancreas. Thin-section images were taken by acquiring images 30-40 seconds after the administration of iodinated contrast (i.e. during the peak of pancreatic arterial perfusion) using helical CT scan. Triphasic study was carried out which included arterial, portal and delayed venous phase for imaging cases of pancreatitis [2]**.**

Inclusion criteria

Patients with clinically suspected pancreatitis with chief complaints of epigastric pain or pain in abdomen which is typically radiating to back, Vomiting & patients coming with c/o acute abdominal pain and diagnosed of pancreatitis on ultrasound and CT.

Exclusion criteria

Patients not ready to give consent & not fitting in inclusion criteria.

Ultrasound criteria used to diagnose acute pancreatitis were

Visualization of pancreas (which was a difficulty in cases of acute pancreatitis)**,** bulky pancreas, heterogenous echotexture (hypoechoic or hyperechoic)**,** extra pancreatic findings (fluid collections, pleural effusion)

CT Criteria to diagnose Acute Pancreatitis were

Bulky pancreas, peripancreatic fat stranding, altered density (heterogenous, hypodense or hyperdense), duct dilatation & extrapancreatic findings (gerota's fascia thickening, stomach wall thickening, and portal vein thrombosis) [2].

RESULTS

Out of these 50 patients, 40 were male and 10 were females. Majority of them were in age group of 30 to 50 years and alcoholism was the most common cause.

Table-1: Age wise distribution of patients with acute pancreatitis

Age group	Male	Female	Total	%age
<20years	01	00	01	6%
21-30years	08	01	09	30%
31-40 years	17	06	23	22%
41-50 years	08	03	11	20%
51-60 years	03	00	03	12%
>60years	03	00	03	10%

Table-2: Distribution of patients depending upon the etiology of pancreatitis

Alcoholism	30 patients
Gallstones	11 patients
Hyperlipidemia	3patients
Drug-induced	1 patients
Trauma	2 patients
Autoimmune	1 patients
Idiopathic	2 patients

FINDINGS ON ULTRASOUND

The pancreas was visualized in only 35 patients out of 50 on ultrasound. In the remaining 15 patients, the pancreas was obscured by bowel gas.

Table-3: Visualisation of pancreas on ultrasound in cases of acute pancreatistis

ULTRASOUND	Visualized	Obscured
Out of 50 patients	35	15 (30%)

SIZE

The size of the pancreas was assessed in the 35 cases in which the pancreas could be visualized. In 27 patients with acute pancreatitis, the pancreas was bulky whereas in 8 patients, it was normal in size In 1 patients of acute on chronic pancreatitis, the pancreas was contracted.

Table-4: Alteration in size visualised on ultrasound in cases of acute pancreatitis

Size	35 patients
Bulky	27 patients (77.14%)
Normal size	08 patients (22.86%)

ECHOGENICITY

In the 35 patients of acute pancreatitis with a visualized pancreas, 20 patients had a hypoechoic echotexture, 10 patients had a heterogenous echotexture and 5 patients had normal echotexture of pancreas.

Table-5: Alteration in echogecities of pancreas in cases of pancreatitis on ultrasound

Echogenicity	Hypoechoic	Heterogenous	Normal	Hyperechoic
Acute Pancreatitis Patients 32	20	10	05	0

EXTRA PANCREATIC FINDINGS

Ascites was the most common finding, being seen in 21 patients with acute pancreatitis. Pleural Effusions, usually on the left side (6cases), were seen in 15 cases of acute pancreatitis. Other findings included gallstones in 11patients.

Table-6: Extra pancreatic findings visualised on ultrasound in cases of pancreatitis

FINDINGS	No. of patients
Ascites	25
Gall stones	11
PV thrombosis	03
Fluid collection	21
Pleural Effusion	06

FINDINGS ON CT

Visualization: The pancreas was visualized in all 35 cases evaluated by CT

Size: The pancreas was bulky in *33* of the 35 patients evaluated. Rest i.e. 2 were of normal size

Extra pancreatic findings on abdominal ct

Stomach wall thickening noted in 24 cases, Gerota‘s fascia thickening seen in 18 patients with acute pancreatitis and portal vein thrombosis in 3 patients.

Table-7: Extrapancreatic findings in cases of acute pancreatitis on ct

FINDINGS	**No. of patients**
Ascites	25 patients
Stomach wall thickening	24 patients
Pleural Effusions	16 patients
Gerota‘s fascia Thickening	20 patients
Gall stones	11 patients
Portal vein thrombosis	3 patients
Fluid collection	12 patients

DISCUSSION

The pancreas is a retroperitoneal organ situated in the anterior pararenal space of the retroperitoneum, where it lies anterior to the perirenal (Gerota’s) fascia and posterior to the parietal peritoneum [2].

Definition and Etiology

Acute pancreatitis is an acute inflammatory disease of the pancreas characterized by auto digestion of the pancreatic parenchyma, interstitial fat necrosis and necrotizing vasculitis, resulted from the inappropriate intracellular activation of proteolytic pancreatic enzymes. The inflammatory process may be limited to the pancreas, spread to surrounding tissues or even involve the remote organs, resulting in multiorgan failure and occasional death [3]. Common causes of acute pancreatitis are gall-stones (30—45%) and alcohol abuse (30-35%) [4,5]. Other causes includes Hyperlipidemia, Trauma, surgery, ERCP, Drugs like azathioprine, mercaptopurine, viral infections (mumps, coxsackie), Autoimmune and Idiopathic.

Pathogenesis: Proposals about possible mechanisms of pancreatic injury, based on animal studies are as Follows

Duct obstruction

Gallstones impacted in the ampulla of Vater can cause pancreatic duct obstruction. Continued pancreatic secretion produces increased ductal pressure which is thought to lead to rupture of small pancreatic ductules which results in extravasation of pancreatic secretions into the interstitium with activation of digestive enzymes and subsequent pancreatitis. Chronic

alcohol ingestion causes secretion of a protein rich pancreatic fluid, leading to deposition of inspissated protein plugs and their calcification. These concretions also may cause obstruction of smaller pancreatic ducts, followed by damage and degeneration of acini and fibrosis [6].

Deranged intracellular transport of pancreatic enzymes [6]

Acinar cell injury

Is an early event in the evolution of many forms of acute pancreatitis? Alcohol may be directly toxic to acinar cells [6].

Clinical features

The main presenting symptom of acute pancreatitis is abdominal pain, typically in the epigastric region which is most of times radiating to the back. The pain is often accompanied by fever, nausea and vomiting. Nausea, vomiting and abdominal distension due to gastric and intestinal hypomotility [7].

On examination

Patient is distressed and anxious with Low grade fever, tachycardia and hypotension. Patient may have jaundice (intrapancreatic common bile duct compression due to pancreatic head edema), Erythematous skin nodules due to subcutaneous fat necrosis. There may be faint blue discoloration around the umbilicus due to hem peritoneum termed as Cullen's sign and bluish discoloration of left flank due to tissue catabolism of the hemoglobin termed as Turners sign.[7] Pulmonary findings in form of basilar rales, atelectasis, pleural effusion may be present bilaterally but usually left side is more common than right. Clinical parameters mentioned above, are associated with severe attack. However these parameters are not reliable prognostic factors and cannot be used as indicators of disease severity [7]. Furthermore old age, hyperlipidemia and obesity are associated with an increased risk of death.

Complications

The complications have been classified as Early Intermediate & Late. Although there is an overlap in the timing of their occurrence early complications occur within 2-3 days of acute attack and are systemic in nature. Abdominal complication usually develops later- within a few weeks or, with decreasing frequency [8]**.**

Early complications

These include systemic complications associated with multiorgan failure. Cardiovascular complications: ECG changes, cardiac and peripheral vascular failure: hypotension and shock. Pulmonary complications: respiratory insufficiency, tachypnea, arterial hypoxemia, and ARDS (Acute Respiratory Distress Syndrome) Renal complications: oliguria, and anuria. Metabolic complications: coagulation factor abnormality, thrombosis, or bleeding (DIC), hyperglycemia, diabetic coma and hypocalcaemia [8].

Intermediate complications - Pseudocysts

Are fluid collections consisting of necrotic material, proteinaceous debris, and enzymatic material that is confined by a fibrous capsule. The administration of intravenous contrast material enhances the margins of such collections and makes the central portion appear less dense. Because of the high protein content of the pseudocyst, the density may appear high, and the cystic nature may not be apparent without intravenous contrast. Pseudocysts are typically located in the pancreas and the immediate per pancreatic area [9]. Gas in pseudocysts suggests Fistula formation, Gas forming infection, surgically produced internal cyst ostomy or any combination of these [10].

Pancreatic abscess

Pancreatic abscess is a severe life threatening problem if not correctly treated. If the organism causing the infection is not gas forming, the appearance of fluid on CT study is indistinguishable from a typical case of supportive or hemorrhagic pancreatitis [2].

Vascular problems

This includes occlusion of vessels commonly the splenic artery and vein, and pseudo aneurysms commonly in the splenic and gastroduodenal arteries [2].

Late complications

Include vascular and hemorrhagic complications, Autodigestive action of extravagated pancreatic enzymes on the pancreatic and per pancreatic vascular structure produce pathological changes responsible for the development of vascular and hemorrhagic complications. Vascular morphological changes affecting the major arteries and veins including inflammation, perivascular fibrosis with narrowing and obstructing strictures, thrombosis, and vascular erosions leading to pseudo aneurysm and massive hemorrhage and splenic vein thrombosis develop in 1-3% patient following pancreatitis. Arterial luminal compromise can lead to segmental colonic or proximal small bowel ischemia and infarction [8].

Laboratory findings

The typical laboratory finding is the increase in the serum and/or urine levels of amylase and lipase [9]. Elevated amylase levels are not specific to acute pancreatitis and may be caused by bowel obstruction, infarction, cholecystitis, or perforated ulcer [10].The duration of hyperlipidemia often exceeds that of hyperamylasemia; however hyperlipidemia is also nonspecific and may also be seen in perforated peptic ulcer, acute cholecystitis and intestinal ischemia [11]. Although both increased serum amylase and lipase are nonspecific as discussed above, serum lipase is

considered more sensitive and specific than serum amylase in the diagnosis of pancreatitis.

Table-8: APACHE II Scoring System [12]

Feature	Acute physiology score (APS)								
Variable	+4	+3	+2	+1	0	+1	+2	+3	+4
Temperature	>=41	30 to 40.9		38.5 to 38.9	36 to 38.4	34 to 35.9	32 to 33.9	30 to 31.9	<=29.9
Mean arterial BP	>=160	130 to159	110 to129		70 to 109		50 to 0		<49
Heart rate	>=180	140 to 179	110 to 139		70 to 109		55 to 6	40 to 54	<=39
Respiratory rate	>=50	35 to 49		25 to 34	12	10 to 11	6 to 9		<5
AaPo2*	>=500	350 to 499	200 to 349		<100	61 to 70		61 to 70	<55
Arterial pH	>=7.7	7.6 to7.69		7.5 to 7.59	7.33 to7.49		7.25 to7.32	7.15 to7.24	<7.15
Serum Bicarbonate	>=52	41 to 51.9		32 to 40.9	23 to 31.9		18 to 21.9	15 to 17.9	<15
Serum Sodium	>=180	160 to 179	155 to 159	150 to154	130 to 149		120 to 129	111 to 119	<=110
Serum Potassium	>=7	6 to 6.9		5.5 to 5.9	3.5 to 5.4	3 to 3.4	2.5 to 2.9		<2.5
Serum creatinine	>=3.5	2 to3.4	1.5 to 1.9		0.6 to 1.4				<0.6
Hematocrit	>=60		50 to 59.9	46 to 49.9	30 to 45.9		20 to 29.9		<20
WBC count	>=40		20 to 39.9	15 to 19.9	3 to 14.9		1 to 2.9		<1

In Table 8 (A), BP = blood pressure; A-aPo2 alveolar-arterial oxygen pressure; Pao2 = partial pressure of oxygen in arterial blood; WBC white blood cell. *Use if percentage of inspired oxygen (Fio2) *>50* percent. §--Use if Fio<*50* percent. v--Use only if no arterial blood gas measurements are available

APACHE II ("acute physiology and chronic health evaluation II") is one of the several ICU scoring systems for calculating severity of disease. It is applied within 24 hours of admission of a patient to an intensive care unit : an integer score from 0 to 71 is computed based on several measurements; higher scores correspond to more severe disease and a higher risk of death

Atlanta Symposium held in 1992, divided acute pancreatitis into two groups as ‘‘mild’’ and ‘‘severe’’ based on the clinical and biochemical findings [13]. This classification was revised in 2008, by the ‘‘Acute Pancreatitis Classification Working Group’’ developing a new morphological classification based on the imaging findings, and divided acute pancreatitis into two groups as ‘‘interstitial edematous pancreatitis’’ and ‘‘necrotizing pancreatitis’’ [14]. The majority of the patients presenting with symptoms of acute pancreatitis belongs to the interstitial edematous group(i.e. mild), which is usually self-limiting and gives good response to the medical conservative treatment and resolves within 48 to 72 hours without any local or systemic (i.e. multiorgan failure) complications.

Minority of patients (20-30%) falls in necrotizing (severe) pancreatitis group which often life is threating with local and systemic complications. Two phases (early and late) of acute pancreatitis having bimodal distribution for mortality has been described in detail by the revised Atlanta classification [14].

The early phase is characterized by exponentially expanding pancreatic and peripancreatic inflammation with ischemia. It occurs within the first week of an episode of acute pancreatitis. Most of these changes can either resolve completely with treatment or at times may progress to irreversible necrosis with fluid collections. Presence of organ failure is the main determinant of the clinical course and disease outcome.

The late phase occurs in the second or subsequent weeks. In contrast to early phase the outcome of late phase largely depends on the degree of secondary infection of the pancreatic necrotic tissue and may result in significant increase in mortality.

Radio imaging in acute pancreatitis

Imaging in patients of acute pancreatitis is undertaken to confirm the clinical diagnosis, to exclude

other causes of abdominal pain mimicking pancreatitis, to investigate the etiology of pancreatitis, and to grade the severity of the disease. Ultrasound (US) is the first-line imaging modality for the confirmation of the diagnosis of the disease and the ruling out of other causes of acute abdomen. Contrast-enhanced computed tomography (CT) plays a significant role in evaluating the extent and evolution of the disease and its complications.

Chest x-ray

Left basal atelectasis, elevated left hemidiaphragm, left pleural effusion [15,16]; as shown in figure no.1.

Abdomen x-rays

Duodenal folds may be thickened, Sentinel loop sign (as shown in figure no.2), the left kidney may be displaced downwards and have a surrounding halo due to edema. Fat necrosis appears as indistinct mottled shadowing. Intra pancreatic gas suggests abscess formation or enteric fistula. Abscess may contain a single fluid level or more commonly multiple bubbles [15,16].

Role of ultrasound

As mentioned earlier , Ultrasound is a quick , cheap, readily available repeatable, free of radiation and easy to perform and can be carried out at the bedside makes it the first-line imaging modality of choice in most centers for the confirmation of the diagnosis and the ruling out of other causes of acute abdomen. Next advantage of US is that, it allows evaluating the gallbladder and biliary tract, to detect gallstones and dilatation of the bile ducts which may be the cause of acute pancreatitis in most of cases. However, there are certain limitations to this modality related to paralytic ileus accompanying in the first 48 hours of the disease those results in non-visualization or obscuration of pancreas. Pancreas may appear normal in the cases of mild acute pancreatitis. In around 30% of the cases, ultrasound is able to detect pancreatic enlargement and diffusely reduced pancreatic echogenicity representing diffuse interstitial edema.

Per pancreatic edema of surrounding adipose tissue may result in blurring of pancreatic contour. There may be per pancreatic fluid collections especially in lesser sac and the left anterior prerenal space. US is used in the characterization of the contents of the fluid collections and the pseudocyst [17-19]. US does not allow the evaluation of the parenchymal perfusion hence is unable to diagnose necrosis of pancreas resulting in inability to make the differential diagnosis of the necrotizing and the interstitial pancreatitis. Use of colour Doppler ultrasound helps to detect vascular complications such as arterial pseudo aneurysm, or portal vein thrombosis arising as a result of pancreatitis.

Role of Computed tomography

Contrast-enhanced CT scan is considered as the gold standard in the evaluation of the patients with acute pancreatitis. It not only establishes the diagnosis of acute pancreatitis as well as allows to stage the severity of the disease [20,21]. The CT attenuation values of normal pancreatic parenchyma on unenhanced CT is 40—50 Hounsfield units (HU) with homogeneous increase in attenuation on administration of intravenous contrast agent to 100-150 HU [21]. CT scan is warranted if the clinical diagnosis is uncertain, or pancreas has not visualized on ultrasound and clinical and laboratory parameters suggests pancreatitis, or clinical findings suggest severe acute pancreatitis (Ranson score ≥ 3, APACHE II score ≥ 8), or there is high suspicion of necrotizing pancreatitis, or those patients who do not improve clinically within 72 hours despite of aggressive medical management or patients who demonstrate early improvement but have symptoms like fever, pain, decrease in hematocrit or hypotension in due course of disease, or high index of suspicion for developing complication [22-24].

Acute pancreatitis on unenhanced CT scan shows localized or diffuse enlargement of the pancreas, ill-defined parenchymal contours and in homogenous pancreatic parenchyma with decreased in parenchymal density and fluid collections in the per pancreatic region. Per pancreatic fat shows increased attenuation secondary to the inflammatory reaction termed as ‘‘stranding’’ [25-27]. Ct scan in early stages of the acute pancreatitis may show reactive pleural or pericardial effusion. In addition to this unenhanced CT scan also helps to detect the calcified gallstones and the parenchymal calcifications in cases of chronic pancreatitis. Contrast enhanced scans shows reduced parenchymal enhancement in case of pancreatic edema or interstitial pancreatitis. Absence of parenchymal enhancement on post contrast study suggests pancreatic necrosis.

There is high accuracy approximately 80-90% in definition of necrosis on contrast enhanced CT scan done after 48-72 hours after the onset of acute attack of pancreatitis. Addition of the arterial phase to contrast enhanced protocol of CT scan while imaging the patients of acute pancreatitis helps in clear delineation of vascular complications, like hemorrhage, pseudo aneurysms [28]. Also the portal venous phase, taken 60-70 s after the administration of intravenous contrast agent, allows better to diagnose pancreatic necrosis as well as to diagnose the extra-pancreatic complications if any.

CT severity index (CTSI)

The CTSI is based on the presence and severity of inflammation and necrosis of pancreas. Total score of 10 points is given based on the severity as depicted in table no.9 [29]. Although this system could be reasonably used to predict overall prognosis in

patients with acute pancreatitis, however this index does not correlate appropriately with subsequent development of extra pancreatic complications, vascular complications and organ failure. Hence an attempt was made to improve the prognostic value of CT in cases of acute pancreatitis in form of modified CT severity index as depicted in table no.10 [30].

Table-9: CT severity index (CTSI) to classify the severity of acute pancreatitis [29]

Description of Pancreas	CT Grade	No. of points assigned	Percentage of pancreas with necrosis	No. of points assigned for percentage necrosis	CTSI
Normal	A	0	0	0	0
Focal or diffuse enlargement	B	1	0	0	1
Peripancreatic fat stranding	C	2	<30	2	4
Single ill-defined collection	D	3	30-50	4	7
Two or more ill-defined collection	E	4	>50	6	10

Table-10: Modified CT Severity Index [30]

Prognostic Indicator	Points
Normal pancreas	0
Intrinsic pancreatic abnormalities with or without inflammatory changes in peripancreatic fat	2
Pancreatic or peripancreatic fluid collection or peripancreatic fat necrosis	4
Pancreatic Necrosis	
None	0
30%	2
>30%	4
Extrapancreatic complications (one or more of pleural effusion, ascites, vascular complications or gastrointestinal tract involvement)	2

CONCLUSIONS

- Maximum numbers of cases are between 30-50 years of age with alcoholism being the major cause of pancreatitis.
- Ultrasonography is cheap, non-invasive, quick &widely available and a safe tool in the imaging and diagnosis of acute pancreatitis and its complications with major limitation of non-visualization of pancreas obscured by bowel gases and inability to detect spread of inflammation to peripancreatic and extrapancreatic regions.
- These limitations are fairly overcome with the use of CT and yields superior results in the evaluation of both acute pancreatitis and its complications.
- Also CT is superior and more accurate in staging of acute pancreatitis and thus helps the clinician to understand the prognosis of patient and helps to decide management plan at the time of hospital admission only.

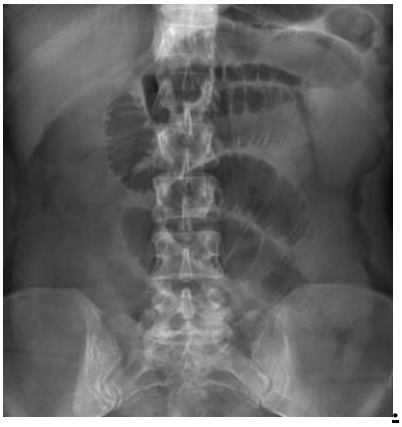

Fig-1: Acute pancreatitis with dilated jejunal loops on erect abdomen x-ray

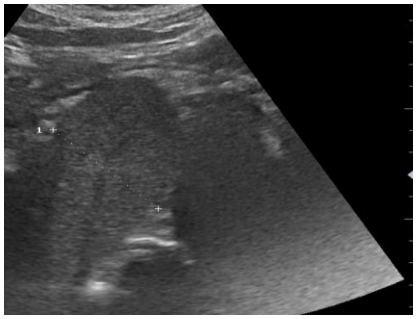

Fig-2: USG image showing Bulky head of pancreas with hypoechoic echotexture in patient of acute pancreatitis

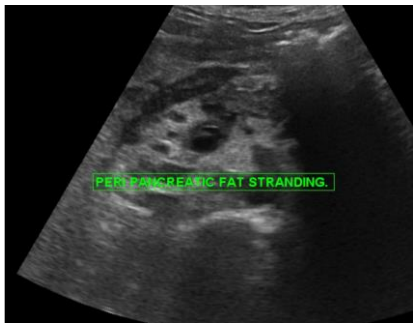

Fig-3: Ultrasound image showing echogenic pancreas with peripancreatic fat stranding in patient of acute pancreatitis

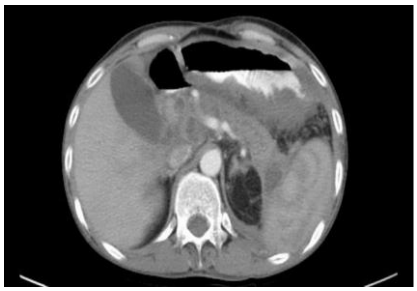

Fig-4: CT image showing fluid collection in patient diagnosed of having acute pancreatitis

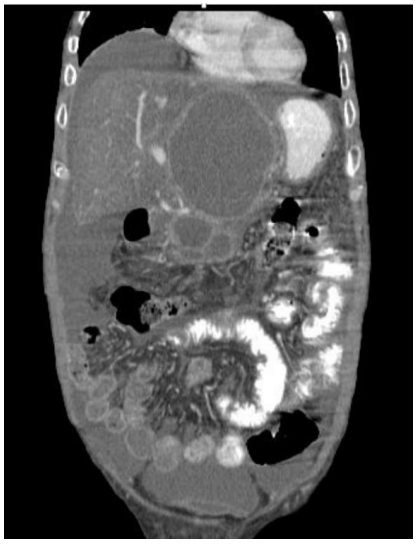

Fig-5: Coronal CT image showing multiple pseudocysts along the pancreas with ascites in the patient of acute pancreatitis

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