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Role of Imaging in Necrotising Pancreatitis

Dr. Nagarjuna Raju.S., Dr. K. Srihari, Dr. Shivani Gogi.
ASSISTANT PROFESSOR, OSMANIA MEDICAL COLLEGE, KOTI, HYDERABAD.
ASSISTANT PROFESSOR, OSMANIA MEDICAL COLLEGE, KOTI, HYDERABAD.
ASSOCIATE PROFESSOR, OSMANIA MEDICAL COLLEGE, KOTI, HYDERABAD.

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Need for the study

Necrotizing pancreatitis is severe form of pancreatitis. presence or absence of necrosis not only increases morbidity and mortality duration of hospital stay & plan of management. Although Diagnosis Acute interstitial edematous pancreatitis are based mainly on signs& symptoms and laboratory finding but the diagnosis of and severity assessment of Acute necrotizing pancreatitis is based mainly on imaging findings.

The present study mainly concentrates on advantages & disadvantages of various imaging modalities especially CT & MRI in the early & prompt diagnosis and severity assessment of pancreatitis and its complications including infection, haemorrhage, pseudo aneurysm formation, venous thrombosis, biliary and Bowel obstruction.

I. INTRODUCTION:

Acute pancreatitis is common clinical problem we encounter in day today practice. It is the commonest pancreatic pathology that causes Emergency Hospital Admissions& has high morbidity and mortality.

Before the era of modern imaging, diagnosis of Acute pancreatitis was mainly based on clinical signs and symptoms and laboratory findings. The invention &advancement of various imaging modalities like CT & MRI has revolutionized process of Diagnosis of acute pancreatitis and its confirmation.

The disease entity of acute pancreatitis is divided in to two morphological subtypes**:** Interstitial oedematous and necrotizing pancreatitis, Necrotizing pancreatitis, themore severe form of pancreatitis is defined as "Necrosis of pancreatic parenchyma with (or) without Necrosis of peripancreatic tissues" [(1).] It occurs as a complication of 20-30% of patients with acute pancreatitis and historically has been associated with high morbidity (34-95%) and mortality (2-39%) [(2,3)].

Outcome of pancreatitis, acute necrotizing pancreatitis mainly dependent on early and prompt diagnosis of this condition and its complications.

During early clinical phase (< 1 week) severity of pancreatitis is determined predominantly by the presence of systemic inflammatory response syndrome and organfailure. The role of imaging is limited during early phase because Morphologicalchanges do not Correlate with clinical findings (or) help predict the subsequent clinical course "[(4).]During the late clinical phase severity and treatment are dictated by presence of clinical feature of persistant systemic signs of ongoing inflammation, features of persistant organ failure & imaging. Imaging is essential in the late phase for diagnosing and evolution of necrotizing pancreatitis and its complications, helping determine when to Implement interventional Radiologic, endoscopic surgical treatment and monitoring treatment Response [(5)].

AIMS AND OBJECTIVES

1. To evaluate the Role of CT and MRI in the diagnosis of Necrotizing pancreatitis and its complications
2. To evaluate the Role of CT and MRI in the prognosis of pancreatitis.
3. Comparitive evaluation of advantages disadvantages in the diagnosis and prognosis Of necrotizing pancreatitis.

ETIOLOGY & PATHOGENESIS OF ACUTE PANCREATITIS

Etiology and pathogens of pancreatitis have been intensively investigated for centuries World wide. [(6)].In 1856 Claude Bernard suggested that bile reflex in to common pancreatic duct could trigger Acute pancreatitis [(7)] several subsequent studies led to theories fuelling the debate until **1901**[(8)] when Eugene oppieproposed gall stone migration into common bile duct is the main cause of pancreatitis [(9)]Since than many other causes of pancreatitis has been discovered.

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There are many causes of pancreatitis which can be easily identified in 75-85% of patient [10]. In the developed countries obstruction is the major cause. developed countries obstructions of common bile ducts (38%)and Alcohol abuse (36%) is the most common causes . where as in India Alcohol abuse is the common case followed by gall stones.[7,11,12,13,14, 15,16,17] Duct obstruction promote pancreatitis by increase duct pressure and subsequent unregulated activation of digestive enzymes.

Alcohol abuse is most common cause in India but the correlation between alcohol and pancreatitis not completely understood [18].

Pancreatic divisium a common congenital anatomical variant of pancreatic duct may lead to pancreatitis because of inadequate patency of minor papillae, wich prevents normal drainage of pancreatic secretions.

Billiary Sludge Refers to Viscous bile suspension that contains cholesterol crystal and calcium bilirubinate granules embedded in stands of gall bladder mucus. Sludge is associated with bile stasis long stranding fasting, distal bile duct obstruction, total parenteral feeding Biliary Sludge is commonly seen in patient with recurrent pancreatitis. cholecystectomy may prevent this recurrent pancreatitis[19]

Intraductal papillary Mucinous tumor might be another cause of acute

pancreatitis. tumor or mucus produced by it obstructs the main pancreatic duct and its side branches. logically the consequence is increased pancreatic duct pressure causing pancreatic hyperstimulation.

Endoscopic retrograde cholangiopancreaticography (ERCP) is a potential cause of acute pancreatitis. Acute hyper amylesemia occurs in 35-70% of patients after the procedure . ERCP has higher risk of inducing Acute pancreatitis when it performed to treat oddispincterobstruction rather than to remove gall stones other risk factor for post ERCP pancreatitis are young Age, female sex number of attempts to consulate papillae, poor emplying of pancreatic duct after opacification.Prevention of pancreatitis might be achieved by temporary stent placing [20]

Hyper calcemia is another rare and inconstant cause of acute pancreatitis.

Drugs rarely can induce pancreatitis have been Reported [21].

There are many Drugs that possibcaly can inducing acute pancreatitis that include HIV drugs like 2', 3' – dideoxynosine (ddI), Azothioprine, Diuretics like frusemide, drugs used is management of inflamatory Bowel disease like 6 mercapto purine,mesalame&sulfaralz rifampicin [21]. PG lankish et all and other authors clearly expressed relation between various durgspancreatitis.[22,23,24,25,26,27]

CLINICAL FEATURES OF ACUTE PANCREATITIS:

Most patients with acute pancreatitis have acute Onset of persistent severe epigastric pain [28]. in some patient the pain may be right upper quadrant on rarely confined to left side.

In patients with gall stone pancreatitis the pain is well localized and onset of painis Rapidin contract, in patient with pancreatitis due to alcohol, heriditory or metabolic cause on set of pain may be less abrupt and pain may be poorly localized. In approximately 50% of patients pain radiates to back Approximately 90% patients have associated nausea vomiting which maybe persistent for several hours[29] patients with severe acute pancreatitis may have dyspnea due to diaphragmatic inflammation.

Pleural effusion on adult respiratory distress syndrome. Approximately 5-10% of patients with pancreatitis mayhave painless disease and have un explained hypotention (eg. Post Operative clinically ill patients on dialysis, organophosphate poisoning , Legionnaires disease)[30,31,32]

LABORATORY FINDINGS

Pancreatic enzymes and products: early in the course of pancreatitis there is synthesis of pancreatic digestive enzymes continue while there in blockage of secretion . As a result digestive enzymesleak out of acinar cells through Baso-lateral membrane to the interstitial space and then in to systemic circulation.

Serum Amylase – Serum amylase raises within 6-12 hours of acute pancreatitis.

Sercum amylase greater than three times limit of normal has sensitivity for the diagnosis of acute pancreatitis of 67 to 83 percent and specificity of 85 – 98%. [33] However elevation of serum Amylase to more than the upper limit of normal may not be seen in Approximately 20 percent of patients with in alcoholic pancreatitis due to inability of parenchyma to produce Amylase andals in 50% of patients with hyper triglysesidemiasassociated with pancreatitis as triglicerideinterfere with amylase assay.[34]

Serum lipase: Serum lipase has sensitivity and specificity for acute pancreatitis ranging from 82 to 100 % [33] serum lipase raised within four to eight hours of Onset of symptoms and peak at 24 hours and return to normal within 8 to 14 days.[35]

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However non specific elevation of lipase base also been reported [33,36]

Various other enzyme products that are recently evaluated areTrypsinogen activationpeptide (TAP), A five Amino acid peptide that in cleaved from trypsinogen to produceactive trypsin is elevated in acute pancreatitis, TAP may be useful in detection of early acute pancreatitis and its predictor of severity of pancreatitis. [37]

Acute pancreatitis associated with elevation of CRP (C-Reactive protein), Il6,IL-8,IL-10,TNF, PMN esterase.[38]Patients with acute pancreatitis may have leukocytosis and elevated haematocrit from haemo concentration, elevated blood urea nitrogen (BUN), hypo calcemia, and Hypoglycemia.

CT SEVERITY INDEX

CT SEVERITY INDEX		
Grading of Pancreatitis	**(Belthazar score)**	
A	Normal pancreas	0
B	Enlargement of pancreas	1
C	Inflamatory changes in pancreas	2
D	Illdefined single peripancreatic fluid collection	3
E	Two moiré poorly defined pierpancreatic fluid collection	4
Pancreatic Necrosis		
	Non	0
	≤ 30%	2
	≥30-50%	4
.	> 50%	6
Total Score -10		
0-3	Mild acute pancreatitis	
4-6	Moderate acute pancreatitis	
7-10	Severe acute pancreatitis	

The original CT severity index has followed internationally and has been very useful. However, it has number of limitations.

It has been found that complications like organ failure do not correlate well with score given by original CTSI.

It has been observed that patients with more than 30% necrosis have similar morbidity and mortality, including additional 50% score was not practically useful.

Because there is limitation A modified CT severity index is proposed by mortel all retrospective Study. On 266 patients underwent contrast MDCT within one week of onset of symptoms three Radiologists were blinded to patients outcome scored the severity of the pancreatitis like old CT severity index and newly proposed modified CT severity index.

MODIFIED CT SEVERITY INDEX

MODIFIED CT SEVERITY INDEX:	
Pancreatic Inflammation	
0	Pancreatic inflammation
2	Intrinsic pancreatic abnormalities with (or) without inflammatory changes in peripancreatic fat.
4	Pancreatic (or)peripnacreatic fluid collection on peripanreatic fat necrosis.
Pancreatic Necrosis	
0	None
2	30% less

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3	30% more
Extra Pancreatic Complications	
2	One or more of pleural effusion, ascites vascular complications, parenchyma complications and gastro intestinal tract involvement.
Total Score-10	
0-3	Mild
4-6	Moderate
7-10	Severe

IMAGING IN NECROTIZING PANCREATITIS:

COMPUTED TOMOGRAPHY:

CT is the primary imaging modality used to assess the morphologic features of necrotizing pancreatitis . In addition to establishing the diagnosis, CT can be used to define the extent and severity of necrotizing pancreatitis and to evaluate for complications, interval change, and treatment response. CT is the most established imaging technique for characterizing the severity of necrotizing pancreatitis, with findings having been shown to correlate with outcome (. Balthazar et al established a CT severity index that graded pancreatitis based on the degree of inflammation, presence of fluid collections, and extent of necrosis. A higher CT severity index score is associated with increased morbidity and mortality. A modified CT severity index by Mortele et al includedextrapancreatic complications (eg, ascites) and vascular complications in the grading system and found that the inclusion of these entities resulted in a stronger correlation with patient outcome.

Although CT can be used to accurately identify necrosis 72 hours after its onset, necrosis cannot be excluded if CT is performed earlier . As a result, to determine whether necrosis is present, CT is ideally performed no earlier than 3–5 days after presentation . the CT protocol for suspected or known necrotizing pancreatitis involves administering water orally and scanning the abdomen and pelvis 40 seconds after the intravenous administration of 100 mL of contrast material (370 mg/mL) at a rate of 3–5 mL/sec. Images are reconstructed at 2-mm intervals in the axial, coronal, and sagittal planes. For established cases of necrotizing pancreatitis, follow-up CT is performed when there is deterioration in the clinical status of the patient or a suspected complication, and as a baseline study prior to discharge or planning intervention .

At approximately 40 seconds after intravenous contrast material administration, normal pancreatic parenchyma demonstrates maximum enhancement (typically, 100–150 HU); this period is considered the pancreatic parenchymal phase. Pancreatic necrosis is suspected when any region of pancreatic parenchyma demonstrates an attenuation of less than 30 HU during the pancreatic parenchymal phase . Although pancreatic necrosis may initially appear homogeneous, the regions of necrosis can become heterogeneous as necrotic tissue gradually becomes liquefied . The severity of necrotizing pancreatitis at imaging is determined on the basis of the extent of parenchymal involvement by necrosis (ie, <30%, 30%–50%, and >50%) . When the extent of parenchymal involvement is less than 30%, the low attenuation due to decreased enhancement of the small region of necrosis may mimic the low attenuation of the gland seen with acute interstitial edematous pancreatitis, making the diagnosis of necrosis less reliable. In these cases, follow-up CT may be required .

Peripancreatic necrosis is a more difficult diagnosis to make at CT, since this modality is not able to demonstrate the presence or absence of fat perfusion. Therefore, the diagnosis of peripancreatic necrosis is suggested by the presence of increased attenuation, linear stranding, and fluid collections interspersed among the peripancreatic fat. Recognition of peripancreatic necrosis is difficult in the first week after onset because the increased attenuation, linear stranding, and fluid collections associated with acute interstitial edematous pancreatitis can have a similar appearance. However, the diagnosis of peripancreatic necrosis may be favored when the regions of increased attenuation have a heterogeneous appearance. After 1 week, the heterogeneous peripancreatic fat and the liquefied components among the fat become more apparent, so that peripancreatic necrosis can be diagnosed with greater confidence . Combined necrosis is diagnosed when imaging features of both parenchymal and peripancreatic necrosis are present . After 4 weeks, the acute pancreatic or peripancreatic inflammation and collections generally evolve into WON, which appears at CT as a heterogeneous or homogeneous collection with a well-defined wall . Approximately 60% of ANCs

evolve into sterile Walled Of Necrosis, 20% are complicated by infection, and the remaining 20% resolve spontaneously .

MR IMAGING:

Although MR imaging is not the first-line imaging modality for evaluating patients with suspected acute necrotizing pancreatitis, it is an acceptable alternative to CT in patients with an allergy to iodinated contrast material. Because imaging may be performed repeatedly, MR imaging may be preferred in young andpregnant patients to minimize radiation exposure. Unenhanced MR imaging can be used in patients with renal impairment . In addition, MR imaging is more sensitive than CT for detecting gallstones and hence is preferred in patients with suspected choledocholithiasis.

When catheter drainage of a fluid collection is contemplated, MR imaging may be helpful in assessing the collection's amenability to drainage by identifying non liquefied material (eg, debris or necrotic tissue) that is difficult to remove with percutaneous catheter drainage (PCD) alone . If non liquefied material is present, endoscopic necrosectomy or surgical débridement may be preferred . MR imaging is less sensitive than CT for detecting gas in collections, the presence of which can suggest infection .

MR imaging protocol for evaluating pancreatitis includes axial and coronal single-shot fast spin-echo T2-weighted, axial fat-saturated fast spin-echo T2-weighted, gradient-echo in-phase and opposed-phase, unenhanced fat-saturated gradient-echo T1-weighted, . Heavily T2-weighted coronal two- and three-dimensional MR cholangiopancreatography (MRCP) images are also acquired.

Peripancreatic stranding, fat heterogeneity, and necrotic collections such as an ANC and WON are best assessed on T2-weighted images, with liquefied components appearing hyperintense and non liquefied components appearing hypointense. WON demonstrates a well-defined, T2-hypointense enhancing wall . MR imaging is more sensitive than CT for the detection of hemorrhage, which is best seen on T1-weighted images . MRCP is useful for detecting choledocholithiasis and mass effect on the common bile duct (CBD), evaluating the integrity of the pancreatic duct, and detecting communication of a collection with the pancreatic duct.

Transabdominal Ultrasonography:

Transabdominal ultrasonography (US) has a limited role in the evaluation of patients with necrotizing pancreatitis. Compared with CT, US is more sensitive for detecting cholelithiasis but less sensitive for detecting distal choledocholithiasis . US has a limited role in evaluating the extent of necrosis and complications, since these findings are often obscured in patients who are large or have large amounts of bowel gas. However, in patients with contraindications for both CT and MR imaging, US may be useful for demonstrating the presence of non liquefied material within a collection.

EndoscopicRetrogradeCholangiopancreatography:

Endoscopic retrograde cholangiopancreatography (ERCP) has no primary role in characterizing the morphology of necrotizing pancreatitis and could lead to complications such as pancreatitis exacerbation, bleeding, and bowel perforation. Because of its less invasive nature, MRCP is preferred for detecting choledocholithiasis and pancreatic ductal strictures or disruptions. Hence, ERCP is generally reserved for therapeutic applications such as CBD stone removal or pancreatic duct stent placement used to treat strictures and disrupted ducts.

Endoscopic US:

Endoscopic US involves using an echoendoscope that generates high-frequency sound waves, which pass through the wall of the stomach or duodenum to help evaluate the pancreatic parenchyma and ductal system. Endoscopic US combines the diagnostic capabilities of US with the interventional advantages of endoscopy. Like transabdominal US, endoscopic US can be used to identify the non liquefied components of collections in preparation for endoscopic drainage and débridement. Endoscopic US is also sensitive for detecting CBD stones, without the risks associated with ERCP .

ComplicationsofNecrotizingPancreatitis:

Infection

Infection occurs as a complication in 20% of patients with necrotizing pancreatitis and is thought to result from bacterial translocation from the gut to adjacent necrotic pancreatic parenchyma. The most common bacterial organisms include Escherichia coli, Staphylococcus aureus, and Enterococcus faecalis, although several other organisms may be found . Infection can occur at any time during the course of the disease but most commonly occurs 2–4 weeks after presentation . Patients with infected necrosis typically present with fever, tachycardia, and an elevated white

blood cell count. Clinical presentation alone is not diagnostic for infection, and patients with sterile necrosis may present with similar symptoms. At imaging, the presence of gas within a collection suggests infection, although gas is found in a minority of cases of confirmed infection (12%–22%), and the absence of gas does not signify the absence of infection . Gas can also be found in uninfected collections as a result of gastrointestinal fistulas . Because there are no symptom constellations or imaging findings that are diagnostic for infection, imaging-guided percutaneous needle aspiration is indicated in patients suspected of being infected . Infected necrosis carries a high mortality rate of 25%–70%; therefore, the diagnosis needs to be pursued aggressively when infection is suspected. When infection is confirmed, some form of intervention is usually indicated .

InflammationandMassEffectonAdjacentorgans:

Necrotizing pancreatitis, with its associated inflammatory changes and collections, may displace and compress adjacent organs. Obstruction of the stomach or bowel and hydronephrosis are possible complications of the mass effect caused by nearby collections and inflamed fat . Inflammatory changes may also secondarily cause bowel wall thickening, mural hyperenhancement, and adjacent fat stranding . Patients with severe gastrointestinal tract obstruction or large abdominopelvic fluid collections are at risk for abdominal compartment syndrome, in which increased intraabdominal pressure results in organ ischemia and further tissue necrosis.

Biliary Obstruction:

Biliary obstruction can result from choledocholithiasis, mass effect from pancreatic inflammation or a collection, or biliary stricture from exposure to pancreatic proteolytic enzymes. A strictured bile duct may appear tapered, compressed, or simply occluded with upstream biliary dilatation, and it may or may not demonstrate mural enhancement . MRCP is particularly helpful for delineating the biliary system, identifying the narrowed or occluded segment, and identifying the cause of the obstruction .

Pancreatic Duct Stricture:

Main pancreatic duct stricture is a late complication of necrotizing pancreatitis. Strictures develop secondary to fibrosis from resolving inflammation, or as a result of healing after the successful interventional drainage of a necrotic collection . Strictures may be single or multiple, may result in upstream dilatation of the pancreatic duct, and can be diagnosed with CT, MRCP, ERCP, or endoscopic US.

Disconnected Pancreatic Duct:

Disconnected pancreatic ducts result from necrosis of the central pancreas (commonly the neck or body) or from a therapeutic intervention that disrupts the main pancreatic duct, and they occur in approximately 40% of patients with pancreatic necrosis . When there is residual upstream functioning pancreatic tissue, a disrupted duct results in persistent leakage of pancreatic fluid from the viable upstream pancreas and leads to accumulation of fluid around the pancreas, pancreatic ascites, or a pancreaticopleural fistula. Although most, if not all, fluid collections are the result of some form of communication with the pancreatic ductal system, disruption of the main duct often leads to persistent and growing collections around the pancreas. At CT or MR imaging, a disrupted duct is suggested by a large or growing collection involving the neck or body of the pancreas and a viable segment of upstream body or tail. The duct in the upstream pancreas may or may not be dilated and may be seen communicating directly with the collection; disruption is suggested when the duct is oriented perpendicular to the collection . A disrupted duct and the presence of a fistula can also be suspected following PCD when there is persistent catheter drainage of amylase-rich fluid, despite the resolution of the fluid collection. Both ERCP and MR imaging with MRCP can help confirm the disruption and identify the site of the fistula .

Pseudoaneurysm:

A pseudoaneurysm develops when an arterial vessel wall is weakened by pancreatic proteolytic enzymes and is a typically late and potentially life-threatening complication of pancreatic necrosis . At CT, MR imaging, or angiography, a pseudoaneurysm appears as a focal outpouching of a vessel within the necrotic region . A mural thrombus may also be seen . At US, turbulent arterial flow may be seen within an anechoic structure . The artery that is most frequently involved by pseudoaneurysm formation in the setting of necrotizing pancreatitis is the splenic artery (up to 10% of patients), followed (in descending order) by the gastroduodenal, pancreaticoduodenal, hepatic, and left gastric arteries . Pseudoaneurysms can rupture into the

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necrotic collection, gastrointestinal tract, peritoneum, or pancreatic parenchyma .

Hemorrhage:

Spontaneous hemorrhage in necrotizing pancreatitis can occur from erosion of vasculature by necrosis or from rupture of a pseudoaneurysm or varices. Hemorrhage can occur within the pancreatic parenchyma, fluid collections, or the gastrointestinal tract . Although its overall rate of occurrence in pancreatitis is not known, spontaneous hemorrhage probably occurs in approximately 1%–5% of cases; mortality rates of 34%–52% have been reported . The splenic artery, portal vein, splenic vein, and other smaller peripancreatic vessels are the most common sources of bleeding . Hemorrhage manifests at CT as a region of high attenuation, typically in an area of necrosis . At MR imaging, the appearance of hemorrhage on T1- and T2-weighted images varies with the age of the bleeding; subacute hemorrhage appears T1 and T2 hyperintense.

Venous Thrombosis:

Venous thrombosis results from a multifactorial process involving local prothrombotic inflammatory factors, reduced venous flow, and mass effect on a venous structure from adjacent necrotic tissue and collections. Acute venous thrombosis appears as focal or complete nonenhancement of an expanded venous structure. In chronic cases, scarring results in a diminutive, less well-visualized vein and multiple collateral vessels . The splenic vein is the most common site for thrombosis (up to 23% of cases of acute pancreatitis); the superior mesenteric and portal veins are less commonly affected . Splenomegaly may result, and collateral vessels may increase the risk of bleeding during subsequent intervention or surgery.

DiagnosticProblemsinNecrotizing Pancreatitis:

Acute Peripancreatic Fluid Collection versus ANC:

An acute peripancreatic fluid collection (APFC) is a collection that develops within 4 weeks after onset of acute interstitial edematous pancreatitis, whereas an ANC is a collection that develops within 4 weeks after onset of acute necrotizing pancreatitis . Both collections have no discernable walls . An APFC contains amylase- and lipase-rich fluid and develops as a result of pancreatic or peripancreatic inflammation, or from a ruptured pancreatic ductal side branch . On the other hand, an ANC is a collection that contains both liquefied and nonliquefied necrotic material .Distinguishing an APFC from an ANC in the first week after onset may not be possible with CT; both collections may be homogeneous and nonenhancing and demonstrate fluid attenuation . If a collection appears heterogeneous, or if hemorrhage or fatty tissue is present, it can be classified as an ANC . Moreover, if pancreatic parenchymal necrosis is present, an associated collection is classified as an ANC. Distinguishing an ANC from an APFC is more challenging when necrosis is solely peripancreatic. Beyond 1 week after onset, collections associated with peripancreatic necrosis become more heterogeneous and are more readily distinguished from APFC . MR imaging is more helpful than CT in this regard, since it can be used to detect nonliquefied components that allow classification of a collection as an ANC .

Pseudocyst versus WON:

Pseudocysts and WON are both late-phase (>4 weeks after onset) collections that develop over time from nonnecrotic (APFC) and necrotic (ANC) collections, respectively . Both pseudocysts and WON have well-defined, nonepithelialized enhancing walls. Pseudocysts contain homogeneous fluid (hypoattenuating at CT, T2 hyperintense at MR imaging) and are only peripancreatic . WON contains necrotic material—often a mixture of fat and fluid—and can involve both pancreatic and peripancreatic tissue. The diagnosis of WON is also favored when a pancreatic collection grows, extends to the paracolic space, and has an irregular border . However, any collection that occupies or replaces pancreatic parenchyma is classified as WON, regardless of its appearance . Pseudocysts are more likely to be associated with main pancreatic ductal dilatation (>3 mm), possibly as a result of the compression of pancreatic parenchyma. In patients with WON, ductal dilatation is less likely to occur because the pancreatic fluid simply leaks into the collection

Differentiating a pseudocyst from WON is important because WON typically does not respond to endoscopic cyst gastrostomy or PCD with small-bore (10-F or smaller) catheters. Treatment of WON typically requires surgical or endoscopic necrosectomy, or PCD using large-bore catheters with frequent irrigation to evacuate the non liquefiedcomponents .

Sterile versus Infected Necrosis:

As described previously, the presence of gas in an area of necrosis, in the absence of previous intervention or spontaneous communication with the bowel, may indicate the presence of infection. CT attenuation measurements cannot be used to distinguish sterile from infected necrosis . With any signs or symptoms of infection, imaging-guided

percutaneous needle aspiration for Gram staining and culture is needed to definitively diagnose infection within a region of necrosis .

Postnecrosectomy Changes versus Infected Collection:

Necrosectomy is a procedure in which necrotic pancreatic or peripancreatic tissue is removed, along with drainage of accompanying fluid collections. Postnecrosectomy imaging findings may mimic infection, since the surgical bed and residual collection may contain tissue, fluid, and gas. The presence of gas is expected when there are indwelling percutaneously placed or surgical catheters or if a stent has been placed between the collection and the stomach following endoscopic necrosectomy. Postnecrosectomy changes are expected to resolve; any interval increase in the size of a collection or formation of a new collection raises the possibility of infection. Microbiologic analysis is necessary to confirm infection .

II. REVIEW OF LITERATE

Loicviremounix MD et.al ,in a prospective of 90 cases of acute pancreatitis condoucted from January 2002 to April 2004 concluded that Non Enhanced MRI seems to be a relative metod of staging Acute pancreatitis in comparison with CECT[73]

Lecesneet.al, in a prospective study conducted in 1999 compared contrast enhanced CT and andnon contrast enhanced MRI concluded that MRCP could be an alternative to contrast CT for intial staging of acute pancreatitis[74].

Sameer Raghuwanshi et .al, in a prospective study of 50 patients conducted from November 2013 to November 2015 concluded that modified ct severity index shows a strong correlation in assessing clinical outcome of patients of acute Pancreatitis.

Thomas L. Bollen et. al, In a Prospective study of 397 consecutive cases of acute pancreatitis between June 2005 and December2007 compared CT severity index Modified CT severity index and APACHE II Concluded that there is no significance difference between CTSI and Modified CTSI in evaluating severity of Acute Pancreatitis. Compared with APACHEII both CTSI were more accurately diagnosed clinically severe disease and had better correlation with need for intervention in pancreatic infection[76].

In 1990 Belthazaret.al , introduced the CTSeverity Index for acute pancreatitis for AP, Wich correlated well with morbidity morbidity and mortality and hospital stay[76].

Leung TK et. Al, in a retrospective study in 2005 evaluated 107 patients and concluded that Belthazar CTSI is superior to Ransons criteria and APACHEII scoring system in predicting out come in patients with Acute Pancreatitis[77].

Gurleyik.G et.al, In a prospective study of 55 patients concluded that CTSI is a reliable method for staging severity of Acute Pancreatitis([78].

I A Banday et.al, in a prospective study of 50 patients in 2015 compared MCTSI and CTSI concluded that MCTSI is a simpler tool and more accurate than Belthazar CTSI in Assessing in severity of Acute Panceatitis[79].

Mortele et.al, proposed a new modified CT severity index in 2004 and conducted a prospective study of 266 patients, compared results of old CT severity index and new modified Ct severity index and concluded that modified CT severity index correlate more closely with patient outcome indices than CT severity index[80].

Bishwanthsheshu et.al, in a prospective study of 60 patients of Acute pancreatitis conducted from march 2014 to march 2016 compared severity of acute pancreatitis using CT severity index and modified Ct severity index and correlate the results with severity grading of Revised Atlanta classification. Concluded that Ct severity index and modified CT severity index showed significant correlation with clinical outcome parameters as well as good concordance with grading of severity as per revised Atlanta classification, also concluded. Modified Ct severity index showed a higher sensitivity where asCT severity index showed higher specificity in differentiating between mild acute pancreatitis and moderate severe disease[81].

ShivanandMelkundi et.al, in a prospective study of 100 patients of acute pancreatitis evaluated complication of acute pancreatitis with CT severity index and modified CT severity index, concluded that there was a significant correlation of severity and outcomes of Acute pancreatitis it is with modified CT severity index than CT severity index[82].

De waele JJ et.al, based on a prospective study of 45 patients of acute pancreatitis in 2007 concluded that Extra pancreatic inflammation on abdominal CT as early predictor of acute pancreatitis and introduced a new scoring system called as Extra Pancreatic Inflammation on CT (EPIC) score.

MERIYANA ARAVANIRAKISet.al, in a prospective study of 30 patients of Acute Pancreatitis conducted in 2004 concluded that MRI is a reliable method of staging severity of acute

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pancreatitisand also concluded MRI is superior in assessing pancreatic duct injury

III. MATERIALS & METHODS

STUDY DESIGN It is a prospective observational study

Patient selection:

All the patients who came with clinical suspicion of Acute Pancreatitis to the department of radio diagnosis for CT were included in the study. They were subjected to CECT.

Technique &inclusion & exclusion criteria:

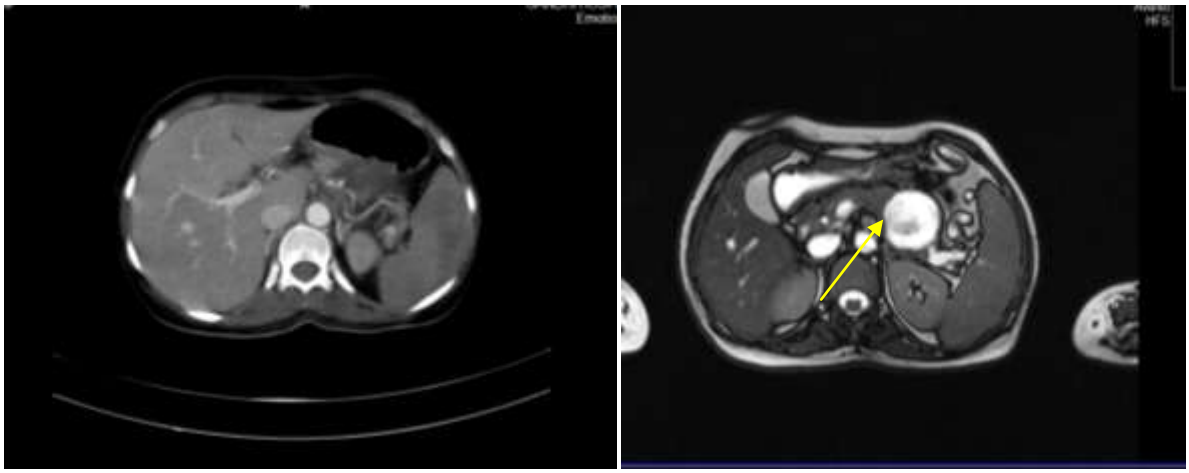
CECT was done on 16slice (SomotomeEmotion of Siemens' Ltd)CT machine. Two protocols were used one is plain ct fallowed by contrast scan(with iv administration of Iohexol } in arterial phase 15 minutes after oral administration of 500ml of water fallowed by 40 secdelay scan in younger patients. Another protocol used wasTri phasic CT after oral administration of 500ml of water in older patients and if clinician specifically requested.

Of total 98 cases 16 cases had normal pancreas were also excluded from the study. Remaining 82 cases had acute pancreatitis, of which 42 patients had no necrosis were also excluded from study.

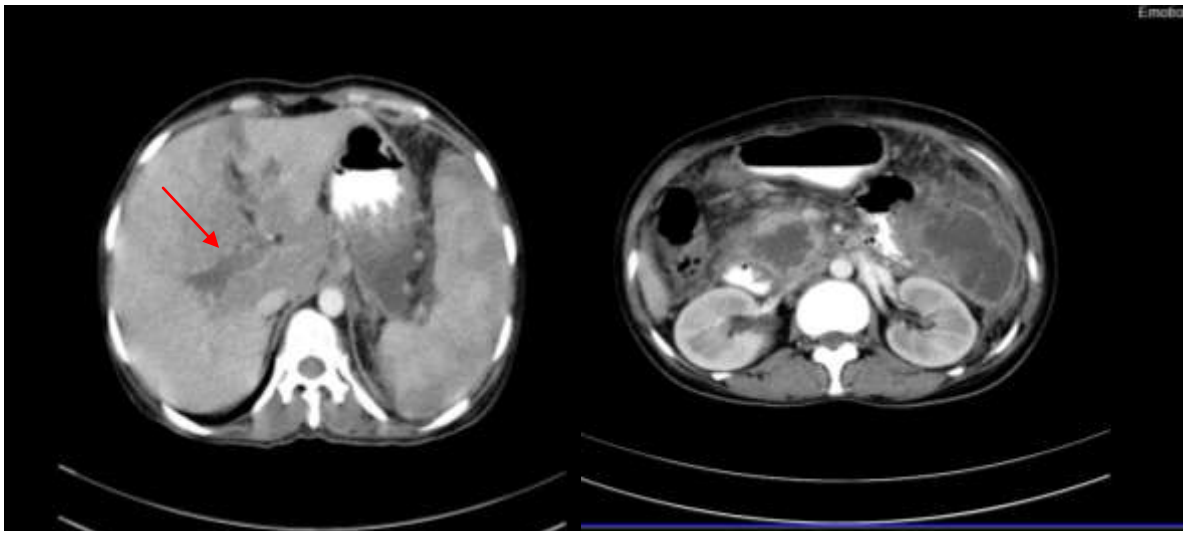
Total 40 cases were included in the study were subjected to MRCP and MRI Abdomen on 1.5Tesla MRI machine(Avanto of seamen's Ltd).MRI was performed using Abdominal coil. A localizer scan was done followed by Axial &coronalT2 Haste (TR 1000-1500,TE100),Axial& coronalT2Haste Fat saturated, Axial &coronal T2 trufi(TR4-5,TE2-3),T2 TSE Respiratory gated3D(TR2000-3000,TE200) images were done for all patients.

MRI imaging findings were compared with CECT imaging findings severity assessment was done based on Modified CT severity index.

CASES 1



CT and corresponding T2 Haste MRI images of pancreatic necrosis in the tail of pancreas with psudo aneurysm of splenic artery(yellow arrows)



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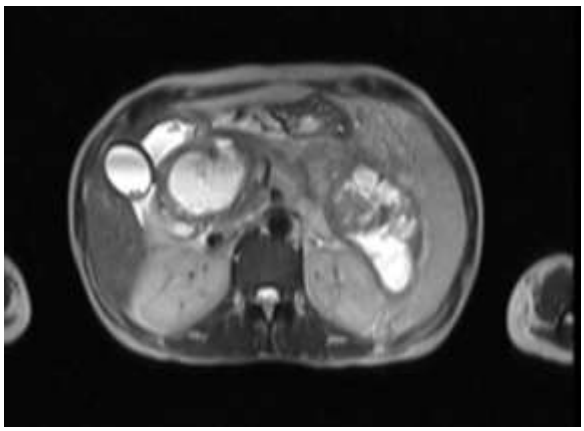
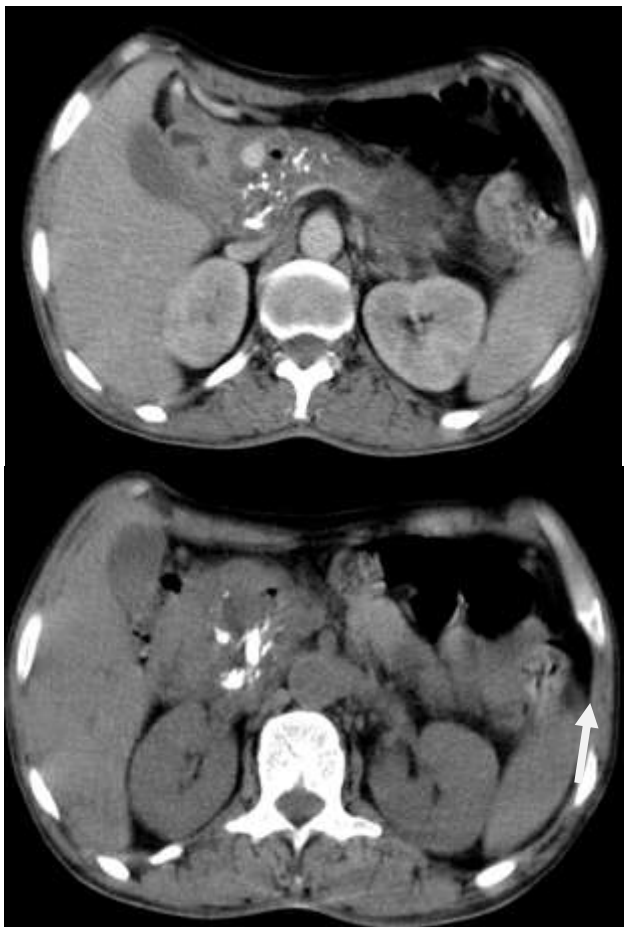


AxialCT and corresponding T2 Haste MRI image showing severe necrosis of pancreas. CT image at different level showing portal vien thrombosis(Redarrow).



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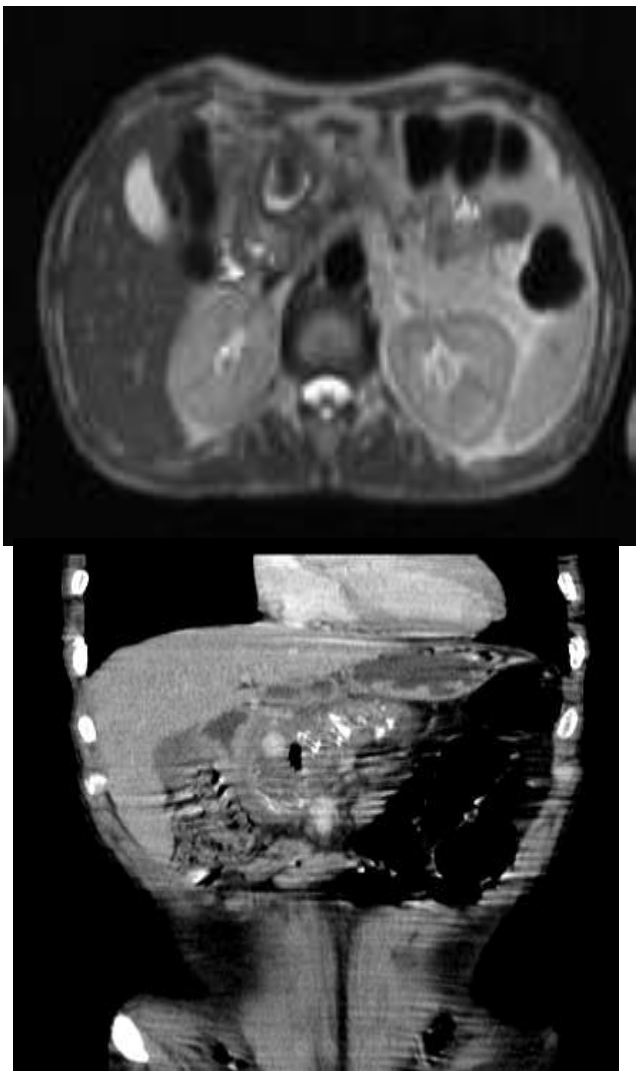
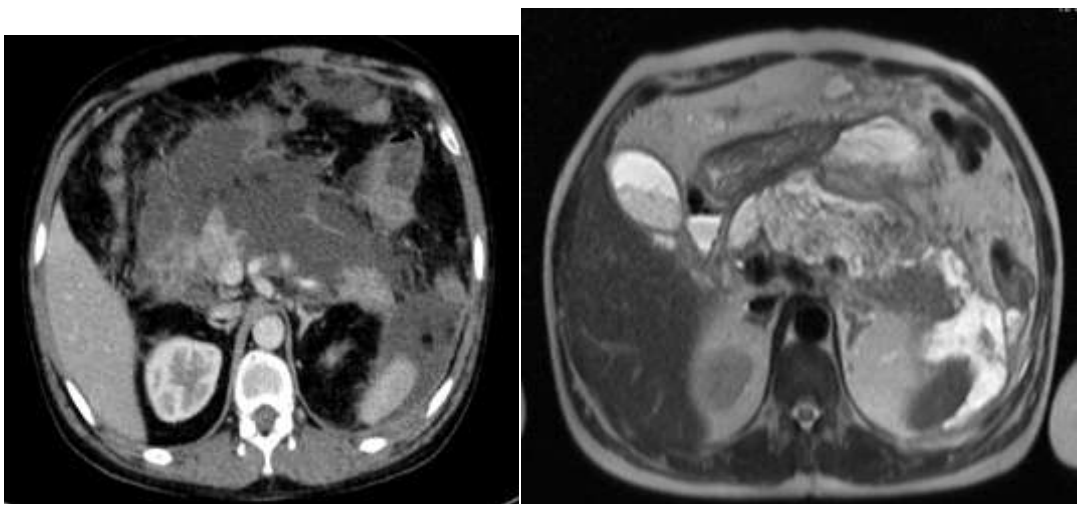


Axial, Plain &contrast and coronal contrast CT images and corresponding Axial Haste MRI image showing psudo aneurysm of gastro duodenal artery.(white arrows)



Axial contrastCT&corresponding T2 Haste MRI Images showing necrosis of body &tail

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IV. DISCUSSION

The present study was undertaken to evaluate role of contrast enhanced CT, (CECT) MRI in diagnosing and prognosticate Acute hecrotizing pancreatitis and to study advantages, disadvantages of CECT, MRI in diagnosis and prognonosis of Acute pancreatitis.

In study population of 82 patients of Acute pancreatitis 40no of patients had necrosis(49%). In the studyconducted by Sameer Raghuwanshi et al, 50% of cases had necrosis.

The study group consisted of 31 Male and 9 Female with a male female sex ratio of 3.4:1 In a prospective study of 50 patients on Acute pancreatitis done by Sameer Raghuwanshi, et al. had a male to female ratio of 2:1and Silverstein et al., had 2:1 ratio in his study. Most common etiology was alchohol abuse wich was associated with 30(75%) cases fallowed by Gallstones wich was seen in 7cases(17.5%).

Infected necrosis is present in 4cases(1 %) in the present study. In the Study conducted SameerRaghuwanshi, et al, Infected necrosis was seen in 8% of cases.

Necrosis appear as non enhancing area with in the pancreas on CECT18no.of(45%)patients had less than 30%,4no.of(7.5%)of patients had 30-50% necrosis18no.of(50%) case s had>50%necrosis. In the study conducted by SameerRaghuwanshi et al, 24% had less than 30% necrosis, 20% patients had 30-50% necrosis, 56% had more than 50% necrosis.

Extra pancreatic and systems complication occurred in 33(82.5%) of patients in the present study. In the study conducted by Sameer Raghuwanshi et al, Extra pancreatic complications wasseenin 68% of patients.

Most common extra pancreatic complication was plural effusion was seen in19 no(47.5%) of cases of which left side was more common. In the study conducted by Sameer Raghuwanshi most common Extrapancreatic complication was also plural effusion which was seen in 46% cases and the study conducted by Belthazar et al also the most common Extra pancreatic complication is plural effusion present in 43% of cases, more common in left side.

Ascitis was found in14(35%) patients in our study which was foundin 34% Sameer Raghuwanshi"sStudy

Vascular complication in 11no (27.5%)of cases. whare as in Thomas L.Bollenet.al,study vascular complications were seen in 8% of cases.Most common vascular complication is venous thrombosis Which is seen in6 no(15%) of cases. Venous thrombosis is appears as Hypo dense filling defect within the vein onCECT. Most common venous thrombosis wassplenicvein thrombosis, followed by portal vein . Isolated splenic vein thrombosis is seen in2(5)%case.portal vein thrombosis is seen in1(2.5%)case. Combined thrombosis is seen in3(7.5%%) cases.

Second most common vascular complication was pseudo aneurysm formation which was seen in 3 cases(7.5%) of which one case is involving gastroduodenal artery, 2 cases involving splenic artery.

Thrid most common vascular complication was haemorrhage which is seen in one case(2.5%)

Severity was graded using modified CT severity index. 29 no of cases(72.5%) have severe pancreatitis (score 8-10). 11no of cases(27.5%) were categorized in moderate pancreatitis (score 6-8). In Sameer Raghuwanshi et al study severe pancreatitis is seen in 44% cases and moderate pancreatitis is seen in 38% of cases, mild pancreatitis was seen in 18% of cases.

Major disadvantages of CT was contrast CT cannot be done in patients with Renal failure, patients with contrast allergy, pregnant patients, another disadvantage is radiation exposure. MRI can be done in such cases.

MRI was done in MR Aventos 1.5T machine. Each case was compared with contrast enhanced CT.

total no of patients diagnosed as having less then 30% necrosis on MRI was20 where on CECT was18.Severity is specifically, positive predictive value and negative predictive value,accuracy of MRI in detecting less than 30% necrosis is 100%,90.91%,90%,100%,95% Respectively.

4no.of cases had 30-50% necrosis on CECT,3no.of cases had 30-50% necrosis of MRI , severity specificity. Positive predictive value and negative predictive value accuracy ofof MRI indetailed 30-50% necrosis over CECT was 75%, 100%,100%, 97.3%,97.5%% Respectively. 18no.of cases had necrosis more than 50% necrosis on CECT.Where as MRI shown necrosis of >50% in 17no .of cases. Sensitivity ,specificity and positive predictive value and negative predictivevalue ,accuracyin. detecting>50%necrosiswas94.4%,100%,100%,95.6 %, 97.5%Respectively

Peripancreatic collections was seen in CECT 27no of cases. Where asPeripancreatic collections were seen in26 no of cases on MRI.

Sensitivity,specificity, positive predictive value, negative predictive value,accuracy of MRI in

detecting peripancreatic collections was96%,100%,100%,92.8%,86. Respectively. Sensitivity can be increased by using fat saturation sequences

In comparison to CECT, MRI more severity in detecting plural effusion.

Ascites was seen in14no of cases on CECT where asAscites was see in 14 no of cases MRI. Severity and specificity, positive predictive value,accuracywas,.100%,100%,100%,100%. Respectively.

Pseudo aneurysm was seen in3no of cases on CECT where as on MRI pseudoaneurysm was seen in 3no of cases. in the experienced had sensitivity detecting pseudo aneurysm was 100%.

Venous thrombosis was seen in 5no. of cases on CECT where as Venous thrombosis seen in 3 no. of cases on MRI. Severity and specificity, positive predictive value, negative predictive value, accuracyare93.7%,100,100,97.1%,98% Respectively.

Thrombosis appeared as loss of flow voids on HASTE(Half Fourier Acquisition Single Shot Turbo Spin Echo) and as filling defect in TRUFI (true fast imaging with steady-state free precession).

Hemorrhagic was seen in2 cases on CECT&on MRI. Hemorrhagic fluid collection are more evident on MRI than CT appear as T1 Hyper intensity and T2 Hypo intensity.

Sensitivity, specificity ,Positive predictive value &negative predictive value of MRI in predicting moderate disease was100%,93.9%,77.78%,100%,95% respectively.

MRI was more accurate in diagnosing Gall Bladder calliculi and ductal communication.

Based on above observation we can conclude even thoughCT is more sensitive and specific ,MRI had comparable sensitivity and specificity in detecting necrotizing pancreatitisand its complications.

MRI has some limitations as it's availability less and it's taken more time, less cost effective, needs breath holding and cannot be done in patients with metallic implants.

MRI is preferred over CT in cases of Renal failure, pediatric population, pregnant female, contrast allergy, and rule out CBD and pancreatic duct and Gall Bladder pathology.

V. SUMMARY & CONCLUSIONS:

1. CECT is preferred investigation in detecting necrosis pancreatitis and its complication and grading of severity
2. MRI has comparable sensitivity with CECT in detecting necrotizing pancreatitis, its complication and severity grading.
3. In cases where CECT cannot be done (Renal failure, paediatric population, pregnant, contrast allergy) MRI can be used as Imaging Investigation.
4. Familiarity with MR appearance improves accuracy of detection of acute necrotizing pancreatitis and its complication.

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