Ct Imaging a Platinum Standard Technique to Diagnose Acute Pancreatitis

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Abstract

*Treatment of patients suffering from acute pancreatitis is beset with initial assessment of disease severity and diversity. Several factors attribute to cause acute pancreatitis which include alcohol, cholelithiasis, trauma, iatrogenic (e.g.post-ERCP) as well as malfunctioning of metabolism in the form of hyperlipidaemia and hypercalcaemia. Infections alike mumps and cytomegalovirus could also be the casuative agents of pancreatitis. Staging at an early period is proportionally related to presence and degree of systematic failure (cardiovascular ,pulmonary ,renal) and severity and extent of pancreatic necrosis in a tremendous form. Severe pancreatitis is prevalent in 20 -30 % population of patients inflicted with acute pancreatitis and is characterized by protracted clinical course, multiorgan failure and pancreatitic necrosis . Some specified peculiar drugs don't lag behind to damage pancreas to a considerable extent due to their side effects on this vital and sensitive organ. Only In 10–30% of cases no virtual change has ever been noticed, thus apparently the same disorder may be categorized under idiopathic pancreatitis, although severe abnormality due to congenital duct anomalies cannot be ruled out. Capacity and potential for complete healing associated with inflammation of pancreas has been especially noticed in the patients aging about 60 years. Pathophysiology reveals pancreatic auto digestion with activated pancreatic enzymes escaping the ductal system and lysing tissue of pancreas and adjacent organs. CT imaging should be repeated if clinical picture envisages abrupt changes, such as occurrence of sudden or intermittent fever or drop in haematocrit***.**

Keywords: *Oedema, Necrosis, Pancreatitis, Contrast enhanced computed tomography, Peripancreatic, Walled-Off Necrosis, Pseudocyst*

I. INTRODUCTION

The latest and revised Atlanta classification (2012) of acute pancreatitis categorises the malcondition into interstitial oedematous pancreatitis and necrotising pancreatitis, (formerly termed mild and severe acute pancreatitis).[1] This morphological classification system has been derived based on findings on contrast-enhanced CT. Summing up the imaging findings in interstitial oedematous pancreatitis include focal or diffuse alternation of the gland, with normal homogeneous enhancement or slightly heterogeneous enhancement of the pancreatic parenchyma, which is causative formidable concern of oedema. In interstitial oedematous pancreatitis, which constitutes 70–80% of cases, the ultrasound (US) images can be normal but common characteristics may involve generalised (or less commonly, focal) enlargement of the gland with reduced reflectivity. Recent UK guidelines further suggest that immediate CT is needed if the clinical and biochemical findings have been found to be inconclusive, especially if the symptoms arouse the possibility of an alternative abdominal emergency.[1] CT is the most solid and vividly viewed imaging technique for staging the acute pancreatitis, if performed with IV contrast medium. Even not depending on, how CT has already been recommended for detection and staging pancreatic cancer, a single venous phase CT image (100–150 mL at 4 mL/s with a fixed delay of 70 s) would be ample to stage acute pancreatitis, and unenhanced or arterial-phase imaging does not make the same necessary. In the clinical context of acute pancreatitis, CT protocol design must be taken into consideration along with radiation exposure, as many patients with severe acute pancreatitis are even in low age group and would require several sessions of CT imaging during the course of their investigation. The clinical grading of preponderance has utilised a number of scoring systems such as Ranson's, APACHE-II, Marshall scoring system or BISAP score. Pancreatic parenchymal necrosis with peripancreatic necrosis establishes its distinguishing characteristic features on imaging by a combination of imaging characteristics observed with pancreatic parenchymal necrosis alone and peripancreatic necrosis. In (mild) interstitial oedematous pancreatitis, proteolytic enzymes devour the acinar cell, causing oedema and swelling of the gland, but the inflammatory response is not a strong and lucid characteristic to expose tremendous necrosis. Peripancreatic necrosis has been observed as heterogeneous areas of non-enhancement that contain non-liquefied materials that furnish a sure sign pertaining to this disorder and appropriate management. In the 2012 classification, necrotising pancreatitis was further classified into (1) parenchymal necrosis alone, (2) peripancreatic necrosis alone and (3) a joint type (areas of pancreatic parenchymal and peripancreatic necrosis) with and

without infection. The pancreatic margins may be difficult to define and interpret ,but still peripancreatic fluid can be visualised. The prevalence of necrosis (necrotizing pancreatitis) can formulate presumptions and indicates that the inflammatory response has triggered cellular apoptosis. Necrotising pancreatitis has been observed in less than 5% of the patients and can be clearly depicted on contrast enhanced CT images due to lack of parenchymal enhancement. The real determinant of morbidity and mortality in patients with acute pancreatitis is the emergence of pancreatic necrosis. Conventional US has not been found to be suitable to establish certainly the complications involved in pancreatitis due to inflammatory response which triggers cellular apoptosis. The CTSI is directly related to morbidity and mortality .Nowadays , it has recently been shown that CT scoring does not supersede clinical scoring systems (BISAP, Ranson's, APACHE-II) for identification of patients with severe pancreatitis. Contrast-enhanced CT is the prime concern in investigation of choice to assess the prevalence and extent of pancreatic necrosis, the extent of peripancreatic inflammation and necrosis and the presence of fluid collections. The CT severity index (CTSI) for staging of acute pancreatitis has already been prepared after considering the volume of collected fluid and the presence or absence of pancreatic necrosis. Serum markers, such as C-reactive protein is confined to appropriately diagnose pancreatic necrosis but their usefulness is still limited to the extent of their specificity and/or availability.[1,2]

Latest Atlanta classification reveal that contrast enhanced CT is the primary concern for assessing and staging patients with acute pancreatitis, an assisting factor to assess complications and to be acquainted with proper response to treatment. All patients with acute pancreatitis need not require contrast enhanced CT imaging during initial stage, if findings are sorted promptly (in the first 24 h after clinical onset). Possibility cannot be ruled out that CT may underestimate the degree of necrosis and in most patients it will not be having full impact on its concrete management in the first few days. The optimal time for performance of contrast-enhanced CT imaging is more than 72 h after the onset of clear cut symptoms. On this burning issue there is an ongoing healthy discussion about the useful purpose of imaging in the diagnosis of acute pancreatitis. Although in mild acute pancreatitis US may suffice the purpose to rule out all sorts of complications and to clearly assess for etiologies such as gallstones, contrast-enhanced CT has emerged as the relevant diagnostic feature occurring in patients with suspected or confirmed acute pancreatitis. Most of the patients with persisting devour or new organ failure and those with continuing pain or appearance of sepsis will require a contrast-enhanced study , being the presence of necrosis as detected on CT has a marked level of coincidence with the risk of local and systemic factors or complications. It has been clearly revealed that early removal of bile duct stones (by ERCP) may have a deep impact on the prognosis in patients with biliary pancreatitis and obstructive jaundice. Thus ,MRCP (or if available endosonography) should be employed to detect choledocholithiasis and to provide genuine evidence in screening and selection of the candidates for ERCP, if performed without delay. MR imaging may also play a vital role in detecting non-liquefied material within a pancreatic or peripancreatic collection.[1]

II. INTERSTITIAL OEDEMATOUS PANCREATITIS

As previously reported, the imaging findings in interstitial oedematous pancreatitis include focal or diffuse enlargement of the gland, with normal homogeneous enhancement or slightly heterogeneous enhancement of the pancreatic parenchyma, which gives rise to an important concern related to oedema. It is still pertinent and even important to highlight, that on a contrast-enhanced CT image obtained in the first few days after presentation with acute pancreatitis, the pancreas may reveal elaborate and adequate enhancement of the pancreatic parenchyma, which cannot be surely characterised as either acute oedematous pancreatitis or necrotising pancreatitis. For grading of acute pancreatitis, a plan pertaining to the CT severity index has already been prepared, which duly takes into consideration the exact location of swollen gland, peripancreatic fluid collections and the presence of necrosis which further serves as an indicator for the prognosis. Under these circumstances it becomes more convenient for the radiologist to conclude that the CT is indeterminate and that contrast-enhanced CT imaging should be repeatedly ensued following 5–7 days, if the patient has not shown improvement up to the desirable limits. Fluid collections can be well observed and ascertained around the pancreas, in the pararenal space along fascial planes, at the mesenteric root or in the bursa omentalis.[1,2]

III. NECROTISING PANCREATITIS

This can be firmly considered as the hallmark of severe acute pancreatitis. The revised Atlanta classification further distinguishes three kinds of acute necrotising pancreatitis: (1) pancreatic parenchymal necrosis alone, (2) peripancreatic necrosis alone, and (3) pancreatic necrosis with peripancreatic necrosis. These three types can be sterile or infected.[1]

The emergence of peripancreatic necrosis alone can be considered as a much better prognosis than parenchymal necrosis. The extent of necrosis may be categorised into (1) less than 30%, (2) 30–50% or (3) more than 50% of the gland. Necrotic

glandular tissue may be observed as an area of non-enhancement within the pancreatic parenchyma. Surgical intervention is the method of choice, although recent studies have shown that a conservative approach avoiding open necrosectomy shows declining trend in the mortality rate considerably (8.3 vs 45%). With passage of time, non-viable or necrotic tissue pancreatic parenchyma or peripancreatic fat gets liquefied. Infected necrosis has been found in 20–70% of patients with necrotic pancreatic tissue and is highly responsible for an estimated 80% of deaths from acute pancreatitis. The availability of gas bubbles within an area of necrotic tissue (in the absence of recent intervention in the region or existence of a passage between gastrointestinal tract and the pancreatic collection/area of necrosis) indicates sure signs of infection. Thus necrosectomy now-a-days is reserved for patients synchronising with concomitant intra-abdominal complications not amenable to conservative therapy.[1,2]

Patients with more than 30% necrosis as established through CT evidence have an overall reported mortality rate upto the extent of 29%. In the study of Heiss et al. two CT features correlated with mortality: first, the presence of necrosis of more than one part (i.e. head, body or tail) of the pancreas and, secondly, the presence of distant fluid collections (in the posterior pararenal space and/or paracolic gutter). Detection of infected part or organ requires fine-needle aspiration. If infection is found to be persist within necrotic tissue, percutaneous drainage may not evacuate thick debris.[1]

A. Pancreatic and Peripancreatic Collections:

Acute pancreatitis may be associated with threshold of pancreatic parenchymal or peripancreatic collections. The acute form of collections have been termed as either acute peripancreatic fluid collections (APFCs) or as acute necrotic collections (ANCs), depending on the availability or non availability of pancreatic necrosis. Necrotising pancreatitis in its three dreadful forms shows clear resemblance and association with ANCs and after a period of 4 weeks by walled-off necrosis (WON). All of these collections can prove as dormant or infected. Thus, in the acute stage interstitial oedematous pancreatitis will be infiltered with APFCs and after a period of 4 weeks by a pseudocyst. APFCs, in particular are self confined and get reabsorbed within the first few weeks, and thereby infection may not be seen further. These collections therefore rarely require any sort of attention until these become infected.[1]

B. Acute Necrotic Collection (ANC)

Necrosis do persists in the pancreas within a month and a persistent collection of the fluid is termed as an ANC. The collected sample is liable to be contaminated to a variable extent due to emergence of pieces of debris and necrotic putrefied fluid material . An ANC establishes contact with the pancreatic duct via common passage. ANC shows progressive liquefaction and putrefaction which covers 3-6 weeks. A subtle change to be described in the latest classification renders any sort of collection derived through pancreatic parenchyma envisaged to be called as an ANC and not a pseudocyst.[1,2]

C. Walled-Off Necrosis (WON)

Acute necrotic collection associated with NP even after 4 weeks following initial emergence of relevant symptoms gets hardened into a thickening of non epithelialised wall and described by a new term WON. Like ANC, WON becomes prepared to invade even the whole pancreas or the associated peripancreatic area. Traditional old term pancreatic abscess has been duly replaced by a new term coined as infected WON. Availability of gas bubbles can ensure the kind and emergence of infection, however, most infected won appear as thick walled fluid collections. It is pertinent to recall that the available air bubbles within a WON is not sure sign of infection as air bubbles can also be observed when pathway between WON and GIT tract may persists. Thus, clinical signs and symptoms of infection should be correlated with laboratory indices of infection such as white cell count and C-reactive protein. Any fluid collection that strongly interferes with the pancreas and proves an obstacle in its normal functioning should be termed a WON following a duration of 4 weeks. Like, ANC, APFC and pseudocysts .WON can be both sterile or infected .Unlike pseudocyst, WON may contain different types of non –liquified material and if found to be severly infected is rarely treated by percutaneous catheter drainage as compared to a pseudocyst. If infection is established by fine needle aspiration and the sample is at least partially liquefied ,percutaneous catheter drainage may be carried out ,but large catheters (upto 30 Fr) need to be required.[1]

D. Pseudocysts

Taking prior factors into consideration, most APFCs resolve spontaneously without clinical sequelae. In some cases, these show their existence for a quite longer period and develop into pseudocysts, which classically have a thin fibrous capsule. Pseudocysts may occasionally even extend upto the mediastinum. More than 70% of pseudocysts resolve spontaneously or decrease in size. Thus, a wait-and-watch policy for more than 6 weeks has been a suggested for asymptomatic pseudocysts. However, these may be involved with more complications, which may arise due to rupture, infection, haemorrhage, pain, biliary or pancreatic duct obstruction, or gastrointestinal tract involvement. Effective treatment may be resorted if supplemented with percutaneous catheter drainage.[1]

E. Vascular Complications

This type of complication can not be regarded as characteristic feature of pancreatitis owing to persistent erosion, vanishment and thrombosis taking place in intrapancreatic and peripancreatic veins and arteries due to gradual deterioration of the quality of pancreatic enzymes or sudden collapse resulting out of compression or suppression due to malformation of existing pseudocyst. Moreover, thrombosis of the blood channels especially splenic artery and vein further leads to vascular degeneration, impairment and obstruction in the related processes of pancreas which accelerates negative feedback or can display retarding impact with regards to splenic vein thrombosis with infarction or splenic haemorrhage that can not be ruled out due to their massive involvement in acute pancreatitis. Devouring of splenic or gastroduodenal artery in the course of time due to prevailing substandard enzymes complicates the normal process with the sudden onset of pseudo aneurysm rupture of which can prove highly fatal as life threatening bleeding may likely be induced. Contrast – enhanced CT imaging firmly establishes an appropriate site of vascular coherence at the appropriate time and in an appropriate manner. However, in serious and cumbersome cases inflicted with severe acute haemorrhage or due to development of pseudo aneurysm; angiography and vascular embolism can be considered as suggestive techniques and may prove to be an alternative to surgery.[1,2]

F. Gastrointestinal Involvement

Net work of the inflammatory process extended from enlarged pancreas can be held responsible for development of oedema, perforation of stomach wall or duodenum and necrosis. The patients involved have been found to be threatening of life risk to the larger extent. The danger may result owing to close association between bowel loops and durable invasion and extension of inflammatory process. Secondary interference of bowel may co-exists when pancreatic enzymes have an easy escape through mesenteric layers or a new aspect pertaining to secondary to vascular complications further arise which are held responsible to disturb the normal metabolic pathway inducing bowel ischaemia.[1]

In recent years, the grading system has been duly revised and redefined on the basis of pancreatic necrosis and hence, the same can be clearly visualized on imaging, and therefore, a new term “CT severity index (CTSI)” is in vogue.[1]

IV. CASE REPORT

A 60-year-old man presents to the emergency department complaining of severe mid-epigastric abdominal pain that radiates to the back. The pain improves when the patient leans forwards or assumes the fetal position and worsens with deep inspiration and movement. He also complains of nausea, vomiting, anorexia and gives a history of heavy alcoholic intake in the past week. He was in distress. He also presented with tachycardia, tachypnoeic, hypotension and was febrile. He looked slightly agitated and confused. He had a past history of type II diabetes mellitus, asthma and obstructive sleep apnoea and hypertension. On examination he appeared diaphoretic with decreased breath sounds over the base of the left lung. He is still unable to eat or drink well due to nausea and vomiting.

V. FINDINGS

PANCREAS:

- Pancreas appears heterogeneous with extensive necrosis of head and body . Multiple irregular walled heterogeneous cystic lesions around the pancreas with multiple air foci and extensive peripancreatic fat stranding.
- On post contrast cyst wall are showing enhancement
- Head – 4.4 cm , Body – 1.9 cm , Tail – 2.8 cm
- F/s/o necrotising emphysematous pancreatitis with multiple walled off necrosis. CTSI-10

SPLEEN:

- Small hypodense area of HU 20-30 noted in the lower pole of spleen measuring 2 x 1.2 cm -likely suggestive of splenic abscess
- B/L adrenal glands appear bulky

THORAX :

- Consolidation with effusion noted on the left side

PERITONEUM :

- Minimal free fluid in the peritoneal cavity – s/o minimal ascites, subhepatic collection noted- Minimal Ascites.
- Multiple subcentimetric pre and para aortic lymph node noted.

VI. DISCUSSION AND CONCLUSION

Perfection can be attained only on concentrating the findings of CT scan owing to acute pancreatitis which presents as an acute condition with symptoms of abdominal pain and enhanced pancreatic enzymes level in the blood and urine. Spectrum of disease reveal peripancreatic inflammation /pancreatic edema /fluid collections. Individual laboratory index is considered as markers of pancreatic injury as well as of inflammatory responses, while promising results have not yet gained clinical acceptance.

Advantages and limitations of the clinical laboratory, radio imaging prognostic indices have been duly analysed and healthy discussion made pertaining to prompt role of CT imaging in evaluation of acute pancreatitis which is thoroughly based on contrast enhanced .CT being imaging modality of

choice, oral and iv contrast differentiate pancreatic tissue from adjacent blood vessels and duodenum. Therefore , emphasis has been virtually laid on CT finding ignoring fictitious parameters confusing the real state of this vital organ without which normal life becomes miserable .With the introduction of CT imaging the patient can be recovered and relapsed.

In many cases the relevant diagnosis is crystal clear pertaining to the patients with a remarked history of heavy intake of liquor, upper abdominal pain and hyper amylasemia or enhanced plasma lipase. In 2008, an international group of experts in the field of pancreatitis, led by the Acute Pancreatitis Classification Group, arrived at the conclusion employing iterative, web-based consultation process headed by a group of experts engaged in this special assignment and revised the Atlanta classification system with an effort to improve clinical diagnosis and management of acute pancreatitis and to standardize norms laid down for peripancreatic fluid collections, pancreatic and/or peripancreatic necrosis and how these entities evolve with the passage of time, as well as to sort out peculiar complications that may arise in future. In addition to it contrast material enhanced CT is being usually employed to assist in concrete evaluation of local pancreatic morphology and to properly diagnose presence and extent of pancreatic necrosis.[1]

CLASSIFICATION OF ACUTE PANCREATITIS[1]:

ATLANTA CRITERIA (1992):

- **MILD ACUTE PANCREATITIS (80% CASES)**
  (Acute interstitial / edematous pancreatitis)
- Acute Absence of organ failure
- Absence of local complications
- **SEVERE ACUTE PANCREATITIS(20% CASES)**
  (Acute haemorrhagic necrotizing [fulminant] pancreatitis)
- Local complications either present or absent
- Organ failure defined as
  - SBP < 90mm Hg
  - PaO2 less than or equal to 60 mmHg
  - GI bleed greater than equal to 500 ml/ 24 hours
  - Creatinine greater than equal to 2mg /dL after rehydration
- Ranson score greater than equal to 3 or APACHE greater than equal to 8.

REVISED ATLANTA CRITERIA (2012)

- MILD ACUTE PANCREATITIS
  - Acute Absence of organ failure
  - Absence of local complications
- **MODERATELY SEVERE ACUTE PANCREATITIS**
  - Local complications either present or absent
  - Transient organ failure < 48 hours
- **SEVERE ACUTE PANCREATITIS**
  1. Persistent organ failure > 48 hours and / or death.

[Defined as a score of 2 or more for one of these (CVS, RENAL, RESPIRATORY) Organ systems using the modified MARSHALL Scoring system.]

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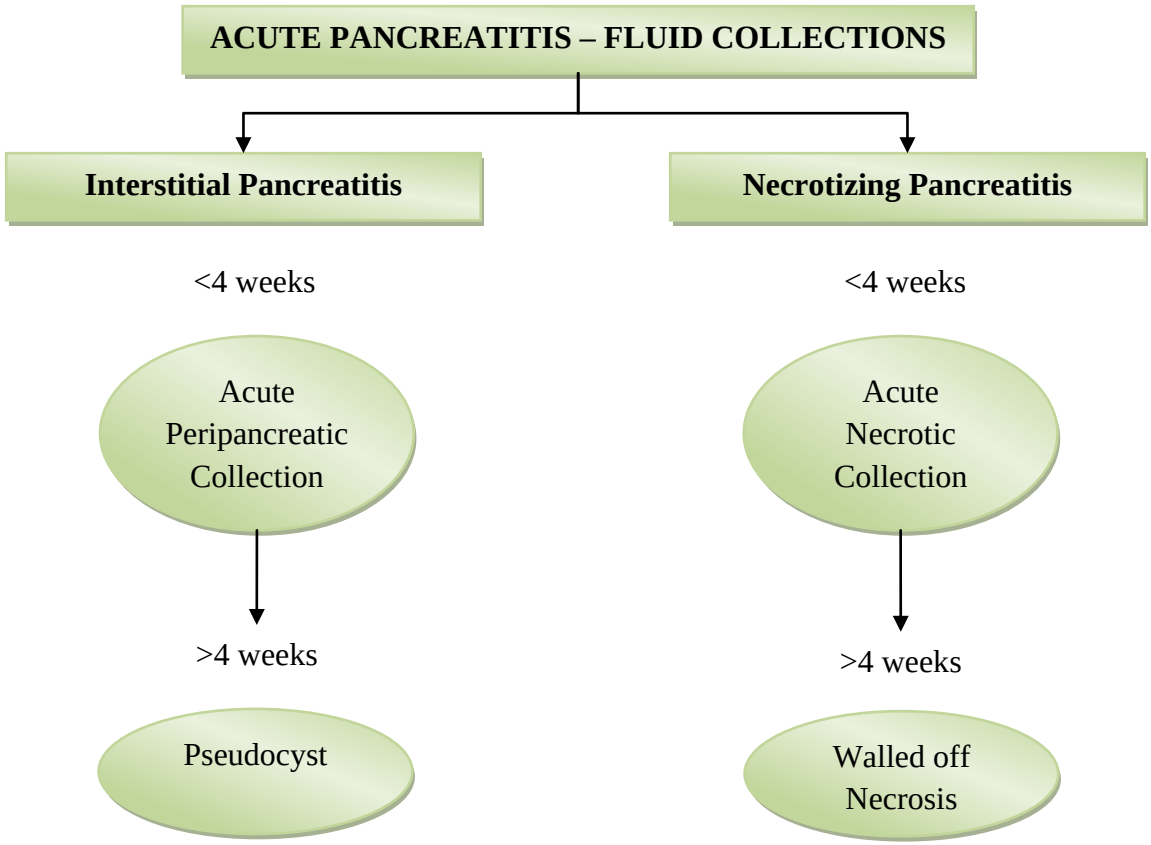


Scoring System For Severity Of Acute Pancreatitis: Ct Severity Index (Ctsi)
Balthazar Grading System:

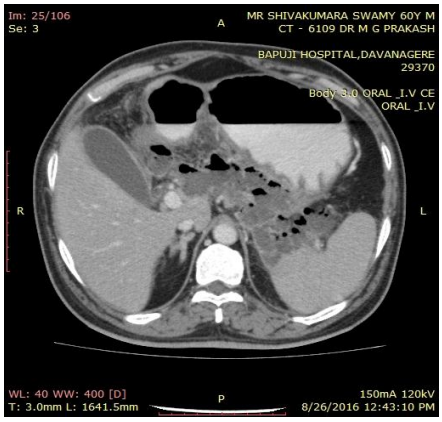
CT FEATURES	SCORE
Grade	
Normal gland	0
Focal / diffuse enlargement	1
Peripancreatic inflammatory change	2
Single pancreatic fluid collection	3
Two or more fluid collections or abscess	4
Necrosis	
None	0
< 30%	2
30 - 50%	4
>50%	6

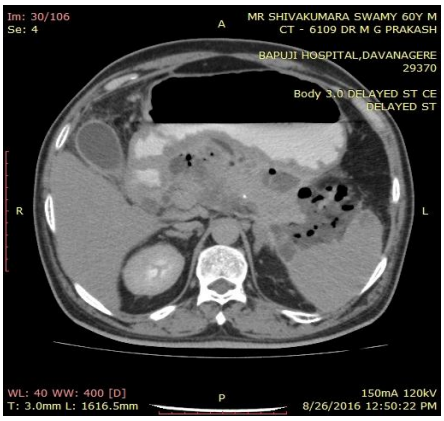
CT severity index (maximum score 10) = CT grade (0–4) +necrosis (0-6)

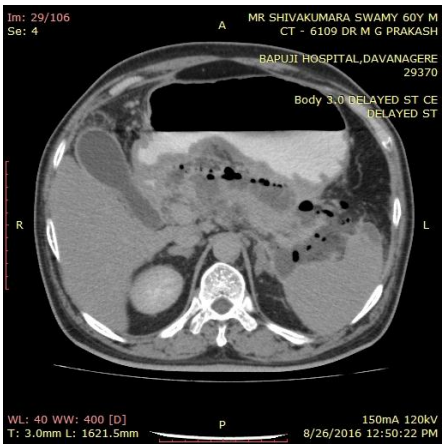
Modified CT Severity Index By Koenraad In 2004

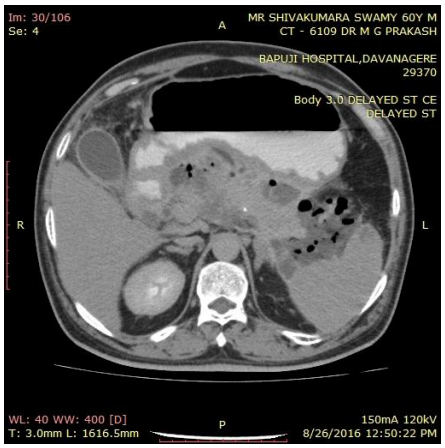
Prognostic indicator	Points
Pancreatic inflammation	
• **Normal pancreas**	0
• **Intrinsic pancreatic abnormalities with or without inflammatory changes in peripancreatic fat**	2
• **Pancreatic or peripancreatic fluid collection or peripancreatic fat necrosis**	4

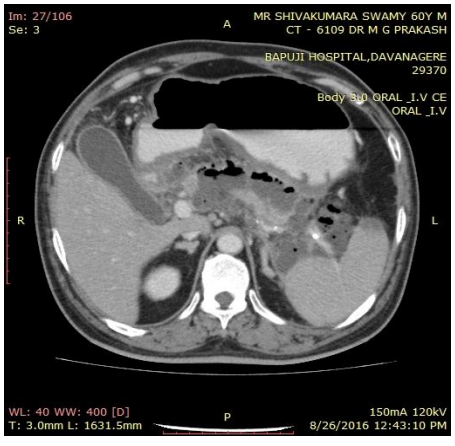
Pancreatic necrosis	
• **None**	0
• **≤ 30%**	2
• **>30%**	4
Extrapancreatic complications (one or more of pleural effusion, ascites, vascular complications, parenchymal complications or gastrointestinal tract involvement.	2

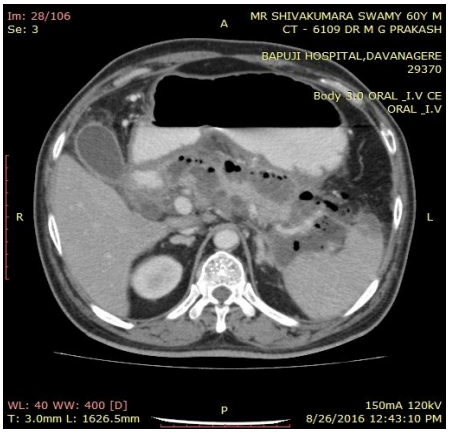


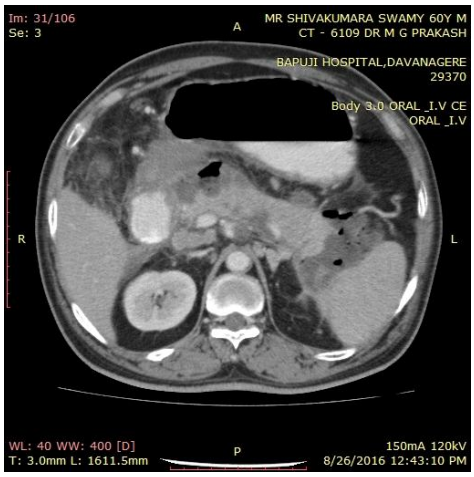








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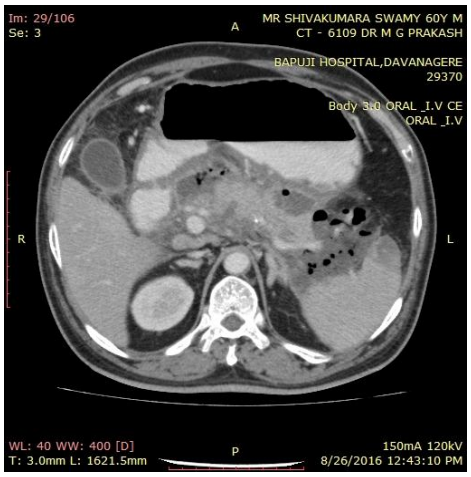
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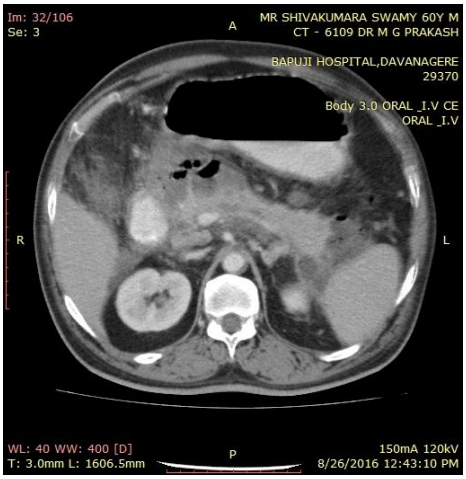
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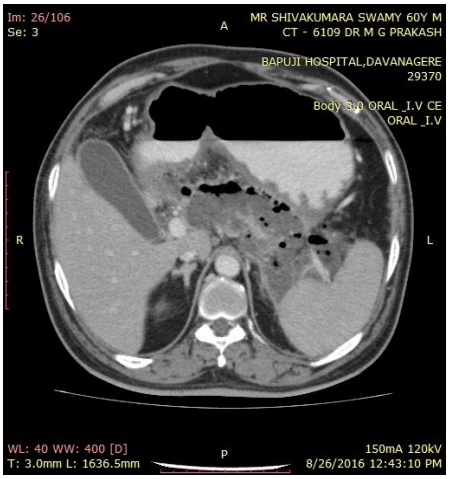
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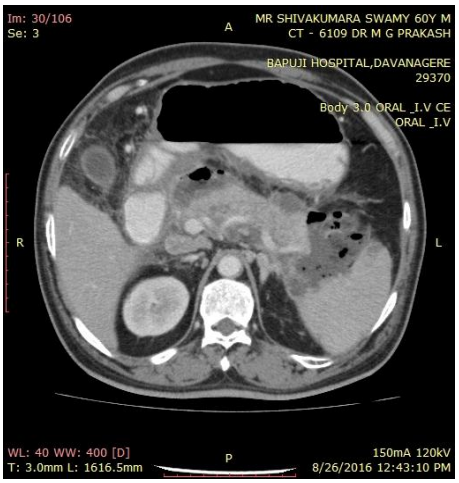
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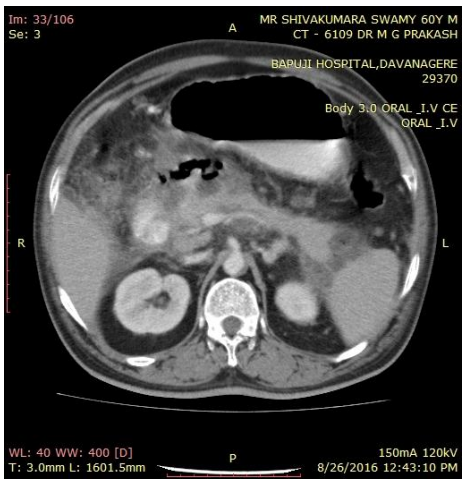
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