CT EVALUATION OF ACUTE PANCREATITIS AND ITS COMPLICATIONS USING MODIFIED CT SEVERITY INDEX

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Abstract

Objectives 1: To grade the CT findings of patients with acute pancreatitis according to MCTSI 2. To correlate the grading systems with patient outcome in terms of a. Organ failure b. Mortality c. Duration of hospital stay 3. To determine sensitivity, specificity and positive predictive value of MCTSI in predicting the above mentioned complications.

Materials and Methods: This study was conducted in the Department of Radio-diagnosis, Mysore medical college and research institute, Mysore from November 2016 to April 2018. The study comprised of a total of 50 patients. Of 50 patients diagnosed with acute pancreatitis who underwent contrast-enhanced MDCT within 3 days of the onset of symptoms during the study period were included in the study. The severity of the pancreatitis was scored using modified CT severity indexes. Patient clinical outcome was scored using parameters such as: mean duration of hospital stay, the need for surgical intervention, occurrences of infection, end organ failure and death. For the modified CT severity indexes, the correlation between the severity of pancreatitis and patient outcome was estimated using the percentage, frequency charts, and chi-square test.

Results: This was a prospective study of 50 cases of clinically diagnosed acute pancreatitis and confirmed by serum amylase and lipase levels. Modified CT severity index grading was done after contrast enhanced CT of abdomen and pelvis in all patients. Correlation of modified CT severity index grades was done with patient outcome taking local and systemic complications, duration of hospital stay. The age group of patients was 15 to 66 years with maximum patients (36%) between 26 and 30 years. 86% of patients were male. Male to female ratio was 8.6:1.4 with male preponderance. Chronic alcohol abuse was the most common cause of pancreatitis (76%), second was gallstones (16%) and others (8%). All patients presented with pain abdomen. 92% had vomiting, 30% patients had fever and 10% of patients had jaundice at the time of presentation. Amylase was elevated in 86% patients. Lipase was elevated in 90% patients. 86% patients had features of pancreatitis on ultrasound and in 14% ultrasound was normal. Pancreatic inflammation was seen in 100% of patients. 30% patients had no evidence of pancreatic necrosis on CT scan. 54% of patients had less than 30% necrosis and only 16% had more than 30% necrosis. 48% patients had no evidence of extrapancreatic complications. 52%
patients had one or more extra pancreatic complications. According to Modified CT Severity Index, 6% patients had mild, 70% patients had moderate and 24% had severe pancreatitis. Duration of hospital stay ranged from 3 to 25 days with mean duration of 9.5 days. Mortality rate was 0%. 38% patients are considered to have end organ failure. Hepatic failure is the most common system failure seen in 22% patients. 36% patients had evidence of systemic infection. 10% patients required surgical interventions.

**Conclusion:** There was highly significant correlation between the MCTSI score and the prediction of end organ failure, systemic infection and duration of hospital stay than CTSI score. MCTSI is a very useful tool for the screening of patients with acute pancreatitis for the classification of severity accurately and to predict the clinical outcome when used before three days of symptom onset. Key words: Computed Tomography; Modified CT Severity Index; Acute pancreatitis; pancreatic necrosis; Patient outcome.

**Introduction:**

Acute pancreatitis is one of the most complex and clinically challenging conditions of all abdominal disorders. Early assessment of the cause and severity of acute pancreatitis is of utmost importance for prompt treatment and close monitoring of patient with a severe disease. Imaging plays an important role in the management of the patient with acute pancreatitis. CT, in particular, has revolutionized pancreatic imaging, and what was once considered a hidden organ may now be accurately and noninvasively imaged.¹ ² Between 10% and 20% of cases of acute pancreatitis are considered severe.³ Treatment of patients with acute pancreatitis based on the initial assessment of disease severity. Severe pancreatitis is characterized by a protracted clinical course, multiorgan failure, and pancreatic necrosis.² Individual laboratory indexes (markers of pancreatic injury, markers of inflammatory response), while promising, have not yet gained clinical acceptance. Numeric grading systems like RANSON and APACHE II are commonly used today as indicators of disease severity. While RANSON score cannot be used for the first 48 hrs, APACHE score is cumbersome to use.⁴ ⁵

Contrast-enhanced computed tomography was later used to evaluate the prognosis of patients with acute pancreatitis. CT severity index was used initially which was popularly called Balthazar scoring system. This scoring system is based on pancreatic morphology, the number of peripancreatic fluid collections and pancreatic necrosis.

Now Modified Computed Tomography Severity Index (MCTSI) has been introduced which differs from the Computed Tomography Severity Index (CTSI) by including the presence of extrapancreatic complications and grading the peripancreatic fluid collection in terms of presence or absence instead of the number of fluid collections.⁵ ⁶

**Objectives:**

1. To determine the value of computed tomography evaluation in the early diagnosis of acute pancreatitis.
2. To evaluate the complications using modified computed tomography severity index.
3. To grade the CT findings of the patient with acute pancreatitis according to modified CT severity index.
4. To correlate the grading systems with patient outcome in terms of
   a. Organ failure
   b. Mortality
   c. Duration of hospital stay
5. To determine sensitivity, specificity and positive predictive value of modified CT severity index in predicting the above-mentioned complications.
Review of Literature:-

Historical background:

The pancreas is truly a noble organ. Known to the ancient Greeks, its name in Greek “pan kreas” translates as “all flesh.” Aristotle believed its function was to protect. The pancreas was apparently first discovered by Herophilus, a Greek anatomist and surgeon. It was in the 18th century that the main duct of Wirsung was described as well as its first cannulations to perform studies on pancreatic secretion.

The digestive action of pancreatic secretions was discovered almost 200 years after the discovery of the pancreas, later, Eberlein in 1834, Purkinje and Pappenheim in 1836, and Valentin in 1844 observed that the emulsification of fat, proteolytic activity, and digestion of starch respectively by pancreatic juice and extracts. It was Bernard who subsequently demonstrated the digestive action of pancreatic juice on sugar, fats, and proteins, using secretions from pancreatic fistula preparations. The histologic structure of the pancreas was first described by Langerhans in 1869 thereafter by Heidenhain.

Kühn introduced the term enzyme and isolated trypsin in 1876 which led to the identification of pancreatic amylase and lipase. It was Cheyvalnikoff in 1889, a student of Pavlov who discovered enterokinase in the duodenal mucosa, which is essential for activation of the proteolytic enzymes. In 1895, Dolinsky stimulated pancreatic secretion by instilling acid into the duodenum; this led to the discovery of secretin by Bayliss and Starling, which proved to be not an enzyme but the first hormone to be identified.

Embryology of Pancreas:

Embryologically, the pancreas is derived from the endoderm of the embryonic foregut and arises from a dorsal and ventral pancreatic bud. The larger dorsal bud is the precursor of the anterior portion of the head as well as body and the tail, while the smaller ventral bud develops into the posterior head and uncinate process. The dorsal and ventral ducts fuse into one major duct, the duct of Wirsung, which empties into the duodenum along with the CBD at the ampulla of Vater.

![Diagram of the pancreas showing the relationship between the dorsal (DP) and ventral (LVP and RVP) pancreatic anlagen.](image)

**Figure 1:** Normal development of the pancreas. The ventral pancreatic anlage is initially paired, with the left lobe subsequently disappearing during development. The ventral pancreatic anlage fuses side by side with the dorsal anlage. (RVP, right ventral pancreatic anlage; LVP, left ventral pancreatic anlage; DP, dorsal pancreatic anlage.
Variations in the pancreatic ductal branching pattern are common (Figure 2) and can predispose to pancreatitis.

**Figure 2:** Colored schematic diagram showing normal and variant pancreatic ductal anatomy. (A) Normal pancreatic ductal anatomy with the main pancreatic duct of Wirsung (thin arrows) emptying into the major duodenal papilla at the ampulla of Vater (thick arrow). (B) Normal variant where an accessory pancreatic duct (thin arrow) empties into the minor papilla (thick arrow). (C) Dorsal-dominant drainage where the main pancreatic duct (thin arrow) empties into the minor papilla and the accessory pancreatic duct (thick arrow) empties into the major papilla. (D) Pancreas divisum with complete separation of the major (thin arrows) and minor pancreatic ducts (thick arrow).

**Anatomy Of Pancreas**:

The pancreas is a soft, elongated, flattened gland measures 12 to 20 cm in length. It is a retroperitoneal organ located in the anterior pararenal space posterior to the stomach and bounded by the c-loop of duodenum on the right side. Composed of the following parts:

1. The head of the pancreas lies within the concavity of the duodenum.
2. The uncinate process emerges from the lower part of the head and lies deep to superior mesenteric vessels.
3. The neck of the pancreas is the constricted part between the head and the body.
4. The body lies behind the stomach.
5. The tail is the left end of the pancreas. It lies in contact with the spleen and runs in the lienorenalligament.

The average normal measurements of the pancreas in CT is as follows: head - 23 +/- 3mm, neck - 19 +/- 2.5mm, body - 20 +/- 3mm and tail - 15 +/- 2.5mm. The AP dimension of pancreas must be smaller than the AP diameter of the adjacent vertebra on CT.
Figure 3: Anatomy of pancreas.

**Head of pancreas 2:**
Uncinate process of pancreas 3: Pancreatic notch 4: Body of pancreas 5: Anterior surface of pancreas 6: Inferior surface of pancreas 7: Superior margin of pancreas 8: Anterior margin of pancreas 9: Inferior margin of pancreas 10: Omental tuber 11: Tail of pancreas 12: Duodenum

**Blood supply of Pancreas:**
**Arterial supply:**
1. The superior pancreaticoduodenal artery a branch of the gastroduodenal artery
2. The inferior pancreaticoduodenal artery from superior mesenteric artery
3. The pancreatic branches of splenic artery the largest of these branches is called the artery of pancreatic magna.

**Venous drainage:**
The body and neck of the pancreas drain into splenic vein; the head drains into the superior mesenteric and portal veins.

**Lymphatic drainage:**
Lymph is drained via the splenic, celiac and superior mesenteric lymph nodes.

Figure 4: Pancreatic blood supply.
Pancreatic duct:
The main pancreatic duct of Wirsung begins near the tail of the pancreas. It is formed from a continuous duct of secretions draining the head of the gland. It passes from left to right and joins with the other ducts to form the main duct of the pancreas. The duct lies midway between the superior and inferior margins. At the level of the major papilla, the duct turns horizontally to join the common bile duct. This portal segment is the ampulla of the bile duct, which terminates in the duodenal papilla and is guarded by the sphincter of Oddi. (Fig-5, Fig-6)

Figure 5:- The pancreatic duct system.

Figure 6:- The pancreatic duct system.
The function of the pancreas\(^{14}\):
The pancreas is a dual-function gland, having features of both endocrine and exocrine glands. The functional unit of the exocrine pancreas is composed of acinar units and the draining ductules.

The part of the pancreas with endocrine function is made up of approximately a million cell clusters called islets of Langerhans.

There are several different types of cells that comprise the following cells:
1. Alpha: Produces glucagon, which raises the level of blood glucose between meals, by converting fat and protein into intermediate metabolites, which eventually are converted to glucose.
2. Beta: Produces insulin and amylin, which lowers the level of blood glucose by inhibiting the secretion of glucagon and slowing the emptying of the stomach.
3. Delta: Produces somatostatin, which inhibits the release of specific hormones and reduces the rate of absorption of food from the content of the small intestine.
4. Gamma: Produces polypeptide, which reduces the appetite.
5. Pancreatic Polypeptide: It is a 36-amino-acid molecule that acts as a cholecystokinin antagonist. It suppresses pancreatic secretion and stimulates gastric secretion.
6. The pancreas is susceptible to a variety of diseases, which include\(^{10,21}\):
7. Pancreatitis: It is characterized by inflammation of the pancreas; pancreatitis can be either acute or chronic.
8. Pancreatic Cancer: The fifth leading cause of cancer death worldwide and has a high mortality rate. The time of diagnosis most often is late, and the mortality rate is high.
9. Type 1 Diabetes: Occurs due to dysfunction of the endocrine part of the pancreas.

Acute pancreatitis:
Definition:
According to the 1992 Atlanta Symposium\(^{15}\), acute pancreatitis was defined as an acute inflammatory process of the pancreas that may also involve peripancreatic tissues and/or remote organ systems.

Historical aspects of acute pancreatitis\(^{16,17}\):
1889:
Fitz classified the severity of acute pancreatitis using evidence of pancreatic hemorrhage and disseminated fat necrosis as morphological features of this disease.
1974-1983:
Ranson and colleagues developed clinical criteria to assess the severity of acute pancreatitis.
1985:
The attenuation values of pancreatic parenchyma during intravenous bolus use have been used as an indicator of pancreatic necrosis and as a predictor of disease severity.
1990-1996:
Balthazar and colleagues introduced the CT Severity Index.
1992-1994:
In Atlanta, the International Symposium on Acute Pancreatitis has classified this entity into mild, acute pancreatitis and severe acute pancreatitis.
2004:
Introduction of Modified CT Severity Index.

Acute pancreatitis is one of the most common diseases affecting the exocrine pancreas. The incidence of acute pancreatitis is increasing, especially in the population, increasing in population over the age of 65.

The overall mortality rate from acute pancreatitis has declined only gradually to approximately 5% to 10%\(^{18}\).

Acute pancreatitis is best defined clinically by the presence of the following criteria\(^{22}\):
1. Symptoms, such as epigastric pain, consistent with the disease;
2. Serum amylase or lipase greater than the upper limit of normal;
3. Radiologic imaging consistent with the diagnosis, usually using ultrasonography, computed tomography (CT), or magnetic resonance imaging (MRI).
Etiology of pancreatitis:

Table 1: Etiology of pancreatitis.

| Common causes | Uncommon causes | Causes for recurrent pancreatitis |
|---------------|-----------------|----------------------------------|
| • Gallstones  | • Vascular causes and vasculitis  |
| • Alcohol     | • Connective tissue disorders    |
| • Hypertriglyceridemia | • Carcinoma pancreas |
| • ERCP        | • Hypercalcemia                |
| • Trauma      | • Peripancreatic diverticulum   |
| • Post-operative | • Pancreas divisum |
| • Sphincter of Oddi dysfunction | • Hereditary pancreatitis |
|               | • Cystic fibrosis             |
|               | • Infectious (mumps, CMV etc) |
|               | • Autoimmune                  |

Table 2: Drugs causing pancreatitis:

| Common causes | Uncommon causes |
|---------------|-----------------|
| • Alpha-methyldopa | Ethinylestradiol/lestrogenol |
| • 5-Aminosalicylate (mesalamine) | Furosemide |
| • Azathioprine  | Isoniazid |
| • Cimetidine   | 6-Mercaptopurine |
| • Cytosine arabinoside | Metronidazole |
| • Dexamethasone | Norethindrone mestranol |
| • Trimethoprim sulfamethoxazole | Valproic acid |
|               | Selindac |

Congenital anomalies causing pancreatitis:

Pancreatic Divisum:
Pancreas divisum is the most common congenital anomaly of the pancreatic ductal system. This condition results from the failure of fusion of the ventral and dorsal pancreatic anlage and relative obstruction to the flow of pancreatic juice is generated because the majority of the gland empties through the dorsal duct (duct of Santorini) into the minor papilla, which is small. The ventral duct (duct of Wirsung), which opens into the major papilla, drains only the ventral pancreatic anlage, which forms the head and uncinate process. Symptoms of pancreatitis can sometimes develop even in patients with a small communication between the ventral and dorsal ducts, which drain separately into the duodenum, representing an anatomic variant (dorsal dominant duct syndrome).
Dorsal duct (arrowhead) draining the majority of the gland into the minor papilla. The smaller ventral duct (arrow) drains the head and uncinate process of the pancreas into the major papilla; the CBD also drains into the major papilla. D – duodenum.

**Long pancreaticobiliary duct:**
There can be an anomalous fusion of the pancreatic and biliary ducts when the ducts have a common channel of greater than 15mm or fuse outside the duodenum, this predisposes the reflux of pancreatic enzymes into the biliary tree and the reflux of bile into the pancreas leading to pancreatitis.

**Annular Pancreas:**
Annular pancreas is a rare congenital anomaly in which a ring of pancreatic tissue surrounds the second part of the duodenum. It occurs in about one in 20,000 persons. There is a bimodal distribution, with one-half of cases manifesting during childhood (commonly with duodenal obstruction) and the other half manifesting in adults in the 4th to 5th decades of life. In adults, annular pancreas may be incidentally detected, or the patient may present with symptoms of peptic ulcer disease (24.8% of cases) or pancreatitis (13.3%).

**Pathophysiology And Course Of The Disease**\(^{25}\):
The pathophysiology of acute pancreatitis is generally considered in three phases.
1. In the first phase, there is premature activation of trypsin within pancreatic acinar cells. Once trypsin is activated, it activates a variety of injurious pancreatic digestive enzymes.
2. In the second phase, there is intrapancreatic inflammation.
3. In the third phase, there is extrapancreatic inflammation.

Determinants of the natural course of acute pancreatitis are pancreatic parenchymal necrosis, extrapancreatic retroperitoneal fatty tissue necrosis, biologically active compounds in pancreatic ascites, and infection of necrosis.

Early in the course of acute pancreatitis multiple organ failure is the consequence of various inflammatory mediators that are released from the inflammatory process and from activated leukocytes attracted by pancreatic injury. During the late course, starting from the second week, local and systemic septic complications are dominant. Bacteriologic analysis of intraoperative smears and aspirates reveals predominantly gram-negative organisms derived from the intestine, most frequently *Escherichia Coli*\(^{26}\).

In the majority of patients, acute pancreatitis is mild. In 10–20%, the various pathways that contribute to increased intrapancreatic and extrapancreatic inflammation result in systemic inflammatory response syndrome (SIRS). In some instances, SIRS predisposes to multiple organ dysfunction or pancreatic necrosis. Necrosis occurs early, within the first 24–48 hours, and with few exceptions remains stable during a given episode of acute pancreatitis.
Symptoms:
Abdominal pain – mild to incapacitating, steady and boring located in the epigastrium and periumbilical region. The pain often radiates to the back, chest, flanks and lower abdomen. The pain is more intense when the patient is supine and is partially relieved by sitting with the trunk flexed and knees were drawn up.
Nausea and vomiting.
Abdominal distention.

Physical Examination:
General examination:
Low-grade fever
Tachycardia
Hypotension
Shock
Jaundice rarely
Erythematous skin nodules
Cullen sign – faint blue discoloration around the umbilicus due to hemoperitoneum.
Grey Turner sign – blue/purple discoloration of the flanks reflects tissue catabolism of hemoglobin.
The Cullen sign and Grey Turner sign are rare findings and their presence indicates severe necrotizing pancreatitis.

Respiratory system:
Basal crepitations
Pleural effusion (usually left sided)

Abdomen:
Tenderness in the epigastric region.
Muscle rigidity may be present but to a lesser extent compared to the pain.
Bowel sounds are diminished or absent.
Pancreatic pseudocyst may be palpable.

Complications:
Local:
Necrosis – sterile/infected.
Pancreatic fluid collections – abscess/pseudocysts.
Involvement of contiguous organs.
Obstructive jaundice.

Systemic complications:
Pulmonary:
Pleural effusion
Atelectasis
Pneumonitis
ARDS

Cardiovascular:
Hypotension
Shock
Sudden death
Pericardial effusion

Hematologic:
DIC
Erosion into major blood vessels
Vessel thrombosis

Gastrointestinal:
Peptic ulcer disease
Erosive gastritis

**Renal:**
Azotemia
ATN

**Metabolic:**
Hyperglycemia
Hypertriglyceridemia
Hypocalcemia
Encephalopathy

**CNS:**
Psychosis
Fat emboli

**Bone:**
Necrosis

The presence of organ failure determines the outcome in cases that are difficult to manage. The respiratory failure (defined as Po2 < 60) is usually the predominant cause. The other important systemic complications are shock (systolic BP of < 90mm Hg and tachycardia > 130), renal failure (serum creatinine > 2mg/dl), abdominal bleeding (>500 ml / 24 hr), central nervous system failure (Glasgow Coma Scale score of less than 6 in the absence of sedation or by the sudden onset of confusion or psychosis), hepatic failure (serum bilirubin levels greater than 100 μmol/L or alkaline phosphatase levels greater than three times the upper limit of the normal range) and hematologic system failure (hematocrit level of less than 20%, WBC of less than 2,000/mm³, or platelet count of less than 40,000/mm³). Out of these, the presence of respiratory failure, shock, renal failure, and abdominal bleeding are the most important predictors of the outcome.

These patients need intense monitoring, correction of the metabolic abnormalities and supportive measures. It is a major cause of mortality in the first 2 weeks after an acute episode of pancreatitis.

The presence of local complications is an important cause of morbidity and may further necessitate interventions. It is a significant cause of mortality after 2 to 3 weeks of an acute episode.

**Classification and definitions of four categories for the severity of acute pancreatitis**:

Table 3:- Classification of the severity of acute pancreatitis.

| Severity category | Local complications                  | Systemic complications          |
|-------------------|-------------------------------------|---------------------------------|
| Mild              | No (peri)pancreatic complication    | and No organ failure            |
| Moderate          | Sterile (peri)pancreatic complication| or Transient organ failure      |
| Severe            | Infectious (peri)pancreatic compiliation | or Persistent organ failure |
| Critical          | Infectious (peri)pancreatic compiliation | and Persistent organ failure |

**Terminologies Associated with pancreatitis**:

**Pancreatic necrosis:**
It refers to focal or diffuse nonviable pancreatic parenchyma and usually peripancreatic fat necrosis. Pancreatic necrosis can be sterile or infected. Peripancreatic necrosis describes necrotic fatty and stromal tissue around the pancreas.

**Acute fluid collection:**
It refers to the fluid located in or near the pancreas that lacks a definite wall and typically occurs early in the course of acute pancrea
Pancreatic pseudocyst:
It refers to the fluid collection that persists for 4 to 6 weeks and becomes encapsulated by a wall of fibrous granulation tissue. When pseudocyst is located within the body of the pancreas, the cyst may contain necrotic pancreatic islet cells or even when the pseudocyst is fluid-appearing with low attenuation on CT. The term for a walled-off fluid-appearing pseudocyst-like structure involving the pancreas is walled-off pancreatic necrosis (WOPN). WOPN is pancreatic necrosis that has liquefied after five to six weeks.

Pancreatic phlegmon:
It refers to the term often used by radiologists to describe an inflammatory mass.

Diagnosis:
There is a general acceptance that a diagnosis of acute pancreatitis requires two of the following three features:
1. Abdominal pain characteristic of acute pancreatitis,
2. Serum amylase and/or lipase ≥3 times the upper limit of normal,
3. Characteristic findings of acute pancreatitis on CT scan.

This definition allows for the possibility that an amylase and/or lipase might be <3 times the upper limit of normal in acute pancreatitis. In a patient with abdominal pain characteristic of acute pancreatitis and serum enzyme levels that are lower than 3 times the upper limit of normal, a CT scan must be performed to confirm a diagnosis of acute pancreatitis. In addition, this definition allows for the possibility that the presence of abdominal pain cannot be assessed in some patients with severely altered mental status due to acute or chronic illness.

Differential Diagnosis of Acute Pancreatitis:
1. Perforated viscus
2. Acute cholecystitis and biliary colic
3. Acute intestinal obstruction
4. Mesenteric vascular occlusion
5. Renal colic
6. Closed-loop intestinal obstruction
7. Inferior wall myocardial infarction
8. Dissecting aortic aneurysm
9. Ruptured ectopic pregnancy.

Severity predictor of Pancreatitis:
Severe Pancreatitis: Clinical Evaluation
Recognition of severe pancreatic injury by means of clinical examination is unreliable. Clinical parameters such as tachycardia, orthostatic hypotension, shock, respiratory distress, and signs of peritonitis are consistent with a severe attack. They are however rarely seen and are not specific, and usually develop late which limits their clinical use.

Flank ecchymosis (Grey-Turner sign) or periumbilical ecchymosis (Cullen sign) are more specific indicators for severe acute pancreatitis and have been associated with a 37% mortality rate. These signs are rarely present, however, and they often appear 48–72 hours after the onset of symptoms.

Diagnosis on the basis of the clinical evaluation was missed in 30%–40% of patients with fatal necrotizing pancreatitis until the time of autopsy. Thus, individual clinical signs have only limited value for the assessment of the severity of acute pancreatitis.

Organ failure:
There is considerable interest among pancreatologists in using organ failure to predict severity. The Atlanta criteria defined which organ systems are of importance: pulmonary, renal, and cardiovascular system.

Multisystem organ failure is defined as two or more organs failing on the same day, rather than one organ failing on one day and another failing on the subsequent day.
Patients with multisystem organ failure or persistent organ failure have a much higher mortality rate approaching 50% as compared with patients with single and transient organ failure.47

Persistent organ failure is defined as lasting greater than 24 hours regardless of intervention. Survival among patients with organ failure at admission has also been shown to correlate with the duration of organ failure. When the organ failure persists for more than 48 hours, mortality is 36%.48

Peritoneal lavage:
Percutaneous recovery of any volume of peritoneal fluid with dark color or recovery of at least 20 mL of free intraperitoneal fluid of dark color portends significant mortality.49 The sensitivity of peritoneal lavage is 36% to 72%, and the specificity is greater than 80% to 100%.

Laboratory Markers:
Hematocrit49:
A high hematocrit on admission or one that fails to decrease after 24 hours of rehydration is thought to be a sign of hemoconcentration due to retroperitoneal fluid loss and thus a marker of severe disease. An elevated hematocrit (>44%) is a predictor for the development of necrosis. The hematocrit should be observed at admission for prognostic purposes and followed prospectively to assist in guiding the rate of intravenous hydration.

Serum Amylase and Lipase Levels50:
Elevated serum amylase and lipase levels, in combination with severe abdominal pain, often trigger the initial diagnosis of acute pancreatitis. Serum lipase rises 4 to 8 hours from the onset of symptoms and normalizes within 7 to 14 days after treatment.

Serum amylase may be normal (in 10% of cases) for cases of acute or chronic pancreatitis (depleted acinar cell mass) and hypertriglyceridemia.

The causes for false positive elevated serum amylase include salivary gland disease (elevated salivary amylase), bowel obstruction, infarction, cholecystitis, and perforated ulcer.

C-reactive protein:
CRP is an acute-phase reactant produced by the liver and is used extensively in Europe as a marker of severe pancreatitis.51

Polymorphonuclear Leukocyte Elastase:
Polymorphonuclear leukocyte elastase rises very early (before CRP) in acute pancreatitis. High levels have been reported to differentiate severe from mild disease.52

Phospholipase A2:
PLA2 is involved in the release of prostaglandins from cell membranes and degrades surfactant in the lung. It may play a role in the pulmonary dysfunctions associated with acute pancreatitis. Levels of catalytic type II PLA2 have been reported to differentiate between mild and severe disease within 24 hours of admission.53

Urinary Trypsinogen Activation Peptide:
The urinary TAP may serve as a near-record predictor of severity in patients with acute pancreatitis.37 Elevated urinary TAP (>30 nmoL/L) correlates with disease severity. The test can be applied within 12 hours of admission. The positive predictive value of an elevated TAP is 80% and the negative predictive value approaches 100%.

Procalcitonin:
This propeptide is a reactant that has been shown to differentiate mild from severe acute pancreatitis within the first 24 hours after symptom onset. A serum strip test has been developed that has a sensitivity of 86% and a specificity of 95% in detecting organ failure.54
Interleukin-6: IL-6isanacute-phase-reactantcytokinethatisproducedby
a variety ofcells and induceshepatic synthesis of CRP. Several studies have shown that it
areasonablygood marker to differentiatemild fromseveredisease, but the testis not readily available.  

Scoring indices: The Atlanta criteria defines severity by the presence of organ failure or pancreatic necrosis on dynamic
contrast-enhanced CT scan, other acceptable markers of severe pancreatitis include three or more of Ranson's
criteria score fornon-gallstone pancreatitis and an Acute Physiology and Chronic Health Evaluation (APACHE-II)
score of greater than eight. 

BISAP (Bedside Index for Severity in Acute Pancreatitis): It is a simple scoring system that includes 5 variables to determine severity yearly in the course of pancreatitis. The
BISAP score provides a single point for 5 parameters:
1. blood urea nitrogen (BUN) greater than 25 mg/dL,
2. impaired mental status,
3. systemic inflammatory response syndrome,
4. age greater than 60, and/or
5. the presence of pleural effusion,

ABISAP score greater than 3 is associated with a seven-to-twelve-fold increase in developing organ failure. The
Marshall Scoring System for organ failure is commonly used by intensivists for patients admitted to an intensive care unit.

Ranson Score
Ranson criteria is a clinical prediction rule for predicting the severity of acute pancreatitis which was introduced in 1974. (Table-4)

Table 4: Ranson criteria for assessment of the severity of AP

Ranson's criteria of severity of acute pancreatitis:

On admission:
1. Age > 55 years
2. White blood cell count > 16000/mm³
3. Glucose > 11.0 mmol/l
4. Lactate dehydrogenase > 350 IU/l
5. Aspartate aminotransferase > 250 U/l

During initial 48 hours:
1. Packed cell volume decrease > 10%
2. Blood urea nitrogen increase > 1.8 mmol/l
3. Calcium < 2 mmol/l
4. Partial pressure of oxygen < 60 mmHg
5. Basedeficit > 4 mmol/l
6. Fluid sequestration > 6 l

Ranson's score interpretation:
1. Score < 3: mild pancreatitis
2. Score 5 to 6: moderate pancreatitis
3. Score 7 to 8: severe pancreatitis

Ranson's score and mortality correlation:
1. Score 0 to 2: 2% mortality
2. Score 3 to 4: 15% mortality
3. Score 5 to 6: 40% mortality
4. Score 7 to 8: 100% mortality
Ranson’s score of ≥8 indicates substantial pancreatic necrosis (at least 30% pancreatic necrosis according to contrast-enhanced CT).

**Ranson criteria drawbacks:**
1. Different scoring for gallstone pancreatitis and non-gallstone pancreatitis.
2. An accurate Ranson’s score takes 48 hours to calculate.
3. Not all laboratories measure all the parameters (e.g., LDH) of Ranson’s criteria.
4. The overall sensitivity of the Ranson score for diagnosing severe disease is only 40% to 88%, and specificity is 43% to 90%.
5. The positive predictive value is approximately 50%, and the negative predictive value is around 90%.

Therefore, the best use of Ranson’s criteria is to exclude severe disease.

**Apache (Acute Physiology Score and Chronic Health Evaluation)**

The first major attempt at a system to quantify the severity of illness in ICU patients was the APACHE system introduced by Knaus et al. in 1981.

**Apache:**
In the original form, APACHE contained 34 physiologic measurements and included many continuous variables. Shortly after its introduction, APACHE system was not liked because of practical problems like the collection of a large number of variables. Apache II

This is the second version of the APACHE system and contained refinements based on experience with the original APACHE system. It contains 12 continuous variables from the original APACHE system and also takes into account the age of the patient, premorbid conditions, and Glasgow comascale.13 (Table 5)

**Table 5:** APACHE score for assessment of the severity of acute pancreatitis.

| Physiologic variable: | Value |
|-----------------------|-------|
| 1. temperature        |       |
| 2. Mean arterial pressure (mmHg) |       |
| 3. Heart rate         |       |
| 4. Respiration        |       |
| 5. Arterial pH        |       |
| 6. PaO2 (mmHg)        |       |
| 7. Serum sodium       |       |
| 8. Serum potassium    |       |
| 9. Serum bicarbonate (mmol/L) Serum creatinine (mg/dl) Serum creatinine (mg/dl) |       |
| 10. White blood cell count |       |
| 11. Glasgow Coma Score |       |

APACHE II score = A + B + C.
APACHE II score > 8 points predicts 11% to 18% mortality.

**Glasgow criteria:**
The Glasgow criterion is valid for both gallstone and alcohol-induced pancreatitis.

(Table 6).

**Table 6:** Glasgow criteria for assessment of the severity of acute pancreatitis.

| Criteria               | Value |
|-----------------------|-------|
| Age (yr)              | > 55  |
| WBC x 10^9/mm² Glucose (mg/dl) | > 15  |
| BUN (mg/dl) LDH (IU/l) Albumin (g/dl) PaO2 |       |
(mmHg) Calcium(mg/dl) | >180  | >45  | >600  | <3.3  | <60  | <8  

Glasgow score interpretation:  
If a patient scores 3 or more indicates severe pancreatitis and the patient should be transferred to ITU.

Balthazar Scoring:  
Developed in the early 1990s by Emil J. Balthazar et al. the Computed Tomography Severity Index (CTSI) is a grading system used to determine the severity of acute pancreatitis. The numerical CTSI has a maximum of ten points and is the sum of the Balthazar grade points and necrosis score.

| Balthazar Grade | Appearance on CT | CT Grade Points |
|-----------------|------------------|----------------|
| Grade A         | Normal CT        | 0 points       |
| Grade B         | Focal or diffuse enlargement of the pancreas | 1 point |
| Grade C         | Pancreatic gland abnormalities and peripancreatic inflammation | 2 points |
| Grade D         | Fluid collection in a single location | 3 points |
| Grade E         | Two or more fluid collections and/or gas bubbles in or adjacent to the pancreas | 4 points |

Table 8: Necrosis Score.

| Necrosis Percentage | Points |
|---------------------|--------|
| No necrosis         | 0 points |
| 0 to 30% necrosis   | 2 points |
| 30 to 50% necrosis  | 4 points |
| Over 50% necrosis   | 6 points |

CTSI's staging of acute pancreatitis severity has been shown by a number of studies to provide a more accurate assessment than APACHE II, Ranson, and CRP level. 69,70.

Modified CT severity index:  
The modified CT severity index correlated more closely with patient outcome measures than the currently accepted CT severity index, with similar interobserver variability. 70

However, the most recent study conducted showed no significant differences between the CTSI and the MCTSI in evaluating the severity of AP. Compared with APACHE II, both CT indices more accurately diagnose clinically severe disease and better correlate with the need for intervention and pancreatic infection. 72

Table 9: Modified CT Severity Index.

| 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      |
### Severe Acute Pancreatitis As Defined By Atlanta Symposium:

#### Early Prognostic Signs:
1. Ranson signs ≥3
2. APACHE-II score ≥8
3. Organ Failure
   - Shock–systolic pressure <90 mmHg
   - PaO2 ≤ 60 mmHg
4. Local Complications
   - Necrosis
   - Abscess
   - Pseudocyst

Others including disseminated intravascular coagulation (platelets ≤100,000/mm³, fibrinogen ≤100 mg/dL, fibrin split products >80 μg/mL), or a severe metabolic disturbance (serum calcium ≤ 7.5 mg/dL).

#### The diagnostic guideline I: look for risk factors of severity at admission

**Risk factors for severe acute pancreatitis**:
- Obesity – BMI > 30
- Old age > 55 yrs
- Organ failure at admission and
- Pleural effusion and/or infiltrates

The importance of established risk factors of severity of acute pancreatitis at admission is to transfer those patients who are most likely to have a severe episode to a step-down unit or an intensive care unit for closer supervision. Gender and etiology have no prognostic significance.

#### Diagnostic Guideline II: Determination Of Severity By Laboratory Tests At Admission Or ≤48 H:

The two tests that are most helpful at admission in distinguishing mild from severe acute pancreatitis are APACHE-II score and serum hematocrit.

It is recommended that APACHE-II scores be generated during the first 3 days of hospitalization and thereafter as needed to help in this distinction.

It is also recommended that serum hematocrit is obtained at admission, 12 h after admission, and 24 h after admission to help gauge adequacy of fluid resuscitation.

In this report, hematocrit ≥44 at admission and failure of admission hematocrit to decrease at 24 h were the best predictors of necrotizing pancreatitis.

#### Diagnostic Guideline III: Determination of Severity during Hospitalization:

Pancreatic necrosis and organ failure are the two most important markers of severity in acute pancreatitis. The distinction between interstitial and necrotizing pancreatitis can be reliably made after 2–3 days of hospitalization, by contrast, enhanced CT scan.

Many patients with acute pancreatitis do not require a CT scan at admission or at any time during the hospitalization. For example, a CT scan is usually not essential in patients with recurrent mild pancreatitis caused by
alcohol. A reasonable indication for a CT scan at admission is to distinguish acute pancreatitis from another serious intra-abdominal condition, such as a perforated ulcer. A reasonable indication for a contrast-enhanced CT scan a few days after admission is to distinguish interstitial from necrotizing pancreatitis when there is clinical evidence of increased severity. The distinction between interstitial and necrotizing pancreatitis can be made much more readily when a contrast-enhanced CT scan is obtained on the second or third day after admission rather than at the time of admission.

Additional contrast-enhanced CT scans may be required at intervals during the hospitalization to detect and monitor the course of intra-abdominal complications of acute pancreatitis, such as the development of organized necrosis, pseudocysts, and vascular complications including pseudoaneurysms.

Contrast-enhanced CT scan is the best available test to distinguish interstitial from necrotizing pancreatitis. Interstitial pancreatitis is characterized by an intact microcirculation and uniform enhancement of the gland. Necrotizing pancreatitis is characterized by disruption of the microcirculation such that devitalized areas do not enhance. Whereas small areas of non-enhancement could represent intrapancreatic fluid rather than necrosis, large areas of non-enhancement clearly indicate a disruption of microcirculation and pancreatic necrosis.

Complications in acute pancreatitis that can be recognized on abdominal CT scan include pancreatic fluid collections, gastrointestinal and biliary complications (such as obstruction of duodenum or stomach, inflammation of the transverse colon, and biliary obstruction), solid organ involvement (such as splenic infarct), vascular complications (such as pseudoaneurysms, splenic vein thrombosis with varices, portal vein thrombosis), and pancreatic ascites. Prompt transfer to an intensive care unit should take place for sustained organ failure. Transfer to an intensive care unit (or possibly a step-down care unit) should be considered if there are signs that suggest that the pancreatitis is severe or is likely to be severe. Additional danger signals that warrant close supervision by physicians and nursing staff in a step-down unit but not necessarily urgent transfer to an intensive care unit include obesity (BMI >30), oliguria with urine output <50 mL/h, tachycardia with pulse >120 beats/min, evidence of encephalopathy, and increasing need of narcotics.

**Overview of acute pancreatitis** 
Overall, 85% of patients have interstitial pancreatitis; 15% (range 4–47%) have necrotizing pancreatitis. Among patients with necrotizing pancreatitis, 33% (range 16–47%) have infected necrosis.

**Table 10:** Mortality in Acute Pancreatitis.

|                      | Median(%) | Range (%)  |
|----------------------|-----------|------------|
| All cases            | 5         | 2-9        |
| Interstitial pancreatitis | 3         | 1-7        |
| Necrotizing pancreatitis | 17        | 8-39       |
| Infected necrosis    | 30        | 14-62      |
| Sterile necrosis     | 12        | 2-44       |

The mortality in the absence of organ failure is 0, with single organ failure is 3% (range 0–8%), with multisystem organ failure 47% (range 28–69%).

Deaths within the first 2 weeks are generally attributed to organ failure; deaths after this interval are generally caused by infected necrosis or complications of sterile necrosis.

**Role of Radiology**

**Conventional imaging**
Radiographic signs of acute pancreatitis include the sentinel loop sign (dilated air-filled duodenum or jejunum), the colon cutoff sign (dilated large bowel to the level of the splenic flexure), loss of the left psoas shadow, ascites, or a gasless abdomen Pleural effusions, atelectasis, or an elevated hemidiaphragm are suggestive of severe acute pancreatitis.

Thickened rugal and duodenal folds, indentation of the stomach, and enlargement of the C-loop of the duodenum are signs of acute pancreatitis on barium meal and follow-through studies.
Ultrasound\textsuperscript{40,56}:
Sonography of patients with acute pancreatitis is often limited by difficulty in visualizing the pancreas because of ileus and overlying bowel gas. Abnormal ultrasound findings like ascites, pleural effusions are seen in 33–90\% of patients with acute pancreatitis.

Interstitial edema in acute pancreatitis is depicted on ultrasound as an enlarged hypoechoic gland. Although ultrasound may be used to identify peripancreatic acute fluid collections, it is not useful for the detection of necrosis, and therefore its main role in the imaging of acute pancreatitis is limited to the detection of cholelithiasis and choledocholithiasis and identification of fluid collections in the peritoneum, retroperitoneum, and pleural spaces.

CT:
Contrast-enhanced CT is the imaging modality of choice for the diagnosis and staging of acute pancreatitis\textsuperscript{57}

Protocol\textsuperscript{58}:
A three-phase (control, pancreatic parenchymal phase (40 seconds), and portal venous phase) protocol can be used for the initial assessment of acute pancreatitis. Positive oral contrast may be administered after the control scan.

Control phase – The use of positive oral contrast material may mask hemorrhage or calculi. So the initial control study may be done with no / negative oral contrast so that hemorrhagic collections and calculi may be identified in this phase.

The pancreatic parenchymal phase is the optimal phase for assessment for necrosis because normal pancreatic tissue enhances the greatest during this phase.

Subsequent imaging with CT is generally performed using a single-phase technique in the portal venous phase.

The suggestion that IV administration of iodinated contrast material can increase the severity and duration of acute pancreatitis has led to conflicting opinions regarding IV contrast usage and at present, the benefits of IV contrast administration appear to outweigh the potential risks\textsuperscript{52}.

CT features:
The pancreas enhances uniformly in mild acute pancreatitis and may be normal or enlarged with a variable amount of increased attenuation in the adjacent fat, termed “stranding”.

Local edema is a common finding and may extend along the mesentery, mesocolon, and hepatoduodenal ligament and into peritoneal spaces. Extension of edematous fluid into the anterior perirenal space may create a mass effect and a halo sign with sparing of the perinephric fat.

Peripancreatic fluid collections consist of exudate, peripancreatic fat tissue necrosis, or hemorrhage.

An organized “pseudocyst” may be formed. Edema is differentiated from fluid collections by the identification of fat islands of normal tissue within edematous fluid\textsuperscript{13}.

Non-enhancement of all or part of the gland is termed “necrosis”. CT is 100\% specific for necrosis if greater than 30\% of the gland is non enhancing\textsuperscript{40}. Necrosis develops between 24 and 48 hours after the onset of acute pancreatitis, and therefore CT within the first 12 hours may be falsely reassuring.

Pancreatic abscess formation is usually observed 4–6 weeks after the onset of acute pancreatitis as an area of low attenuation containing pus and a thick wall that may enhance after IV contrast administration. Air bubbles may be found within the collection.

Necrosis and abscess are considered among the most important imaging features of acute pancreatitis because they have prognostic relevance and may need intervention by either interventional radiologists or by the surgeons\textsuperscript{40}.

International Symposium, held in Atlanta, GA, in 1992, established a clinical based classification system for acute pancreatitis\textsuperscript{2}. The goal was to establish international standards of definitions of acute pancreatitis and its
complications to make possible valid comparisons of the severity of illness and the results of therapy and also to establish a
ground for future trials. This was a group of 40 international authorities from six medical disciplines and 15
countries.

Interstitial pancreatitis was defined as focal or diffuse enlargement of the pancreas with enhancement of the
parenchyma that is either homogeneous or slightly heterogeneous in response to IV contrast. There may be
inflammatory changes in peripancreatic fatty tissue characterized by a hazy appearance.

Pancreatic necrosis was defined as diffuse or focal areas of nonviable pancreatic parenchyma that was typically
associated with peripancreatic fat necrosis. The criteria for the CT diagnosis of necrosis included focal or diffuse
well-marginated zones of nonenhanced pancreatic parenchyma greater than 3 cm in size or greater than 30% of the
pancreas.

An extrapancreatic fluid collection was defined as pancreatic fluid that extravasates out of the pancreas during acute
pancreatitis into the anterior pararenal spaces and other areas as well.

A pancreatic pseudocyst was defined as a collection of pancreatic juice enclosed by a non epithelialized wall that
occurs as a result of acute pancreatitis, pancreatic trauma, or chronic pancreatitis. It is generally believed that a
period of at least 4 weeks is required from the onset of acute pancreatitis to form a well-defined wall composed of
granulation and fibrous tissue. 59

Severe pancreatitis was defined as pancreatitis associated with organ failure and/or local complications (necrosis,
pseudocyst).

![Figure 8: Axial CECT section of the normal pancreas in the arterial phase.](image)

**MRI**: 
MRI may be performed using unenhanced and contrast-enhanced T1-weighted and fat-suppressed T2-weighted
sequences. Heavily T2 weighted thick slab and thin sections are obtained for delineation of the ductal anatomy.

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An enlarged edematous gland that is low signal on T1-weighted and high signal on T2-weighted MRI is observed. Acute pancreatitis is sometimes associated with pancreatic ductal dilatation, which can be clearly identified and examined on T2-weighted images.

T2-weighted images are also useful for the detection of acute pancreatic collections and pseudocyst.

It has the advantage of demonstrating possible choledocholithiasis, the presence or absence of ductal distention, disruption or leakage of the pancreatic duct, and the size, location, and possible communication of a pseudocyst with the pancreatic duct. In addition, it better demonstrates local hemorrhage in or around the pancreas and helps assess the internal consistency and drainability of fluid collections which may influence the choice of treatment.

The pancreatic duct disconnection occurs when necrosis affects the ductal epithelium and an isolated segment of viable pancreatic tissue is disconnected from the duodenum. This creates persistent fistulation and inflammation with an increased incidence of infection. Diagnosis of disconnection of the main pancreatic duct requires visualization of a necrotic region of at least 2 cm in size, viable pancreatic tissue proximal to the necrosis.

Early MRCP may sometimes be of limited value for identifying the cause of acute pancreatitis because collections may compress the pancreatic and biliary ducts obscuring gallstones. MRCP may be of benefit when iodinated contrast administration is contraindicated or if disconnection of the main pancreatic duct is suspected.

MRI has not been widely used in the care of patients with acute pancreatitis. While CT scan remains the primary imaging technique to evaluate patients with acute pancreatitis, recent reports have indicated that MRI has some advantages: no concern regarding radiation exposure, the greater ability of MRI as compared to CT to distinguish necrosis from fluid, and the overall reliability of MRI as compared to CT scan in staging the severity of acute pancreatitis and its complications. Accurate identification of retained bile duct stones and pancreatic duct leaks. Disadvantages of MRI include lack of availability when urgently needed, variation in quality among centers, and the difficulty of supervising a critically ill patient undergoing MRI.

In the imaging of acute pancreatitis using MRI the same descriptive terminology as that used in CT is used.

![Figure 9](image-url)

Figure 9: (a) Axial T2 Weighted and (b) gadolinium-enhanced
T1 Weighted image of the normal pancreas:

Figure 10: (a) MRCP and (b) ERCP depicting the normal anatomy of the pancreas, common bile duct, common hepatic duct, and intrahepatic ducts:

Role of intervention radiology:

CT is the modality of choice to guide the intervention procedures because the retroperitoneal location and the adjacent bowel loops can be better evaluated by CT.
Transhepatictransgastric, b) transgastric, c) gastrospenic, d) left anterior paranrenal space to body and tail of the pancreas, e) paravertebral, f) right paranrenal, g) pancreatic head through the duodenum, h) transhepatic, i) gastrocolic to head and body of pancreas j) transmesocolic.

L: liver, ST: stomach, LS: lesser sac, C: colon.

The most common approaches include left anterior para renal space approach for tail collections and gastrocolic for head and body collections.

**Treatment:**

**Treatment Guideline I: Supportive Care**

Prevent hypoxemia and ensure adequacy of fluid resuscitation.

It is recommended that supplemental oxygen is administered until there is no further threat of hypoxemia. Aggressive IV fluid replacement is of critical importance to counteract hypovolemia caused by third space losses, vomiting, and diaphoresis.

The abdominal pain is relieved with a parenterally administered narcotic medication.

**Treatment Guideline II: Transfer To An Intensive Care Unit**:

Prompt transfer to an intensive care unit should take place for sustained organ failure. In particular, sustained hypoxemia, hypotension refractory to a bolus of IV fluids, and possibly renal insufficiency that does not respond to a fluid bolus (such as a serum creatinine > 2.0 mg/dL) warrant prompt transfer to an intensive care unit.

**Treatment Guideline III: Nutritional support**:

In general, oral intake is usually initiated when the abdominal pain has subsided, abdominal tenderness has markedly decreased, nausea and vomiting have ceased, bowel sounds are present.

**Treatment Guideline IV: Use of Prophylactic Antibiotics in Necrotizing Pancreatitis**:

The use of prophylactic antibiotics to prevent pancreatic infection is not recommended at this time among patients with necrotizing pancreatitis.

**Treatment Guideline V: Treatment of Infected Necrosis**:

CT-guided percutaneous aspiration with Gram’s stain and culture is recommended when infected necrosis is suspected. Treatment of choice in infected necrosis is surgical debridement. It is impossible to distinguish these
conditions clinically unless CT scan shows evidence of air bubbles in the retroperitoneum. Another technique is percutaneous catheter drainage of infected necrosis.

**Treatment Guideline VI: Treatment of Sterile Necrosis**: 
Sterile necrosis is best managed medically during the first 2–3 wks. After this interval, if abdominal pain persists and prevents oral intake, debridement should be considered. This is usually accomplished surgically, but percutaneous or endoscopic debridement is a reasonable choice.

**Treatment Guideline VII: Role of ERCP and Biliary Sphincterotomy in Gallstone Pancreatitis**: 
ERCP is indicated for clearance of bile duct stones in patients with severe pancreatitis, in those with cholangitis, in those who are poor candidates for cholecystectomy, in those who are postcholecystectomy, and in those with strong evidence of persistent biliary obstruction.

**Material and Methods:**
Cases of suspected acute pancreatitis referred to the department of Radio-diagnosis, Mysore Medical College and Research Hospital to the contrast-enhanced computed tomography on the clinical suspicion/diagnosis of acute pancreatitis, altered biochemical parameters (serum amylase, serum lipase) in favor of acute pancreatitis were included in this study. The study period was between 1/11/2016 – 31/04/2018.

**Study Design:**
Hospital-based prospective study.

**Study Area:**
Mysore Medical College and Research Institute, Mysore.

**Sample Size:**
As per the biostatistician's opinion the minimal sample size required was 32 cases for satisfactory statistical analysis. However, a total number of 50 cases are included under the advice of the biostatistician.

**Statistical Analysis:**
Sensitivity, Specificity, and Positive Predictive value are analyzed.

**Method of Collection of Data:**
Cases of acute pancreatitis are graded according to the modified CT severity index. The patients are assessed on the 5th day after the initial CT examination and on the day of discharge as follows:

- Development of organ failure defined as:
  1. Shock - systolic BP less than 90 mmHg
  2. Respiratory failure - PO2 less than 60 mmHg
  3. Renal failure - serum creatinine > 2 mg / dl
  4. Gastrointestinal bleed > 500ml / 24 hr

Number of days of hospital stay.
Mortality.
Equipment:

![Dual slice Computed Tomography scanner](image)

**Figure 12:-** Dual slice Computed Tomography scanner (GE health care system).

**Imaging Protocol:**
Plain and post-contrast study of the abdomen and pelvis was done. It consists of the acquisition of contiguous axial sections, of thickness 7 mm of abdomen and pelvis and 5 mm in the region of interest in cranio-caudal direction from the level of the xiphisternum to pubic-symphysis before and after administration of oral and intravenous iodinated contrast. Positive oral contrast was administered after the control scan. 100 ml of the non-ionic contrast agent are given intravenously at the rate of 3 ml/sec. The study was done with the pancreatic parenchymal phase at 40 sec and venous phase at 90 sec. Reconstruction was done with a slice thickness of 3 mm. All images were viewed in a range of soft tissue window settings.

Clinical details, laboratory and computed tomography findings of the cases were recorded as per the proforma.

The severity of pancreatitis was scored using modified CT severity index and classified into three categories (mild, moderate and severe). The modified index is a 10-point scoring system derived by assessing the degree of pancreatic inflammation (0 to 4 points), pancreatic necrosis (0 to 4 points) and extrapancreatic complications (0 or 2 points). Clinical outcome parameters included the length of hospital stay, the need for surgical intervention, and the occurrence of infection, organ failure and death.

Collected data were analyzed using descriptive statistics by using tabulations, graphs, charts and proportions, percentages.
Inclusion criteria:
1. All the patients who are suspected/diagnosed with acute pancreatitis based on clinical and laboratory findings (serum amylase & serum lipase).
2. Patients who diagnosed acute pancreatitis on ultrasound
3. Patients who present as acute on chronic pancreatitis.

Exclusion criteria:
1. Chronic pancreatitis.
2. Pancreatic trauma.

Ethical committee clearance:
Ethical clearance was obtained from the institution.

Results and Analysis:
This study was conducted in the Department of Radio-diagnosis, Mysore medical college and research institute, Mysore from November 2016 to April 2018. The study comprised of total of 50 patients.

Study group characteristics:
Agedistribution:
Table 11: Age distribution of the study group.

| Age group (in years) | Number of patients | Percentage |
|----------------------|--------------------|------------|
| 15-25                | 13                 | 26         |
| 26-35                | 18                 | 36         |
| 36-45                | 9                  | 18         |
| 46-55                | 7                  | 14         |
| Above 55             | 3                  | 6          |
| Total                | 50                 | 100        |

The study included patients between the age group ranging from 15 years to 66 years with a mean age of 34.32 yrs. The maximum number of patients was seen in the age group of 26-35 years of the age group which consisted of 18 (36%) patients. (Graph 1 and Table 9)
Graph 1: Age distribution of patients with acute pancreatitis.

Sex distribution:
Table 12: Sex distribution of the study group.

| Gender | Number of patients | %  |
|--------|--------------------|----|
| Male   | 43                 | 86 |
| Female | 7                  | 14 |
| Total  | 50                 | 100|

In our study, out of 50 cases, 43 (86%) were male and 7 (14%) were female, with a male to female ratio of 8.6:1.4. (Graph 2, Table 10)

Graph 2: Gender distribution of patients with acute pancreatitis
**Etiology of pancreatitis:**

Table 13: - Etiology of pancreatitis.

| ETIOLOGY      | Number of patients (n=50) | %   |
|---------------|---------------------------|-----|
| Alcohol       | 38                        | 76  |
| Gallstones    | 8                         | 16  |
| Others        | 4                         | 8   |
| **Total**     | **50**                    |     |

In our study, 38 of 50 patients were alcoholic comprising of 76%. 8 (16%) patients had gallstones and remaining patients were grouped as others which consisted of 4 patients. (Graph -3, Table-11)

![Graph 3: Etiology of pancreatitis.](image)

**Diagnosis of pancreatitis:**

Mode of presentation:

Table 14: - Mode of presentation.

| Mode of presentation | Number of patients (n=50) | %   |
|----------------------|---------------------------|-----|
| Pain abdomen         | 50                        | 100 |
| Vomiting             | 46                        | 92  |
| Fever                | 15                        | 30  |
| Jaundice             | 5                         | 10  |

All 50 patients (100%) presented with pain abdomen. 46 patients (92%) had vomiting. 15 (30%) patients had fever and 5 (10%) of patients had jaundice at the time of admission. (Graph -4 and Table-12).
Graph 4: Mode of clinical presentation of acute pancreatitis.

Laboratory diagnosis:
Table 15: Patients with elevated amylase and lipase levels.

| Lab parameters | Elevated (%) | Normal (%) |
|----------------|--------------|------------|
| Amylase        | 86           | 14         |
| Lipase         | 90           | 10         |

Amylase was elevated in 43 (86%) patients at presentation. Lipase was elevated in 45 (90%) patients at presentation (Graph 5 and Table 13).

Graph 5: Patients with elevated amylase and lipase levels.

AMYLASE  

LIPASE
Ultrasonography:
Table 16: Patients with normal or abnormal findings on ultrasonography.

| USG          | Normal | Abnormal |
|--------------|--------|----------|
| Number of patients | 7      | 43       |
| Percentage   | 14     | 86       |

In 7 (14%) patients, ultrasound was normal. 43 (86%) patients had abnormal ultrasound findings such as bulky pancreas with altered echogenicity, peripancreatic fat stranding, fluid collection, ascites, and pleural effusion (Graph 6, Table 14).

Graph 6: Patients with normal or abnormal findings on ultrasonography.

Computed tomographic evaluation:
After diagnosing Acute Pancreatitis based on the clinical presentation, biochemical parameters, and ultrasonography, patients were subjected to CT scan of the abdomen according to the standard protocol. The severity of the pancreatitis was assessed by assigning points system by using Modified CT Severity Index and CT Severity Index.

Modified CT Severity Index:
The modified index is a 10-point scoring system derived by assigning points to the degree of pancreatic inflammation (0 to 4 points), pancreatic necrosis (0 to 4 points), and extrapancreatic complications (0 or 2 points). All patients were graded into mild (score 0-3), moderate (score 4-6), or severe (score 7-10).

Pancreatic inflammation:
Table 17: Pancreatic inflammation.

| Score | Number of patients | Percentage(%) |
|-------|--------------------|---------------|
| 0     | 0                  | 0             |
| 2     | 24                 | 48            |
| 4     | 26                 | 52            |

In our study, 24 (48%) patients had intrinsic pancreatic abnormalities with or without inflammatory changes in peripancreatic fat, towards whom 2 points were assigned. Remaining 26 (52%) patients had peripancreatic fluid collection or peripancreatic fat necrosis, to whom 4 points were assigned. (Graph 7, Table 15)
Inflammation in CT:
Pancreatic necrosis:
Table 18: Number and percentage of patients with scoring based on the pancreatic necrosis.

| Pancreatic necrosis | Number of patients | Percentage (%) |
|---------------------|--------------------|----------------|
| Nil (0)             | 15                 | 30             |
| <30% (2)            | 27                 | 54             |
| >30% (4)            | 8                  | 16             |

A total of 15 (30%) patients had no evidence of pancreatic necrosis on CT scan.

27 (54%) patients had less than 30% necrosis to which 2 points were assigned. Only 8 patients had more than 30% necrosis, to which 4 points were assigned (Table 16, graph 8).

Pancreatic necrosis:
Extrapancreatic complications:

Table 19: Number and percentage of patients with scoring based on the extrapancreatic complications.

| Extra pancreatic complications | Number of patients | Percentage(%) |
|--------------------------------|--------------------|---------------|
| absent (0)                     | 24                 | 48            |
| Present (2)                    | 26                 | 52            |

In our study, 13 patients (26%) had both ascites and pleural effusion. 9 patients (18%) had only isolated ascites. 4 patients had isolated left pleural effusion and 2 patients had isolated bilateral pleural effusion. 1 patient is found to have splenic vein thrombosis and 3 found to have portal vein thrombosis.

Graph 9: Percentage of patients with extra pancreatic complications based on MCTSI.

Modified CTSI Severity Index:

Table 20: Number and percentage of patients with grades of severity assessment based on MCTSI.

| Severity   | Number of patients | Percentage(%) |
|------------|--------------------|---------------|
| Mild (0-2) | 3                  | 6             |
| Moderate (4-6) | 35              | 70            |
| Severe (8-10) | 12              | 24            |

Modified CTSI Severity index was calculated by adding points assigned to each parameter. The severity of pancreatic is classified into three categories: mild (0-2 points), moderate (4-6 points) and severe (8-10 points). According to the Modified CT Severity Index, the patients were graded into mild (n=3), moderate (n=35) and severe (n=12), i.e. 6% patients had mild, 70% patients had moderate and 24% had severe pancreatitis (Table 18 and Graph -10).
Graph 10: Percentage of patients with grades of severity assessment.

Based on MCTSI:

Table 21: Distribution of CT grade in patients with AP using MCTSI.

| CT grade | Number of patients (n=50) | %  |
|----------|---------------------------|----|
| 2        | 3                         | 6  |
| 4        | 15                        | 30 |
| 6        | 20                        | 40 |
| 8        | 6                         | 12 |
| 10       | 6                         | 12 |

Graph 11: Distribution of CT grade in patients with AP.

CT Severity Index:
The severity of pancreatitis is scored using CT severity index and classified into three categories (mild, moderate, and severe).
he CTSIs a 10 point scoring system derived by assigning points to the degree of pancreatic inflammation (0 to 4 points) and pancreatic necrosis (0 to 6 points).

**Table 22:** Number and percentage of patients with grades of severity assessment based on CTSI.

| Severity | Number of patients | Percentage (%) |
|----------|--------------------|----------------|
| Mild     | 25                 | 50             |
| Moderate | 21                 | 42             |
| Severe   | 4                  | 8              |

According to the CTSI severity index, the patients were graded into mild (n=25), moderate (n=21) and severe (n=4). 50% of patients had mild, 42% of patients had moderate and only 8% of patients had severe pancreatitis as per CTSI score (Graph 12, Table -20).

**Graph 12:** Number and percentage of patients with grades of severity assessment based on CTSI.

**Comparison of total patients belonged to each severity indexes based on MCTSI and CTSI:**
The graph clearly depicts the distribution of patients in each category. In CTSI scoring system, 25 patients (50%) belonged to mild category, 21 (42%) patients had moderate and only 4 (8%) patients belonged to severe pancreatitis. But in MCTSI scoring system, 3 (6%) patients found to have mild pancreatitis and 35 (70%) patients found to have moderate and 12 (24%) patients severe pancreatitis.

The discrepancy is attributed to the inclusion of extrapancreatic complications in MCTSI scoring system. Hence, two extrapoints were added to the severity index in addition to the pancreatic inflammation and necrosis.
Graph 13: Percentage of patients each severity index based on MCTSI and CTSI.

Table 23: Patients developing Pseudocyst as a consequence of AP:

| Pseudocyst | Number of patients (n=50) | % |
|------------|---------------------------|---|
| Absent     | 35                        | 70 |
| Present    | 15                        | 30 |

Graph 14: Patients developing pseudocyst as a consequence of AP.

Table 24: Patients developing walled off necrosis in AP.

| Abscess     | Number of patients (n=50) | % |
|-------------|---------------------------|---|
| WON absent  | 47                        | 94 |
| WON present | 3                         | 6  |
**Graph 15:-** Patients developing WON in AP.

**Patient outcome parameters:**
Clinical outcome of patients in our study was assessed by the following parameters: end-organ failure, evidence of systemic infection, surgical intervention and the duration of hospital stay.

**End organ failure:**
Patients are followed up for the presence or absence of dysfunction in six separate organs as defined. End-organ failure is defined as the presence or absence of dysfunction in any of the six separate organs as defined.

Respiratory failure: PaO2 of less than 60 mmHg or the need for ventilatory support.

Cardiovascular system: Systolic blood pressure of less than 90 mmHg or the absence of hypovolemia with signs of peripheral hypoperfusion or the need for continuous infusion of vasopressor or inotropic agents to maintain asystolic blood pressure of more than 90 mmHg.

Central nervous system failure: Glasgow Coma Scale scores lesser than 6.

Renal failure: Either serum creatinine level that exceeded 300 μmol/L (3 mg/dL) or urine output of less than 500 mL/24 hours or less than 180 mL/8 hours by the need for hemodialysis or peritoneal dialysis.

Hepatic failure: Serum bilirubin levels greater than 100 μmol/L (3 mg/dL)

**Hematologic system failure:** Hematocrit level of more than 50%, WBC of less than 2,000/mm3, or platelet count of less than 40,000/mm3 of 50 patients (38%) are found to have end-organ failure. Hematologic system failure was seen in 11 (22%) patients in the acute pancreatitis in our study. Cardiovascular system failure was seen in 4 (8%), Renal failure was seen in 2 (4%) patients. Respiratory failure was seen in 1 patient (2%) and CNS failure was seen in 2 (4%) patient. Of these 19 patients who developed end-organ failure, 10 patients had moderate and severe pancreatic failure. These patients had moderate and severe pancreatic failure respectively (p=0.002) (Table 22, Graph 16).

**Table 25:** Number and percentage of patients who developed EOF based on MCTSI.

| Severity | Number of patients | Number of patients who developed EOF | Percentage (%) |
|----------|-------------------|-------------------------------------|----------------|
| Mild     | 3                 | 0                                   | 0              |
| Moderate | 35                | 10                                  | 28             |
| Severe   | 12                | 9                                   | 75             |
Graph 16: Percentage of patients who developed EOF based on MCTSI.

According to CTSI, of these 19 patients, 5 patients had mild, 11 patients had moderate, and 3 patients had severe pancreatitis. 26% of patients who had mild pancreatitis had end-organ failure, whereas end-organ failure was seen in 57% and 15% of patients who had moderate and severe pancreatitis, respectively (p=0.012). The above statistics show that a highly significant correlation exists between the prediction of end-organ failure with the classification according to the MCTSI (p=0.002) than CTSI (p=0.012).

Systemic infection:
A total of 15 (36%) patients who had fever and leukocytosis were considered to have systemic infection. Of these 15 patients, none of them had mild, 9 patients had moderate, and 1 patient had severe pancreatitis according to the MCTSI. Systemic infection is seen in 40% and 60% of patients who had moderate and severe pancreatitis, respectively. (p=p=0.001)(Graph-17, Table-23)

Table 26: Number and percentage of patients who developed systemic infections based on MCTSI.

| Severity | Number of patients | Number of patients who developed infections | Percentage (%) |
|----------|--------------------|---------------------------------------------|----------------|
| Mild     | 3                  | 0                                           | 0              |
| Moderate | 35                 | 9                                           | 25.7           |
| Severe   | 12                 | 10                                          | 83.3           |

Graph 17: Percentage of patients who developed systemic infections based on MCTSI.
According to CTSI, of these 15 patients, 6 patients had mild, 7 patients had moderate, and 2 patients had severe pancreatitis. 40% of patients who had mild pancreatitis had systemic infection, whereas systemic infection was seen in 46% and 13% of patients who had moderate and severe pancreatitis respectively (p = 0.172) (Figure 21).

With these statistical values, it can be concluded that there is a highly significant correlation between the prediction of systemic infection with the classification according to the MCTSI (p = 0.001), compared to the classification according to CTSI, which is not statistically significant (p = 0.172).

**Surgical Intervention:**
Surgical intervention was required in 5 (10%) patients. Four patients or USG guided aspiration had pseudocyst. One patient underwent surgical necrosectomy. The statistical p-value is not significant. (Table 24 and graph 18).

| Intervention                  | Number of patients (n=50) | %   |
|-------------------------------|---------------------------|-----|
| Surgical necrosectomy         | 1                         | 2   |
| USG - PC Aspiration           | 4                         | 8   |

**Graph 18:** Percentage of patients who needed Intervention in AP.

**Duration of Hospital Stay:**
Duration of hospital stay in our study was ranging from 3 to 25 days with a mean duration of 9.5 days. The mean duration of hospitalization in mild, moderate, and severe classes of Acute Pancreatitis according to Modified dCTSI Severity Index was 3, 7, and 20 days respectively. (Graph 19) Whereas it was 6, 12, and 17 days respectively as per the CTSI Severity Index.

The above values show that mean duration of hospitalization correlates well with the severity classification based on the MCTS I.
Graph 19: Mean Duration of hospitalization in severity based on MCTSI.

Table 28: Patient outcomes and duration of hospitalization in severity based on Modified CT Severity Index.

| MCTSI                      | MILD | MODERATE | SEVERE |
|----------------------------|------|----------|--------|
| Total number of patients   | 3    | 35       | 12     |
| Mean duration of hospitalization (in days) | 3    | 7        | 20     |
| Surgical intervention      | 0 (0%) | 2 (5.7%) | 3 (25%) |
| Infection                  | 0 (0%) | 9 (25%)  | 6 (50%) |
| End-organ failure          | 0 (0%) | 10 (28.5%) | 9 (75%) |

Graph 20: Patient outcomes based on MCTSI
Mortality rate:
The mortality rate in our study was 0%.

Table 29: Accuracy of CT grading in predicting systemic complications with EOF.

|                | Systemic complication absent | Systemic complication present | Total |
|----------------|-----------------------------|-------------------------------|-------|
| CT grade (8-10)| 3                           | 9                             | 12    |
| CT grade < 8   | 28                          | 10                            | 38    |
| Total          | 31                          | 19                            | 50    |

Sensitivity = True positive / True positive + False negative
Specificity = True negative / True negative + False positive
Positive predictive value = True positive / Total positives
Negative predictive value = True negative / Total negatives
Sensitivity = 100%
Specificity = 93.7%
Positive predictive value = 38.29%
Negative predictive value = 100%

Table 30: Accuracy of CT grading in predicting local complications.

|                | Local complication absent | Local complication present | Total |
|----------------|--------------------------|---------------------------|-------|
| CT grade moderate and severe | 29                       | 18                        | 47    |
| CT grade mild | 3                        | 0                         | 3     |
| Total          | 32                       | 18                        | 50    |

Sensitivity = 100
Specificity = 90.3%
Positive predictive value = 75%
Negative predictive value = 73.6%

Discussion:
This was a prospective study conducted from November 2016 to April 2018 in Mysore medical college and research institute. 50 cases diagnosed as acute pancreatitis based on history and serum amylase and lipase levels were included in this study. These patients underwent CECT of the abdomen and pelvis and were graded according to the modified CT severity index. The grades were correlated with patient outcome in terms of systemic complications leading to end-organ failure, local complications, duration of hospital stay.

Study group characteristics:
Age incidence:
The mean age of patients in the study was 34.32 years. Maximum number of patients was seen in the age group of 26-35 years of the age group which consisted of 18 (36%) patients. The age group affected reflects the etiologies of alcohol and gallstones which are common in the age group 26 to 35 yrs.

Sex distribution:
Most of the patients were male (86%) as compared to female (14%). Alcohol was the most common etiological factor in our study which is common in males, a high M:F ratio was observed.

These observations were similar to that of a study conducted by Lankish et al on 602 patients of acute pancreatitis which showed no correlation between age, gender with the severity of acute pancreatitis. The study also showed the maximum incidence of acute pancreatitis in the age group of 31 to 40 years similar to our study.
was the mean duration of stay in moderate grade and 20 days was the mean stay in the severe grade of pancreatitis.

Fewer patients in the mild grade in our study may possibly be due to the morphologic severity of pancreatitis.

Similarly, most of the patients were of moderate CT severity (70%) and minimum patients had a mild grade (7%).

Assessment of severity of acute pancreatitis:
The CT grades were classified into 2, 4, 6, 8 and 10 according to the MCTSI. We further classified the grades into mild (grade 2), moderate (grade 4 and 6) and severe (grade 8 & 10). The previous studies by Bollen et al 47 and Mortele et al 46 have classified grade 2 as mild, grade 4 and 6 as moderate and grade 8 and 10 as severe similar to our study.

The maximum patients were seen to fall in the grade 6 category (40%) and minimum patients (6%) were seen in grade 2 category. Similarly, most of the patients were of moderate CT severity (70%) and minimum patients had a mild grade (6%). Severe pancreatitis was present in 24 percent of patients. According to the study by Bollen et al 47, the morphologic severity of pancreatitis was graded as mild in 86 (44%), moderate in 75 (38%), and severe in 35 (18%) cases. In contrast to our study, their study had patients with severe pancreatitis as the minimum number of patients. Fewer patients in the mild grade in our study may possibly be explained by decreased use of CECT in mild cases of AP as CECT in not indicated in mild forms unless the diagnosis of AP is by itself doubtful or development of complications is suspected.

Most of the patients needed ward stay ranging from 2 to 25 days. 3 days was the mean stay in mild grade, 6 days was the mean duration of stay in moderate grade and 20 days was the mean stay in the severe grade of pancreatitis.

### Table 31: Age and sex distribution comparison in different studies.

| Study series   | Total no. of patients | Male | Female | M: F | Age in years |
|----------------|-----------------------|------|--------|------|--------------|
| Present study  | 50                    | 43   | 7      | 8.6:1.4 | 15-66 (mean-34) |
| Mortele et al 71 | 66                    | 37   | 29     | 1.2:7  | 19-87 (mean-53) |
| Bollen et al 72 | 179                   | 107  | 89     | 1.2:1  | 21-91 (mean-45) |
| Freeny et al 73 | 34                    | 26   | 8      | 3.2:1  | 31-71 (mean-56) |
| Bollen et al 74 | 150                   | 84   | 66     | 1.2:1  | 21-91 (mean-54) |
| Jauregui et al 72 | 30                   | 19   | 11     | 1.7:1  | 18-82 (mean-45) |

### Table 32: Etiological factors comparison in different studies.

| Study series   | Biliary stones | Alcohol abuse | Miscellaneous |
|----------------|----------------|---------------|---------------|
| Present study  | 16%            | 76%           | 8%            |
| Bollen et al 63 | 34%            | 22%           | 44%           |
| Dugernier et al 74 | 22%            | 60%           | -             |
| Freeny et al 73 | 20%            | 35%           | -             |
| Jauregui et al 72 | 53%            | 27%           | -             |

Etiology:
Chronic alcohol abuse is the most common etiological factor in our study constituting 76% of cases. Similar results were observed by Dugernier TL 74 and Freeny et al 73. In contrary, studies done by Bollen et al 63 and Jauregui et al 72 showed biliary stones as the predominant etiological agent.
The most common segment of the total duration of hospital stay was from 8 to 14 days (41%). A strong correlation was seen between patient's CT grade and total duration of hospital stay. A study by Mortele et al published in 2004 showed a significant correlation between MCTSI grade of pancreatitis and length of hospital stay (3 days for mild pancreatitis, 8 days for moderate and 12 days for severe grades). The differences between both the studies regarding the number of days of stay may be due to the differences in protocols regarding management in the individual hospital, the preferences of the treating doctors and the current standards and advances in the management of acute pancreatitis.

The local complications identified in the study were pseudocysts and abscess formation. Pseudocyst was seen in 15 patients (30%) in our study. Pseudocyst formation occurred in 50% of patients in a study conducted by Gonzalez et al. WOPN was detected in 3 patient (6%). The total percentage of patients developing local complications in the study was 36%. Presence of local complications was positively associated with CT grading. No local complications were seen in patients with mild pancreatitis. About 35% of patients with moderate pancreatitis and 60% of patients with severe pancreatitis had developed local complications.

In our study intervention was needed in form of surgical debridement in one patient with grade 10 of AP. Radiological intervention was needed in 4 patients (5%). Aspiration of pseudocyst was needed in 4 patient with the severe grade of pancreatitis. Thus patients who need an intervention have more severe CT grades. This is similar to the study by Bollen et al which demonstrated that development of local complications and the need for intervention was significantly associated with grade of pancreatitis.

19 of 50 patients (38%) are found to have end organ failure. Of these 19 patients who developed end organ failure, 10 patients had moderate and 9 patients had severe pancreatitis according to the MCTSI. End organ failure is seen in 28% and 75% of patients who had moderate and severe pancreatitis respectively. A significant association was noted between the development of systemic complications and grading of AP by Bollen et al.

A total of 15 (36%) patients developed systemic infection. Of these 15 patients, none of them had mild, 9 patients had moderate and 6 patients had severe pancreatitis according to the MCTSI. Systemic infection is seen in 40% and 60% moderate and severe pancreatitis respectively.

No mortality due to pancreatitis was observed in our study. In the study by Bollen et al mortality was seen in 6% of patients and in 1.5% of patients in the study by Mortele et al.

The accuracy of MCTSI in predicting the systemic complications were as follows: Sensitivity = 100%, Specificity = 93.8%, Positive predictive value = 38.2%, Negative predictive value = 100%.

The accuracy of MCTSI in predicting the local complications were as follows: Sensitivity = 100%, Specificity = 90.32%, Positive predictive value = 75%, Negative predictive value = 73.6%.

Table 33: Comparison table between the present study and others studies.

| Study series | Oursudy | Morteletal 60 | Bollenetal 63 |
|--------------|---------|---------------|---------------|
| Total number of patients | 50 | 66 | 196 |
| MCTSI | | | |
| Mild | 3 (6%) | 34 (52%) | 86 (44%) |
| Moderate | 35 (70%) | 22 (33%) | 75 (38%) |
| Severe | 12 (24%) | 10 (15%) | 35 (18%) |
| Mild | 25 (50%) | 42 (63%) | 136 (69%) |
|                      | CTSI Moderate | CTSI Severe | Modified CTSI Moderate | Modified CTSI Severe |
|----------------------|--------------|------------|------------------------|----------------------|
|                      | 21 (42%)     | 19 (28%)   | 41 (21%)               |                      |
| Duration of hospital stay in days | 3-25(mean-9.5) | 0-34(mean-7) | 0-113 (mean-6)         |          |
| Surgical intervention | 5 (10%)      | 10 (15%)   | 19 (10%)               |          |
| Infection            | 15 (35%)     | 21 (32%)   | 7 (4%)                 |          |
| End-organ failure    | 19(38%)      | 9 (14%)    | 38 (19%)               |          |
| Death                | 0 (0%)       | 2 (30%)    | 11 (6%)                |          |

The MCSTI accurately correlated with pancreatic inflammation and the need for intervention compared with APACHE II. CT is the modality of choice for detecting the local complications. Thus the MCTSI is as useful as APACHE II in predicting the severity of AP in terms of the number of days of hospital stay and organ failure and is better than APACHE II in detecting the local complications and confirming necrosis in AP. But MCTSI has a disadvantage that the study cannot be carried out within 48 hrs as this is the time taken to demonstrate necrosis. However, APACHE II score can be calculated even at the time of admission.

Recommendations on the basis of observations from our study:
1. We would propose that APACHE II can be used in early stages of AP and be supplemented by MCTSI which can be used after 48 hrs after admission to further manage the patients.
2. The grading of AP can be classified as mild (grade 2 and grade 4), moderate (grade 6) and severe (grade 8 and 10) contrary to other previous studies which classified it into mild (grade 2), moderate (grade 4 and grade 6) and severe (grade 8 and 10) as grade 2 and 4 patients had similar outcome.
3. Patients who have a severe grade of AP should be transferred to a center which has the facility of ICU if needed.
4. As patients with the moderate and severe grade of AP have a higher possibility of local complications a follow-up study with ultrasound / CT may be considered in these patients.

The limitations of the study are as follows:
1. The small sample size.
2. Nonrandomized study.
3. Patients with mild acute pancreatitis could not be included in the present study as a CT scan is not indicated in all cases of pancreatitis.

Summary:
This was a prospective study of 50 cases of clinically diagnosed acute pancreatitis and confirmed by serum amylase and lipase levels. CT severity index and Modified CT severity index grading were done after contrast-enhanced CT of abdomen and pelvis in all patients. Correlation of CT severity index and modified CT severity index grades was done with patient outcome taking local and systemic complications and duration of hospital stay as parameters.
1. The age group of patients was 15 to 66 years with maximum patients (36%) between 26 and 30 years.
2. 86% of patients were male. Male to female ratio was 8.6:1.4 with male preponderance.
3. Chronic alcohol abuse was the most common cause of pancreatitis (76%), the second was gallstones (16%) and others (8%).
4. All patients presented with pain abdomen. 92% had vomiting. 30% of patients had fever and 10% of patients had jaundice at the time of presentation.
5. Amylase was elevated in 86% of patients. Lipase was elevated in 90% of patients.
6. 86% of patients had features of pancreatitis on ultrasound and in 14% ultrasound was normal.
7. Pancreatic inflammation was seen in 100% of patients. Of these, 30% of patients had no evidence of extrapancreatic complications. 54% of patients had less than 30% necrosis and only 16% had more than 30% necrosis.
8. 48% of patients had no evidence of extrapancreatic complications. 52% of patients had one or more extrapancreatic complications.
9. According to the Modified CT Severity Index, 6% of patients had mild, 70% of patients had moderate and 24% had severe pancreatitis.
10. Duration of hospital stay ranged from 3 to 25 days with a mean duration of 9.5 days.
11. Mortality rate was 0%.
12. 38% of patients were considered to have end organ failure. Hepatic failure is the most common systemic failure seen in 22% of patients. 36% of patients had evidence of systemic infection. 10% of patients required surgical interventions.

Conclusion:

1. Grading by modified CT severity index has a significant correlation with the necessity of ICU admission, duration of ICU stay and total duration of hospital stay.
2. Modified CT grading correlates directly with the development of local and systemic complications.
3. Modified CT severity index can be used to predict the possibility of developing local and systemic complications and necessity of ICU admission.
4. Modified CT severity index can predict the need for interventions.
5. Extrapancreatic complications, when included in the CT scoring system (MCTSI), were significantly correlated with end-organ failure and adverse clinical outcomes. Hence, MCTSI may be more useful in scoring systemic than CT.
6. MCTSI is a very useful tool for the screening of patients with acute pancreatitis for the classification of severity accurately and to predict the clinical outcome when used within three days of symptom onset.

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