"COMPARATIVE STUDY OF RANSON'S VERSUS APACHE II SCORING SYSTEMS IN PREDICTING THE CLINICAL OUTCOME IN PATIENTS WITH ACUTE PANCREATITIS"

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M.S. (GENERAL SURGERY) BRANCH-I

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I hereby declare that the dissertation entitled **"COMPARATIVE STUDY OF RANSON'S VERSUS APACHE II SCORING SYSTEMS IN PREDICTING THE CLINICAL OUTCOME IN PATIENTS WITH ACUTE PANCREATITIS"** is a bonafide and genuine research work carried out by me under the guidance of **Dr.P. AMUTHA , M.S.,** Professor, Department of General Surgery, Madurai Medical College, Madurai.

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CONTENTS

1. INTRODUCTION

Acute pancreatitis is a common entity encountered during routine surgical practice and it poses a great challenge to the treating surgeon. It is a protean disease capable of wide clinical variation, ranging from mild discomfort to severe consequences.

It is an inflammatory condition of the pancreas that is painful and at times deadly. Despite the great advances in critical care medicine over the past 20 years, the mortality rate of acute pancreatitis has remained at about 10%. Diagnosis of pancreatic problems is often difficult and treatments are therefore delayed because the organ is relatively inaccessible. There are no easy ways to see the pancreas directly without surgery, and available imaging studies are often inadequate.

2. AIMS AND OBJECTIVES

Present study was aimed at analyzing patients admitted to Department of General Surgery, Madurai Medical College with a diagnosis of acute pancreatitis during the period between December 2015 and May 2017 with the following

OBJECTIVES:

- To assess the severity of acute pancreatitis using Ranson's scoring system and APACHE II scoring system.
- To compare these two scoring systems with respect to their accuracy in predicting the outcome in cases of acute pancreatitis.

3. REVIEW OF LITERATURE

Thomas L Bollen et al (2012, April)[2] did a comparative study of radiological and clinical scoring systems in acute pancreatitis in 346 consecutive patients and found that CTSI (contrast-enhanced CT only) demonstrated the highest accuracy but this was not statistically significant. Hence he concluded that a CT on admission solely for severity assessment in Acute pancreatitis is not recommended.

Rawad Mounzer et al (2012, March)[3] did a Comparative study of existing clinical scoring systems to predict persistent organ failure in patients with acute pancreatitis and found that the Glasgow score was the best classifier at admission for predicting severity although all scoring systems showed modest accuracy.

Fabre A et al (2012, Feb)[4] studied 48 children with acute pancreatitis. Ranson's, Glasgow and CT severity index were calculated in all patients. For Ranson's score sensitivity was 56% and specificity was 85% for predicting severity, compared to 80% and 86% respectively for CT severity score. So he concluded that for paediatric cases of acute pancreatitis CT severity index is best for predicting severity.

Zhang WW et al (2011, September)[5], investigated the correlation between CT pancreatic inflammatory infiltration degree of severe acute

pancreatitis (SAP) and the clinical disease severity in 83 patients and found that among the CT severity indexes, the score of extra-pancreatic inflammation spread is superior and has good correlation with APACHE II and Ranson's scores.

Su Mi Woo et al (2011, May)[6] prospectively studied 44 patients with acute pancreatitis comparing serum Procalcitonin with Ranson's, APACHE II, Glasgow and Balthazar CT Severity Index scores in predicting severity of acute pancreatitis and found that accuracy of serum procalcitonin was 77% compared to 93% for Ranson's score and 77% for APACHE II score. He inferred Serum PCT was a promising simple biomarker and had similar accuracy as APACHE II scores in predicting severity of acute pancreatitis.

Chavarri Herbozo CM et al (2011, Jan)[7] compared hemoconcentration, APACHE II and Ranson's as early predictors of severity in patients with acute pancreatitis in Peru in 151 patients and found area below the ROC curve of 0.89 and 0.68 for APACHE II and Ranson's scores respectively. Hence he concluded that Hemoconcentration and Ranson's score are not as useful as APACHE II score in predicting severity in acute pancreatitis.

Ekrem Kaya et al[8] (2007) prospectively studied 199 patients with acute pancreatitis and found that CRP > 142 mg/L, BUN > 22 mg/dL, LDH > 667

U/L, base excess > -5, CT severity index > 3 and APACHE score > 8 were related to morbidity and mortality.

Yuk Pang et al[9] (2006) prospectively studied 101 patients of acute pancreatitis. Of these 11.9% patients had severe pancreatitis. Ranson's, APACHE II and APACHE O scores were performed in all patients on admission as well as after 48 hours. AUC for the three scores on admission were 0.549, 0.904 and 0.904 respectively. AUC for the same scores after 48 hours were 0.808, 0.955 and 0.951 respectively. So they concluded that APACHE II is more accurate in predicting severity than Ranson's score. Addition of Obesity as a criterion did not improve the accuracy.

Masahiko Hirota et al[10] (2006) did a review of literature about the various severity scoring systems predicting severity in acute pancreatitis to compare them with the new validated JPN scoring system. Examination of the results of 1240 patients showed that the JPN score had almost the same value for assessment as the APACHE II score and the Ranson's score.

Ting-Kai Leung et al[11] (2005) reviewed 121 patients who underwent helical CT within 48 hours after the onset of symptoms of a first episode of AP between 1999 and 2003. They also reviewed Ranson's and APACHE II scores in the same patients and classified 85 patients (79%) as having mild acute pancreatitis (CTSI<5) and 22 patients (21%) as having severe acute

pancreatitis. They concluded that Balthazar computed tomography severity index is superior to Ranson's criteria and APACHE II scoring system in predicting acute pancreatitis outcome.

Taylor SL et al[12] (2005) did a retrospective chart review of 49 patients diagnosed as acute pancreatitis. They calculated Ranson's, Glasgow, MOSS and APACHE II scores in all patients. They studied if these scores were predictive of patient outcome in the form of length of hospital stay. They found that Glasgow and MOSS showed correlation with patient outcome when APACHE II and Ranson's did not, although authors did agree that sample size was too small to change practice based on this study.

Chatzicostas et al[13] (2003) prospectively studied 78 patients with acute pancreatitis. Data pertinent to scoring systems were recorded 24 hours (APACHE II and III scores), 48 hours (Ranson's score) and 72 hours (Balthazar computed tomography severity index) after admission. Statistical analysis was performed by using receiver operating characteristic curves and by comparing likelihood ratios of positive test (LRPT). LRPT were 2.4157 for Ranson's, 4.0980 for APACHE II, 3.6670 for APACHE III score and 11.2157 for the Balthazar score. Balthazar Computed Tomography Severity Index is Superior to Ranson's Criteria and APACHE II and III Scoring Systems in predicting Acute Pancreatitis Outcome. However the Ranson's

and APACHE scores perform slightly better with respect to organ failure prediction.

Chatzicostas C et al[14] (2002) prospectively studied 153 patients with acute pancreatitis. Data pertaining to the scoring systems were recorded 24 (the APACHE II scores) and 48 hours (the Ranson's score) after admission. Analysis was performed by using receiver operating characteristic curves (ROC), area under curve (AUC), and by comparing likelihood ratios of positive test (LRPT). AUC for Ranson's was found to be significantly larger than AUC for APACHE II and APACHE III scores (0.817, cut-off > or = 3; 0.618, cut-off > or = 10; and 0,676, cut-off > or = 42 respectively). Ranson's score achieved the highest sensitivity and the lowest false-negative rate, but the positive and negative predictive values and LRPT were of similar extent for all three scores. Ranson's criteria proved to be as powerful a prognostic model as the more complicated APACHE II and III scoring systems, but with the disadvantage of a Lankisch PG et al[15] (2002) prospectively studied 326 patients with a first attack of acute pancreatitis. The following parameters for the severity of the disease were used: Atlanta classification, Ranson's score, Imrie score and Balthazar score (CT) in addition to APACHE II. In 74 (28%) of the 262 patients with interstitial pancreatitis, the APACHE II score was at least eight points, indicating severe pancreatitis

(overestimation of the disease), whereas the score was less than eight in 41 (64%) of 64 patients with necrotizing pancreatitis (underestimation). Sensitivity was 36%; specificity was 72%; the positive predictive value was 24%; and the negative predictive value was 82%. So they concluded that APACHE II score is unreliable to diagnose necrotizing pancreatitis.

Williams M et al[16] (1999) retrospectively analysed 273 patients with acute pancreatitis. Objective was to assess concordance between length of stay as well as death, and Ranson's criteria, APACHE III score and modified Glasgow Coma score. APACHE III scores >30 at 96 hours, 5 or more Ranson's criteria, and a modified Imrie score of >3 predicted those who died or had multiple complications. Those patients with combined 48-hr and 96-hr APACHE III scores of >60 either died or had hospitalizations of >60 days. They found that magnitude of correlation between the length of stay and the 96-hr APACHE III and modified Imrie is larger than that between length of stay and Ranson's criteria.

Paredes Cotoré JP et al[17] (1995) prospectively studied 113 patients with acute I scores were analyzed. Sensitivity of Ranson's was 79% and APACHE II was 86%. They concluded that APACHE II system was the best for the early detection of severe acute pancreatitis.

Vesentini S et al[18] (1993) Prospectively compared C-reactive protein level, Ranson's score and contrast-enhanced computed tomography in the prediction of septic complications of acute pancreatitis in 59 consecutive patients. Although all prognostic indices correlated significantly with sepsis, multivariate logistic regression analysis showed that the only variables predictive of the risk of subsequent sepsis were the presence and extent of necrosis. So they concluded that CT severity index is better than Ranson's.

Roumen RM et al[19] (1992) retrospectively studied 5 Scoring systems for predicting outcome in acute hemorrhagic necrotizing pancreatitis in 39 patients. These included Ranson's, Imrie, APACHE II, multiple organ failure (MOF) and Sepsis Sensitivity Score (SSS). Sensitivity in prediction of death was best with APACHE II score greater than 9 (96%) and Ranson's score greater than or equal to 3 (95%). Of the five scores, MOF greater than or equal to 4 gave the best equilibration between sensitivity (73%) and specificity (76%) and the strongest prediction of lethal outcome (80%). They found that APACHE II scoring is best for grading the severity of disease on admission to intensive care, while the MOF score is best for monitoring the degree of organ dysfunction and the intensity of supportive treatment.

Larvin M et al[20] (1989) compared the value of APACHE-II score with Ranson's and Imrie scores in the evaluation and monitoring of acute pancreatitis in 290 attacks. At 48 hour, APACHE-II was most accurate, and correctly predicted outcome in 88% of attacks, compared with 69% for Ranson's and 84% for Imrie scores.

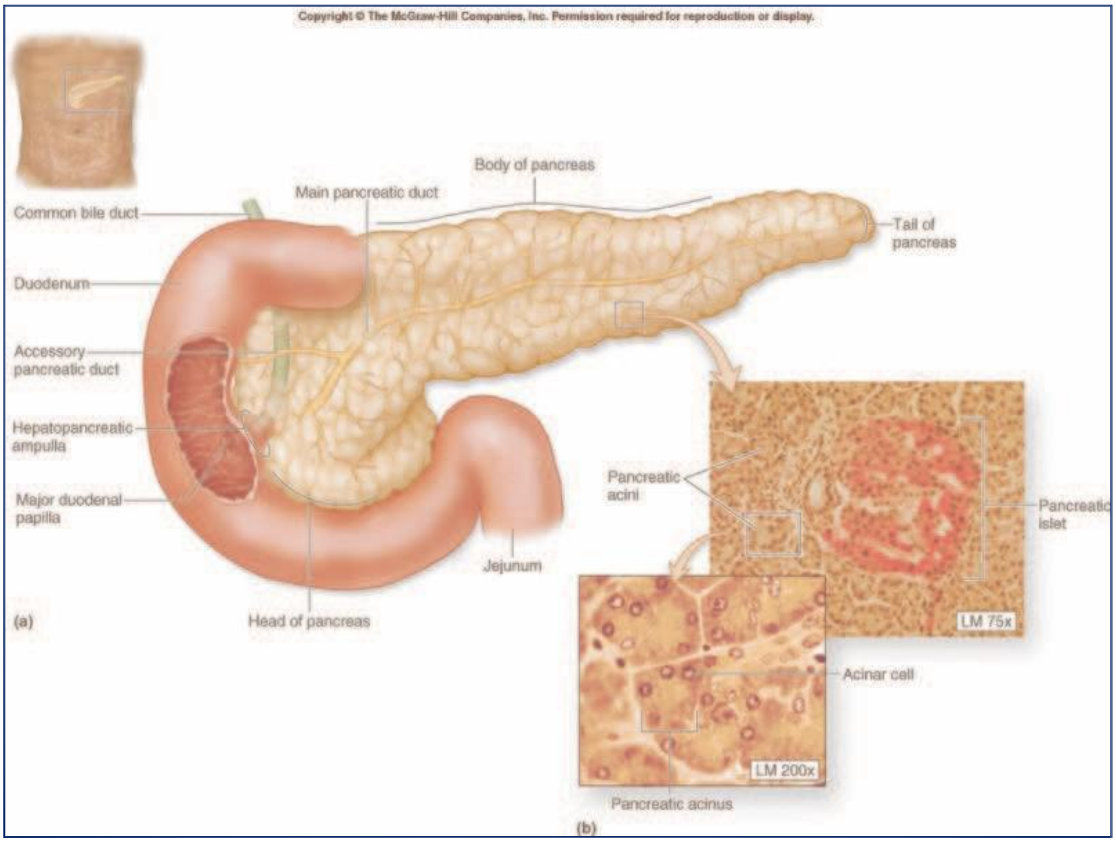
APACHE-II predicted 73% of pancreatic

collections at 48 hours, compared with 65% for Ranson's and 58% for Imrie scores. They concluded that APACHE II is best for monitoring the progression of acute pancreatitis.

HISTORY

- Earliest account of acute pancreatitis comes from the fatal illness of Alexander the great.
- Reginald Fitz presented his 1st landmark paper on acute pancreatitis in 1889[21].
- Opie (1873 -1971) proposed the common channel theory regarding the pathogenesis of acute pancreatitis.
- Comfort et al described the pathogenesis of alcohol induced pancreatitis in 1946.
- Comfort and steinberg were 1st to describe hereditary pancreatitis in 1952.

ANATOMY



The pancreas lies posterior to the stomach and lesser omentum in the retro peritoneum of the upper abdomen. It extends from the medial edge of the duodenal C loop to the hilum of spleen, lies anterior to the inferior vena cava, aorta, splenic vein and left adrenal gland.

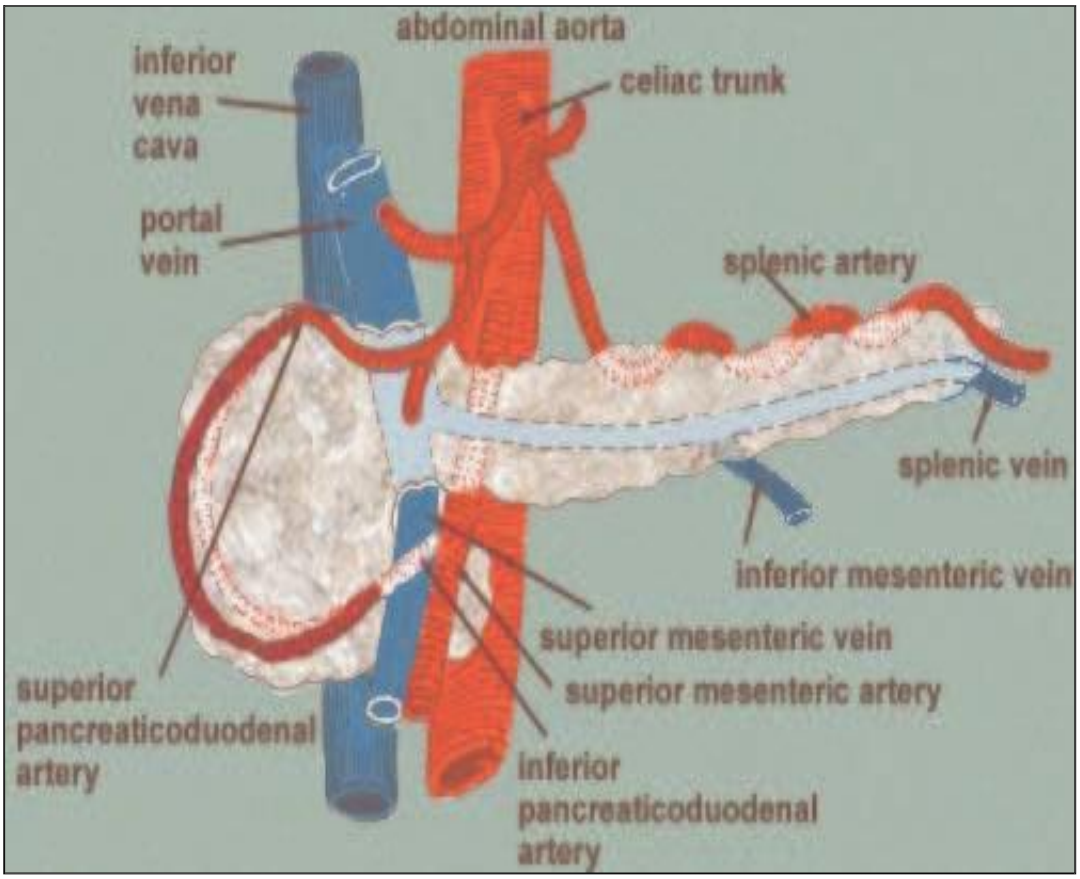


Figure No 2 – Blood supply of Pancreas

Arterial supply of pancreas is derived from celiac trunk and superior mesenteric artery through splenic and pancreatico-duodenal arteries. Venous drainage of pancreas drains into the portal, splenic and superior mesenteric veins. Lymphatics follow the blood vessels to the pancreatico-splenic nodes and pyloric lymphnodes, efferents of which drain into the celiac, hepatic and superior mesenteric lymphnodes. Nerve supply of pancreas is derived from the vagus and splanchnic nerves.

PHYSIOLOGY

Pancreas plays a vital role in the digestion and absorption of food from the gut and plays an important role in glucose homeostasis. Humoral control is by two hormones -- secretin and pancreozymin, liberated from duodenum and proximal jejunum. Secretin induces watery alkaline secretion rich in bicarbonate. Pancreozymin produces juice rich in enzymes namely amylase, lipase, trypsinogen.

EXOCRINE FUNCTIONS

TRYPSIN	PANCREATIC LIPASE
CHYMOTRYPSIN	PHOSPHOLIPASE A2
ELASTASE	COLIPASE
CARBOXYPEPTIDASE A & B	RIBONUCLEASE AMYLASE
DEOXYRIBONUCLEASE	

ENDOCRINE FUNCTIONS

ALPHA CELLS	- GLUCAGON BETA CELLS
- INSULIN	
DELTA CELLS	- SOMATOSTATIN
F CELLS	- PANCREATIC POLYPETIDE

ETIOLOGICAL FACTORS[23]

I. Toxic	Alcohol Organophosphorus and other toxic substances
II. Metabolic	Hyperlipidemia Hypercalcemia Venoms (scorpion, spiders)
III. Mechanical	Cholelithiasis Congenital malformations ❖ Pancreas divisum ❖ Annular pancreas Anatomical variants: ❖ Duodenal duplication ❖ Duodenal diverticulum ❖ Choledochal cyst ❖ Ampullary dysfunction ❖ Trauma
IV. Infections	Virus : Mumps, Coxsackie A, HIV, CMV Bacteria : Mycobacterium tuberculosis Parasites : Ascaris Others : Mycoplasma
V. Drugs	Furosamide Thiazide 6 Mercaptopurine Azathioprine Valproic acid Tetracyclin Trimethoprim - sulfamethoxazole Metronidazole Estrogen Isoniazid Sulindac L – asparagenase Acetaminophen

VI.	Miscellaneous	Vascular ❖ Vasculitis ❖ Embolisms ❖ Hypercoagulability Autoimmune disorders ❖ Sjogren syndrome ❖ Primary sclerosing cholangitis ❖ Celiac disease ❖ Autoimmune hepatitis

Table No 1 - Etiology of acute pancreatitis

Biliary pancreatitis

It is the most common cause of acute pancreatitis. It has been observed that an episode of acute pancreatitis is frequently preceded by passage of stone into duodenum. Stone can be retrieved from stools in roughly 90% patients with stone induced pancreatitis.

Various proposed mechanism for biliary pancreatitis are:

1. **"**Common channel theory**"** proposed by Opie[24] -- Biliary stone gets lodged in the common channel between bile duct and pancreatic duct which results in reflux of bile into pancreatic duct resulting in pancreatitis.
2. **"**Duct obstruction theory**"**-- Recent studies have shown that bile reflux is neither necessary nor sufficient to cause pancreatitis; hence duct obstruction theory has been proposed[25]. Accordingly, stone induced

duct edema leads to duct obstruction and duct hypertension which in turn triggers pancreatitis.

Alcohol induced pancreatitis

It is the most frequent cause for morphologically defined chronic pancreatitis though it can also cause acute episodes. There is no threshold rate of consumption below which acute pancreatitis doesn't occur. It has been observed that mean alcohol consumption in alcohol induced pancreatitis is 150 – 175 g/day. Mean duration for the same is 18+/-11 yrs for males and 11+/-8 yrs for females[26].

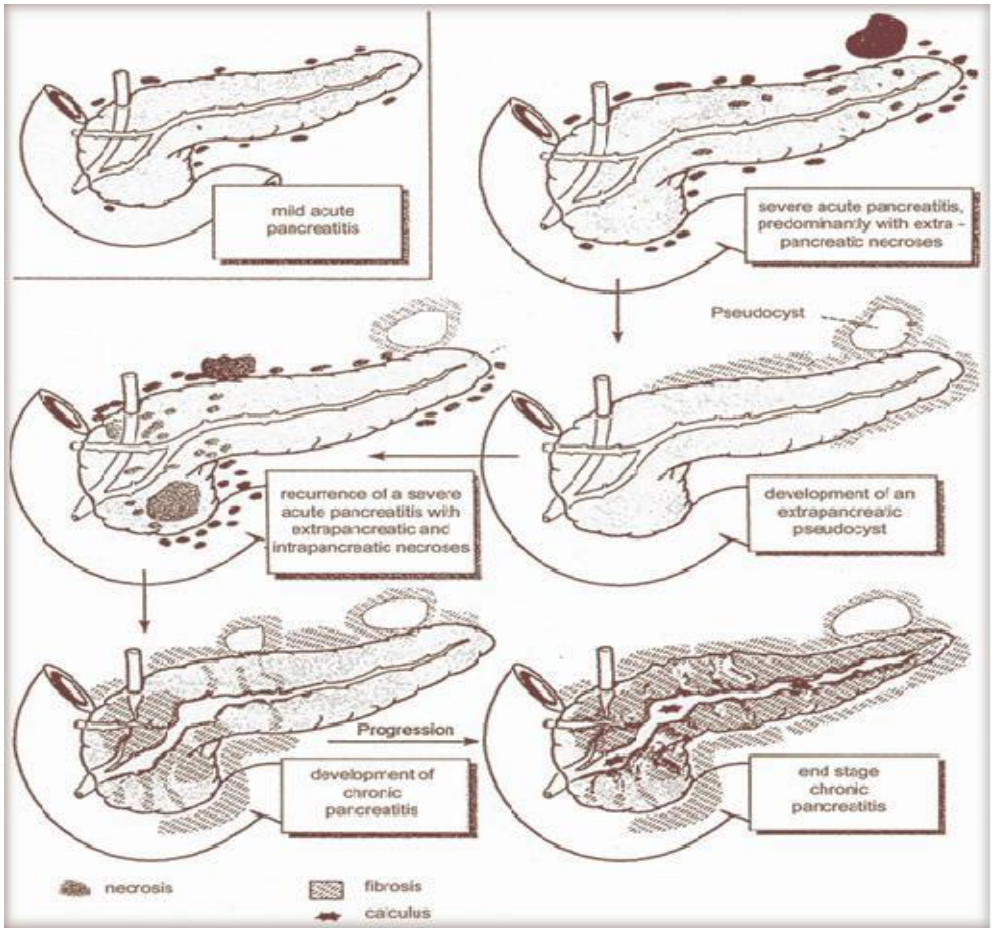


Figure No 3 - Natural history of alcohol induced pancreatitis

Various proposed mechanisms for alcohol induced pancreatitis are:

Alcohol induces spasm of sphincter of oddi resulting in ductal hypertension.

Alcohol induces hypertriglyceridemia which leads to increased production of free fatty acids which in turn are toxic to pancreatic acinar cells.

It also stimulates intrapancreatic generation of free radicals which injure the acinar cells.

It reduces pancreatic blood supply by affecting microcirculation and hence induces pancreatic ischemia.

It stimulates acinar cells to secrete pancreatic juice which is rich in proteins.

a. Protein rich fluid leads to formation of protein plug leading to duct obstruction.
b. There is secretion of enzymes which overwhelm the protective enzymes leading to pancreatic auto digestion.

Idiopathic Pancreatitis

In about 20% cases of acute pancreatitis, no cause can be identified in spite of extensive work-up. Probable mechanisms in these are:

- ❖ Gall bladder sludge or microcrystals.
- ❖ Sphincter of oddi dysfunction leading to ductal hypertension.
- ❖ Subclinical mutations in cystic fibrosis gene (CFTR gene).

PATHOGENESIS

Exact mechanism is not known. Concepts which have been proposed are based on the few experimental animal studies available. Most accepted mechanism is[27]

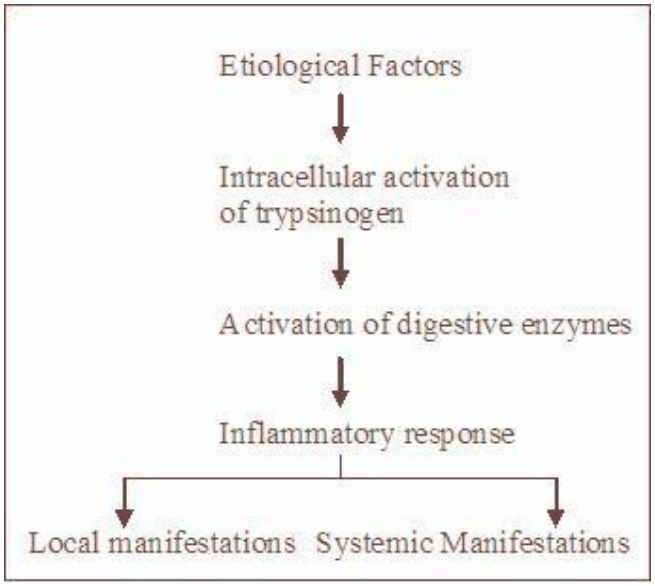


Figure No 4 - Mechanism of development of acute pancreatitis

Following are some of the known concepts in the pathogenesis of acute pancreatitis:

1. Only 10% of alcohol abusers will develop the disease. Also every individual with gall stone or hypercalcemia do not develop the disease. Similarly severity of the disease varies from one patient to the other. Reason behind all these is not yet known.
2. Acute Pancreatitis begins within the acinar cells as shown by animal models in which the main pancreatic duct was ligated.

3. The exocrine pancreas synthesizes and secretes various digestive enzymes like trypsinogen, chymotrypsinogen, lipase, amylase etc. These get activated only in duodenum. Trypsin which is derived from trypsinogen is the principal activator of all these enzymes. Even normally a small proportion of trypsinogen gets activated spontaneously inside the acinar cells. But the various protective mechanisms present within pancreas wash out the activated Trypsin so that there won't be any damage to the gland. These include, Serine protease inhibitor Kazal type 1 (SPINK1) Mesotrypsin Enzyme Y 1-antitrypsin, 2-macroglobulin[28]
4. Once these defensive mechanisms are overcome, there is intracellular activation of enzymes which is also favoured by lysosomal enzymes like catepsin B which lead to pancreatic self digestion.
5. Trypsin also activates other pathways, such as complement, coagulation or fibrinolysis, extending the process outside the gland which is responsible for systemic manifestation of the disease.
6. Occasionally this acute inflammatory process is associated with a systemic inflammatory response syndrome (SIRS) mediated by cytokines and pancreatic enzymes released in to general circulation that may affect distant organs, giving rise to respiratory distress, renal failure, myocardial depression and shock or metabolic

alterations. Finally, a MODS may with vital risk of necrotic tissue infection, a situation translocation of intestinal pathogens plays an important role.[29] occur where

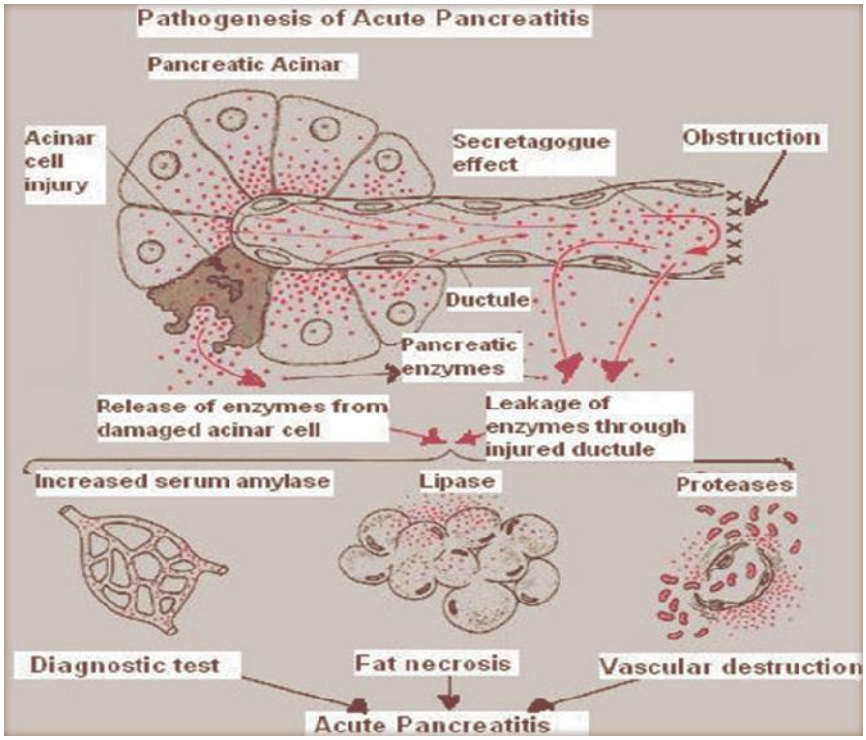


Figure No 5 - Pathogenesis of acute pancreatitis

7. Genetic factors implicated in pathogenesis of acute pancreatitis are
   - ❖ Cationic trypsinogen gene (*PRSS1*)
   - ❖ Cystic fibrosis transmembrane conductance regulator gene (*CFTR*)[30]
   - ❖ Polymorphisms in SPINK1

CLINICAL FEATURES

Symptoms:

Pain abdomen : Most common symptom. Typically pain

- Located in upper abdomen[epigastrium / right hypochondrium]
- Radiates to back
- Abrupt in onset reaching to maximum level within hours
- Very severe
- Stabbing type
- Constantly present throughout the episode
- May be referred to shoulder because of pleuritic component
- Typically relieved by leaning forward or lying down on one side with drawing up of legs.

Nausea, vomiting and severe retching

Physical findings:

Typically pancreatitis patients are seen rolling around in the bed or moving around trying to find the most comfortable position for pain relief unlike those with hollow viscus perforation who will be lying still in the bed.

Per Abdomen:-

Tenderness either localized to epigastrium or diffuse all over abdomen

Guarding and rigidity

Absent bowel sounds due to paralytic ileus

Subcutaneous fat necrosis leading to subcutaneous tenderness and edema.

Retroperitoneal haemorrhage leading to bluish discolouration in

- Umbilical area – Cullen's sign
- Loin – Grey Turner's sign
- Groin – Fox's sign

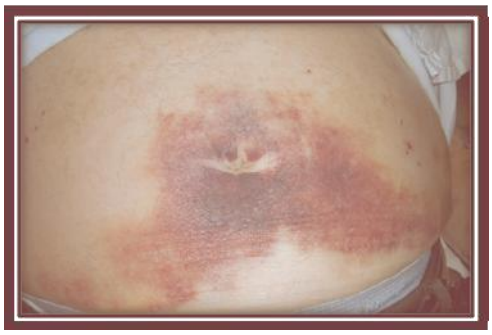


Figure No 6 - Cullen's sign

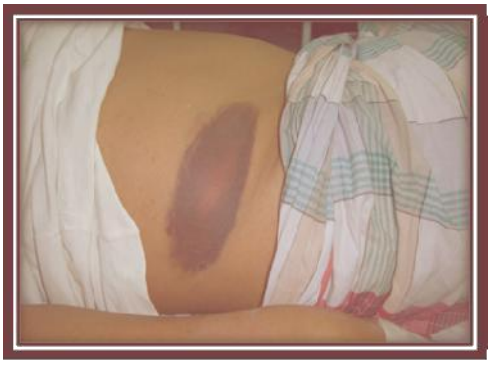


Figure No 7 - Grey Turner's sign

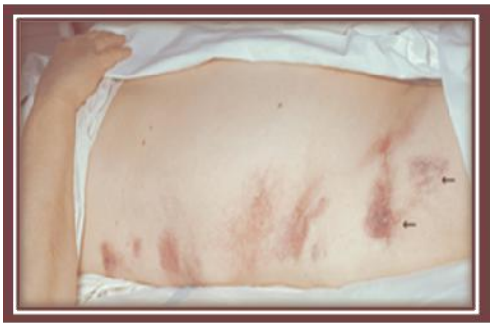


Figure No 8 - Fox's sign

General examination:-

- Tachycardia / hypotension and tachypnea related to hypovolemic state.
- Hyperthermia related to release of pro inflammatory cytokines.
- Jaundice which may be a cause i.e., due to cholelithiasis or may be the effect
    - i.e., due to cholestasis or biliary obstruction secondary to compression by edematous pancreatic head.

Decreased breath sounds in basal lung fields secondary to atelectasis or pleural effusion.

Diagnosis:-

Diagnosing acute pancreatitis requires clinical, serological and imaging correlation. Various serum markers used in the diagnosis and prognosis of acute pancreatitis are:

Laboratory Test	Time of onset (hours)	Purpose	Clinical observation / Limitations
Alanine transaminase	12 to 24	Diagnosis and etiology	Associated with gallstone pancreatitis; threefold elevation or greater in the presence of acute pancreatitis has appositive predictive value of 95 percent pancreatitis.
Amylase	2 to 12	Diagnosis	Most accurate when at least twice the upper limit of normal; amylase levels and sensitivity decrease with time from onset of symptoms.
C-reactive protein	24 to 48	Predictive of severity	Late marker; high levels associated with pancreatic necrosis.
Interleukin-6	18 to 48	Predictive of severity	Early indication of severity
Interleukin-8	12 to 24	Predictive of severity	Early indication of severity
Lipase	4 to 8	Diagnosis	Increased sensitivity in alcohol-induced pancreatitis; more specific and sensitive pancreatitis
Phospholipase A_2	24	Predictive of severity	Associated with development of pancreatic necrosis and pulmonary failure
Procalcitonin	24 to 36	Predictive of severity	Early detection of severity; high concentrations in infected necrosis
Trypsinogen activation peptide	Within a few hours	Diagnosis and predictive of severity	Early marker for acute pancreatitis and close correlation to severity

Table No 2 - Serum markers for the diagnosis of acute pancreatitis[31,32]

Among these, the two most important markers for diagnosis are serum amylase and lipase.

Serum amylase

It is the most common serum marker used in the diagnosis of acute pancreatitis.

It begins to elevate 2-12 hr after the onset of symptoms and remains elevated for 3-6 days.

- If it remains elevated for more than 1week it indicates development of a complication.
- Urinary levels remain elevated longer than serum levels.

Mechanism of hyperamylasemia in pancreatitis:

- Older theory- Normally amylase is secreted from apex of acinar cells. In acute pancreatitis, it is secreted from basolateral surface. So it has better access to lymphovascular system.
- Recent theory- In acute pancreatitis, there is loss of cell to cell adhesions.

So amylase has better access to vascular system.

It is only diagnostic but has no prognostic value. So that even in severe cases, there may be mild elevation. In 10% of cases of lethal pancreatitis, serum amylase may be normal or near normal[33].

Extrapancreatic sources of amylase

- Salivary gland
- Lung
- Ovary
- Prostate

False positive elevation of amylase levels

- Acute cholecystitis
- Intestinal ischemia
- Hollow viscus perforation
- Intestinal obstruction

False negative elevation of amylase levels

- Acute on chronic pancreatitis
- Severe pancreatitis with overwhelming necrosis
- Acute pancreatitis due to hypertriglyceridemia

Macroamylasemia

It occurs in 0.5 % patients. In this condition, there is normal levels of amylase. But amylase is bound to a high molecular weight protein. So it is not excreted by kidneys. So levels remain elevated. But in this situation, urinary amylase will be very low unlike as in cases of acute pancreatitis.

Amylase to creatinine clearance ratio:

- More reliable than the usual serum levels.
- Mainly useful to differentiate actual elevation from macroamylasemia.
- Amylase/Creatinine Clearance Ratio = Urine Amylase (U/L) x Serum Creatinine (mg/dL) x 100% / Serum Amylase (U/L) x Urine Creatinine (mg/dL).
- Value greater than 5 % indicates acute pancreatitis.

Serum lipase

- Advantage: More specific than amylase[34].
- Disadvantage: Levels remain elevated for one week. So not sensitive to detect development of complications.

Other blood investigations

Increased haemoglobin, haematocrit, blood urea nitrogen and creatinine due to hypovolemia.

Hypoalbuminemia secondary to fluid replacement with crystalloids.

Hyperbilirubinemia which may be a cause or effect of acute pancreatitis.

Hypochloremic metabolic alkalosis secondary to excessive vomiting.

Hypocalcemia secondary to

- Hypoalbuminemia
- Calcium sequestration into pancreatic fat necrosis

- ❖ Bone calcium does not respond to paratharmone
- ❖ Associated hypomagnesemia

Hyperglycaemia due to

- ❖ Associated Diabetes
- ❖ Increased glucagon release
- ❖ Increased catecholamine release

Imaging studies

A. X Ray abdomen:

Not much useful in diagnosis of acute pancreatitis. But may show following signs due to paralytic ileus.

- ❖ Sentinel loop sign
- ❖ Colon cut off sign
- ❖ Renal halo sign

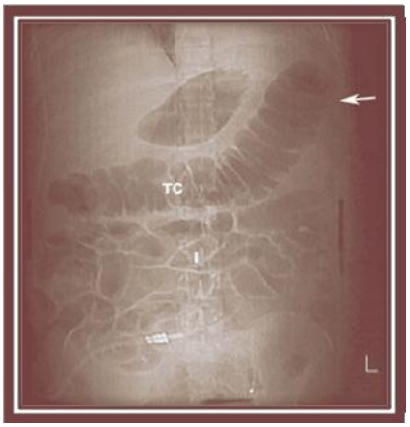


Figure No 9 - Colon cut off sign

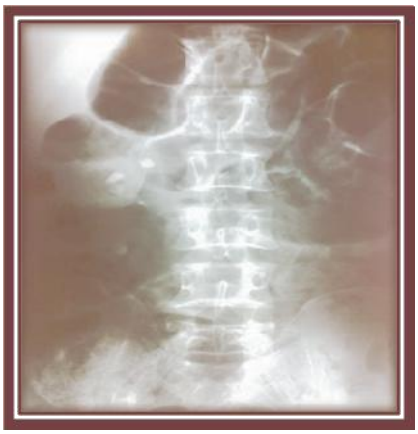


Figure No 10 - Sentinel loop sign

B. USG abdomen

Limited value during acute episode because of presence of intestinal gas shadows.

It can demonstrate

- ❖ Biliary stone
- ❖ Dilated pancreatic duct
- ❖ Bulky edematous pancreas

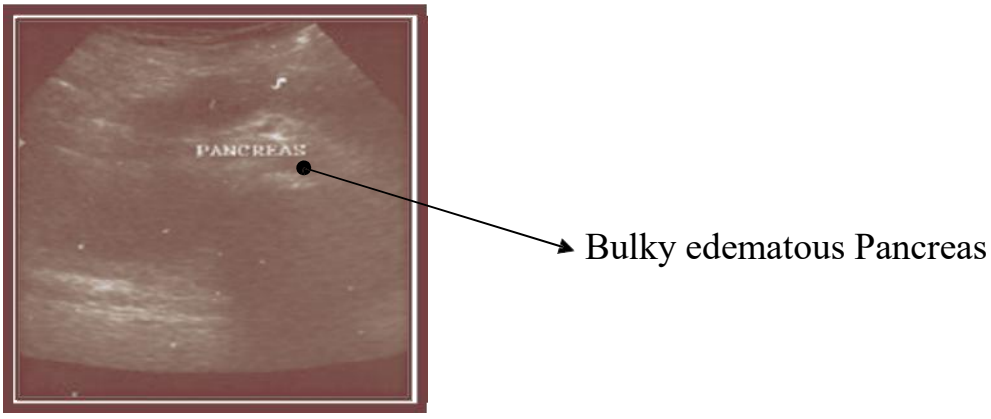


Figure No 11 - USG abdomen showing edematous pancreas

C. CECT abdomen:-

Investigation of choice for the diagnosis of acute pancreatitis as well as detection of complications.

Features suggestive of acute pancreatitis are

- ❖ Enlargement of pancreas
- ❖ Loss of peri pancreatic fat plane
- ❖ Areas of decreased density
- ❖ Localized fluid collection

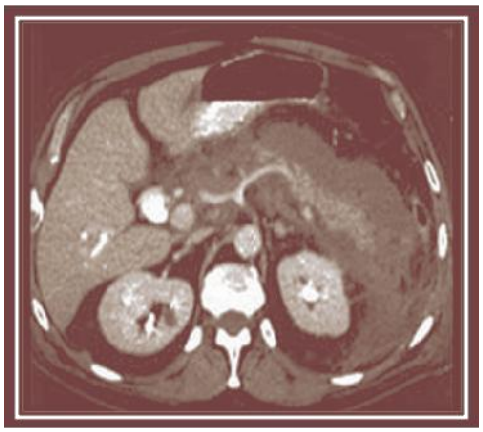


Figure No 12 - CECT abdomen showing edematous pancreas with peri pancreatic fat stranding

- ❖ Main value of CT is in detection of pancreatic necrosis.

Normally on CT abdomen, viable pancreas enhances by > 50 HU on IV administration of contrast material. Non viable pancreas does not show

such enhancement. Features suggestive of pancreatic necrosis on CECT abdomen are:

- ❖ Non enhancement of >30% parenchyma of pancreas.
- ❖ Area of > 3cm of pancreas that does not enhance.

Ideal timing of CECT abdomen in cases of acute pancreatitis is controversial because if it is done too early after diagnosis it may miss the necrosis. So most authors prefer to do it 48 hours after the diagnosis. Sensitivity of CECT abdomen to detect necrosis at 4 days is 100%[35].

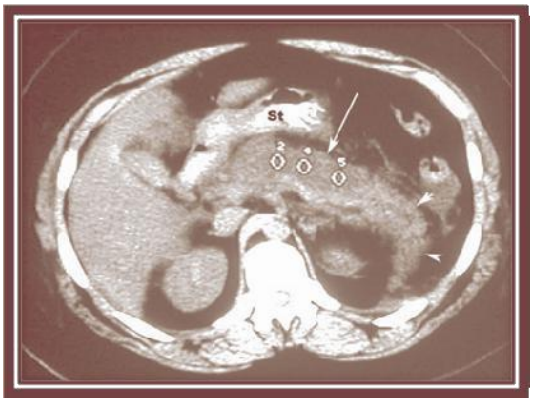


Figure No 13 - CECT abdomen showing non enhancing areas within pancreas suggestive of necrosis

D. ***MRI abdomen:***

- ❖ It is a reliable method of staging acute pancreatitis severity[36].
- ❖ Can predict prognosis of the disease, pancreatic duct disruption.
- ❖ No advantage over CT abdomen.
- ❖ Indications:
    - o Patients with renal dysfunction.
    - o Patients with allergy to contrast material.

Following table compares various imaging modalities used in acute pancreatitis:

Imaging technique	Effectiveness
Contrast-enhanced computed tomography	78 percent sensitivity and 86 percent specificity for severe acute pancreatitis
Endoscopic ultrasonography	100 percent sensitivity and 91 percent specificity for gallstones
Magnetic resonance cholangiopancreatography	81 to 100 percent sensitivity for detecting common bile duct stones
	98 percent negative predictive value and 94 percent positive predictive value for bile duct stones
	As accurate as contrast-enhanced computed tomography in predicating severity of pancreatitis and identifying pancreatic necrosis
Magnetic resonance imaging	83 percent sensitivity and 91 percent specificity of severe acute pancreatitis
Transabdominal ultrasonography	87 to 98 percent sensitivity for the detection of gallstones.

Table No 3 - Comparison of various imaging modalities for diagnosis of acute pancreatitis[37,38,39]

COMPLICATIONS

Acute pancreatitis can range in severity from the most benign self limiting conditions to the most severe cases which are associated with various systemic manifestations which may end up in death of the patient. Complications associated with acute pancreatitis can be classified into local / regional/ systemic.

Local	Fluid collections
	Pancreatic ascites/pleural effusion
	Pancreatic pseudocyst
	Pancreatic necrosis
	Infected pancreatic abscess
	Haemorrhage/pseudoaneurysm

Regional	Venous thrombosis
	Paralytic ileus
	Intestinal obstruction
	Intestinal ischemia/necrosis
	Cholestasis

Systemic	Systemic inflammatory response syndrome
	Multiple-organ-dysfunction syndrome
	ARDS/pulmonary failure
	Renal failure
	Cardiovascular complications
	Hypocalcemia
	Hyperglycemia
	Disseminated intravascular coagulopathy
	Protein calorie malnutrition
	Encephalopathy

Table No 4 - Complications of acute pancreatitis

ATLANTA symposium[40] held in1992 has given the following definitions for acute pancreatitis and its complications to maintain uniformity across the world.

Acute pancreatitis	An acute inflammatory process of the pancreas with variable involvement of other regional tissues or remote organ organ systems
	Associate with raised pancreatic enzyme levels in blood and / or urine
Severity	
Mild acute pancreatitis	Associated with minimal organ dysfunction and uneventful recovery; lacks the features of sever acute pancreatitis. Usually normal enhancement of pancreatic parenchyma on contrast-enhanced computed tomography
Severe acute pancreatitis	Associated with organ failure and / or local complications such as necrosis, abscess, or pseudocyst
Predicted severity	Ranson's Score>=3 or APACHE II Score >=8
Organ failure and systemic complications	
Shock	Systolic blood pressure <90mm hg
Pulmonary insufficiency	PaO_2<6mm Hg
Renal failure	Creatinine>=177μmol/L or <=2mg/dL after rehydration
Gastrointestinal bleeding	500ml in 24 hours
Disseminated intravascular coagulation	Platelets <=100,000/mm^3, fibrinogen<1.0g/L and fibrin-split products>80μg/L
Severe metabolic disturbances	Calcium <=1.87 mmol/L or <=7.5mg/dL
Local Complications	
Acute Fluid Collections	Occur early in the course of acute pancreatitis, are located in or near the pancreas and always lack a wall of granulation of fibrous tissue. In about half of patients, spontaneous regression occurs. In the other half, an acute fluid collection develops into a pancreatic abscess or pseudocyst
Pancreatic necrosis	Diffuse or focal areas(S) of non-viable

	pancreatic parenchyma, typically associated with peripancreatic fat necrosis. Non-enchanced pancreatic parenchyma>3cm or involving more than 30% of the area of the pancreas
Acute pseudocyst	Collection of pancreatic juice enclosed by a wall of fibrous or granulation tissue, which arises as a result of acute pancreatitis, pancreatic trauma or chronic pancreatitis, occurring at least 4 weeks after onset of symptoms, is round or ovoid an most often sterile; when pus is present lesion is termed a “pancreatic abscess”
Pancreatic abscess	Circumscribed, intra-abdominal collection of pus, usually in proximity to the pancreas, containing little or no pancreatic necrosis, which arises as consequence of acute pancreatitis or pancreatic trauma. Often 4 weeks or more after onset Pancreatic abscess and infected pancreatic necrosis differ in clinical expression and extent of associated necrosis.

Table No 5 - Atlanta definitions of acute pancreatitis and its complication

Prognostic scoring system

Treatment of acute pancreatitis is mainly based on the severity. So it is of prime importance to grade these patients into mild or severe. Hence a number of scoring systems have been proposed. As can be seen with their numbers none is considered to be the gold standard. Let us see the most important ones.

BISAP (Bedside Index for Severity in Acute Pancreatitis) Scoring System

BUN >25mg/dL
Impaired mental status (Glasgow Coma Scale Score<15)
SIRS
SIRS is defined as two or more of the following
1) Temperature of <36 or > 38^{o}C
2) Respiratory rate > 20 breaths/min or PaCO2 < 32 mm Hg
3) Pulse > 90 beats/min
4) WBC < 4,000 or > 12,000 cells/mm^{3} or > 10% immature bands
Age> 60 years
Pleural effusion detected on imaging
One point is assigned for each variable within 24 hours of presentation and added for a composite score of 0 -5

Table No 6 - BISAP scoring system[41]

- Total score of more than 2 indicates severity.

Ranson's Scoring System

- It is one of the most widely used scoring systems for acute pancreatitis.
- First proposed in 1974[42].

On admission:	After 48 hrs:
•Age - >55yrs	•Fall in hematocrit > 10%
•WBC - > 16000/l	•Fluid sequestration >6 L
•Blood glucose > 200 mg/dl	•Serum calcium < 8 mg/dl
•Serum LDH > 350 IU/L	•PaO2 < 60mmHg
•Serum AST >250 IU/L	•Increase in BUN > 5 mg/dl
	•Base deficit > 4mmol/L

Table No 7- Ranson's scoring system

- Total score of more than 3 indicates severity.
- Main disadvantage is that it is possible to assess the severity only after 48 hours.

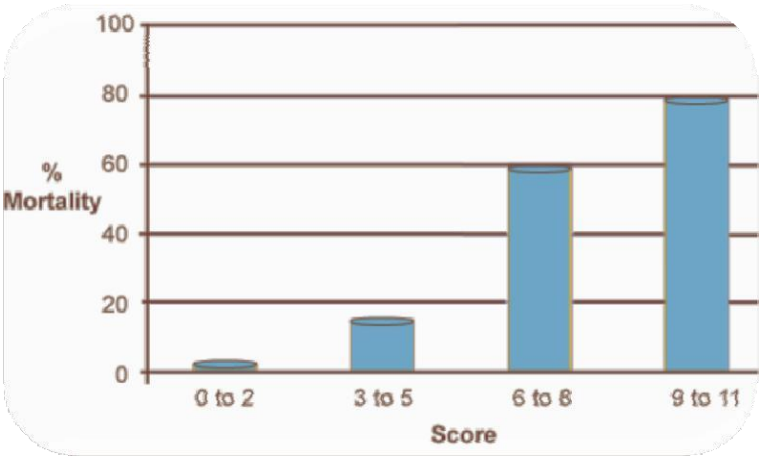


Figure No 14 - Prediction of mortality according to Ranson's score

Glasgow Scoring System

- It is a scoring system initially developed by Imrie in 1978[43].
- It initially included 7 criteria to be measured within 48 hours of admission.
- Later it was modified in 1984 to as mentioned below.

On Admission	
Age	>55 years old
WBC Count	15*10^9 cell/L
Blood glucose level	>10mmol/L
Serum urea level	16mmol/L
PaO2	8kPa (60mmHg)

Within 48 hours	
Serum calcium	< 2mmol/L
Serum albumin	<32g/L
LDH	>600IU/L
AST / ALT	>600IU/L

Table No 8 - Imrie [Modified Glasgow] scoring system

- Total score of more than or equal to 3 indicates severity.

APACHE II Scoring System

- It means Acute Physiology And Chronic Health Evaluation[44].
- It is a physiological scoring system based on 14 criteria.
- Total score of more than 8 indicates severity.
- Advantages over other systems
    - Severity can be assessed within 24 hours unlike others where 48 hours are required.
    - Severity can be assessed continuously through out the clinical course of the disease.
    - Prognosis can also be assessed after interventions like debridement.

Disadvantages:

- Cumbersome
- Not specific for pancreatitis

Modifications:

- APACHE 3[45] - Here 5 additional criteria are taken into account to increase the accuracy.
- APACHE O - Here clinical assessment of obesity is also taken into account.

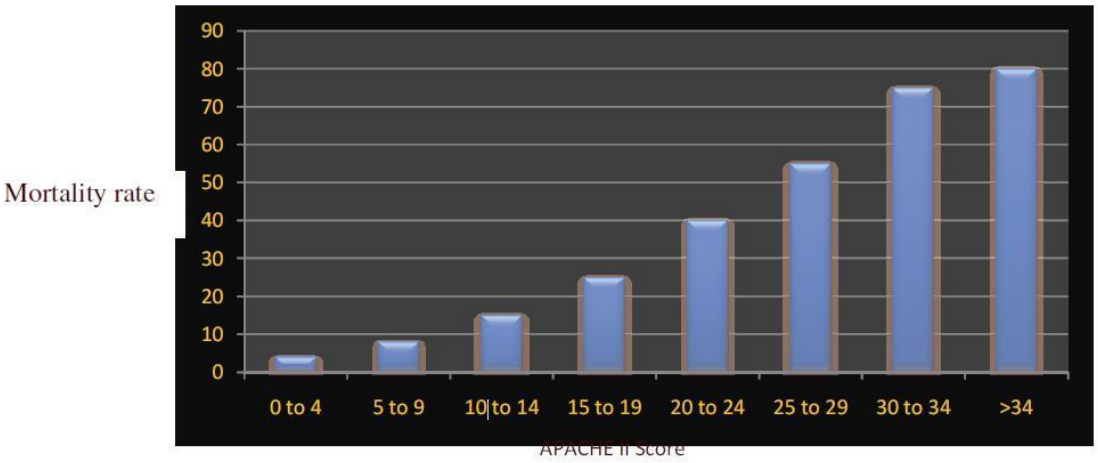


Figure No 15 - Mortality rate according to APACHE II scoring system

Physiologic Variable	High Abnormal Range					Low Abnormal Range				
	+4	+3	+2	+1	0	+1	+2	+3	+4	Points
Temperature - rectal (°C)	≥41°	39 to 40.9°		38.5 to 38.9°	36 to 38.4°	34 to 35.9°	32 to 33.9°	30 to 31.9°	≤29.9°	
Mean Arterial Pressure - mm Hg	≥160	130 to 159	110 to 129		70 to 109		50 to 69		≤49	
Heart Rate (ventricular response)	≥180	140 to 179	110 to 139		70 to 109		55 to 69	40 to 54	≤39	
Respiratory Rate (non-ventilated or ventilated)	≥50	35 to 49		25 to 34	12 to 24	10 to 11	6 to 9		≤5	
Oxygenation: A-aDO2 or PaO2 (mm Hg) a. FIO2 ≥0.5 record A-aDO2 b. FIO2 <0.5 record PaO2	≥500	350 to 499	200 to 349		<200 PO2>70	 PO2 61 to 70		 PO2 55 to 60	 PO2<55	
Arterial pH (preferred) Serum HCO3 (venous mEq/l) (not preferred, but may use if no ABGs)	≥7.7 ≥52	7.6 to 7.69 41 to 51.9		7.5 to 7.59 32 to 40.9	7.33 to 7.49 22 to 31.9		7.25 to 7.32 18 to 21.9	7.15 to 7.24 15 to 17.9	<7.15 <15	
Serum Sodium (mEq/l)	≥180	160 to 179	155 to 159	150 to 154	130 to 149		120 to 129	111 to 119	≤110	
Serum Potassium (mEq/l)	≥7	6 to 6.9		5.5 to 5.9	3.5 to 5.4	3 to 3.4	2.5 to 2.9		<2.5	
Serum Creatinine (mg/dl) Double point score for acute renal failure	≥3.5	2 to 3.4	1.5 to 1.9		0.6 to 1.4		<0.6			
Hematocrit (%)	≥60		50 to 59.9	46 to 49.9	30 to 45.9		20 to 29.9		<20	
White Blood Count (total/mm3) (in 1000s)	≥40		20 to 39.9	15 to 19.9	3 to 14.9		1 to 2.9		<1	
Glasgow Coma Score (GCS) Score = 15 minus actual GCS										
A. Total Acute Physiology Score (sum of 12 above points)										
B. Age points (years) ≤44=0; 45 to 54=2; 55 to 64=3; 65 to 74=5; ≥75=6										
C. Chronic Health Points (see below)										
Total APACHE II Score (add together the points from A+B+C)										

Table No 9 - APACHE II scoring system

Atlanta Criteria for Severity of Acute Pancreatitis

Severity Criteria	Definition
Organ failure with one or more	
Shock	Systolic blood pressure < 90mmHg
Pulmonary insufficiency	PaO2 < 60 mmHg
Renal failure	Serum Creatinine level > 2mg/dL after rehydration
Gastrointestinal tract bleeding	500 mL in 24 hours
Local Complications	
Pancreatic Necrosis	More than 30% of the parenchyma or more than 3cm
Pseudocyst	Collection of pancreatic juice enclosed by a wall
Abscess	Circumscribed collection of pus containing little or no pancreatic necrosis
Ranson's Score	>3
APACHE II Score	>8

Table No 10 - Atlanta criteria for assessing severity of acute pancreatitis

Balthazar CT Severity Scoring System

In contrast to the previous scoring systems, this is a radiological scoring system based on CECT abdomen.

- First developed by Balthazar et al in 1985[46], considered to be more accurate than the clinical scoring system.

Grade	CT Findings
A	Normal
B	Focal of diffuse enlargement of the pancreas, including irregularities of contour and inhomogeneous attenuation
C	Pancreatic gland abnormalities in grade B plus per pancreatic inflammation
D	Grade C plus a single fluid collection
E	Grade C plus 2 or more fluid collection and / or the presence of gas inor adjacent to the pancreas

Table No 11 - Balthazar CT severity scoring system

Grades beyond "C" indicate severity.

CT Grade	Assigned Score	Percent necrosis	Assigned Score
A	0	None	0
B	1	<30	2
C	2	30-50	4
D	3	>50	6
E	4		

Table No 12 - CT severity Index

Score greater than 5 indicates severity.

Other prognostic factors:

These are used sometimes to assess the severity though their role in routine clinical practice is not yet clear.

1. C Reactive Protein
2. IL 6
3. IL 8
4. Soluble IL 2 receptor
5. TNF Alfa
6. Trypsinogen Activating Peptide
7. Serum procalcitonin
8. Polymorphonuclear elastase

MANAGEMENT

Management of acute pancreatitis is quite complicated and is associated with a lot of controversies in every aspect.

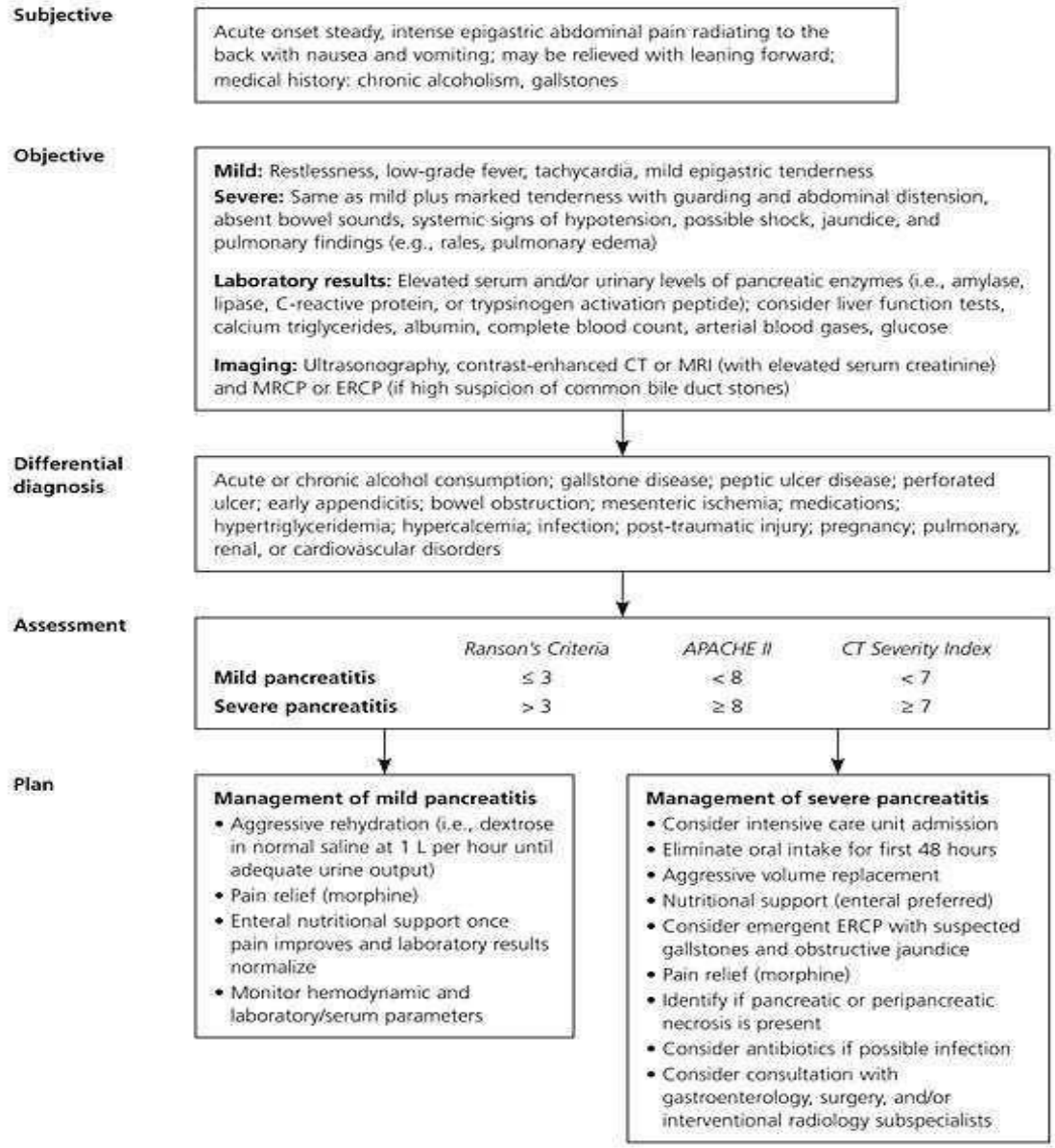


Figure No 16 - Management summary of acute pancreatitis

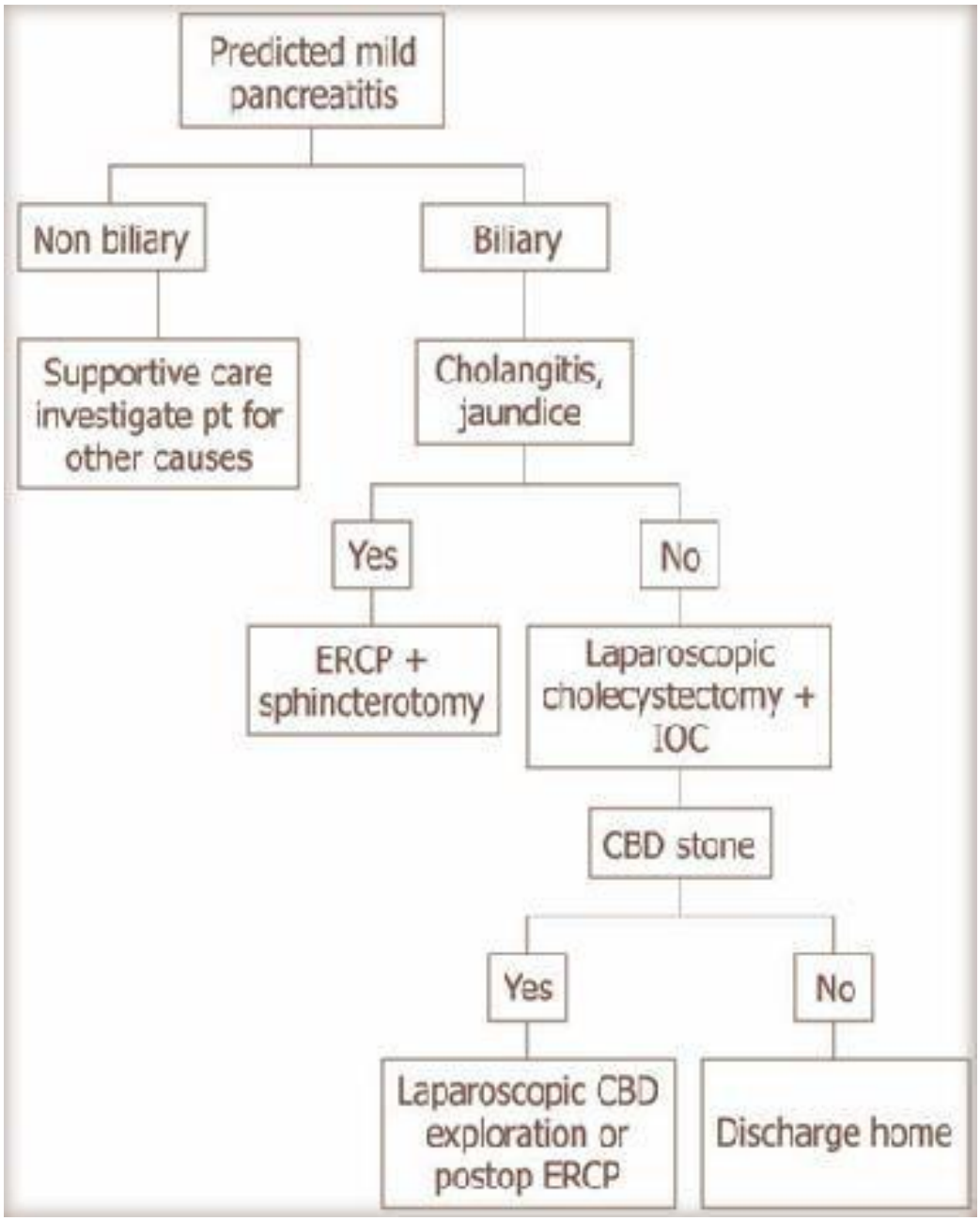


Figure No 17 - Algorithm for management of mild and severe acute pancreatitis

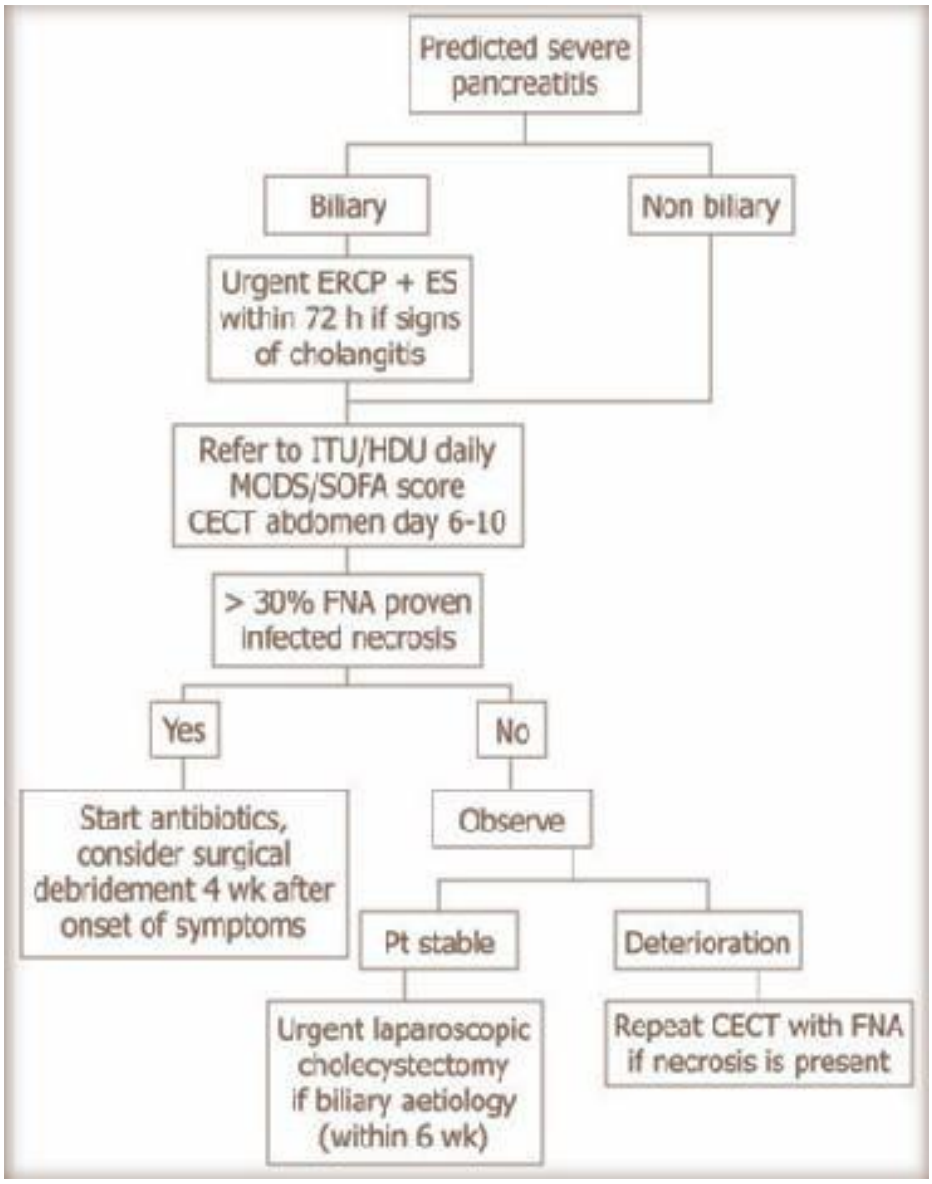


Figure No 17 - Algorithm for management of mild and severe acute pancreatitis

Treatment of acute pancreatitis involves 3 main components.

- ❖ Initial management of the acute episode
- ❖ Surgical management
- ❖ Management ofcomplications

Management of acute episode:

I. Fluid management

- ❖ Most important initial step.
- ❖ Fluid loss
    - o External – repeated vomiting / inadequate intake secondary to nausea
    - o Internal – fluid sequestration into areas of inflammation/ pulmonary parenchyma / soft tissues of the body
- ❖ Fluid loss leads to hypovolemia, hemoconcentration and in severe cases results in development of renal failure.
- ❖ Fluid resuscitation in the range of 200 ml/hr may be required.
- ❖ Close monitoring of cardiovascular and renal functions is a must.
- ❖ Studies have shown that improper fluid sequestration can increase the development of necrosis[47].

II. Electrolyte management

Patients with acute pancreatitis may develop various electrolyte

imbalances which require appropriate management. These include:

- ❖ Hypochloremic alkalosis secondary to repeated vomiting.
- ❖ Hypoalbumenemia
- ❖ Hypocalcemia
- ❖ Hypomagnesemia

III. Pain management

- ❖ Pain is considered to be the worst pain suffered by most individuals.
- ❖ Patient controlled analgesia is preferred.
- ❖ In most cases use of narcotics is required for adequate pain control.
- ❖ Mepiridine and analogues are the preferred medications.
- ❖ Morphine needs to be avoided because at least theoretically it aggravates biliary spasm.

IV. Role of Nasogastric tube

Previously Nasogastric aspiration was done routinely in all patients with the assumption that it decreases the pancreatic stimulation. There are no studies to support this concept. So placement of Nasogastric tube should be individualized. It is indicated in those with

- ❖ Severe vomiting to prevent aspiration pneumonia
- ❖ Severe retching to prevent Mallory weiss tears
- ❖ Paralytic ileus

V. Nutritional support:

- Classical teaching – avoid enteral nutrition based on the concept that enteral feeding leads to pancreatic stimulation and further aggravates pancreatic injury.
- Recent studies have shown that enteral feeding is feasible, safe and even desirable[48].
- Advantages of enteral feeding over TPN
  - Supports mucosal integrity and hence decreases septic complications
  - Easier
  - Lower complication rate
  - Lower cost
- Studies have shown that enteral feeding leads to
  - Lower APACHE scores and CRP levels.[49]
  - Lower septic and total complications.

To summarize, TPN is recommended in cases of severe acute pancreatitis with paralytic ileus. Otherwise enteral feeding is recommended.

VI. Role of prophylactic antibiotics

- Role of antibiotics is considered controversial.
- Basis for the use of antibiotics.

Sepsis is the leading cause of death among patients with severe pancreatitis. Incidence of local infections increases with increase in the

extent of necrosis and duration of acute pancreatitis. Incidence of local sepsis at 1 week is 29% and at 3 week is 71%[50]. Organisms commonly implicated are E. coli, Klebsiella, streptococcus, Staphylococcus and pseudomonas.

- Disadvantages of routine use of antibiotics
  - Development of multidrug resistant organisms
  - Fungal super infection
- Current evidence shows that use of prophylactic broad spectrum antibiotics in patients with severe pancreatitis is associated with decreased mortality[51].
- Current recommendation:
  - Mild cases – no need for antibiotics
  - Severe cases – antibiotics are recommended.
- Duration of use of antibiotic: 1-4 weeks. Most authors recommend it for 2 weeks[52].
- Commonly used regimens are Imipenem alone or Imipenem in combination with Cilastatin and cefuroxime. Some have recommended use of anti fungals like Fluconazole[53].

Surgical Management

"A 10-minute surgical discussion of acute pancreatitis should include 9 minutes of silence!!" -- Dictum followed in late 19th century.

In the modern day practice things have changed, thanks to better understanding of the natural history of the disease, basic pathophysiology of pancreatitis and better anaesthetic facilities.

Indications for surgical intervention in Necrotizing Pancreatitis
1. Diagnostic uncertainty
2. Intra-abdominal catastrophe unrelated to necrotizing pancreatitis
3. Infected necrosis documented by FNA or extraluminal gas on CT
4. Severe sterile necrosis
5. Symptomatic organized pancreatic necrosis

Table No 13 - Indications for surgery in case of acute pancreatitis

Surgical approach to the treatment of pancreatic necrosis	
Open surgery approaches	**Minimally invasive approaches**
Pancreatic resection	Minimally invasive approaches
Necrosectomy + wide tube drainage	Laparoscopic necrosectomy
Necrosectomy + relaparotomy (Staged reexploration)	Laparoscopic assisted percutaneous drainage
Necrosectomy + laparotomy ±open packing	Laparoscopic transgastric necrosectomy

Necrosectomy + drainage + closed continuous lavage	Percutaneous necrosectomy and sinus tract endoscopy
	MRI- radiologically assisted necrosectomy

Table No 14 - Surgical options for pancreatic necrosis

The basic and universal aspect of necrosectomy is débridement of necrotic peripancreatic and pancreatic tissue. As Necrosis is an ongoing process, it has been addressed in a variety of ways, as follows:

- Closed packing
- Open drainage
- Closed high-volume lavage of the lesser sac
- Repeated, planned necrosectomy with abdominal wall closure

Necrosectomy and Closed Packing

The lesser sac is accessed through the base of the mesocolon. A thorough, blunt necrosectomy is followed by packing with multiple, stuffed Penrose drains; however, it is associated with a high incidence of intra-abdominal abscess.

Necrosectomy and Open Drainage

An initial blunt necrosectomy is followed by marsupialization of the lesser sac by suturing the omentum to the abdominal wall fascia. Daily

unpacking and gentle irrigation are done.

Necrosectomy and Closed Lavage

It combines operative débridement (of only necrotic tissue to minimize loss of pancreatic parenchyma) with subsequent high-volume lavage of the lesser sac using a peritoneal dialysate.

Planned, Repeated Necrosectomy and Delayed, Primary Abdominal Wall Closure

The initial celiotomy and necrosectomy is followed by a planned, repeated necrosectomy every 48 hours. The lesser sac is entered through the gastrocolic ligament, and all areas of necrosis are unroofed and bluntly débrided. Multiple soft drains are placed in both paracolic gutters and the lesser sac. Subsequently, a nonadherent elastic drape or Adaptic gauze is used to line the lesser sac, which is then packed with moist laparotomy pads. Packing keeps the lesser sac open and readily accessible for repeated necrosectomy. Temporary abdominal closure is obtained with a zipper. Planned reoperation is scheduled at approximately 48-hour intervals until the necrotic processes have been controlled or resolved.

They include laparoscopic necrosectomy, laparoscopic assisted percutaneous drainage, laparoscopic transgastric necrosectomy, percutaneous necrosectomy and sinus tract endoscopy, MRI– radiologically assisted necrosectomy and Video- assisted retroperitoneal debridement.

4. MATERIAL AND METHODS

Source of Data

Patients admitted to Surgical wards in Madurai Medical College Hospital, Madurai.

Method of Collection of Data

A time bound prospective study was conducted on patients admitted with acute pancreatitis during the study period from December 2015 to December 2017. All the patients were subjected to detailed clinical examination, laboratory investigations and radiological imaging.

Inclusion Criteria

Patients with confirmed diagnosis of acute pancreatitis based on clinical / laboratory / radiological investigations.

Exclusion Criteria

- Age less than 16 years; as physiological thresholds are calibrated for adults.
- Patients with acute on chronic pancreatitis.

Sample Size

After considering both inclusion and exclusion criteria, total number of patients included in the study were 100.

All the 100 patients were subjected to both Ranson's and APACHE II scoring systems. Scoring was done on admission/time of diagnosis and at 48 hours. The scores were compared with the clinical severity which was graded according to Atlanta criteria and also compared with the clinical outcome.

Methods of Statistical Analysis

Independent t test was used to examine differences in age; fisher's exact test for sex; and chi square test for etiology were used. Sensitivity, specificity, positive predictor value, negative predictor value and accuracy were calculated. A "p" value of less than 0.05 was considered to be statistically significant. Data analysis was performed using SPSS software.

5. OBSERVATION AND RESULTS

The study was conducted in Madurai Medical College Hospital.

Total number of patients studied were 100.

According to the Atlanta Criteria, 62 patients were classified as Mild Acute Pancreatitis and 38 patients were classified as Severe Acute Pancreatitis.

Sex Distribution of the Study Population

Sex	Mild	Severe	Total
Male	56	36	92
Female	6	2	8

Table No 16 – Sex Distribution

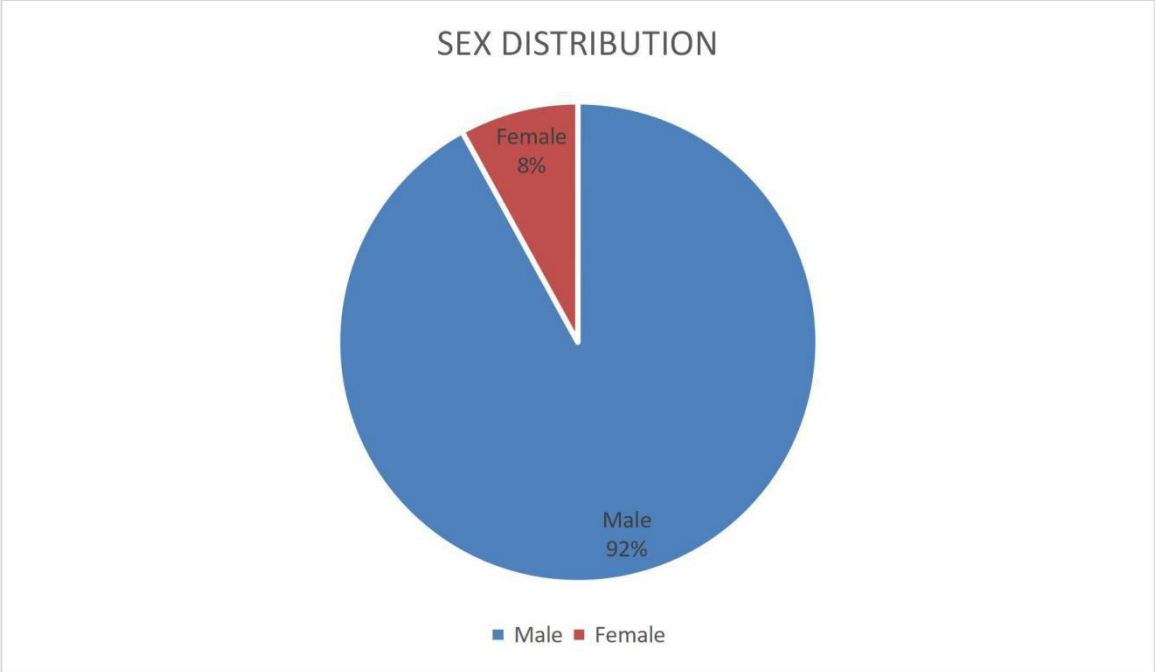


Figure No 19 – Sex Distribution

Of the 100 patients, 92 were Male (92.5 %) and 8 were Female (7.5%). There was no statistical significance of Sex (p=0.545) on the severity of the disease.

Etiology of Acute Pancreatitis

Etiology	Mild	Serve	Male	Female	Total
Alcohol	65	23	88	0	88
Gall stones	6	2	0	8	8
Idiopathic	2	2	4	0	4

Table No 17 – Etiology

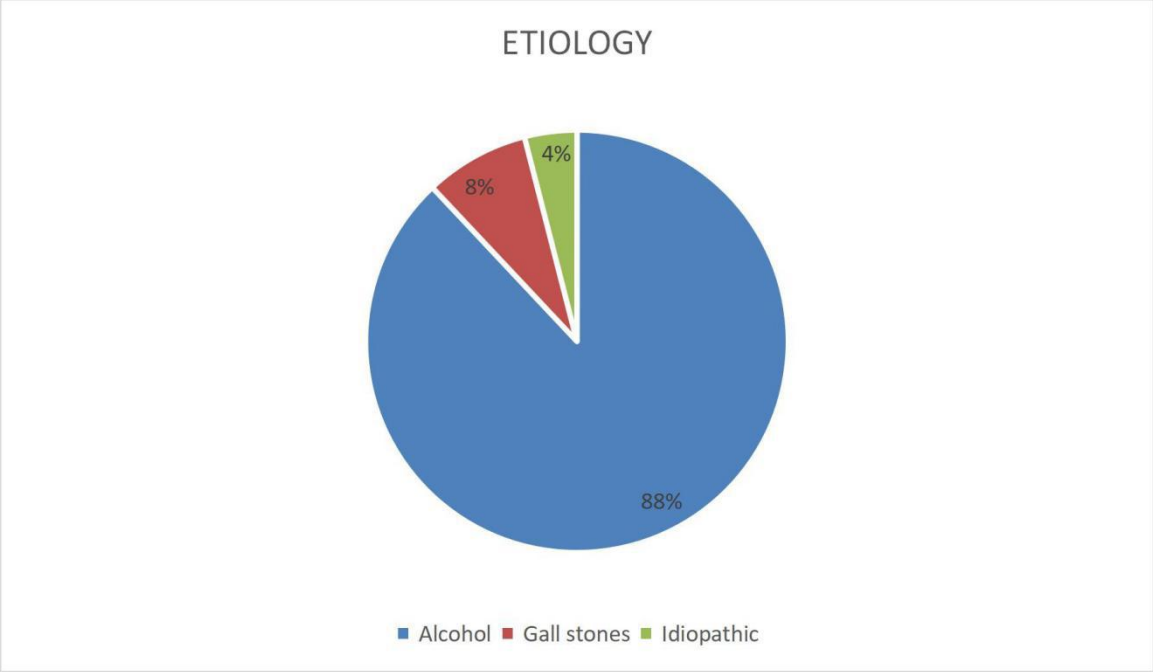


Figure No 20 – Etiology

Out of 100 patients, 30 (74%) had Alcohol induced Acute Pancreatitis, 3 (8%) had Gall Stones induced Acute Pancreatitis and 7 (18%) had Idiopathic Acute Pancreatitis. There was no statistical significance of Etiology (p=0.943) on the severity of the disease.

Outcome of Patients

No of patients without complicated	No of Patients with complicated	complicated			
		Local complications			System complications
		Pseudo cyst	Pancreatic necrosis	Hemorrhagic pancreatitis	SIRS
60	40	16	15	6	3

Table No 18 – Outcome of Patients

- Out of 100 patients
- 60% had uncomplicated outcome
- 40% of patients with any complication
- 6.4% of patients developed pseudo cyst
- 6% of patients developed Pan – Neurosis
- 3& hgic paneer

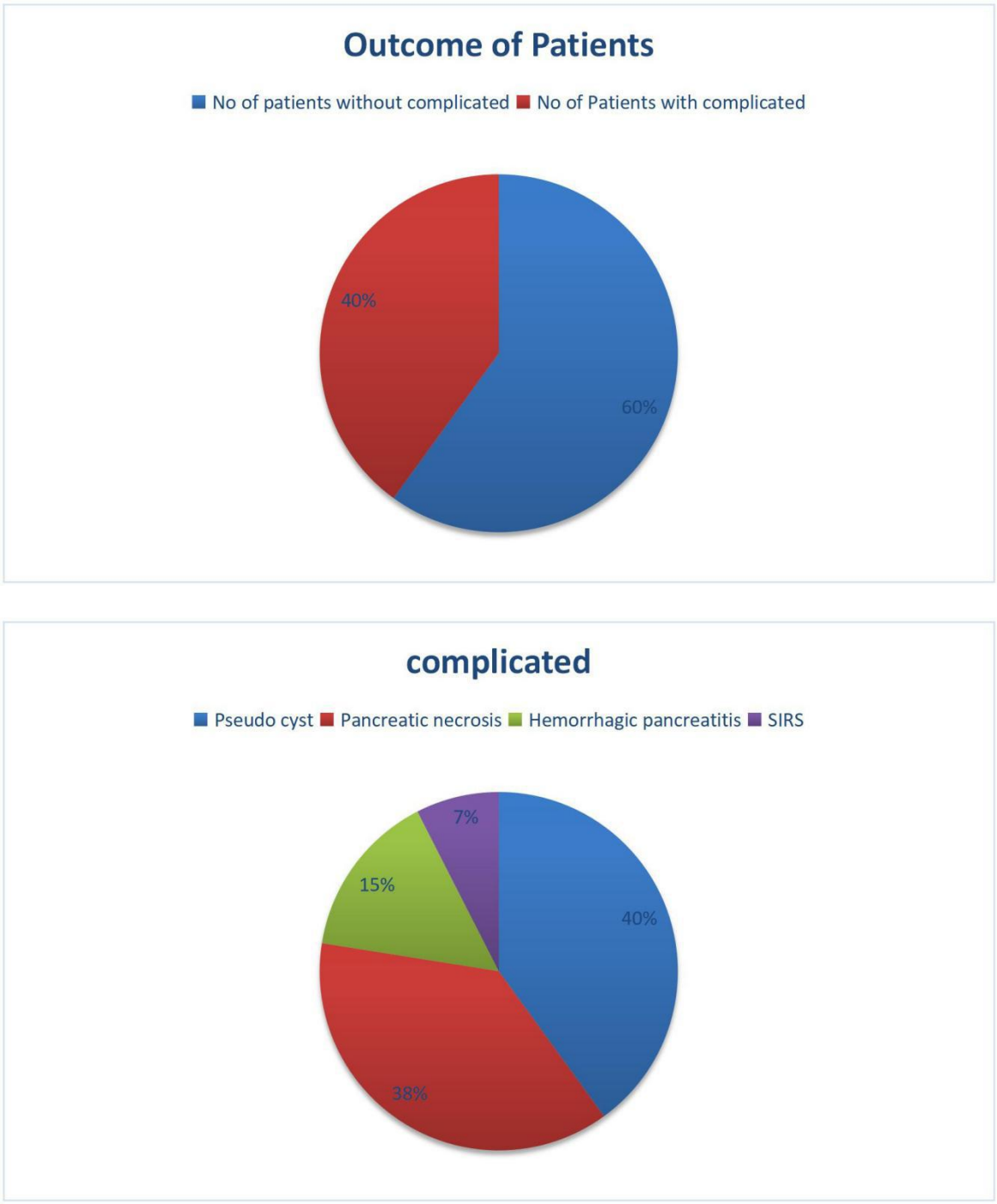


Figure No 21 – Outcome of Patients

Out of 100 patients with acute pancreatitis, 25 patients (62.5 %) had an uncomplicated outcome.

15 patients (37.5 %) developed complications, of which 14 patients (93.4 %) developed local complications and 1 patient (6.6 %) developed systemic complication. Of the local complications, 6 patients developed Pseudo Cyst, 6 patients developed pancreatic necrosis, and 2 developed hemorrhagic pancreatitis. The patient who developed systemic complication (SIRS) had a fatal outcome.

Surgical intervention was performed in one patient. Exploratory Laparotomy with necrosectomy was done and the patient eventually recovered.

Outcome of patients based on different cut-off Ranson's Score

Ransons score	Uncomplicated outcome	Complicated outcome			
		Local complications			Syst complications
		Pseudo cyst	Pancreatic necrosis	Hemorrhagic pancreatitis	SRS
<=3	39	3	0	0	0
>3	25	10	15	5	0
>5	0	0	0	0	3

Table No 19 – Outcome for different Ranson's Score

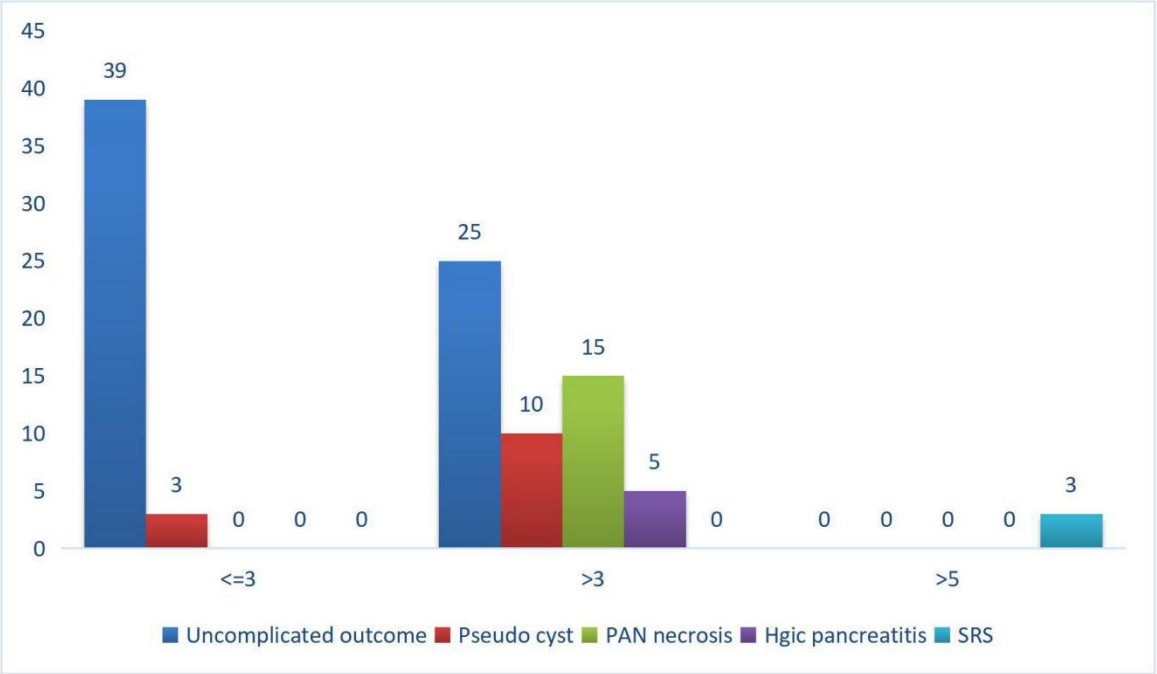


Figure No: 23 – Outcome for different Ranson's Score

Out of 42 patients <+ 3

32.85% are uncomplicated

7.14 % are complicated

Out of 55 patients > 3

45.45% Are uncomplicated

18.18% are complicated – pseudo cyst

27.27% are complicated – Pan necrosis

9.09% are complicated – Hgic pancreatitis

Of the 25 patients (62.5 %) who had Ranson's score of less than or equal to 3, 24 (96 %) had an uncomplicated outcome and one (4 %) developed

Pseudo Cyst. No patient in this group had Pancreatic Necrosis or any major organ failure. There were no deaths in this group.

15 patients (37.5 %) had Ranson's score of more than 3, one (6.6 %) of them had an uncomplicated course and 14 patients (93.4 %) developed complications, 13 had local complication and one had systemic complication.

One patient (2.5 %) had Ranson's score more than 5 and developed systemic complication (SIRS) and had fatal outcome.

Of the 25 Patients with Ranson's Score < = 3, 96 % had an uncomplicated mild course. The inference being Ranson's Score < = 3 predicts an uncomplicated outcome – mild acute pancreatitis.

Of the 15 Patients with Ranson's Score > 3, 93.4 % developed complications. The inference being Ranson's score > 3 predicts a complicated outcome -- severe acute pancreatitis.

Outcome of patients based on different cut-off APACHE II

Apache II Score	Uncomplicated outcome	Complicated outcome			
		Local complications			Syst complications
		Pseudo Cyst	PAN Necrosis	Hemorrhagic Pancreatitis	SIRS
<=8	57	3	0	0	0
>8	4	6	9	2	0
>12	1	6	6	3	3

Table No 20 – Outcome for different APACHE II Score

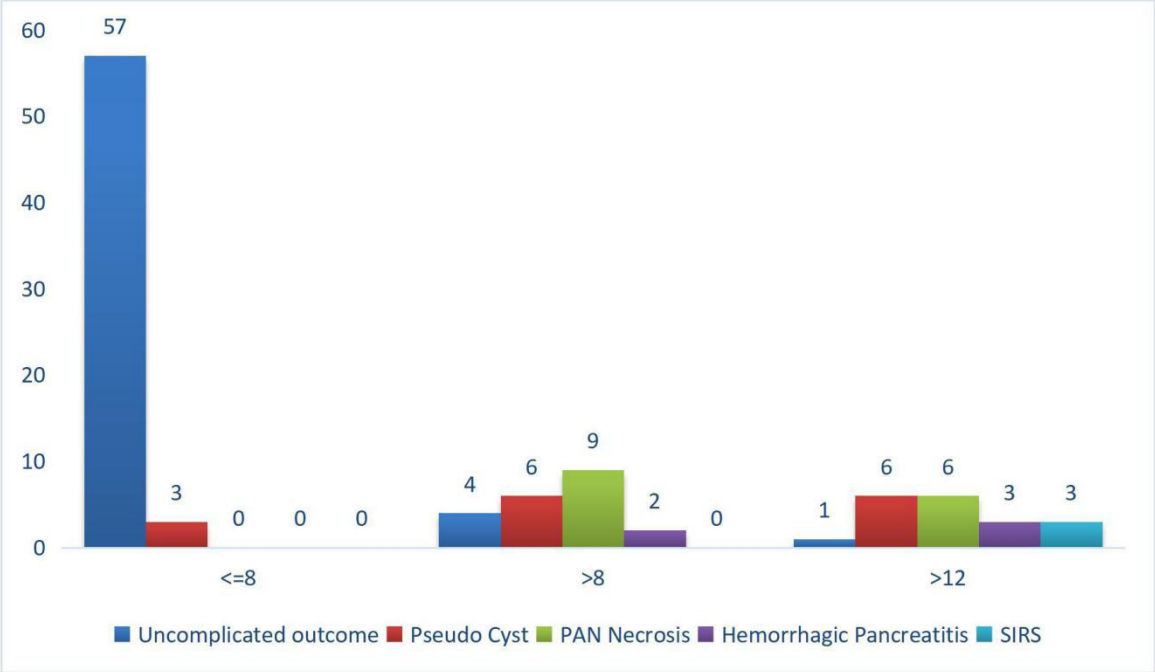


Figure No 24 – Outcome for different Apace II Score

Apache II score <8Uncomplicated outcome were 57%. Local complications: pseudo cyst were 5.26%.

Apache II score >8Uncomplicated outcome were 4%. Local complications: pseudo cyst were 35.29%, pancreatitis necrosis were 55.97%, Hemorrhagic pancreatitis were 11.17%.

Apache II score >12 Uncomplicated outcome were 1%. Local complications: pseudo cyst were 33.3%, pancreatitis necrosis were 33.3%, Hemorrhagic pancreatitis were 16.6%. and SIRS were 16.6%.

Of the 25 patients (62.5 %) who had APACHE II score less than or equal to 8, 24 patients (96 %) had an uncomplicated outcome. One patient (4 %) developed Pseudo Cyst. No patient in this group had Necrosis or major organ failure or death.

15 patients (37.5 %) had APACHE II score more than 8, one (6.6 %) of them had an uncomplicated course and 14 patients (93.4 %) developed complications, 13 developed local complications and one developed systemic complication. Of the 7 patients who had APACHE II score more than 12, all 7 patients (100 %) developed complications.

Of the 25 patients who had APACHE II score < = 8, 96 % had an uncomplicated outcome. The inference being APACHE II score < = 8 predicts an uncomplicated outcome -- mild acute pancreatitis.

Of the 15 patients with APACHE II score > 8, 93.4 % developed complications. APACHE II score > 8 predicts a complicated outcome -- severe acute pancreatitis.

Mean of Ranson's and APACHE II Score

Ranson's	Mean
Mild	2.40
Severe	4.53
Over All	3.20

Table No 21 – Mean Ranson's Score

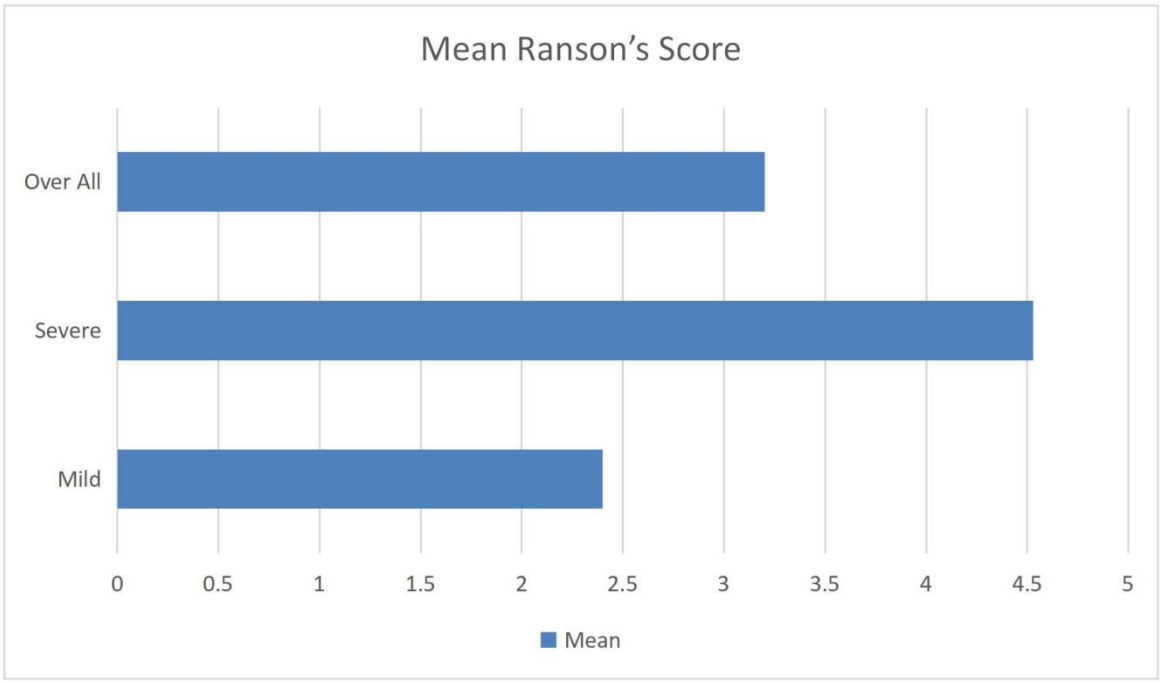


Figure No 25 – Mean Ranson's Score

APACHE II	Mean
Mild	5.28
Severe	12.27
Over All	7.90

Table No 22 – Mean APACHE II Score

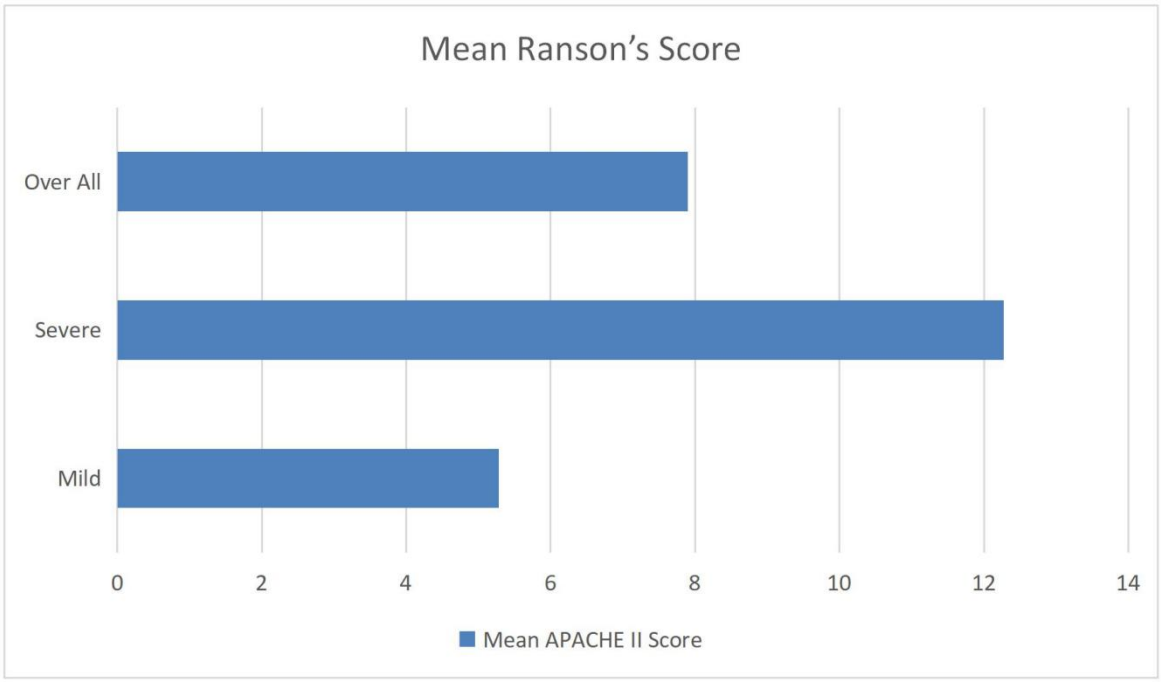


Figure No 26 – Mean APACHE II Score

Ranson's Score and APACHE II Score in severe acute pancreatitis were significantly higher than those in the mild cases ($p < 0.001$).

Prediction of severity by Ranson's Score

Table No 23 –

Ranson's Score	Sensitivity	Specificity	PPV	NPV	Accuracy
>=3	100	56	57.69	100	72.5
>=4	93.33	96	93.33	96	95
>=5	53.33	100	100	78.1	82.5

Prediction of severity by Ranson's Score

Ranson's score of greater than or equal to 4 predicted 93% of severe attacks and 96% of mild attacks with a PPV of 93.33 and NPV of 96 and accuracy of 95.

Ranson's score of greater than or equal to three predicted more number of severe attacks (100%) but less number of mild attacks (56%) with PPV of 57.69 and NPV of 100 and accuracy of 72.5.

Ranson's score of greater than or equal to 5 predicted less number of severe attack (53%) and branded more severe attacks as mild attacks.

Ranson's score of greater than or equal to 4 had the best sensitivity, specificity and accuracy.

Prediction of severity by APACHE II Score

Apache II Score	Sensitivity	Specificity	PPV	NPV	Accuracy
>=8	100	80	75	100	35
>=9	93.33	96	93.33	96	95
>=10	86.66	100	100	92.6	95
>=11	80	100	100	89.2	92.5

Table No 24 – Prediction of severity by APACHE II Score

APACHE II score of greater than or equal to 9 predicted 93.33% of severe attacks and 96% of mild attacks with a PPV of 93.33 and NPV of 96 and accuracy of

95. APACHE II score of greater than or equal to 10 also had the same accuracy.

APACHE II score of greater than or equal to 8 predicted more number of severe attacks (100%) but less number of mild attacks (80%) with PPV of 75 and NPV of 100.

APACHE II score of greater than or equal to 11 predicted less number of severe cases and labelled more number of severe cases as mild .

APACHE II score of more than or equal to 9 had the best sensitivity, specificity and accuracy.

Prediction of Major Organ failure and Pancreatic collection by Ranson's Score

Ranson's Score	Sensitivity	Specificity	PPV	NPV	Accuracy
Pancreatic Collection	93.33	96	93.33	96	95
Major Organ Failure	100	64.1	6.66	100	65

Table No 25 – Prediction of organ failure & pancreatic collection by Ranson's Score

The Ranson's scores were very sensitive for prediction of systemic complications (100%) but less sensitive for prediction of local complications(93.33).

Prediction of Major Organ failure and Pancreatic collection by APACHE II Score

APACHE II Score	Sensitivity	Specificity	PPV	NPV	Accuracy
Pancreatic Collection	93.33	96	93.33	96	95
Major Organ Failure	100	64.1	6.66	100	65

Table No 26 – Prediction of organ failure & pancreatic collection by APACHE II Score.

APACHE II scores showed higher sensitivity in the prediction of systemic complications(100%) than in the prediction of local complications(93.33%).

Prediction of Severity by the two scoring Systems

	Sensitivity	Specificity	PPV	NPV	Accuracy
Ranson's Score	93.33	96	93.33	96	95
APACHE II Score	93.33	96	93.33	96	65

Table No 27 – Prediction of severity by Ranson's and APACHE II scoring systems

As Sensitivity, Specificity, Positive Predictive Value, Negative Predictive Value and Accuracy are found to be the same for Ranson's and APACHE II scores, Ranson's scoring system is equally efficacious as APACHE II scoring system in the prognostication of acute pancreatitis.

Hospital Stay

The mean duration of hospital stay was 6.60 days for mild cases .

The mean duration of hospital stay was 9.31 days for severe cases.

The duration of hospital stay was not statistically significant.

6. DISCUSSION

Acute Pancreatitis is an increasing common abdominal emergency. Assessment of severity of acute pancreatitis is important for early identification of patients who may benefit from additional supportive and specific therapeutic procedures. Many different scoring systems have been devised for the assessment of severity of acute pancreatitis, which are divided into two types : The first type attempts to correlate laboratory and clinical markers specific to pancreatitis with subsequent outcome and disease severity, the most widely used in this group is Ranson's Score. The second type of scoring system is the application of non specific physiological scoring system, which was originally created for use in general population of critically ill patients like APACHE II scores.

Ideal predicting criteria should be simple, non-invasive, accurate and quantitative; and the assessment tests should be readily available at the time of diagnosis.

In this study we compare the classical and simple Ranson's scoring system with the more cumbersome APACHE II scoring system. We have classified the severity of acute pancreatitis in this study based on the Atlanta criteria.

In this study, acute pancreatitis was found 12 times more commonly in males than females and the mean age was 37.5 years. These results do not

match with the results of the study of Larvin et al where male is to female ratio was 47:53 and mean age was 62 years.

In the present study alcohol was the etiological factor in 74 % of patients and gall stones in 8 %, contrary to alcohol being 22 % and gall stones 43 % in Larvin et al. The etiology had no significant influence on the scores or the final outcome of acute pancreatitis, suggesting that once the pathogenic mechanisms have initiated the disease, the course and outcome of acute pancreatitis are not influenced by underlying etiological factors. Some authors have published similar results as in the study by Su Mi Woo et al[6].

Out of the 40 cases in this study, 25 patients (62.5 %) had mild acute pancreatitis and 15 patients (37.5 %) had severe acute pancreatitis. The percentage of severe cases was higher in our study as compared to most of the other studies. In the study by Larvin et al 20 % of all the cases were severe. Mortality in our study was 2. 5 % and mortality in the study by Larvin et al was 7.6 %. Mortality was less in our study.

In our study the mean Ranson's and APACHE II scores calculated during the first 48 hours showed significantly higher values for severe than for mild cases of acute pancreatitis. The mean Ranson's score in mild and severe cases was 2.40 and 4.53 respectively. The mean APACHE II score was 5.28 and 12.27 for mild and severe cases respectively.

Comparing outcomes in patient groups based on a range of Ranson's and APACHE II scores, it was observed that complications like Pseudo Cysts, Pancreatic Necrosis, major organ failure and deaths were more common when Ranson's score exceeded 3 and APACHE II scores exceeded 8. Contrary to expectation Pseudo Cyst was observed in one patient whose Ranson's and APACHE II scores were 3 and 8 respectively. These patients presented to hospital later than 48 hours after the onset of symptoms by which time the severity of the attack has subsided and the recorded scores were spuriously low. It can therefore be concluded that patients with Ranson's score more than 3 and APACHE II score of more than 8 are high risk patients.

In our study Ranson's score of greater than 3 and APACHE II score of greater than 8 had the highest sensitivity, specificity and accuracy for the prediction of severity of acute pancreatitis.

In our study the Ranson's and APACHE II scoring systems were very sensitive for the prediction of systemic complications (100%) but less sensitive for prediction of local complications (93.33%). This is comparable to the study by Larvin et al, where the sensitivity to detect systemic complications was higher (76%) than to detect local complications (73%).

In our study the sensitivity, specificity, positive predictor value, negative predictor value and accuracy of Ranson's and APACHE II scores are comparable.

	Sensitivity	Specificit	PPV	NPV	Accuracy
Ranson's	93.33	96	93.33	96	95
APACHE	93.33	96	93.33	96	95

Table No 28 – Accuracy of Ranson's and APACHE II scoring systems

As sensitivity, specificity and accuracy of Ranson's and APACHE II scores are comparable in our study, Ranson's is as powerful a prognostic scoring system as APACHE II.

Comparison of diagnostic performance of Ranson's and APACHE II Score with

Larvin et al[20] and Wilson et al[54]

	Ranson's Scoring System			APACHE II Scoring System		
	Present			Present		Wilson et
Sensitivity	93.33	75	87	93.33	71	68
Specificity	96	68	71	96	91	67
PPV	93.33	37	49	93.33	67	40
NPV	96	91	94	96	93	87
Accuracy	95	69	75	95	87	68

Table No 29 – Comparison of Ranson's and APACHE II scoring systems with Larvin & Wilson et al

The sensitivity, specificity, positive predictive value, negative predictive value, accuracy in the present study were higher than the studies by Larvin et al and Wilson et al and the correlation between Ranson's and APACHE II scores were also higher in the present study compared to the other studies.

Comparison of diagnostic performance of Ranson's and APACHE II Score with Su Mi Woo[6] et al and Constantinos Chatzicostas[14] et al.

	Ranson's Scoring System			APACHE II Scoring System		
	Present	Su Mi	Constantinos	Present	Su Mi	Constantinos
Sensitivi	93.33	89.50	82	93.33	78.9	58
Specifici	96	96	74	96	76	78
PPV	93.33	94.4	48	93.33	71.4	43
NPV	96	92.3	93	96	82.6	86
Accurac	95	93.2	76	95	77.3	73

Table No 30 – Comparison of Ranson's and APACHE II scoring systems with Su Mi Woo & Constantinos et al

The sensitivity, specificity, positive predictive value, negative predictive value, accuracy in the present study were higher than the studies by Su Mi Woo et al et al and Constantinos et al. In the study by Su Mi Woo et al and Constantinos et al the sensitivity and specificity of Ranson's were higher than that of the APACHE II scoring system. Whereas in the present study the sensitivity and specificity of Ranson's is the same as that of the APACHE II scoring system.

Comparing with the study by Arif A Khan et al[55] the accuracy of APACHE II scoring system in the study by Arif et al was 75 % and in the present study accuracy was 95 %.

Several theories may explain how the Ranson's score performed as good as the APACHE II scoring system. First, the Ranson's score has always been a specific predictor of outcome in patients with pancreatitis whereas the APACHE II score was developed to encompass a wide variety of disease processes. Secondly, we studied a relatively small population of patients in which the proportion of severe pancreatitis was quite high. A larger study from multiple centres might prove different results. Thirdly, the Ranson's scoring system performed well in the study as a significant number of cases were secondary to alcohol intake (Ranson's scoring system was derived using data from a predominantly alcoholic patient population).

The Ranson's scoring system is a simple scoring system wherein the laboratory tests required are simple, routine and readily available out of hours compared to the more cumbersome APACHE II scoring system, the only disadvantage being a 24 hour delay. According to our study, the Ranson's scoring system still accurately predicts the outcomes in patients with acute pancreatitis and it compares favourably with the physiological scoring systems in the prediction of disease severity for pancreatitis.

7. CONCLUSION

From this study, we can conclude Ranson's scoring system is not inferior to APACHE II scoring system in predicting the severity of acute pancreatitis. Ranson's scoring system is a simple, cheap, easy to remember, recollect, and calculate scoring system. Moreover, Ranson's scoring system was developed specifically for acute pancreatitis. In the developing world, where cost effectiveness of each test is important, Ranson's scoring system can be used in place of APACHE II scoring system. The Ranson's scoring system accurately predicts the outcome in patients with acute pancreatitis and compares favourably with the physiological scoring systems in the prediction of disease severity for acute pancreatitis, the only disadvantage being a 24 hour delay.

The Ranson's scoring system proved to be as powerful a prognostic model as the more complicated APACHE II scoring system even in the present era of advanced investigations.

8. SUMMARY

In the present study:

100 cases of acute pancreatitis were studied.

According to the Atlanta criteria, 62.5 % were mild acute pancreatitis and 37.5% were severe acute pancreatitis.

37.5 % of patients were in the age group of 31 to 40 years.

92.5 % of patients were male.

Alcohol in-take was the cause in 74 % of patients.

Common complications were pseudo cyst of pancreas and pancreatic necrosis.

Mean Ranson's score for mild and severe cases were 2.40 and 4.53 respectively;

Mean APACHE II score for mild and severe cases were 5.28 and 12.27 respectively.

Ranson's score of more than 3 and APACHE II score of more than 8 had the best accuracy for predicting severity of acute pancreatitis.

The Ranson's and APACHE II scores showed higher sensitivity in prediction of systemic complications than in the prediction of local complications.

2.5 % of patients were treated surgically.

Mean duration of hospital stay was 6.6 days for mild cases and 9.3 days for severe cases.

Over all mortality rate was 2.5 %.

Sensitivity, Specificity, Positive Predictor Value, Negative Predictor Value and Accuracy were 93.33, 96, 93.33, 96 and 95 respectively for both the Ranson's and APACHE II scoring systems.

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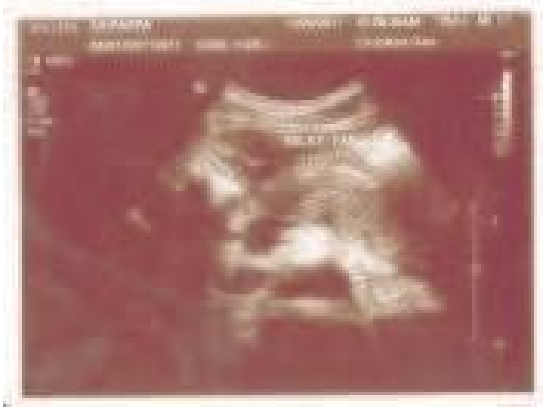
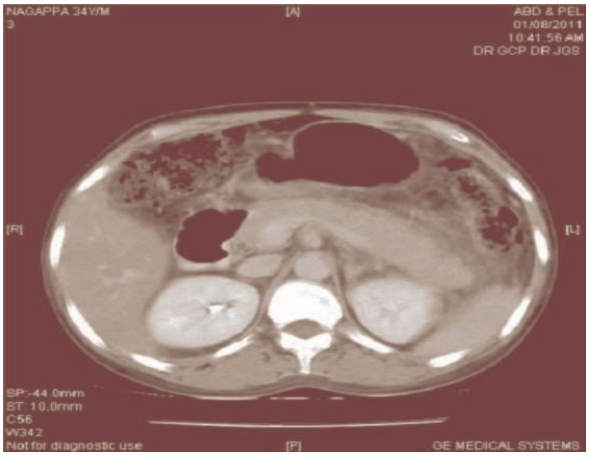
10. ANNEXURES

ANNEXURE 1 – PHOTOGRAPH



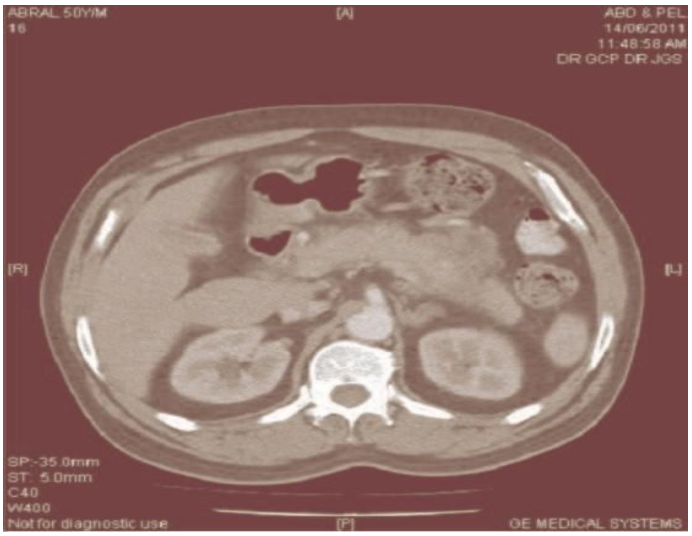
Ultrasound Abdomen of a patient showing diffuse bulky edematous pancreas

(Ranson's Score - 3, APAHCE II Score - 8)



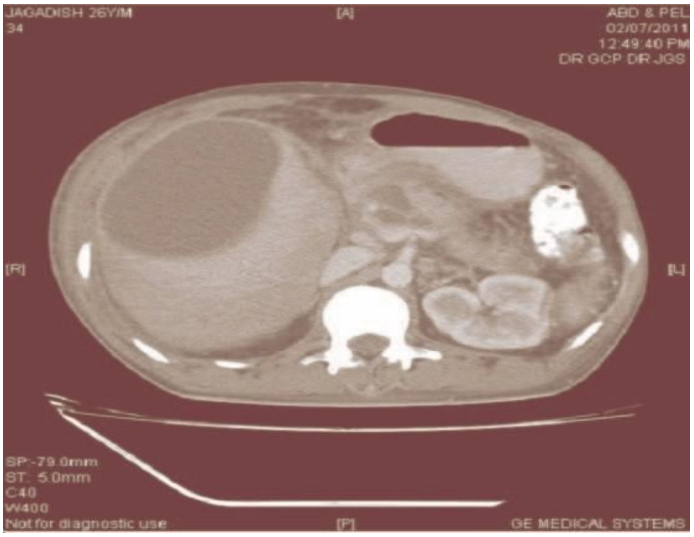
CECT abdomen showing diffuse enlargement of the pancreas with peripancreatic fat stranding

(Ranson's Score - 2, APAHCE II Score - 4)



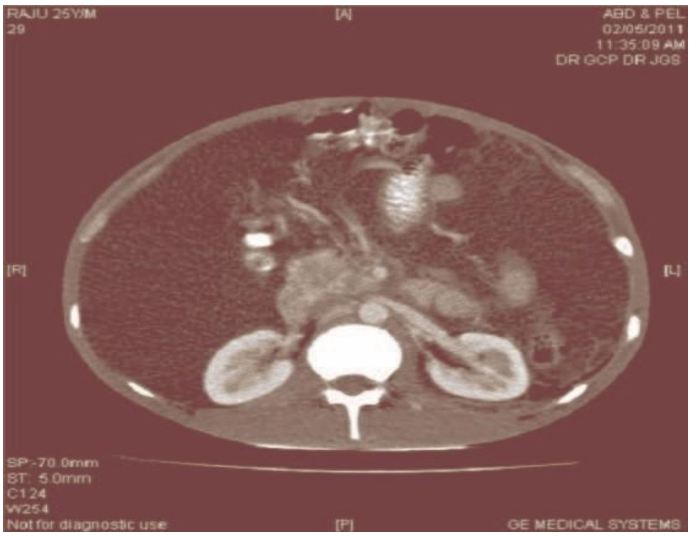
CECT abdomen showing diffuse enlargement of the pancreas

(Ranson's Score - 2, APAHCE II Score - 6)



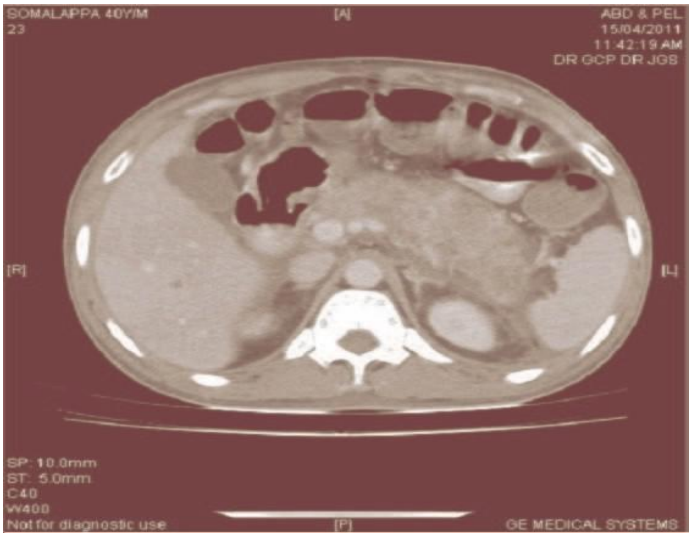
CECT abdomen showing Pseudo cyst of pancreas

(Ranson's Score - 3, APAHCE II Score - 8)



CECT abdomen showing Necrotizing pancreatitis

(Ranson's Score - 5, APAHCE II Score - 13)



CECT abdomen showing Necrotizing pancreatitis

(Ranson's Score - 5, APAHCE II Score - 14)

ANNEXURE 2 – PROFORMA

A. General Information

1. Name :

2. Age :

3. Sex :

4. I.P. Number :

5. Date of Admission :

B. Symptoms

1. Pain Abdomen :

2. Vomiting :

C. Examination

1. General

a. GCS : b. Pulse : c. BP : d. Respiratory Rate : e. Body Temperature : f. Signs of Dehydration :

2. Abdomen

a. Tenderness : b. Guarding : c. Epigastric Mass : d. Retroperitoneal Haemorrhage :

3. Respiratory System

a. Basal Atelectasis :

b. Pleural Effusion :

D. Investigations

1. Serum Amylase :

2. PCV

- On admission :
- At 48 hours :

3. TC :

4. RBS :

5. Blood Urea

- On admission :
- At 48 hours :

6. Serum Creatinine :

7. Serum Sodium :

8. Serum Potassium :

9. Serum Calcium :

10. SGOT :

11. LDH :

12. Fluid Sequestration :

13. Base Deficit :

14. PaO2 :

15. Arterial pH :

16. USG Abdomen :

17. CECT Abdomen :

E. Complication

1. Local

Pseudocyst :

Pancreatic Necrosis :

Pancreatic Abscess :

2. Regional

Venous Thrombosis :

Paralytic Ileus :

Cholestasis :

3. Systemic

Renal Failure :

ARDS :

SIRS / MODS :

Others :

F. Severity Score

- Atlanta Clinical Classification:
- Ranson's Score :
- APACHE II Score :

G. Prognosis

- Hospital Stay :
- Recovered / Expired

S.No	IP.No	Name	Age	Sex	Etiology	Treatment: Conservative (c) / Operative (o)	Duration of Hospital stay (Days)	Complications	Mortalilty	Atlanta Classification	Ranson's Score	APACHE II Score
1	13681	Aavathu muthu	62	M	Alcohol	C	13	Pancreatic Necrosis		Severe	5	10
2	17298	Ashok kumar	36	M	Alcohol	C	10	Pancreatic Necrosis		Severe	5	14
3	65890	Asoul Mamid	63	M	Alcohol	C	3			Mild	2	1
4	36281	Bala subramaniyan	58	M	Alcohol	C	10	Haemorrhagic Pancreatitis		Severe	4	11
5	76218	Balasundarm	46	M	Alcohol	C	14			Mild	2	6
6	23330	Balasundarm	36	M	Alcohol	C	7	Pseudo Cyst		Severe	4	14
7	13982	Chinadurai	42	M	Alcohol	C	6	Haemorrhagic Pancreatitis		Severe	5	14
8	65473	Esaki Konar	30	M	Alcohol	C	4	Pseudo Cyst		Severe	4	9
9	52259	EsakkiRaja	65	M	Alcohol	C	3			Mild	3	6
10	21298	Ganesh	33	M	Alcohol	C	6	Pseudo Cyst		Severe	5	11
11	73106	George Nariyan	37	M	Alcohol	C	10			Mild	2	4
12	76449	Iengo	29	M	Alcohol	C	4			Mild	1	3
13	22665	Jeyachandaran	45	M	Alcohol	C	6	Pseudo Cyst		Severe	5	11
14	19995	John	28	M	Alcohol	C	3			Mild	2	1
15	34866	Johns	45	M	Idiopathic	C	5			Mild	3	7
16	97855	Kabin	35	M	Alcohol	C	3			Mild	2	6
17	97860	Kabis	35	M	Alcohol	C	5			Mild	3	7
18	74340	Kalyana konar	30	M	Alcohol	C	2	Pancreatic Necrosis		Severe	5	12
19	56378	Kalyanakonar	30	M	Alcohol	C	4			Mild	2	8
20	74785	Kannan	42	M	Alcohol	C	24	Pseudo Cyst		Severe	3	8
21	500	Kannan	56	M	Alcohol	C	10			Mild	2	8
22	13987	Kannan	32	M	Alcohol	C	6	Haemorrhagic Pancreatitis		Severe	5	14
23	34365	Kannan	28	M	Idiopathic	C	11	Pancreatic Necrosis		Severe	4	12
24	76163	Karthik	22	M	Alcohol	C	5			Mild	2	4
25	8895	Karupasamy	54	M	Alcohol	C	3			Mild	3	6
26	27618	Kasi	60	F	Gallstones	C	10			Mild	4	9
27	68332	Krishnakumar	24	M	Alcohol	C	7			Mild	3	4

28	19056	Lakshmi	38	F	Gallstones	C	14	pseudo cyst		Severe	4	14
29	5570	Lakshmi	28	F	Gallstones	C	3			Mild	3	6
30	12559	Maharajan	58	M	Alcohol	C	2			Mild	3	5
31	56374	Maheswari	33	F	Gallstones	C	10	Haemorrhagic Pancreatitis		Severe	4	11
32	43851	Manisamy	42	M	Alcohol	C	3			Mild	2	1
33	31500	Mariammal	36	F	Gallstones	C	2			Mild	3	5
34	3432	Mariappan	36	M	Idiopathic	C	3			Mild	2	6
35	48260	Muniyandi	53	M	Alcohol	C	11	Pancreatic Necrosis		Severe	4	11
36	48280	Muniyandi	53	M	Alcohol	C	4			Mild	1	3
37	17891	Muniyandi	42	M	Alcohol	C	4	Pseudo cyst		Severe	5	14
38	55215	Murugan	58	M	Alcohol	C	6	Haemorrhagic Pancreatitis		Severe	5	14
39	68563	Murugan	55	M	Alcohol	C	6	Pseudo Cyst		Severe	5	11
40	34228	Muthu kumar	31	M	Alcohol	C	2	Pancreatic Necrosis		Severe	5	12
41	22543	Muthukrishnan	55	M	Alcohol	C	6			Mild	2	6
42	20193	Muthukumar	53	M	Alcohol	C	24	Pancreatic Necrosis		Severe	5	13
43	19100	Muthulakshmi	23	F	Gallstones	C	8			Mild	1	3
44	63862	Muthusamy	60	M	Alcohol	C	4			Mild	1	3
45	12987	Mydeen	29	M	Alcohol	C	4	Pseudo Cyst		Severe	4	9
46	5750	Narayana perumal	58	M	Alcohol	C	13			Mild	2	4
47	70778	Naruoin sroorth	31	M	Alcohol	C	10			Mild	4	9
48	56193	Pakiyasamy	60	M	Alcohol	C	13	Pancreatic Necrosis		Severe	5	10
49	64181	Palaniselvam	34	M	Alcohol	C	2			Mild	3	5
50	31237	Paniarasan	33	M	Idiopathic	C	10	Pancreatic Necrosis		Severe	5	14
51	64120	Pannuraj	40	M	Alcohol	C	5			Mild	3	6
52	82672	Paramasivam	31	M	Alcohol	C	4	Pseudo cyst		Severe	5	14
53	128/27	Parvathy	80	F	Gallstones	C	7			Mild	3	8
54	5224	Paulraj	30	M	Alcohol	C	4			Mild	2	8
55	26387	Peratchi	57	M	Alcohol	C	10			Mild	2	4
56	26785	petchiammal	30	F	Gallstones	C	6			Mild	2	6
57	47892	Pillai	46	M	Alcohol	C	6			Mild	2	1
58	5568	RajaPandi	28	M	Alcohol	C	7	Pseudo Cyst		Severe	4	14
59	74340	RajaPandi	40	M	Alcohol	C	3			Mild	2	4
60	13642	Rajesdran	29	M	Alcohol	C	5	SIRS	Expired	Severe	6	17
61	23419	Rajsus	37	M	Alcohol	C	5	SIRS	Expired	Severe	6	17
62	34524	Rama moorthy	43	M	Alcohol	C	24	Pseudo Cyst		Severe	3	8

63	17030	Ramaiah	46	M	Alcohol	C	4			Mild	3	8
64	11980	Ramaiya	37	M	Alcohol	C	11	Pancreatic Necrosis		Severe	4	12
65	18905	Ramar	70	M	Alcohol	C	5			Mild	2	4
66	48264	Ramasamy	69	M	Alcohol	C	13			Mild	2	4
67	56919	Ramesh	33	M	Alcohol	C	7	Pseudo Cyst		Severe	4	14
68	73370	Ramesh	21	M	Alcohol	C	8			Mild	1	3
69	5568	Ramesh	36	M	Alcohol	C	10			Mild	2	4
70	5210	Ramesh	35	M	Alcohol	C	10			Mild	2	8
71	2332	Ramesh	40	M	Alcohol	C	5			Mild	3	7
72	11900	Ramhya	37	M	Alcohol	C	10			Mild	4	9
73	15310	Ramsun	47	M	Alcohol	C	5			Mild	3	6
74	64856	Samsudeen	23	M	Alcohol	C	3			Mild	3	4
75	10302	Sangaralingam	20	M	Alcohol	C	3			Mild	3	7
76	109142	Sankaralingam	20	M	Alcohol	C	7			Mild	3	4
77	30989	santhnakumar	45	M	Alcohol	C	7			Mild	3	8
78	31389	santhnakumar	45	M	Alcohol	C	3			Mild	3	6
79	67413	Selva raj	57	M	Alcohol	C	6			Mild	2	6
80	39714	Selvin	27	M	Alcohol	C	3			Mild	3	7
81	7085	Shahul Hammed	22	M	Alcohol	C	10	Haemorrhagic Pancreatitis		Severe	4	11
82	30684	Shankar	47	M	Alcohol	C	24	Pancreatic Necrosis		Severe	5	13
83	77133	Shanmugam	39	M	Alcohol	C	2	Pancreatic Necrosis		Severe	5	12
84	5220	Shanmugam	47	M	Alcohol	C	3			Mild	3	6
85	12987	Sivanathan	41	M	Alcohol	C	4	Pseudo Cyst		Severe	4	9
86	2088	Solasivam	19	M	Alcohol	C	14	pseudo cyst		Severe	4	14
87	24691	Sornam	50	M	Alcohol	C	3			Mild	2	4
88	56374	Sorwan	62	M	Alcohol	C	3			Mild	3	7
89	66041	Sri Murugan	33	M	Alcohol	C	5	SIRS	Expired	Severe	6	17
90	32757	Sukumar	16	M	Alcohol	C	8			Mild	1	3
91	35393	Sundar	30	M	Alcohol	C	13			Mild	2	4
92	13705	Suriyapanidan	13	M	Alcohol	C	3			Mild	3	4
93	10686	Tamil selvan	55	M	Alcohol	C	24	Pancreatic Necrosis		Severe	5	13
94	563	Tausik	30	M	Alcohol	C	11	Pancreatic Necrosis		Severe	4	11
95	17500	Vandimalayan	35	M	Alcohol	C	24	Pseudo Cyst		Severe	3	8
96	12484	Vandimurugan	34	M	Alcohol	C	10	Pancreatic Necrosis		Severe	5	14
97	23707	Vandiralayan	36	M	Alcohol	C	7			Mild	3	8
98	311612	Vandiralayan	35	M	Alcohol	C	3			Mild	3	6

99	3435	Veeramuthu	33	M	Alcohol	C	6			Mild	2	1
100	74344	Yousf	52	M	Alcohol	C	4			Mild	3	8

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ETHICS COMMITTEE CERTIFICATE

Name of the Candidate	:	Dr.M.Vivek
Course	:	PG in MS., General Surgery
Period of Study	:	2015-2018
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	Comparative study of Ranson's versus apache II scoring systems in predicting clinical outcome in patients with acute pancreatitis
Ethical Committee as on	:	27.07.2017

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

Member Secretary

Chairman

Prof Dr V Nagaraajan
M.D., MNAMS, D.M., Dsc.,(Neuro), Dsc (Hon)
CHAIRMAN
IEC - Madurai Medical College
Madurai

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CERTIFICATE - II

This is to certify that this dissertation work titled COMPARATIVE STUDY OF RANSON'S VERSUS APACHE II SCORING SYSTEMS IN PREDICTING THE CLINICAL OUTCOME IN PATIENTS WITH ACUTE PANCREATITIS of the candidate **Dr . Vivek M**. with registration Number 221511123 for the award of MASTER DEGREE in the branch of GENERAL SURGERY. I have personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows ZERO percentage of plagiarism in the dissertation.

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