

**COMPARITIVE STUDY BETWEEN BISAP SCORE AND RANSON
SCORE IN PREDICTING SEVERITY OF ACUTE PANCREATITIS**



By

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In

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Under the guidance of

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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation "**COMPARITIVE STUDY
BETWEEN BISAP SCORE AND RANSON SCORE IN PREDICTING
SEVERITY OF ACUTE PANCREATITIS**" is a bonafide and genuine research work carried out by me under the guidance of PROF.P. RAGUMANI., M S., Professor, Institute of General Surgery, MMC & RGGGH, CHENNAI.

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is a bonafide

Research work done by Dr. R. ARUNKUMAR in partial

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ABSTRACT

Acute pancreatitis has widely variable clinical and systemic manifestations spanning the spectrum from a mild, self-limiting episode of epigastric pain to severe, life-threatening, multiorgan failure. Since the morbidity and mortality of Acute Pancreatitis differ markedly between mild and severe disease (mild < 5% vs severe 20–25%), it is very important to assess severity as early as possible. Various scoring systems like APACHE II scoring, RANSONS scoring and BISAP have been used to asses Severity in Acute Pancreatitidis. Among these BISAP and RANSONS scoring systems have been considered to be predictive and most widely used. The need of a scoring system with maximum accuracy and simplicity has been emphasized upon. BISAP has the advantage over Ranson score of being calculated within 24hrs of admission. Ranson score seems to perform accurate prediction of persistent organ failure. This study aims at evaluating the predictive value of BISAP scoring in comparison to RANSONS SCORE.

AIM AND OBJECTIVE

1. To assess the accuracy of BISAP scoring system vs RANSON scoring system in predicting Severity in an attack of acute pancreatitis.
2. To compare predictability of organ failure between BISAP scoring and RANSONS Scoring system

METHODOLOGY

In this study, 60 in-patients presenting with features of acute pancreatitis to Rajiv Gandhi Govt. General Hospital from November 2013 to September 2014 have been studied. It is a prospective and retro prospective study.

Keywords : acute pancreatitis, BISAP score, Ranson score

OBSERVATION AND RESULTS

- No. of patients in the study – 60
- Most common age of presentation is 4 th decade of life.
- Males are most commonly affected.
- Alcohol consumption is the most common etiology in our study.
- 38 patients had mild disease
- 22 patients had a complicated course
- 16 patients had moderately severe course
- 6 patients had severe course.
- Most common local complication is pseudocyst.
- Mortality rate in our study is 5 %
- Ranson's score of more than 3 and BISAP score of less than or equal to 3 had the best accuracy of predicting severity of acute pancreatitis.

- Both Ranson's score and BISAP score showed higher sensitivity in prediction of systemic complications than that of local complications.
- No patients were treated surgically.
- Sensitivity , specificity, positive predictive value, negative predictive value and accuracy were 93.33,96,93.33,96 and 95 respectively for both Ranson's score and BISAP scoring system.

CONCLUSION

From this study, we can conclude that BISAP scoring system is not inferior to Ranson's scoring system in predicting the severity of acute pancreatitis. BISAP scoring system is very simple, cheap, easy to remember and calculate. BISAP scoring system accurately predicts the outcome in patients with acute pancreatitis. Moreover the values in BISAP score are instantaneous and there is no time delay. Ranson's score takes a minimum of 24 hours.

Thus, BISAP score has proved to be a powerful tool in predicting the severity of acute pancreatitis in par with Ranson's score

ABBREVIATIONS

ABBREVIATION	EXPANSION
ICU	INTENSIVE CARE UNIT
MODS	MULTIORGAN DYSFUNCTION SYNDROME
SIRS	SYSTEMIC INFLAMMATORY RESPONSE SYNDROME
CRP	C-REACTIVE PROTEIN
ERCP	ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY
ARDS	ACUTE RESPIRATORY DISTRESS SYNDROME
APACHE	Acute physiology and chronic health evaluation
BISAP	BED SIDE INDEX FOR SEVERITY OF ACUTE PANCREATITIS
CSI	CT SEVERITY INDEX
TPN	TOTAL PARENTRAL NUTRITION

AP	ACUTE PANCREATITIS
LDH	LACTATE DEHYDROGENASE
AST	ASPARTATE TRANSAMINASE
BUN	BLOOD UREA NITROGEN
AUC	AREA UNDER CURVE

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Introduction

Acute pancreatitis is a common entity encountered during routine surgical practice and it poses a great challenge to the treating surgeon. “Acute pancreatitis is defined as a pancreatic inflammatory process, with peripancreatic and multi-organ involvement causing multi-organ dysfunction syndrome (MODS), with increased mortality rate”. Following statement grossly summarises its consequences.

“Acute pancreatitis is the most terrible of all calamities that occur in connection with the abdominal viscera. The suddenness of its onset, the illimitable agony that accompanies it, and the mortality attendant upon it , all render it the most formidable of catastrophes”

Lord Moynihan, 19251

Fitzgerald remarked about biliary pancreatitis

“Never in the medical history have so many owed to a single stone”

AIM AND OBJECTIVE OF THE STUDY

The present study was aimed at analysing sixty number of patients admitted in Rajiv Gandhi Government General Hospital, Chennai – 3 with a diagnosis of acute pancreatitis during September 2013 to august 2014 with the following objectives

1. To assess the accuracy of BISAP scoring system vs RANSONS scoring system in predicting Severity in an attack of acute pancreatitis.
2. To compare predictability of organ failure between BISAP scoring and RANSONS Scoring system

REVIEW OF LITERATURE

Thomas L. Bollen et al (2012, April) did comparative study of radiological and clinical scoring system in acute pancreatitis in 346 patients and found that CECT abdomen demonstrated the highest accuracy but this was not statistically significant. Hence he concluded that CT abdomen on admission for assessment of severity in acute pancreatitis is not recommended.

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

Fabre A et al (2012, Feb) studied 48 children with acute pancreatitis Ranson's , Glasgow, 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 the 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 indices, the score for extra pancreatic inflammation spread is superior.

“B U Wu et al7, using (CART) analysis, developed a clinical scoring system, for prediction of hospital mortality in acute pancreatitis. The BISAP scoring system was validated on data collected from 18,256 acute pancreatitis cases from 177 hospitals in 2004-2005. The accuracy of the BISAP score for mortality prediction was measured by the area under the receiver operating characteristic curve (AUC). The creditability of BISAP score was further validated by comparing with APACHE II score. BISAPscore is a accurate and simple method for the early identification of patients with acute pancreatitis who are at increased risk for in- hospital mortality”.

“Vikesh k. singh et al, BISAP score was analysed among 397 cases of acute pancreatitis admitted to their hospital. BISAP scores were calculated on all cases within 24h of presentation. The ability of the BISAP score to predict mortality was analysed. Of 397 cases, 14(3.5%) deaths were observed. With increasing BISAP score, mortality has been found to be increased (p<0.0001). Thus calculating BISAP score within 24h of presentation is an easiest way to identify patients at risk of increased mortality and to develop intermediate markers of severity”.

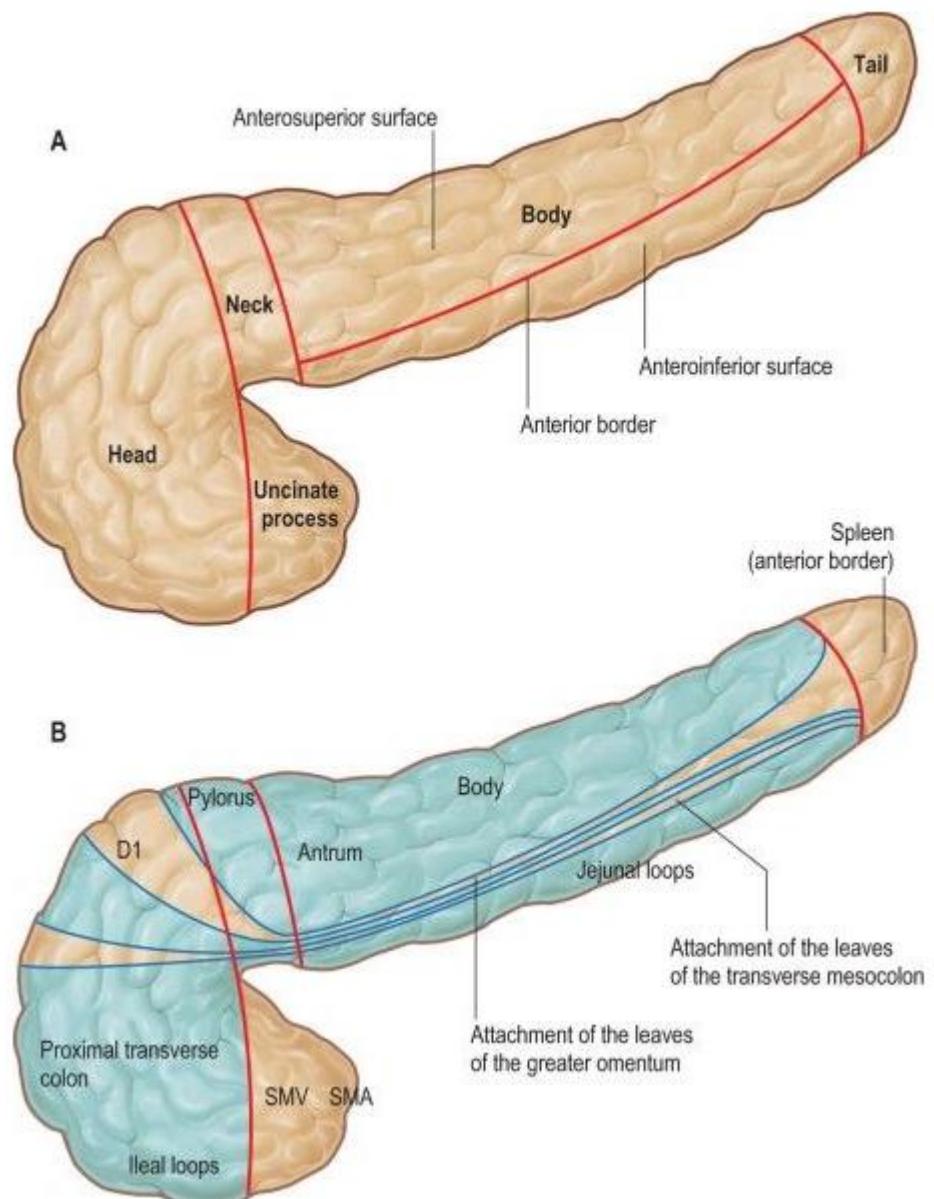
HISTORY

- Earliest account of acute pancreatitis comes from the fatal illness during the regime of Alexander the great.
- Reginald Fitz presented his 1 st landmark paper on acute pancreatitis in 1889.
- Opie 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 first studied hereditary pancreatitis in 1952.
- Strong association between pancreatitis and CFTR gene has been proposed in 1998.

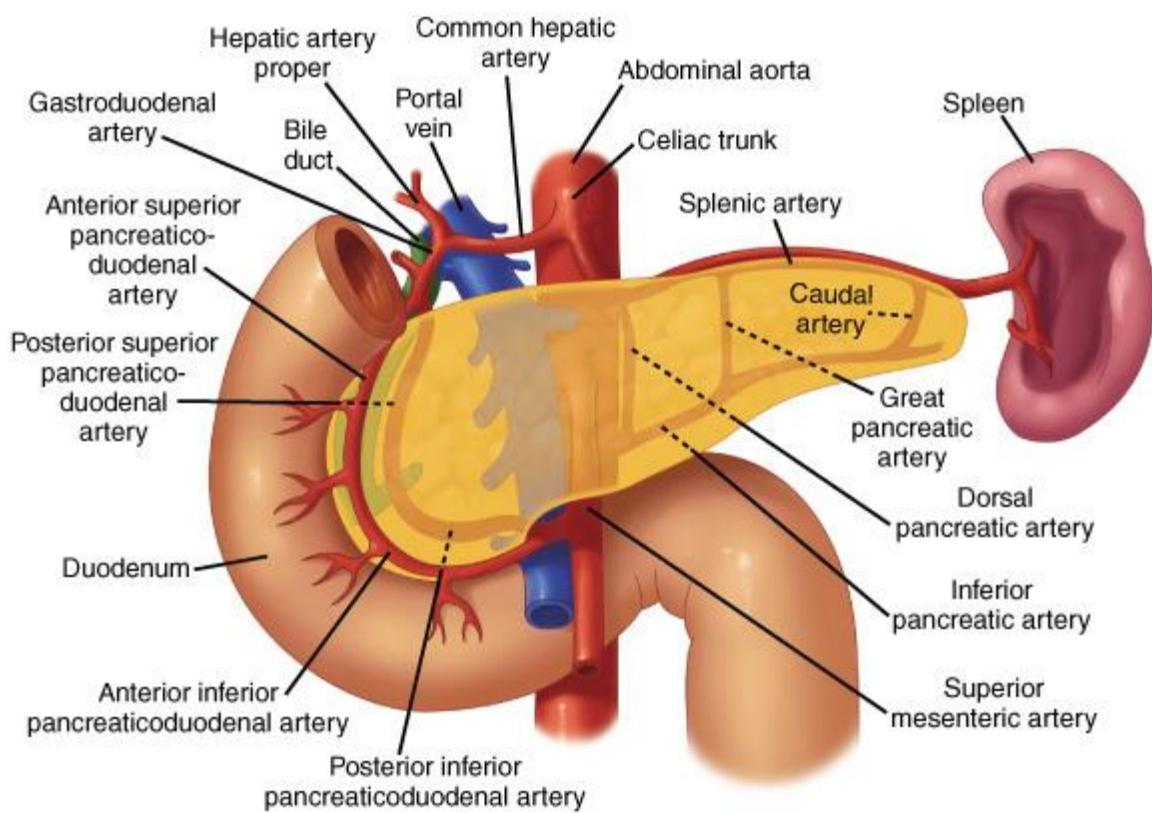
ANATOMY

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 IVC, aorta, splenic vein and left adrenal gland.

Blood supply of pancreas is from the celiac artery, and the superior mesenteric artery through splenic and pancreatico – duodenal arteries. Venous 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 from the vagus and splanchnic nerves.

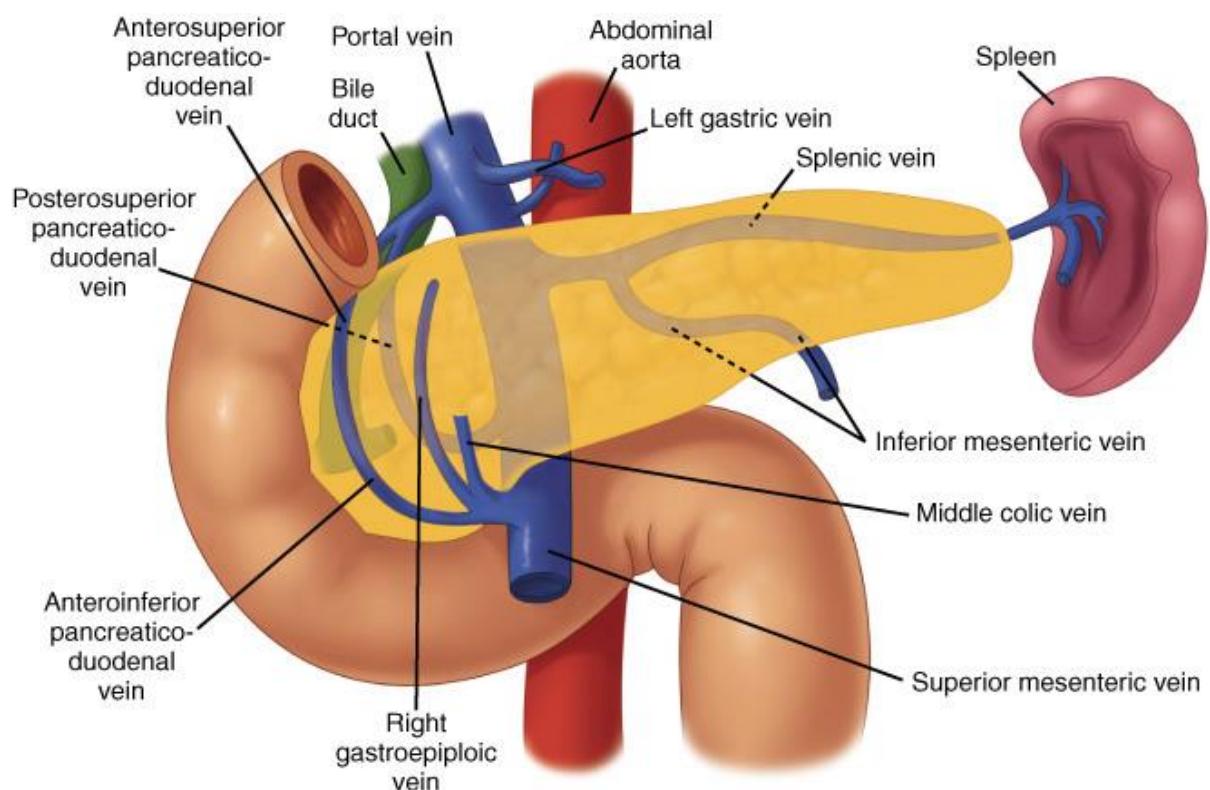


Relations of pancreas



Source: Brunicardi FC, Andersen DK, Billiar TR, Dunn DL, Hunter JG, Matthews JB, Pollock RE; *Schwartz's Principles of Surgery*, 9th Edition; <http://www.accessmedicine.com>
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Arterial supply of pancreas



Source: Brunicardi FC, Andersen DK, Billiar TR, Dunn DL, Hunter JG, Matthews JB,

Pollock RE; *Schwartz's Principles of Surgery, 9th Edition*; <http://www.accessmedicine.com>

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Venous drainage of pancreas

PHYSIOLOGY

Pancreas helps 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 and trypsinogen.

EXOCRINE FUNCTIONS	ENDOCRINE FUNCTIONS
Trypsin	Alpha cells - Glucagon
Chymotrypsin	Beta cells - Insulin
Elastase	Delta cells - Somatostatin
Carboxypeptidase A & B	F cells - Pancreatic polypeptide
Pancreatic lipase	
Amylase	
Colipase	
Phospholipase A2	
Ribonuclease	
Deoxyribonuclease	

ETIOLOGY AND CLASSIFICATION

Acute pancreatitis may be classified based on pathology, aetiology, disease severity, or the presence of necrosis. In approximately 10 - 20% of patients, no aetiology is identified. Some patients may have microlithiasis and/or “sphincter of Oddi dysfunction (SOD)”, as the aetiology of AP. With the increasing knowledge and understanding of the role of genetic abnormalities in hereditary and idiopathic chronic pancreatitis (CP), it is possible that these abnormalities will be implicated in idiopathic AP. Furthermore, polymorphisms in inflammatory mediators may influence disease severity.

Clinically, acute pancreatitis may be classified as mild or severe disease. Severe acute pancreatitis can cause organ failure; and/or local complications, such as - necrosis, abscess, or pseudocyst. Approximately 10 - 20% of patients, develop severe disease. [APACHE]), serum markers (e.g., interleukin [IL]-6, C-reactive protein, and trypsinogen activation peptide) and imaging modalities (contrast enhanced computed tomography [CT] scan) have been used to predict severity. Complicated courses are more common in SAP with mortality rates from 5 to 20%¹¹. In contrast, mild AP is the more frequent presentation and is associated with minimal or transient organ dysfunction and uneventful recovery.

The presence of pancreatic necrosis - is the single best predictor of outcome during AP. "Pancreatic necrosis is a diffuse or focal area of nonviable pancreatic parenchyma, typically associated with peripancreatic fat necrosis, which is observed as non enhanced pancreatic parenchyma on a contrast CT scan". The degree of necrosis can predict morbidity and mortality. Approximately 30% of patients with pancreatic necrosis, develop infection with a death rate of - 6 to 40% and a morbidity of - 80%.

ETIOLOGICAL FACTORS

A. Toxic	<ul style="list-style-type: none"> • Alcohol • Organo phosphorus
B. Metabolic	<ul style="list-style-type: none"> • Hyperlipidemia • Hypercalcemia • Venoms (spider, Scorpion)
C. Mechanical	<ul style="list-style-type: none"> • Cholelithiasis • Congenital malformations <ul style="list-style-type: none"> - Pancreatic divisum - Annular pancreas <p>Anatomical variants</p> <ul style="list-style-type: none"> - Duodenal duplication - Duodenal diverticulum

	<ul style="list-style-type: none"> - Choledochal cyst - Ampullary dysfunction <p>Trauma</p>
D. Infections	<ul style="list-style-type: none"> • Virus – Mumps, coxsackie A, HIV, CMV • Bacteria - Mycobacterium tuberculosis • Parasites – ascaris • Others - mycoplasma
E. Drugs	<ul style="list-style-type: none"> • Furosemide • Thiazide • 6 mercaptopurine • Azathioprine

SPECIFIC ETIOLOGIES

GALLSTONES

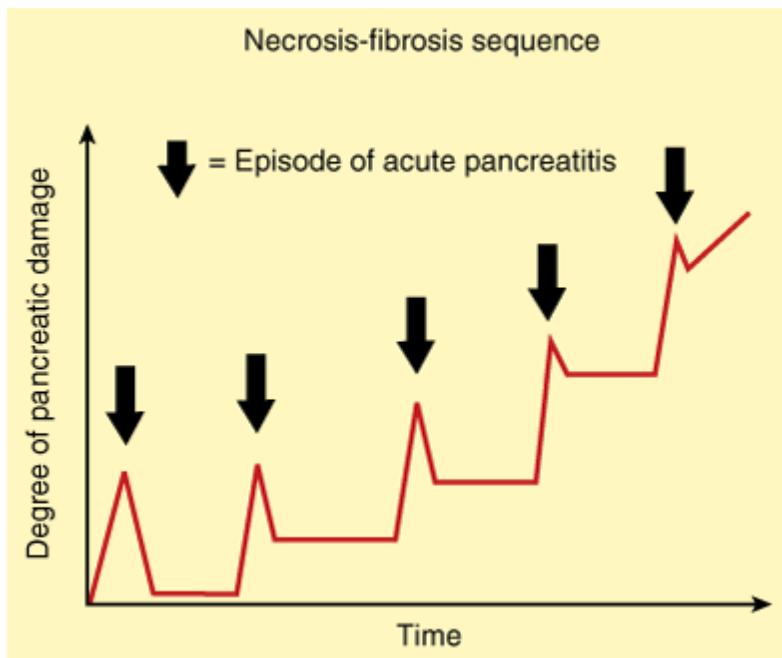
Gallstones are implicated in the majority of AP cases. Although gallstones are common, they rarely cause pancreatitis. It is estimated that over a 20- to 30-year period, the risk of developing pancreatitis due to gall stones in patients with asymptomatic cholelithiasis is approximately 2%. Small gallstones, particularly those smaller than 5 mm in size, increase the risk of AP. Additionally, a long common channel at the junction of the bile and pancreatic ducts may increase this risk. The specific mechanism by

which gallstones produce pancreatitis is still debated, - “but most biliary pancreatitis is precipitated by the obstruction of the ampulla by stones which may be transient or persistent”. In the vast majority of patients, these stones pass into the intestine. Bile crystals, like stones, can cause AP. Patients with microlithiasis may present with recurrent abnormalities in aminotransferases and the evidence of microscopic crystals in bile. Treatment by cholecystectomy eliminates the risk of recurrence.

ALCOHOL

Alcoholic pancreatitis presents as AP, although in most patients, it occurs in the presence of already established chronic pancreatitis (CP). It is the most common cause - of recurrent pancreatitis. The incidence of pancreatitis is low (about 5%) in alcohol abusers. This estimate suggests that in addition to alcohol ingestion, other factors, such as genetic background or environmental influences, may affect patient susceptibility. Several major physiological mechanisms may lead to the development of alcoholic pancreatitis, including -

- abnormal SOD spasm,
- obstruction of the small ducts by protein material
- toxic effect of alcohol and its metabolites.



Multiple hit theory

Several episodes of acute pancreatitis cause increasingly more organized inflammatory changes that finally result in chronic inflammation and scarring.

HYPERLIPIDEMIA

Hyperlipidemia is a cause of AP and CP. Triglyceride levels more than 1000 mg/dL is required for pancreatitis development. The probable disease mechanism is generation of toxic-free fatty acids by the action of lipase on high triglyceride levels in the pancreatic capillary beds, which leads to endothelial damage with the recruitment of inflammatory cells, thrombosis, and ischemia. Following a bout of AP, patients require lipid-lowering medication, as well as treatment of concomitant diabetes and alcohol cessation.

ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY

AP is the most common complication of ERCP. Prospective studies have documented an incidence of approximately 5% with most cases being mild pancreatitis. Risk factors include young age, normal pancreatic ducts, operator inexperience, multiple injections of the pancreatic duct with acinarization, pancreatic sphincterotomy, SOD, and biliary or pancreatic sphincter manometry. Several strategies might reduce the incidence of this complication, including the use of protease inhibitors (gabexate mesilate), somatostatin, IL-6 antibodies, and temporary pancreatic duct stenting.

STRUCTURAL

A variety of conditions that obstruct the pancreatic duct chronically or intermittently may cause AP and include SOD, pancreas divisum, and benign and malignant pancreatic duct strictures. SOD is determined by measuring pressures through the sphincter segment at the time of ERCP. This condition is considered when all other possible etiologies have been eliminated, because performing ERCP with sphincter manometry is also likely to precipitate an attack of AP. “Pancreas divisum - a condition in which dorsal and ventral pancreas fail to fuse. Therefore, the secretion of the larger dorsal pancreas drains through the small minor papilla. This common

variant occurs in 7% of the population, and very few of these patients develop pancreatitis". In a very small subset, AP may develop.

Finally, patients with pancreatic adenocarcinoma may rarely present with unexplained AP, which has led to the recommendation that patients older than 45 years of age with unexplained pancreatitis should undergo an ERCP or endoscopic ultrasonography to evaluate for the possibility of an underlying malignancy

Acute Biliary Pancreatitis

Gallstones account for between 30 and 50% of AP. It is the most frequent cause of the first AP episode. Most studies exclude patients with microlithiasis; thus, the incidence is likely higher. The wide variation in incidence is noted within and between countries, dependent on the population studied and extent of alcohol use in the community. Gallstone pancreatitis is most common in women between the ages of 50 and 70. However, AP occurs more frequently in males than in females with gallstone disease. The risk for severe disease is similar to that observed for other etiologies, but some studies suggest biliary patients have a higher mortality than alcoholic pancreatitis. This higher mortality may be secondary to the increased risk of cholangitis and the older age of presentation. Biliary pancreatitis may be recurrent if the gallstones are left untreated, although it is

not a cause of CP. Recurrence rates are uncertain but may be as high as 30% in the absence of cholecystectomy or biliary sphincterotomy.

Acute Alcoholic Pancreatitis

Excess alcohol intake is the most common etiology of AP in males. Overall, it is the second most common etiology for AP (30%), yet several studies from North America suggest that it may be the most common etiology of AP in the continent. Because alcohol causes recurrent AP, it becomes the predominant etiology when relapses are included in the analysis. Although the incidence of AP is increasing, recent studies do not show any increase in the incidence of alcoholic pancreatitis. Most attacks of acute alcoholic pancreatitis represent an acute attack on CP; however, in most cases, the structural and functional aspects of the pancreas are unknown and the attacks are therefore assumed to be AP. Despite the fact that alcoholic pancreatitis is complicated by severe disease, it is a less common cause of fatal pancreatitis.

Other Etiologies :

Other etiologies identified include pancreatic cancer in 1% of cases, post-ERCP in 2-3%, medications in 1%, miscellaneous causes in 2%, and unknown causes in 15-23% of first attacks of AP.

Recurrent Acute Pancreatitis

Bouts of recurrent AP are most commonly alcohol-related (60%); other etiologies include unknown causes (17%) and untreated gallstones (19%). Recurrent AP appears to be relatively benign and is associated with a low mortality rate.

Natural History and Long-Term Outcome

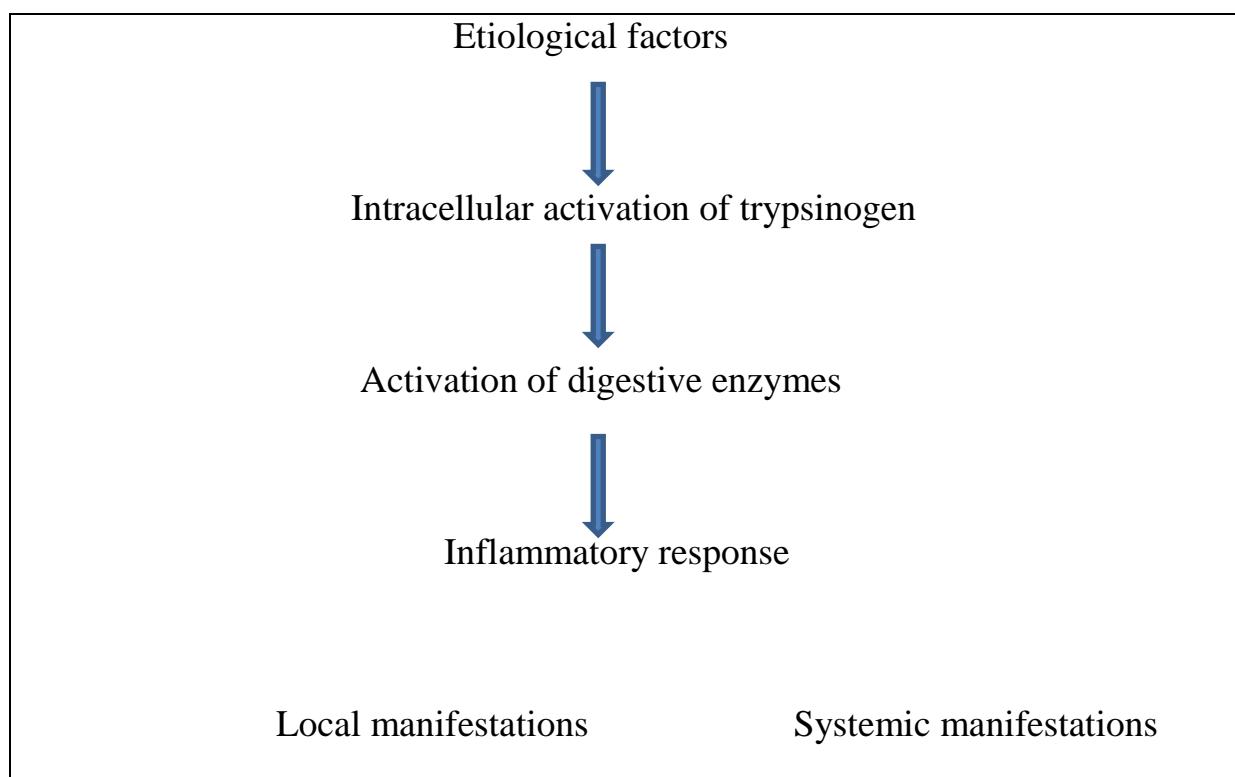
The majority of patients with mild pancreatitis recover uneventfully and once the etiological factor is identified and removed, there are no long-term complications or recurrences. 10 - 20% of patients with AP - develop severe disease and have a complicated hospital course. The incidence of necrosis is 6 - 20%. In patients with necrotizing pancreatitis, long-term followup has demonstrated pancreatic ductal changes on ERCP, although the clinical significance of this evidence is uncertain. Following necrosectomy for necrotizing pancreatitis, approximately 50% of patients will develop long-term pancreatic exocrine and endocrine dysfunction, most have a good function of pancreas. The development of insufficiency of pancreas- varies with the necrosis extent ; and resection of pancreas.

Mortality

The mortality of AP is reported in the literature as being between 1.3 and 10%. A range of 2 - 5% likely represents a true mortality because the higher rates are indicated in studies from referral centers and probably do not include patients with mild disease. Overall, studies suggest a reduction in mortality in the last decade. Gender is not an independent risk factor for AP. When necrotizing pancreatitis is considered, the mortality rate is between 14 and 30%. Approximately half of this mortality is seen in the first 2 weeks. Mortality appears to be influenced by age, etiology (higher in patients with idiopathic, post-ERCP pancreatitis, and gallstone), presence of organ failure on admission and, most importantly, the presence of pancreatic necrosis. Additionally, patients with severe AP transferred to tertiary care facilities for management have higher mortalities. Most studies suggest that approximately 10 - 20% of fatal pancreatitis is missed with the diagnosis only being made at autopsy. The missed diagnosis appears in patients who present without abdominal pain, with acute respiratory failure or neurological changes, and/or normal serum enzymes or pancreatic imaging.

PATHOGENESIS

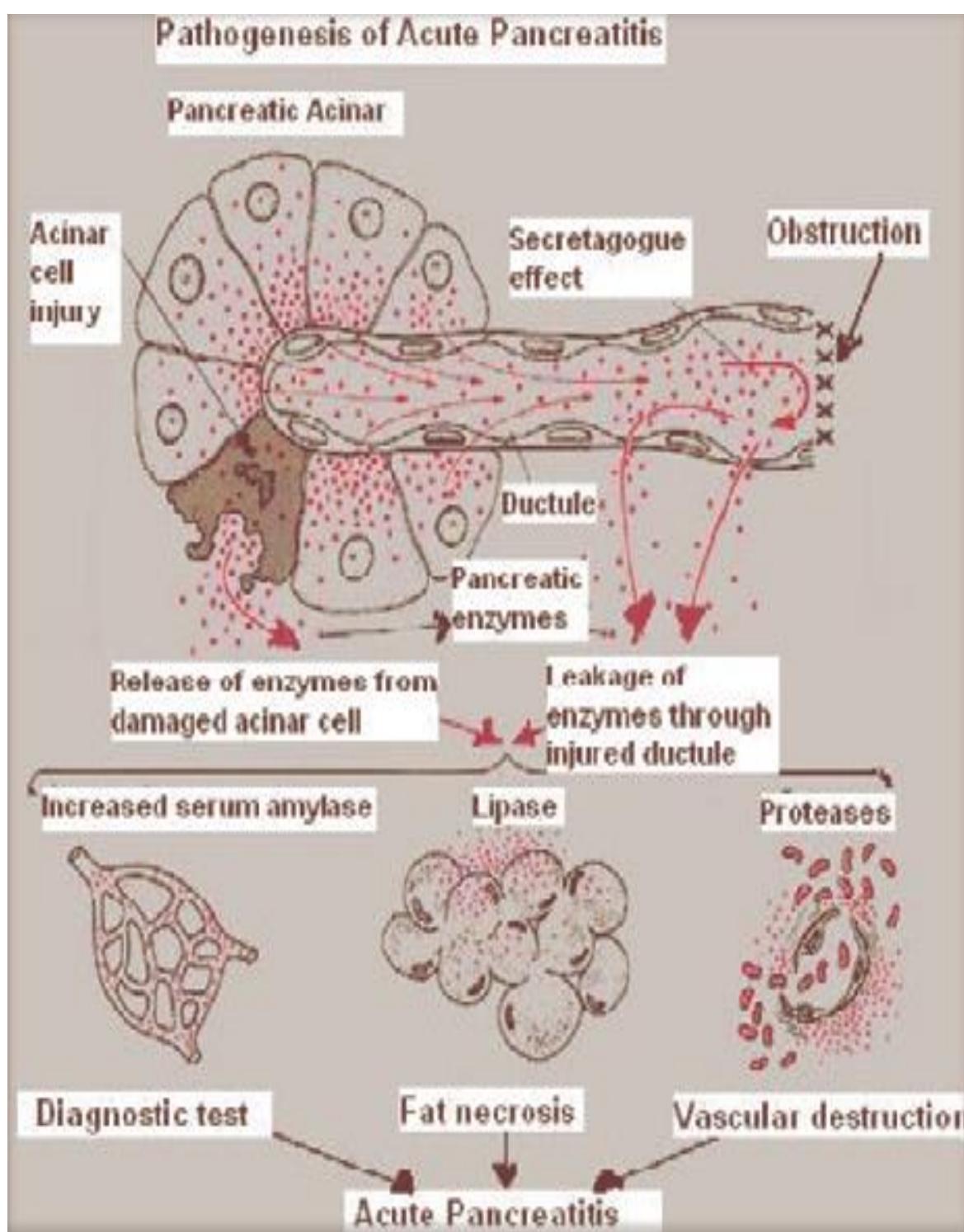
Exact mechanism is not known. Concepts which have been proposed are based on the few experimental animal studies available. Most accepted mechanism is



The following are some 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. they 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 wont be any damage to the gland. These include :
 - Serine protease inhibitor Kazal type 1 (SPINK1)
 - Enzyme Y
 - α 1 antitrypsin
 - α 2 macroglobulin
 - Mesotrypsin
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 systematic 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 into general circulation that may affect the distant organs, giving rise to respiratory distress, renal failure, myocardial depression and shock or metabolic alterations. Finally a MODS may occur with vital risk of necrotic tissue infection, a situation where translocation of intestinal pathogens play an important role.

7. Genetic factors implicated in pathogenesis of acute pancreatitis are

- Cationic trypsinogen gene (PRSS1)
- Cystic fibrosis transmembrane conductance regulator gene (CFTR)
- Polymorphisms in SPINK1

CLINICAL FEATURES

Pain abdomen : most common symptom

- Located in upper abdomen (epigastrium/ rt 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

1. Nausea
2. Vomiting
3. Severe retching

Physical findings:

Typically pancreatitis patients are seen rolling around in the bed or moving around trying 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
- Retro peritoneal haemorrhage leading to bluish discolouration in
 - ✓ “Umbilical area – Cullen’s sign”
 - ✓ “Loin - Grey Turner’s sign”
 - ✓ “Groin - Fox’s sign”

General examination :-

- Tachycardia/ hypotension and tachypnea related to hypovolemic state
- Hyperthermia related to release of pro inflammatory cytokines.
- Jaundice
- Decreased breath sounds in basal lung fields secondary to atelectasis pleural effusion

Differential diagnosis for acute pancreatitis

- “Biliary pain/acute cholecystitis
- Perforation of bowel/stomach
- Mesenteric ischemia or infarction
- Closed- loop intestinal obstruction
- Inferior wall MI
- Dissecting aortic aneurysm
- Ruptured ectopic pregnancy”

Laboratory Tests

The diagnosis of AP is usually based on the appropriate clinical features and is confirmed by laboratory and imaging tests. Leakage of

pancreatic enzymes into the circulation is a hallmark of AP. Although amylase and lipase constitute a small fraction of all pancreatic enzymes, they are the easiest and the quickest enzymes to measure. Typically, the elevation of serum amylase in AP is above threefold of the normal values. Amylase levels are usually increased within a few hours of disease onset, but they may be cleared from the serum rather quickly. Serum amylase usually remains elevated for 3 to 5 days in uncomplicated AP.

Causes of Increased Serum Amylase Activity

- Pancreatic disorders
- Pancreatic cancer
- Abdominal emergencies
- Acute cholecystitis
- CBD obstruction
- Perforated viscus
- Intestinal ischemia
- appendicitis
- Ruptured ectopic pregnancy
- Salivary gland diseases
- Renal insufficiency
- Macroamylasemia

- Diabetic ketoacidosis
- HIV infection/AIDS
- Sphincter Oddi stenosis or spasm
- drugs- morphine
- Acute pancreatitis
- Acute salpingitis

Because many conditions can cause hyperamylasemia, the specificity of elevated serum amylase level is less than 70%. Very high elevations of serum amylase (more than fivefold normal), however, are rarely associated with diseases other than AP. Elevations of three- to fivefold normal are commonly seen in the absence of acute pancreatitis in patients with renal failure, as a result of decreased clearance of the enzyme. Measurements of urinary amylase and the amylase-to-creatinine ratio may be helpful to distinguish AP from other causes of hyperamylasemia, but such measurements are infrequently employed.

Serum amylase isoenzyme measurements may improve the diagnostic accuracy of serum amylase alone. In healthy people, less than half of all circulating amylase originates in the pancreas, whereas the remainder is of salivary origin. Serum pancreatic isoamylase (P-isoamylase) accounts for

the elevated total serum amylase level in AP and tends to persist for several days. However, pancreatic isoamylase can be elevated in some other gastrointestinal disorders and in renal insufficiency, making it difficult to diagnose AP based on P-isoamylase levels alone without additional diagnostic parameters.

The elevation of serum lipase generally parallels the serum amylase level in AP. However, the serum lipase level often remains elevated longer, making it more useful to diagnose pancreatitis after symptoms have subsided. Lipase is considered more specific than amylase for pancreatic tissue injury, despite that lipase is also produced by numerous other gastrointestinal tissues. Another potential advantage of lipase is that it is generally not elevated in diabetic ketoacidosis or macroamylasemia.

Both amylase and lipase are widely available and are, in general, rapidly available from hospital laboratories. In practice, combining the measurement of serum amylase and lipase somewhat enhances the diagnostic accuracy for AP. A normal amylase or lipase level makes the diagnosis of AP unlikely, except in the presence of hyperlipidemia. Very high levels of serum triglyceride (one of the causes of AP) can interfere with the

laboratory assay for both amylase and lipase; dilution of the serum may be necessary in this situation to reliably measure the elevations of amylase or lipase.

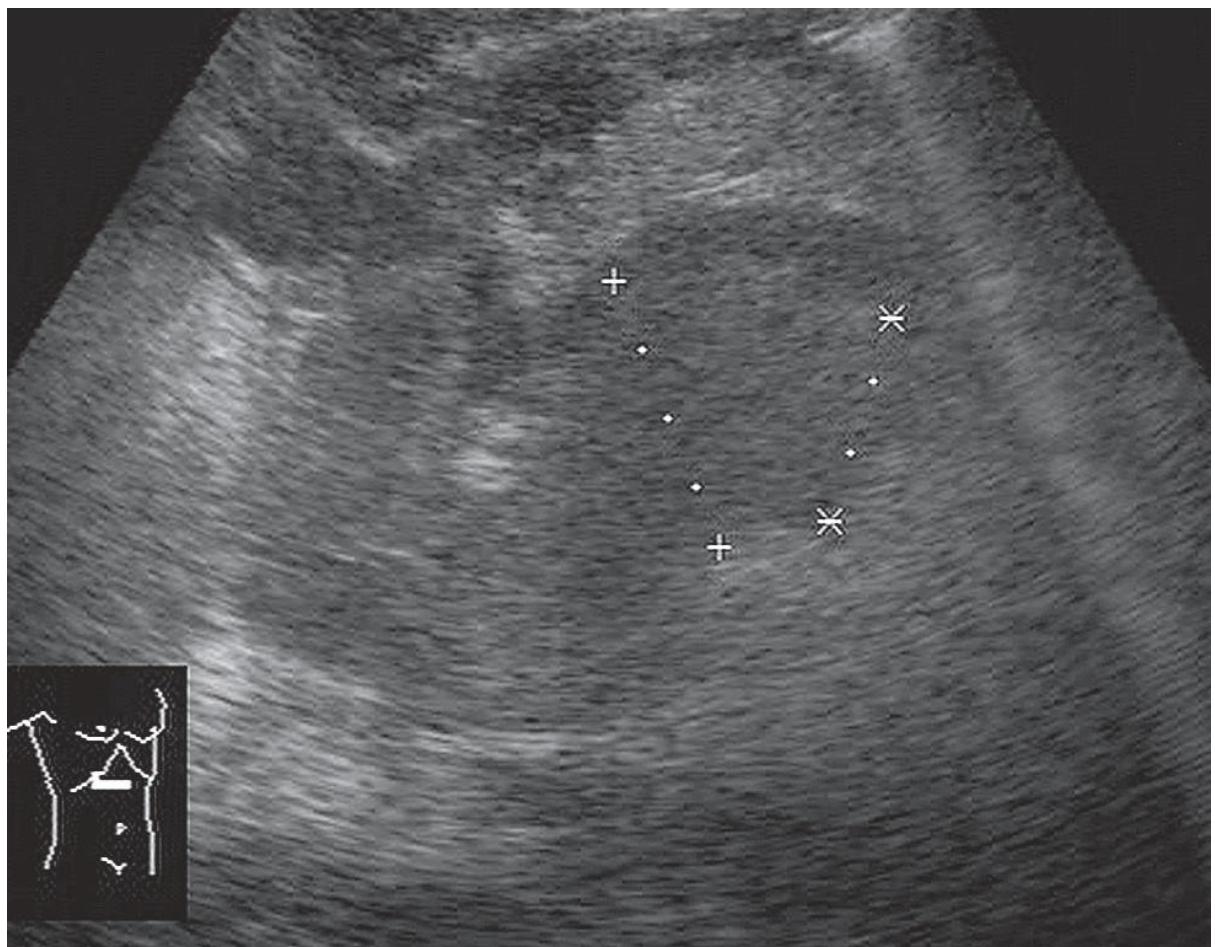
In some patients with chronic pancreatitis, acute abdominal pain can be the result of focal acute inflammation of the gland, and serum amylase and lipase levels may remain normal. It is important to note that a correlation has not been found between the degree or trend of serum amylase and lipase elevation with the amount of structural damage of the pancreas or severity of AP.

Pancreatic enzymes, such as “serum trypsin, chymotrypsin, elastase, ribonuclease, and phospholipase A2”, have been all reported to be raised in AP, but assays to measure these enzymes are not readily available for clinical use, and their specificity has not been defined. The use of other clinically available laboratory tests may have a role in determining the etiology of AP. For example, elevated bilirubin and hepatic transaminases, particularly alanine aminotransferase more than 80 IU/L should raise the suspicion of gallstone pancreatitis.

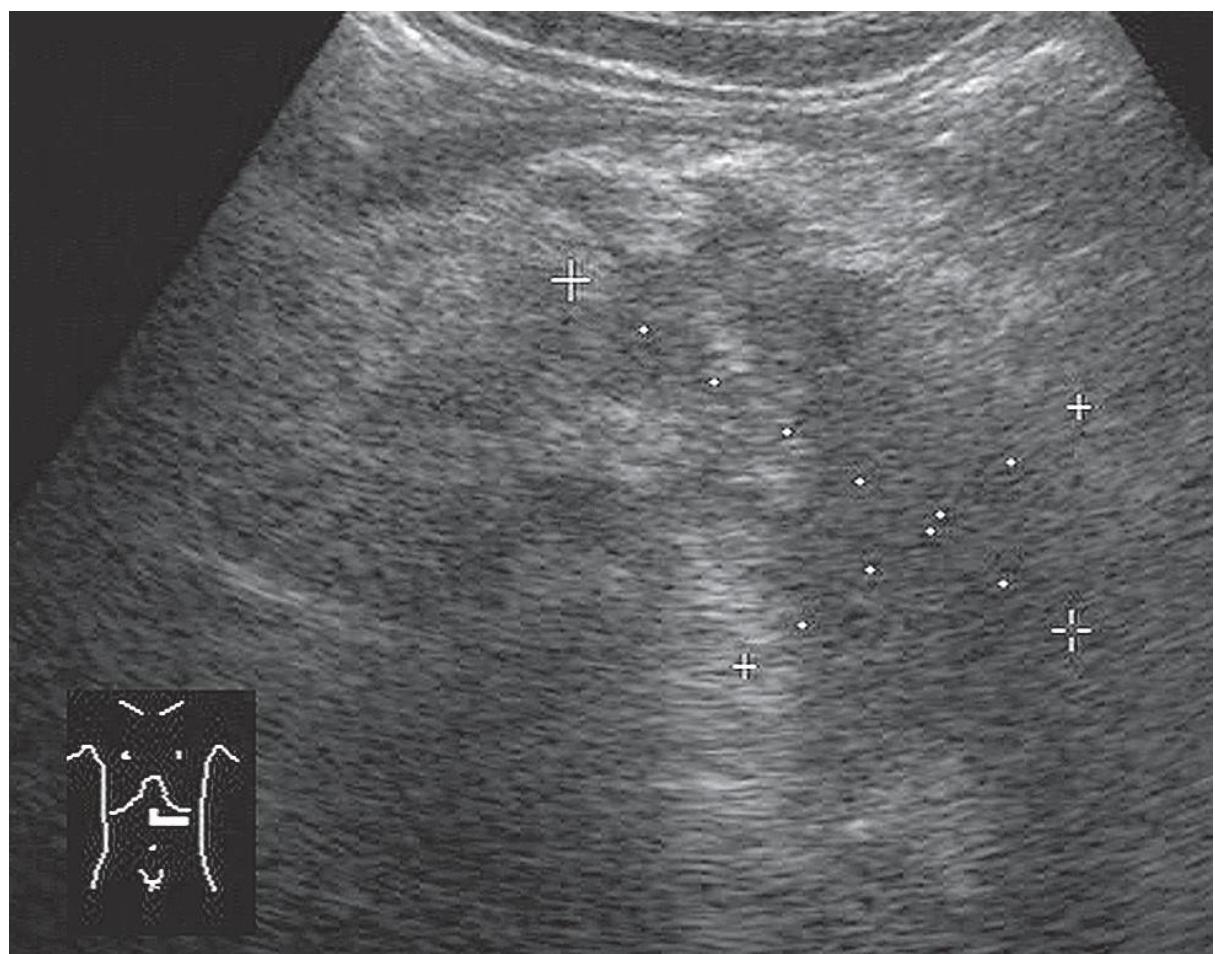
IMAGING

1. ULTRASONOGRAPHY

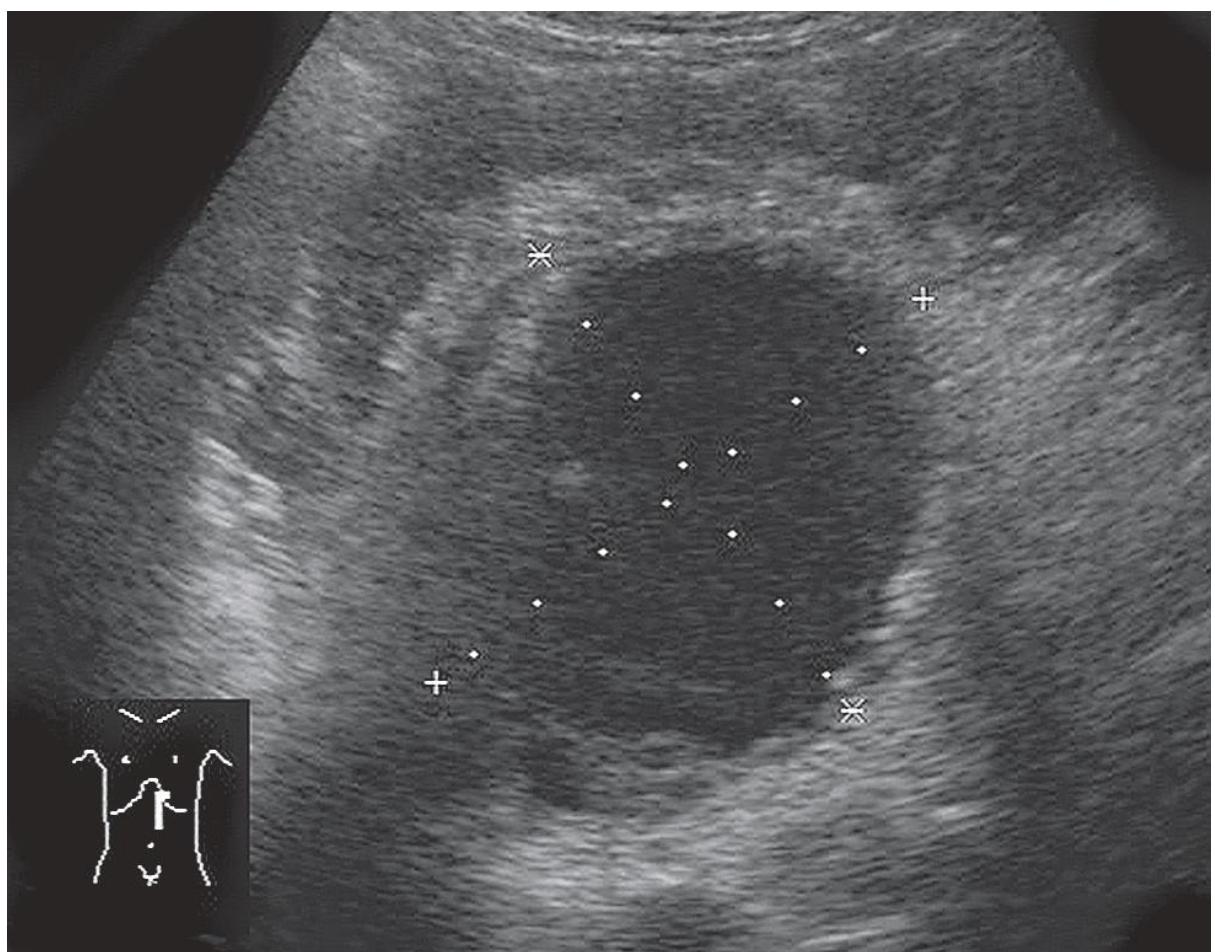
Transabdominal ultrasonography is widely available, relatively inexpensive, and quite safe. Unfortunately, pancreatic imaging by ultrasound has limitations from overlying bowel gas and surrounding fat planes, which tend to be exaggerated in the acutely inflamed pancreas owing to ileus and peripancreatic edema. Thus the sensitivity and specificity of this modality for diagnosing AP is low. Nonetheless, transabdominal ultrasonography is useful in the early stages of AP to search for gallbladder stones or sludge, evaluate for dilation of the common bile duct caused by choledocholithiasis, and analyze for other possible causes of severe abdominal pain.



Collection in the peri pancreatic area



Pancreatic necrosis



Pseudocyst of pancreas



Pancreatic abscess

2. COMPUTED TOMOGRAPHY SCAN

The computed tomography (CT) scan, particularly when done with helical or multidetector technology, is valuable in the diagnosis and management of AP. However, not every patient with AP requires a CT scan. CT is mainly indicated if the initial diagnosis is in doubt or for prognostic purposes in severely ill patients as in the section on Risk Stratification. The role of CT is both to document the appropriate findings that confirm the diagnosis of AP and to exclude other intraabdominal catastrophes that can mimic AP (e.g., a perforated viscus). CT scan findings, which support the diagnosis of AP, include

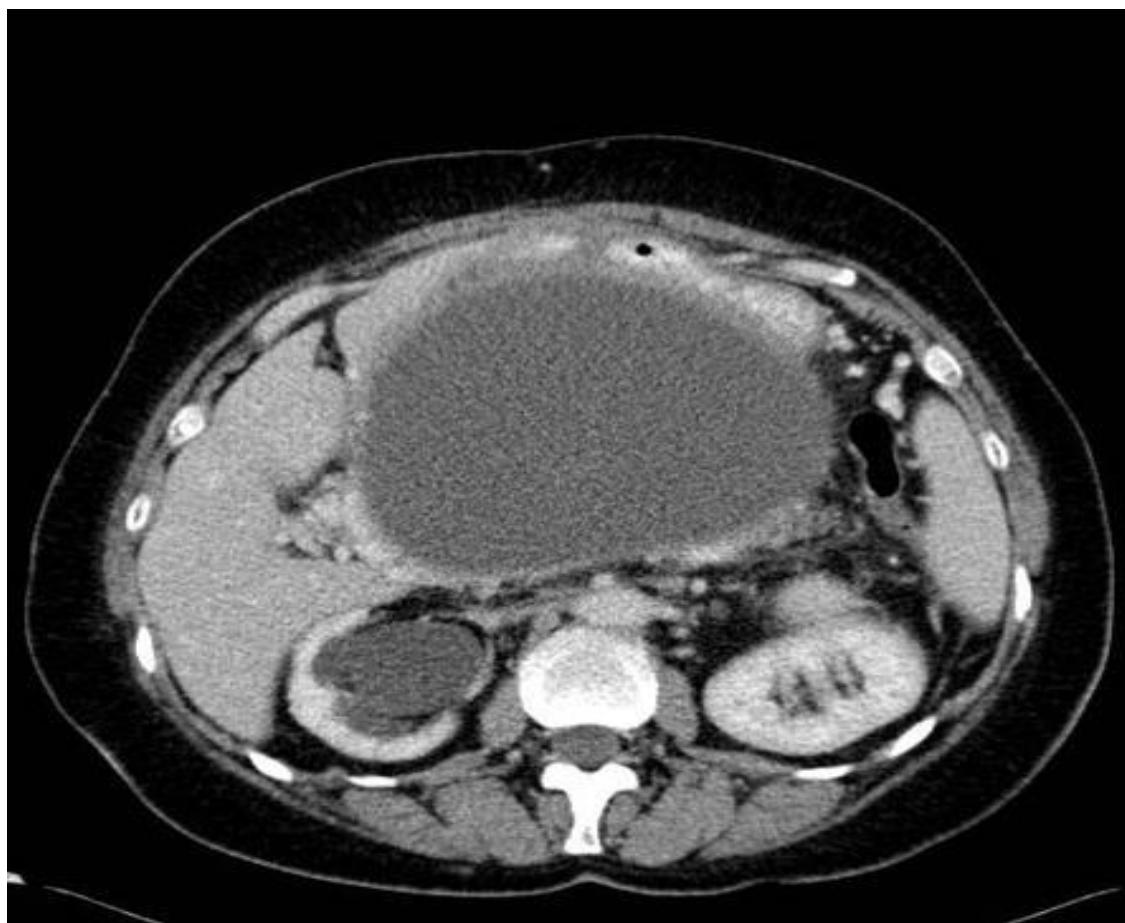
- “diffuse or segmental enlargement of the pancreas”,
- “irregularity of the pancreatic contour with obliteration of the peripancreatic fat planes”,
- “areas of decreased density within the pancreas,”
- “fluid collections in the pancreas or outside the gland in the lesser sac or pararenal spaces”.

The frequency of these findings varies according to the severity of pancreatitis, and these findings do not require intravenous administration of contrast material to be identified.

Intravenous contrast-enhanced computed tomography (CECT) is mainly used to differentiate pancreatic necrosis from interstitial pancreatitis or to

monitor for pancreatitis complications in selected cases (i.e., to assist in estimating prognosis or managing patients with AP, rather than simply confirming a diagnosis). Normal CT findings have been reported in 24 67% of patients with mild AP.

Controversy exists as to whether intravenous contrast early in the clinical course exacerbates the severity of AP. Although deleterious effects of intravenous contrast have been observed in animal models of experimental pancreatitis, studies in humans have yielded conflicting results. Many authors agree that CECT scans are unnecessary in patients with mild AP and should be done for those patients; who present with a more complicated- clinical course. Additionally, early CECT may underestimate the degree of pancreatic necrosis that may develop over time from the disruption of pancreatic microvascular circulation that usually occurs in the first 12 - 24 hours of AP. At present, it is recommended that CECT be obtained 3 - 4 days after the onset of SAP for optimal assessment of pancreatic necrosis.



Ct abdomen showing pseudocyst of pancreas

3. MAGNETIC RESONANCE IMAGING

Currently, magnetic resonance imaging (MRI) has no advantage Over CT scan in the management of AP. MRI has a comparable specificity and sensitivity for diagnostic and severity assessment of AP^{17,18}. Its cost, availability, and contraindication in patients with metallic implants has limited the application of MRI in AP to date.

4.'ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY'

ERCP has no role in diagnosing AP. Therapeutic application of ERCP in moderate to severe acute gallstone pancreatitis has been shown by several controlled clinical trials to lower morbidity and mortality when compared to traditional medical treatment alone. ERCP is also utilized in the differential diagnosis and elective treatment of recurrent unexplained pancreatitis secondary to sphincter Oddi dysfunction, pancreatic divisum, and microlithiasis.

5. ENDOSCOPIC ULTRASOUND

The diagnostic role of endoscopic ultrasound (EUS) in AP is still evolving; it is not readily available in all institutions. In recent studies, the immediate application of EUS for suspected biliary AP may aid in the diagnosis of gallstone pancreatitis, thereby helping to triage patients for therapeutic ERCP with endoscopic sphincterotomy and stone removal.

TREATMENT

GENERAL CONSIDERATIONS

- The patient needs adequate iv fluids and adequate pain control.
- NPO is usually done until nausea and vomiting have stopped.
- Abdominal pain is managed through analgesics. Morphine may be used..
Although morphine can increase SOD tone and to increase serum amylase values, it is used in the management of AP.
- Nasogastric aspiration is not useful in pancreatitis. This modality is used to reduce ileus and intractable nausea and vomiting.
- PPI s and H 2 receptor blocking agents are not useful.
- Each patient should be monitored for any signs of early organ failure like fall in blood pressure and drop in O2 saturation or renal failure via close following of vital signs and urinary output.
- Blood gas measurements and O 2 supplementation are helpful in patients with increased rate of respiration
- Any patient who exhibit signs of early organ dysfunction should be shifted to ICU because deterioration can be rapid and fatal.

FLUID RESUSCITATION

- “Maintaining adequate intravascular volume in severe acute pancreatitis require 5 to10 litres of IV daily for the first several days”
- “A Swan-Ganz catheter is used to measure central venous pressure. “
- “Aggressive fluid resuscitation may not prevent pancreatic necrosis. Haemodilution to a haematocrit value of 30% with dextran 60 solution improved the pancreatic oxygenation and microcirculation and. When the haematocrit decreased to 25%, packed red blood cells should be transfused to maintain a haematocrit close to 30%.”

RESPIRATORY CARE:

- Hypoxemia (saturation of oxygen < 90%) requires oxygen, ideally administered through nasal prongs or face mask if needed.
- Endotracheal intubation and assisted ventilation are used if nasally administered oxygen does not correct hypoxemia.
- Acute Respiratory Distress Syndrome is a serious respiratory complication of acute pancreatitis because leads to severe dyspnoea, progressive hypoxemia, and higher mortality. It occurs between the 2 nd and 7 th day. Chest X-ray shows multilobar pulmonary infiltrates. Treatment is mechanical ventilation with positive end- expiratory pressure.

CARDIOVASCULAR CARE

Severe acute pancreatitis can cause arrhythmias , congestive heartfailure, MI, and cardiogenic shock.. If hypotension is present even after adequate fluid resuscitation, intravenous dopamine may be used to maintain systemic blood pressure. Dopamine does not affect the pancreas blood micro circulation.

METABOLIC COMPLICATIONS:

- Hyperglycemia may be present during the first several days of severe pancreatitis but usually comes to normal as the inflammatory process subsides. Blood glucose levels fluctuate widely, and insulin should be administered with caution.
- Hypocalcemia may be due to low serum albumin concentration causes no symptoms and requires no treatment. Reduced serum ionized calcium may cause neuromuscular irritability.
- If the patient also has hypomagnesemia, magnesium replacement should restore serum calcium level to normal. Causes of magnesium depletion include vomiting, loss of magnesium in urine, and deposition of magnesium in areas of fat necrosis.

ANTIBIOTICS

- Antibiotics are essential for extra pancreatic infections such as ‘cholangitis, catheter-acquired infections, septicaemia , UTI and pneumonia’.
- In severe acute pancreatitis, prophylactic antibiotics - is not recommended
- For sterile necrosis, antibiotics are not essential.
- . In Infected necrosis (i) initial CT-guided needle aspiration - for Gram stain; and culture to look for antibiotic sensitivity is done
- (ii)Antibiotics may be given empirically .

ENDOSCOPIC THERAPY

Urgent Removal of gallstones in gallstone pancreatitis

- In patients with mild acute pancreatitis,if found to have cholelithiasis, a cholecystectomy is done before leaving the hospital to prevent a recurrent acute pancreatitis
- For necrotizing biliary AP, cholecystectomy is postponed until active inflammation subsides in order to prevent infection.
- “use of antifungal agents along with antibiotics is not routinely recommended”

NUTRITIONAL THERAPY

- “In mild acute pancreatitis, oral feeds can be started immediately, if there is no abdominal pain , nausea and vomiting”
- “In mild acute pancreatitis starting feeds with a low-fat solid diet appears safe as a clear liquid diet”
- “In severe acute pancreatitis, enteral route of nutrition is used to avoid infections. Parenteral nutrition is avoided and may be given if the enteral route is not available or not tolerated” .
- Nasogastric and nasojejunal delivery are equally efficacious.

ERCP in acute pancreatitis

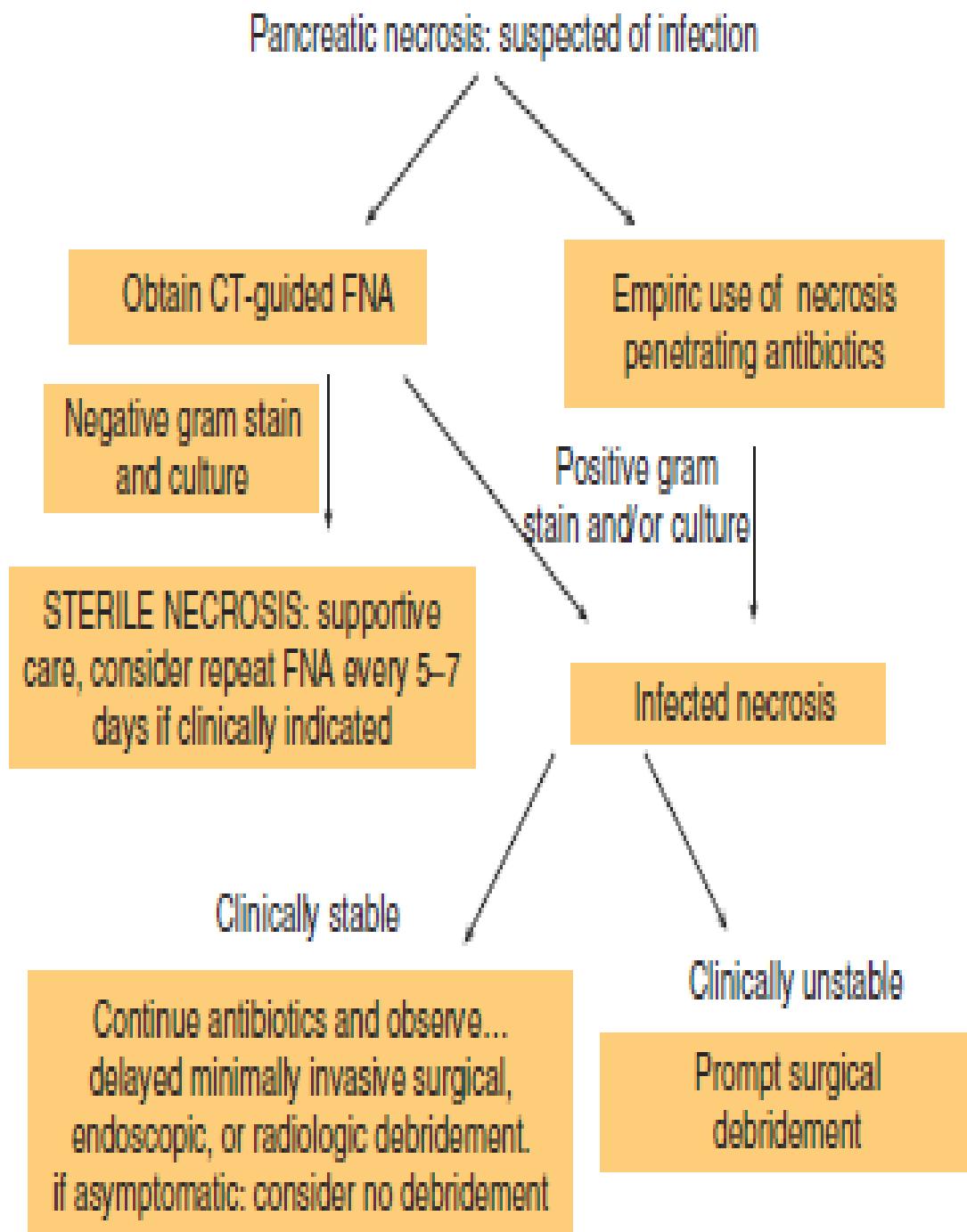
- “Patients of acute pancreatitis with associated acute cholangitis should undergo ERCP within 24 hours of admission .”
- ERCP is not useful in patients with pancreatitis due to cholelithiasis without biliary obstruction
- MRCP or EUS is used for ERCP to screen for CBD stone in the absence of cholangitis.
- To prevent post-ERCP pancreatitis, rectal (NSAID) suppositories are utilized.

SURGERY

- “In patients with mild acute pancreatitis,if found to have cholelithiasis, a cholecystectomy is done before leaving hospital to prevent a recurrence

of acute pancreatitis”

- “In patients with necrotizing biliary acute pancreatitis, cholecystectomy is postponed until active inflammation subsides in order to prevent infection.”
- “The presence of asymptomatic pseudocysts and pancreatic and / or extrapancreatic necrosis is managed conservatively.”
- “In patients with infected necrosis who are stable , drainage should be delayed for > 1 month to allow liquefaction of necrotic contents and development of a fibrous wall around the necrosis (walled-off necrosis)”
- “In patients with infected necrosis who are symptomatic , minimally invasive methods of necrosectomy are preferred over open necrosectomy”



COMPLICATIONS OF ACUTE PANCREATITIS

Local

- Sterile - pancreatic necrosis
- Infected - pancreatic necrosis
- Pancreatic Abscess
- Pseudocyst
- GI bleeding:

Pancreatitis- related:

- Splenic artery rupture
- damage to splenic vessel;
- damage to portal vein;
- thrombosis of splenic vein leading to gastro- esophageal varices

Non- pancreatitis – related:

- “Mallory- Weiss tear”
- “Gastropathy;”
- Mucosal ulcers in stomach
- Infarction
- Hematoma
- small / large bowel fistula/obstruction.
- Hydronephrosis(Rt side)

Systemic

- Lung failure
- “Renal failure”.
- Shock (circulatory failure)
- Increase in blood glucose
- Decrease in serum calcium
- DIC
- Subcutaneous nodules due to fat necrosis
- Retinopathy
- Psychosis

LOCAL COMPLICATIONS

PANCREATIC NECROSIS

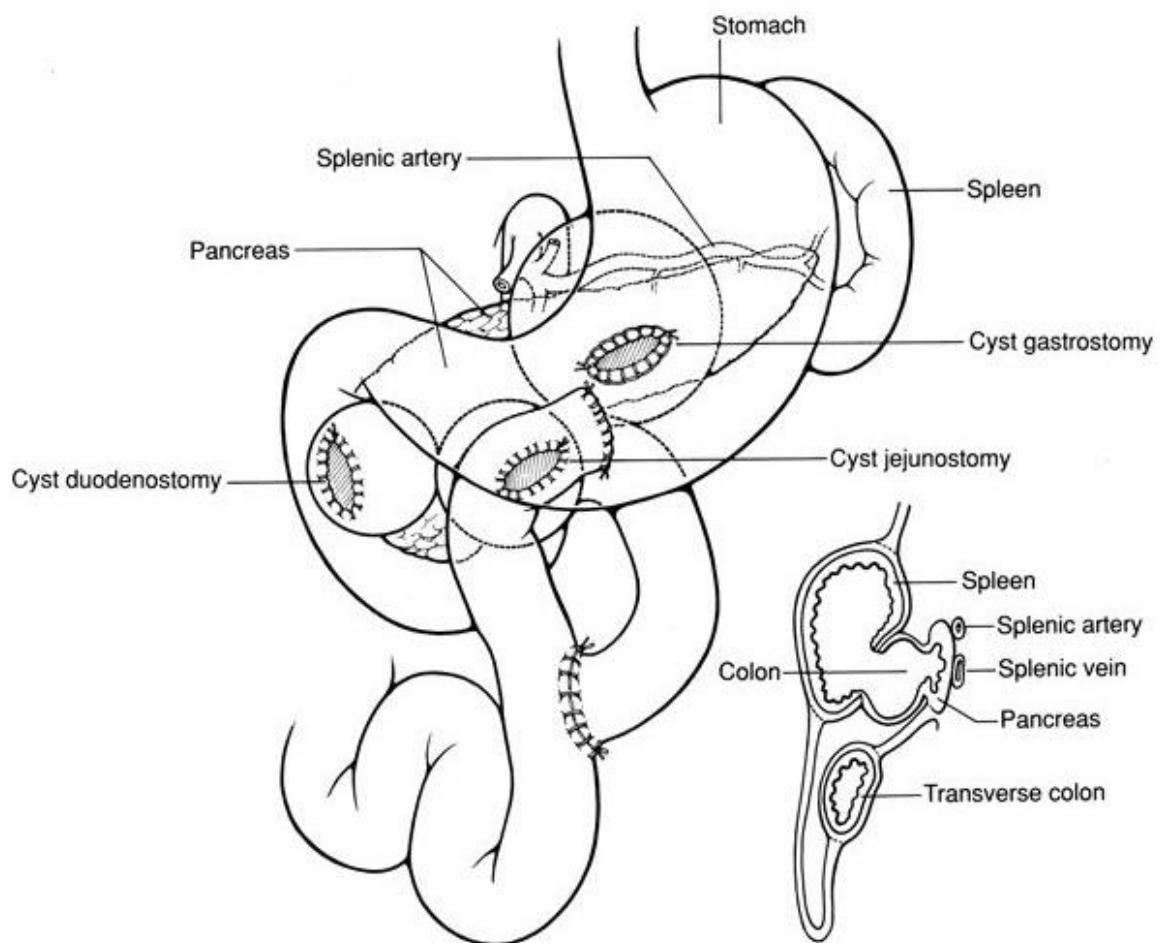
In about 20% of patients with acute pancreatitis, CT shows necrosis. Pancreatic infection is uncommon in interstitial pancreatitis but may occur in 20% to 50% of individuals with necrotizing pancreatitis. Infection typically appears within the first 2 weeks of illness. In comparison, as mentioned previously, pancreatic abscess due to acute pancreatitis may occur after 4 weeks.

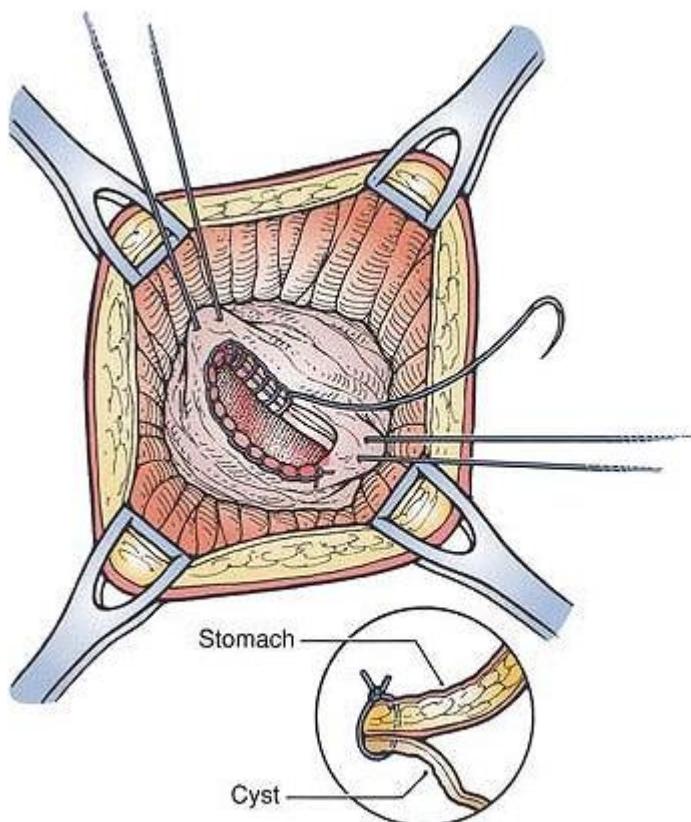
PANCREATIC PSEUDOCYST

- A pseudocyst occurs secondary to AP, trauma, or CP.
- It contains a high concentration of pancreatic enzymes and varying amounts of tissue debris.

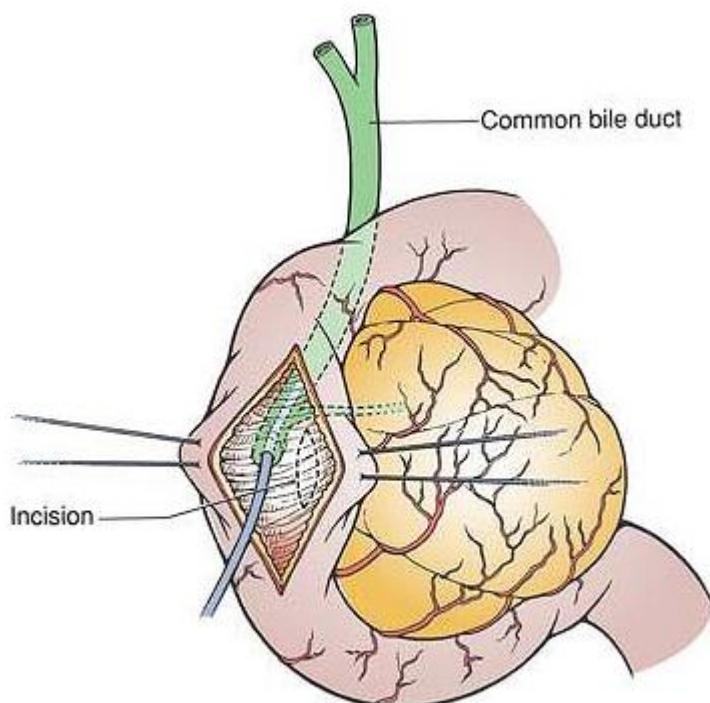
- Most pseudo cysts are sterile.
- Regardless of size,- an asymptomatic pseudocyst does not require treatment.
- USG is done every 3 to 6 months.
- In 2 studies, there were no deaths among patients treated either medically or surgically.
- Pseudocysts can be complicated by
 - infection,
 - intracystic haemorrhage,
 - rupture leading to pancreatic ascites.
- pseudocysts can migrate into the chest or other unusual locations.
- if an individual develops pain, chills, and fever, abscess formation should be suspected
- Treatment –
 - surgical,
 - radiologic
 - endoscopic drainage.
- No randomized prospective trials have compared these methods.
- Surgical drainage of a pseudocyst is possible with a cystogastrostomy or cystoduodenostomy if the pseudocyst wall is broadly adherent to the stomach or duodenum.

- Other options are a Roux-en-Y cystojejunostomy and pancreatic resection if the pseudocyst is in the tail.
- Surgical mortality is less than six percent.
- Pseudocyst recurrence after internal drainage occurs in 15% of cases and is more common if the main pancreatic duct is obstructed downstream from the surgical anastomosis.
- For this reason, a preoperative ERCP is usually performed to determine whether there is duct obstruction. In the presence of duct obstruction, a resection of the pseudocyst is preferred.
- Percutaneous catheter drainage is effective treatment to drain and close both sterile and infected pseudocysts.
- As with surgical drainage, percutaneous catheter drainage may fail if there is obstruction of the main pancreatic duct downstream from the pseudocyst.
- Therefore, an ERCP is usually performed before catheter drainage is attempted. Two endoscopic methods to decompress a pancreatic pseudocyst are (1) an endoscopic cyst- gastrostomy or cyst-duodenostomy and (2) insertion of a stent through the ampulla directly into the pancreatic duct and then into the pseudocyst itself.

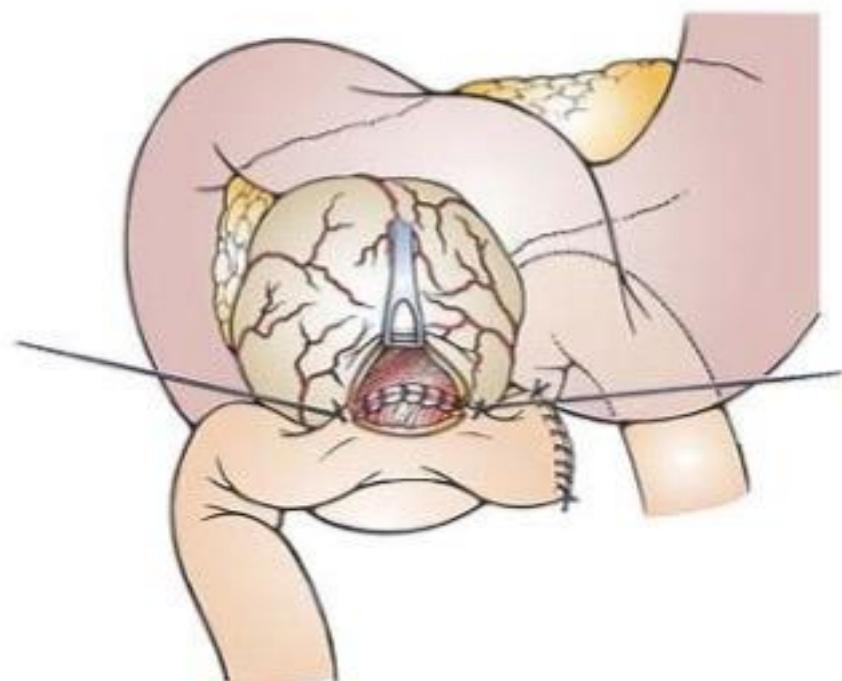
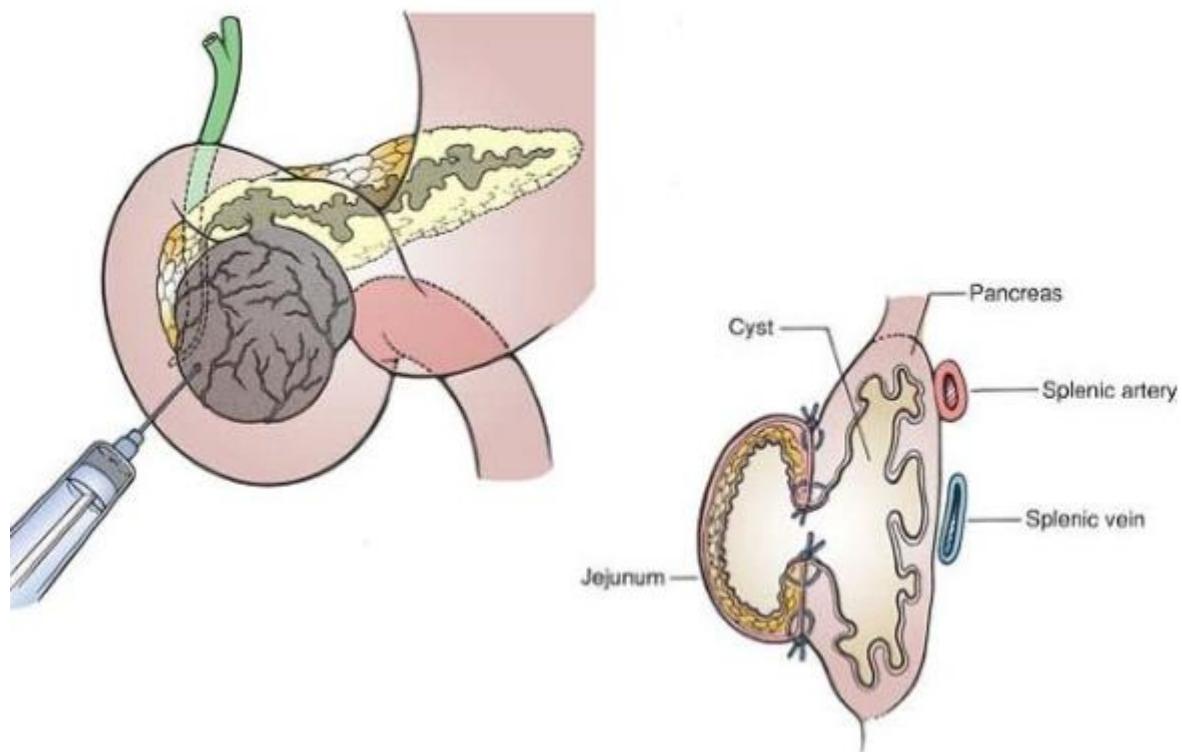




cysto gastrostomy



cysto duodenostomy



Roux-en-Y Drainage of a Pseudocyst

Steps in Procedure—Cyst Gastrostomy

Steps of cysto gastrostomy

- Upper midline incision
- Explore abdomen
- Confirm retrogastric location
- Place two stay sutures on anterior surface of stomach, centered on cyst
- Create longitudinal gastrotomy
- Oversew edge of cystogastrostomy with running lock stitch
- Check hemostasis
- Close gastrotomy and cover with omentum
- Close abdomen without drains

Steps of cyst duodenostomy

- Upper midline incision
- Explore abdomen and confirm cyst adherent to duodenum but not stomach
- (Kocher maneuver performed)
- Open bile duct and place #3 Bakes dilator or other cannula through ampulla
- Two stay sutures on anterior surface of duodenum over ampulla
- Generous longitudinal duodenotomy
- Choose site for cyst duodenostomy away from ampulla (usually medial to ampulla)
- Aspirate to confirm cyst and exclude blood in cyst
- Create opening into cyst
- Perform full-thickness biopsy of cyst wall
- Running lock stitch to oversew cyst duodenostomy (avoid Ampulla)
- Close duodenostomy and cover with omentum
- Close abdomen without drains

Cysto gastrostomy is done if the pseudocyst is broadly adherent to the wall of the stomach or duodenum.

Endoscopic approach

- Insertion of a double – pigtail stent through the stomach into the pseudocyst.
- Insertion of a transpapillary pancreatic duct stent into the cyst.
- catheter is removed after 3 to 4 weeks if closure of the pseudocyst is seen on CT.

GASTROINTESTINAL BLEEDING:

Causes

- stress induced mucosal gastropathy,
- Mallory- Weiss tear
- alcoholic gastropathy.

Alternatively, bleeding can be due to the inflammatory aspects of the pancreatitis- due to irritative effects of liberated activated pancreatic enzymes on vascular structures or pressure necrosis of in inflammatory debris or fluid collections on surrounding structures.

- Rupture of the splenic artery, splenic vein, or portal vein may be seen.

Treatment is surgical ligation and resection.

- Variceal bleeding occurs.
- Pseudocysts lead to pseudoaneurysm, seen on dynamic contrast-enhanced CT. If the pseudoaneurysms bleed, arteriography with embolization is the treatment of choice.

bleeding into the pancreatic duct occurs (hemosuccus pancreaticus),

SPLENIC COMPLICATIONS

Splenic complications of acute pancreatitis include

- intra splenic pseudocysts
- infarction
- necrosis of the spleen,
- splenic rupture
- hematoma.

Some of these complications can be life- threatening and require emergency splenectomy.

INTESTINAL COMPLICATIONS

- Pressure necrosis from inflammatory debris from the tail of the pancreas can obstruct or perforate the bowel or can fistulize into the small or large intestine.
- The most common site is – the left colon. Treatment is frequently surgical.

SYSTEMIC COMPLICATIONS:

Organ Dysfunction

Respiratory insufficiency is the most common systemic complication associated with pancreatitis. The causes are multifactorial and include pleural effusions, pneumonia, atelectasis, and ARDS. Oxygen supplementation, antibiotics, pleurocentesis, and assisted ventilation may be necessary. Renal complications are due to hypovolemia causing prerenal azotemia or hypotension leading, to acute tubular necrosis. These are treated with an increase in intravenous fluid administration for the case or hemofiltration or hemodialysis for the latter.

Shock is usually caused by hypovolemia secondary to third space losses, vomiting, and interstitial visceral edema. Other uncommon sources are myocardial infarction and pericardial effusions. Fluid replacement in severe acute pancreatitis is best accomplished via central venous monitoring. As mentioned previously, mortality is substantially raised with increasing organ dysfunction, especially if shock is involved.

Metabolic disturbances

Hyperglycemia and hypocalcemia are common in severe disease. Hypoglycemia is usually transient, is due to insulin deficiency from presumed islet cell necrosis hyper glucagonemia. It is uncommon for these complications to require aggressive treatment.

Fat necrosis

Fat necrosis occurs in subcutaneous tissue, bone, retroperitoneal tissue, peritoneum, mediastinum, pleura and pericardium, fat cells are necrotic and are associated with a diffuse inflammatory infiltration. The subcutaneous lesions are circumscribed, tender, red nodules that are adherent to the skin but are movable over deeper structures. Most commonly they occur over the ankles, figures, knees, and elbows. The lesions may drain. Through the skin, rarely, there is also necrosis of adjacent tendons or involvement of joints, particularly the metatarsal, interphalangeal, wrist, knee, and ankle joints. The lesions usually resolve after days to weeks.

Coagulation Disorders:

- Mild pre DIC defects are common in acute pancreatitis
- D-dimer levels increase in the blood.
- Full-blown DIC with a bleeding diathesis is rare.

MISCELLANEOUS COMPLICATIONS

Pancreatic encephalopathy consists of a variety of central nervous system symptoms occurring in acute pancreatitis, including agitation, hallucinations, confusion, disorientation, and coma. A similar syndrome may be due to alcohol withdrawal, and other causes are possible, such as electrolyte disturbances (e.g., hyponatremia) and hypoxia. Purtscher's retinopathy (discrete flame-shaped haemorrhages with cotton-wool spots) can cause sudden blindness; it's believed to be due to microembolization in the choroidal and retinal arteries.

RISK STRATIFICATION IN ACUTE PANCREATITIS

Early evaluation of AP severity is essential to allow the clinician to unit admission. Severe pancreatitis can be defined by various systems that predict complications and mortality or by the development of the complication itself. Thus, there is a difference between a predictive system that suggests complications may develop and the actual development of a complication. This section focuses on methods to predict morbidity and mortality. Severe pancreatitis can be predicted by clinical criteria, multiple factor scoring detect severe pancreatitis is similar to the accuracy of the multiple factor scoring systems. Several of these scoring systems have been developed to assist the clinician in the assessment of the severity of AP. The most commonly used systems are the Ranson criteria, the modified Glasgow scoring system, and the Acute Physiology And Chronic Health Evaluation II (APACHE II)

Ranson Criteria and the Modified Glasgow System:

They rely on a collection of clinical and biochemical variables measured within the first 48 hours of admission. Clearly, from looking at these systems, many of the variables are factors that any clinician would be attuned to in managing a critically ill patient and the scoring systems merely place these variables within a numerical framework. Using these systems,

it is only possible to predict severity after 48 hours have passed. Higher Ranson or Glasgow scores predict severe disease with reasonable sensitivity. Mortality is less than 5% in patients with Ranson score of 0, in comparison to 10% for those with a criteria of 3 - 5, and 60% for those with a Ranson score greater than 6. Thus, many patients with higher Ranson scores do not die and, in fact, do not develop organ failure or other complications. The same is true for the modified Glasgow scoring system. Therefore, the Ranson and modified Glasgow scoring systems lack specificity. It should also be noted that there are separate Ranson scoring systems for alcohol-induced and biliary pancreatitis, and the total score cannot be calculated unless all factors are measured after 48 hours of observation. The most important roles of the Ranson and Glasgow scoring may be to exclude severe disease. A Glasgow or Ranson score of 0 or 1 virtually guarantees that complications will not develop and that mortality will be negligible. A second important use of these scoring systems is for clinical research, in characterizing disease severity for comparison between studies.

VARIABLES OF THE RANSON CRITERIA AND MODIFIED GLASGOW SYSTEM

Ranson Criteria

For Acute Non-Gallstone Pancreatitis

Upon admission:

1. Age >55 years
2. WBC >16,000/mm³
3. Glucose >200 mg/dL
4. LDH >350 IU/L
5. AST >250 IU/L

Within 48 hours:

1. Drop in HCT >10%
2. Serum Ca <8 mg/dL
3. Base deficit >4 mEq/L
4. Increase BUN >5 mg/dL
5. Fluid deficit >6 L
6. Arterial PO₂ <60 mmHg

For Acute Gallstone Pancreatitis

Upon admission:

1. Age >70 years
2. WBC >18,000/mm³
3. Glucose >220 mg/dL
4. LDH >400 IU/L
5. AST >440 IU/L

Within 48 hours:

1. Drop in HCT >10%
2. Serum Ca <8 mg/dL
3. Base deficit >5 mEq/L
4. Increase BUN >2 mg/dL
5. Fluid deficit >6 L
6. Arterial PO₂ <60 mmHg

Modified Glasgow System

- Arterial PO₂ <60 mmHg
- Serum albumin <3.2 g/dL
- Serum Ca <8 mg/dL
- WBC >15,000/mm³
- AST >200 IU/L
- LDH >600 IU/L
- Glucose >180 mg/dL
- BUN >45 mg/dL

CT has also become routinely used in the prediction and determination of disease severity. The initial CT grading system, which did not require intravenous contrast administration, was developed by Balthazar and Ranson. However, using CT alone also has a relatively high false-positive rate (i.e., many patients with grade C and even D pancreatitis recover without

developing organ failure or dying). Combining the CT grading system with Ranson prognostic signs further improves the prognostic capacity when compared to either system alone. Patients with grade D or E are almost certain to develop complications, and they have a significantly increased risk of mortality, and this risk is augmented by the coexistence of a high Ranson score. Those patients with grade C pancreatitis and a Ranson score less than 3 routinely do well, whereas those with grade C pancreatitis and a Ranson score more than 3 are much more likely to develop complications and/or die. A grade of A or B strongly predicts an uncomplicated outcome^{17,36}. These grading systems are based on non-CECT scans. CECT can also be used to determine the presence of pancreatic necrosis. Interstitial pancreatitis (the absence of necrosis) is defined by homogeneous and uniform intravenous contrast enhancement of the pancreas, which requires rapid scanning over the pancreas timed to the infusion of intravenous contrast. Necrosis is defined by inhomogeneous enhancement with intravenous contrast, especially when large areas of the pancreas are entirely devoid of enhancement.

Pancreatic necrosis per se is not always associated with other clinical features of severe disease (e.g., organ failure or infected necrosis), but the presence of necrosis markedly increases the chance of developing these severe clinical markers. Particularly, pancreatic necrosis puts patients at risk for

infection of the devitalized tissue, one of the most severe complications of AP.

CT scans with intravenous contrast enhancement is our only method currently available to identify necrosis.

Given that the multiple factor scoring systems are complex and that CT scans are expensive, there has been continued interest in identifying simpler or less expensive methods to predict severity. Several clinical and serum markers of disease severity have been proposed, which include routine laboratory tests and novel markers of disease severity. Despite the diagnostic importance of elevated serum amylase and lipase in AP, numerous studies have demonstrated that elevated levels of these enzymes have no prognostic value in AP. This is the reason why they are excluded in any AP severity scoring system. Hemoconcentration more than 44% at presentation has been demonstrated by several investigators to be a reasonably accurate early marker that predicts pancreatic necrosis and organ failure³⁷⁻³⁹. In contrast, Whitcomb et al. showed that an admission hematocrit of 40% or below predicts a low risk of pancreatic necrosis and may reduce the need for diagnostic CT scans.

Computed Tomography Grading System

Grade A: Normal findings

Grade B: Focal or diffuse pancreatic enlargement

Grade C: Inflammation of the pancreas and pancreatic fat

Grade D: Peripancreatic fluid collection in single location (within anterior para-renal space)

Grade E: Two or more fluid collections or the presence of peripancreatic gas

More novel serum tests have also been evaluated. C-reactive protein (an acute-phase reactant) is cheap, widely available, and commonly used in Europe as a measure of severity. A level of 150 mg/L of C-reactive protein has been proposed as a criterion for distinguishing mild AP from SAP25. Other markers, such as trypsinogen activation peptide, interleukin-6, and polymorphonuclear elastase, have been shown in research studies to be of value to predict severe necrotizing pancreatitis, but commercial assays are not yet available for clinical use. Clinical or demographic features may also predict disease severity. Obesity has been shown in several studies to be a risk factor for the severe outcome of AP, and it is associated

with an increased risk of mortality²⁵. Advanced age and comorbid diseases are also risk factors for morbidity and mortality from AP. Other clinical parameters like hypovolemic shock, massive pleural effusion, prolonged hypoxia, and body echymosis are indicative of a complicated course and a higher risk of mortality.

Many steps have already been predicting the severity of AP. The ability to accurately predict outcome would allow the improved use of intensive and intermediate care unit beds and would allow specific therapy (once available) to be directed at those patients most likely to benefit. However, the ideal grading system or the predictive marker of choice does not yet exist. Careful and repeated clinical evaluation by skillful clinicians remains an important part of detecting complications early.

Multiple factor scoring systems are useful adjuncts but remain complex, difficult to use, and all have a high false-positive rate. CT scans are widely used and seem to provide the best addition to clinical assessment, both to confirm the diagnosis and/or rule out alternative diagnoses and estimate the disease severity.

BISAP SCORE

The ability to stratify patients early in their course is a major step to improving future management strategies in acute pancreatitis. The Ranson and modified Glasgow score contain data not routinely collected at the time of hospitalization. In addition both require 48hr to complete, missing a potentially valuable early therapeutic window. APACHE II was originally developed as an intensive care instrument and requires the collection of a large number of parameters, some of which may not be relevant to prognosis in acute pancreatitis.

“B U Wu et al7, using classification and regression tree (CART) analysis, a clinical scoring system was developed for prediction of in hospital mortality in acute pancreatitis. The scoring system was derived on data collected from 17,992 cases of acute pancreatitis from 212 hospitals in 2000-2001. The BISAP scoring system was validated on data collected from 18,256 acute pancreatitis cases from 177 hospitals in 2004-2005. The accuracy of the BISAP scoring system for prediction of mortality was measured by the area under the receiver operating characteristic curve (AUC). The performance of the new scoring system was further validated by comparing its predictive accuracy with that of APACHE II. A new mortality based prognostic scoring system for use in acute pancreatitis has been derived and validated. BISAP is a

simple and accurate method for the early identification of patients at increased risk for in-hospital mortality.”

“Vikesh K. Singh et al⁹, BISAP score was evaluated among 397 consecutive cases of acute pancreatitis admitted to their institution between June 2005 and December 2007. BISAP scores were calculated on all cases using data within 24h of presentation. The ability of the BISAP score to predict mortality was evaluated using trend and discrimination analysis. The optimal cutoff score for mortality from the receiver operating curve was used to evaluate the development of organ failure, persistent organ failure, and pancreatic necrosis. Among 397 cases, there were 14(3.5%) deaths. There was a statistically significant trend for increasing mortality ($p<0.0001$) with increasing BISAP score. The area under the receiver operating curve for mortality by BISAP score in the prospective cohort was 0.82(95% confidence interval: 0.70, 0.95), which was similar to that of the presentation validation cohort by B U Wu¹⁰. BISAP score more or equal to 3 was associated with an increased risk of developing organ failure (odds ratio=7.4, 95% confidence interval: 2.8, 19.5), persistent organ failure (odds ratio=12.7, 95% confidence interval: 4.7, 33.9) and pancreatic necrosis (odds ratio=3.8, confidence interval: 1.8, 8.5). Thus the BISAP score represents a simple way to identify patients at risk of increased mortality and the development of intermediate markers of severity within 24 hrs of presentation”

“Individual components of the BISAP scoring system

- “BUN > 25 mg/dl
- Impaired mental status (Glasgow Coma Scale Score < 15)
- SIRS is defined as two or more of the following:
 - (1) Temperature of < 36 or > 38 °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/mm³ or > 10% immature bands
- Age > 60 years
- Pleural effusion detected on imaging”

One point is assigned for each variable within 24 hrs of presentation

BISAP, bedside index for severity in acute pancreatitis; SIRS, systemic inflammatory response syndrome.

MATERILAS & METHODS

METHODS OF COLLECTION OF DATA:

All patients who present at Rajiv Gandhi government general hospital , diagnosed as acute pancreatitis

Acute pancreatitis was defined as 2 or more of the following

- Characteristic abdominal pain
- Increased levels of Serum amylase and/or lipase 3 times the normal value
- Ultrasonography of the abdomen within first 7 days of hospitalization demonstrating changes consistent with acute pancreatitis

BISAP score & Ranson's score are calculated in all such patients based on data obtained within 48 hours of hospitalisation

A CT or MRI or USG of the abdomen, obtained at any time in the first 7 days of hospitalization, was required to differentiate necrotizing from interstitial pancreatitis. Organ failure was defined as a score of ≥ 2 in one or more of the three (respiratory, renal and cardiovascular) out of the five organ systems initially described in the Marshall score. Organ failure scores were calculated for all patients during the first 72 hours of hospitalization based on the most extreme laboratory value or clinical measurement during each 24h

period. Duration of organ failure is defined as transient (≤ 48 h) or persistent (≥ 48 h) from the time of presentation

Criteria for organ failure based on Marshall scoring system

Organ system			Score		
	0	1	2	3	4
Respiratory (PaO ₂ / FiO ₂)	>400	301- 400	201- 300	101- 200	<101
Renal(serum creatinine, mg/dl)	<1.5	>1.5 to <1.9	>1.9 to <3.5	>3.5 to <5.0	>5.0
Cardiovascular(SBP, mm hg)	>90	<90, fluid responsive	<90, fluid unresponsive	<90, pH<7.3	<90, pH<7.2

ATLANTA CLASSIFICATION :

Acute pancreatitis is an acute inflammatory process of the pancreas with variable involvement of other tissues or remote organ systems.

- *Mild acute pancreatitis* is associated with minimal organ dysfunction and an uneventful recovery.
- *Severe acute pancreatitis* is associated with distant organ failure and/or local complications such as necrosis, abscess, or

pseudocyst.

- *Acute fluid collections* occur early in the course of acute pancreatitis, are located in proximity to the pancreas, and always lack a wall of granulation/fibrous tissue.
- *Pancreatic necrosis* is a diffuse or focal area(s) of nonviable pancreatic parenchyma, which is typically associated with peripancreatic fat necrosis.
- *Pancreatic abscess* is a circumscribed intra-abdominal collection of pus, usually near the pancreas, containing little or no pancreatic necrosis, that arises as a consequence of acute pancreatitis or pancreatic trauma.
- *Acute pseudocyst* is a collection of pancreatic fluid enclosed by a wall of fibrous or granulation tissue, which arises as a consequence of acute pancreatitis, pancreatic trauma, or chronic pancreatitis.

ATLANTA REVISION

Mild acute pancreatitis	Absence of organ failure Absence of local complications
Moderately severe acute pancreatitis	1. Local complications AND/OR 2. Transient organ failure (<48 hrs)
Severe acute pancreatitis	Persistent organ failure > 48 hrs

Source of data

Patients admitted to surgical wards in RGGGH, Chennai

Method of collection of data

Prospective and retro prospective study was conducted on patients admitted with acute pancreatitis during the study period from November 2013 to September 2014. All the patients were subjected to detailed clinical examination, laboratory investigations and radiological imaging with their consent.

Inclusion criteria

Patients with history and clinical findings suggestive of acute pancreatitis with evidence of bulky edematous pancreas on USG/CT abdomen.

Exclusion criteria

Chronic pancreatitis

Acute on chronic pancreatitis

Sample size

After considering both inclusion and exclusion criteria , total number of patients included in the study were 60.

All the 60 patients were subjected to both BISAP and Ranson's 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 revised Atlanta criteria and persistent organ failure graded by Modified Marshall scoring system is used to assess both scores' reliability in predicting organ failure.

Method of statistical analysis

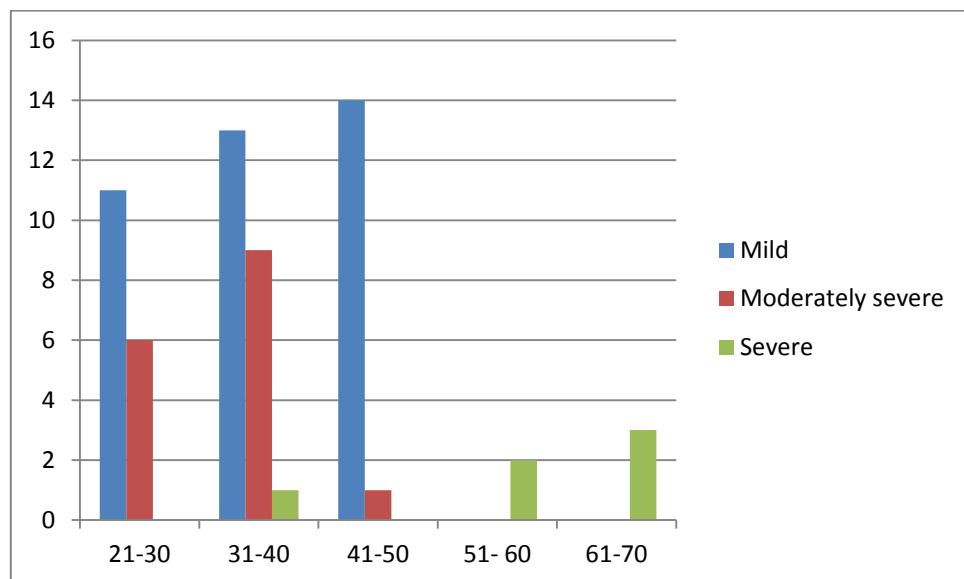
Independent t test was used to examine differences in age; fischer's exact test for sex; and chi square test for etiology were used. Sensitivity, specificity, positive predictive value, negative predictive value and accuracy were calculated. A "p" value of less than 0.05 was considered to be statistically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

OBSERVATION AND RESULTS

- This study was conducted in Rajiv Gandhi government general hospital Chennai from august 2013 to august 2014
- Total number of patients studied were 60
- According to Altanta Revised criteria, 30 patients had mild pancreatitis, 20 patients had moderately severe pancreatitis, 10 patients had severe pancreatitis.
- Of the 60 patients, 37 patients had ranson's score less than or equal to 3. 23 patients had a score of more than 3.
- Of the 60 patients, 39 patients had a BISAP score less than or equal to 3, 21 patients had a score more than 3.

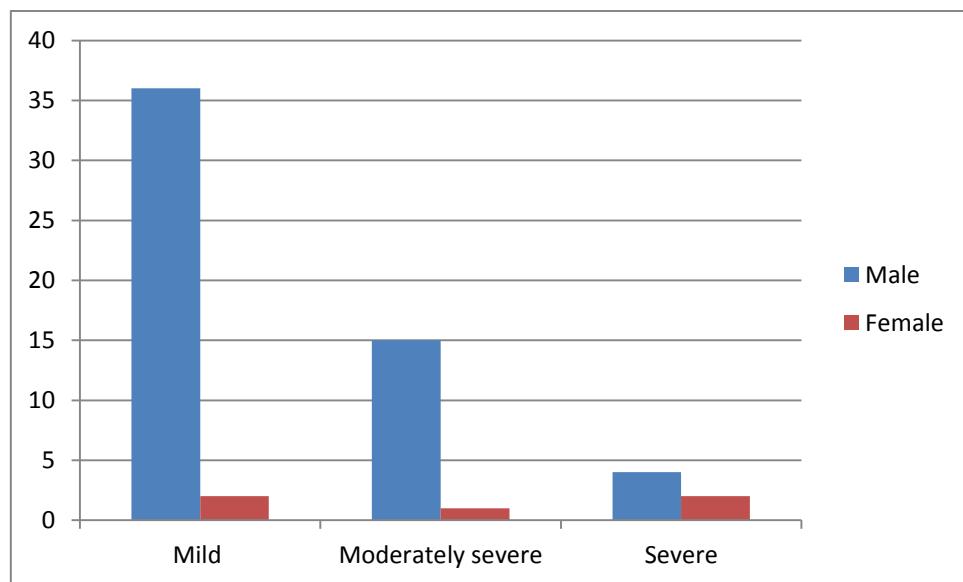
Age distribution of the study population

Age (Years)	No. of patients			
	Mild	Moderately severe	Severe	Total
21-30	11	6	0	17
31-40	13	9	1	23
41-50	14	1	0	15
51- 60	0	0	2	2
61-70	0	0	3	3



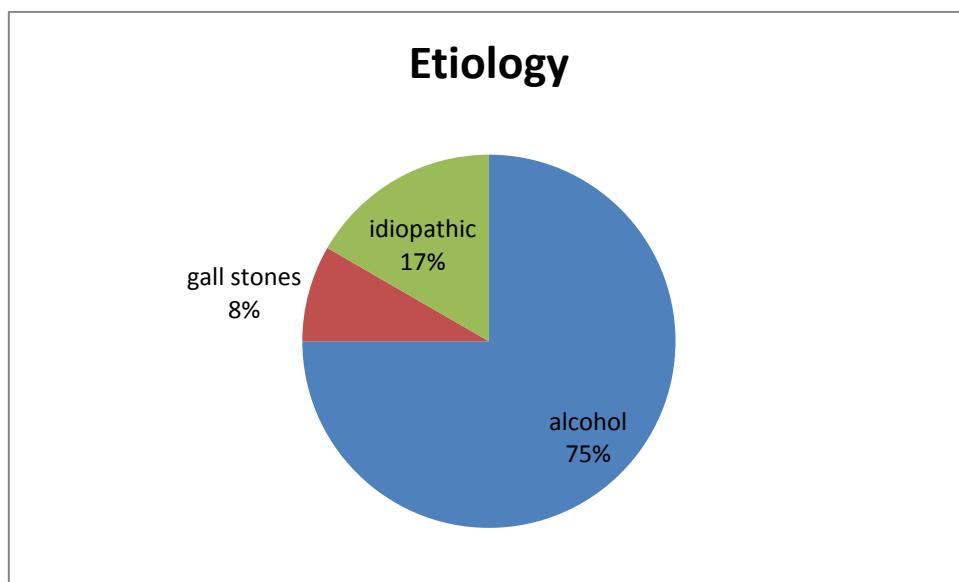
Sex distribution of the study population

Sex	Mild	Moderately severe	Severe	Total
Male	36	15	4	55
Female	2	1	2	5



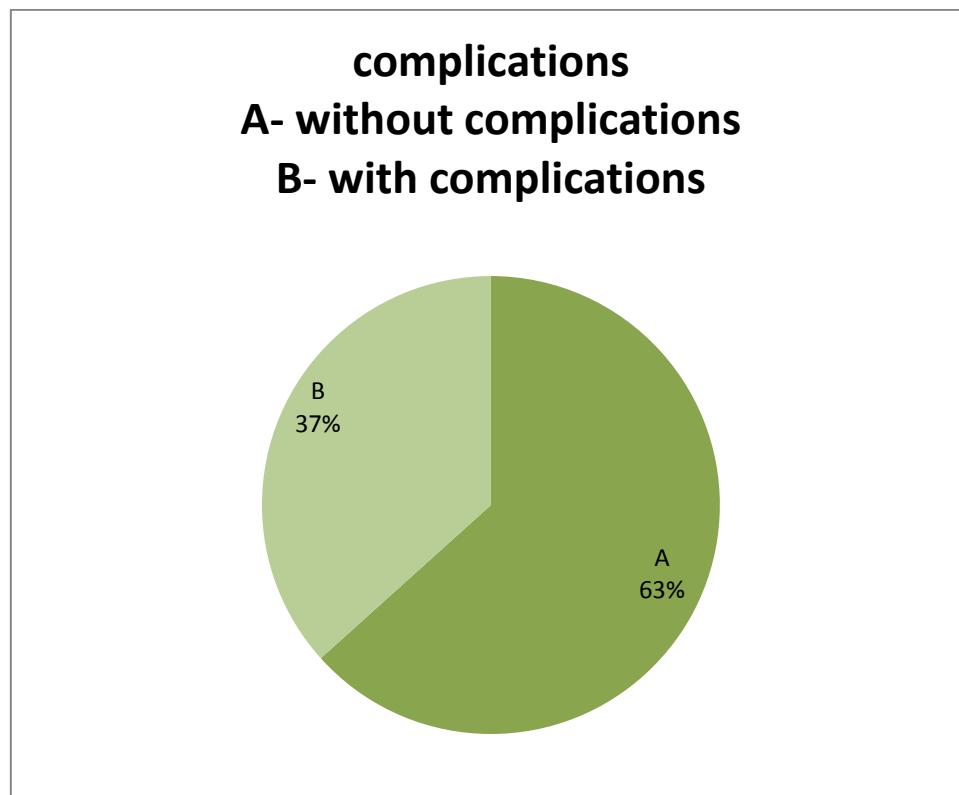
Etiology of acute pancreatitis

Etiology	Mild	Moderately severe	Severe	Total
Alcohol	28	13	4	45
Gall stones	3	2	0	5
Idiopathic	7	1	2	10



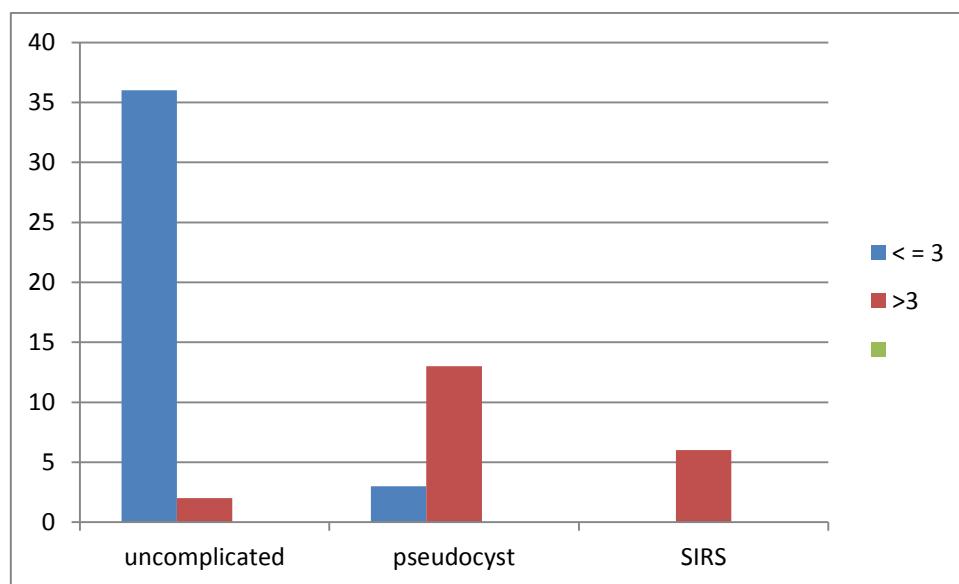
Outcome of patients

No. of patients without complications	No. of patients with complications	Complications			
		Local complications			Systemic complications
		Pseudocyst	Pancreatic necrosis	Haemorrhagic pancreatitis	SIRS
38	22	16	0	0	6



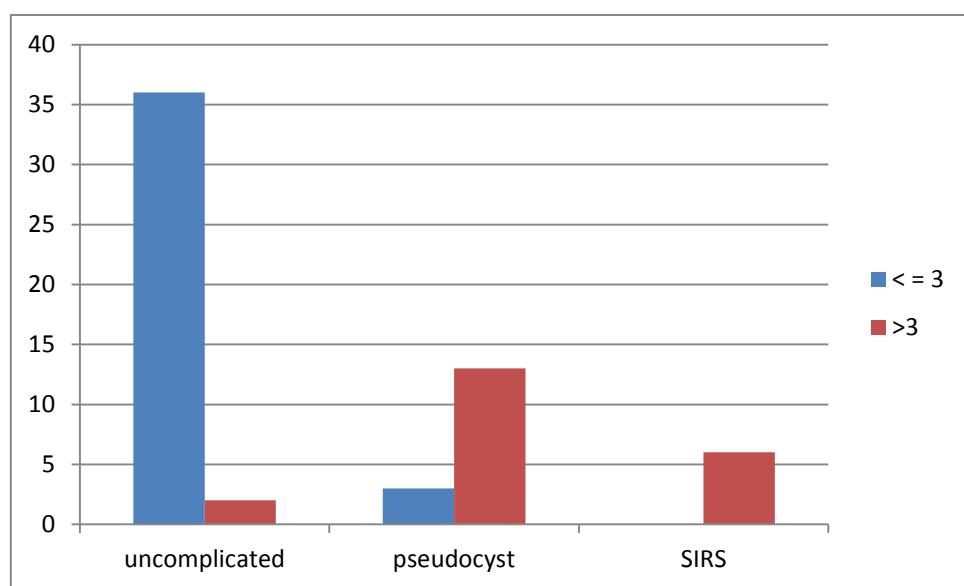
Outcome of patients based on different cut off Ranson's score

Ranson's score	Uncomplicated outcome	Local complications			Systemic complications
		Pseudocyst	Pancreatic necrosis	Haemorrhagic pancreatitis	
≤ 3	36	1	0	0	0
>3	2	11	0	0	3
> 5	0	4	0	0	3

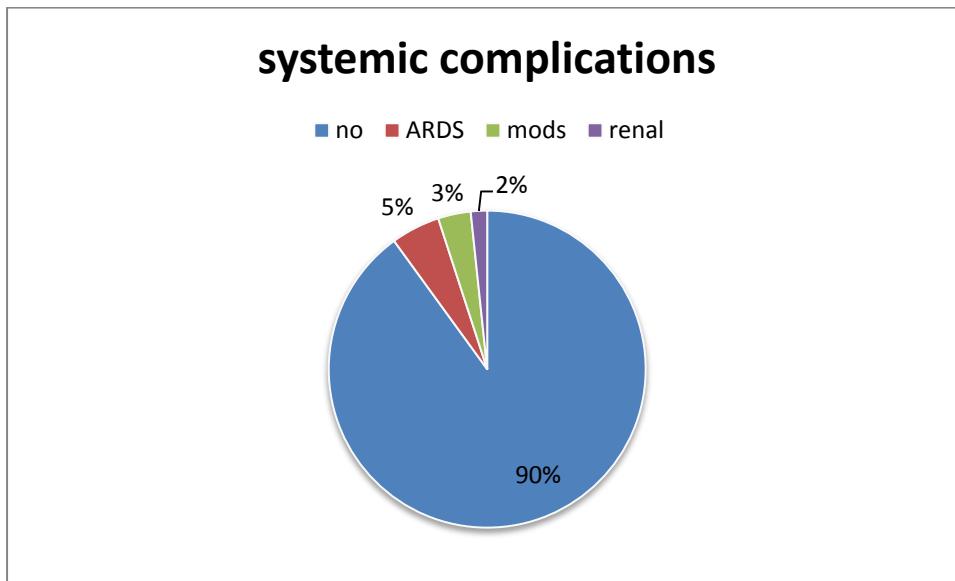


Outcome of patients based on different cut off BISAP score

BISAP score	Uncomplicated outcome	Local complications			Systemic complications
		Pseudocyst	Pancreatic necrosis	Haemorrhagic pancreatitis	
≤ 3	36	3	0	0	0
>3	2	13	0	0	6



Out of 60 patients, 10 % of patients developed organ failure. Organ failure may be transient or persistent. 3 patients had transient organ failure. 3 patients developed persistent organ failure.



ARDS – 5 %

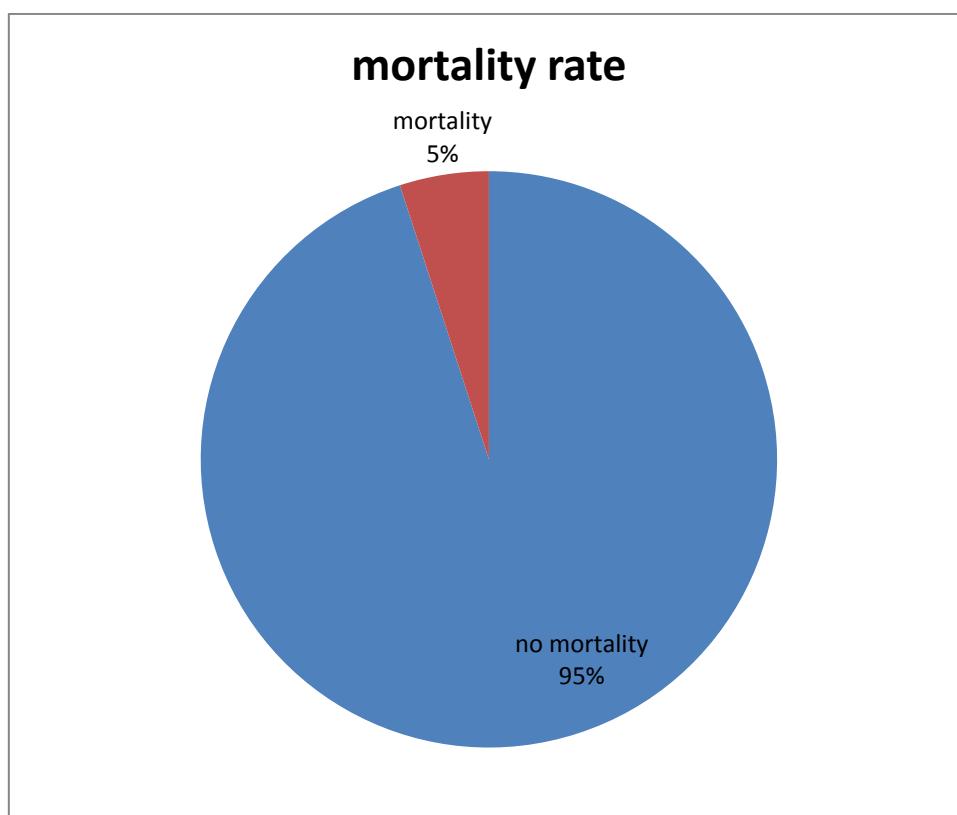
MODS – 3 %

Renal failure – 2 %

Transient organ failure developed in 3 patients

Persistent organ failure developed in 3 patients who died.

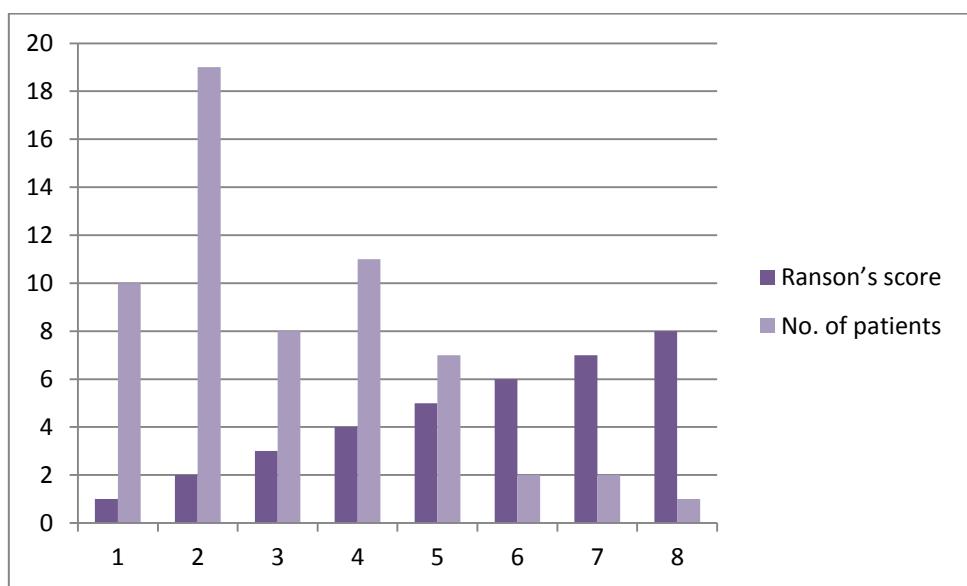
Among 60 patients, 3 patients died



Mortality rate is 5%

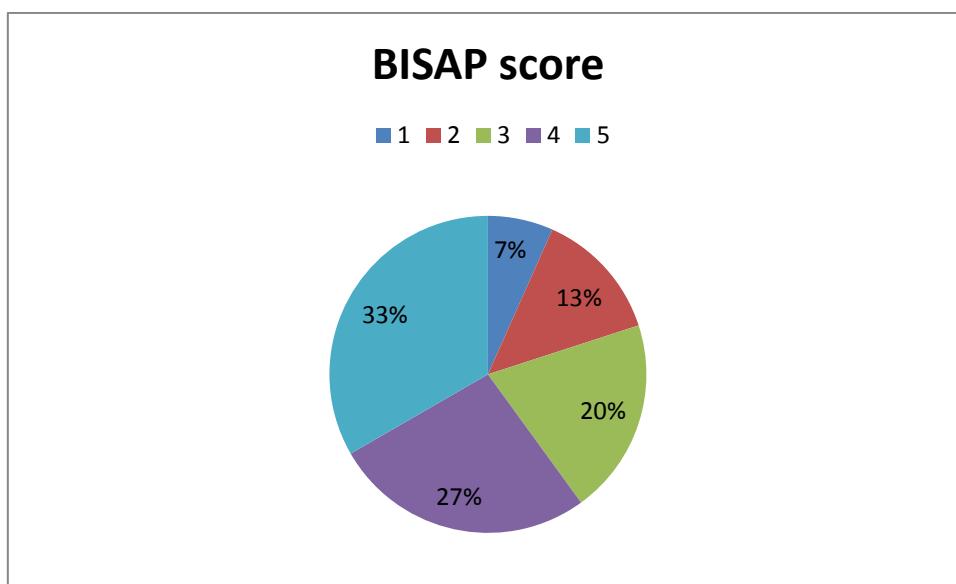
Ranson's score

Ranson's score	No. of patients
1	10
2	19
3	8
4	11
5	7
6	2
7	2
8	1



Mean of ranson's score – 3.08

BISAP score	No. of patients
1	10
2	20
3	9
4	19
5	2



Mean of BISAP score among 60 patients is 2.7

Prediction of severity by Ranson's score

Ranson's score	Sensitivity	Specificity	Positive predictive value	Negative predictive value	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

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

Ranson's score of greater than or equal to 3 predicted more number of severe attacks (100%) but less number of mild attacks (56%) with a 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 attacks (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 BISAP score

BISAP score	Sensitivity	Specificity	Positive predictive value	Negative predictive value	Accuracy
<=3	93.33	96	93.33	96	95
> 3	86.66	100	100	92.6	95

BISAP score of less than or equal to 3 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.

BISAP score of less than or equal to 3 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

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 BISAP score

BISAP score	sensitivity	specificity	PPV	NPV	Accuracy
Pancreatic collection	93.33	64.1	93.33	96	95
Major organ failure	100	64.1	6.66	100	65

BISAP score was more accurate prediction of systemic complications (100 %)

but less sensitive for prediction of local complications(93.33)

Prediction of severity by Ranson and BISAP scoring systems

	sensitivity	specificity	PPV	NPV	Accuracy
Ranson's score	93.33	96	93.33	96	95
BISAP score	93.33	96	93.33	96	95

As sensitivity , specificity, positive predictive value, negative predictive value and accuracy are found to be the same for Ranson's and BISAP scores, BISAP scoring system is equally efficacious as Ranson scoring system in predicting the severity of acute pancreatitis.

DISCUSSION

The study includes 60 patients with acute pancreatitis.

The majority of patients of acute pancreatitis present with a mild disease, however approximately 20% runs severe course and require appropriate management in an intensive care unit.

Multi-organ dysfunction syndrome, the extent of pancreatic necrosis, infection and sepsis are the major determinants of mortality in acute pancreatitis. Pancreatic necrosis is considered as a potential risk for infection, which represents the primary cause of late mortality. Occurrence of acute respiratory (arf), cardiovascular (cvf) and renal failures (rf) can predict the fatal outcome in sap. A wide range of mortality (20%-60%) has been reported in sap. Early diagnosis and prognostic evaluation are extremely important and may reduce the morbidity and mortality associated with sap.

On account of differences in outcome between patients with mild and severe disease , it is important to define that group of patients who will develop severe pancreatitis, predicting which still represents challenge for the clinician. Interestingly, when seeking medical attention (usually 12 to 24 hours after the onset of pain) most patients do not exhibit multiple organ dysfunction, which is likely to emerge by the second or third day.

"Identification of patients at risk for mortality early in the course of acute pancreatitis is an important step in improving outcome" write dr b u wu , from brighamand womens hospital and harvard medical school in boston , massachusetts, and colleagues, "current methods of risk stratification in acute pancreatitis have important limitation". Most patients of acute pancreatitis recover without complications, the overall mortality rate of this illness is between 2-5%^{2 3}. Multiple risk stratification tools for acute pancreatitis have been developed, but their clinical usefulness is limited. Older measures and modified Glasgow score use data that are not routinely collected at the time of hospitalization . In addition, both require 48hrs, thereby missing potentially valuable early therapeutic window⁴. The APACHE II score is the most widely used prediction system currently but it requires the collection of large number of parameters. APACHE II was originally developed as an intensive care instrument and requires the collection of large number of parameters, some of which may not be relevant to prognosis".

For this purpose a simple and accurate clinical scoring system that is bedside index for severity in acute pancreatitis (BISAP) scoring system was developed. This scoring system used for stratifying patients according to their

risk of hospital mortality and is able to identify patients at increased risk of mortality prior to the onset of organ failure. Data or BISAP score collected within the first 24hr of hospitalization. Ranson score was calculated within 48 hours. The ability to stratify patients early in their course is a major step to improving management strategies in acute pancreatitis. Out of 60 patients, 38 patients had mild pancreatitis (63.33%). Majority of patients, the disease was self-limiting. 22 patients had severe pancreatitis (27.7 %)

Among 60 patients in our study, 55(91%) were males and 5(9%) were females.. However it was found that there was male predominance when stratifying mortality on the basis of sex in severe acute pancreatitis. BISAP score more than 3 was above 40 years of age. With respect to etiological factors of the acute pancreatitis, we found alcohol being the most common cause of acute pancreatitis. The proportion of two main causes greatly depends on the geographical and cultural variations. Alcohol is the main cause in the United States of America and Finland, gallstones in southern Europe , whereas central and northern Europe sees a similar frequency of the two factors or a predominance of alcohol.

In our study, out of 60 patients , 55(90%) had no organ failure,

6(10%) patients developed organ failure. Out of 6 patients 3 (50%) patients had transient organ failure and 3(50%) had persistent organ failure. Mortality was seen in 3 patients, who presented with persistent organ failure. Study was done by vikesh k singh et al, . We found similar results compare to studies done by vikesh k singh et al. According to a recent study, the mortality rates among severe acute pancreatitis patients have decreased from 50-58% in 1978-1982 to 12- 18% in 1993-1997. The overall mortality in our study was 5 % which is similar compared to other studies

Both ranson and BISAP were equal in predicting the severity of acute pancreatitis. Both were equally efficacious in assessing the predictability of organ failure.

Conclusion

From this study, we can conclude that BISAP scoring system is not inferior to Ranson's scoring system in predicting the severity of acute pancreatitis. BISAP scoring system is very simple, cheap, easy to remember and calculate. BISAP scoring system accurately predicts the outcome in patients with acute pancreatitis. Moreover the values in BISAP score are instantaneous and there is no time delay. Ranson's score takes a minimum of 24 hours.

Thus, BISAP score has proved to be a powerful tool in predicting the severity of acute pancreatitis in par with Ranson's score.

Summary

- No. of patients in the study – 60
- Most common age of presentation is 4 th decade of life.
- Males are most commonly affected.
- Alcohol consumption is the most common etiology in our study.
- 38 patients had mild disease
- 22 patients had a complicated course
- 16 patients had moderately severe course
- 6 patients had severe course.
- Most common local complication is pseudocyst.
- Mortality rate in our study is 5 %
- Ranson's score of more than 3 and BISAP score of less than or equal to 3 had the best accuracy of predicting severity of acute pancreatitis.
- Both Ranson's score and BISAP score showed higher sensitivity in prediction of systemic complications than that of local complications.
- No patients were treated surgically.
- Sensitivity , specificity, positive predictive value, negative predictive value and accuracy were 93.33,96,93.33,96 and 95 respectively for both Ranson's score and BISAP scoring system.

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ABBREVIATIONS

ABBREVIATION	EXPANSION
ICU	INTENSIVE CARE UNIT
MODS	MULTIORGAN DYSFUNCTION SYNDROME
SIRS	SYSTEMIC INFLAMMATORY RESPONSE SYNDROME
CRP	C-REACTIVE PROTEIN
ERCP	ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY
ARDS	ACUTE RESPIRATORY DISTRESS SYNDROME

APACHE	Acute physiology and chronic health evaluation
BISAP	BED SIDE INDEX FOR SEVERITY OF ACUTE PANCREATITIS
CSI	CT SEVERITY INDEX
TPN	TOTAL PARENTRAL NUTRITION
AP	ACUTE PANCREATITIS
LDH	LACTATE DEHYDROGENASE
AST	ASPARTATE TRANSAMINASE
BUN	BLOOD UREA NITROGEN
AUC	AREA UNDER CURVE

PROFORMA**I. PARTICULARS OF THE PATIENT:**

IP No:

Name:

Age:

Sex:

Address:

Socio - economic status:

Date of Admission:

Date of Discharge:

Positive history:

Positive clinical findings:

Final Diagnosis:

USG abdomen:

BISAP score - total score-

CRITERIA	YES	NO
<ul style="list-style-type: none"> • “BUN > 25 mg/dl 		
<ul style="list-style-type: none"> • SIRS • (1) Temp of < 36 or > 38 ° C • (2) Resp rate > 20 /min or PaCO₂ < 32 mm Hg • (3) Pulse > 90 beats/min • (4) WBC < 4,000 or >12,000 cells/mm³ or >10% immature bands 		
Impaired mental status (GCS Score < 15)		
SIRS		
Age.60 years		
Pleural effusion		

Ranson score – total score

Criteria	Yes	No
Upon admission:		
1. Age >55 years		
2. WBC >16,000/mm ³		

3. Glucose >200 mg/dL 4. LDH >350 IU/L 5. AST >250 IU/L		
Within 48 hours: 1. Drop in HCT >10% 2. Serum Ca <8 mg/dL 3. Base deficit >4 mEq/L 4. Increase BUN >5 mg/dL 5. Fluid deficit >6 L 6. Arterial PO ₂ <60 mmHg		

CT abdomen:

Severity based on Atlanta criteria – Mild/ Moderately severe/Severe:

Organ complications:

Any operative procedure:

Condition on discharge:

Follow up:

Comments and summary:

Originality

0

GradeMark

0

PeerMark

acute pancreatitis

BY 221211002-MS GENERAL SURGERY ARUNKUMAR.R

21%
SIMILAR--
OUT OF 0

Introduction

Acute pancreatitis is a common entity encountered during routine surgical practice and it poses a great challenge to the treating surgeon. "Acute pancreatitis is defined as a pancreatic inflammatory process, with peripancreatic and multi-organ involvement causing multi-organ dysfunction syndrome (MODS), with increased mortality rate". Following statement grossly summarises its consequences.

" Acute pancreatitis is the most terrible of all calamities that occur in

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INSTITUTIONAL ETHICS COMMITTEE
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CERTIFICATE OF APPROVAL

To

Dr.R.Arunkumar,
Postgraduate MS (General Surgery),
Institute of General Surgery,
Madras Medical College,
Chennai – 600 003.

Dr.R.Arunkumar,

The Institutional Ethics Committee has considered your request and approved your study titled "**Comparative study between BISAP score and RANSONS score in predicting severity of acute pancreatitis**" No.21072014.

The following members of Ethics Committee were present in the meeting held on 01.07.2014 conducted at Madras Medical College, Chennai-3.

1. Dr.C.Rajendran, M.D.,	: Chairperson
2. Dr.R.Vimala, M.D., Dean, MMC, Ch-3	: Deputy Chairperson
3. Prof.B.Kalaiselvi, M.D., Vice-Principal, MMC, Ch-3	: Member Secretary
4. Prof.R.Nandhini, M.D., Inst.of Pharmacology, MMC	: Member
5. Dr.G.Muralidharan, Director Incharge, Inst.of Surgery	: Member
6. Prof.Md.Ali, M.D., D.M., Prof & HOD of MGE, MMC	: Member
7. Prof.K.Ramadevi, Director i/c, Inst.of Biochemistry, MMC	: Member
8. Prof.Saraswathy, M.D., Director, Pathology, MMC, Ch-3	: Member
9. Prof.Tito, M.D., Director i/c, Inst.of Internal Medicine, MMC	: Member
10.Thiru S.Rameshkumar, Administrative Officer	: Lay Person
11.Thiru S.Govindasamy, B.A., B.L.,	: Lawyer
12.Tmt.Arnold Saulina, M.A., MSW.,	: Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.


Member Secretary, Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003

S NO	Name	Age/Sex	IPNO	DOA	DOD/ Death	BUN(mg/dl)	GCS	SIRS	Age≥60	Pedal Edema	BISAP score	RANSON's score	Glucose	WBC COUNT	LDH	AST	Drop in HCT(in %)	Serum calcium(mg/dl)	Base deficit(>4meq/l)	Increase in BUN > 5	Fluid deficit > 6l	Arterial po2	Etiology	Severity	Complication
1	Ramu	38/M	75884	02.11.2013	08.11.2014	27	15	no	nil	nil	1	1	221	9943	102	109	2	10.1	A	mild	nil				
2	Rajesh	29/M	78663	12.11.2013	19/11/2014	26	15	no	nil	nil	2	2	165	10895	367	98	3	7.9	2	3	3	99	A	mild	nil
3	Kumar	37/M	79003	16/11/2013	25/11/2013	28	13	yes	nil	yes	4	4	251	16698	390	87	3	10.2	2	2	5	94	A	moderate	pseudocyst
4	Mani	50/M	81557	28/11/2013	12-10-13	30	15	no	nil	nil	2	2	175	9782	134	287	6	7.3	2	3	3	96	A	moderate	pseudocyst
5	Mani	23/M	82344	03.12.2013	09.12.2013	27	14	no	nil	yes	3	3	290	8375	387	89	4	7.9	2	4	4	99	I	mild	nil
6	Guruppa	45/M	82999	05.12.2013	13.12.2013	21	14	no	nil	nil	2	2	280	16890	145	132	8	8.9	3	4	3	99	I	mild	nil
7	Venkatesan	40/M	83455	12-12-13	19/12/2013	23	14	no	nil	nil	2	2	296	9862	176	95	3	7.8	2	3	5	96	I	mild	nil
8	Sivakumar	47/M	83997	15/12/2013	27/12/2013	32	13	yes	nil	yes	4	5	267	16745	189	300	5	7.2	3	3	3	95	A	moderate	pseudocyst
9	Dhayalan	35/M	84540	23/12/2013	30/12/2013	27	14	no	nil	nil	2	2	168	9925	154	298	6	9.5	5	4	4	99	A	mild	nil
10	Muthukannan	35/M	11158	05.01.2014	13/01/2014	25	13	no	nil	yes	3	3	276	16325	390	89	6	9.4	3	2	6	99	A	mild	nil
11	Muthu	31/M	12134	15/01/2014	27/01/2014	34	13	yes	nil	yes	4	4	289	10362	385	286	6	7	4	3	3	96	G	moderate	pseudocyst
12	Kumar	38/M	12256	21/01/2014	28/01/2013	28	15	no	nil	yes	2	2	342	9624	135	294	8	9.3	3	3	4	99	A	mild	nil
13	Chengaiyah	60/M	13785	28/01/2014	30/01/2014 expired	42	10	yes	yes	yes	5	8	356	17845	394	286	12	6.9	2	4	5	59	A	severe	MODS
14	Sekar	33/M	14645	02-02-14	02-10-14	26	15	no	nil	yes	2	2	298	9024	124	90	4	7.9	3	3	6	99	A	mild	nil
15	Chandran	61/M	14726	09.02.2014	11/02/2014 death	45	11	yes	yes	yes	5	7	198	10834	398	288	11	7	6	6	2	67	I	severe	MODS
16	Muthammal	47/F	14938	10.02.2014	16.02.2014	35	14	yes	nil	yes	4	4	367	16534	399	106	4	7.6	3	3	4	96	I	mild	nil
17	Sambu Singh	24/M	15375	18/02/2014	25.02.2014	27	15	no	nil	yes	2	2	156	16999	143	94	6	7.8	2	3	5	99	A	mild	nil
18	Murugesan	31/M	18472	5.3.2014	12.03.2014	24	15	no	NIL	yes	2	2	298	16453	135	103	7	10.1	3	3	4	99	A	mild	nil
19	Siva	42/M	18923	09.03.2014	16.03.2014	46	13	yes	nil	yes	4	5	292	16896	377	287	8	7.2	2	3	3	99	A	mild	nil
20	Murugan	33/M	19475	18/03/2014	25.03.2014	35	14	no	nil	nil	2	2	303	8278	164	106	6	7.8	2	2	4	96	A	mild	nil
21	Punniya moorthy	28/M	19803	21/03/2014	30/03/2014	28	12	yes	nil	yes	4	5	387	16734	382	300	2	7.2	3	4	5	96	A	moderate	pseudocyst
22	Kumaravel	42/M	21901	28/03/2014	04-04-14	25	15	no	nil	nil	1	1	264	8635	154	96	4	9.3	3	3	4	99	A	mild	nil
23	Jayaraman	45/M	22231	01.04.2014	02/04/2014 death	30	14	yes	nil	yes	4	4	322	9934	324	311	3	7.8	2	4	5	78	A	severe	ARDS

24	Kumar	38/M	22300	02.04.2014	09.04.2014	28	13	no	nil	yes	3	2	298	9452	376	98	8	9.2	3	3	3	99	A	mild	nil
25	Thiyagaraja	29/M	22532	5.04.2014	13.04.2014	36	15	no	nil	nil	1	1	143	9624	200	132	2	7.8	2	2	4	99	A	mild	nil
26	Murali	31/M	22834	08.04.2014	14.04.2014	28	15	no	nil	yes	2	2	300	10452	212	114	4	7.2	3	3	5	96	I	mild	nil
27	Soundaraman	38/M	23001	11.04.2014	17.04.2014	30	15	no	nil	nil	1	1	186	8425	198	111	7	7.6	2	4	4	99	A	mild	nil
28	Perumal	39/M	25983	24/04/2014	30.04.2014	28	15	no	nil	yes	2	2	295	16549	165	107	5	10.3	3	3	2	99	A	mild	nil
29	Kannan	41/M	28542	02.05.2014	09.05.2014	31	13	no	nil	yes	3	3	134	9653	178	300	4	6.9	2	4	4	99	G	mild	nil
30	Rajendran	49/M	31894	09.05.2014	16/05/2014	19	15	no	nil	yes	1	1	285	9563	145	168	6	9.4	2	2	3	99	A	mild	nil
31	Muthukannan	40/M	32168	10.05.2014	20/05/2014	32	13	yes	nil	yes	4	6	326	17345	395	87	11	6.9	5	2	4	95	A	moderate	pseudocyst
32	Stephen	28/M	32983	13.05.2014	20/05/2014	19	15	no	nil	yes	1	1	173	17564	165	46	6	9	3	3	3	96	A	mild	nil
33	Srinivasan	46/M	34167	18/05/2014	27/05/2014	29	13	yes	nil	yes	4	5	352	16429	438	322	8	7.7	3	3	4	97	A	moderate	pseudocyst
34	Velmurugan	27/M	35108	20/05/2014	27/05/2014	32	15	no	nil	nil	1	1	298	9255	185	123	3	8.8	2	2	5	99	A	mild	nil
35	Krishnan	43/M	37934	23/05/2015	30/05/2014	38	13	no	nil	yes	3	3	296	8943	342	102	6	7.1	3	3	3	99	A	mild	nil
36	Kamala	35/F	37948	23/05/2014	30/05/2014	27	15	no	nil	nil	1	1	364	9354	142	124	9	10	2	2	4	99	G	mild	nil
37	Stella	29/F	39980	28/05/2014	06-03-14	30	14	yes	nil	yes	4	5	304	17398	352	294	3	7.4	3	3	5	96	I	moderate	pseudocyst
38	Issakimuthu	39/M	41003	01.06.2014	06-10-14	36	13	yes	nil	yes	4	5	284	18423	342	296	5	6.8	2	4	3	99	A	moderate	pseudocyst
39	Manoharan	50/M	42836	04.06.2014	06-11-14	30	15	no	nil	yes	2	2	276	10253	198	110	4	7.4	3	2	4	99	A	mild	nil
40	Palani	41/M	43346	05.06.2014	06-12-14	28	15	no	nil	yes	2	2	284	9283	202	114	6	7.8	2	3	2	99	A	mild	nil
41	Kamalakkannan	22/M	44749	11.06.2014	20/06/2014	30	12	no	nil	yes	3	4	123	8354	201	116	7	9.5	3	4	5	95	A	moderate	pseudocyst
42	Veni	42/F	49984	13/06/2014	22/06/2014	35	13	yes	nil	yes	4	5	287	16329	400	284	3	7	2	2	4	97	G	moderate	pseudocyst
43	Sulthan anil	60/M	50084	14/06/2014	21/06/2014	30	15	no	nil	yes	2	2	287	9845	176	110	4	7.8	3	4	3	98	A	mild	nil
44	Renganathan	65/M	50523	19/06/2014	26/06/2014	29	14	no	yes	no	2	2	246	16987	168	117	2	9	3	3	5	99	A	mild	nil

45	Sathish	24/M	51673	23/06/2014	30/06/2014	34	13	no	nil	yes	3	3	265	10946	379	286	4	10	3	2	4	96	I	mild	nil
46	Munusamy	36/M	52002	26/06/2014	06-02-14	32	14	no	nil	no	2	2	365	16333	142	98	8	9.6	2	4	3	99	A	mild	nil
47	Kumar	30/M	52999	28/06/2014	07-07-14	28	13	yes	nil	yes	4	7	341	16230	395	274	5	7.5	6	6	5	94	A	moderate	pseudocyst
48	Subburaman	55/M	53102	01.07.2014	13/07/2014	30	14	yes	nil	yes	4	6	300	16999	390	299	7	7.9	3	7	4	76	A	severe	renal failure
49	Babu	38/M	53258	02.07.2014	07-11-14	28	14	no	nil	yes	3	4	322	16984	440	134	5	7.4	2	2	4	95	A	moderate	pseudocyst
50	Ramesh	26/M	53724	05.07.2014	07-12-14	30	15	no	nil	yes	2	2	378	16864	198	142	3	9	3	3	4	99	I	mild	nil
51	Sivagnanam	36/M	54007	08.07.2014	17/07/2014	25	14	yes	nil	yes	4	4	163	9824	415	367	11	6.9	3	2	3	96	A	moderate	pseudocyst
52	Ayyanar	30/M	54389	11.07.2014	17/07/2014	18	15	no	nil	yes	1	1	152	9200	192	98	6	7.8	2	2	5	99	A	mild	nil
53	Radhakrishnan	33/M	56329	24.07.2014	08-06-14	32	12	yes	nil	yes	4	4	355	16437	400	89	5	6.9	2	2	4	85	A	severe	ARDS
54	Babu	25/M	57825	30/07/2014	08-06-14	30	14	no	nil	yes	3	3	306	9674	398	75	3	7.2	3	3	5	96	A	mild	nil
55	Jeyashankar	27/M	58106	02.08.2014	20/08/2014	29	13	yes	nil	yes	4	4	290	16554	193	86	5	7.2	2	2	4	98	A	moderate	pseudocyst
56	Manikandan	30/M	58946	08.08.2014	15/08/2014	21	15	no	nil	yes	2	3	298	16892	182	400	2	10	3	2	4	99	I	mild	nil
57	Vadivel	33/M	59389	12.08.2014	24/08/2014	36	12	yes	nil	yes	4	4	299	16002	387	98	7	7.4	3	2	4	84	A	severe	ARDS
58	Malliga	58/F	59468	13.08.2014	20/08/2014	26	15	no	nil	yes	2	3	322	9456	398	93	6	13	2	4	3	99	G	mild	nil
59	Raja	37/M	59700	15/08/2014	24/08/2014	32	13	yes	nil	yes	4	4	298	16888	402	102	4	7.6	2	3	2	96	A	moderate	pseudocyst
60	Andavar	25/M	60289	18.08.2014	25/08/2014	32	15	no	nil	nil	1	1	146	8756	400	110	3	11	3	4	4	99	A	mild	nil