A DISSERTATION ON

"COMPREHENSIVE ANALYSIS OF ETIOLOGY, PROGNOSIS AND CLINICAL OUTCOME OF ACUTE PANCREATITIS IN A TERTIARY CARE CENTER"

Dissertation Submitted to

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for the Award of the degree

M.S. (General Surgery)

Branch –I

INSTITUTE OF GENERAL SURGERY,

MADRAS MEDICAL COLLEGE, CHENNAI.

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CERTIFICATE

This is to certify that, the dissertation entitled **"COMPREHENSIVE ANALYSIS OF ETIOLOGY, PROGNOSIS AND clinical OUTCOME OF ACUTE PANCREATITIS IN A TERTIARY CARE CENTER"** is the bonafide work done by **Dr.B.GUNASEKARAN** during his **M.S. (General Surgery)** course 2015-2018, done under my supervision and is submitted in partial fulfillment for the requirement of the M.S. (BRANCH-I) - General Surgery, May 2018 examination of The Tamilnadu Dr.M.G.R Medical University.

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DECLARATION

I hereby, declare that this dissertation titled **“COMPREHENSIVE ANALYSIS OF ETIOLOGY, PROGNOSIS AND CLINICAL OUTCOME OF ACUTE PANCREATITIS IN A TERTIARY CARE CENTER”** represents a genuine work of mine. The contributions of any supervisors to the research are consistent with normal supervisory practice, and are acknowledged. I also affirm that this bonafide work or part of this work was not submitted by me or any others for any award, degree or diploma to any other university board, either in India or Abroad. this is submitted to the Tamil Nadu Dr. M.G.R Medical University, Chennai in partial fulfillment of the rules and regulations for the award of master of surgery degree Branch- I (General Surgery).

Date:

Place: **Dr. B. GUNASEKARAN**

ACKNOWLEDGEMENT

As I walk down the memory lane, I realize with a deep sense of humility that what I have done now would not have been possible, but for certain mentors, who have enlightened my path to wisdom.

While I put these words together it is my special privilege and great pleasure to record my deep sense of gratitude to my respected Professor and guide **Prof. R.A.PANDYARAJ M.S.,FRCS.,** but for whose constant guidance, help and encouragement this research work would not have been made possible . The unflinching academic, moral and psychological support will remain ever fresh in my memory for years to come . Words cannot simply express my gratitude to them for imparting me the surgical skills I have acquired.

I would like to express my sincere thanks to **Dr.D.MANIVANNAN,M.S., Dr.D.VINODH,M.S., Dr.G.VIMALA,M.S., Dr.T.PAULIA DEVI M.S.,** Assistant Professors of Surgery, for all of them have given me invaluable advice, guided me and have been most kind and patient to me.

All along the way, I have been supported and encouraged by all my Associate Professors and Assistant Professors who helped me to reach where I am.

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I thank the Dean, MMC & RGGGH for permitting me to conduct this study.

I would be failing in my duty if I do not show my deep sense of gratitude to all the patients who have helped me to become a surgeon and especially those who consented to be part of this study.

With deep reverence, I salute my parents and my family and I thank the almighty for blessing me with a wonderful family to whom I have dedicated this thesis.

DR. B. GUNASEKARAN

LIST OF ABBREVIATIONS USED

MMC	-	Migrating Myoelectric Complex
CCK	-	Cholecystokinin
PP	-	Pancreatic Polypeptide
CECT	-	Contrast enhanced computer tomogram
ERCP	-	Endoscopic retrograde cholangio pancreaticogram
USG	-	Ultrasonogram
TG	-	Triglyceride
TPN	-	Total parenteral nutrition
MMR	-	Mumps Measles Rubella
TB	-	Tuberculosis
CBD	-	Common Bile Duct
MODS	-	Multi organ dysfunction syndrome
ARDS	-	Acute respiratory distress syndrome
TAP	-	Trypsinogen activated peptide
CTSI	-	Computerised tomogram severity index
MRI	-	Magnetic resonance imaging
EUS	-	Endoscopic ultrasonogram
SIRS	-	Systemic inflammatory response syndrome
FNA	-	Fine needle aspiration
APACHE	-	Acute Physiology and Chronic Health Evaluation
BISAP	-	The Bedside Index for Severity in Acute Pancreatitis

TABLE OF CONTENTS

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CERTIFICATE OF APPROVAL

To
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Dear Dr.B.Gunasekaran,

The Institutional Ethics Committee has considered your request and approved your study titled **"COMPREHENSIVE ANALYSIS OF ETIOLOGY, PROGNOSIS AND CLINICAL OUTCOME OF ACUTE PANCREATITIS IN A TERTIARY CARE CENTRE" - NO.23012017 (II).**

The following members of Ethics Committee were present in the meeting hold on **19.01.2017** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2.Dr.M.K.Muralidharan,MS.,M.Ch.,Dean, MMC,Ch-3	:Deputy Chairperson
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12.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
13.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

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

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
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CERTIFICATE

This is to certify that this dissertation work titled **"COMPREHENSIVE ANALYSIS OF ETIOLOGY, PROGNOSIS AND clinical OUTCOME OF ACUTE PANCREATITIS IN A TERTIARY CARE CENTER"** of the candidate **Dr.B.GUNASEKARAN, M.B.B.S.,** with registration Number **221511004** for the award of **MS Branch-I,** in **General Surgery.** I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the upload thesis file contains from introduction to conclusion pages and result shows **1% (One percentage)** of plagiarism in the dissertation.

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INTRODUCTION

Acute pancreatitis is recognized as a difficult area both for the patients and surgeons since the impending threat to the patient if not addressed at time. Several causes have been incriminated for acute pancreatitis, among which alcohol and gall stone disease remain the leading causes.

Acute pancreatitis can be classified as mild and severe. Mild pancreatitis is explained by interstitial edema of the gland and it is usually a self limiting form. Whereas in severe pancreatitis, there is pancreatic necrosis, severe systemic inflammatory response and multi organ failure which may lead to mortality. Hence it is crucial to identify risk stratification tools for the disease, which aids in the management.

Several causes have been incriminated for acute pancreatitis, among which alcohol and gall stone disease remain the leading causes. Though standard text books describes the gall stone disease as the most common cause this study aims to identify the common etiology of the regional population since the alcohol related pancreatitis is on the rise.

Various scoring systems had been developed to define the severity and prognosis of the acute pancreatitis in the past. The earliest of which was developed by Ranson in 1974. It predicts the severity of the disease,

which is based on multiple (11) parameters that are obtained at admission and after 48 hours. Ransons's score has low positive predictive value (50%) and high negative predictive value (90%).

Hence its main use is to rule out acute pancreatitis and also predicts a severe attack. The major disadvantage Ranson's and as well as older Glasgow criteria being, many of the parameters which are components of this scoring, on a routine are not collected at admission,. as six of the parameters are assessed only after 48 hours, it does not predict the severity of the disease at admission.

Hence an early therapeutic window to initiate the appropriate therapy is missed. The APACHE II, which is the common scoring used worldwide, had been originally developed as a risk stratification tool in intensive care setting but it takes into account a huge list of parameters which are not always available at primary centers. Moreover the score may not be related to the severity. So, an accurate, and practically simple bedside scoring system was developed, the BISAP.

BISAP, Bedside index for severity in Acute Pancreatitis, scoring system picks patients with high morbidity and risk of mortality, before organ failure sets in. Details for this scoring is collected within 24 hours of admission, which helps in identifying patients who are at risk of

developing severe disease, and helps in treating the same early and effectively, thereby decreasing the mortality and morbidity.

CECT considered to be the excellent and gold standard imaging modality for diagnosis for diagnosis, establishing the extent of disease process and grading its severity. This can be used as a prognostic indicator as it accurately identifies the pancreatic complications.

AIMS AND OBJECTIVES

Acute pancreatitis is a common cause of morbidity and mortality in the working population of our society. Several etiological agents have been incriminated for acute pancreatitis, among which alcohol and gall stone disease remain the leading causes. Our study would like to identity the most common cause among our population hence it will help the society in developing preventive strategies for the same. Even though there have been several prognostic scoring systems defined for acute pancreatitis, BISAP and modified CTSI remain the reliable clinical and radiological tools, respectively. We would like to correlate these scores with the clinical outcome in our tertiary set-up which may aid to start the early appropriate treatment strategy.

AIMS:

1. To identify most common etiological agent of acute pancreatitis in our institution

2. To correlate the existing clinical (BISAP) and radiological (Modified CTSI) prognostic scoring systems in Acute pancreatitis with the clinical outcomes of patients in our institution.

HISTORY AND REVIEW OF LITERATURE

The pancreas was first discovered by Herophilus, a Greek anatomist and surgeon. The word pancreas was first mentioned in the writings of Eristratos during (310-250B.C). And 400 years later, Rufus, an anatomist / surgeon of Ephesus, gave the name “pancreas”. It is written in Greek language, the word meant “pan: all, kreas: flesh”

In 1642, A German surgeon, Johann Georg Wirsung, discovered the pancreatic duct - “The Duct of Wirsüng” at San Francisco Monastery in Padua.

First landmark paper on Acute Pancreatitis was released on1889 in *Boston Medical and Surgical journal* by Fitz.

Ranson scoring introduced in 1974 for assessing the severity of pancreatitis.

In 1994, CT severity index (CTSI) was developed by Balthazar and his colleagues and in 2004 it is modified by Mortele et al,.

In 2008, the BISAP, Bedside index for severity in Acute Pancreatitis was proposed for the early recognition of patients with high mortality.

ACUTE PANCREATITIS

DEFINITION

Pancreatitis is an “inflammation of glandular parenchyma lead to injury or destruction of acinar components associated with little or no fibrosis of the pancreas”.

Acute pancreatitis is best diagnosed when 2 of the 3 following criteria presents in a patient.

1. Symptoms consistent with pancreatitis

2. Serum lipase or amylase level more than 3 fold rise of the laboratory’s upper range of normal limit

3. Radiologic features suggestive of pancreatitis

The most common cause of acute pancreatitis is gallstones and alcohol. A study done in New Delhi, India, reveals gall stones and alcoholism were identified to be the etiology in 49% and 23.6% cases, respectively.

The remaining 10% form includes large group of other causes of acute pancreatitis. Thus include hypercalcemia, hypertriglyceridemia, drug induced, hereditary causes, sphincter of Oddi dysfunction, pancreatic neoplasms, pancreatic divisum and others.

ETIOLOGY

ALCOHOL

Excessive ethanol consumption is one of the common cause of acute pancreatitis worldwide and it is more prevalent in young men (30 - 45 years of age) than in women. However, only 5% to 10% of patients who drink alcohol develop acute pancreatitis. Heavy alcohol abuse, smoking and genetic predisposition contribute to acute pancreatitis. As compared with non smokers, the relative risk of alcohol induced pancreatitis in smokers is 4.91.

The nature of alcohol consumed is less important than a daily consumption of between 100 to 150 g of ethanol. In a patient with a history consumption on alcohol, with absence of other causes of pancreatitis, the initial attack of acute pancreatitis is thought to be due to alcohol.

The “secretion with blockage” concept reveals that ethanol consumption causes increased tone of sphincter of Oddi. Hence it is a metabolic toxin to pancreatic acinar cells, where it can disrupt enzyme synthesis and secretion. Ethanol causes a brief secretary increase which followed by inhibition. This causes enzyme proteins to precipitate within the duct. Calcium then precipitate within the protein matrix which

resulting in multiple ductal obstructions. Ethanol also increases the ductal permeability.

GALL STONES

The mechanism of gallstone induced pancreatitis is not clear. Bile reflux backinto the pancreatic duct, or ductal obstruction at the level of ampulla due to stone/edema due to passage of stone have been proposed to cause pancreatitis.

An impacted gallstone in the distal bile duct obstructs the pancreatic duct, causing increased pressure within the ductal system, thereby causing damage of acini and ductal epithelial cells.

Acute pancreatitis is most frequently due to gallstones as it causes obstruction of pancreatic ducts. But only 3% to 7% of patients who have gallstones will develop an attack of acute pancreatitis in their lifetime. Gallstone pancreatitis is more common in women than men since gallstones are more frequent in women.

Acute pancreatitis occurs more commonly when a patient develops a stone, less than a diameter of 5mm. The larger stones may not move down the cystic duct to go on to obstruct the pancreatic duct or ampulla. Intermittent and continuous obstruction of the ampullary orifice due to a

gallstone or edema caused by a passing stone is the initiating factor in the pathogenesis of gallstone related pancreatitis.

Causes of Acute Pancreatitis

More Common Causes	*Comments*
Gallstones and microlithiasis	Most common cause
Alcohol abuse	Alcohol-related disease usually occurs only occurs after >5–10 y of heavy drinking
Drugs	More common in older patients, HIV-positive persons, or in those receiving immunomodulating agents
ERCP	Can be a trigger, particularly if performed by an inexperienced clinician or if the patient has sphincter of Oddi dysfunction
Hyperlipidemia	Usually with extremely elevated triglyceride levels (>1000 mg/dL)
Hypercalcemia	Commonly caused by hyperparathyroidism or cancer, can be a trigger by increasing activation of trypsinogen
Genetic	Hereditary, and research has linked gene mutations in cationic trypsinogen (PRSS1), SPINK1, or CFTR genes with acute and chronic pancreatitis
Autoimmune pancreatitis	Diffuse "sausage shaped" finding on imaging with rim enhancement or ductal abnormalities.
Infections	Includes viruses: mumps, coxsackievirus, cytomegalovirus, varicella, HSV, HIV; bacteria: *Mycoplasma, Legionella, Leptospira, Salmonella; Parasites: Toxoplasma, Cryptosporidium, Ascaris*; and fungi: *Aspergillus*
Idiopathic	Accounts for approximately 15%–20% of cases; causes include sphincter of Oddi dysfunction, microlithiasis, and biliary sludge; anatomical abnormalities
Less common causes	
Cystic lesions of the pancreas	More likely if cysts involve the main duct, such as pancreatic intraductal papillary mucinous tumor
Cystic fibrosis	Rare, occurs when some viable pancreatic tissue remains
Pancreas divisum	Controversial as a cause so exclude all other causes first
Pancreatic cancer	Focal pancreatitis can indicate an underlying mass
Penetrating peptic ulcer	Rare, clue is thickening of the duodenal wall
Postsurgical	Such as ischemia related to bypass surgery
Trauma	History is usually compelling
Tropical pancreatitis	Endemic in some parts of Asia and Africa
Vasculitis	Rare even in patients with vasculitis

ERCP = endoscopic retrograde cholangiopancreatography; HSV = herpes simplex virus.

Microlithiasis denotes “aggregates <5 mm in diameter, of cholesterol mon o hydrate crystals or calcium bilirubinate granules detected as “sludge” within the gallbladder” on ultrasonography or on

examination of bile obtained by ERCP. Causative role for microlithiasis in acute pancreatitis remains controversial.

Although, cholecystectomy or endoscopic sphincterotomy will reduce the risk of recurrent attacks of pancreatitis in patients with microlithiasis.

TUMOURS

Neoplasms, by possibly causing obstruction of the pancreatic duct, can cause in repeated episodes of acute pancreatitis. They are common particularly in persons above 40 years of age. The commonest neoplasm which presents like this is intraductal papillary mucinous neoplasm. Acute pancreatitis can be the initial presentation in patients with adenocarcinoma of the pancreas.

Metastases from other cancers like lung and breast to the pancreas have also caused pancreatitis. Sometimes and adenoma from the ampulla can also cause obstruction of the ductal system and subsequent attack of acute pancreatitis.

MEDICATIONS

Drugs are not a very common cause, to note as a important etiology of acute pancreatitis. Drug- induced pancreatitis msy account for <2 % of cases.

The most common agents include sulfonamides, metronidazole, erythromycin, tetracyclines, didanosine, thiazides, furosemide, 3- hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) reductase inhibitors (statins), azathioprine, 6-mercaptopurine, 5-aminosalicylic acid, sulfasalazine, valproic acid, and acetaminophen. More recently, antiretroviral agents used for the treatment of AIDS have been implicated in AP.

Clearly linked to pancreatitis	Weaker association	Questionable association
Azathioprine	Sulfasalazine	Acetaminophen
6-Mercaptopurine	Captopril	Cyclosporin
Trimethoprim-sulfamethoxazole	Alfa-interferon	Cytarabine
Pentamidine	Estrogens	Erythromycin
2',3'-Dideoxyinosine (ddI)	Aminosalicylic acid	Roxithromycin
Asparaginase	Corticosteroids	Ketoprofen
Methyl-dopa	Corticotropin	Metolazone
	Acetaminophen	Octreotide
	Sulindac	
	Tetracycline	
	Metronidazole	
	Thiazide diuretics	
	Furosemide	
	Isotretinoin	
	Valproic acid	

The most common mechanism of drug induced pancreatitis is a “hypersensitivity reaction”. Aminosalicylates, metronidazole, and tetracycline group of drugs, act by this mechanism. This usually occurs between the 4th to 8th week of starting on the drug, and does not depend

on the dose. On challenging the patient with the same drug again, recurrent attack occurs with an early onset, in a few days or even hours of the dose.

The next mechanism is said to be due to accumulation of a products of a drug which is toxic, and typically presents after months. Sodium valproate and didanosine (DDI) falls in this category. Drugs proucing hypertriglyceridemia like thiazide diuretics, tamoxifen, isotretinoin also belong to this category.

Some drugs are intrinsically toxic, and a high dosage of these can result in pancreatitis (Example: erythromycin, paracetamol).

In short, Drug-induced pancreatitis is mild and self limited. The diagnosis should only be considered after excluding alcohol, gallstones, hypertriglyceridemia, hypercalcemia, and neoplasm.

METABOLIC DISORDERS

HYPERCALCEMIA

Very rarely, hypercalcemia of any cause is associated with acute pancreatitis. Possible mechanisms include deposition of calcium salts in the pancreatic ductal system and activation of trypsinogen within the pancreatic parenchyma. Primary hyperparathyroidism attributes to very minimal of, less than 0.5% of all cases of acute pancreatitis. Pancreatitis

can also be due to other causes of hypercalcemia, that include metastatic bone disease, Total parentral nutrition, sarcoidosis, vitamin D overdose, and infusion of calcium in high doses during cardiopulmonary bypass surgery.

HYPERTRIGLYCERIDEMIA

Hypertriglyceridemia is could be the third most common cause of pancreatitis, responsible for 2% to 5% of cases. Serum triglyceride levels more than 1000 mg/dL may result in acute attacks of pancreatitis. But recent studies suggest that the serum Triglycerides need to be even more higher to precipitate acute pancreatitis, that is above 2000 mg/dL. The obvious lactescent serum due to excessive concentrations of chylomicrons. The mechanism of hypertriglyceridemia causing acute pancreatitis is unclear, but the release of free fatty acids by lipase can damage pancreatic acinar cells and endothelial cells. The hydrolysis of Triglycerides by lipase releases free fatty acids that causes free radical damage and can directly injure the cell membranes. Disorders of lipoprotein metabolism are commonly divided into primary (genetic) and secondary causes, including diabetes mellitus, hypothyroidism and metabolic syndrome.

INFECTIONS

The diagnosis of acute pancreatitis caused by an infective agent needs evidence of acute pancreatitis, evidence of an active infection, and the absence of the common cause of acute pancreatitis.

Acute pancreatitis had been associated with

Viruses (mumps, coxsackievirus, hepatitis A, B, C, and herpesviruses, including HSV, CMV, VZV, and EBV), MMR vaccine,

Bacteria (Mycoplasma, Salmonella, Legionella, Leptospira, brucellosis and TB); fungi (Aspergillus, Candida)

Parasites (Toxoplasma, Cryptosporidia, Ascaris lumbricoides, Clonorchis sinensis).

C. sinensis and A. lumbricoides cause pancreatitis by obstructing the main pancreatic duct.

TRAUMA

The penetrating and blunt trauma, both can cause acute pancreatitis. usually other intra abdominal organs are also involved. Laparotomy is compulsory in every case of penetrating injury for the assessment of injuries and to treat them accordingly. In Blunt injuries to

the abdomen pancreatic injury caused by compression of pancreas against the spine.

VASCULAR DISEASES

Ishacemia to the pancreas is rarely related to acute pancreatitis. In most of instances, it is mild but a severe necrotizing pancreatitis may occur. SLE and polyarteritis nodosa can cause vasculitis in pancreas.

Other causes include Atheromatous embolization of cholesterol plaques after trans abdominal angiography, hemorrhagic shock, intra operative hypotension, ergoid overdose and transcatheter arterial embolization for hepatocellular carcinoma.

IATROGENIC

Iatrogenic pancreatitis is commonly due to ERCP, which can cause significant morbidity. Asymptomatic hyperamylasemia occurs after 35% to 70% of ERCPs.

Post-ERCP pancreatitis is thought to be multifactorial, involving a combination of chemical, hydrostatic, enzymatic, mechanical, and thermal factors. Acute pancreatitis occurs in 5% of diagnostic ERCPs, 7% of therapeutic ERCPs, and upto 25% in those with suspected SOD or in those with a history of post-ERCP pancreatitis.

PANCREATIC DIVISUM

Pancreas divisum is the most common congenital anomaly of pancreas, the majority of whom never develop pancreatitis.

Obstruction of the minor papilla is the causative factor in pancreatic divism. Genetic factors may have a role to play in patients suffering from pancreatitis, who have pancreas divisum.

POST-OPERATIVE STRESS

Pancreatitis can be secondary to surgeries of the gastro intestinal tract or thoracic cavity. Pancreatitis occurs after 0.4% to 7.6% of cardiopulmonary bypass surgeries. 27% of patients undergoing cardiovascular surgery gets hyperamylasemia, and 1% develops acute necrotizing pancreatitis. Pancreatitis may occur following liver transplantations. Postoperative pancreatitis is associated with higher morbidity as compared to other causes.

MISCELLANEOUS

Crohn’s disease and Celiac disease has an uncertain association with the development of acute pancreatitis. Hyperamylasemia in these diseases is thought to be due to disruption of small bowel mucosal barrier.

Smoking also suggested to have causative role in acute pancreatitis. Pancreatitis has been seen in cases of severe burns.

Acute pancreatitis resulting from autoimmune disorders is rare, may be seen in type II hypersensitivity disea

CLINICAL FEATURES

Diagnosis of an acute pancreatitis by clinical history and examination is often challenging, as it mimics other causes of acute abdomen.

The diagnosis of acute pancreatitis is based on two or more of the following criteria:

1. Characteristic Severe abdominal pain
2. Serum lipase or amylase level more than 3 fold rise of the laboratory's upper range of normal limit
3. Contrast enhanced computed tomography (CECT) findings consistent with acute pancreatitis.
4. Usually, when the first two criteria are present, and CECT is not required for diagnosis.
5. Other upper abdominal conditions that look similar like acute pancreatitis are perforated peptic ulcer, small bowel gangrene and acute cholecystitis. Since these pathologies often have a fatal

outcome without surgery, quick intervention is needed in the small number of cases in which doubt persists.

DIAGNOSIS

PANCREATIC ENZYMES

The diagnosis of acute pancreatitis made on at least a 3 fold elevation (of upper limit of normal value according the laboratory's reference) of serum amylase or lipase in the blood.

SERUM AMYLASE

Pancreatic disease presents with elevated pancreatic isozyme of amylase, and specifically measuring this isozyme improves the accuracy of diagnosis. But this is not used routinely.

Total amylase is measured routinely since it is cheaper and easier. It increases 6 to 12 hours after the onset of symptoms and persists in blood for about 3-5 days. It is cleared from the blood rapidly with a short half life of 10 hrs.Renal clearance is less than 25 %.

Serum amylase is neither very sensitive nor specific. Sensitivity is about 85%. It may be normal or only mildly elevated in severe pancreatitis, or in chronic pancreatitis because of very little remnant of acinar tissue.

Hypertriglyceridemia induced pancreatitis is associated with normal level of amylase.

Upto 50% of patients with elevated amylase levels may truely have no evidence of pancreatic disease. Elevated amylase levels is suspicious rather than diagnostic of pancreatitis. Hyperamylasemia may be seen in asymptomatic.

SERUM LIPASE

The sensitivity of serum lipase for the diagnosis of acute pancreatitis is like that of serum amylase and is above 85%. But Lipase has higher specificity in diagnosing acute pancreatitis as it is not affected by other causes of hyperamylasemia. Serum lipase level is almost always raised on the first day of onset of the disease, and it remains increased for longer period, thus providing a higher sensitivity. Combining amylase and lipase does not improve diagnostic accuracy and only increases cost.

ROUTINE BLOOD INVESTIGATIONS

The neutrophil count is markedly elevated in severe disease, and is not related to the presence of infection.

The blood glucose also may be high and seen with high levels of serum glucagon.

Liver enymes (AST, ALT and ALP) and bilirubin may also be rised in pancreatitis induced by gallstones.

It should be noted that the decrease in serum calcium seen in patients with acute pancreatitis is mainly due to the decreased serum albumin.

MCV shows some difference in ethanol and non-ethanol related causes of acute pancreatitis. Alcoholic patients used to have higher MCV due to the toxic effects of alcohol on erythrocytosis in the marrow.

IMAGING STUDIES

PLAIN X- RAY ABDOMEN

Plain abdominal radiograph may show no specific finding in mild disease to focal ileus of a segment of small bowel ("sentinel loop"). The "colon cut-off sign is seen in severe disease.

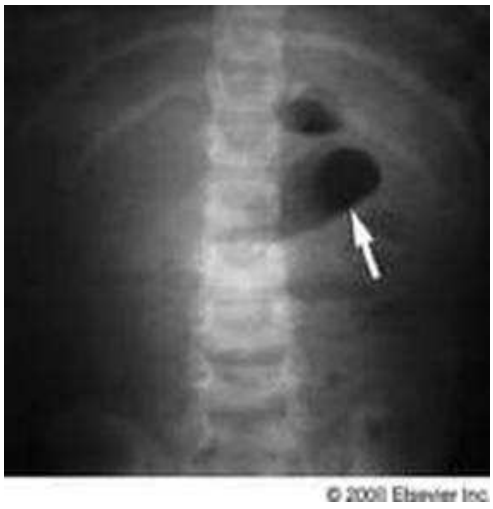


Fig. No.1, Sentinel Loop Sign

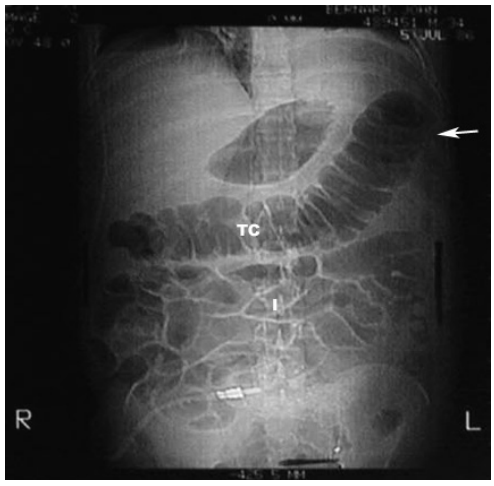


Fig.No.2. Colon cut-off sign

And importantly, X-ray abdomen helps to rule out other acute abdominal pathologies, which may need immediate intervention.

Appearance of dilated loops of bowel depends on the location as well as spread of pancreatic exudates. Gastric abnormalities are due to the exudation in the lesser sac, causing forward displacement of the stomach, with separation of contour of the stomach from transverse colon. Small bowel dilatation is due to inflammation in near the small bowel mesentery, and include ileus of one or more loops of jejunum ("the sentinel loop"), of the distal ileum or cecum. Generalized ileus can occur in severe disease.

Spread of the exudate to specific regions of the colon, may produce spasm of that area of the colon with no air distal to that point ("the colon cut-off sign"), or dilated colon proximal to the spasm.

ULTRASOUND ABDOMEN

Abdominal ultrasound is useful in the initial 24 hours of admission, to identify gallstones, CBD dilatation due to choledocholithiasis, and ascites.

Ascites is commonly seen in patients with moderate to severe pancreatitis, as protein rich fluid extravasates from the intravascular compartment to peritoneal cavity. Pancreas is uniformly enlarged and

hypoechoic, and obscured by bowel gas. Ultrasound is used to serially monitor the size of pseudocyst.

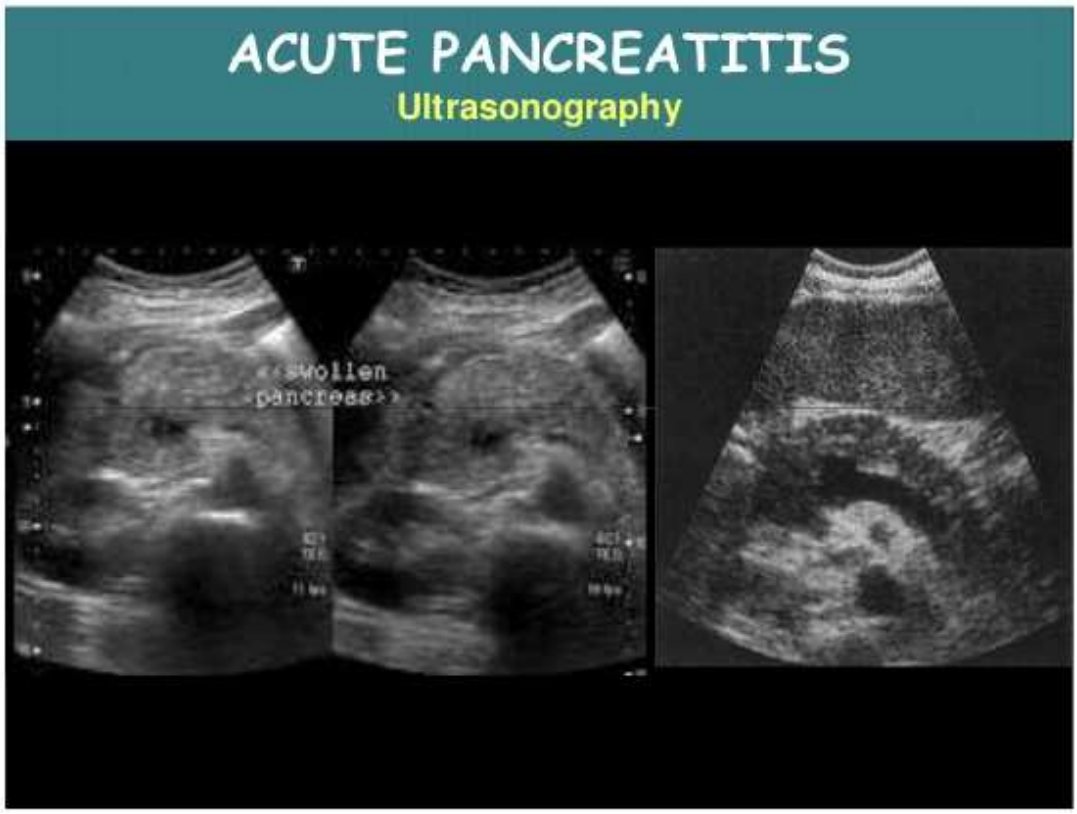


Fig. No.3.USG in Acute Pancreatitis

CECT ABDOMEN

CECT is the most important mode of imaging in diagnosis acute pancreatitis and its intra abdominal complications.

The main indications for CECT in acute pancreatitis are

- ✓ To rule out the other causes of acute abdomen
- ✓ To assess the severity the disease

- ✓ To find the complications of acute pancreatitis

Helical CT is the commonly used imaging technique. CT taken after oral contrast followed by intravenous contrast helps in identifying pancreatic necrosis.

If there is a normal perfusion, it may be due to interstitial pancreatitis and defects in perfusion is due to necrosis of pancreas.

Pancreatic necrosis, may not be seen on CT upto 48-72 hours after the onset of the disease. The presence of air bubbles on CT indicates infected necrosis or pancreatic abscess.

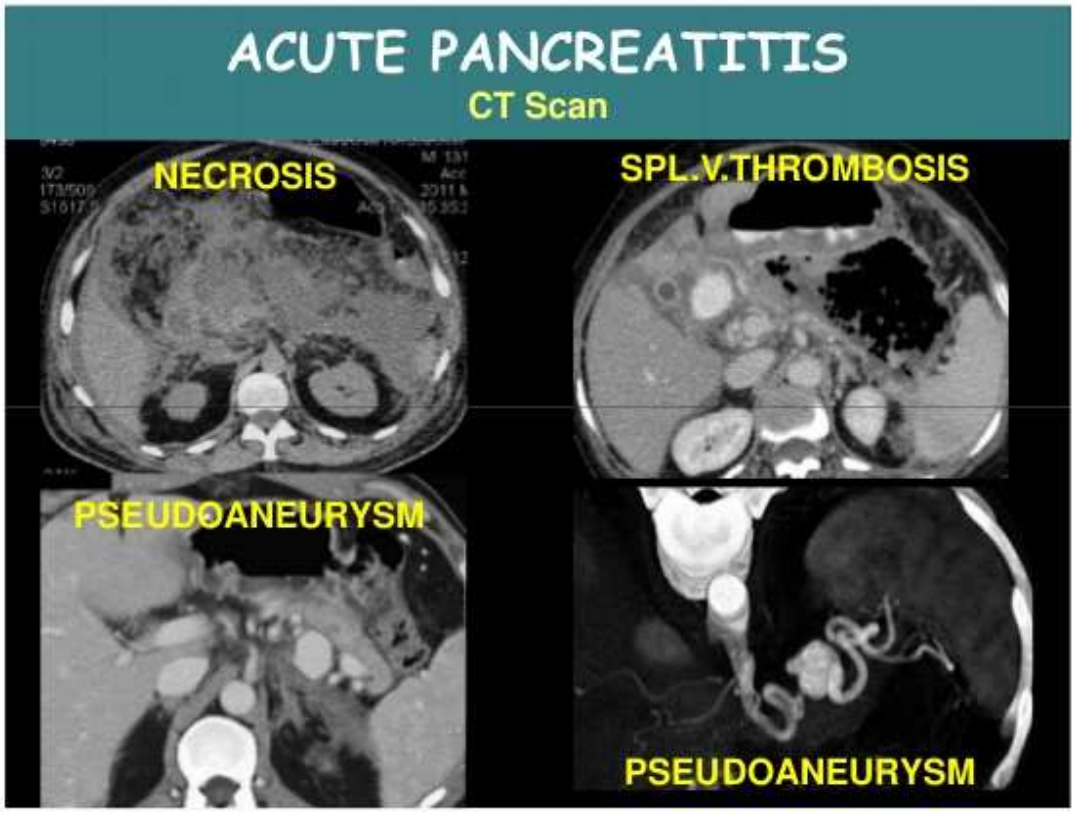


Fig. No. 4. CECT in Acute Pancreatitis

Very early CT may fails to detect an evolving necrosis, which becomes well demarcated after 48-72 hours after the onset of symptoms. CT is not useful in diagnosing necrosis or in predicting the severity within 24 hours of onset of symptoms of illness. CT severity index was developed by Balthazar and colleagues in 1994.

The sensitivity of identifying pancreatic necrosis using CECT scan approaches 100% at 96 hours of diagnosis. CT scans also used as a diagnostic and therapeutic modality in infected pancreatic necrosis. CT guided aspiration of necrosis can be done, when the patient is not improving clinically or in patients who have clinical decline

CT SEVERITY INDEX SCORE:

CT severity index (CTSI) was developed by Balthazar and colleagues in 1994 for distinguishing mild, moderate and severe forms of acute pancreatitis.

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

LIMITATIONS

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

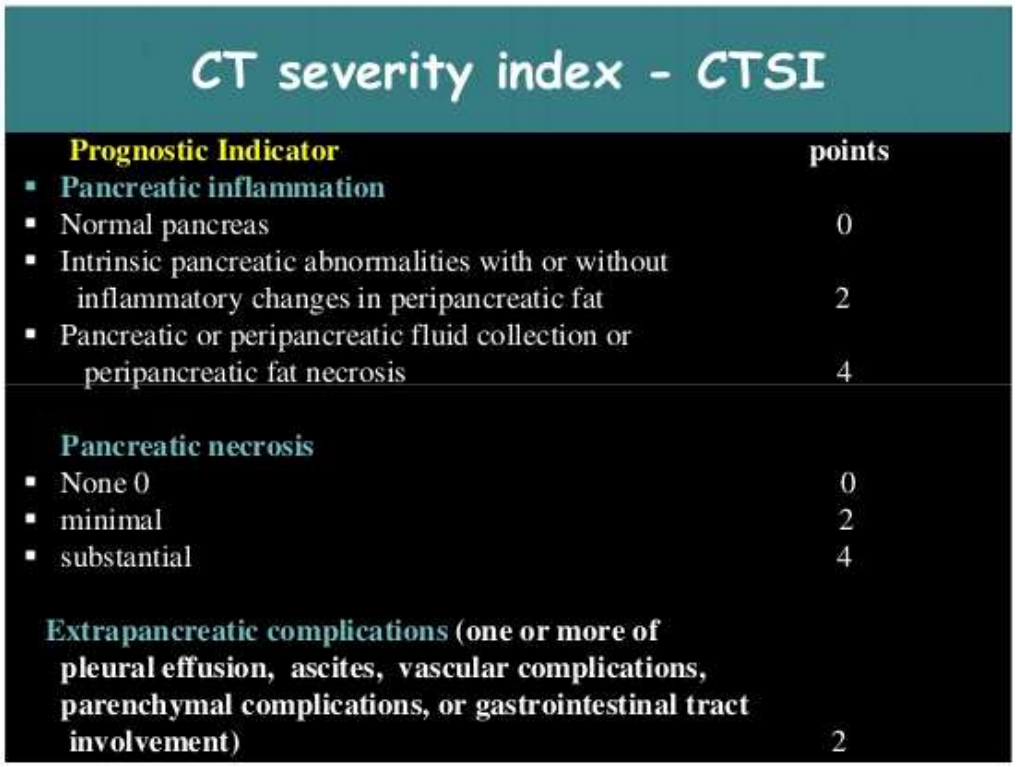
- Inter observer variability with original CTSI can result in different scores for the same patient
- It has been observed that patients with >30% necrosis have similar morbidity and mortality, thus including an additional 50% in the score was not practically useful.

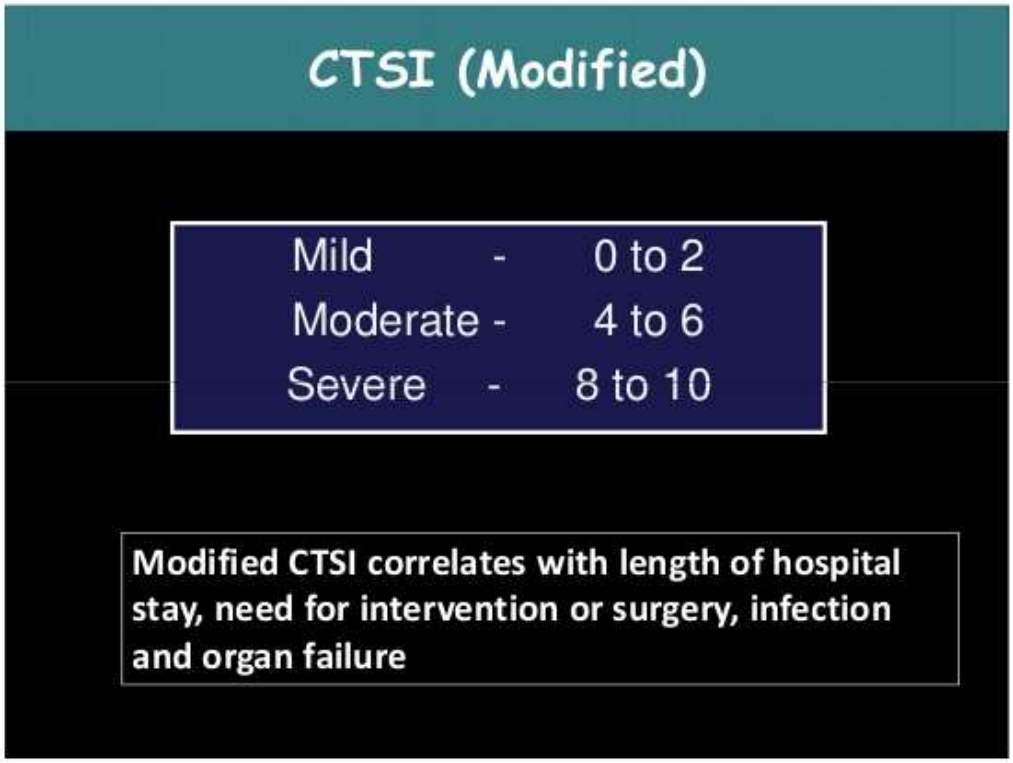
BALTHAZAR GRADING OF CT SEVERITY OF ACUTE PANCREATITIS

Inflammatory Process	*Grade*	*Score*	*Subtotals*
Normal	A	0	
Focal or diffuse enlargement	B	1	
Contour irregularity			
Inhomogeneous attenuation			
Grade B *plus* peripancreatic haziness/mottled densities	C	2	
Grades B, C *plus* one ill-defined peripancreatic fluid collection	D	3	
Grades B, C *plus* two ill-defined fluid collections or gas	E	4	
Necrosis:			
None	0	0	
<30%		2	
50%		4	
>50%		6	
Total			

These limitations have resulted in the creation of the modified CTSI which correlates more closely with patient outcome. The modified CT severity index is an extension of the original CTSI which was developed by Mortele et al in 2004.

MODIFIED CT SEVERITY INDEX:





MRI

MRI abdomen gives similar information like CT in identifying the severity of the disease. MRI is as useful as CT in identifying necrosis and fluid collections.

MRI is better than CT, and equal to EUS and ERCP in detecting the choledocholithiasis, ductal or ampullary pathologies and neoplasms. The use of IV secretin, before MRCP helps in better delineation of the pancreatic duct. This is particularly useful in the management of patients with idiopathic and recurrent pancreatitis.

ENDOSCOPIC ULTRASOUND AND ERCP

EUS is not that useful in early phase of acute pancreatitis. EUS during an acute attack of and weeks following an episode, shows signals indistinguishable from chronic pancreatitis and malignancy. But after a month, particularly in patients with idiopathic pancreatitis, EUS may be useful in identifying the presence of small tumours, pancreas divisum, and CBD stones. EUS is equal to MRCP and ERCP but more sensitive than either abdominal ultrasound or CT in detecting common bile duct stones. ERCP considered to be safe in acute pancreatitis, such as in the setting of biliary pancreatitis, with raising serum bilirubin and biliary sepsis.

ASSESSMENT OF SEVERITY

Table 1. Clinical Scoring Systems Developed to Classify the Severity of Acute Pancreatitis

Clinical Score	First Validation Study, Year (Reference)	Country	Outcomes Predicted in the First Validation Study
Ranson score/criteria	Ranson et al, 1974 (5); Ranson and Pasternack, 1977 (6)	United States	Severity (death, ≥7 d in the intensive care unit)
Glasgow score/criteria	Blamey et al, 1984 (7)	United Kingdom	Severity (mortality, surgery, complications)
Simplified prognostic criteria	Agarwal and Pitchumoni, 1986 (8)	United States	Severity (complications)
APACHE II	Wilson et al, 1990 (9) Larvin and McMahon, 1989 (10)	United Kingdom	Severity, mortality
Japanese Severity Score (original)	Ogawa et al, 2002 (11)	Japan	Mortality
Logistic Organ Dysfunction Score	Halonen et al, 2002 (12)	Finland	Mortality
Multiple Organ Dysfunction Score	Halonen et al, 2002 (12)	Finland	Mortality
SOFA	Halonen et al, 2002 (12)	Finland	Mortality
SIRS score	Ogawa et al, 2002 (11)	Japan	Mortality, severity (Multiple Organ Dysfunction Score)
	Buter et al, 2002 (13)	United Kingdom	Mortality, severity (Multiple Organ Dysfunction Score)
APACHE III	Liu et al, 2003 (14)	United States	Mortality
BALI score	Spizer et al, 2006 (15)	United States	Mortality
Early Warning Score	Garcea et al, 2006 (16)	United Kingdom	Mortality, severity (Atlanta criteria)
Mortality Probability Model	Göçmen et al, 2007 (17)	Turkey	Mortality, severity (Atlanta criteria)
Panc 3 score	Brown et al, 2007 (18)	United States	Severity (Atlanta criteria)
Pancreatitis Outcome Prediction Score	Harrison et al, 2007 (19)	United Kingdom	Mortality
Simple Prognostic Score	Ueda et al, 2007 (20)	Japan	Mortality, severity (infection, organ failure)
SAPS	Göçmen et al, 2007 (17)	Turkey	Mortality, severity (Atlanta criteria)
BISAP	Wu et al, 2008 (4)	United States	Mortality
Harmless Acute Pancreatitis Score	Lankisch et al, 2009 (21)	Germany	Severity (necrosis, need for ventilation or dialysis, death)
Japanese Severity Score (revised)	Ueda et al, 2009 (22)	Japan	Mortality

APACHE = Acute Physiology and Chronic Health Evaluation; BISAP = Bedside Index of Severity in Acute Pancreatitis; SAPS = Simplified Acute Physiology Score; SOFA = Sequential Organ Failure Assessment; SIRS = Systemic Inflammatory Response Syndrome.

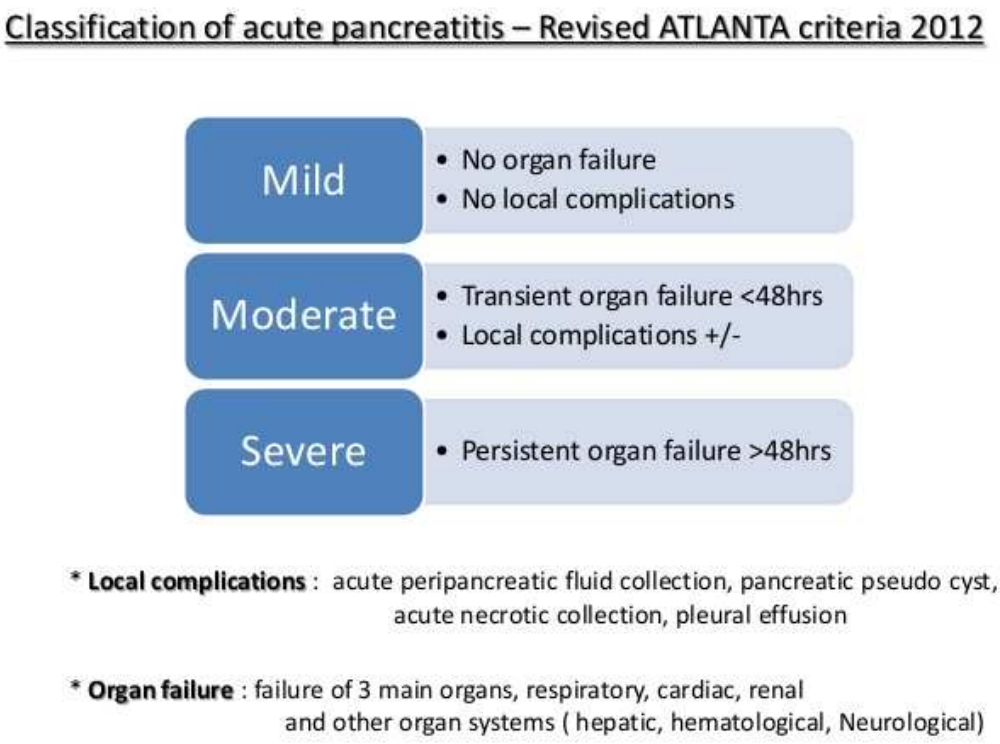
Numerous classification systems had been devised in the past.

The widely accepted classification system, for severity in acute pancreatitis is the Atlanta classification, was devised in 1992. Atlanta 1992 classification, parts acute pancreatitis into two groups: mild and severe. Severe pancreatitis, defined by the presence of organ failure, local pancreatic complications on imaging studies like "acute fluid collection, pancreatic necrosis, pseudocyst and pancreatic abscess" and/or poor prognostic scores (Ranson's ≥3 and/or APACHE-II≥8). Atlanta 1992 has developed a universally applicable classification system, that

successfully used in clinical studies and helped in the comparison of data from different institutes for over many years.

However there are limitations in the 1992 Atlanta classification of acute pancreatitis, and improved understanding of the pathogenesis of acute pancreatitis, the 1992 classification has been revised in 2012.

The revised of the Atlanta classification (Atlanta 2012) divides, acute pancreatitis severity into 3 groups: Mild, Moderate and Severe.



Mild acute pancreatitis: It is characterized by absence of the organ failure and local/systemic complications.

Moderately severe acute pancreatitis: It is characterized by transient organ failure (recovers within 48 hours) and/or local/systemic complications.

Severe acute pancreatitis: It presents as persistent organ failure that can involve one or multiple organs.

Necrotizing pancreatitis is defined as the "The presence of parenchymal necrosis and/or necrosis of peripancreatic fat."

The updated Atlanta classification, includes the patients with peri pancreatic necrosis only that is, without necrosis of pancreatic parenchyma, in the category of Necrotizing Pancreatitis.

Edematous interstitial pancreatitis usually follows a mild course, however a small subset of patients may suffer a fulminant attack and die within 2 to 5 days. These patients have severe disease, but not included in the criteria of necrotizing pancreatitis.

SCORING SYSTEMS

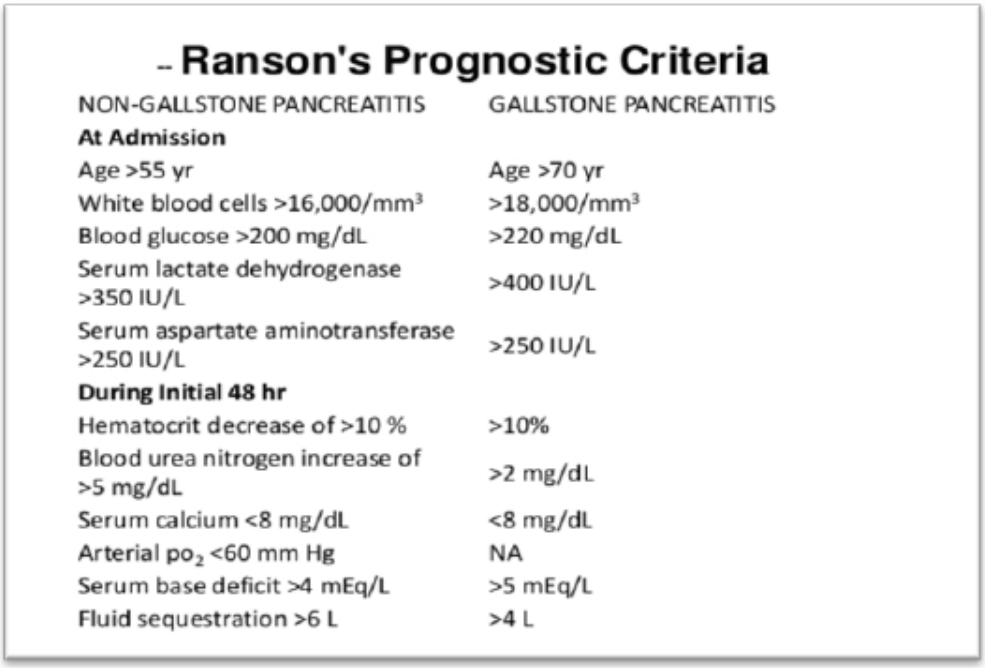
COMMONLY USED PREDICTIVE LABORATORY SCORING SYSTEMS AND THEIR CUTOFF FOR PREDICTED SEVERE PANCREATITIS

TABLE 88-1 Commonly Used Predictive Laboratory Scoring Systems in Acute Pancreatitis and Their Cutoff for Predicted Severe Pancreatitis

Predictive Score	Cutoff
APACHE II	≥8 in first 24 hours
BISAP	≥3 in first 24 hours
Modified Glasgow (or Imrie)	≥3 in first 48 hours
Ranson	≥3 in first 48 hours
Urea at admission	>60 mmol/L
C-reactive protein	>150 U/L in first 72 hours

I) RANSON'S SCORING SYSTEM

This is the earliest scoring system designed to assess the severity of acute pancreatitis. It was introduced by Ranson and his colleagues in 1974. It predicts the severity of the disease based on multile(11) parameters, that are collected at the time of admission and or 48 hours later.



Ranson's Prognostic Criteria

NON-GALLSTONE PANCREATITIS	GALLSTONE PANCREATITIS
At Admission	
Age >55 yr	Age >70 yr
White blood cells >16,000/mm^3	>18,000/mm^3
Blood glucose >200 mg/dL	>220 mg/dL
Serum lactate dehydrogenase >350 IU/L	>400 IU/L
Serum aspartate aminotransferase >250 IU/L	>250 IU/L
During Initial 48 hr	
Hematocrit decrease of >10 %	>10%
Blood urea nitrogen increase of >5 mg/dL	>2 mg/dL
Serum calcium <8 mg/dL	<8 mg/dL
Arterial po_2 <60 mm Hg	NA
Serum base deficit >4 mEq/L	>5 mEq/L
Fluid sequestration >6 L	>4 L

If three or more criteria are positive, Severe pancreatitis is diagnosed. The original criteria was analyzed in patients who actually suffered from alcoholic pancreatitis, that was modified 8 years later, for those patients with gallstone disease. Higher the Ranson's scores suspects a more severe disease. The mortality rate of acute pancreatitis, directly correlates with the number of parameters positive.

Mortality rate in mild pancreatitis if the scores <2, is 2.5% and in severe pancreatitis if the scores >3, it is 62%. The incidence of local and complications of acute pancreatitis relates with Ranson's score. This criteria is still commonly used in the United States and Europe.

The Ranson criteria has many drawbacks, which include

1. The criterias are more complicated

2. There are two different lists based on the etiology

3. It takes 48 hours to calculate the score

4. Validation beyond 48 hours has not been identified

5. Some of the parameters in the criteria are not used routinely in all centers

The sensitivity of the Ranson's criteria is only 40% to 88%, and the specificity is only 43% to 90%.

The positive predictive value is around 50%, and the negative predictive value around 90%.

II. MODIFIED GLASGOW CRITERIA:

This criteria is useful in both alcoholic and biliary pancreatitis.

The score ≥ 3 indicates, the severe disease requires ICU care.

P - PaO2 < 8kPa or < 60 mmhg

A - Age > 55 years old

N - Neutrophilia with WBC count > 15x109/L

C - Calcium < 2mmol/L or < 8 mg/dl

R - Renal function, Urea >16mmol/L or > 45 mg/dl

E – Enzymes: serum LDH > 600 IU/L: AST > 200 IU/L

A - Albumin < 3.2g/dL

S - Sugar: > 10mmol/L or > 180 mg/dl

III. AGA GUIDELINES

A. The American Gastroenterological Association has given guidelines for predicting the severity of pancreatitis.

1. Prediction of severe disease be performed using the APACHE II system, using a cutoff of ≥ 8.
2. Patients with severe disease and those with other severe co morbid conditions, should be considered for admission to an ICU or intermediate medical care unit.
3. In patients with predicted severe disease, with APACHE II score of ≥8 and patients with features of organ failure during the initial 72 hours, rapid bolus CECT should be performed after 72 hours of

illness to assess the EXTEND of pancreatic necrosis. CT should be used selectively based on the clinical features in patients who do not has these criteria.

B. Laboratory tests can be used in addition to clinical judgment and the APACHE II score. A serum C reactive protein >150 mg/L at 48 hours is preferred.

IV. APACHE II SCORING

It is abbreviated as Acute Physiology and Chronic Health Evaluation (APACHE II) score.

It is the most widely studied scoring system in acute pancreatitis.

It has a good negative predictive value and a modest positive predictive value, at predicting severity of acute pancreatitis and can be calculated daily. Decreasing tend during the first 2 days will suggest a mild attack, whereas increasing trend denotes a severe attack. The mortality is less than 4% with a score < 8 and it is 11 to 18% with a score > 8.

APACHE II scoring is based on the patient's age, previous health status, and 12 routine physiologic measurements. An APACHE II score of 8 or more, defines severe pancreatitis. It has the advantage of being

used daily and has similar positive and negative predictive values like the Ranson's score at 48 hours after admission.

The major advantage of the APACHE II scoring system is that it can be used in monitoring patient’s response to treatment. However, Ranson and the Glasgow scales are mainly meant to assess the severity at presentation.

The APACHE-II system assesses 12 variables, for age, and for chronic health status, thus generating a total score.

The 12 variables are

1. Temperature
2. Respiratory rate
3. Heart rate,
4. Mean arterial blood pressure
5. Arterial pH
6. Oxygenation,
7. Serum creatinine
8. Serum sodium
9. Serum potassium,
10. Hematocrit;
11. WBC count
12. GCS

Since the age and severe chronic health problems reflect a decreased physiological reserve, they have been directly incorporated into APACHE II.

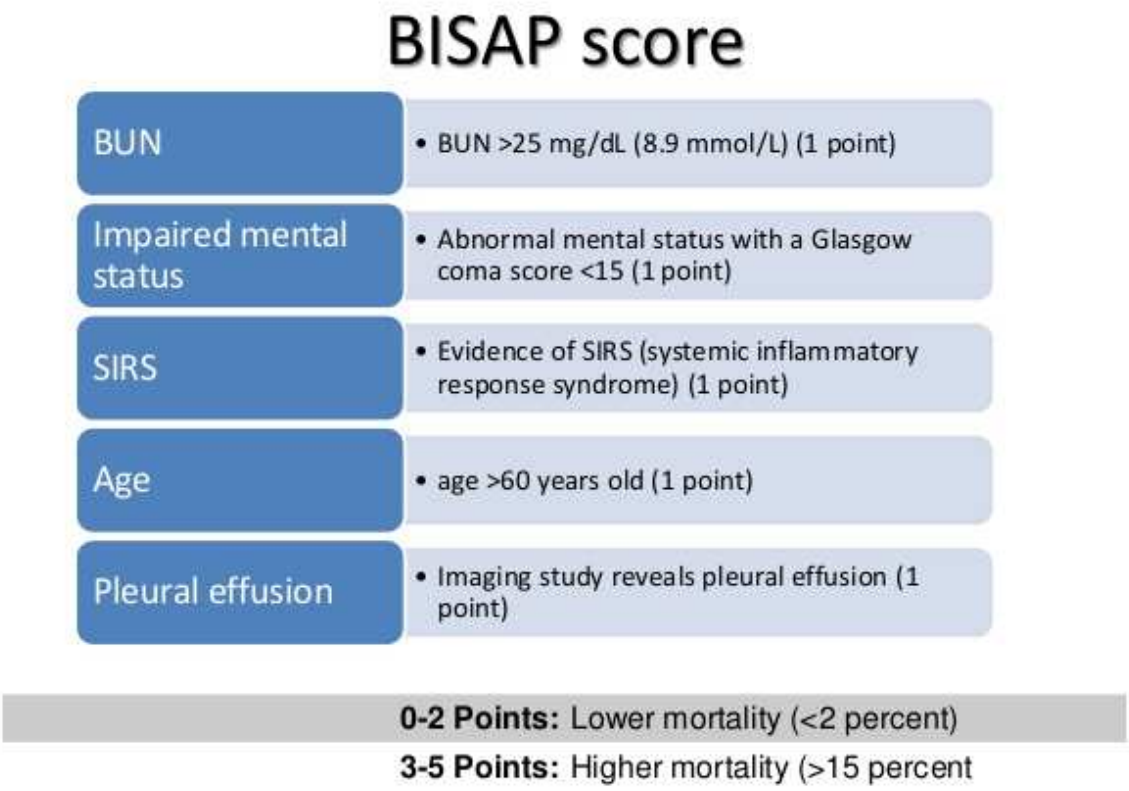
The laboratory tests which are required are simple, routine and readily available and can be done daily on basis. Most patients survive if APACHE-II scores are 9 or less during the first 48 hours. Patients with APACHE-II scores of 13 or more have a high likelihood of mortality.

The range of the APACHE II scoring is wide, thus providing a better delineation between the mild and severe attacks since the varying weights are assigned to increasingly abnormal values, instead all or no judgements.

At initial presentation, sensitivity is 34% to 70%, and specificity is 76% to 98%. At 48 hours, the sensitivity is less than 50%, but specificity is about 90% to a Score of ≥ 2 indicates presence of organ failure. These scores were calculated within 72 hours of hospitalisation. The organ failure was classified as Transient if lasts less than 48 hrs and Persistent if continue s beyond 48 hrs.

V. BISAP - The "Bedside Index for Severity in Acute Pancreatitis":

This scoring system has been devised recently for early identification of patients with risk of mortality.



The BISAP score was developed and validated retrospectively on a large population based study, done by Cardinal Health Clinical Outcomes Research Database, Marlborough, USA.

This system was published recently for clinical and research purpose, for its accuracy and reliability in patient stratification.

The BISAP score:

1) Blood urea nitrogen (BUN) >25 mg / dl.
2) Impaired mental status (GCS < 15).
3) signs of SIRS.

4) Age > 60 years.
5) Pleural effusion

Systemic Inflammatory Response Syndrome is identified by presence of 2 or more of the following features:

I. Pulse rate > 90/min.
II. Respiratory rate > 20/min or PaCO 2 < 32 mm Hg.
III. Temperature >100.4 F or < 96.8 F / < 36 or > 38 ° C.
IV. WBC count >12,000 or < 4,000 cells/mm3, or presence of more than
V. 10% immature blasts.

One point is given for each variable present for a total of 5, thus the score ranges from 0 to 5.

The presence of a pleural effusion was determined by a CT scan, chest X ray or abdominal ultrasound that is obtained within 24 h of presentation. Imaging is obtained within 24 h of admission at the hospital and for transferred patients was also reports are collected and reviewed.

A BISAP score of 3 or more is associated with high mortality and can predict the necrosis and organ failure very well.

The Great advantage of this sytem is, it is simple and easy to calculate and can be done at a primary heath care level that hels in

transferring the high risk cases to the higher centers since this is usually done at the time of admission or within 24 hrs of hospitalization.

MANAGEMENT OF ACUTE PANCREATITIS

GENERAL CONSIDERATIONS

Patients with acute pancreatitis requires timely and aggressive parentral hydration to maintain hemodynamic stability and to adequately supply the kidneys and pancreas.

These patients need adequate pain alleviation to eliminate and significantly reduce the pain. The patients are kept on nil per oral until the nausea and vomiting settles. Abdominal pain can be treated with opioids. Opioid dosage is monitored carefully and adjusted on according to the needs. Although morphine has been implicated to increase the tone of sphincter of Oddi, and serum amylase, it is used in treating the pain in acute pancreatitis and it has not been shown to affect prognosis adversely.

Nasogastric intubation is not been shown to be beneficial in mild pancreatitis hence not routinely used.

It is used for treat gastric ileus or intractable nausea and vomiting. Similarly, routine administration of proton pump inhibitors or H2 receptor blockers have not been shown to be that beneficial.

The patient has be monitored carefully for signs of early organ failure like hypotension, respiratory failure, or renal failure by monitoring the vital signs and urinary output closely. Tachypnea should not be considered to be due to abdominal pain. Measuring oxygen saturation and, if needed, arterial blood gas measurement is advised, and also oxygen supplementation is needed in cases of hypoxia. Patients those exhibit signs of early organ dysfunction should be transferred quickly to an ICU, as clinical deterioration can be rapid and lethal.

FLUID RESUSCITATION

Recommendations regarding aggressive volume replacement are based on expert opinion, laboratory experiments and retrospective as well as prospective clinical studies. The inflammatory process progresses early in the course of acute pancreatitis, there is extravasation of proteinicious intravascular fluid, into the peritoneal cavity as well as retroperitoneum, resulting in hemo concentration and decreased renal perfusion with elevation of blood urea. This leads to the reduced perfusion pressure into the pancreas results in microcirculatory changes which cause pancreatic necrosis.

Hence at presentation if the hematocrit more than 44% and a failure of initial hematocrit to decrease at 24 hours, have been shown to be result in necrotizing pancreatitis. An elevated/ rising blood urea

nitrogen is associated with increased mortality. Early aggressive IV fluid repletion to restore intravascular volume is of foremost importance. The aim is to provide adequate intravascular volume to reduce the hematocrit and blood urea nitrogen, thus increasing pancreatic perfusion.

Ringer lactate may be the ideal solution for initial hydration. Due to its bicarbonate content and stable pH, this isotonic solution, may stop the development of metabolic acidosis.

It is important to recognize that aggressive early volume infusion, will require caution in certain patients (such as elderly patients or those with a history of cardiac and/or renal disease) to avoid complications as volume overload, pulmonary edema, and abdominal compartment syndrome.

PULMONARY CARE

Hypoxia (SpO2 <90%) requires oxygen supplementation, ideally by nasal prongs/face mask. If nasal oxygen fails to correct hypoxemia, or if there is respiratory fatigue, early endotracheal intubation and assisted mechanical ventilation is required. It is important to use a Swan-Ganz catheter to identify if hypoxemia is due to congestive heart failure or due to primary pulmonary damage.

Acute respiratory distress syndrome (ARDS), is the most dreaded respiratory complication of acute pancreatitis. ARDS is associated with severe dyspnea, progressive hypoxia, and results in increased mortality. It usually occurs between the second and seventh day of onset of disease (but can be present at admission) and consists of increased pulmonary alveolar capillary permeability resulting in pulmonary edema. Treatment for this is endotracheal intubation, with positive end expiratory pressure ventilation, with low tidal volumes to protect the lungs from barotrauma.

ANTIBIOTICS

Antibiotics are not usually indicated in mild acute pancreatitis. However, antibiotics would be needed in pancreatic sepsis (e.g., infected necrosis and abscess) and non pancreatic sepsis (e.g., IV catheter sepsis, uro sepsis, or pneumonia).

A recent updated metaanalysis clearly demonstrated that there is no beneficial effect in the routine use of systemic antibiotic prophylaxis in pancreatitis.

NUTRITION

In severe acute pancreatitis, especially with pancreatic necrosis, 4 to 6 weeks of parentral nutritional support may be necessary. Earlier TPN was the standard method of feeding patients with severe acute

pancreatitis. Enteral nutrition is cheaper as well as safer, and is preferred nowadays. Enteral nutrition is hypothesized to decrease small bowel bacterial translocation, and to improve intestinal mucosal barrier function, thus reducing bacterial translocation and resultant complications. The optimal route for the administration of enteral feeding, either through a nasojejunal/gastric tube is yet to be studied.

SURGICAL TREATMENT

Cholecystectomy is routinely performed in patients with gallstone pancreatitis, and it is suggested that in mild or severe gallstone pancreatitis, cholecystectomy should be performed as soon as the patient has recovered from the acute inflammatory process has subsided.

An another potential role for surgery in pancreatitis is to remove pancreatic necrosis (necrosectomy) or drain a pancreatic abscess.

Sterile necrosis can be managed non-operatively or by percutaneous drainage since the mortality of this condition without surgery is less than 5%.

The methods of necrosectomy operations that had been recommended include necrosectomy with closed continuous irrigation through indwelling catheters, necrosectomy with closed drainage without irrigation, or necrosectomy and open packing.

BILIARY PANCREATITIS

Gallstones are the common cause of acute pancreatitis all over the world. Most patients will pass off the offending stone during early hours of acute pancreatitis, but they have additional stones which are capable of inducing further episodes. The issue of when to operate is controversial. Generally either urgent intervention (cholecystectomy) within the first 48 to 72 hours of admission, or a delayed intervention after 72 hours, during the same admission is performed. Cholecystectomy and open common duct clearance is possibly the wise treatment for an otherwise healthy patient with obstructive pancreatitis.

However, patients who are at a high risk for surgery are treated by endoscopic sphincterotomy, with removal of stones by ERCP If in acute biliary pancreatitis, in which obstruction persists after 24 hours of observation, emergency endoscopic sphincterotomy and stone extraction is done. Routine ERCP examination of the bile duct is not advised in cases of pancreatitis, as the possibility of finding the residual stones is less, and also the risk of iatrogenic pancreatitis is high. Patients who are suspected to have an impacted stone in the distal common bile duct or ampulla should have a confirmation by radiologic imaging (CT scan, MRCP, or endoscopic ultrasonography) before intervention is done.

COMPLICATIONS:

A. Pancreas
   1. Edema, inflammation, local fat necrosis
   2. Necrosis, hemorrhage
   3. Phlegmon
   4. Pseudocyst: pain, rupture, hemorrhage, infection, obstruction of gastrointestinal tract (stomach, duodenum, colon)
   5. Abscess

B. Contiguous organs
   1. Extension of inflammation, fat necrosis, hemorrhage into peritoneum and retroperitoneum
   2. Thrombosis of adjacent blood vessels
   3. Ileus, bowel obstruction, perforation, infarction
   4. Pancreatic ascites: disruption of main pancreatic duct, leaking pseudocyst
   5. Obstructive jaundice

C. Systemic
   1. Cardiovascular: shock, hypovolemia, peripheral vasodilation, pericardial effusion, nonspecific ECG changes, sudden death
   2. Pulmonary: pleural effusion, pulmonary edema, adult respiratory distress syndrome, atelectasis, pneumonitis, mediastinal abscess
   3. Renal: renal failure, acute tubular necrosis, renal artery or vein thrombosis
   4. Hematologic: disseminated intravascular coagulation (DIC)
   5. Metabolic: hypocalcemia, hypoglycemia, hyperglycemia, hypertriglyceridemia
   6. Gastrointestinal: erosive gastritis, peptic ulcer, hemorrhage, bowel obstruction, portal vein thrombosis, variceal hemorrhage
   7. Nervous system: encephalopathy, retinopathy (sudden blindness), psychosis, fat emboli
   8. Distant fat necrosis (skin, bones, joints)

MATERIALS AND METHODS

Study design: Prospective and retrospective study

Period of Study : October 2016 – September 2017

Setting: Institute of General Surgery, Rajiv Gandhi Govt. General Hospital . The study was conducted after obtaining the Institutional Ethical Committee approval.

INCLUSION CRITERIA

- Patients with a clinical picture consistent with the diagnosis of acute pancreatitis, along with radiological evidence of inflamed pancreas will be considered to have acute pancreatitis.
- First episode of Acute Pancreatitis
- Age > 18 years and Age < 70 years

Individual components of the BISAP scoring system:

1) BUN > 25 mg/dl
2) Impaired mental status (Glasgow Coma Scale Score < 15)
3) SIRS-SIRS is defined as two or more of the following:
    a. Temperature of < 36 or > 38 ° C
    b. Respiratory rate > 20 breaths/min or PaCO2 < 32 mm Hg

c. Pulse > 90 beats/min

d. WBC < 4,000 or >12,000 cells/mm3 or >10% immature bands

4) Pleural effusion detected on imaging

5) Age > 70 years

One point is assigned for each variable within 24 hrs of presentation. A CECT of the abdomen, obtained at any time in the first 7 days of hospitalization.

EXCLUSION CRITERIA

- Proven cases of chronic pancreatitis.
- Hereditary pancreatitis.
- Acute pancreatitis patients with organ failure at or within 24hrs of presentation
- Pregnancy
- Chronic kidney disease
- Traumatic pancreatitis with head injury
- Mental retardation

METHODS

First 50 patients attending the general surgery department with clinical features of Acute Pancreatitis are evaluated clinically and subjected to laboratory and radiological investigations as per the designed proforma.

Data pertinent to the scoring systems will be recorded within 24 h of admission to the hospital. Once diagnosis is established the patient disease severity will be assessed by BISAP scoring system

Sample Size : 50 Patients

Source of Study: Patients diagnosed as acute pancreatitis in Institute of General Surgery, Rajiv Gandhi Govt. General Hospital. 50 of them are to be selected on the basis of non probability (purposive) sampling method.

Statistical Analysis:

All the patients included in the study has to answer the questionnaire regarding the history of Alcoholism, Gall stone disease, Trauma, Drug intake and family history of dyslipidemia.

Their vital signs were recorded immediately after admission. They all were subjected to complete blood count, random blood sugar, renal function test, Serum amylase/lipase, serum calcium, lipid profile and liver function tests.

An abdominal Xray and USG abdomen was done as early as possible, that is within 24 hours of presentation.

They are subjected to CECT during hospitalization, usually after initial stabilisation that is between 48 to 96 hours.

For each of 50 patients included in the study, BISAP scores and modified CTSI scores were calculated.

The collected data were analysed with IBM. SPSS statistics software 23.0 Version.To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables.The Receiver Operator Characteristic (ROC) curve analysis was used to find the Sensitivity ,Specificity ,PPV and NPV on BISAP Score with CTSI Score.used.To find the significance in categorical data Chi-Square test was used .

Biliary Pancreatitis was presence of gall stones/biliary sludge in the gall bladder or bile duct, which was documented by any radiological methods.

Alcoholic Pancreatitis was considered, when the patient found to have regular high intake of alcohol daily, or if there was binge of alcohol

consumption prior to the onset of illness and has no signs of other etiologies present.

Idiopathic pancreatitis was the one with no identifiable etiological factor based on the history and after all investigations.

Patients were observed prospectively till discharge from the hospital.

Survivors were defined as patients discharged alive from the hospital and non-survivors were those who died from pancreatitis or its complications during hospitalization.

Improvement is defined as resolution of symptoms with decreasing trend of enzymes and resolving radiological features.

OBSERVATION AND RESULTS

The collected data were analysed with IBM. SPSS statistics software 23.0 Version.To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables.The Receiver Operator Characteristic (ROC) curve analysis was used to find the Sensitivity ,Specificity ,PPV and NPV on BISAP Score with CTSI Score.used.To find the significance in categorical data Chi-Square test was used .

Table 1 Descriptive statistics

	N	Minimum	Maximum	Mean	Std. Deviation
AGE	50	19	65	37.26	9.354
HEART RATE	50	68	130	93.68	14.265
RESPIRATORY RATE	50	14	30	19.48	4.077
LEUCOCYTOSIS	50	4100	27800	11884.00	5552.104
AMYLASE	50	416	1400	864.94	224.740
LIPASE	50	486	1320	761.64	165.651
TRYGLYCERIDES	50	97	1150	205.28	178.789
CALCIUM	50	7	10	8.46	.676
LENGTH OF STAY	50	2	18	6.58	3.494
BUN	50	17	54	25.76	9.492
BISAP TOTAL	50	0	4	.94	1.185
PANCREATIC INFLAMMATION	50	0	4	1.16	1.283
PANCREATIC NECROSIS	50	0	2	.32	.741
EXTRA PANCREATIC	50	0	2	.28	.701
CTSI TOTAL	50	0	8	1.68	2.189
Valid N (listwise)	50				

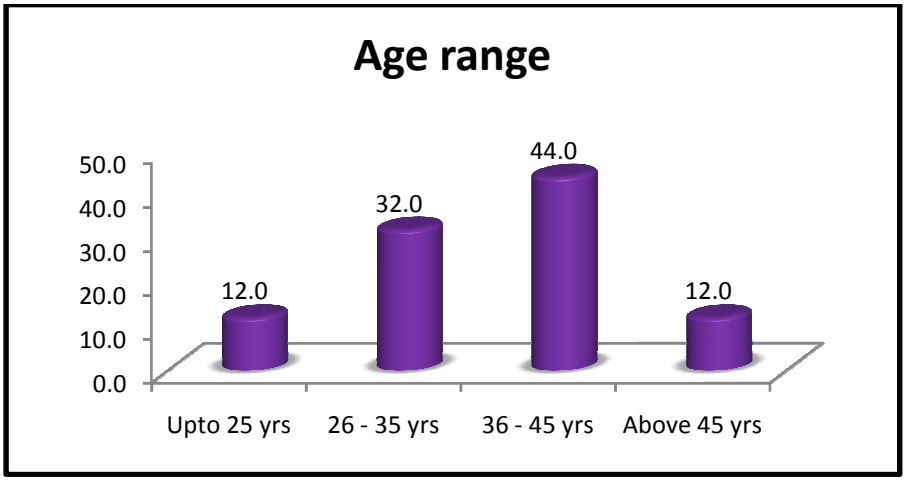
AGE DISTRIBUTION

Table 2 Age Distribution

AGE	Frequency	Percent
Upto 25 yrs	6	12.0
26 - 35 yrs	16	32.0
36 - 45 yrs	22	44.0
Above 45 yrs	6	12.0
Total	50	100.0

In our study, the patients ranged from the age of 19 to 65 years, and 44% were between 35 to 45 years. That is the Adult men of productive age group is affected mostly.

Graph 1: Age Distribution



The mean age is 37 years

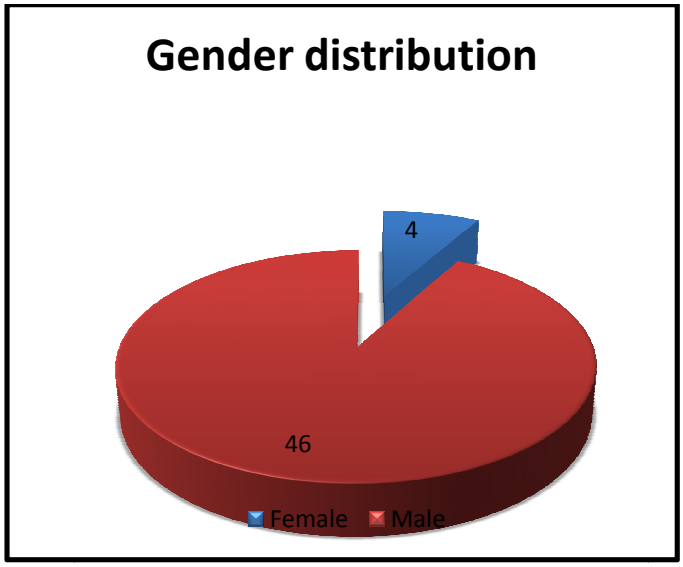
GENDER DISTRIBUTION

Table 3: Gender Distribution

Gender	Frequency	Percent
Female	4	8.0
Male	46	92.0
Total	50	100.0

Obviously Men are involved more than women. This could be due to high prevalence of the alcohol related pancreatitis in this study population which belongs to society with male dominance towards alcoholism.

Graph 2 Gender Distribution



Men (92%) are involved mostly than the women (8%).

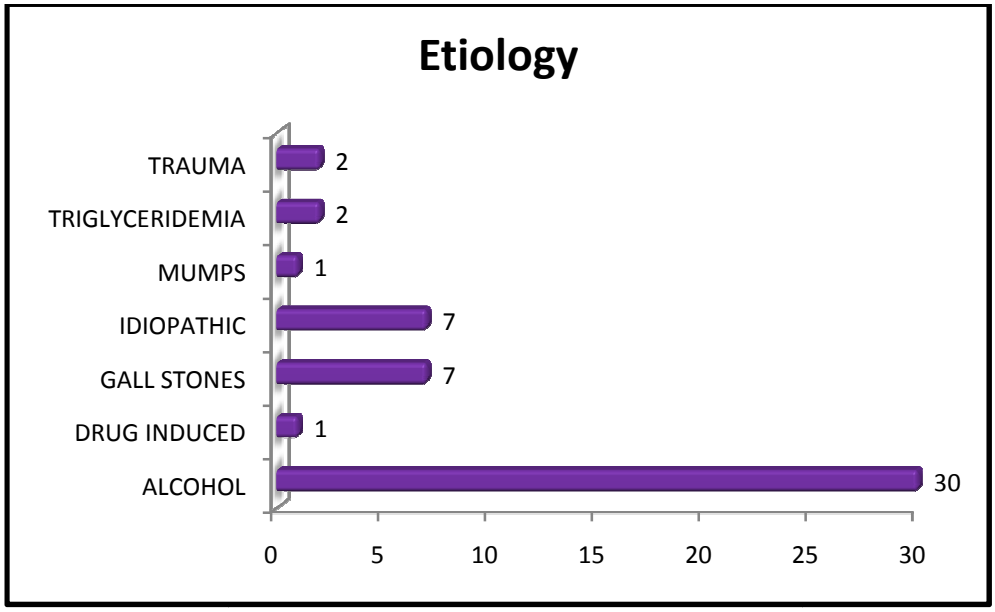
ETIOLOGY

Table 4: Etiology

	Frequency	Percent
ALCOHOL	30	60.0
DRUG INDUCED	1	2.0
GALL STONES	7	14.0
IDIOPATHIC	7	14.0
MUMPS	1	2.0
TRIGLYCERIDEMIA	2	4.0
TRAUMA	2	4.0
Total	50	100.0

History of consumption of alcohol and the possibility of it being the etiological factor were found in 30 patients. Most of the patients belong to middle age that the productive population. They are either chronic alcoholics who consume more than 100 grams of alcohol or frequent binge drinkers. They had no other attributable causes after a brief evaluation.

Graph 3: Etiology



Gall stone disease was attributed in 7 patients. It was confirmed by USG and CT scan. 6 cases were treated conservatively and one case required emergency ERCP to retrieve the calculi, for the resolution of the inflammation. These cases were advised to undergo laparoscopic cholecystectomy and none of them presented with recurrence.

Hyperlipidemia and as causative factor presented in 2 patients who had familial history.

There was clear cut history of blunt trauma with CT scan showed isolated pancreatic laceration presented in 2 cases. One had it by the steering wheel while driving a lorry and another one got it by hitting against the handle bar of two wheeler.

History of Medication in 1 patient who was recently started on anti retro viral therapy. He had resolution of symptoms after the change of regimen.

Mumps was diagnosed in one patient and it was found be the cause. He was a young boy presented with fever, abdominal pain and parotid swelling. He was subjected to imaging for his abdominal pain which confirmed the pancreatitis. Serum IgM for mumps was found positive.

No cause could be attributed in rest of the 7 patients. They all were evaluated for any attributable cause and finally subjected to upper GI scopy to visualize the ampulla to rule out ampullary pathologies like worm infestations, neoplasms. Three of them were subjected to MRI/MRCP to rule out ductal or ampullary causes. However no causative factor was found.

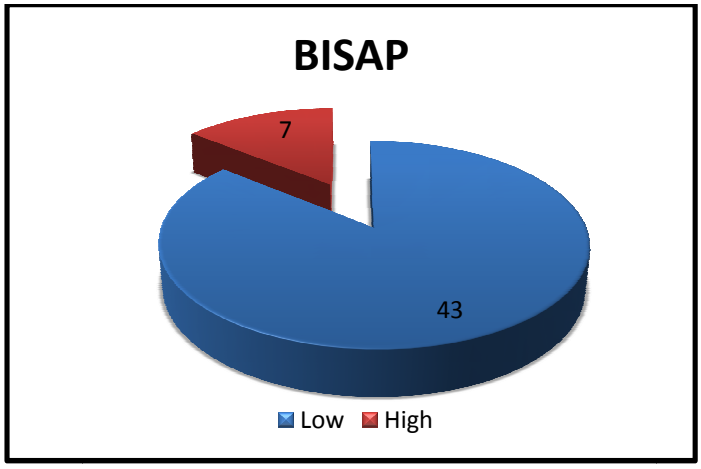
Table 5 SIRS

SIRS	Frequency	Percent
No	28	56.0
Yes	22	44.0
Total	50	100.0

Nearly half of the patients had signs of systemic inflammation and the most common sign was the fever. However most of the cases with positive signs, improved without any complications.

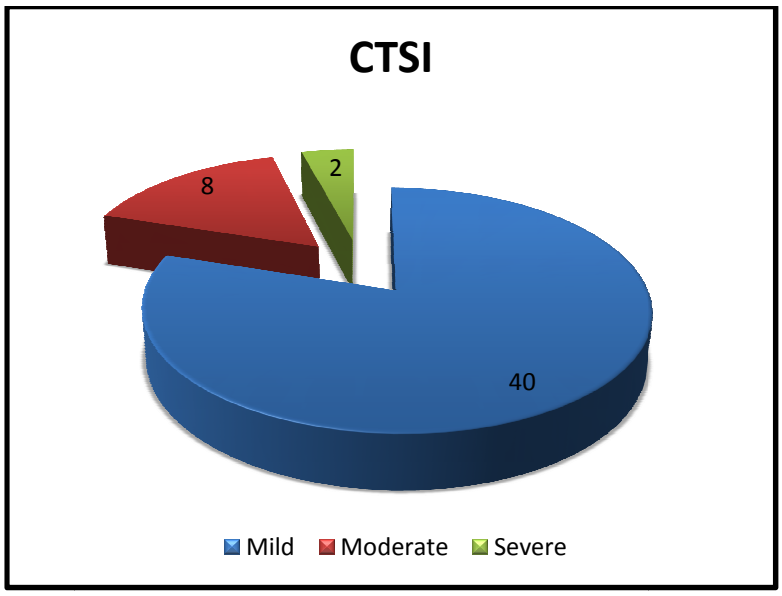
BISAP and CTSI SCORE:

Graph 4 BISAP Score



86% of patients had BISAP score less than 3 (mild) and 14% of patients had more or more than 3 (severe).

Graph 5 CTSI Score



. The cases with mild, moderate and severe CTSI score were 76%, 20% and 4% respectively. Only two cases with BISAP score 3 or more had CTSI >6.

Recurrence was seen in 4 cases and all of these patients were Alcoholics. They continued to drink after remission of the first episode and presented with recurrence during the study.

OUTCOME

Table 6 Outcome

	Frequency	Percent
DEATH	2	4.0
IMPROVED	39	78.0
LOCAL COMPLICATION	6	12.0
SYSTEMIC COMPLICATION	3	6.0
Total	50	100.0

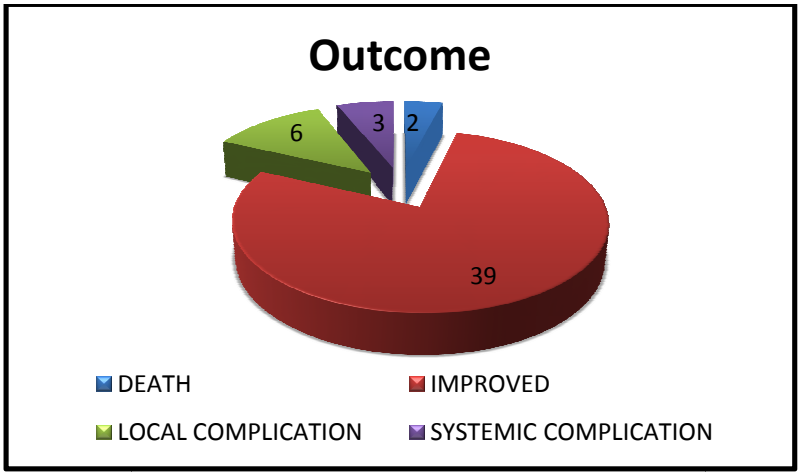
The majority of the cases improved without sequlae. That is 78% of the cases those who all had BISAP score of less than 3.

Local complications like psedocyst, superior mesentric thrombosis occured in 6 cases.

Systemic complication (sepsis, ARDS) occured in 3 cases. 2 cases expired during the study.

Complications occured in cases who had BISAP score more than 3 but patients with moderate severe CTSI score also developed systemic complications.

Graph 6: Outcome



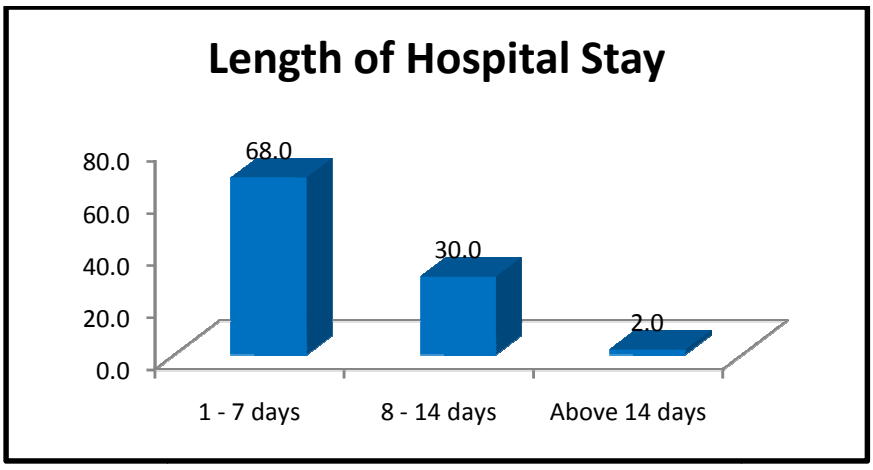
Two cases died during the study. One patient had alcohol as the cause. He presented with septicemia with SIRS. He was referred from a district hospital 2 days after onset of symptoms. Patient required ventilatory and inotrophic support since admission. He was treated at a high dependancy unit. He had BISAP score of 4 and it was calculated retrospectively. He was subjected to CECT after stabilising his renal functions. His modified CTSI score was 8. He didn't came out of sepsis and expired on 6th day.

Where as the another patient who died suffered a blunt (steering wheel) injury to abdomen. He was admitted and evaluated at a private hospital where CECT was done with modified CTSI score of 8. His

retrospectively collected BISAP was 4. He was referred to our institute on the second day with multi organ dysfunction syndrome in a moribund state. He didn't survived the second day.

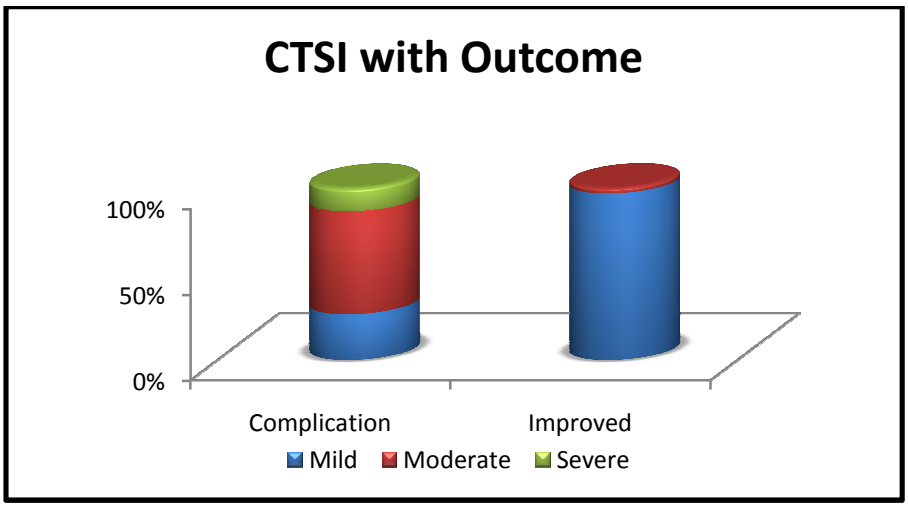
Two cases of recurrence seen during the study period. The both individuals were chronic alcoholics who resumed their course of alcohol after resolution of the first episode.

Graph 7: Length of Hospital Stay



Patients had mild disease according to BISAP and CTSI recovered early and returned home within a week that is 68%. Those with BISAP score of less than 3 never required hospital stay more than a week. Those with severe BISAP score more 3 or more and CTSI score 6 or more needed longer stay. The average length of hospital stay is 7 days.

Graph 8: CTSI with Outcome



The all 38 cases had mild CTSI score Low BISAP score and 2 cases who had severe CTSI score had high BISAP score.

Table 7 CTSI with Outcome

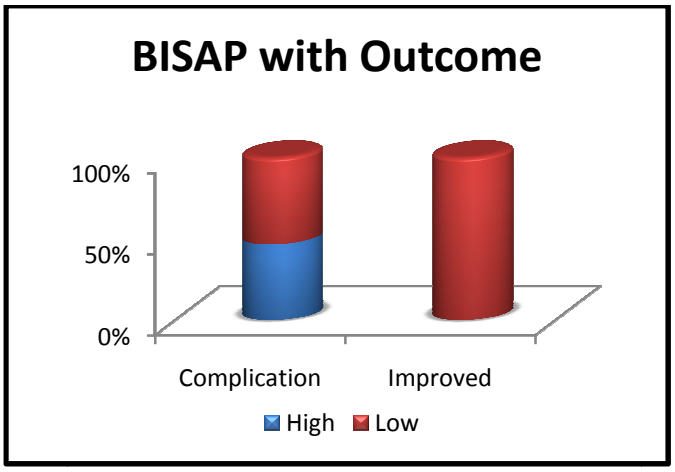
			OUTCOME		
			Complication	**Improved**	**Total**
CTSI TOTAL	Mild	Count	2	38	40
		% within OUTCOME	26.7%	97.1%	76.0%
	Moderate	Count	7	1	8
		% within OUTCOME	60.0%	2.9%	20.0%
	Severe	Count	2	0	2
		% within OUTCOME	13.3%	0.0%	4.0%
Total		Count	15	35	50
		% within OUTCOME	100.0%	100.0%	100.0%

5 cases with moderate CTSI score had low BISAP and another 5 with moderate CTSI had high BISAP.

Table 8. BISAP with Outcome

			OUTCOME		
			Complication	**Improved**	**Total**
BISAP	High	Count	7	0	7
		% within OUTCOME	46.7%	0.0%	14.0%
	Low	Count	8	35	43
		% within OUTCOME	53.3%	100.0%	86.0%
Total		Count	15	35	50
		% within OUTCOME	100.0%	100.0%	100.0%

Graph 9. BISAP with Outcome



BISAP score picks up the complications more clearly than the CTSI score. The 35 cases with low score never developed complications and all the cases with high score developed complications whereas the CTSI scores are overlapping.

Table 9. CTSI TOTAL * OUTCOME
Cross tabulation

		OUTCOME		
		Complication	Improved	Total
CTSI TOTAL	Severe	2	0	2
	M&M	8	40	48
Total		10	40	50

Sensitivity	93.3
Specificity	100.0
PPV	100.0
NPV	72.9
Accuracy	56.7

The all 7 patients with high BISAP score developed complications where the CTSI missed to pick the complications in 5 cases. Importantly the all patients with low BISAP score never developed complications whereas 5 patients with moderate severity in CTSI did not develop complications.

Table 10. BISAP TOTAL * OUTCOME
Cross tabulation

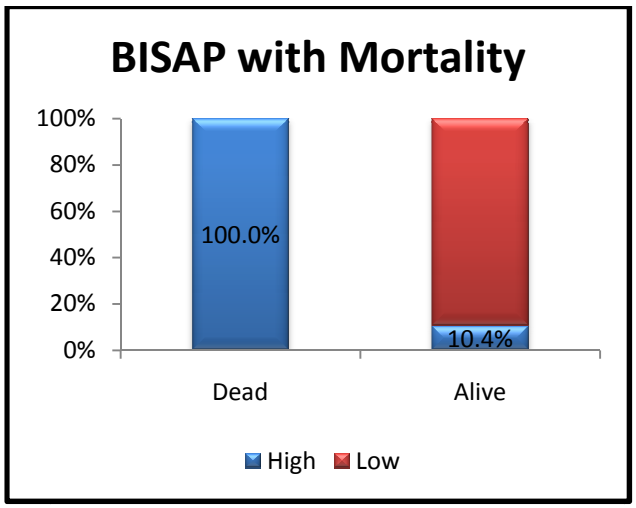
		OUTCOME		
		Complication	Improved	Total
BISAP TOTAL	High	7	0	7
	Low	8	35	43
Total		15	35	50

Sensitivity	86.7
Specificity	100.0
PPV	100.0
NPV	81.4
Accuracy	73.3

The sensitivity of BISAP is higher than the CTSI. Both cases had identified the high risk cases equally that is specificity and positive predictive value. BISAP has higher negative predictive value which can make the difference in health care delivery. Though both are equal in specificity BISAP score stays ahead in accuracy of predicting the prognosis of acute pancreatitis.

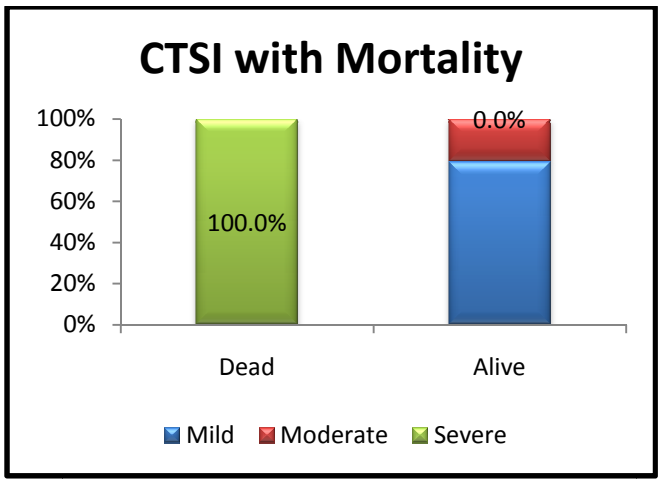
MORTALITY

Graph 10



The mortality rate was 4% in our study. The expired patients had high BISAP score. 10% of cases with high BISAP score survived.

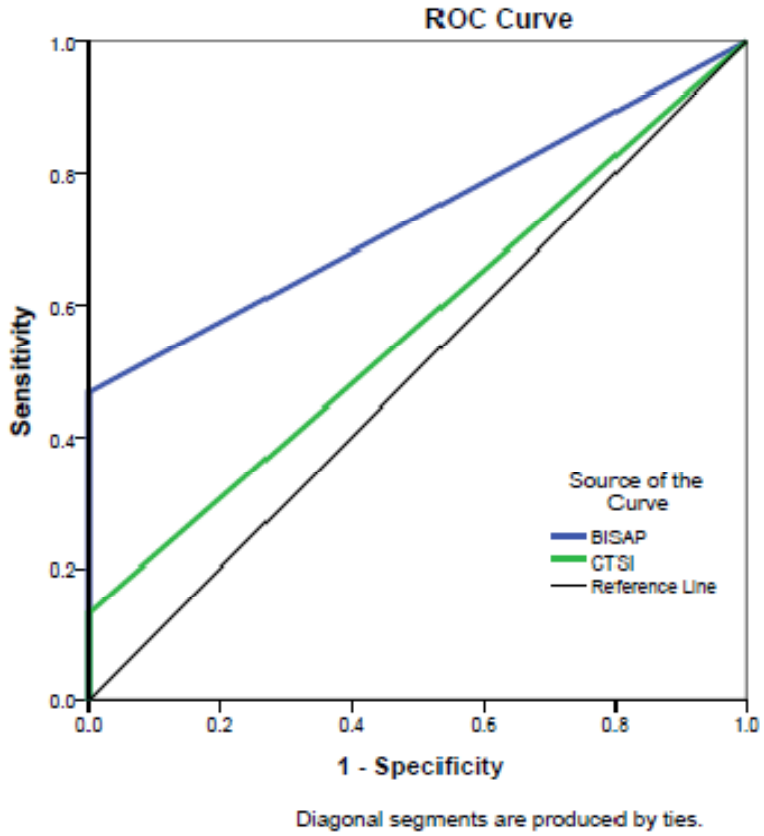
Graph 11



The patients with severe CTSI score were died Where as cases with moderate and mild severity have survived. Thus CTSI score predicts the mortality clearly.

RECEIVER OPERATING CHARACTERISTIC CURVE

Graph 12. ROC Curve



Diagonal segments are produced by ties.

Area Under the Curve

Test Result Variable(s)	Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
BISAP	.733	.089	.010	.559	.908
CTSI	.567	.093	.459	.384	.749

The accuracy of the BISAP and CTSI scores in predicting severity and mortality was determined by plotting receiver operating characteristic (ROC) curves, and calculating the area under curve (AUC). The more the area under the curve more the accuracy. The area under curve (AUC) for BISAP score in predicting severity and mortality was 0.733 and 0.567, respectively, better than that of CTSI.

DISCUSSION

- ✓ The most of the articles state that gall stone as the common cause and alcohol related pancreatitis is on increasing trend. Few studies shows Alcohol and gall stones were equally related to pancreatitis.
- ✓ In an article published in Medical council on alcohol stated that abstinence from alcohol after the first episode protects against the recurrence.
- ✓ In a study conducted at Banaras Hindu University concluded that no single scoring system is ideal for predicting the severity of acute pancreatitis and a system can be chosen according to the institutional fascility.
- ✓ In a journal Published in American journal of gastroenterology, a prospective analysis of BISAP scoring in assessing the mortality showed statistically significant mortality rate when BISAP score was high.
- ✓ In Our study Alcohol is the most common etiology and BISAP serves as a simple and reliable prognostic score.

LIMITATIONS OF THIS STUDY

- Only Small number of patients are included in this study.
- The common etiology found this study were found to be different from worldwide accepted one, hence might not be correct to compare with other studies.
- Variation in timing of presentation of patients to the hospital after onset of symptoms can alter with assessment of the scoring systems.

SUMMARY

- The prediction scores and management tools keeps on developing which means there is a definite dilemma in risk stratification and appropriate treatment strategy that need to be started at the appropriate time,
- The list of causative factors goes a long way and common causes as described in standard texts may not be applicable to all regions as found in this study.
- Alcohol is the major cause in this study. The prevalence of Alcoholism in the regional population may attribute to this situation. Hence Alcoholism can be considered as a social disorder in this population and policy makers may consider the remedies since it affects mainly the adult men of productive age. Most of the cases treated in this government facility belong to middle or low socioeconomic status, so the disability of the adult population can potentially affect the economical growth and quality of life of their own and ultimately of society/state.
- All these patients with alcohol related disease are counseled along with their family and it was empathized that the Alcoholism is the primary diseases. They all were referred to de addiction centers.

- Gall stone disease is the next common cause in our study, that too in females. Since this is the common cause all over the world, all the patients with acute pancreatitis must be screened for gall stones.
- Once the common cause are excluded the possible etiological factor must be sought for to attain early remission and to take steps to prevent the disease in future. As recurrent attacks clearly result in morbidity it is always better to spend time and money on further evaluation to identify a cause before concluding it as idiopathic. Usually the detailed history and clinical examination will give a clue towards the etiology.
- BISAP score is the recently developed, reliable and easy system to stratify the risk in Acute pancreatitis. It can be calculated in center which has a basic laboratory and Xray/USG facility. Usually these are available in the district head quarter hospitals. The sophisticated facilities or special training is not required to calculate this and there is no long waiting time as happens with the CT scan. Once the score is known, the center for the management of the disease, can be clearly decided. As the high risk cases must to be treated at high dependency units this decision can potentially influence the outcome. However gall stone complicating can be referred to the appropriate center for further management.

- The cases with low BISAP score need not undergo with CECT since the negative predictive value of BISAP of almost 100%. CECT is available only in the higher centers and it involves transportation, more time, higher cost and long waiting time. CECT is not immediately available since it cannot be in the in the intense phase of the disease. And also CECT carries the risk of contrast allergy and radiation exposure.
- This study shows that BISAP score is the best in predicting the prognosis than the CTSI. The sensitivity and negative predictive value of BISAP is more than the CTSI. In this study BISAP correlates well with the outcome as the patients with low score(35) did not develop complications and all patients with high score (7) developed complications. Whereas the CTSI scores were overlapping. Hence the patients with low BISAP scores, <3, need not undergo CT scan unless specifically required and can be treated at district level centres. This reduces the cost and saves time for the both, patients as well as the service providers. Even it can reduce the over load of cases at the higher centers.
- As BISAP is a bedside study and calculated at the time of admission, clinically it can be valued higher than the CTSI which is usually done after 48 hours.

CONCLUSION

- ✓ Men were most commonly affected than women with a ratio of 9:1.
- ✓ The age group affected were in 35 to 45 years of age with mean age of 39.
- ✓ Alcohol is the most common etiological factor for acute pancreatitis in this regional population.
- ✓ The morbidity rate is 26% and the mortality rate in patients with severe pancreatitis was 4%.
- ✓ The BISAP score is more accurate in predicting disease severity and significantly than CTSI in this study.

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PMCID: PMC4576607
Irshad Ahmad Banday,1 Imran Gattoo,2 Azher Maqbool Khan,3 Jasima Javeed,4 Ghanshyam Gupta

PROFORMA

PATIENT DETAILS:

Name: Age: Sex:

IP No. :

ON ADMISSION:

MAIN COMPLAINTS:

ASSOCIATED COMPLAINTS :

HISTORY: ALCOHOLISM
GALL STONE DISEASE
TRAUMA
DRUG INTAKE
FAMILY HISTORY

CLINICAL EXAMINATION:

Pulse : BP :

RR : Temp :

Dehydration : Icterus :

Spo2:

CVS : RS :

P/A : CNS:

INVESTIGATIONS :

CBC/RFT				
TC				
DC				
Hb %				
PCV				
RBC				
Platelets				
Glucose				
Urea				
Creatinine				
Na^+/K^+				

Sr. Amylase				
Sr. Lipase				
Total Bili				
Dir. Bili				
SGOT				
SGPT				
Total Protein				
Sr. Albumin				

CXR :

USG Abdomen :

CECT Abdomen :

BISAP SCORE:

CTSI SCORE:

FOLLOW UP :

PATIENT CONSENT FORM

Study Detail	:	**"**COMPREHENSIVE ANALYSIS OF ETIOLOGY, PROGNOSIS AND CLINICAL OUTCOME OF ACUTE PANCREATITIS IN A TERTIARY CARE CENTER**"**
Study Centre	:	Rajiv Gandhi Government General Hospital, Chennai.
Patient's Name	:	
Patient's Age	:	
In Patient Number	:	

Patient may check (☑) these boxes

- I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ☐
- I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐
- I understand that sponsor of the clinical study, others working on the sponsor's behalf, the Ethics committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

- I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ☐

- I hereby consent to participate in this study ☐

- I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests and to undergo treatment ☐

Signature/thumb impression
Patient's Name and Address:

Signature of Investigator
Study Investigator's Name: