A dissertation on

A COMPARATIVE EVALUATION OF CLINICAL AND RADIOLOGICAL SCORING SYSTEMS IN THE EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS

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ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate : DR. SUJITH M. JOSE

Course : POST GRADUATE IN M.S GENERAL SURGERY

Period of Study : ONE YEAR

College : COIMBATORE MEDICAL COLLEGE AND HOSPITAL

Dissertation Topic : "A COMPARATIVE EVALUATION OF CLINICAL AND RADIOLOGICAL SCORING SYSTEMS IN THE EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS"

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This is to certify that this dissertation titled " **A COMPARATIVE EVALUATION OF CLINICAL AND RADIOLOGICAL SCORING SYSTEMS IN THE EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS** " is the bonafide work of **Dr.SUJITH M. JOSE** postgraduate student in M.S General Surgery, Coimbatore Medical College and Hospital, Coimbatore. This study was undertaken in the Department of General Surgery, Coimbatore Medical College and Hospital, Coimbatore during the period July 2016 to June 2017 in the partial fulfillment of the requirement of the **"The Tamil Nadu Dr. M.G.R. Medical University"**for the award of M.S. Degree in General Surgery. This dissertation has not been submitted in part or fully to any other University or Board. It gives me great pleasure to forward this dissertation.

HOD
Prof. Dr. V. Elango M.S.,
Head of the Department
Department of General Surgery
Coimbatore Medical College
Coimbatore

GUIDE
Prof.Dr.A. Nirmala MS., DGO.,
Professor of Surgery
Department of General Surgery
Coimbatore Medical College
Coimbatore

Dr.B.Asokan M.S., MCh.,
The Dean
Coimbatore Medical College and Hospital
Coimbatore

DECLARATION

The dissertation titled **" A COMPARATIVE EVALUATION OF CLINICAL AND RADIOLOGICAL SCORING SYSTEMS IN THE EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS "** is being submitted by me to **"The Tamil Nadu Dr. M.G.R. Medical University"** in partial fulfillment of the regulation for the completion of the M.S General Surgery Degree Examination to be held in 2017. This work has been carried out in the Department of General Surgery, Coimbatore Medical College and Hospital, Coimbatore under the guidance of **Dr.A.Nirmala M.S., DGO.,** Professor of General Surgery, Coimbatore Medical College and Hospital, Coimbatore.

Date:

Place: Coimbatore **Dr.Sujith M. Jose**

CERTIFICATE – II

This is to certify that this dissertation work titled **“ A COMPARATIVE EVALUATION OF CLINICAL AND RADIOLOGICAL SCORING SYSTEMS IN THE EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS ”** of the candidate **DR.SUJITH M. JOSE** with registration Number **221511316** for the award of **M.S in the branch of General Surgery**, I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains 82 pages from Introduction to Conclusion and the result shows **0% (Zero)** percentage of plagiarism in the dissertation.

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ACKNOWLEDGEMENT

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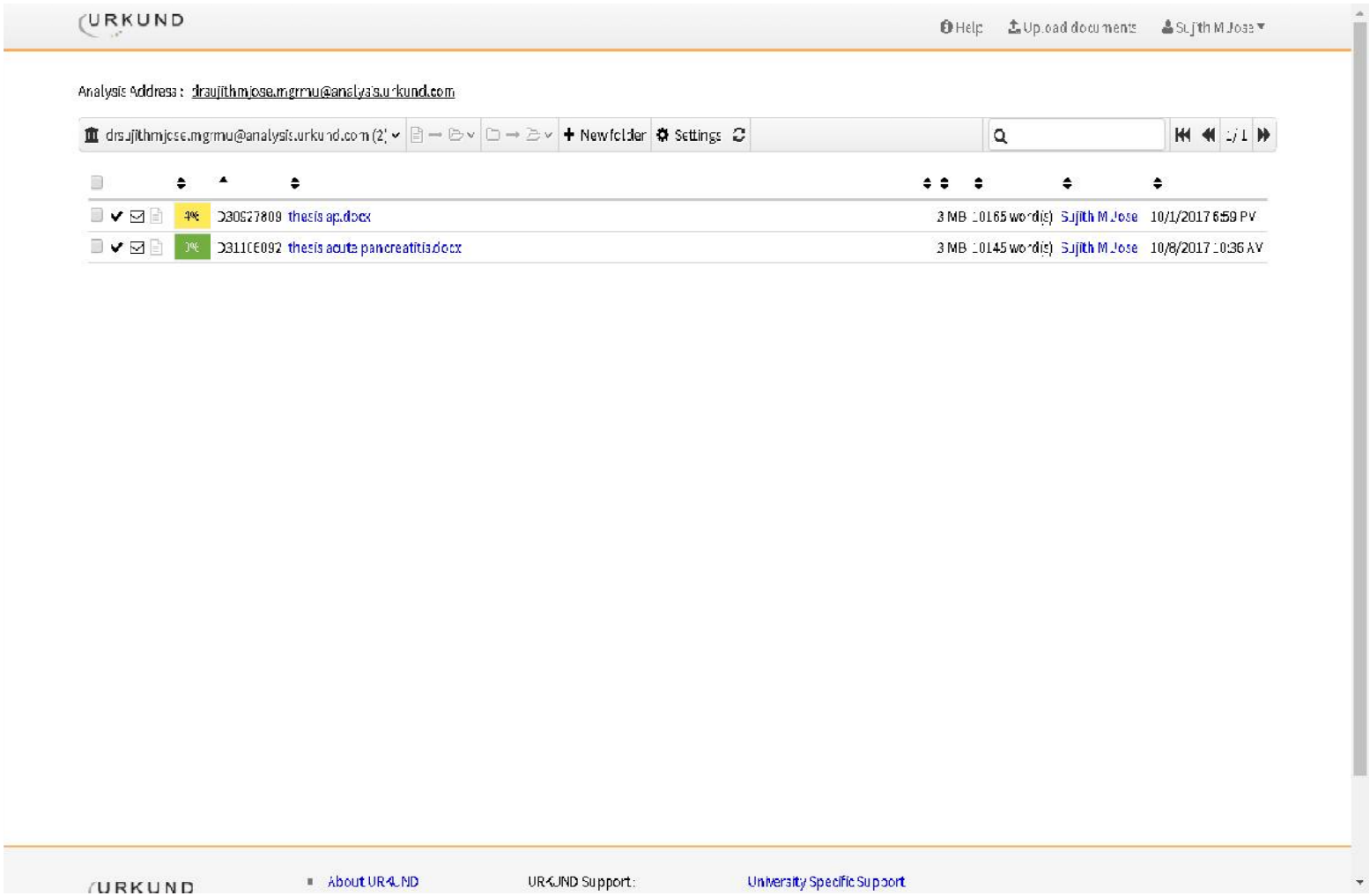
And above all, to God Almighty, for all his kindness and blessings showed upon me.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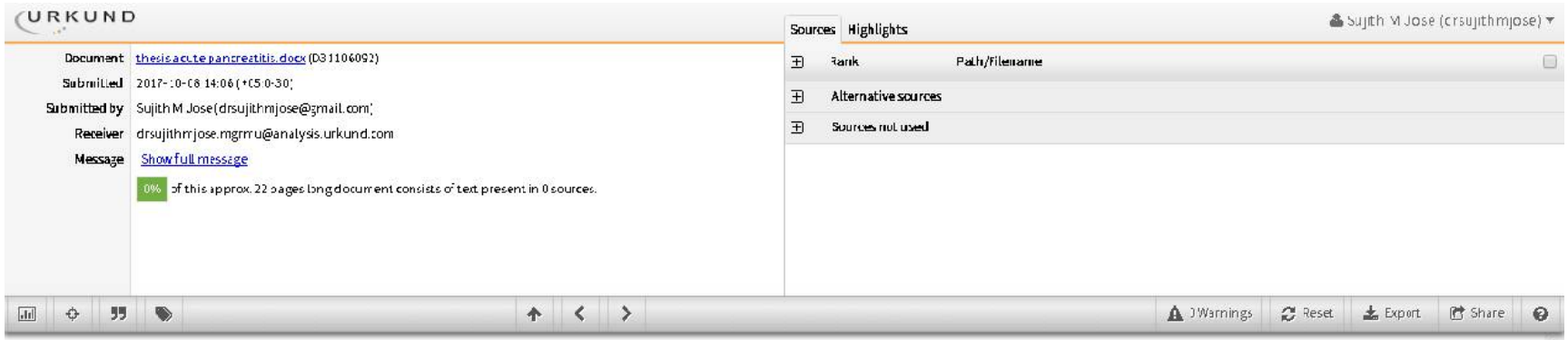

INTRODUCTION Acute pancreatitis is an acute inflammatory process involving pancreas and peri-pancreatic tissue, with a range of severity as well as various local and systemic complications. It is a common disorder causing a substantial burden on the healthcare system. The process involved in acute pancreatitis is complex in which pancreatic damage is caused by pancreatic enzyme activation, which results in an acute inflammatory response. Usually, the clinical course of acute pancreatitis is mild and it resolves without any sequelae and carries the essentially minimal risk of mortality. But severe disease associated with Multi-Organ Dysfunction Syndrome (MODS) is present in 10-20% of patients. In this subset of patients, mortality rate reaches up to 30%. However, the individual patient response to pancreatic injury is highly variable and unpredictable. Clinical biomarkers play a vital role in early patient triage, management and in predicting the development of life-threatening complications. Patient with severe disease benefit from early detection of organ failure, antibiotic administration, and treatment for the etiological factors. The patient outcome can be increased by early detection of the severity of disease and triaging patients correctly towards intensive care units based on severity. Gallstones and alcoholism are associated with 80% of cases of acute pancreatitis. Individual association of alcohol and gallstones varies geographically. In a population where alcohol consumption is common, the incidence of alcohol induced pancreatitis is substantially high. Trauma and drugs also cause acute pancreatitis. Severe acute pancreatitis is defined by revised Atlanta classification of 2012 by the presence of organ failure that persists more than 48 hours. Organ failure is determined by assessing Cardiovascular, Respiratory and Renal systems. Different scoring systems are being used to predict the severity of pancreatitis which includes APACHE II score, with 14 criteria and the RANSON'S score with 11 criteria. MODS score with 12 criteria and BISAP score with 5 criteria are the newer scoring system. Balthazar described CT severity index which was modified into MCTSI (Modified CT Severity Index) by Silverman et al in 2004. CTSI is calculated using CT scan features of acute pancreatitis and pancreatic necrosis. BISAP score is a 5 point bedside score. It is inexpensive to perform and easy to obtain. BISAP uses 5 points: Blood Urea Nitrogen (BUN) > 25mg/dl, impaired mental status evidenced by disorientation or disturbance in mental status, the presence of SIRS, age > 60 years and Pleural Effusion. BISAP score has been shown to be accurate in predicting the severity of acute pancreatitis in the western population. The aim of our study is to apply BISAP score to a semi-urban population in Coimbatore and assess the accuracy of BISAP and MCTSI in the prediction of severity, pancreatic necrosis, and

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ABBREVIATIONS

AP	:	Acute Pancreatitis
APACHE	:	Acute Physiology And Chronic Health Evaluation
APFC	:	Acute Peripancreatic Fluid Collection
ARDS	:	Acute Respiratory Distress Syndrome
BISAP	:	Blood Urea Impaired Mental Status SIRS Age Pleural Effusion
CARS	:	Counter Anti Inflammatory Response Syndrome
CBD	:	Common Blie Duct
CCK	:	Cholecystokinin
CECT	:	Contrast Enhanced Computed Tomography
CKD	:	Chronic Kidney Disease
CT	:	Computed Tomography
CTSI	:	Computed Tomograph Severity Index
ERCP	:	Endoscopic Retrograde Cholangio Pancreatography
GCS	:	Glasgow Coma Scale
IL	:	Interleukin
MCTSI	:	Modified Computed Tomograph Severity Index
MODS	:	Multi Organ Dysfunction

MRI	:	Magnetic Resonance Imaging
NPV	:	Negative Predictive Value
PAN	:	Polyarteritis Nodosa
PPV	:	Positive Predictive Value
SAP	:	Severe Acute Pancreatitis
SIRS	:	Severe Inflammatory Response Syndrome
TNF	:	Tumor Necrosis Factor
USG	:	Ultrasonography
VARD	:	Video Assisted Retroperitoneal Debridement

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INTRODUCTION

Acute pancreatitis is an acute inflammatory process involving pancreas and peri-pancreatic tissue, with a range of severity as well as various local and systemic complications. It is a common disorder causing a substantial burden on the healthcare system. The process involved in acute pancreatitis is complex in which pancreatic damage is caused by pancreatic enzyme activation, which results in an acute inflammatory response.

Usually, the clinical course of acute pancreatitis is mild and it resolves without any sequelae and carries the essentially minimal risk of mortality. But severe disease associated with Multi-Organ Dysfunction Syndrome (MODS) is present in 10-20% of patients. In this subset of patients, mortality rate reaches upto 30%.However,theindividual patient response to pancreatic injury is highly variable and unpredictable. Clinical biomarkers play a vital role in early patient triage, management and in predicting the development of life-threatening complications.

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Gallstones and alcoholism are associated with 80% of cases of acute pancreatitis. Individual association of alcohol and gallstones varies geographically. In a population where alcohol consumption is common, the incidence of alcohol-induced pancreatitis is substantially high. Trauma and drugs also cause acute pancreatitis.

Severe acute pancreatitis is defined by revised Atlanta classification of 2012 by the presence of organ failure that persists more than 48 hours. Organ failure is determined by assessing Cardiovascular, Respiratory and Renal systems.

Different scoring systems are being used to predict the severity of pancreatitis which includes APACHE II score, with 14 criteria and the RANSON'S score with 11 criteria. MOSS score with 12 criteria and BISAP score with 5 criteria are the newer scoring system. Balthazar described CT severity index which was modified into MCTSI (Modified CT Severity Index) by Silverman et al in 2004. CTSI is calculated using CT scan features of acute pancreatitis and pancreatic necrosis.

BISAP score is a 5 point bedside score. It is inexpensive to perform and easy to obtain. BISAP uses 5 points: Blood Urea Nitrogen (BUN) > 25mg/dl, impaired mental status evidenced by disorientation or disturbance in mental status, the presence of SIRS, age > 60 years and Pleural Effusion. BISAP score has been shown to be accurate in predicting the severity of acute pancreatitis in the western population.

The aim of this study is to apply BISAP score to a semi-urban population in Coimbatore and assess the accuracy of BISAP and MCTSI in the prediction of severity, pancreatic necrosis, and mortality in acute pancreatitis. If proved to be significant, we could avoid performing CT scan in patients with mild acute pancreatitis, which will drastically reduce the cost of treatment.

AIM AND OBJECTIVE

To compare BISAP (Blood Urea Nitrogen >25mg/dl, Impaired Mental Status, Systemic Inflammatory Response Syndrome, Age >60 and Pleural Effusion) score with modified computed tomography severity index(MCTSI) in predicting:

a) Severity

b) Pancreatic Necrosis

c) Mortality in patients with Acute Pancreatitis.

REVIEW OF LITERATURE

HISTORY OF PANCREATITIS

Pancreas originated from a Greek word which means “all flesh”. In TALMUD written between 200BC and 200AD, thepancreas was referred to as the finger of liver[1]. When William Harvey described circulation in Middle Ages, the pancreas was considered only as a pad to protect major vessels. It was Dr.Nicholas Tulp who first published a clear description of acute pancreatitis. In 1652 Dr.Reginald Filtz was the first to classify acute pancreatitis. He classified pancreatitis into hemorrhagic, suppurative and gangrenous forms. Pancreatic autodigestion as the pathophysiology behind acute pancreatitis was first postulated by Chiari. In 1896. Lord Moynihan quoted acute pancreatitis as[1]:

“ The most terrible of all calamities related with the abdominal viscera. The suddenness of its onset, the illimitable agony which accompanies it and the mortality attendant upon it, all renders it the most formidable of catastrophies.”

It is believed that Alexander the Great (323 BC) died out of Acute Pancreatitis.

ANATOMY OF PANCREAS

The pancreas is a retroperitoneal organ. It is divided into head, neck, body, and tail. Head constitute 30% of the gland by mass. Body and tail constitute rest 30%. Head occupies the space within the ‘C’ loop of duodenum[2]. The position of head corresponds to the body of second lumbar vertebrae. Behind the neck of pancreas lies aorta and superior mesenteric vessels. Superior mesenteric vein joins the splenic vein to form portal vein behind the neck of the pancreas. Tip of pancreatic tail reaches upto hilum of spleen.[3]

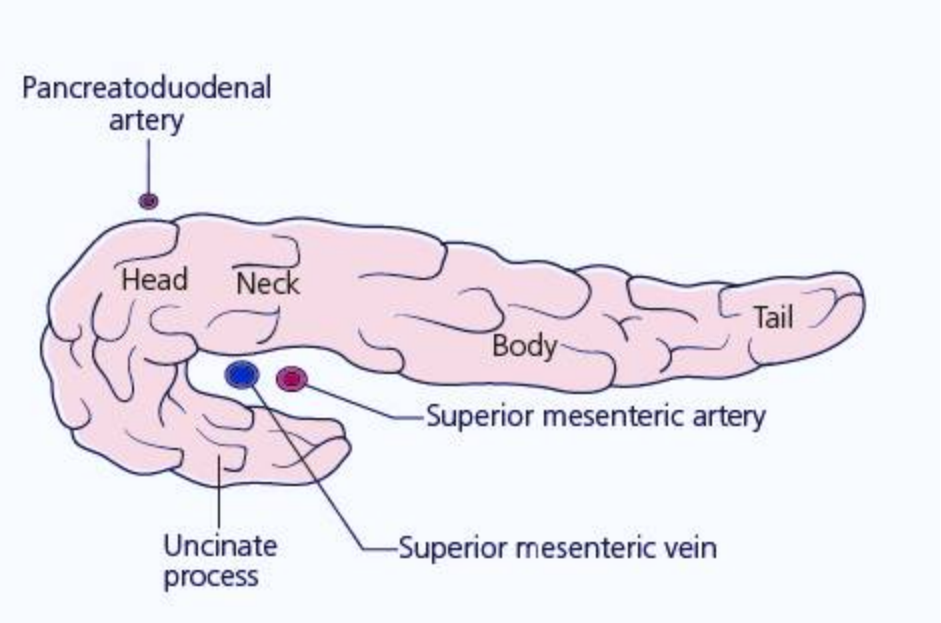

Figure 1. The five parts of the pancreas. The division between body and tail is arbitrary

The weight of pancreas is 80 grams. 90% of its mass is composed of exocrine acinar cells. Main pancreatic duct branches into interlobular, intralobular ducts, ductules and acni. The lining of pancreatic duct is by columnar epithelium. In ductules it becomes cuboidal. [4]Clusters of endocrine cells called Islets of Langerhans[1] are distributed throughout the pancreas. B cells producing insulin constitute 75% of islet cells. A cells producing glucagon contribute 20% of islet cells. Rest is formed by D cells which produce somatostatin. Pancreatic portal system is formed by capillaries that drain islet cells to portal vein.[5]

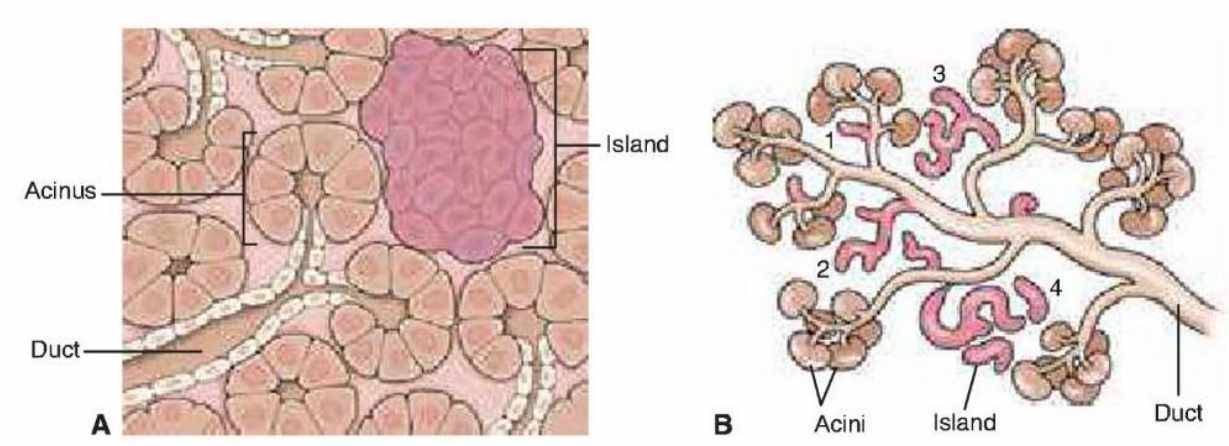

Figure 2. A:Pancreatic acini and islet cells B: Four progressive stages in the organization of islands.

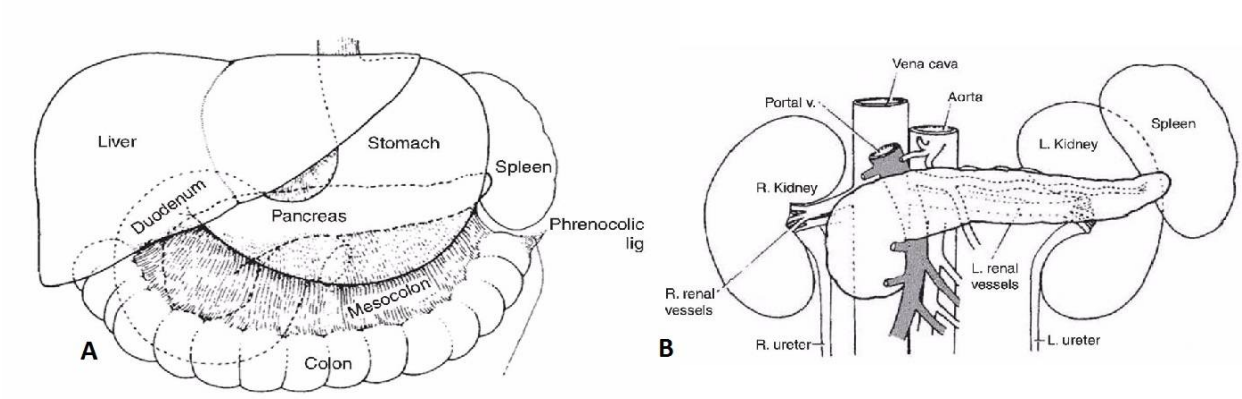

Figure 3. Relations of pancreas A: Anterior relation B: Posterior relation

PANCREATIC EMBRYOGENESIS

From the dorsal side of the duodenum, closed pancreatic duct arises by 26 days of gestation. Ventral bud arises from the base of hepatic diverticulum by day 32. The 2 buds come in contact by 37 days of gestation and its fusion occurs by the end of 6th week.[6] Head and uncinate process are formed from the ventral bud. The main pancreatic duct of Wirsung is formed from the ventral duct and distal portion of dorsal duct.[4] Proximal dorsal duct forms the duct of Santorini. All these processes of duct fusion occur by the6th week. Pancreatic acini and islets begin to appear by the3rd month. Annular pancreas is formed due to malrotation of ventral bud.

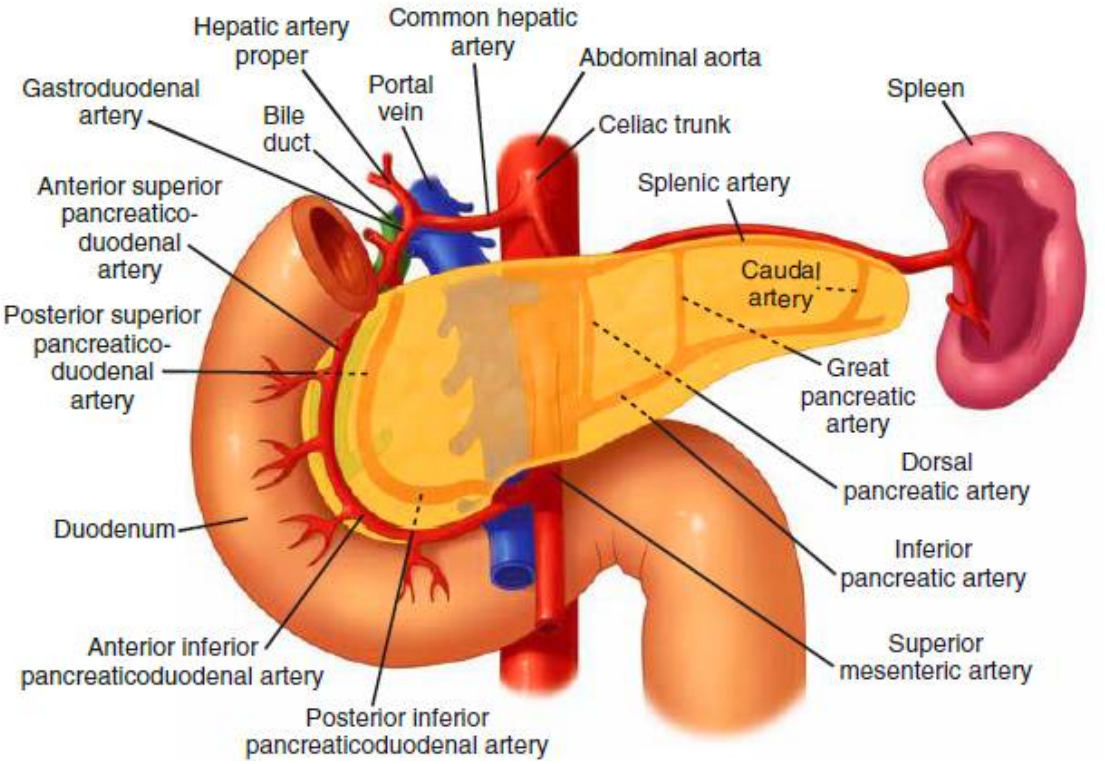

Figure 4. Blood supply of pancreas

PHYSIOLOGY

After meals, pancreas secretes digestive enzyme in an alkaline bicarbonate rich fluid of pH 8.4. Secretin is produced from duodenal mucosa.[7]

It produces bicarbonate rich fluid in response to food. Duodenal mucosa produces cholecystokinin.CCK is responsible for enzyme release.. Volume of secretion is increased by vagal stimulation.[2]

DEFINITION

Pancreatitis is defined as inflammation of parenchyma of the pancreas. It is divided into acute and chronic pancreatitis based on disease process and clinical presentation.[8]Acute Pancreatitis presents as an acute abdomen with abdominal pain and is usually associated with a raised pancreatic enzyme in blood or urine.[9] It is due to the prematureactivation of enzymes within the pancreas, leading to autodigestion of pancreas.[10]

ETIOLOGY

Many factors have been identified to cause acute pancreatitis. But the exact mechanism in all cases is poorly understood.Gallstones and alcohol cause 80% of acute pancreatitis.[3] In some cases no specific etiological agents are identified. In order to identify uncommon and modifiable risk factors, a systemic approach to acute pancreatitis is important.[11]Etiology plays an important role in determining the median age of presentation of aute pancreatitis. For example, the median age of presentation of alcohol induced pancreatitis is in the 3rd and 4th decade and gallstone and trauma induced pancreatitis present is in the sixth decade. Etiology also influences the gender difference in presentation. In males alcohol more often causes pancreatitis but in female gall stones[12].Etiological factors of Acute

Pancreatitis are classified into metabolic factors, mechanical factors, vascular factors and infection.[13]

METABOLIC FACTOR

- Alcohol
- Hyperlipoproteinemia[11]
- Hypercalcemia
- Hyperparathyroidism[10]
- Drugs like 5-Aminosalicylates[11], 6 mercaptopurine, azathioprine, tetracyclinesvalproic acid, L-asparginase and diuretics like frusemide and thiazides.[3]
- Scorpion venom

MECHANICAL FACTOR

- Cholelithiasis[14]
- Postoperative patients
- Congenital anomalies like Pancreatic Divisum[4]
- Post abdominal trauma
- Post ERCP[15]
- Pancreatic duct obstruction by tumor, ascaris.

VASCULAR FACTOR

- Postoperative (cardiopulmonary bypass)
- PAN(Poly Arteritis Nodosa)
- Thromboembolism

INFECTION

- Coxsackie B virus
- Cytomegalovirus
- Mumps & Cryptococcus

Surgeries causing pancreatitis include[8]:

a. Surgeries in and near pancreas

- 1.Pancreatic Biopsy
- 2. Distal Gastrectomy
- 3.Splenectomy
- 4.CBD exploration

b. Surgeries using low systemic perfusion like

- Cardiac bypass(cardiopulmonary bypass)
- Cardiac transplantation

GALLSTONES

Gall stone is an important etiology for the development of acute pancreatitis. In patients with acute pancreatitis, the liver function test is usually deranged. Also gall stones are retrieved from the faeces of patients with acute pancreatitis within 10 days of an attack of AP. These factors provides evidence to support that passage of gallstones may be the factor behind the development of Acute Pancreatitis[14].

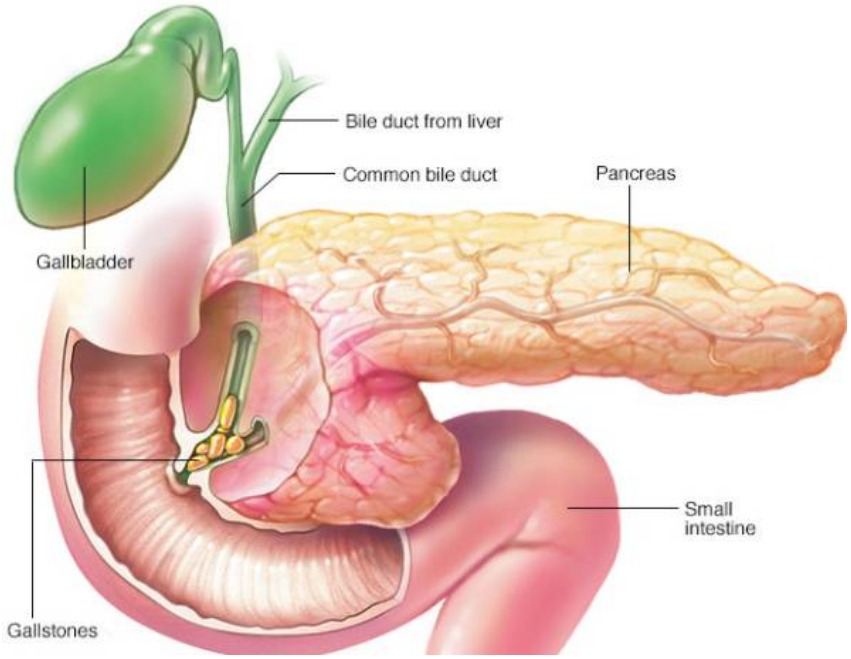

Figure 5. Gallstone causing pancreatitis

The mechanism by which gallstone causes acute pancreatitis is explained by “common channel” hypothesis. It is proposed that reflux of bile into pancreatic duct occurs when gall stones lodge in distal part of common

bile duct near Ampulla of Vater. Another study suggests that transient incompetence to the sphincter is caused by the passage of stone through the sphincter. This incompetence will lead to reflux of bile and duodenal fluid to pancreatic duct[16]. A third study propose that gallstone may obstruct pancreatitis duct directly leading to ductal hypertension which leads to back pressure and ductal disruption and extravasation of pancreatic juices.[17]

ALCOHOL

Alcohol consumption is related with acute pancreatitis, recurrent pancreatitis, and chronic pancreatitis. Amount of alcohol consumed is more important than the type of alcohol consumed. The pattern of drinking is also important. There is always a history of excess alcohol consumption prior to the first attack. Ethanol damages acinar cells and disturbs its metabolic activity. Acute pancreatitis is triggered by secretory burst following alcohol intake coupled with ethanol induced spasm of the sphincter of Oddi.[10] Ethanol increases the ductal permeability which leads to leakage of pancreatic enzymes causing damage to pancreatic parenchyma. Protein content of pancreatic juice is increased by alcohol thereby forming protein plug and cause obstruction to pancreatic outflow.[10]

IATROGENIC

Procedures like biopsy of pancreas, distal gastrectomy, exploration of common bile duct, repair of aortic aneurysm and retroperitoneal lymph node excision are some iatrogenic causes of acute pancreatitis. Splanchnic hypoperfusion with cardiopulmonary bypass and cardiac transplant can lead to pancreatitis as pancreas is highly susceptible to ischemia[18]. Worldwide post ERCP accounts for 3rd most common identified etiological factor for AP. If contrast is used repeatedly in ERCP, the chance of developing AP is much higher.

TECHNIQUES TO REDUCE ERCP- INDUCED PANCREATITIS

Avoid solely diagnostic ERCPs to the maximum. It should be replaced by low risk tests such as MRCP. Perform ERCP as a therapeutic procedure whenever possible. Limiting the number of attempts of pancreatic duct cannulation and limiting the number of contrast injection into pancreatic duct and slow injection of contrast are other methods to reduce ERCP induced pancreatitis. A transpapillary pancreatic duct stent must be placed in high risk patients.

HEREDITARY PANCREATITIS[19]

It is an autosomal dominant disorder due to mutation of cationic trypsinogen gene (PDSS-1). Mutation in this gene causes premature activation of trypsinogen to trypsin and promote acute pancreatitis. Mutation in SPINK -1 protein gene will also lead to acute pancreatitis.

PANCREATIC DIVISUM

Pancreatic divisum is a common congenital disorder seen in 7% of population.The absence of fusion of dorsal and ventral bud leads to this anomaly. Pancreatic divisum may result in a stenosed or inadequately patent minor duct papilla which prevents normal drainage of pancreatic secretion. This may result in increased intraductal pressure. The relation between pancreatic divisum and pancreatitis is highly controversial. Another controversial issue is the relation between Sphincter of Oddi Dysfunction and acute pancreatitis. This also results in increased intraductal pressure. Biliary sludge is a viscous suspension of bile and it consists of cholesterol crystals and calcium bilirubinate granules which are embedded in strands of gallbladder mucus. Sludge is associated with bile stasis and biliary duct obstruction. In patients with recurrent acute pancreatitis, biliary sludge is commonly seen. Such patients are benefited by cholecystectomy.

PATHOPHYSIOLOGY OF ACUTE PANCREATITIS

Acute pancreatitis is the end result of abnormal enzyme activation within the pancreatic acinar cells.[20] The earliest step is co-localization of zymogen granules and lysosomes inside the acinar cells. This step occurs well before the elevation of serum amylase and onset of pancreatic edema.[21]Once cathespin-B in lysosomes and trypsinogen in zymogen granules are brought in contact by co–localization induced by pancreatitis,trypsinogen gets activated to trypsin[22]. This trypsin induces leak of colocalized organelles,releasing more cathespin-B into cytoplasm leading to apoptosis and necrosis of acinar cells.

Intra acinar enzyme activation induces autodigestion of normal pancreatic parenchyma, leading to the release of proinflammatory cytokines such as tumor necrosis factor –alpha (TNF –α) and interleukins IL-1,IL-2 & IL-6.[20]These inflammatory cytokines recruit neutrophils and macrophages into the pancreatic parenchyma and cause release of more TNF-α, IL-1 and IL-6,reactive oxygen species,prostaglandins, platelet activating factors and leukotrienes.[4] These inflammatory mediators increase the permeability and damages the microcirculation and further aggravates pancreatitis. In severe cases, local hemorrhage and pancreatic necrosis occurs.[23]

In 80-90%of cases, this inflammatory cascade is self-limiting. In rest of patients,a vicious cycle of pancreatic injury ,local and systemic inflammatory reaction occurs. This leads to them assive release of inflammatory mediators into systemic circulation causing Multi Organ Dysfunction .[16] Active neutrophils causes acute lung injury and induce Adult Respiratory Distress Syndrome. Most common cause of death in Acute Pancreatitis is Multi Organ Dysfunction Syndrome (MODS).[24]The first sign of MODS in Acute Pancreatitis is impaired lung function due to ARDS. Mortality in first two weeks is due to multi-organ dysfunction. Death in late phase is due to sepsis related complications.

To summarize, cathepsin B mediated intra acinar cell activation of digestive enzymes leads to acinar cell injury which triggersinflammatory response. The released proinflammatory cytokines propagate response locally and systemically.[24]

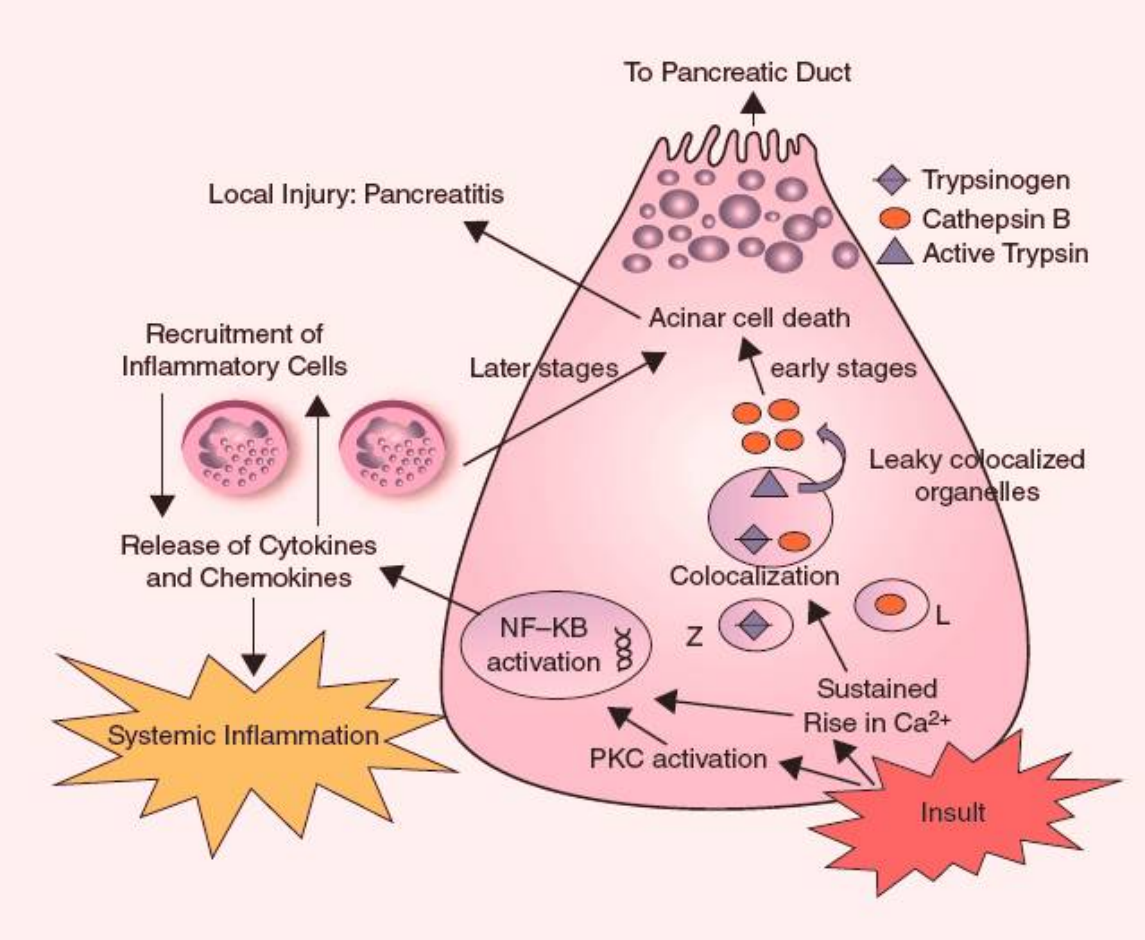

Figure 6. Stimulation of acinar cells leads to colocatisation of lysosomes and zymogen.Intracytosolic calcium is needed for colocalisation.Cathepsin B activates trypsingen to trypsin. Trypsin results in apoptosis.Mitochondria releases cytochrome c. Released cytokine attracts inflammatory response cells, finally leading to systemic and local inflammatory response.

CLINICAL PRESENTATION OF ACUTE PANCREATITIS

Most common symptom of pancreatitis is pain. Maximum intensity is reached within minutes and pain persists for hours. Pain is constant and refractory to the usual dose of analgesics[10]. Pain is first experienced in

epigastrium and it may gradually localize to either upper quadrant or diffusely over entire abdomen.

Radiation of pain to back is present in 50%of cases. Patient experience mild relief of pain on stooping forward. Acute pancreatitis mimics most of the acute abdominal conditions. Nausea, vomiting ,retching may be associated with pain.[25]

On examination, the general appearance can vary from normal to severely ill depending on the severity of the disease. In patients with severe acute pancreatitis tachypnea, tachycardia and hypovolemia may be present[9]. Due to the presence of inflammation, the patient may have elevated body temperature. Acute swinging pyrexia suggest cholangitis.[26]

Bleeding into the fascial planes can produce discoloration of abdominal wall. Bluish discoloration of flanks is known as Grey Turner sign and that of umbilicus is known as Cullen's sign. Subcutaneous fat necrosis leads to small red tender nodules on the skin of legs.[27]

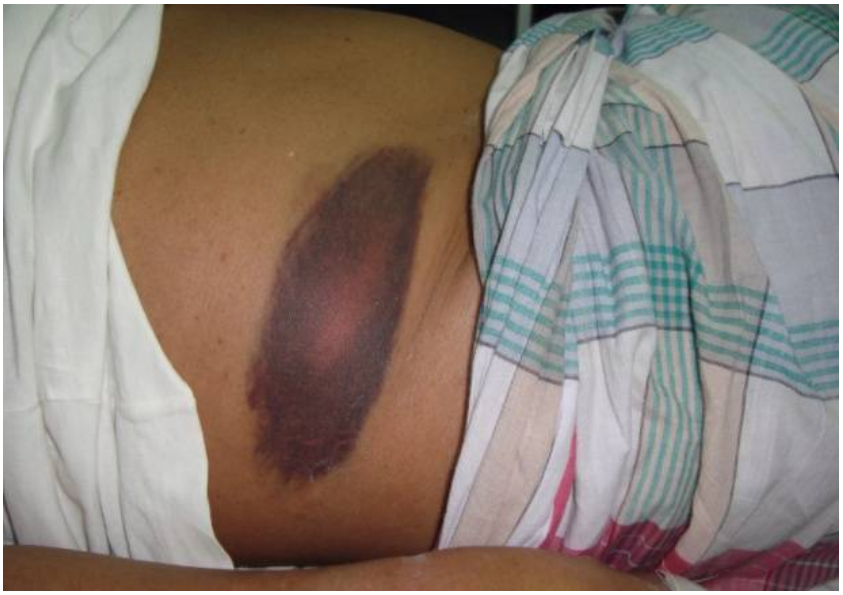

Figure 7. Grey Turner sign

On abdominal examination, distension may be present if there is ileus. Ascites may be present presenting as shifting dullness. Guarding in the upper abdomen is commonly seen but rigidity is rarely seen.[3] Pleural effusion present in 10-20% patients signs of metabolic derangement and hypoxemia may be seen in severe cases.

INVESTIGATIONS

Serum amylase level usually increases to 3-4 fold level. It is one of the diagnostic criteria of acute pancreatitis. [28]A normal serum amylase level does not exclude acute pancreatitis. If the patient presents late, amylase value may be high. Serum lipase level is more sensitive and specific than serum amylase. The single best investigation for acute pancreatitis is

contrast enhanced CT[29]. Sentinel loop sign, colon cut-offsign and renal halo sign are seen in plain X-ray abdomen in acute pancreatitis. Because of spasm of distal bowel overlying inflamed pancreas, the bowel is focally dilated proximally and is called sentinel loop sign. Colon cut off sign is the focal dilation of mid transverse colon. It is due to the extension of peripancreatic inflammation and bowel spasm at splenic flexure. Widening of C- loop of duodenum occurs due to edema and inflammation of pancreatic head. Calcification may be present in X-ray. Chest X-ray may show pleural effusion or findings of ARDS.

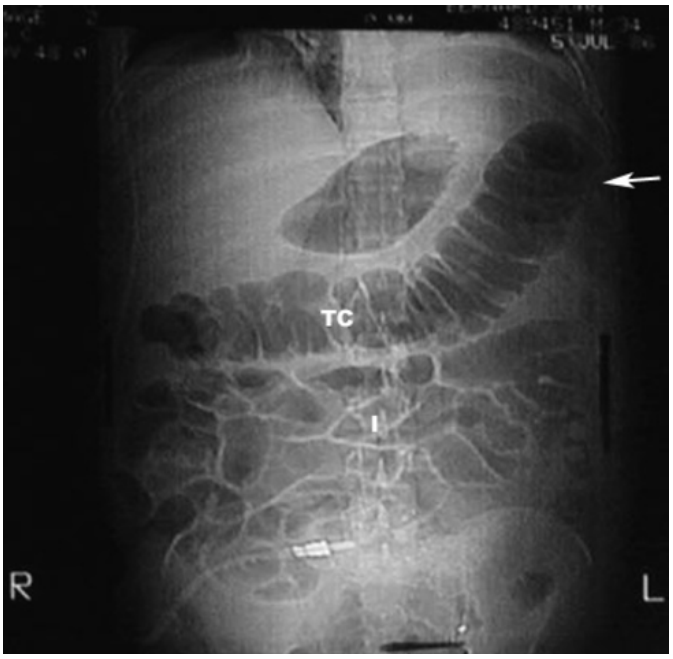

Figure 8. Colon cut off sign shown by arrow

In ultrasonography, swollen and edematous pancreas may be visualized. Gallstones can also be demonstrated in USG.

Indications of contrast enhanced CT in Acute Pancreatitis are[29]:

- When there is diagnostic uncertainty.
- In patient with severe acute pancreatitis.
- In the presence of organ failure and signs of sepsis.
- In the presence of localized complications such as fluid collection, pseudocyst, and pseudoaneursym.

DIAGNOSIS

The diagnosis of Acute Pancreatitis requires two of the following three features:[30]

1.Abdominal pain consistent with acute pancreatitis (onset of an acute, severe, persistent, epigastric pain which often radiates to the back)

2. Serum amylase or lipase level at least three times higher than the upper limit of normal range.

3.Characteristic sign of Acute Pancreatitis on contrast enhanced computed tomography (CECT), magnetic resonance imaging (MRI) or transabdominal ultrasonography.

CLASSIFICATION OF ACUTE PANCREATITIS

Acute pancreatitis is classified into 2 types:[31]

1. Acute interstitial oedematous pancreatitis

2. Acute necrotizing pancreatitis

ACUTE INTERSTITIAL OEDEMATOUS PANCREATITIS

It accounts for around 80-90% of cases. it is a milder form of Acute Pancreatitis and usually, resolves in one week. The pancreas is diffusely enlarged(occasionally localized)due to inflammation[23]. In contrast CT there is homogenous enhancement of contrast in pancreas with no evidence of necrosis in pancreatic parenchyma and peripancreatic tissue. The peripancreatic fat shows haziness or mild stranding due to inflammation ,minimal peripancreatic fluid may also be present.

ACUTE NECROTIZING PANCREATITIS

It is characterized by the presence of tissue necrosis in pancreatic parenchyma, peripancreatic tissue or both. It is a more aggressive form of acute pancreatitis and most commonly manifests as necrosis of both pancreatic & peripancreatic tissue, less commonly as necrosis of peripancreatic tissue alone and rarely as pancreatic necrosis alone.[32] The

extent of necrosis is underestimated by early CECT as compromise of pancreatic perfusion and features of peripancreatic necrosis evolve over several days. Necrotising pancreatitis has a variable course as the necrotic tissue may remain sterile or get infected, may remain solid or liquefy, may persist or disappear over time[9].

COMPLICATION OF ACUTE PANCREATITIS

LOCAL COMPLICATIONS

When there is persistent abdominal pain, secondary elevation in pancreatic enzyme level, progressive organ dysfunction or signs of sepsis, local complications of Acute Pancreatitis are suspected. Local complications are acute peripancreatic fluid collection, pancreatic pseudocyst, acute necrotic collection, walled off necrosis, gastric outlet obstruction, colonic necrosis, splenic or portal vein thrombosis.[2]

1.ACUTE PERI - PANCREATIC FLUID COLLECTION

The peripancreatic fluid collection is associated with interstitial edematous pancreatitis with no necrosis within first four weeks after onset of disease. Imaging shows a homogenous collection of fluid adjacent to pancreas without a well defined wall encapsulation.

2.PANCREATIC PSEUDOCYST

A pseudocyst is a collection of pancreatic juices that arise as a consequence of acute pancreatitis, pancreatic trauma or chronic pancreatitis and is enclosed by a nonepithelialized wall (Atlanta International Symposium 1992).Imaging shows well circumscribed homogenous fluid collection with well defined wall[33].Pseudocyst occurs in 5-15% of patient who has APFC. The wall is made of collagen or granular tissue and fluid is rich in amylase.

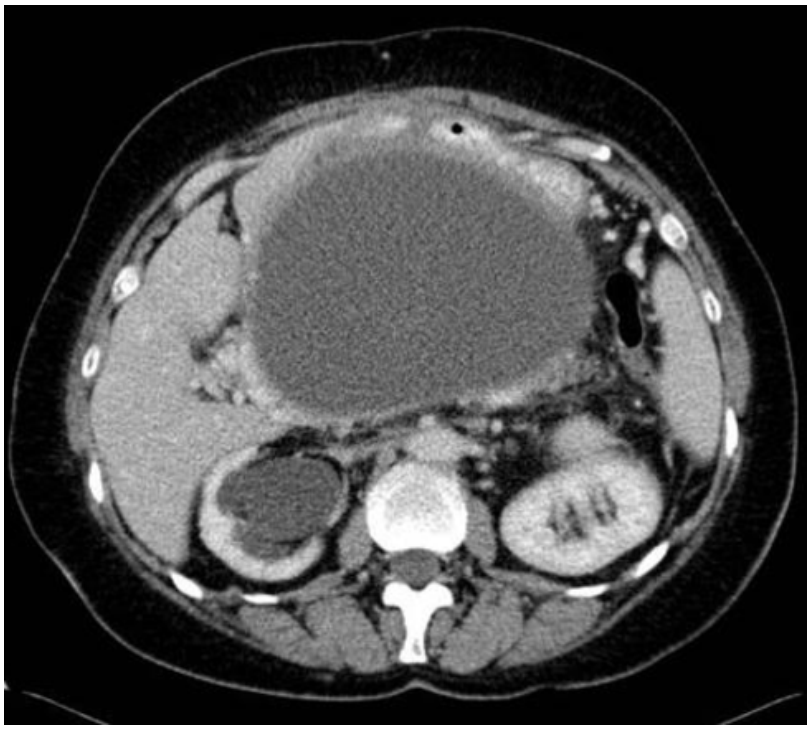

Figure 9. Pancreatic Pseudocyst

3.ACUTE NECROTIC COLLECTION

It is a collection of solid and liquid components without a well defined wall involving pancreatic extra pancreatic tissue or both in the setting of necrotizing pancreatitis. Imaging studies shown an encapsulated heterogenous collection of varying density.

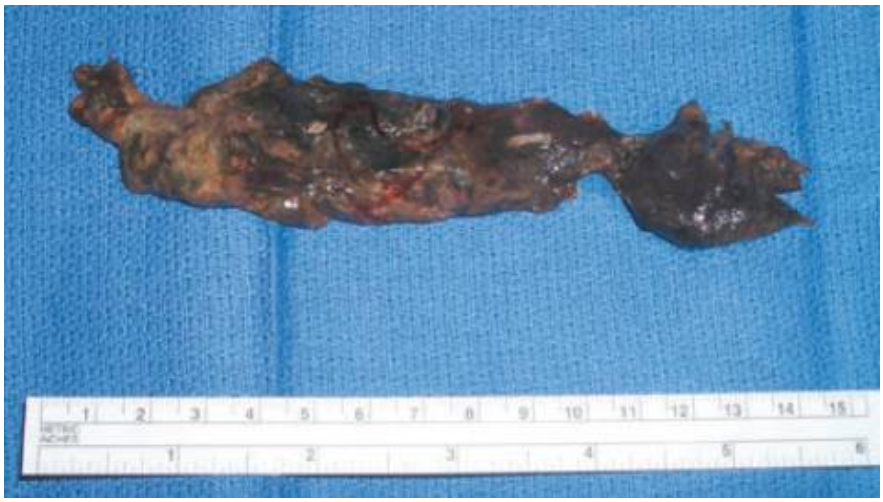

Figure 10. Pancreatic Necrosis

4.WALLED OFF NECROSIS

It is an encapsulated collection in the setting of necrotizing pancreatitis, four weeks after onset of disease. Imaging shows heterogeneous collection with varying degree of localization with well defined wall.[10]

5.INFECTED PANCREATIC NECROSIS

The necrotic tissue rarely gets infected. Infection can be suspected when there are signs of ongoing sepsis and confirmed by image guided fine needle aspiration, which may show frank pus or presence of bacteria and fungi on microscopic examination. Infected pancreatic necrosis requires antibiotic and the infected material needs to be drained.

Pancreatic ascites, pancreatopleuralfistula,pancreatocutaneous fistula and vascular complications are other local complications of acute pancreatitis

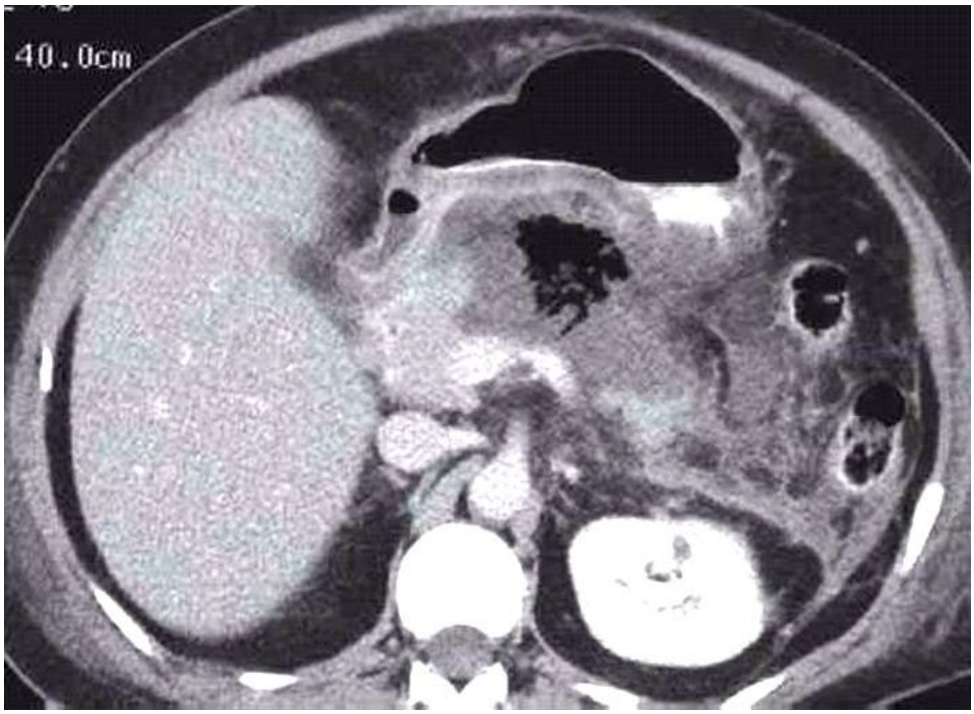

Figure 11.Infected Pancreatic Necrosis. The air bubble in necrotic debris indicates infected necrosis

SYSTEMIC COMPLICATIONS

Worsening of pre-existing comorbidities such as coronary artery disease, obstructive pulmonary disease in the setting of acute pancreatitis is considered under systemic complications.

ACUTE PANCREATITIS IN PREGNANCY

The incidence of acute pancreatitis in pregnancy is 0.1%. Gestational sex hormones like estrogen have cholestatic effect and therefore gallstones are the most common cause of acute pancreatitis in pregnancy. As in other cases of acute pancreatitis, the presentation is with typical epigastric pain which radiates to back. As serum lipase levels are unaffected by pregnancy, it is of high diagnostic significance in pregnancy. In a normal pregnancy, there is mild elevation in serum amylase levels. Cholelithiasis and bile duct dilation are preferably detected by abdominal ultrasonography. CT is avoided in pregnancy. Due to the presence of overlying gravid uterus and bowel gas, the pancreas is usually poorly visualized in pregnancy. The course of acute pancreatitis is usually mild during pregnancy and respond well to medical therapy. For symptomatic choledocholithiasis during pregnancy, an endoscopic sphincterotomy is a safe option.

SEVERITY OF ACUTE PANCREATITIS

The revised Atlanta classification of acute pancreatitis (2012) is an international multidisciplinary classification of Acute Pancreatitis severity. It is an update of 1991 Atlanta classification of acute pancreatitis. It classifies acute pancreatitis into Mild, Moderate and Severe acute pancreatitis.[9]

In mild pancreatitis[32] there is no organ failure. Moderate acute pancreatitis is defined by transient organ failure less than 48 hrs. Severe acute pancreatitis is defined by persistent organ failure that persists longer than 48 Hrs.

Organ failure is defined by :

- Shock – Systolic Blood Pressure of<90 mmHg
- Pulmonary Insufficiency –Pao2 <60 mm Hg at room air or need for mechanical ventilation
- Renal Failure – Serum creatinine level>2 mg/dl after rehydration or hemodialysis
- Gastrointestinal Bleeding>500ml/24 hrs

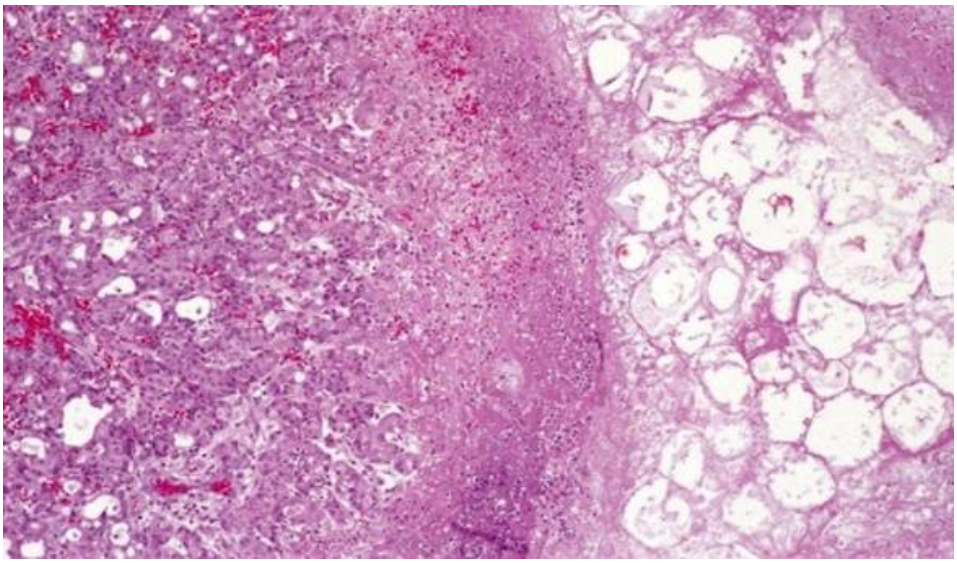

Figure 12. Histology of acute pancreatitis showing hemorrhageand inflammatory cells.

ASSESSMENT OF SEVERITY OF ACUTE PANCREATITIS

There are different scoring systems that are designed to evaluate the severity of Acute Pancreatitis.

RANSONS SCORE[34]

The earliest scoring system is Ransons (1979). It predicts the severity of disease on the basis of 11 parameters obtained at the time of admission & 48 hours later[35]. The advantage of this scoring system is that it does not predict the severity of the disease at the time of admission because six parameters are assessed only after 48 hours of admission.

RANSONS SCORE FOR NON GALLSTONE PANCREATITIS

At Presentation

- Age > 55 years
- Blood glucose level > 200 mg/ dl
- Lactate dehydrogenase level > 350 IU/L
- Aspartate amino transferase > 250 IU/L

After 48 hours of admission

- Hematocrit decrease > 10 %
- Serum Calcium level < 8 mg /dL
- Base deficit > 4 mEq/L
- Blood urea nitrogen level increase > 5mg/dL
- Fluid requirement > 6 litres
- PaO2 < 60 mm Hg

In the case of Gallstone pancreatitis, cut off values for some parameters changes. Age more than 70 years, Blood glucose level more than 220mg/dl, WBC more than 18,000 cells/mm3 and LDH more than 400 IU/liter are taken at admission. In the after 48 hours calculation, base deficit more than 5mEq/L, blood urea nitrogen level increase more than 2mg/dl and fluid requirement more than 4 litres are taken into consideration.

APACHE II

Acute physiology and chronic health evaluation(APACHE II)score also addresses the severity of acute pancreatitis. It 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. The advantage of APACHE II scoring system is that it can be used on admission and repeated at any time. The disadvantage is that it is complex and not specific for acute pancreatitis[36]

Variables that determine APACHE II score are :[31]

- Rectal Temperature
- Mean Arterial Blood Pressure
- Heart Rate
- Respiratory Rate
- Oxygenation (PaO_2)
- Arterial pH and Bicarbonate level
- Serum Sodium, Potassium and Creatinine
- Hematocrit and White Blood Cell count
- Glasgow Coma Scale

CT SEVERITY INDEX (CTSI)[29]

Balthazar and his associates established the CT severity index using imaging characteristics. This index correlates CT finding with the patient's outcome.

Following are the parameters considered in CTSI for acute pancreatitis:

PANCREATIC INFLAMMATION	POINTS
❖ Normal Pancreas	0
❖ Enlargement of the Pancreas	1
❖ Peripancreatic inflammation	2
❖ Single acute peripancreatic fluid collection	3
❖ Two or more peripancreatic fluid collection	4

PANCREATIC NECROSIS	
❖ None	0
❖ Less than 30 %	2
❖ 30% to 50%	4
❖ More than 50%	6
MAXIMUM POINTS	**10 POINTS**

CT severity index is now rarely used in practice. It is replaced by Modified CT severity index in 2004.It accounted for all the potential limitations of CTSI. It simplified the evaluation of pancreatic parenchymal necrosis and peripancreatic inflammatory changes. It also considered extrapancreatic complications in the assessment scale.

Parameters considered in the MCTSI are the following.

PROGNOSTIC INDICATOR	**POINTS**
PANCREATIC INFLAMMATION	
Normal Pancreas	Point 0
Intrinsic pancreatic abnormality	Point 2
Peripancreatic fluid collection	Point 4
PANCREATIC NECROSIS	
Absent	Point 0
< 30 percentage	Point 2
≥ 30 percentage	Point 2
EXTRA PANCREATIC COMPLICATIONS	Point 2

Extrapancreatic complications considered in the index are pleural effusion, ascitis, vascular complications, parenchymal complications and gastrointestinal tract involvement.

BISAP SCORE

Wu et al introduced BISAP score. It is simple 5 point bedside score and is easy to obtain and inexpensive. The BISAP score was originally retrospectively derived and validated based on a large population data [37]. Parameters assessed in BISAP score are:

❖ BUN	: Blood urea nitrogen > 25 mg /dl	1 Point
❖ Impaired Mental Status: GCS less than 15		1 Point
❖ SIRS	: Evidence of SIRS	1 Point
❖ Age	: Age more than 60 years	1 point
❖ Pleural effusion	: Evidenced by imaging study	1 point

PHASES OF ACUTE PANCREATITIS

Acute pancreatitis is divided into early and late phases, in pathophysiological terms.

A.EARLY PHASE

Early phase[22] occurs in the 1st week after onset. In this phase disease manifest as a systemic inflammatory response. Hence, host response to cytokine cascade lead to systemic inflammatory response(SIRS)or compensatory anti-inflammatory syndrome(CARS)and may progress to organ failure which may be single or multiple (multi-organ failure).In this phase ,clinical severity and treatment are mainly determined on the basis of type and degree of this organ failure

B. LATE PHASE

Late phase generally starts in the2nd week and can last for weeks to months. Late phase occurs only in patients with moderately severe or severe pancreatitis. This phase is defined by persistent organ failure and by local complications

MANAGEMENT OF ACUTE PANCREATITIS

Management of acute pancreatitis involves accurate diagnosis, proper triaging of patients, high quality supportive care, monitoring for early detection and treatment of complications and prevention of relapse.[16]

TRIAGE

All patients diagnosed to have acute pancreatitis should be hospitalized for supportive therapy and proper management. All cases of the first episode of pancreatitis must be admitted and evaluated to determine the specific cause. Patients with early signs of organ failure must be monitored in an intensive care unit. General supportive therapy to prevent complications, directed therapy for specific causes of pancreatitis and early recognition and treatment of complication are theprimary goal of therapy.

MANAGEMENT OF:

A.SYSTEMIC INFLAMMATORY RESPONSE SYNDROME PHASE

Adequate fluid resuscitation and adequate pain relief are the most important considerations in this phase.[31] Fluid should be given attaining a goal of 1mg/kg/hr urine output in this phase. In the initial 24 hours of severe acute pancreatitis close monitoring and intravenous fluid, supplementation are pivotal. Crystalloids are the ideal fluid of choice.[25]

B.COUNTERACTIVE ANTIINFLAMMATORY RESPONSE SYNDROME PHASE

Infection of pancreatic necrosis and peripancreatic collections must be thought of, if the patient deteriorates even after initial resuscitation. Decision on intervention is made based on clinical status of the patient.[2]

PREVENTION OF INFECTION

Many prophylactic strategies are tried to prevent infection as the infection is associated with increased mortality in acute pancreatitis. Enteral bacteria is the usual culprit of infection. These bacteria cross the mucosal barrier in the first 24 hours of disease. Intravenous antibiotics, enteral nutrition, selective bowel decontamination and enteral probiotics are tried to reduce infection.

Recent studies show that early enteral nutrition reduces small bowel bacterial overgrowth and improve intestinal mucosal barrier functions.

In patients with mild pancreatitis, oral feeding can be started after one day. In severe pancreatitis, enteral nutrition by nasojejunal feeding tube can be started by 3 days.

Recent studies disproved the beneficial effect of prophylactic systemic antibiotics in acute pancreatitis. Since gut is the source of bacteria responsible for the infectious complications,in acute pancreatitis,selective bowel decontamination with Norfloxacin ,Colistin and Amphotericin are under consideration[9].

INTERVENTIONS IN ACUTE PANCREATITIS

In the first 2 weeks, i.e phase of systemic inflammatory response syndrome, there is no room for intervention for pancreatic necrosis[38]. Acute interventions are justified only in the presence of :

- abdominal compartment syndrome
- bowel ischemia/ perforation
- severe bleeding unresponsive to angiographic coiling

Abdominal Compartment Syndrome is defined as intra abdominal pressure, more than 20 mm Hg with signs of new organ failure.

In severe biliary pancreatitis and cholestasis (Bilirubin 2.3mg/dl or dilated common bile duct) ERCP with endoscopic sphincterotomy reduces the risk of progression to complications.[39]

In the second counter anti inflammatory response syndrome phase(CARS),there is increased the risk of systemic infection or sepsis caused by secondary infection of pancreatic necrosis. The most accepted indication for intervention is documented on a suspected infection of pancreatic/ peripancreatic necrosis with signs of sepsis. The intervention can be done radiologically,endoscopically or surgically[40].

Choice and timing of intervention is determined by a multidisciplinary team. Based on the current literature postponding the intervention preferable until 4 weeks after onset of the disease is accepted as the strategy of choice. By this time the collection will be encapsulated and is referred as “walled off necrosis”. However, the length of interval is determined by the completeness of encapsulation and clinical condition of the patient.[41]

TYPES OF INTERVENTION FOR TREATING INFECTED NECROSIS

A .CATHETER DRAINAGE

It is the least invasive technique for treating infected necrosis. The drain is placed percutaneously through left retroperitoneum ortransabdominally. If patient fails to improve after adequate drainage, next step is performing necrosectomy. Percutaneous drain can be used as a

roadmap for necrosectomy. This two step approach ,first drainage, and second drain guided minimally invasive necrosectomy is called the STEP UP APPROACH

B . MINIMALLY INVASIVE NECROSECTOMY

It is video-assisted retroperitoneal debridement (VARD) procedure. In this procedure, Zero degree laparoscope is introduced guided by the previously placed drain. All necrotic material in reach are removed under direct vision. After debridement two large bore drains are kept into the empty cavity and continuously lavaged with 0.9% normal saline is done for 3 days

C .ENDOSCOPIC TRANSLUMINAL NECROSECTOMY

Endoscopic transluminal / transgastric necrosectomy is performed when VARD is not feasible. This procedure needs to be repeated multiple times to remove sufficient amount of necrotic material.

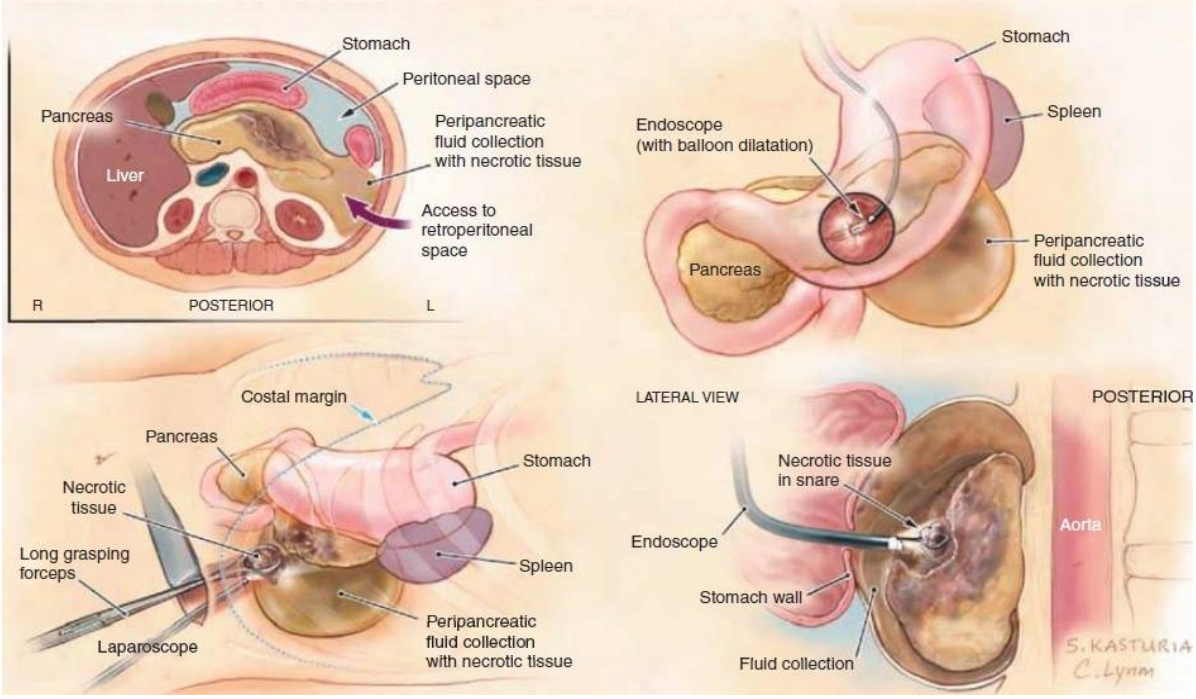

Figure 13.Video Assisted Retroperitoneal Debridement and Endoscopic Transluminal Necrosectomy

D. OPEN NECROSECTOMY

It is done in two ways. The first method includes laparotomy with placement of a retroperitoneal lavage system after complete necrosectomy [41]. Drains placed in lesser sac are continuously lavaged with normal saline. In the second method, after removing all necrotic tissue and debris,the resulting cavity is packed with gauge stuffed pentrose drains. These gauze stuffed drains are removed one by one after one week.

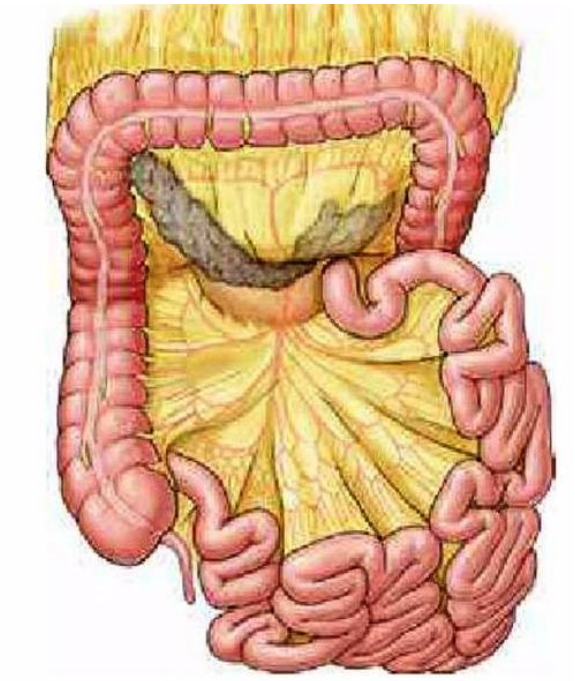

Figure 14. Necrosis is often seen through the transverse mesocolon after opening the abdomen.

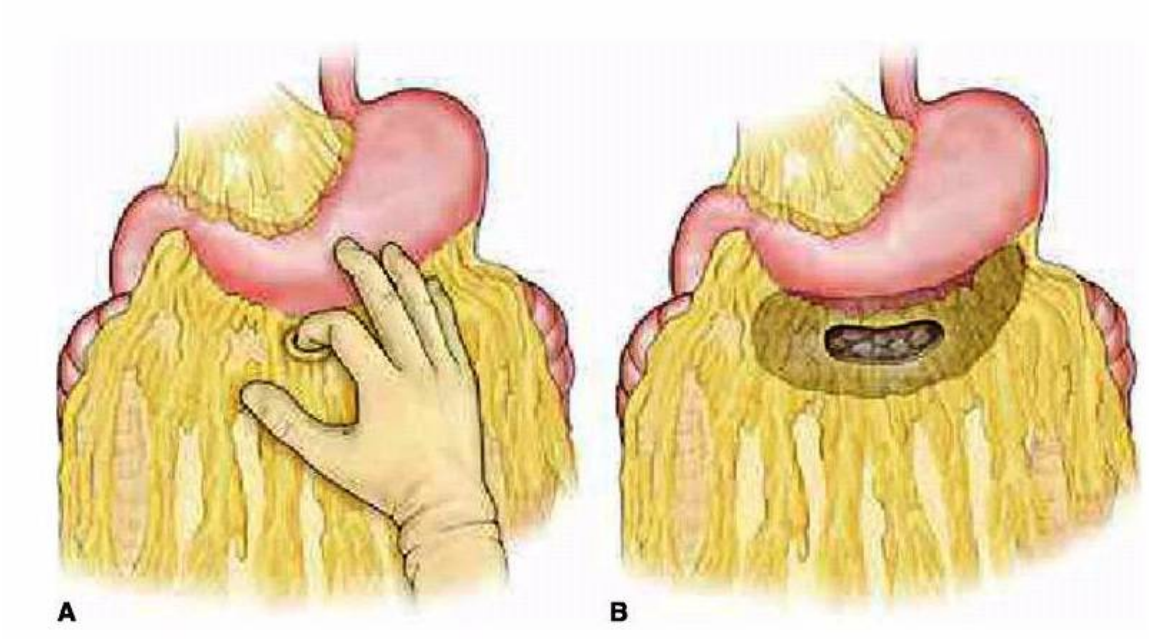

Figure15. A. During open necrosectomy, a small window is made ingastrocolic omentum B. Necrosis accessed through lesser sac by extending the window

FUTURE DIRECT OF RESEARCH

Till now the treatment of acute pancreatitis remains non specific and mainly supportive, focusing mostly on intensive care. Recent immune modulatory therapeutic attempts in experimental models is driven by the presence of inflammatory response syndrome during Acute Pancreatitis.[20] It includes cytokines, chemokines, immune cells and other inflammatory mediator blockade. Recent studies in animal models showed that TNF antagonism by either TNF receptor blockade or anti-TNF antibodies protected them from local intra pancreatic damage, systemic complications, and overall mortality. Administration of Infliximab appears to decrease serum amylase levels in both edematous and necrotic pancreatitis in murine models[17]

Pro-inflammatory cytokines such as IL- 6 and IL- 1 are released in acute pancreatitis with their plasma concentration correlates with severity of the disease and occurrence of multi organ failure. But anti inflammatory mediators such as IL-10 levels seems to be inversely proportional to the severity of pancreatitis

Blockade of the IL-1 receptor by either targeted genetic disruption or pharmacological agents reduced the extent of intra-pancreatic damage and

systemic complications. IL-10 has been shown to have a protective effect in several models of acute pancreatitis. It significantly ameliorates organ specific damage in the pancreas and peripancreatic tissue including lungs and liver.

Studies showed that when PAF antagonists were applied therapeutically, local intra pancreatic damage and micro circulating derangements were significantly reduced. Most promising among them are Lexipafant.

Expression of adhesion molecules is central for the development of endothelial barrier dysfunction, transmigration of neutrophils and development of organ dysfunction. Treatment with antibodies against adhesion molecules like ICAM – 1 and platelet endothelial cell adhesion molecules – 1 has shown to be effective in experimental models[42]

In murine model, Hydrocortisone has reduced mortality and blood cytokine levels. Treatment of acute pancreatitis by immune modulation currently represents an attractive and highly promising concept.

MATERIALS AND METHOD

The study was concluded in the Department of General Surgery, Coimbatore Medical College Hospital from July 2016 to June2017. This study was approved by the ethical committee of Coimbatore Medical College Hospital. Nature, methodology, and risks involved in the study were explained to the patient and informed consent was obtained. The information collected from the patients and their case records were kept confidential. All provisions of the Declaration of Helsinki were followed in this study.

STUDY DESIGN

Diagnostic Test Evaluation.

STUDY POPULATION

100 patients who were admitted to general surgical ward and diagnosed to have Acute Pancreatitis during the study period from July 2016 to June 2017 were included in the study.

INCLUSION CRITERIA

All patients admitted with the diagnosis of Acute Pancreatitis based on the presence of at least two of the following three criteria:

1. Characteristic epigastric abdominal pain, with or without radiation to the back.
2. Serum amylase or lipase levels elevated to at least three times the upper limit of normal.
3. Characteristic finding of Acute Pancreatitis on abdominal CT scan.

EXCLUSION CRITERIA

Patients with pre existing Chronic Kidney Disease (CKD) which may be associated with elevated Blood Urea Nitrogen values were excluded from the study as they may result in a high BISAP score.

METHOD

All patients with Acute Pancreatitis presenting to the Department of General Surgery who fit the inclusion criteria were included in the study after obtaining informed consent. Extensive demographic, radiographic and laboratory data which includes complete haemogram, serum electrolytes, renal function test, liver function test, serum amylase, lipid profile, chest X-ray, USG abdomen etc were collected. BISAP score was calculated using data from the first 24 hours from admission. A score of 1 is given for each criteria for a maximum score of 5.

CRITERIA FOR BISAP SCORE:

- BUN more than 25mg/dl
- Abnormal mental status with GCS < 15
- Evidence of SIRS
- Age > 60 years
- Presence of Pleural Effusion on X Rays

One point is given for each score.

SIRS is defined by the presence of >2 of the following criteria:

- Pulse rate > 90/min
- Respiratory rate > 20/min or PaCO2 < 32mm Hg
- Temperature > 100.4^{0}F or 96.8 ^{0}F
- WBC count > 12,000 or

  < 4,000 cells/ mm^{3} or

  >10% immature neutrophils.

MODIFIED CT SEVERITY INDEX

MCTSI was calculated from CECT within 48 hours.

Modified CT Severity Score is calculated as follows:

Normal Pancreas	Point 0
Intrinsic Pancreatic Abnormality with Peripancreatic fat stranding	Point 2
Peripancreatic fluid collection	Point 4
Pancreatic Necrosis	
Absent	Point 0
< 30 percent	Point 2
≥ 30 percent	Point 2
Extra Pancreatic Complications	Point 2

Using above Score total point is calculated.

Patients were closely monitored during the entire stay in hospital and evidence of organ failure documented. Patients were classified as mild acute pancreatitis and severe acute pancreatitis based on the presence of organ failure that persists for more than 48 hours.

Organ failure is defined by

i) Shock (Systolic BP <90 mm Hg)

ii) Pulmonary Insufficiency (PO_2 <60mm Hg at room air or need of mechanical ventilator)

iii) Renal Failure (serum creatinine>2mg/dl after rehydration or hemodialysis)

Pancreatic necrosis was assessed from CECT. Pancreatic necrosis is defined as lack of enhancement of pancreatic parenchyma with contrast. Comparison of prediction of severity of acute pancreatitis by BISAP and MCTSI score is the primary outcome of interest and comparison of prediction of mortality and pancreatic necrosis by both scores is the secondary outcome of interest.

STATISTICAL ANALYSIS

The data collected were entered in Microsoft Excel. The categorical data were expressed as percentage. The continuous data were expressed in Mean ± Standard Deviation. For the comparison of two groups, Unpaired T test was used. When more than two groups were compared ANOVA was used as the statistical tool. Categorical data and influence of the factors on severity were assessed using chi square test. For all analytical purpose, SPSS Software version 21.0 was used a value less than 0.05 was considered significant.

RESULTS AND OBSERVATION

AGE DISTRIBUTION

Table 1. Age Distribution

AGE	NO OF PATIENTS	PERCENTAGE
< 30 years	24	24%
31-40 years	41	41%
41-50 years	20	20%
51-60 years	6	6%
> 60 years	9	9%

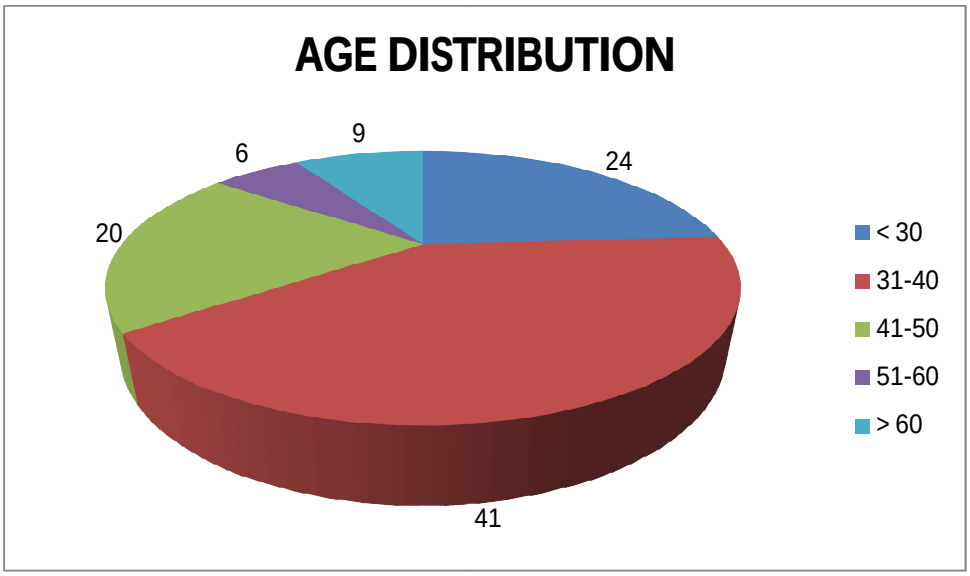

Chart 1. Age Distribution

Of the total of 100 patients studied in the study, most of the patients (41%) were in the age group of 31 – 40 years. 15% of patients were above 50 years and 24% of patients were below 30 years of age.

SEX DISTRIBUTION

Table 2. Sex Distribution

SEX	NO OF PATIENTS	PERCENTAGE
MALE	97	97%
FEMALE	3	3%

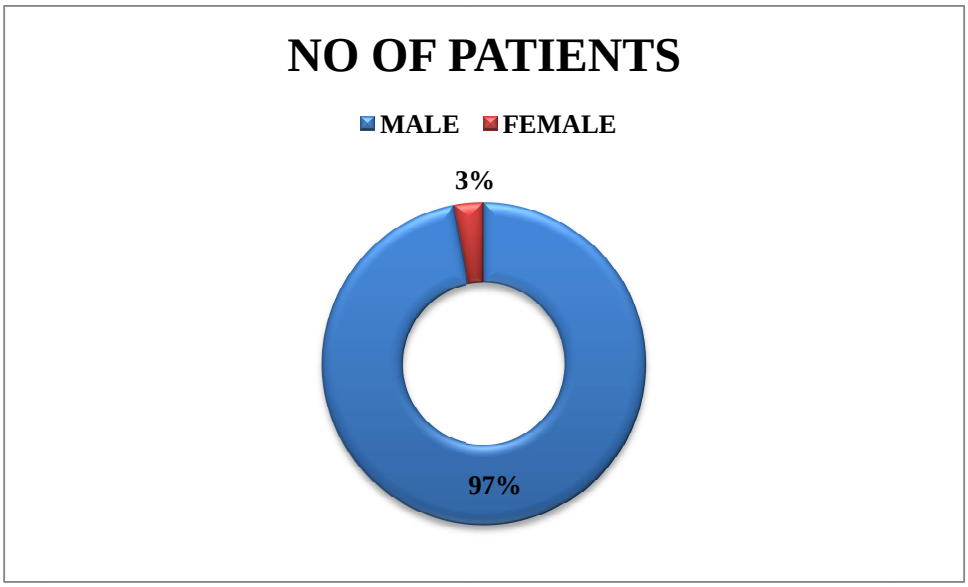

Chart 2. Sex Distribution

97% of patients in the study are males. Other Indian studies also revealed similar gender distribution pattern, the majority being males. This male preponderance is due to high incidence of alcohol consumption in a male population of India.

ETIOLOGY

Table 3. Etiology of Acute Pancreatitis

ETIOLOGY	NO OF PATIENTS	PERCENTAGE
ALCOHOL	46	46%
GALL STONES	27	27%
IDIOPATHIC	24	24%
POST ERCP	2	2%
HYPERLIPIDEMIA	1	1%

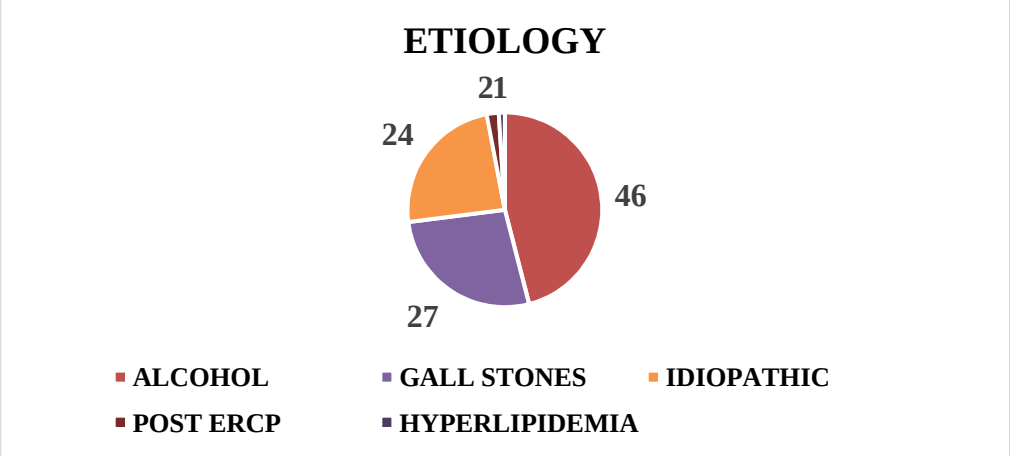

Chart 3. Etiology of Acute Pancreatitis

In our study, alcohol was found to be the most common etiology of acute pancreatitis contributing 46% of cases followed by gallstones contributing 27% of cases. Hyperlipidemia and Post ERCP were also found to be rare causes contributing 1 and 2 % respectively.

SIGNS AND SYMPTOMS

Table 4. Signs and Symptoms

SYMPTOMS	PRESENT	ABSENT
PAIN	100%	0%
RADIATION	64%	36%
VOMITING	78%	22%
GUARDING	88%	12%

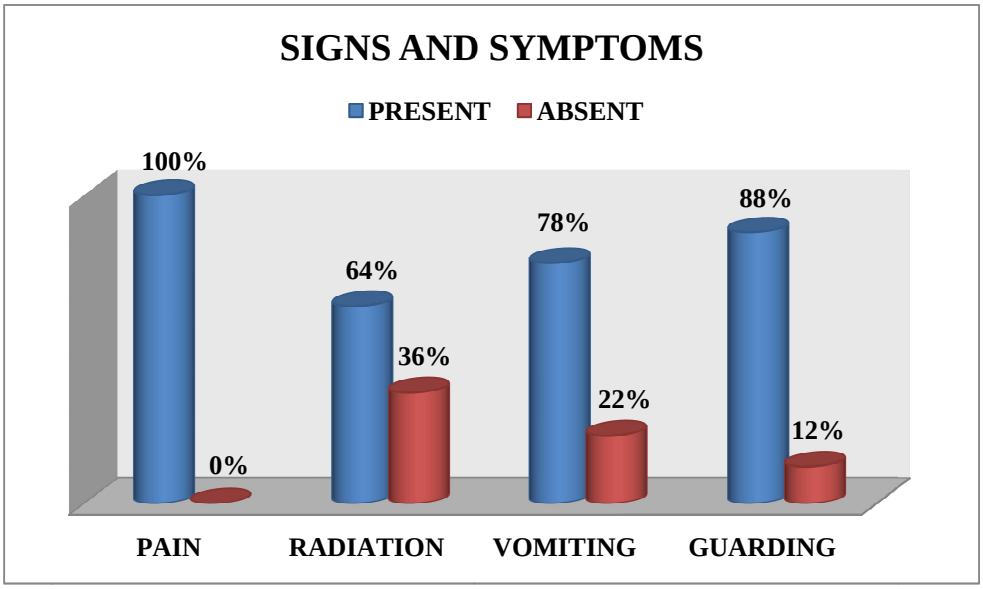

Chart 4. Signs and Symptoms

In our study, all patients presented with abdominal pain which was radiated to back in 64% of cases. The pain was associated with vomiting in 78% of cases.On examination, guarding was present in 88% of cases.

HOSPITAL STAY

Table 5. Hospital Stay

HOSPITAL STAY	NO OF PATIENTS	PERCENTAGE
< 5 DAYS	50	50%
5-10 DAYS	24	24%
> 10 DAYS	26	26%

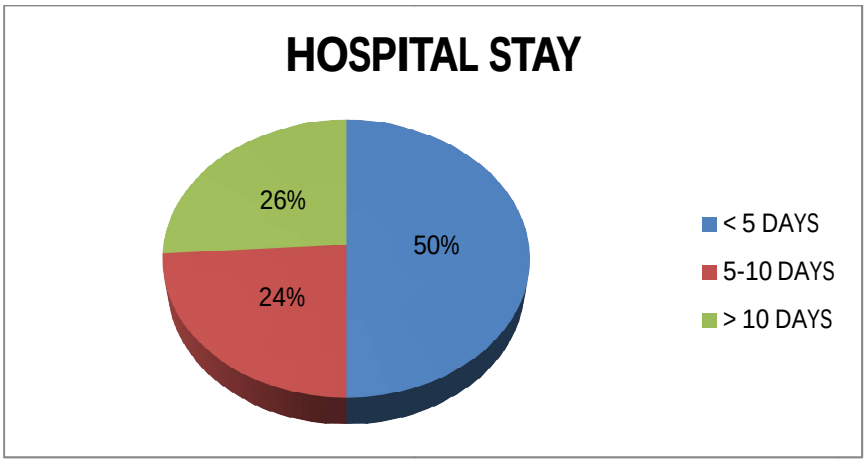

Chart 5. Hospital Stay

Most of the cases of acute pancreatitis (42%) were discharged in less than 5 days. 24% of patients stayed in the hospital for 5 – 10 days.In 26% of patients, the stay prolonged for more than 10 days. The length of hospital stay was found to be directly related to BISAP score and MCTSI score for discharged patients.

STATISTICS OF AGE, HOSPITAL STAY AND AMYLASE LEVEL

Table 6. Statistics of Age, Hospital Stay and Amylase level

		Age	Hospital Stay	Amylase
Mean		39.65	9.52	563.02
Median		38.00	5.50	464.50
Std. Deviation		12.373	8.736	232.255
Range		56	37	1000
Minimum		15	3	309
Maximum		71	40	1309
Percentiles	25	32.00	4.00	384.25
	50	38.00	5.50	464.50
	75	46.75	11.00	734.25

The mean age of patients in the study is 39 years. The minimum age is 15 years and the maximum age is 71. The mean length of hospital stay is 10 days. Minimum hospital stay was for 3 days and maximum for 40 days. Mean amylase level of the patients in the study was 563 IU. The highest amylase value was 1309 IU and lowest was 309 IU.

SEVERITY (BY ATLANTA CLASSIFICATION)

Table 7. Severity classification based on Atlanta classification

SEVERITY	NO OF PATIENTS	PERCENTAGE
SEVERE	29	29%
MILD	71	71%

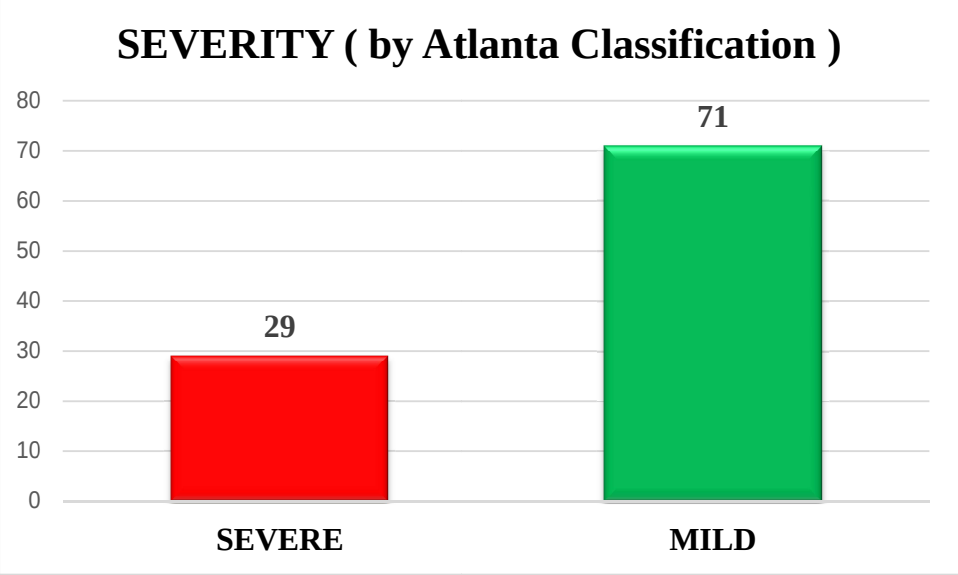

Chart 6. Severity classification based on Atlanta classification

Of the total 100 cases, 29 cases fulfilled the criteria of Severe Acute Pancreatitis (SAP) by Atlanta Classification. This was taken as the standard to compare BISAP score and MCTSI.

ORGAN FAILURE IN SEVERE ACUTE PANCREATITIS

Of the total 29 cases of SAP diagnosed to have organ failure that persisted more than 48 Hrs, majority (48%) was found to had Cardiovascular System failure which presented as shock and hypotension.35% of patients had renal failure and Respiratory failure was found in 35% of patients

A.RESPIRATORY FAILURE IN SEVERE PANCREATITIS PATIENTS

Table 8. Respiratory failure among severe pancreatitis

RESPIRATORY FAILURE	NO OF PATIENTS(N=29)	PERCENTAGE
PRESENT	10	35%
ABSENT	19	65%

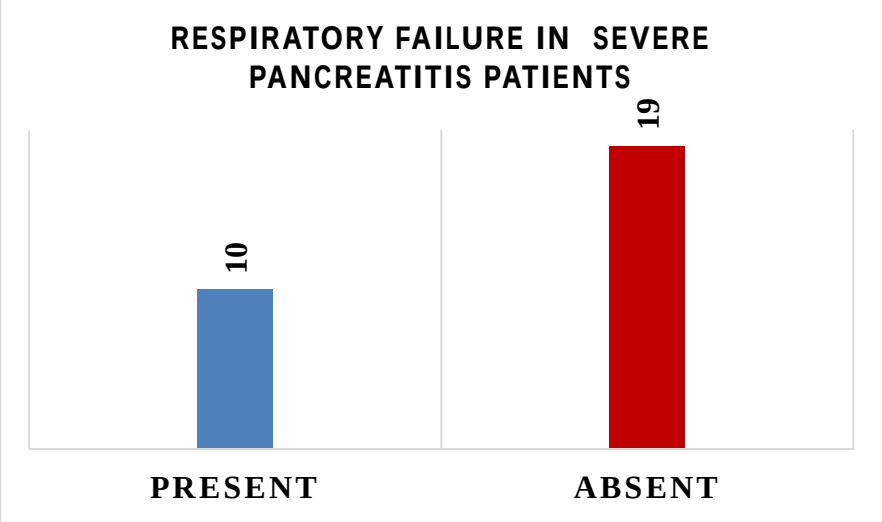

Chart 7. Respiratory failure among severe pancreatitis

CARDIAC FAILURE IN SEVERE PANCREATITIS PATIENTS

Table 9.Cardiac failure among severe pancreatitis

CARDIAC FAILURE	NO OF PATIENTS(N=29)	PERCENTAGE
PRESENT	14	48%
ABSENT	15	52%

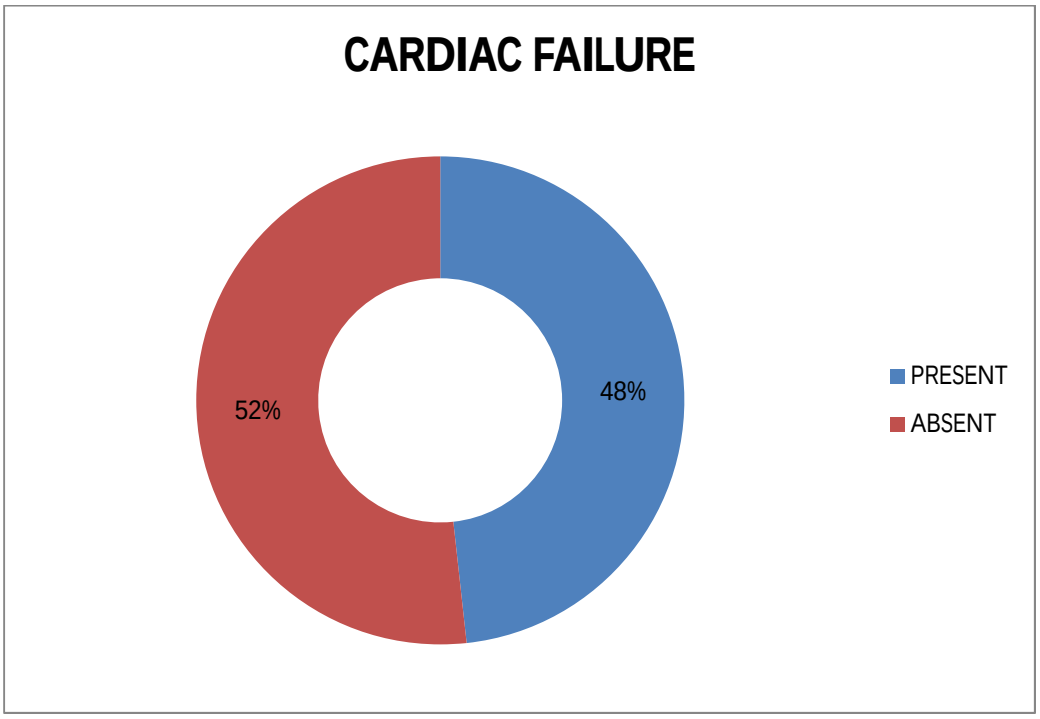

Chart 8. Cardiac failure among severe pancreatitis

RENAL FAILURE IN PATIENTS WITH SEVERE PANCREATITIS

Table 10.Renal failure among severe pancreatitis

RENAL FAILURE	NO OF PATIENTS(N=29)	PERCENTAGE
PRESENT	10	35%
ABSENT	19	65%

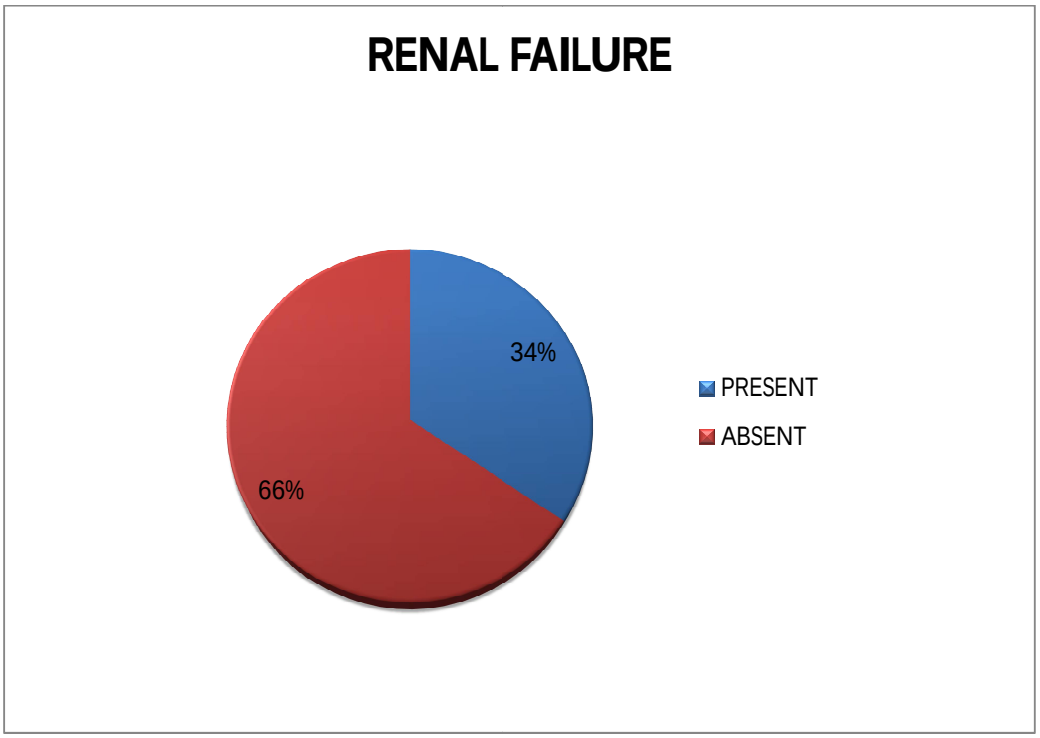

Chart 9. Renal failure among severe pancreatitis

BISAP SCORE

The proportion of subjects with acute pancreatitis stratified with BISAP point score is presented in table.11. A significant trend of disease severity was seen with increasing BISAP score.

Table 11.Stratification of patients based on BISAP score

BISAP	NO OF PATIENTS	PERCENTAGE	SAP
ZERO	40	40%	0
ONE	20	20%	3
TWO	16	16%	7
THREE	15	15%	11
FOUR	9	9%	8

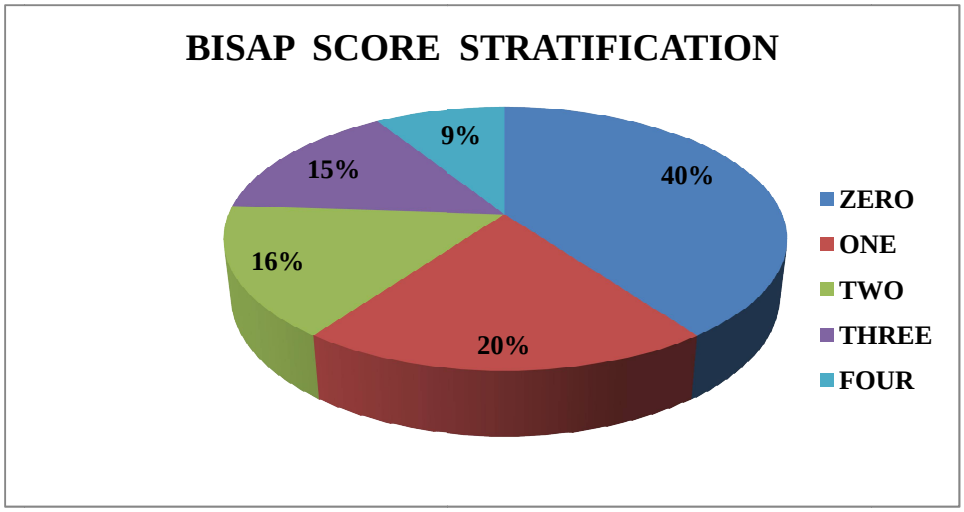

Chart 10.Stratification of patients based on BISAP score

MODIFIED CT SEVERITY INDEX

The proportion of subjects with acute pancreatitis stratified with MCTSI score is presented in table.12.

Table 12. Stratification of patient based on MCTSI score

MCTSI SCORE	NO OF PATIENTS	PERCENTAGE	SAP
ZERO	31	31%	0
TWO	38	38%	9
FOUR	15	30%	8
SIX	11	22%	7
EIGHT	5	10%	5

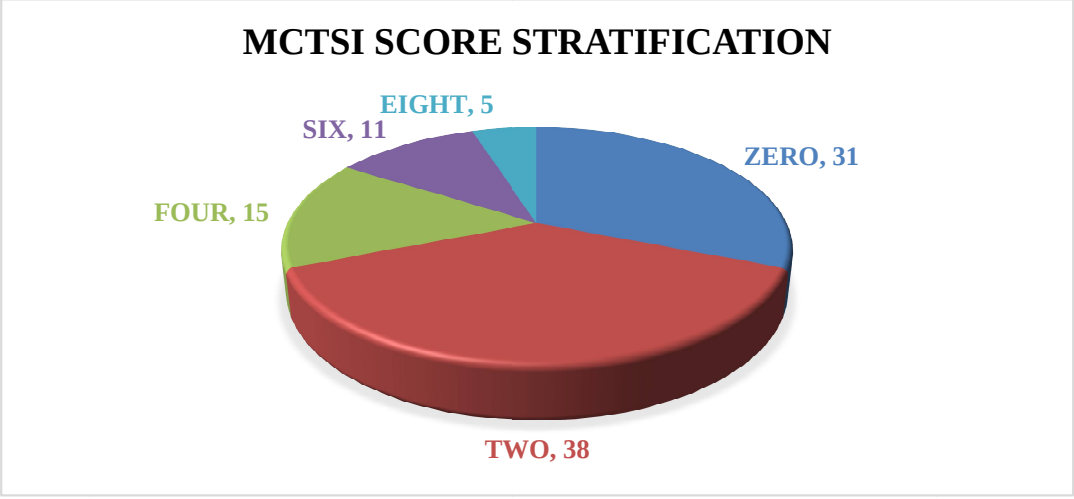

Chart 11. Stratification of patients based on MCTSI score

RECEIVER OPERATING CHARACTERISTIC CURVE

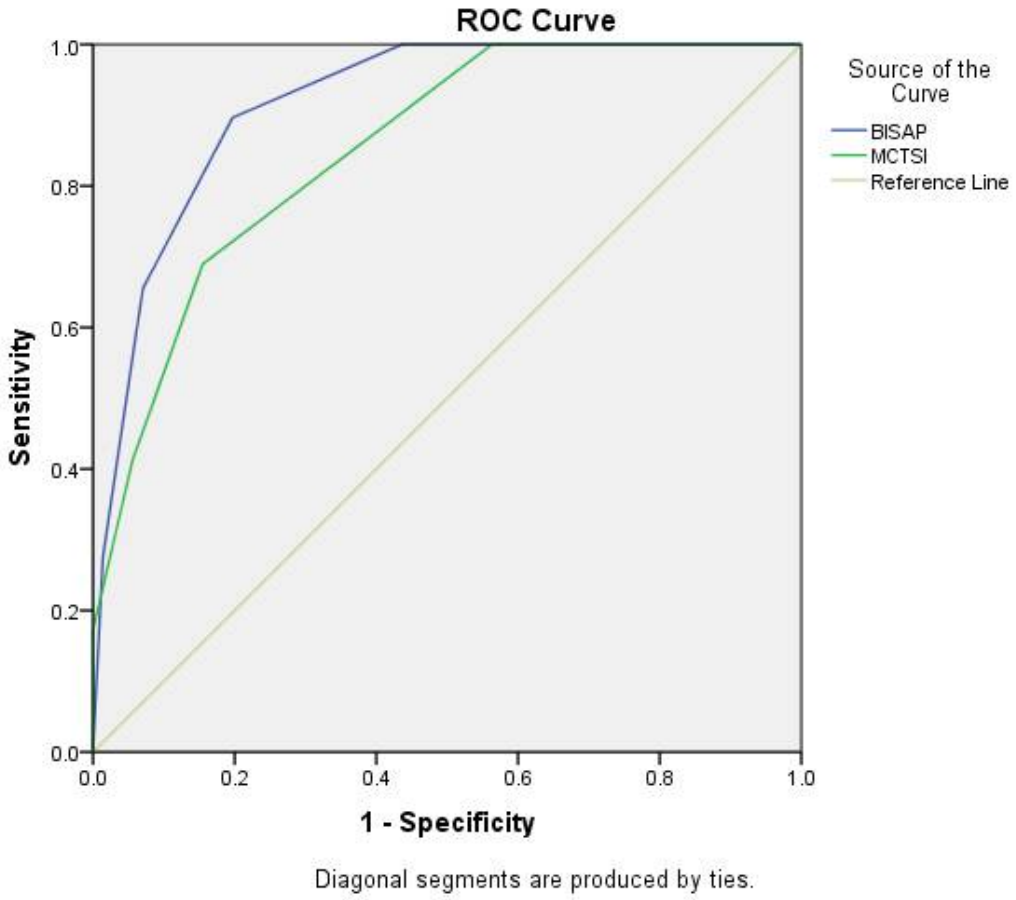

Chart 12. Receiver Operating Characteristic curve of severity

Reciever Operator Characteristic Curves of BISAP and CTSI scores in predicting severity were plotted and pairwise comparison was done. The AUC for BISAP and CTSI was 0.917 (95% CI 0.864–0.970) and 0.853 (95% CI 0.777– 0.928), respectively. On the basis of highest sensitivity and specificity values generated from the receiver – operating characteristic curves, the following cutoff's were selected for assessing severity:

BISAP score 3, and a CTSI 4.

STRATIFICATION BASED ON CUT-OFF OF BOTH SCORES

Table 13. BISAP ≥ 3 vs BISAP < 3

BISAP SCORE	NO OF PATIENTS	PERCENTAGE
BISAP >= 3	24	24%
BISAP < 3	76	76%

Table 14. MCTSI ≥ 4 vs MCTSI <4

MCTSI	NO OF PATIENTS	PERCENTAGE
MCTSI >= 4	31	31%
MCTSI < 4	69	69%

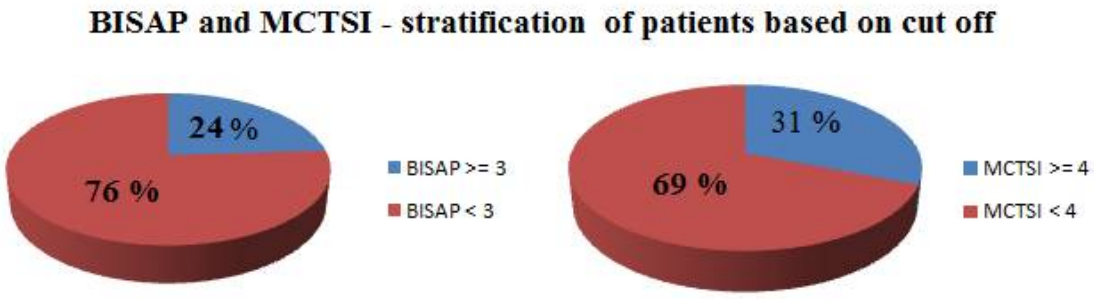

Chart 13.A. BISAP ≥ 3 vs BISAP< 3. B. MCTSI ≥ 4 vs MCTSI <4

Among 100 patients 24% of patients had BISAP ≥ 3 and 76% of patients had BISAP < 3. 31% of patients had MCTSI ≥ 4 and 69 had MCTSI < 4.

SERUM AMYLASE – SEVERITY CORRELATION

Table 15.serum amylase – severity correlation

SEVERITY	MEAN	SD
SEVERE	793.86	163.83
MILD	468.73	214.91

P VALUE 0.001 UNPAIRED T TEST

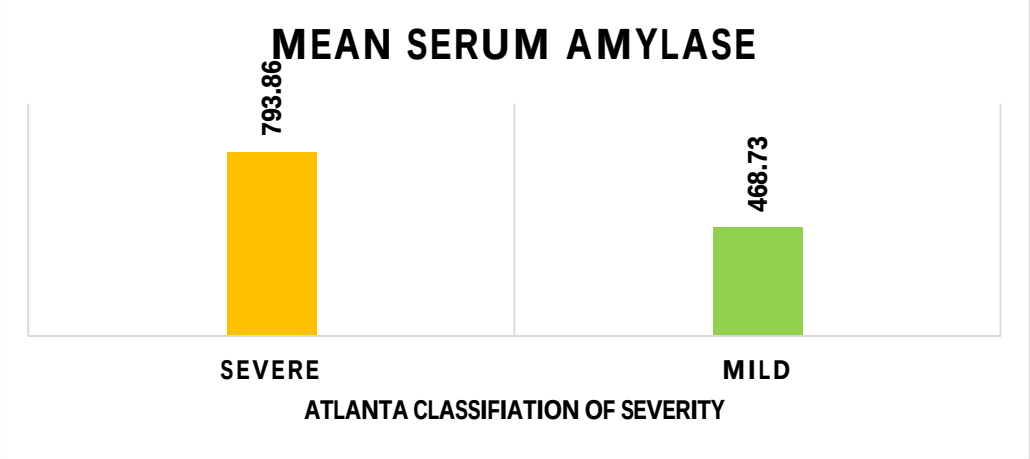

Chart 14.serum amylase – severity correlation

The mean S.Amylase level of patients with Severe Acute Pancreatitis (SAP) was found to be 793.86 ± 163.83 U/L and those with mild acute pancreatitis is 468.73 ± 214.91 U\L. P Value was found to be 0.001 which was statistically significant. Both levels are above three times the normal S.Amylase level.

AMYLASE LEVEL CORRELATION WITH BISAP SCORE

Table 16.Amylase correlation with BISAP score

BISAP SCORE	MEAN	SD
ZERO	403.6	67.26
ONE	483.6	121.66
TWO	631.37	240.56
THREE	785.2	165.15
FOUR	956.11	188.26

P-VALUE : 0.001 ANOVA

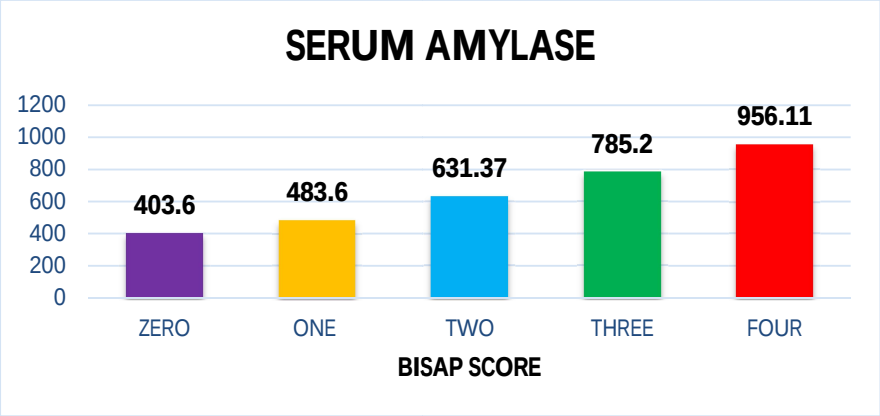

Chart 15.Amylase correlation with BISAP score

S.Amylase level stratified by the BISAP point score is presented in Table.16 and plotted in Chart.15. The P Value was calculated using ANOVA test and was found to be 0.001 which is statistically significant.With the increase in BISAP score, there is an increasing trend in the S.Amylase level.

AMYLASE LEVEL CORRELATION WITH MCTSI

Table 17.Amylase correlation with MCTSI

MCTSI SCORE	MEAN	SD
ZERO	434.34	102.89
TWO	475.68	142.55
FOUR	682.13	195.36
SIX	851.09	233.5
EIGHT	1025.8	222.62

P VALUE 0.001 ANOVA

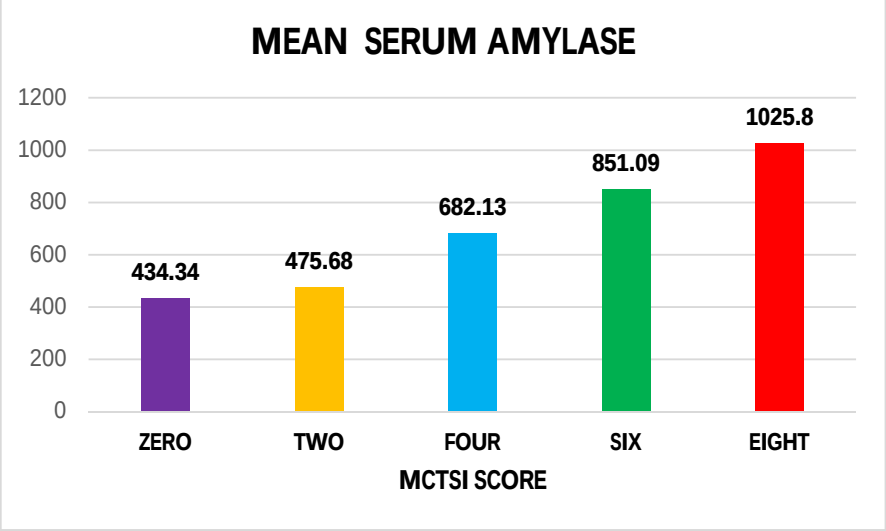

Chart 16. amylase correlation with MCTSI

S.Amylase level stratified by the MCTSI score is presented in Table.17 and plotted in Chart16. The P Value was calculated using ANOVA test and was found to be 0.001 which is statistically significant.With the increase in MCTSI, there is an increasing trend in the S.Amylase level.

PREDICTION OF SEVERITY OF DISEASE WITH BISAP SCORE

Table 18.Prediction of severity with BISAP score

BISAP SCORE	SEVERE	MILD
BISAP >= 3	19	5
BISAP < 3	10	66
TOTAL	29	71

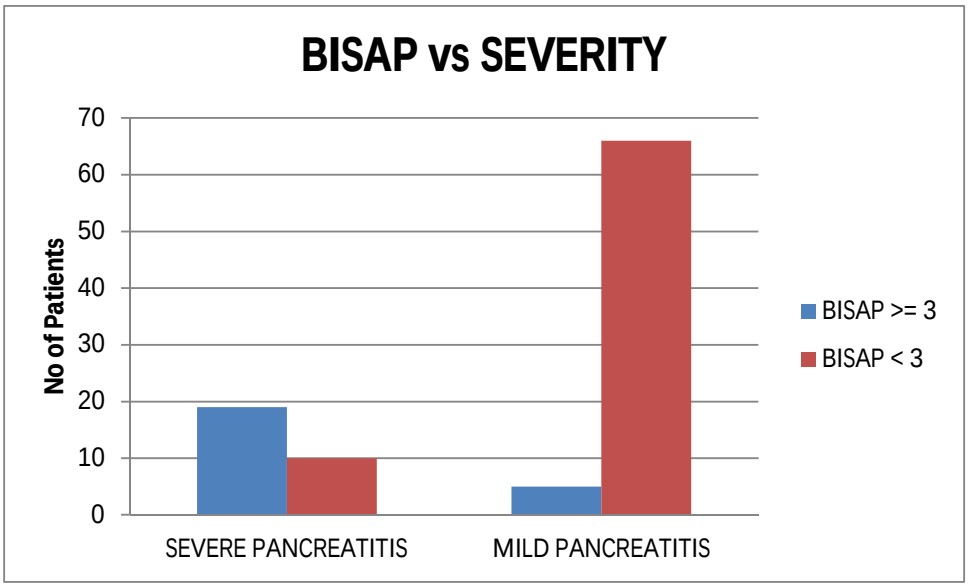

Chart 17.Prediction of severity with BISAP score

Severe and mild pancreatitis cases with BISAP ≥ 3 and BISAP < 3 are tabulated in the table18.Among the total 29 cases of severe acute pancreatitis, 19 cases were found to have BISAP score ≥ 3 and 10 cases had a score < 3.

PREDICTION OF SEVERITY WITH MCTSI

Table 19.Prediction of severity with MCTSI

MCTSI	SEVERE	MILD
MCTSI >= 4	20	11
MCTSI < 4	9	60
TOTAL	29	71

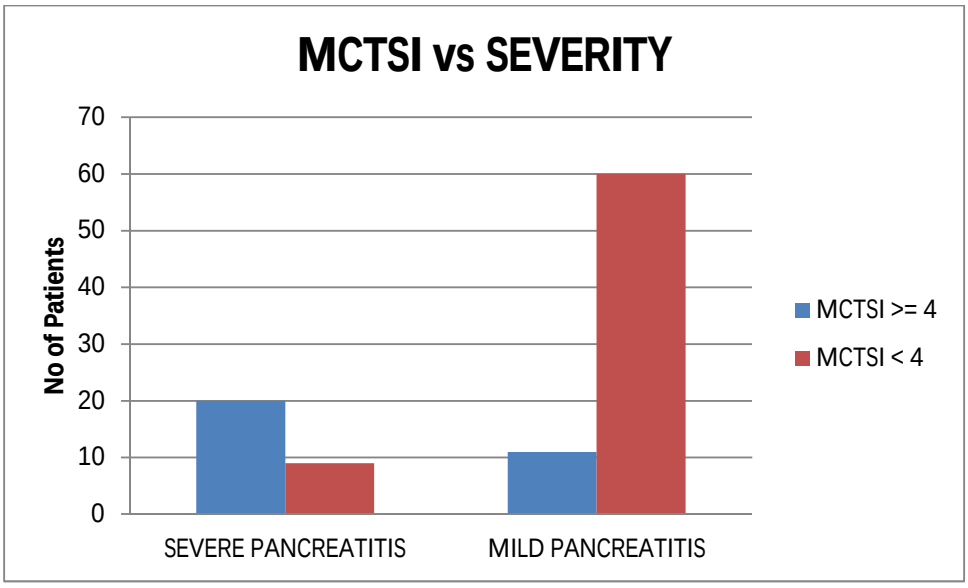

Chart 18.Prediction of severity with MCTSI

Severe and mild pancreatitis case with MCTSI ≥ 4 and MCTSI < 4 are tabulated in the table19.Of the 29 cases of SAP, 20 cases had MCTSI ≥ 4 and 9 cases had score < 4.

SENSITIVITY, SPECIFICITY, PPV, NPV COMPARISON OF BISAP AND MCTSI IN PREDICTING SEVERITY

Table 20. Sensitivity, Specificity, PPV, NPV comparison of BISAP and MCTSI in predicting severity

SCORING SYSTEM	BISAP	MCTSI
SENSITIVITY	65.52%	68.97%
SPECIFICITY	92.96%	84.51%
POSITIVE PREDICTIVE VALUE	79.17%.	64.52%
NEGATIVE PREDICITIVE VALUE	86.84%	86.96%

.

The Specificity, Sensitivity. Positive Predictive Value and Negative Predictive Value of both BISAP and MCTSI in predicting the severity of acute pancreatitis was calculated. The Specificity and Positive Predictive Value of BISAP score were higher than the MCTSI in predicting the severity. Comparable Negative Predictive Values were seen for both BISAP score and MCTSI. The ROC analysis for severity showed BISAP score had AUC of 0.917 which was more than MCTSI score which had AUC of 0.853. Hence the accuracy of BISAP score in predicting the severity of acute pancreatitis is more when compared with MCTSI.

PANCREATIC NECROSIS

Table 21. Pancreatic necrosis in patients with SAP

NECROSIS	NO OF PATIENTS	PERCENTAGE
PRESENT	19	19%
ABSENT	81	81%

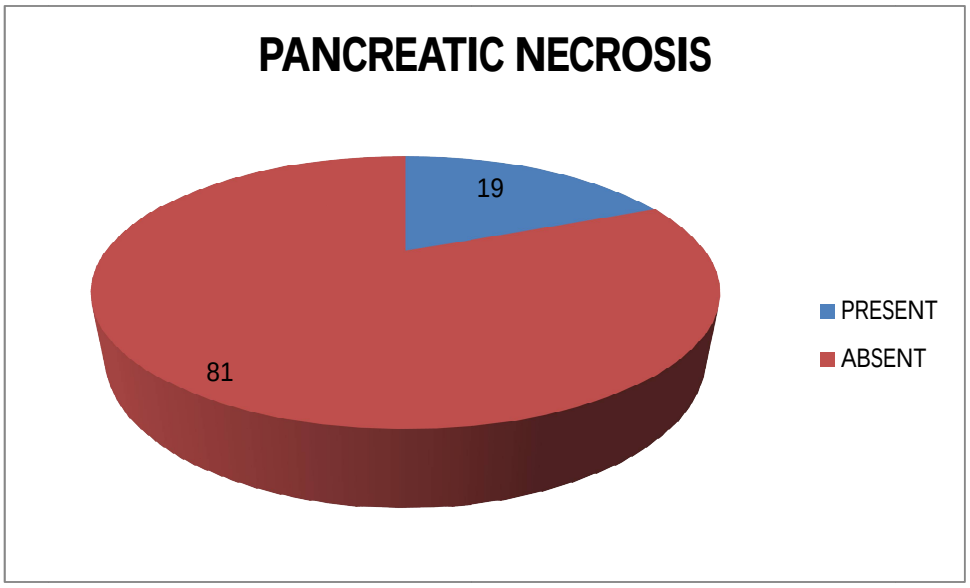

Chart 19.Pancreatic necrosis in patients with SAP

Of the total 100 patients, CT scan done within 48Hrs revealed pancreatic necrosis in 19 cases.

BISAP SCORE STRATIFICATION IN PANCREATIC NECROSIS

Table 22. BISAP score stratification for Necrosis

BISAP SCORE	NECROSIS IN SEVERE CASES	NECROSIS IN MILD CASES
BISAP >=3	6	3
BISAP < 3	1	9
TOTAL	7	12

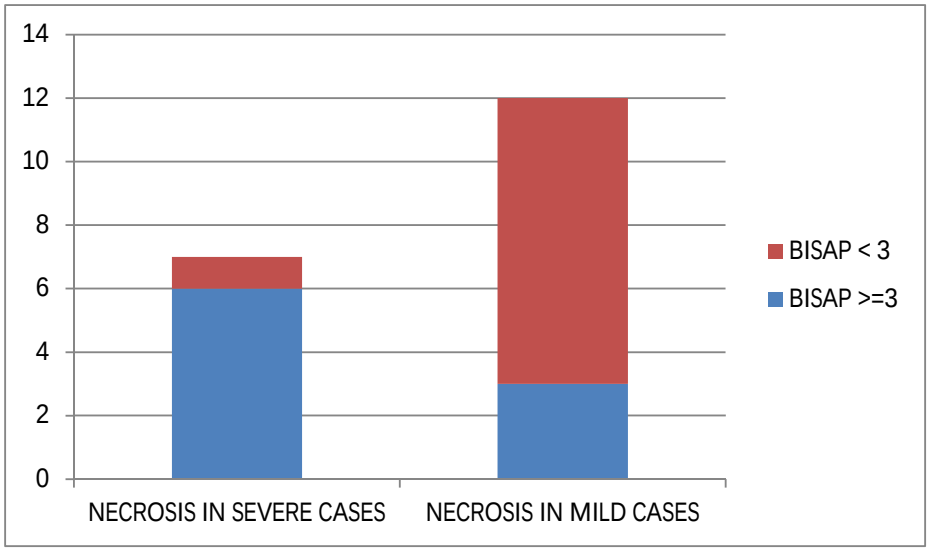

Chart 20.BISAP score stratification for Necrosis

By performing CT scan in the first 48 Hrs, necrosis was identified in 7 out of 29 severe cases and 12 out of 71 mild cases of acute pancreatitis. Of the 7 severe cases, 6 cases had BISAP ≥3. Of the 12 mild cases, 3 showed BISAP ≥ 3.

MCTSI STRATIFICATION IN PANCREATIC NECROSIS

Table 23.MCTSI score stratification for Necrosis

MCTSI	NECROSIS IN SEVERE CASES	NECROSIS IN MILD CASES
MCTSI >= 4	7	9
MCTSI < 4	0	3
TOTAL	7	12

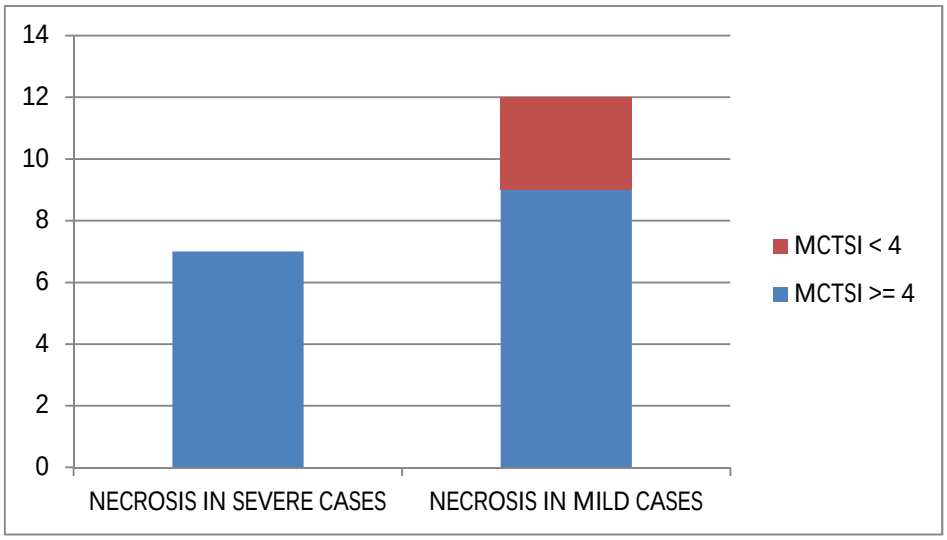

Chart 21.BISAP score stratification for Necrosis

Out of the 19 cases of pancreatic necrosis, all 7 severe cases had MCTSI ≥ 4 and 9 out of 12 mild cases of pancreatitis had MCTSI ≥ 4.

COMPARING BISAP AND MCTSI IN PREDICTING NECROSIS

Table 24.Comparison of BISAP and MCTSI in predicting Necrosis

PREDICTION OF NECROSIS	BISAP	MCTSI
SENSITIVITY	85.71%	100.0%
SPECIFICITY	75.00%	75.00%
POSITIVE PREDICTIVE VALUE	66.67%	83.75%
NEGATIVE PREDICTIVE VALUE	90.00%	100.00%
ODDS RATIO	3.96	23

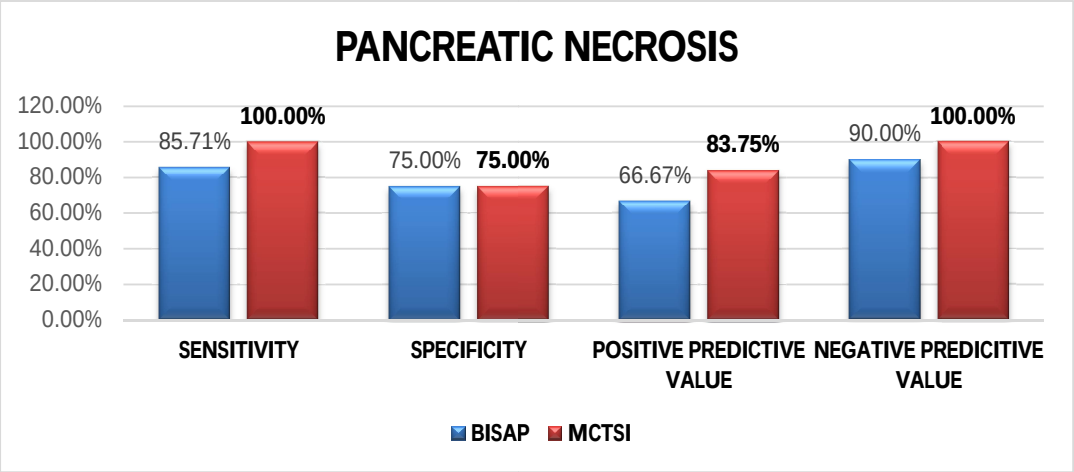

Chart 22.Comparison of BISAP and MCTSI in predicting Necrosis

The Sensitivity, Specificity, Positive Predictive Value and Negative Predictive Value of both BISAP score and MCTSI in predicting pancreatic necrosis were calculated. MCTSI was found to have higher sensitivity and positive predictive value. MCTSI also had a negative predictive value of 100%.Patients with MCTSI ≥ 4 had 23 times chance of having pancreatic

necrosis than MCTSI < 4. Hence MCTSI is more accurate in predicting necrosis compared to BISAP score.

MORTALITY

Table 25.Mortality Statistics

OUTCOME	NO OF PATIENTS	PERCENTAGE
DIED	8	8%
DISCHARGED	92	92%

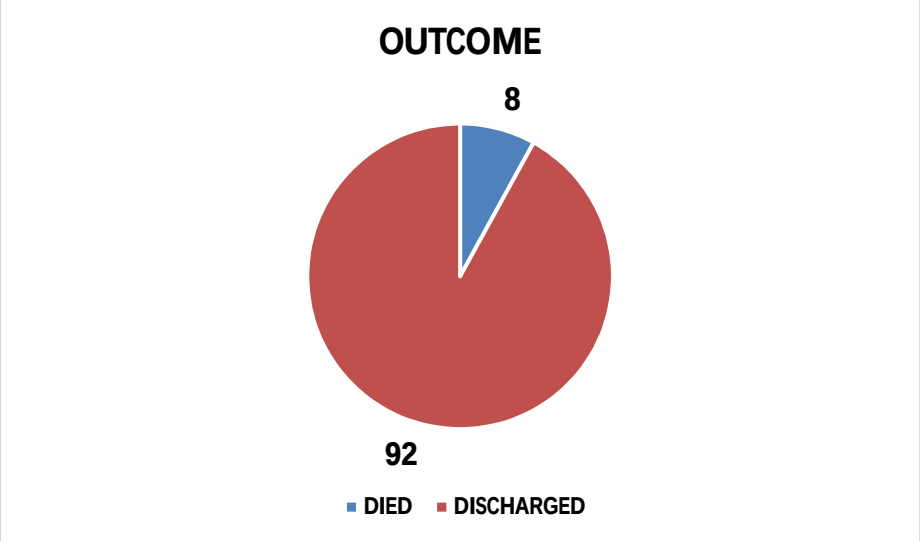

Chart 23.Mortality Statistics

Of the total 100 patients in the study, 8 patients died in the course of treatment. 92 patients were discharged. All the dead patients were diagnosed to have severe acute pancreatitis.All of them were having BISAP score ≥ 3 and CTSI ≥ 4.

MORTALITY vs BISAP SCORE

Table 26.Mortality vs BISAP score

BISAP SCORE	DEAD	DISCHARGED
BISAP >= 3	8	16
BISAP < 3	0	76

P VALUE :0.001 ODDS RATIO: 38

CHI SQUARE TEST

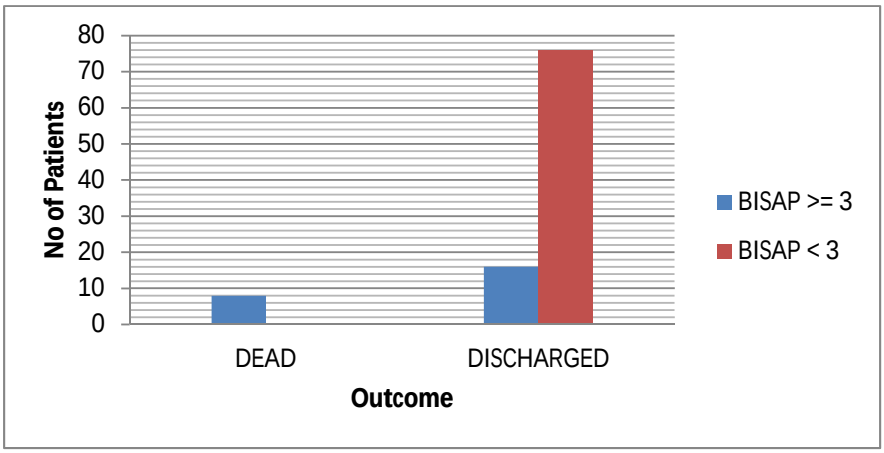

Chart 24.Mortality vs BISAP score

Among the 8 expired patients in the study all of them had a BISAP score ≥ 3. 16 out of 92 discharged patients also had a BISAP score ≥ 3.P value was calculated to be 0.001 which was statistically significant. The odds ratio was calculated to be 38. Based on BISAP score patient who has score ≥ 3 has Thirty Eight fold higher chance of ending up in mortality than patients with a score < 3 , which is statistically significant.

MORTALITY vs MCTSI SCORE

Table 27.Mortality vs MCTSI score

MCTSI	DEAD	DISCHARGED
MCTSI >= 4	8	23
MCTSI < 4	0	69

P VALUE 0.001 ODDS RATIO 24

CHI SQUARE TEST

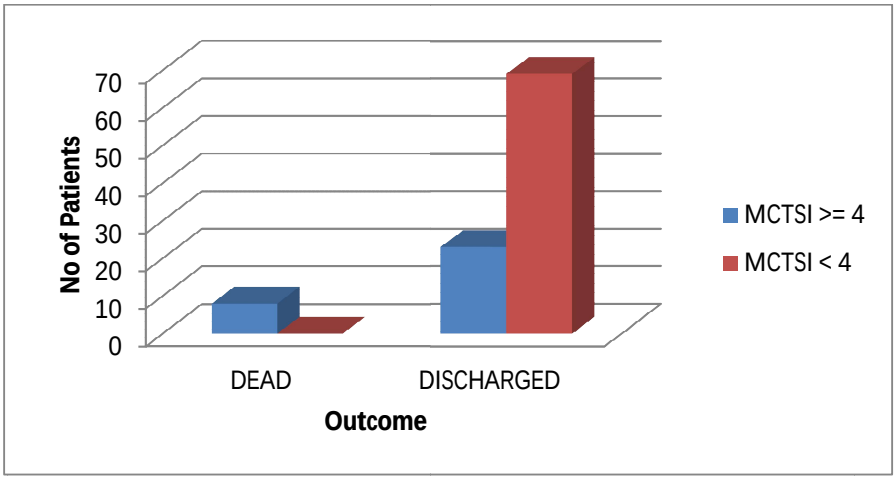

Chart 25.Mortality vs MCTSI score

Among the 8 expired patients in the study all of them had a MCTSI score ≥ 4. 23 out of 92 discharged patients also had a MCTSI score ≥ 4. P value was calculated to be 0.001 which was statistically significant. Odds ratio was calculated to be 24. Based on MCTSI score patient who has score ≥ 4 has Twenty Four fold higher chance of ending up in mortality which is statistically significant. Comparing BISAP and MCTSI, BISAP having high odds ratio predicts mortality more accurately.

DISCUSSION

Acute pancreatitis is a condition with high incidence and is associated with significant mortality rates. Therefore determining the severity in patients with acute pancreatitis is important in triaging patients to either wards or intensive care units to provide the best outcome. The present study compares BISAP score which is a clinical scoring system with MCTSI, which is a radiological score in predicting severity, mortality, and necrosis in 100 patients with acute pancreatitis.

DEMOGRAPHY

The median age for acute pancreatitis in the study done by Cornfield et al on 418 patients was 61 years. Nordestgaard et al did a study on 51 patients with acute pancreatitis with a mean age of 44 years. In the present study mean age of patients presenting with acute pancreatitis is 39 years.

In this study males were 97% and females were 3%.This is in contrast to most western studies where both sexes is equally affected. An Indian study conducted by Vaidya et al revealed similar age and sex distribution. This male preponderance is due to the significant incidence of alcohol consumption in the male population of rural India.

Alcohol and gallstone are the most common etiological factors of acute pancreatitis. In the original BISAP study by Wu et al, gallstone contributed 23.8% cases and alcohol was responsible in 21.1% cases. In the present study alcohol is the most common etiological agent contributing 46% followed by gallstones contributing 27%. Other Indian studies also showed the similar distribution in etiological agents. This may be attributed to the difference in dietary, social, genetic and cultural factors between Indian population and Western population

COMPARISON OF SCORING SYSTEMS

In our study 29 out of 100 patients (29%) developed severe acute pancreatitis. The AUC for prediction of severity by BISAP and MCTSI score are 0.917 (95% CI 0.864 – 0.970) and 0.853 (95% CI 0.777 – 0.928) respectively. The in-hospital mortality rate is 8%

In 2010, Papachristou et al conducted a study of 185 patients which showed AUC for predicting severity in acute pancreatitis for BISAP and MCTSI as 0.81 and 0.84 respectively. A study done by Gompertz et al in 2012 noted BISAP score $\geq$ 3 had a sensitivity, specificity, positive and negative predictive values of 71.4, 99.1, 83.3 and 98.3 % respectively in predicting severity. The present study also had high specificity and negative

predictive value compared with that of MCTSI ≥4 in predicting severity in acute pancreatitis.

Singh and colleagues from Harvard Medical School studied 397 cases of acute pancreatitis.They observed that cases with BISAP score ≥3 were 4 times more likely to develop pancreatic necrosis than those with a score < 3.Case with MCTSI ≥ 4 were 18 times more likely to develop pancreatic necrosis compared with cases with a score < 4. In our study also cases with BISAP score ≥ 3 had a 4 times more chance to develop pancreatic necrosis than those with a score < 3. Also, patients with MCTSI ≥ 4 has 23 times more chance to develop pancreatic necrosis than those with score < 4. A study by Yadhav et al concluded that MCTSI predicts pancreatic necrosis more accurately than BISAP score. Present study also derived at the same conclusion

Wu et al showed in their study that 18% of patients with BISAP ≥ 3 died and only 1% of those with a score < 3 died in the study.In the present study 50% of patients with BISAP score ≥ 3 died and no patients with BISAP < 3 expired. In the present study patients with BISAP ≥3 had thirty eight times more chance of ending up in death compared to those with BISAP < 3. These results are comparable to other similar studies.

CONCLUSION

Individual response to pancreatic injury is highly variable and unpredictable in most of the times. To classify patients with acute pancreatitis into mild and severe groups, BISAP is a reliable prognostic tool. The components of BISAP are clinically relevant and easy to obtain. The sensitivity of BISAP score ≥ 3 in predicting severe acute pancreatitis was found to be 65.52%. The negative predictive value of BISAP score in predicting severity is much higher at 86.84%.If only BISAP score of 0 or 1 were considered, only 3 out of 60 patients (5%) had severe pancreatitis. Hence it is safe to consider that patients with BISAP score 0 or 1 will be having mild pancreatitis and CT scan can be avoided in such patients. However, 7 out of 16 patients(43%) with BISAP score 2 were found to have acute pancreatitis. Therefore CT scan must be done in all patients with BISAP score 2. AUC concludes that BISAP score is an ideal tool in predicting severity in Acute Pancreatitis.

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DATA COLLECTION PROFORMA

Name : Age:

Sex: Case Number:

IP No: Length of Hospital Stay:

Serum Amylase: Etiology:

SIGNS AND SYMPTOMS

Abdominal Pain	Present	Absent
Guarding	Present	Absent
Rigidity	Present	Absent
Vomiting	Present	Absent

Pulse Rate: Respiratory Rate:

Temperature: WBC count:

SIRS: Present / Absent

Pancreatic Necrosis: Present / Absent

Blood Urea Nitrogen:

Impaired Mental Status : Yes / No

SIRS: Yes / No

Age more than 60: Yes / No

Pleural Effusion: Yes / No

BISAP Score: **MCTSI :**

ORGAN FAILURE:

Respiratory System	Present	Absent
Cardiovascular System	Present	Absent
Renal System	Present	Absent

SEVERITY: Mild / Severe

OUTCOME: Dead / Discharged

CONSENT FORM

PART 1 0F 2

Dear Volunteers,

We welcome you and thank you for your interest in participating in this research project. Before you participate in this study, it is important for you to understand why this research is being carried out.this form will explain you all the relevant details of this research .it will explain the nature,thepurpose,thebenefits,the risks, the discomforts,the precautions and the information about how this project will be carried out. It is important that you read and understand the contentsof the form carefully.

TITLE OF THE PROJECT: "A COMPARATIVE EVALUATION OF CLINICAL AND RADIOLOGICAL SCORING SYSTEMS IN THE EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS"

Name of the investigator:

What is the purpose of this project/study?

To compare BISAP and MCTSI in predicting the severity in acute pancreatitis

How will the study be carried out?

All patients of acute pancreatitis who the fit inclusion criteria will be included in study. For this research only data will be collected and no intervention will be done.

- Following datas will be collected: Demographics (age,gender)
- Etiology
- Blood investigations
    a) Complete blood count
    b) Blood urea and creatinine
- Imaging studies(ultrasonography/CECT),chest X-ray
- Clinical and severity scores and radiological scores

Following is the Inclusion criteria: All patients admitted with the diagnosis of Acute Pancreatitis based on the presence of at least two of the following three criteria:

1. Characteristic epigastric abdominal pain , with or without radiation to the back.
2. Serum amylase or lipase levels elevated to at least three times the upper limit of normal.
3. Characteristic finding of Acute Pancreatitis on abdominal CT scan.

What is the expected duration of the subject participation? No additional stay in hospital is required than the usual course.

What are the benefits to be expected from the researchto the participant or to others and the post-trial responsibilities of the investigator?

No direct benefit to the participants. At the end of study we will be able to find some factors which predict the severity in early phase of acute pancreatitis.

What are the risk factors expected from the study to the participants?

No risk as this is only an analytical study.

Whether my participation in the study will be kept confidential ?

Yes,confidentiality of records will be maintained.

Is there provision of free treatment for research related injury? No intervention will be done for research purpose. So no research related injury is expected.

Can I withdraw from study when I want?

Yes. You can withdraw at any time without giving reason and this decicsion will not affect your regular medical care.

PARTICIPANT CONSENT FORM

PART 2 OF 2

Participants name: Address:

Title of the project:

“A COMPARATIVE EVALUATION OF CLINICAL AND RADIOLOGICAL SCORING SYSTEMS IN THE EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS”

The details of the study have been provided to me in writing and explained to me in my own language. I confirm that I have understood the above study and had the opportunity to ask questions. I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without the medical care that will normally be provided by the hospital being affected. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose. I have been given information sheet giving details of the study. I fully consent to participate in the above study.

I also consent /do not consent to use my stored biological samples for future scientific purposes –if applicable

Signature of the participant: Date:

Signature of the witness: Date

Signature of the investigator: Date:

ஒப்புதல் படிவம்

பெயர் :

வயது :

பாலினம் :

முகவரி:

கோவை அரசு மருத்துவக்கல்லூரி மருத்துவமனையில் மருத்துவர். R$ɹj;vk;n$h ! ; தலைமையில் நடைபெறும் இந்த ஆய்வில் முழு சம்மதத்துடன் கலந்துகொள்ள சம்மதிக்கிறேன் .இந்த ஆய்வில் என்னை பற்றி விவரங்களை பாதுகாப்புடன் இந்த ஆய்வில் வெளியிட ஆட்சேபணை இல்லை என்று தெரிவித்துக் கொள்கிறேன் .எந்த நேரத்திலும் ஆய்வில் இருந்து எந்த நேரத்திலும் விலக்கிக்கொள்ளும் உரிமை உண்டு என்று அறிவேன் .

இடம் :

தேதி:

கைகெயாப்பம் /ரேகை

KEY TO MASTER CHART

Gender			
	Male	1	
	Female	2	
Etiology			
	Alcohol	1	
	Gall Stones	2	
	Idiopathic	3	
	Post ERCP	4	
	Hyperlipidemia	5	
Signs and Symptoms		Present	Absent
	Abdominal Pain	1	2
	Radiation to Back	1	2
	Guarding	1	2
	Vomiting	1	2
Atlanta Criteria for classification of Pancreatitis			
	Severe pancreatitis	1	
	Mild Pancreatitis	0	
Organ failure		Present	Absent
	Respiratory	1	0
	CVS	1	0
	Renal Failure	1	0
Necrosis		Present	Absent
		1	0
Outcome	Death	1	
	Discharged	0	

							SIGNS/SYMPTOMS								ORGAN FAILURE				
Sl.No	Name	Age	Sex	IP No	Hospital Stay	Etiology	Pain	Radiation	Vomiting	Guarding	Amylase	BISAP	Atlanta Class	MCTSI	resp fail	cvs fail	renal fail	Necrosis	Outcome
1	vellingiri	50	1	6531	5	1	1	1	1	1	389	0	0	0	0	0	0	0	0
2	Veerappan	65	1	6821	6	1	1	1	2	1	451	0	0	0	0	0	0	0	0
3	Murugavel	26	1	6966	4	2	1	1	1	2	643	2	1	2	1	0	0	0	0
4	Sateesh Kumar	36	1	7031	17	1	1	2	1	1	698	3	0	4	0	0	0	0	0
5	Selvam	40	1	7186	10	3	1	1	2	1	421	0	0	2	0	0	0	0	0
6	Anand	47	1	7252	5	1	1	1	1	1	397	0	0	2	0	0	0	0	0
7	Sakthivel	25	1	7459	3	2	1	1	1	1	512	1	1	2	0	1	0	0	0
8	Goutham	15	1	7682	4	1	1	2	2	1	743	2	0	2	0	0	0	0	0
9	Francis	48	1	8216	3	3	1	1	1	1	432	1	0	0	0	0	0	0	0
10	Varatharajan	32	1	8337	4	1	1	2	1	1	388	0	0	0	0	0	0	0	0
11	Velusamy	40	1	8416	12	2	1	2	1	2	402	0	0	2	0	0	0	0	0
12	Anand	26	1	8617	7	1	1	1	2	1	514	2	1	2	0	0	1	0	0
13	Chandran	49	1	8911	20	2	1	2	1	1	597	2	0	4	0	0	0	1	0
14	Kajamoideen	35	1	9436	4	1	1	2	1	1	409	0	0	0	0	0	0	0	0
15	Nagarajan	37	1	10396	5	1	1	1	1	1	383	0	0	2	0	0	0	0	0
16	kaliyappan	65	1	11672	24	2	1	1	1	1	1212	3	1	6	0	0	1	0	0
17	SureshKumar	39	1	11689	4	1	1	1	1	1	533	0	0	0	0	0	0	0	0
18	Ashok Kumar	24	1	23168	13	3	1	2	2	1	612	0	0	2	0	0	0	0	0
19	Ganeshan	59	1	28823	3	2	1	1	1	1	678	1	1	2	0	1	0	0	0
20	Varatharaj	54	1	22220	24	3	1	1	1	1	912	3	1	4	0	0	1	0	0
21	Chinnaraj	40	1	31615	5	3	1	2	1	1	803	2	0	0	0	0	0	0	0
22	Nagaraj	38	1	36311	3	2	1	1	1	1	1209	4	1	8	1	0	0	1	1
23	Arun Kumar	29	1	36558	11	1	1	2	1	1	504	0	0	2	0	0	0	0	0
24	Sikkandar Bhasha	26	1	40046	6	2	1	1	2	1	612	0	0	0	0	0	0	0	0
25	Sivakumar	35	1	47733	5	2	1	2	1	1	499	1	0	2	0	0	0	0	0
26	Mahendran	33	1	47978	4	1	1	1	1	1	1309	4	1	8	0	1	0	1	1
27	raghupathy	40	1	44409	36	1	1	1	1	1	917	3	1	8	0	1	0	1	0
28	Kaja	45	1	92625	5	1	1	2	1	1	509	1	0	2	0	0	0	0	0

29	kannan	40	1	64057	6	3	1	1	1	2	399	0	0	0	0	0	0	0	0
30	Babu	28	1	64669	10	2	1	2	2	1	457	0	0	2	0	0	0	0	0
31	Mani	44	1	64793	8	1	1	1	1	1	876	4	1	6	1	0	0	0	1
32	Suresh Kumar	34	1	71171	4	1	1	2	1	1	596	1	0	0	0	0	0	0	0
33	Jaganathan	46	1	71176	7	3	1	1	1	1	432	0	0	2	0	0	0	0	0
34	Arun Kumar	30	1	71229	6	2	1	1	1	1	769	3	1	6	0	1	0	0	1
35	Murugavel	35	1	73923	3	1	1	2	1	1	601	1	0	0	0	0	0	0	0
36	Sareeg Dher	23	1	74504	8	3	1	1	2	1	413	0	0	2	0	0	0	0	0
37	Ammasai	68	2	4917	3	2	1	1	1	1	781	4	1	8	1	1	1	1	1
38	Suresh	47	1	74865	6	1	1	2	1	1	612	1	0	2	0	0	0	0	0
39	Krishnakumar	35	1	76395	5	1	1	2	1	1	347	0	0	0	0	0	0	0	0
40	Karuppanan	71	1	39641	8	1	1	1	1	2	708	3	1	4	0	1	0	0	0
41	Kiran	25	1	41788	12	2	1	1	1	1	339	1	0	2	0	0	0	0	0
42	Jayachandran	29	1	41828	4	1	1	2	1	1	400	0	0	0	0	0	0	0	0
43	Ganeshan	35	1	9152	16	3	1	1	1	1	748	3	1	4	1	0	0	0	0
44	Gukan	29	1	43414	38	2	1	2	1	1	812	4	0	6	0	0	0	1	0
45	Dinesh	25	1	45283	4	2	1	2	2	1	413	0	0	2	0	0	0	0	0
46	manikandan	38	1	10092	10	3	1	1	1	1	913	4	1	8	1	1	1	1	1
47	Surendran	40	1	51192	3	2	1	1	1	1	316	0	0	0	0	0	0	0	0
48	Surya	28	1	85320	5	1	1	1	1	1	801	4	1	6	0	0	1	0	1
49	Balan	32	1	86442	3	1	1	2	1	1	421	0	0	0	0	0	0	0	0
50	Palanisamy	64	1	26207	25	2	1	1	1	1	697	3	1	4	1	0	1	0	0
51	Manoharan	37	1	86996	9	3	1	1	1	1	410	0	0	2	0	0	0	0	0
52	Selvaraj	35	1	90423	6	3	1	2	2	1	543	2	1	2	0	1	0	0	0
53	Siddharth	23	1	79408	4	1	1	2	1	2	366	0	0	0	0	0	0	0	0
54	Faharudeen	50	1	79488	5	3	1	1	1	1	345	0	0	2	0	0	0	0	0
55	Suresh	37	1	80524	12	1	1	1	1	1	547	1	0	4	0	0	0	1	0
56	Raju	52	1	51438	28	1	1	1	1	1	794	3	1	6	0	1	0	1	0
57	Mani	35	1	81005	32	2	1	1	1	1	659	3	0	6	0	0	0	1	0
58	Arokyadas	37	1	2503	5	3	1	2	2	1	361	0	0	0	0	0	0	0	0
59	Velusamy	38	1	2625	4	3	1	1	1	2	494	1	0	2	0	0	0	0	0
60	Suresh	37	1	83681	20	1	1	1	1	1	669	3	1	4	0	1	0	0	0

61	Tirupathi	35	1	9382	22	1	1	1	1	1	338	2	0	4	0	0	0	1	0
62	Krishnakumar	35	1	11124	3	2	1	2	2	1	376	1	0	2	0	0	0	0	0
63	Krishnasami	54	1	17376	3	1	1	2	1	1	443	0	0	0	0	0	0	0	0
64	Velusami	37	1	28319	11	3	1	1	1	1	865	3	1	4	1	0	0	0	0
65	Nagaraj	40	1	18174	18	2	1	1	1	1	470	3	0	4	0	0	0	0	0
66	Najeeb	38	1	29735	7	4	1	2	1	2	377	0	0	2	0	0	0	0	0
67	Chandrashekar	67	1	36862	4	1	1	2	2	1	412	1	0	0	0	0	0	0	0
68	Veerasamy	40	1	20981	4	3	1	1	1	1	877	4	1	6	0	1	0	0	1
69	Sentil Kumar	42	1	36154	8	1	1	1	1	1	312	0	0	2	0	0	0	1	0
70	Raja	22	1	38140	4	3	1	1	2	1	376	0	0	0	0	0	0	0	0
71	Nataraj	45	1	91542	27	1	1	1	1	1	1027	4	1	4	0	0	1	0	0
72	Nagaraj	40	1	38930	4	1	1	1	1	1	330	1	0	0	0	0	0	0	0
73	chandran	41	1	39866	5	1	1	2	2	1	406	0	0	0	0	0	0	0	0
74	Padmanabhan	46	1	57166	3	3	1	2	1	2	514	1	0	0	0	0	0	0	0
75	Jaya	49	2	10636	6	5	1	1	1	1	876	2	1	2	1	0	0	0	0
76	Ajith Kumar	19	1	35315	34	2	1	1	1	1	1269	2	0	6	0	0	0	1	0
77	Rajeev	45	1	82664	4	1	1	1	2	1	430	0	0	0	0	0	0	0	0
78	Bunnan	57	1	70230	4	1	1	2	1	1	315	1	0	2	0	0	0	0	0
79	Rajan	35	1	75533	9	1	1	1	1	2	356	2	0	4	0	0	0	1	0
80	Stephan	37	1	32318	40	3	1	2	1	1	875	3	0	6	0	0	0	1	0
81	Arumugam	49	1	31730	4	2	1	1	1	1	344	0	0	0	0	0	0	0	0
82	Jude Charles	30	1	77562	4	4	1	1	2	1	309	0	0	2	0	0	0	0	0
83	Sakthivel	26	1	82679	9	3	1	1	2	1	766	2	1	2	0	0	1	0	0
84	Dharman	54	1	86629	4	2	1	2	1	2	379	0	0	0	0	0	0	0	0
85	Murugeshan	65	1	82407	10	1	1	1	1	1	743	1	1	2	0	1	0	0	0
86	Arun Kumar	22	1	82858	6	2	1	1	1	1	522	2	0	2	0	0	0	1	0
87	Musthafa	50	1	84242	7	1	1	2	1	1	320	0	0	2	0	0	0	0	0
88	Shekar	36	1	80643	12	3	1	1	2	1	365	1	0	2	0	0	0	0	0
89	rangasamy	46	1	86301	5	1	1	1	1	1	370	0	0	0	0	0	0	0	0
90	Chithra	38	2	75429	30	3	1	2	1	1	418	2	1	6	1	0	0	1	0
91	Gopinath	37	1	73904	5	1	1	1	1	2	441	2	0	0	0	0	0	0	0
92	Mohan	24	1	87908	5	2	1	1	2	1	367	0	0	2	0	0	0	1	0

93	Parthibhan	23	1	87872	4	1	1	1	1	1	398	0	0	0	0	0	0	0
94	Mani	40	1	98564	5	1	1	2	1	1	459	2	1	2	0	0	1	0	0
95	Chinnan	64	1	79761	5	2	1	1	1	2	345	1	0	0	0	0	0	0	0
96	Santhosh	20	1	34501	13	1	1	1	2	1	367	0	0	2	0	0	0	0	0
97	Subbayan	70	1	87961	3	2	1	1	1	1	453	1	0	0	0	0	0	0	0
98	Jagan	34	1	86432	16	1	1	2	1	1	786	3	1	4	0	1	0	0	0
99	ramu	41	1	68710	10	3	1	1	2	1	814	2	0	4	0	0	0	1	0
100	venkadachalam	47	1	12111	4	1	1	1	1	1	365	0	0	2	0	0	0	0	0