A DISSERTATION ON

"A COMPARATIVE STUDY BETWEEN BISAP SCORE AND APACHE IISCORE IN ASSESSING THE SEVERITY OF ACUTE PANCREATITIS BASED ON THE REVISED ATLANTA CLASSIFICATION"

SUBMITTED

TO

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In partial fulfillment of the regulations for the award of the

DEGREE OF M.S (GENERAL SURGERY)

BRANCH-I

DEPARTMENT OF GENERAL SURGERY

STANLEY MEDICAL COLLEGE AND HOSPITAL

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CERTIFICATE BY THE INSTITUTION

This is to certify that dissertation **"A COMPARATIVE STUDY BETWEEN BISAPSCORE AND APACHE II SCORE IN ASSESSING THE SEVERITY OFACUTE PANCREATITIS BASED ON THE REVISED ATLANTACLASSIFICATION"** is a bonafide record of work done by **Dr.V.NEDUNCHEZHIAN** in theDepartment of General Surgery, Stanley Medical College, Chennai, during his Post Graduate Coursefrom MAY 2017- MAY 2020. This is submitted in partial fulfillment for the award of **M.S. DEGREE EXAMINATION- BRANCH I (GENERAL SURGERY)** to be held in May 2020 under the **Tamilnadu DR.M.G.R. Medical University, Chennai.**

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CERTIFICATE BY GUIDE

This is to certify that this dissertation entitled**"A COMPARATIVE STUDY BETWEEN BISAP SCORE AND APACHE II SCORE IN ASSESSING THE SEVERITY OF ACUTE PANCREATITIS BASED ON THE REVISED ATLANTA CLASSIFICATION"**is the bonafide work done by the candidate **Dr. V. NEDUNCHEZHIAN**Post Graduate Student (MAY 2017 to MAY 2020) in the Department of General Surgery, Stanley Medical College, Chennai-1,with registration number **221711060**under my guidance and supervision for the award of **M.S.Degree** Examination , Branch-I **(GENERAL SURGERY)** to be held in May 2020 under the Tamilnadu DR.M.G.R. Medical University, Chennai.

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DECLARATION

I **Dr. V. NEDUNCHEZHIAN,** solemnly declare that this dissertation entitled **"A COMPARATIVE STUDY BETWEEN BISAP SCORE AND APACHE II SCORE IN ASSESSING THE SEVERITY OF ACUTE PANCREATITIS BASED ON THE REVISED ATLANTA CLASSIFICATION",** is a bonafide work done by me in the department of general surgery, Govt. Stanley Medical College and Hospital, Chennai under the supervision of **Prof. Dr. T. SIVAKUMAR M.S.,**This dissertation is submitted to the Tamilnadu Dr. M.G.R. Medical university, Chennai in partial fulfillment of the university regulations for the award of M.S,Degree (General Surgery), Branch – I Examination to be held in May 2020.

DATE:

PLACE:

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I sincerely thank the members of Institutional Ethical Committee, Stanley Medical College for approving my dissertation topic.

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I am extremely thankful to my patients who consented and participated to make this study possible.

ABBREVIATIONS

BISAP	–	Bedside Index for Severity in Acute Pancreatitis
APACHE II	–	Acute Physiological Age and Chronic Health Evaluation
AP	–	Acute Pancreatitis
MAP	–	Mild Acute Pancreatitis
SAP	–	Severe Acute Pancreatitis
MODS	–	Multi Organ Dysfunction Syndrome
ARF	–	Acute Renal Failure
RF	–	Respiratory Failure

INTRODUCTION

Acute pancreatitis is the most common gastrointestinaldisease for which patients are acutely hospitalized.Around 80% of patientswith acute pancreatitis have a mild disease course wheresymptoms usually resolve within 1 week.Approximately20% of patients develop severe acute pancreatitis withorgan failure and/or necrotizing pancreatitis. Necrotizingpancreatitis is defined by pancreatic parenchymal necrosisand/orperipancreatic fat necrosis.Those patients areat risk for a persistent systemic inflammatory responsesyndrome and/or (multiple) organ failure. Sterile pancreaticnecrosis and sterile peripancreatic collections canusually be treated successfully with conservative measures.However, 30% of patients develop secondary infection ofnecrosis, most often 3 to 4 weeks after the onset of disease.When secondary infection of necrosis occurs, morbidityand mortality increase dramatically.Overall mortalityin severe pancreatitis is high (15% to 30%) comparedwith mild pancreatitis (0% to 1%).

AIMS AND OBJECTIVES OF THE STUDY

- To Evaluate the Efficacy of BISAP score and APACHE II score to Assessing theSeverity and Mortality in Acute Pancreatitis based on the Revised Atlanta Classification.

- Stratification of the patients with Acute Pancreatitis according to their scores at the time of hospitalization.

- Thereby to Predict the Appropriate Point for Early and Timely Intervention.

MATERIALS AND METHODS

STUDY SETTING

Department of General Surgery, Govt. Stanley Medical College and Hospital, Chennai. The study was conducted after obtaining the Institutional Ethical Committee Approval.

DURATION

- 12 MONTHS

STUDY DESIGN

- Comparative Analytical Study

SAMPLE SIZE 100

PATIENT SELECTION

First 100 Patients Attending the Surgical Emergency Ward with Clinical features of Acute Pancreatitis are Admitted and Evaluated as per the designed proforma. Data pertinent to the scoring systems will be recorded within 24 hours of admission to the Hospital.

METHODS:

WRITTEN INFORMED CONSENT WILL BE OBTAINED FROM ALL SUBJECTS BEFORE ENROLMENT IN THE STUDY.

INCLUSION CRITERIA

- Age more than 20 years including both sexes.
- Serum amylase/serum lipase equal to or more than 3 times the upper limit of normal
- Radiological evidence of presence of acute pancreatitis.

EXCLUSION CRITERIA

- Age less than 20 years
- Chronic pancreatitis
- Hereditary pancreatitis.
- Patient with comorbidities like COPD, RENAL IMPAIRMENT AND IMMUNOSUPPRESSIVE STATE, etc.
- Traumatic pancreatitis associated with other visceral Injuries.

STATISTICAL ANALYSIS

1) For Eachof 100 patients included in the study. APACHE II and BISAP scores were calculated by using the APACHE II prognostic system in the manner described by KNAUSet.Al and the cardinal health database system for BISAP scoring and Recorded within 24hours of admission to the hospital.

2) Patients were classified to have Mild or Severe Acute Pancreatitis according to the Revised Atlanta classification guidelines.

3) APACHE II score of more than or equal to 9 and BISAP score more than or equal to 3 are expected to predict the severe acute pancreatitis.

4) Patients were observed prospectively until discharge or death.

REVIEW OF LITERATURE

HISTORY OF PANCREAS

The pancreas was generally ignored, both as an organ and as a seat of disease.

The pancreas was first discovered by Herophilus, a Greek anatomist cum surgeon, born in 336 BC on the Asiatic side of the Bosporus in Chalcedon.

The word pancreas first mentioned in the writings of Eristratos. Rufus, an anatomist gave the name “pancreas”. The word meant “all flesh”.

Galen, Physician of Rome and the Roman Emperor, taught that the pancreas serves as a cushion to protect the large vessels lying behind it.

In March 2, 1642, Johan George Wirsung, discovered the pancreatic duct at San Fracisco Monastery in Padua, Italy. But it was named by his collegue as “the duct of wirsung”. Where as papilla, the enlargement of that duct at its junction with the Common Bile Duct which projects into the Duodenum, were first described by vater in 1720. Santorini described accessory duct in 1734, that bears his name.

In 1869, Paul Langerhans, a student of the famous Berlin institute of pathology, headed by eminent professor Rudolph Virchow, who describe the islets of pancreas that was subsequently known as the “Islets of Langerhans”, an endocrine system which lies with in the pancreas.

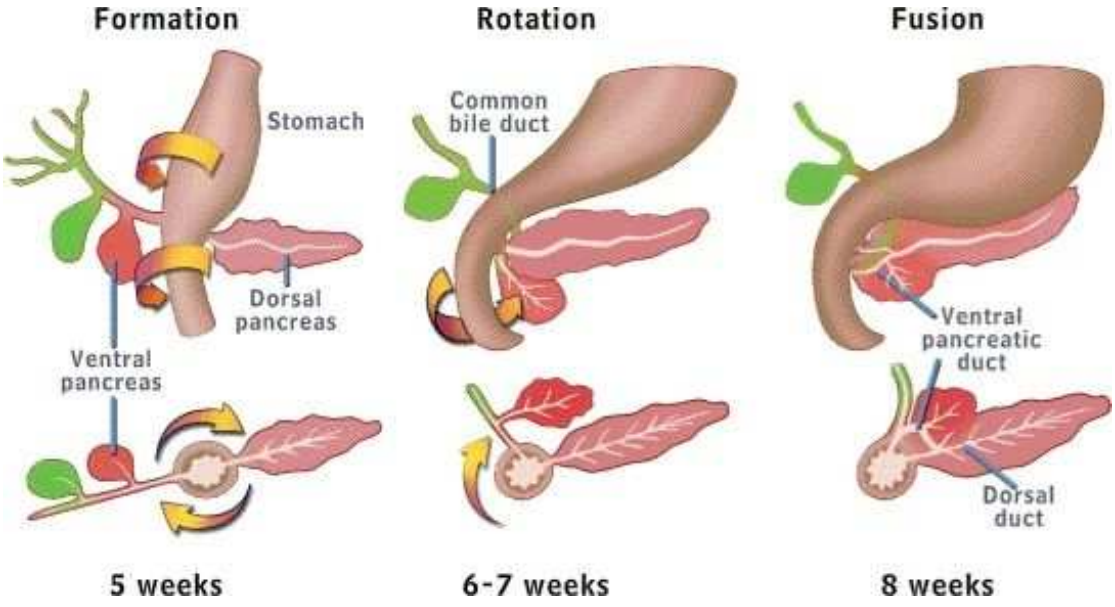
Since 1898, many surgeons undertook various steps for the resection of tumour of ampulla and Head of pancreas. Allen O. Whipple (1881-1963), son of American missionaries in Persia, was recognized as the “ Father of Pancreatic Surgery” for his successful single stage pancreatic head tumours.

In 1992, at the Atlanta Symposium, the clinically oriented classification system was established for Acute Pancreatitis.

EMBRYOLOGY

The pancreas develops from two separate growths(buds) of tissue, both of which arise from the distalforegut and develop within the mesenteries. The small ventral pancreatic bud branchesfrom the hepatic diverticulum in the ventral mesenteryand therefore shares a duct drainage systemwith the liver. The larger dorsal pancreatic bud formsin the dorsal mesentery. Rotation of the foregut to theright causes the ventral pancreatic bud and bile ductto rotate to the original dorsal aspect (now on the lefthand side) of the gut tube, where it joins and fuseswith the dorsal bud.

The ventral bud forms the pancreatichead and uncinate process whereas the dorsalbud forms the pancreatic neck, body and tail. Themain pancreatic duct, which joins the common bileduct to drain into the second part of the duodenumvia the major duodenal papilla, is formed by a unionof the duct systems of the ventral bud and the distalpart of the dorsal bud. The accessory pancreaticduct, which drains via the minor duodenal papilla, isformed from the duct system in the proximal part ofthe dorsal bud.Pancreatic tissue can be located in numerous ectopicpositions including within the stomach, duodenumor jejunum, or in an ileal diverticulum. Malformationof the ventral pancreatic bud, possibly as a result ofits bifurcation, can lead to an annular pancreas wherepancreatic tissue surrounds, and therefore obstructs,the second part of the duodenum.



DEVELOPEMENT OF PANCREAS

GROSS ANATOMY

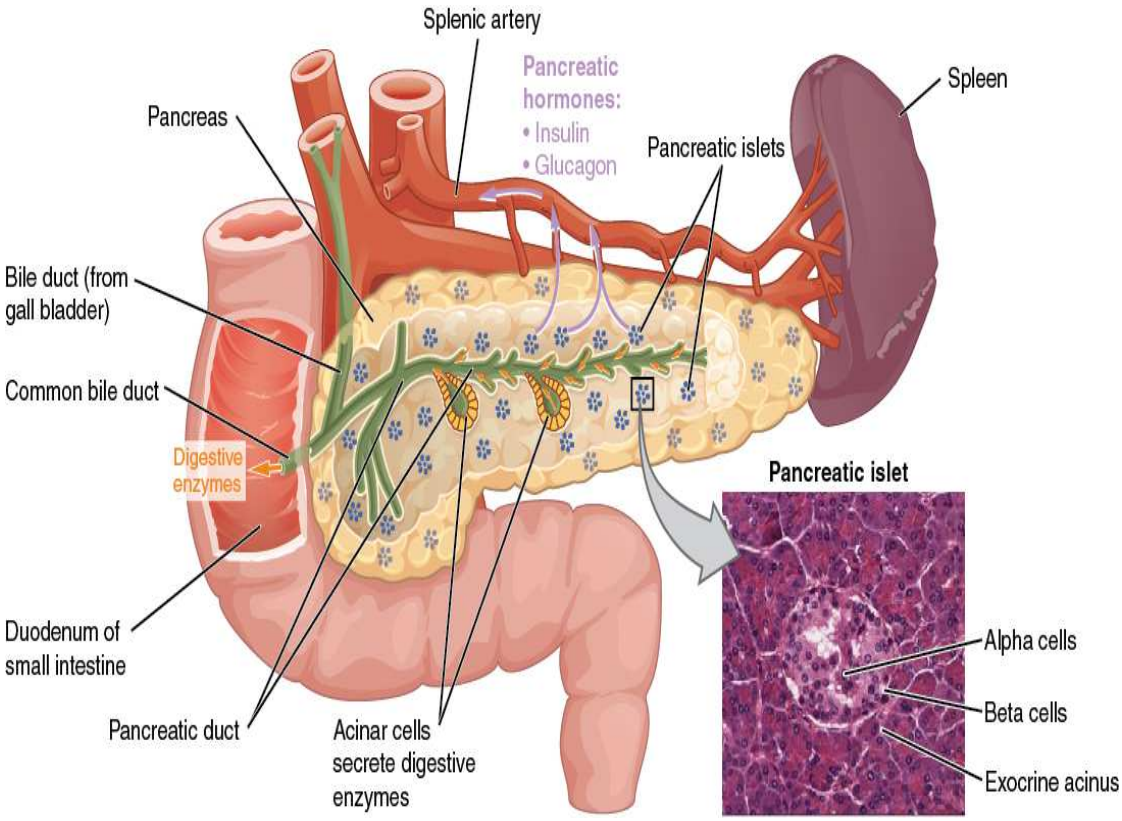
The pancreas is both an endocrine and an exocrine gland. It is about 15 cm long and weighs about 80 g. It possesses a headwith an uncinate process, a neck, a body and a tail, and liesobliquely across the posterior abdominal wall, crossing the 1st lumbar vertebra and the aorta and inferior vena cava. The head, the expanded right extremity of the gland, bears inferiorly the **uncinate process**. The body, triangular in section, has anterior, inferior and posterior surfaces; the tail is the narrow left extremity and lies in the lienorenal ligament.

RELATIONS

The head lies within the curve of the duodenum Anteriorly it is covered, from above downwards, by the pylorus,the transverse colon and the small intestine; posteriorly itlies on the inferior vena cava, the right renal vessels and thebile duct. The **uncinate process** lies on the left renal vein and the aorta and is crossed by the superior mesenteric vessels. The**neck**overlies the portal vein and is behind the pylorus and thegastroduodenal artery. Above the **body** is the coeliac artery, and the common hepatic and splenic arteries run along its superiorborder. Anteriorly lie the stomach and lesser sac. Inferiorly itssurface is covered by the peritoneum of the greater sac and it isrelated to coils of small intestine. The transverse mesocolon isattached by its mesentery to its anterior surface.

The body, fromright to left, lies on the aorta and superior mesenteric artery,the left crus of the diaphragm, the left renal vessels and theleft kidney, and the splenic vein runs behind it throughout itslength, being joined by the inferior mesenteric vein.

The **pancreatic duct** traverses thelength of the gland to the head of the pancreas, where it joinsthe bile duct in the ampulla before opening into the secondpart of the duodenum. An accessory duct drains the uncinated process and usually drains into the ampulla, but it may openseparately into the duodenum about 3 cm proximal to themain duct.



ANATOMY OF PANCREAS

BLOOD SUPPLY

This is from the splenic and superior and inferior pancreaticoduodenalarteries. The veins drain to the splenic vein and, viathe pancreaticoduodenal veins, to the superior mesenteric vein.

NERVE SUPPLY

This is from the thoracic splanchnic nerves and thevagus via the coeliac plexus. Pain fibres, whose cell bodies arelocated in the 6th to 10th thoracic segments, are conveyedwith the sympathetic nerves. Pancreatic pain is commonlyreferred to the back.

LYMPHATIC DRAINAGE

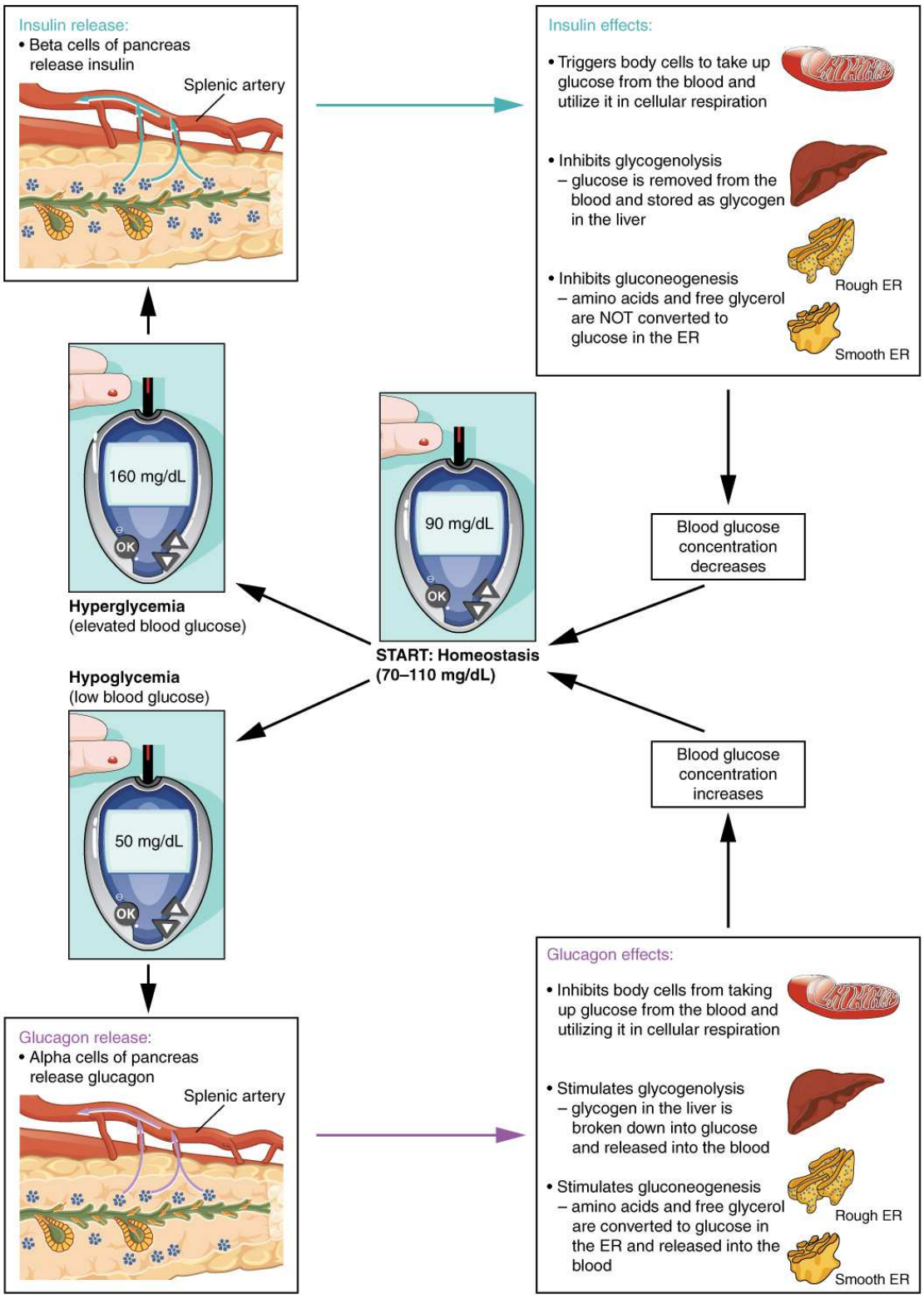
This is via suprapancreatic nodes to the preaortic coeliac nodes.

PHYSIOLOGY

Despite the disparate functions of the endocrine andexocrine parts of the pancreas, the two different componentscoordinate to regulate and respond to food digestionby secreting different hormones and digestive enzymes,with a regulatory feedback system in place. Pancreas mediates body’s energy metabolism throughislet cells of Langerhans.

Regulation based on the actions of Insulin and Glucagon. Insulin Raises the protein synthesis and reduces the lipolysis and glycogenolysis. Especially aftera meal or in a hyperglycemic state. Glucagon, on theother hand, is viewed as the hormone of energy release. Which raises the blood glucose by gluconeogenesis,glycogenolysis and lipolysis. Thus counteracts theeffects of insulin.β cells secrete insulin whenglucose is ingested enterally compared to the parenteralroute, indicating that a feed-forward mechanism in thedigestive tract is activated, anticipating the rise in blood glucose through*incretins*. glucose-dependentinsulinotropicpeptide, also known as gastric inhibitorypeptide (GIP), and glucagon-like peptide-1 GLP-1).

Bothare secreted by endocrine cells located in the smallintestinal epithelium when the concentration ofglucose increases which stimulate the β cells to secrete more insulin. Hence,the great interest in the pharmaceutical industry to developincretin-based therapies to treat diabetes, In addition toGLP-1's inhibitory effect on glucagon secretion and theability to increase food transit time in the stomach.Humoral inhibitors include somatostatin,amylin, leptin, and pancreastatin. Insulin secretion stimulated by vagus nerve and sympathetic systeminhibits it. Other substance like vasoactive intestinal peptide, substance p andneurotensinstimulatedby pancreas.

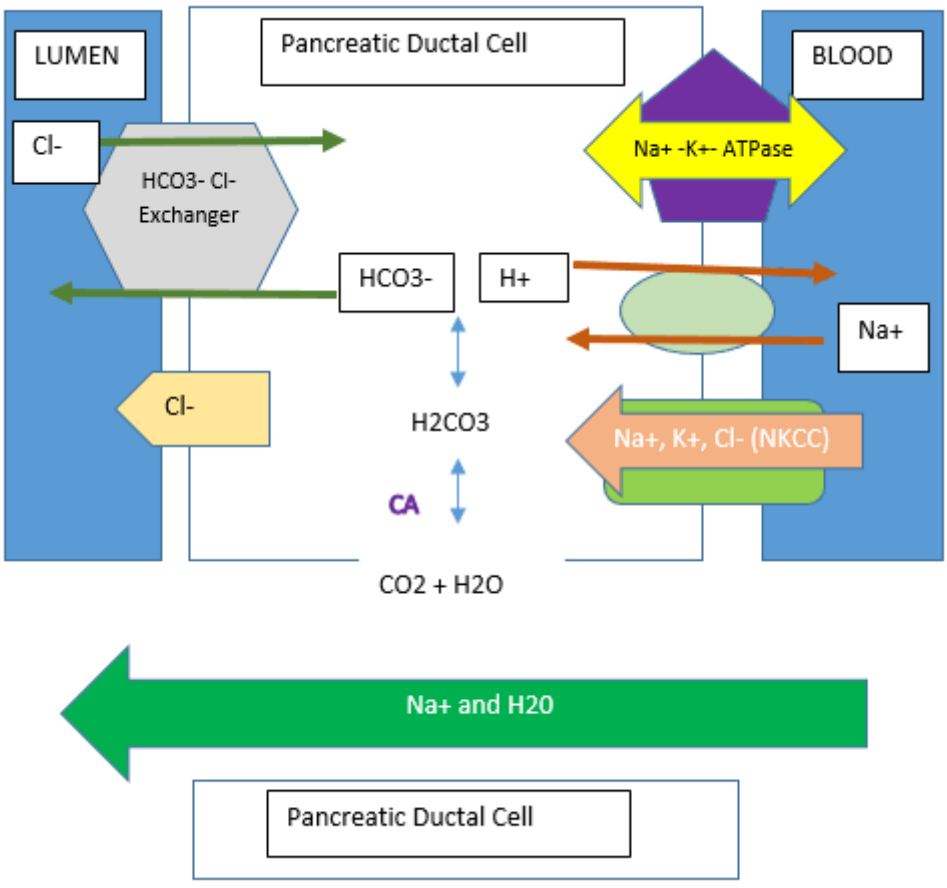


ENDOCRINE PANCREATIC SECRETION AND FUNCTIONS

Exocrine secretion function mediated by cholecystokinin and parasympatheticvagal discharge. The exocrine function is traditionallydivided into three phases: (1) the cephalic phase, whichis triggered by the sight and smell of food, comprises 10%to 20% of pancreatic excretion; (2) the gastric phase,which is triggered by food entering the stomach andgastric distention, comprises 15% to 20% of enzymeexcretion; and (3) the intestinal phase, which is triggeredby acidification of the duodenum and proximal jejunum,comprises 60% to 70% of meal-stimulated pancreaticexcretion.The exocrine portion of the pancreas iscomprised of a ductal tree along with a mass of acinarcells. Acidification and entry of fatty acids along with bilesalts in the duodenum stimulate secretin and VIP, in turnleading to the release of a bicarbonate-rich fluid fromductal cells. Vagal stimulation and the entry of eitherpeptides or fatty acids into the duodenum cause releaseof CCK and acetylcholine, producing the secretion of adigestive enzyme–rich fluid from the acinar cells.

Currently, the most widely accepted model of bicarbonatesecretion from the ductal cells involves the diffusionof carbon dioxide into the cell from the circulation, whereit is hydrated by carbonic anhydrase to form H2CO3.H2CO3 dissociates into H+ and HCO3−. The bicarbonateis transported into the ductal space by a chloride/bicarbonate exchanger. Secretin binds to receptors onthe basolateral membrane, activating adenylate cyclase toproduce cyclic adenosine monophosphate (cAMP).

cAMPin turn activates the cystic fibrosis transmembrane regulator(CFTR) on the luminal cell surface, allowing for thepassage of chloride into the ductal space. The passage ofbicarbonate and chloride across the ductal cell membranegenerates an ionic and osmotic gradient causing sodiumand water to follow.Defects in CFTR lead to both acuteand chronic pancreatitis through ductal and glandularobstruction secondary to the inability to hydrate the ductalmolecules in the lumen.



EXOCRINE PANCREATIC SECRETION

The lack of chloride ionsflowing into the lumen prevents the formation of an ionicand osmotic gradient. Therefore, sodium and water donot cross into the lumen, producing a low volume, thickenedsecretion and subsequent blockage. Pancreatitis israrely a complication in individuals with mutations ofboth CFTR alleles because this results in rapid destructionof the pancreas beginning in utero. Patients experiencethe loss of acinar cells, which are a necessary nidus forpancreatitis, leading to pancreatic insufficiency.Along with bicarbonate secretion, the second arm ofpancreatic exocrine function involves the release of digestiveenzymes from the acinar cells. Digestive enzymes aresynthesized in their inactive form within acinar cells andare packaged into zymogen granules. The granules migrateto the cell surface and fuse to the cell membrane releasingtheir contents in response to vagal stimulation, peptides,and fatty acids. Some enzymes, including amylase, lipase,RNAse, and DNAse are synthesized in their active forms,but most (trypsinogen, chymotrypsinongen, procarboxypeptidaseandproelastase) are inactive upon release. Theintestinal brush border enzyme, enteropeptidase, cleavestrypsinogen to its active form, trypsin. Trypsin cleaves andactivates the remaining digestive enzymes. More than 40mutations in cationic trypsinogen *(PRSS1)*, the gene thatencodes trypsin, have been uncovered.

The mutationsoften cause the premature activation of trypsinogen totrypsin, producing a condition characterized by recurrentepisodes of pancreatitis ultimately leading to pancreaticinsufficiency.

Serum amylase is increased 2.5 times higher than normal level in acute pancreatitis within 6hrs. Major drawback in serum amylase level analysis is less specificity to diagnose acute pancreatitis.The amylase-to-creatinine ratio (ACR) may useful in differentiatingacute pancreatitis from other conditions which produce raised amylase level.

Serum lipase level is morespecific in diagnosing pancreatic tissue damage becauselipase is only produced in the pancreas. Lipase raised in alcoholic pancreatitis and the amylase levelincreased in gallstone pancreatitis, hence the lipase-to-amylaseratio has been useful to distinguish between this two.

Hormones	Islet Cell	Functions
Insulin	β (beta cell)	Decreased gluconeogenesis, glycogenolysis, fatty acid breakdown, and ketogenesis
		Increased glycogenesis, protein synthesis
Glucagon	α (alpha cell)	Opposite effects of insulin; increased hepatic glycogenolysis and gluconeogenesis
Somatostatin	γ (delta cell)	Inhibits GI secretion
		Inhibits secretion and action of all GI endocrine peptides
		Inhibits cell growth
Pancreatic polypeptide	PP (PP cell)	Inhibits pancreatic exocrine secretion and section of insulin
		Facilitates hepatic effect of insulin
Amylin (IAPP)	β (beta cell)	Counterregulates insulin secretion and function
Pancreastatin	β (beta cell)	Decreases insulin and somatostatin release
		Increases glucagon release
		Decreases pancreatic exocrine secretion
Ghrelin	ε (epsilon cell)	Decreases insulin release and insulin action

IAPP = islet amyloid polypeptide.

ACUTE PANCREATITIS

Definition:

Acute pancreatitis is “an inflammatory disease, associated with little or nofibrosis of the pancreas”. There are several initiating factors, which includegallstones, alcohol, trauma, and infections, and, rarely hereditary.

Etiology of acute pancreatitis:

There are so many different factors have been implicated in the causation ofthis disease. On the basis of worldwide data, the most common cause aregallstones, account for about 45 percent of cases. Alcoholism is the secondcommon cause**,** in about 35 percent of cases. The disease occurs at higher rate in young men and older women. Femalesare more prone to have gall stone pancreatitis and males are more prone to havealcohol induced pancreatitis.

CAUSES OF ACUTE PANCREATITIS:

Alcohol

Biliary tract disease

Obstructive causes:

- Choledocholithiasis
- Ampullary carcinoma or pancreatic malignancy

- Papillary obstruction by worms/foreign bodies
- Pancreas divisum with minor duct obstruction
- Choledochocele
- Duodenal diverticula at periampullary region
- Spasm sphincter of Oddi

Toxins or drugs:

- *Toxins*:- ethanol/methanol, scorpion sting, organophosphorous compounds
- *Drugs*:- **Definite cause**
- 5-Aminosalicylate (ASA)
- 6-Mercaptopurine (6-MP)
- Azathioprine
- Cytosine arabinoside (cytarabine)
- Didanosine
- Diuretic agents
- Estrogens, etc.

Probable Cause

- Acetaminophen
- α-Methyl-DOPA
- L-Asparaginase
- Isoniazid (INH)
- Phenformin, etc.

Trauma:

- External / surgical traumatic injury to the abdomen.
- Iatrogenic injury- postoperative trauma, post ERCP, post endoscopicsphincterotomy and manometry of sphincter of Oddi

Metabolic abnormalities:

- Hypercalcemia
- Hypertriglyceridemia **Inherited conditions**

Infection:

- Parasitic:- ascariasis, Clonorchissinensis
- Viral:-mumps, rubella, hepatitis A, B, non-A, non-B, coxsackie B, echovirus, adenovirus, CMV, varicella, EBV, HIV.
- Bacterial:- mycoplasma pneumoniae, Campylobacter jejuni, Myco.tuberculosis, MAC, legionella pneumophila, leptospiral infection

Vascular causes:

- Hypo perfusion causing ischemia (e.g., after major cardiac vascular surgery)
- Athero-embolism
- Vasculitis-SLE, PAN, malignant hypertension
- **Miscellaneous causes:**
    - Peptic ulcer penetration
    - Cystic fibrosis
    - Crohn's disease
    - Reye's syndrome
    - Hypothermia

Idiopathic causes

The two major causes of acute pancreatitis are biliary calculi,which occur in 45-50% of patients, and alcohol abuse, which accounts for 35% of cases.Gallstone pancreatitis is thoughtto be triggered by the passage of gallstones down the commonbile duct. If the biliary and pancreatic ducts join to share acommon channel before ending at the ampulla, then obstructionof this passage may lead to reflux of bile or activated pancreaticenzymes into the pancreatic duct. Patients who havesmall gallstones and a wide cystic duct may be at a higherrisk of passing stones.

The proposed mechanisms for alcoholic pancreatitis include the effects of diet, malnutrition, directtoxicity of alcohol, concomitant tobacco smoking, hypersecretion,duct obstruction or reflux, and hyperlipidemia. Theremaining cases may be due to rare causes or be idiopathic.

Among patients who undergo ERCP, 1–3% develop pancreatitis,probably as a consequence of duct disruption andenzyme extravasation. Patients with sphincter of Oddidysfunctionor a history of recurrent pancreatitis, and those whoundergo sphincterotomy or balloon dilatation of the sphincter,carry a higher risk of developing post-ERCP pancreatitis.

Patients who have undergone upper abdominal or cardiothoracicsurgery may develop acute pancreatitis in the postoperativephase, as may those who have suffered blunt abdominaltrauma.

Hereditary pancreatitis is a rare familial condition associatedwith mutations of the cationic trypsinogen gene.Patients have a tendency to suffer acute pancreatitis while intheir teens, progress to chronic pancreatitis in the next twodecades and have a high risk (possibly up to 40%) of developingpancreatic cancer by the age of 70 years.Occasionally, tumours at the ampulla of Vater may causeacute pancreatitis.

It is important to check the serum calciumlevel, a fasting lipid profile, autoimmune markers and viraltitres in patients with so called idiopathic acute pancreatitis.

It is equally important to take a detailed drug history andremember the association of corticosteroids, azathioprine,asparaginase and valproic acid with acute pancreatitis. Statins(taken over a long time) and gliptins have been linked withpancreatitis, but the evidence is slim. It is essential to excludetiny gallstones. A careful search for the etiology must bemade in all cases, and no more than 20% of cases should fallinto the idiopathic category.

PATHOPHYSIOLOGY

Acute pancreatitis occurs in varying degrees of severity, the determinants ofwhich are multifactorial**.** It is generally believed that acute pancreatitis is triggeredby digestive enzymes which got activated inside acinar cells. This was thought tobe counter acted by endogenously secreted pancreatic enzyme inhibitor. Theultimate severity depends upon the event that subsequently occurs following theacinar cell injury. The events are activation and recruitment of inflammatory cell and mediates the inflammation.Large amounts of liberated digestive enzymes however overwhelm the system as awhole.

There are three reasons for this theory:

(a) The pancreas is digestible by the activated enzymes of the duodenum.

(b) Activated digestive enzymes are present within the pancreas in severe pancreatitis

(c) The Pancreatitis histology is suggestive of a coagulative necrosis.

However, the mechanism(s) of erroneous activation are not fully understood.

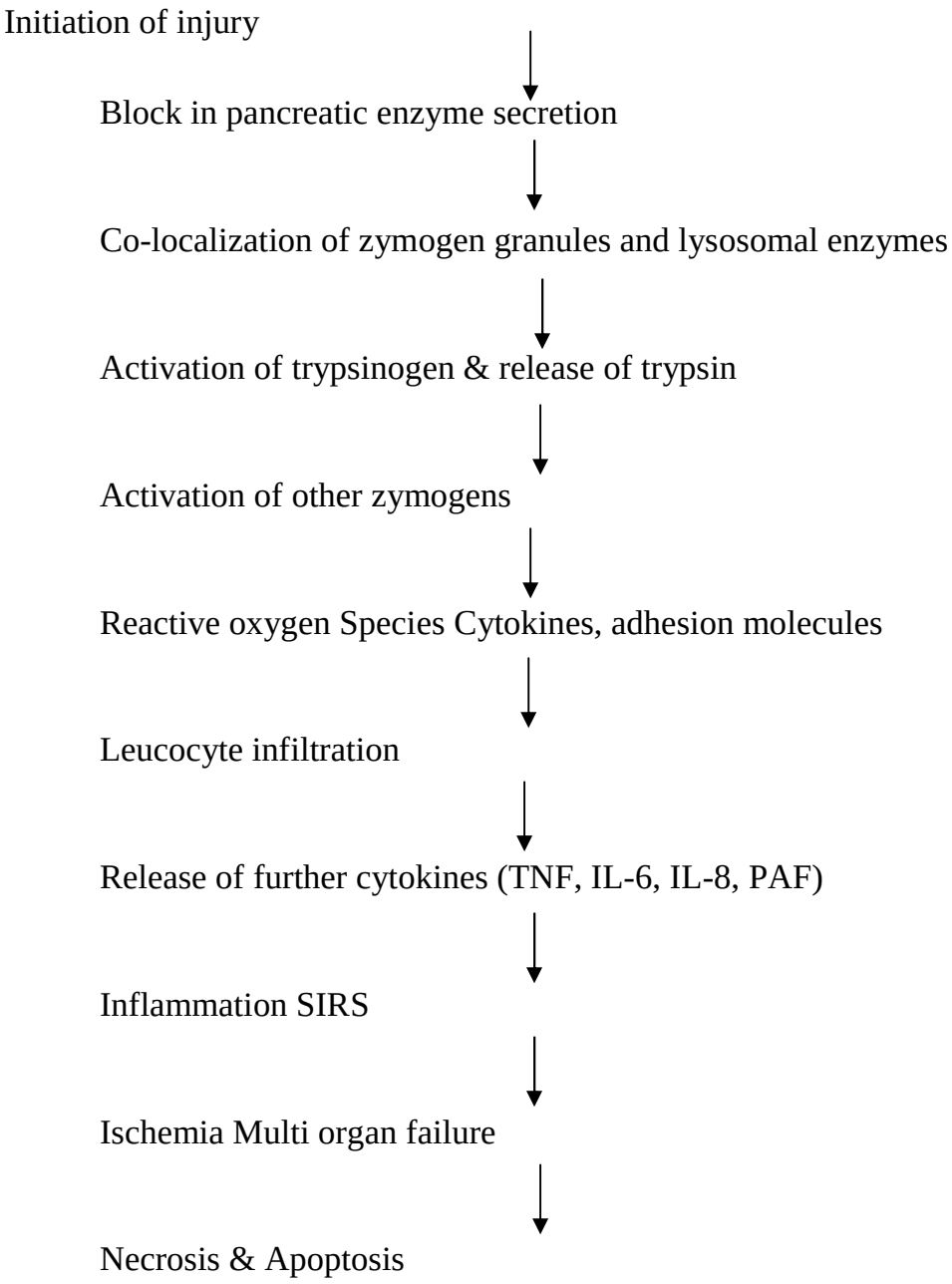
According to "***colocalization hypothesis***" digestive enzymes are confined withincytoplasmic vacuoles that also contain the lysosomal hydrolase Cathepsin B,which activates trypsinogen. Recent studies suggest that cathepsin Bactivity inhibition by highly specific inhibitor,

CA-74me, protects against intraacinarcell activation of trypsinogen and hence pancreatitis.

These findings suggest that the trypsinogen is activated because it erroneously colocalises in cytoplasmic vacuoles with cathepsin B.

Recent studies suggest that trypsin, once activated inside the colocalized vacuoles(appears similar to autophagic vacuoles), mediates thepermeability of these organelles and release of their contents into the cytosol.Cathepsin B is

one of the enzymes released into the cytosolduring pancreatitis. Which causes apoptotic cell death by permeabilizing mitochondrialmembranes, which allows cytochrome C to be released into the cytosol and the apoptoticdeath of the acinar cells.



Schematic representation of the mechanisms of pathogenesis ofacute pancreatitis.

CLINICAL PRESENTATION:

The clinical presentation, diagnosis, and management of an acute attack ofpancreatitis are similar regardless of whether that attack is acute or chronicpancreatitis. The acute pancreatitis can mimic like acute abdomen and shouldnever be excluded in differential diagnosis.Abdominal pain, nausea, and vomiting are the predominant symptoms. Eachepisode begins with severe pain, following a substantial amount of meal. Thecardinal symptom is usually epigastric pain, but can occur anywhere in theabdomen or lower chest. The pain was described as "knifing" or "boring through"to the back, while leaning forward the pain might be relieved(***Mohmadian prayerposition***). Pain starts 12-48 hours after a bout of alcohol or after a large meal incase of gall stone pancreatitis. Pain became generalized once peritonitis has been sets in.

Peritoneal dialysis, post-operative situations, legionnaire's disease are wellknown for the occurrence of uncommon painless pancreatitis.If patient develops generalized paralytic ileus abdominal distension andvomiting can occur. The vomiting may lead to gastro esophageal tears (i.e.,Mallory-Weiss syndrome) and upper gastrointestinal bleeding. Vomiting is moreintense in necrotizing pancreatitis than in edematous pancreatitis. Althoughvomiting and retching may be relieved by passage of a nasogastric tube, the painusually persists even after gastric decompression.

Fever is an important sign. Fever in the first week is due to acuteinflammation mediated by cytokines. Fever in the second or third week is

due toinfected pancreatic necrosis. Fever in gall-stone induced pancreatitis, may be dueto cholangitis and mandates prompt biliary decompression.

PHYSICAL FINDINGS:

On examination, the patient may be tachypneic, hypotensive, and hyperthermic, and have tachycardia. The temperature was minimally raised inuncomplicated pancreatitis. Mild guarding might be present overthe epigastric and left hypochondrial region. The bowelsounds may be decreased or absent. There is usually no palpable swelling or masses. Theabdomen may have minimal ascites and left sided pleural effusion.With increasing severity, there are sequestrations of fluid in the retroperitoneum that leads to life threatening intravascular fluid loss. This leads tohemoconcentration. There might be bleeding into the retro peritoneum or peritoneal cavity which might be dissect into the soft tissues and appearsas a bluishdiscoloration around the umbilicus (Cullen's sign) or in the flanks (GreyTurner'ssign) and the inguinal region (Fox's sign).

The severe intravascular fluid loss may lead to acute renal shutdown withelevated BUN and creatinine levels. And also there may be hyperglycemia,hypoalbuminemia, and hypocalcemia that are sufficient enough to produce tetanyin few cases.

DIAGNOSIS:

The clinical diagnosis is one of exclusion and diagnosis may be difficultdespite the plenty of investigation that are available.

SERUM PANCREATIC ENZYMES:

Serum pancreatic enzyme estimation is the gold standard for diagnosis.The reason is pancreatic acinar cells synthesize, store, and secrete a large amountof digestive enzymes (e.g., amylase, lipase, trypsinogen, and elastase), the levels ofwhich are elevated in the serum of most patients.Because of the ease of measurement, serum amylase levels are measuredmost often. Serum amylase concentration will increase immediately reaches thepeak value within several hours after the onset of disease and remains elevated for3 to 5 days before returns back to normal. There was no significant correlationbetween the magnitude of serum amylase rise and severity of pancreatitis. But,there are many nonpancreatic causes of hyperamylasemia (e.g., biliary tractdisease, intestinal obstruction, mesenteric ischaemia, acute appendicitis, mumps,parotitis, impaired amylase excretion etc.), that make the interpretation of thismarker difficult.

In contrast, a patient with acute pancreatitis may have a normalserum amylase level, which could be due to several reasons like patients with Hyperlipidemia; values might appear to be normal because of interference by lipidswith chemical determination of serum amylase. The urinary amylase clearancefrom the circulation increases during pancreatitis; therefore, the urinary amylaselevels might be more sensitive than serum levels. For these reasons, it isrecommended to measure the urinary amylase concentrations, which usuallyremain elevated for several days after serum amylase levels have returned back tonormal. In patients with severe pancreatitis associated with significant necroticdamage, the pancreas may not release large amounts of enzymes into thecirculation. It is important to recognize that, in patients with severe pancreatitis,frequent measurement of serum enzymes is not needed. Patients with alcoholicpancreatitis, in general, have a smaller increase in serum amylase levels. Becausehyperamylasemia can be observed in many extra pancreatic diseases, measuringpancreatic-specific amylase (p-amylase) rather than total amylase, whichalso includes salivary amylase, makes the diagnosis more specific(88 to 93%).

The serum lipase estimation has been found to have high sensitivity andspecificity in the diagnosis as there are no other sources of lipase. Totalamylase is having a sensitivity of 84%, the serum P- amylase has 95% and lipasehas 93%. Specificities for amylase, P-mylase and lipase respectively are-88%,93% and 96%, respectively. Thus P-amylase is the enzyme with the higherdiagnostic value.The rise of lipase: amylase has been found to

differentiate alcoholic fromnonalcoholic pancreatitis. The serum (SGPT) alanine aminotransferase level rise ofthree or more times above the base-line value has great specificity in diagnosinggallstonepancreatitis.Immunologic assay like serum trypsinogen or immune lipase are generallyless specific than the lipase assay. The increased urinary level of activationpeptides released during either trypsinogen, procarboxypeptidase, orprophospholipase activation, may aid in predicting the severity of an attack.Leucocyte migration and activation has considered as major determiningfactor of local & systemic complications.Although methemalbumin levels sometimes rise during attacks of severepancreatitis, and methemalbuminemia is indicative of a poor prognosis,methemalbumin levels are usually not measured. Circulating levels of severalinflammatory mediators and acute phase reactants(e.g., IL-1, 6, TNF-alpha, and CRP) also increase during pancreatitis, and themagnitude of those increases can be used to predict the severity of an attack.C-reactive protein is readily available in all centers and vales > 120mg/L, after 72hours are closely related to necrotizing pancreatitis.

IMAGING:

In general, the plain chest and abdominal radiographs can be useful in themanagement by identifying other causes for the patient's symptoms (e.g.,pneumonia, perforated hollow viscous, mechanical bowel obstruction).

Plainabdominal X-ray findings are either generalized or local ileus (known as sentinelloop), colon “cut-off” sign or “renal halo” sign. A chest

radiograph may show leftpleural effusion, elevated left hemi diaphragm or basal atelectasis.

ULTRASONOGRAPHY:

Abdominal ultrasound (US) examination is the gold standard forconfirmation of gallstones pancreatitis. It also helpful to detect extra pancreaticductal dilations & pancreatic edema, swelling, free peritoneal fluid andperipancreatic acute fluid collections (PFCs).It may not be sensitive in about 20%of cases, due to bowel gas interference with the imaging.

CT SCAN:

The contrast-enhanced computed tomography (CECT), has become goldstandard for

- Diagnosis
- Assessing the severity
- Detection of complications of acute pancreatitis.

The Balthazar scoring system and other similar grading systems haveincorporated various CT findings such as inflammation and fluid collections in &around the pancreas to correlate radiographic appearance with

morbidity andmortality.Early CT scans often fail to detect evolving necrosis, which become welldemarcated by 2 to 3 days after the onset of symptoms. The CT scans are notuseful in diagnosing necrosis or predicting the severity within the 24 hours of onsetof illness. The sensitivity for identifying pancreatic necrosis using contrastenhancedCT scan approaches 100%, 4 days from diagnosis. CT scans also beenuseful in the early diagnosis of infected pancreatic necrosis and image guidedaspiration of necrosis, when patient not improving clinically or who experienceclinical decline. In the patient with moderate renal impairment or allergy tointravenous contrast material, magnetic resonance imagining (MRI) may be useful.MRI has been found to have sensitivity and specificity similar to contrast-enhancedCT for detecting severe acute pancreatitis.ERCP should be done in patients with acute pancreatitis , whose clinicalcourse fails to improve despite full intensive care support, and in whom ampullaryor common bile duct stone impaction is suspected, based on ultrasonography, orclinical/biochemical signs of cholangitis. It may also be helpful in patients withrecurrent attacks of acute pancreatitis, without any obvious cause. It is useful incorrecting potentially correctable lesions such as CBD stones with impaction,pancreas divisum, ampullary stenosis, pancreatic duct stenosis etc.

FACTORS DETERMINING THE SEVERITY OF PANCREATITIS:

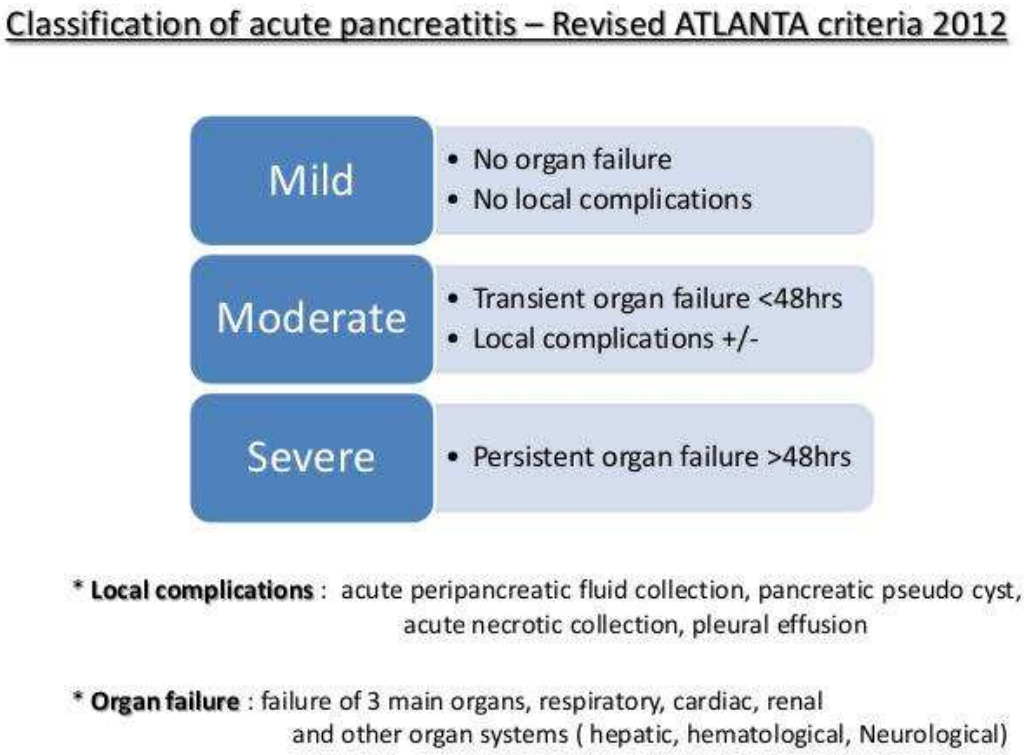
The severity of acute pancreatitis varies significantly. Some may have mildform of the disease that is self-limiting, while others suffer a more severe andsometimes lethal attack. Severity in acute pancreatitis is multifactorial which is important to detect the cause as early as possible to decrease the morbidity and mortality associated with the disease.

#**Severity of Acute Pancreatitis as Defined in the Revised Atlanta Classification**

REVISED ATLANTACLASSIFICATION

Complications	**Mild**	**Moderate**	**Severe**
Local complications	No	Yes	Yes
Systemic complications	No		
Transient organ failure	-	Yes	Yes
Persistent organ failure	-	No	Yes
Exacerbation of preexisting comorbidity	-	Yes	Yes

Local complications: acute peripancreatic fluid collection, pseudocyst formation, pancreatic necrosis and pleural effusion.



Scoring Systems in Acute Pancreatitis

Cutoff for PredictedSevere Acute Pancreatitis

- APACHE II ≥8 in first 24 h*
- BISAP ≥3 in first 24 h
- Modified Glasgow (or Imrie) ≥3 in first 48 h
- Ranson≥3 in first 48 h
- Urea at admission >60 mmoL/L

- C-reactive protein >150 U/L in first 72 h

*After onset of symptoms.

APACHE, Acute physiology and chronic health evaluation; *BISAP,* bedsideindex for severity in acute pancreatitis

TREATMENT:

There are two phases in evolution of an acute attack of pancreatitis. Bothphases are overlapping on each other.The initial phase, which lasts for 1 to 2 weeks, involves an acuteinflammatory and autodigestive process that takes place within and around thepancreas. It may have systemic effects as well.The second phase, that may last for weeks or months, is primarilycharacterized by the development of local complications that are, themselves, theresults of necrosis, infection and pancreatic duct rupture.Immediate management inpancreatitis patients is early diagnosis, assessing the severity, treating the major symptoms,and haltering the disease progression. The treatment for acute pancreatitis islargely supportive. Since 15-30 % patients develop severe pancreatitis, so each andevery patient should be treated aggressively. The main aim of the treatment is‘allowing rest to the gland’ by oral feed and fluids restriction. The goal of initialmanagement consists of adequate fluid replacement, correction of electrolyteimbalance, nutritional support and prevention of local & systemic complications.

Early management of severe acute pancreatitis.

- Admission to HDU/ICU
- Analgesia
- Aggressive fluid rehydration
- Supplemental oxygen
- Invasive monitoring of vital signs, central venous pressure, urineoutput, blood gases
- Frequent monitoring of haematological and biochemicalparameters (including liver and renal function, clotting, serumcalcium, blood glucose)
- Nasogastric drainage (only initially)
- Antibiotics if cholangitis suspected; prophylactic antibiotics can beconsidered
- CT scan essential if organ failure, clinical deterioration or signs ofsepsis develop
- ERCP within 72 hours for severe gallstone pancreatitis or signs ofcholangitis
- Supportive therapy for organ failure if it develops (inotropes,ventilatory support,haemofiltration, etc.)
- If nutritional support is required, consider enteral (nasogastric)feeding
- CT, computed tomography; ERCP, endoscopic retrogradecholangiopancreatography; HDU,high-dependency unit; ICU, intensive careunit.

MANAGEMENT OF PAIN

Good analgesics should be given to these patients as the pain can be verysevere in intensity. Most patients require narcotic analgesics. Meperidine ispreferred as morphine induces spasm of the sphincter of Oddi, which can, at leasttheoretically, worsen biliary pancreatitis.

FLUID AND ELECTROLYTE MANAGEMENT

Aggressive fluid resuscitation is important to replenish extravascular, or"third space," fluid losses, which may be considerable. The fluid resuscitation is of utmost importance to preventsystemic complications, mainly acute renalinsufficiency, that may occur with hypovolemia. Transudation of the fluid fromintravascular space into the areas of inflammation (i.e., peripancreatic,retroperitoneum and into the pulmonary parenchyma and soft tissues elsewhere inthe body) is the principle cause of hypovolemia. Furthermore, studies have shownthat inadequate resuscitation may add upon as a significant risk that leads to furtherpancreatic injury.

Banks and colleagues have showed that while aggressive fluid resuscitationmight not prevent the progression to develop pancreatic necrosis. The degree andintensity of monitoring depends upon the disease severity.During the first several days of a severe attack, circulating levels of

manyproinflammatory factors, including cytokines and chemokines, are elevated. Thisso-called ***"cytokine storm"***, in many cases, triggers the systemic immune responsesyndrome, and as a result, the hemodynamic parameters of these patients mayresemble those of sepsis associated with other disease states. Heart rate, cardiacoutput, and cardiac index usually rise, and total peripheral resistance falls.Hypoxemia can also occur as a result of the combined effects of increasedintrapulmonary shunting and a pancreatitis-associated lung injury that closelyresembles that seen in other forms of ARDS. Fluid management, though critical,may be difficult when hypovolemia is combined with respiratory failure of ARDS.Measurement of central filling pressures, using a Swan-Ganz or centralvenous pressure catheter, can be helpful in guiding fluid management, particularlywhen hypovolemia is combined with lung injury.

NASOGASTRIC DECOMPRESSION

The nausea and vomiting of pancreatitis can result in significant fluid as wellas electrolyte losses and retching can lead to gastro-esophageal mucosal tears andresult in upper gastrointestinal bleeding (i.e., the Mallory-Weiss syndrome).

Forsymptomatic relief and toincrease patient comfort, nasogastric decompression maybe needed, although the institution of nasogastric drainage does not shown to alterthe eventual outcome of an attack.

PROPHYLACTIC ANTIBIOTICS

Infection is a serious complication of acute pancreatitis and is the mostcommon cause of death. It is mostly caused by the enteric bacteria and was seencommonly in necrotizing pancreatitis. Local infection were common with largeramounts of pancreatic necrosis, and this increases in incidence as time progressesfor at least the first 3 weeks in the course of the disease. Aerobic and anaerobicgastrointestinal floras are the primary organisms involved, and infections may beeither mono or polymicrobial in nature. The predominant microbes seen were E.coli (35%),Kleb.pneumoniae (25%), Streptococcus (25%),Staphylococcus(15%), and Pseudomonas (10%).The association of high mortality with pancreatic infection has been the rationale behind the use of prophylactic antibiotics widely in patients with pancreatic necrosis. In severe pancreatitis, beneficial effects have beenobserved with regimens that included imipenem alone, imipenem withcilastatin, metronidazole and third-generation cephalosporin (cefuroxime). Because*Candida* species are common inhabitants of the upper GI tract, *Candida* sepsis andsecondary fungal infection of pancreatic necrosis is a risk in severe disease, andmany surgeons advocate empirical therapy with fluconazole in severe acutepancreatitis.The duration of treatment has not defined clearly.

A treatment course of1week to 4 weeks has been recommended commonly, but many of them limit thetreatment to 2 weeks.According to the current UK guidelines (Johnson 2005), the duration ofantibiotic prophylaxis is 1 to 2 weeks.

NUTRITIONAL SUPPORT

Classically speaking, the enteral feeding should be limited, therebypancreatic stimulation and further pancreatic injury by the release of proteolyticenzymes can be avoided. Recent data, suggests that such strict limitations ofenteral nutrition may have been unnecessary. Most of the severe acute pancreatitispatients found to have prolonged course of illness with hyper catabolic state andileus that have led to a generous use of parenteral nutrition in them.

The points favoring enteral nutrition are

- It might feasible, safe, and desirable in severe pancreatitis.
- It has the advantage of avoiding the high cost of total parenteral nutrition(TPN) as well as its associated catheter-related complications.
- The use of enteral nutrition may support intestinal mucosal integrity byavoiding the alteration in intestinal permeability & barrier function as seenwith use of TPN.

COMPLICATIONS OF ACUTE PANCREATITIS.

Systemic

(More common in the first week)

Cardiovascular

- Shock
- Arrhythmias

Pulmonary

- ARDS

Renal failure

Haematological

- DIC

Metabolic

- Hypocalcaemia
- Hyperglycaemia
- Hyperlipidaemia

Gastrointestinal

- Ileus

Local

(Usually develop after the firstweek)

Acute fluid collection

Sterile pancreatic necrosis

Infected pancreatic necrosis

Pancreatic abscess

Pseudocyst

Pancreatic ascites

Pleural effusion

Portal/splenic vein thrombosis

Pseudoaneurysm

Neurological

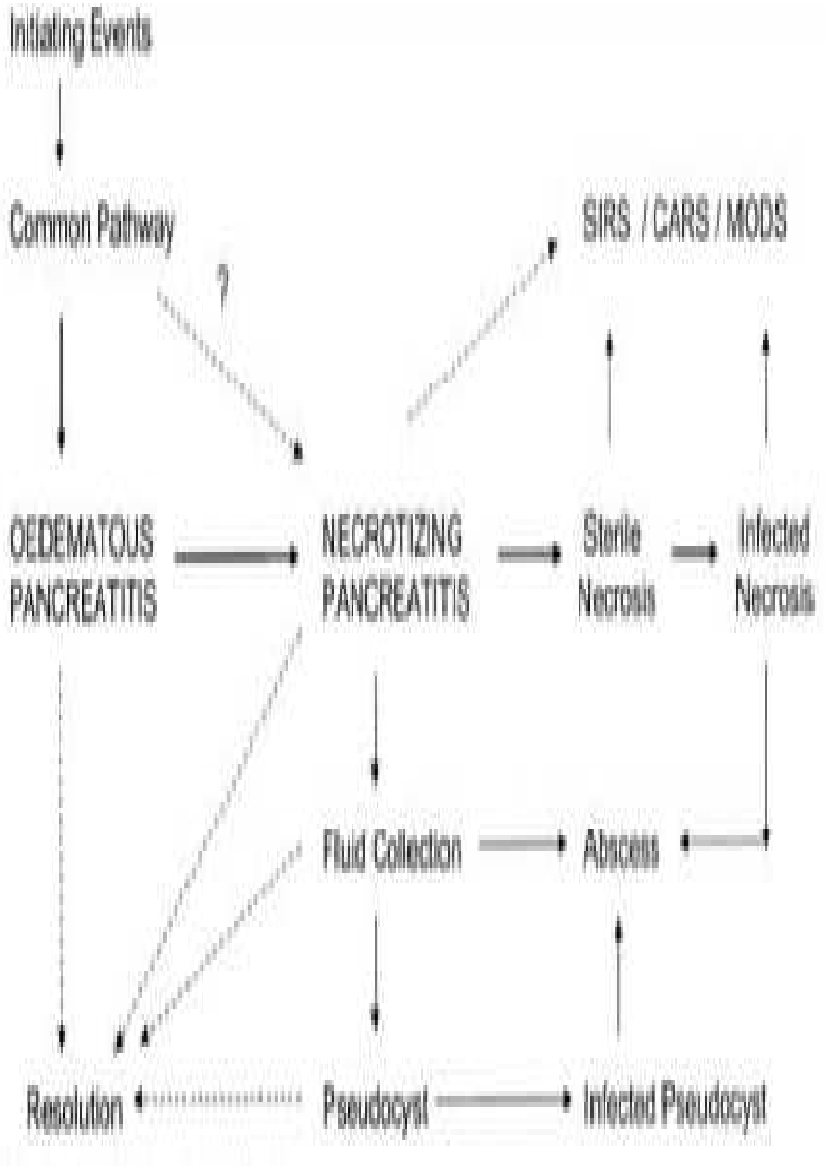
- Visual disturbances
- Confusion, irritability
- Encephalopathy

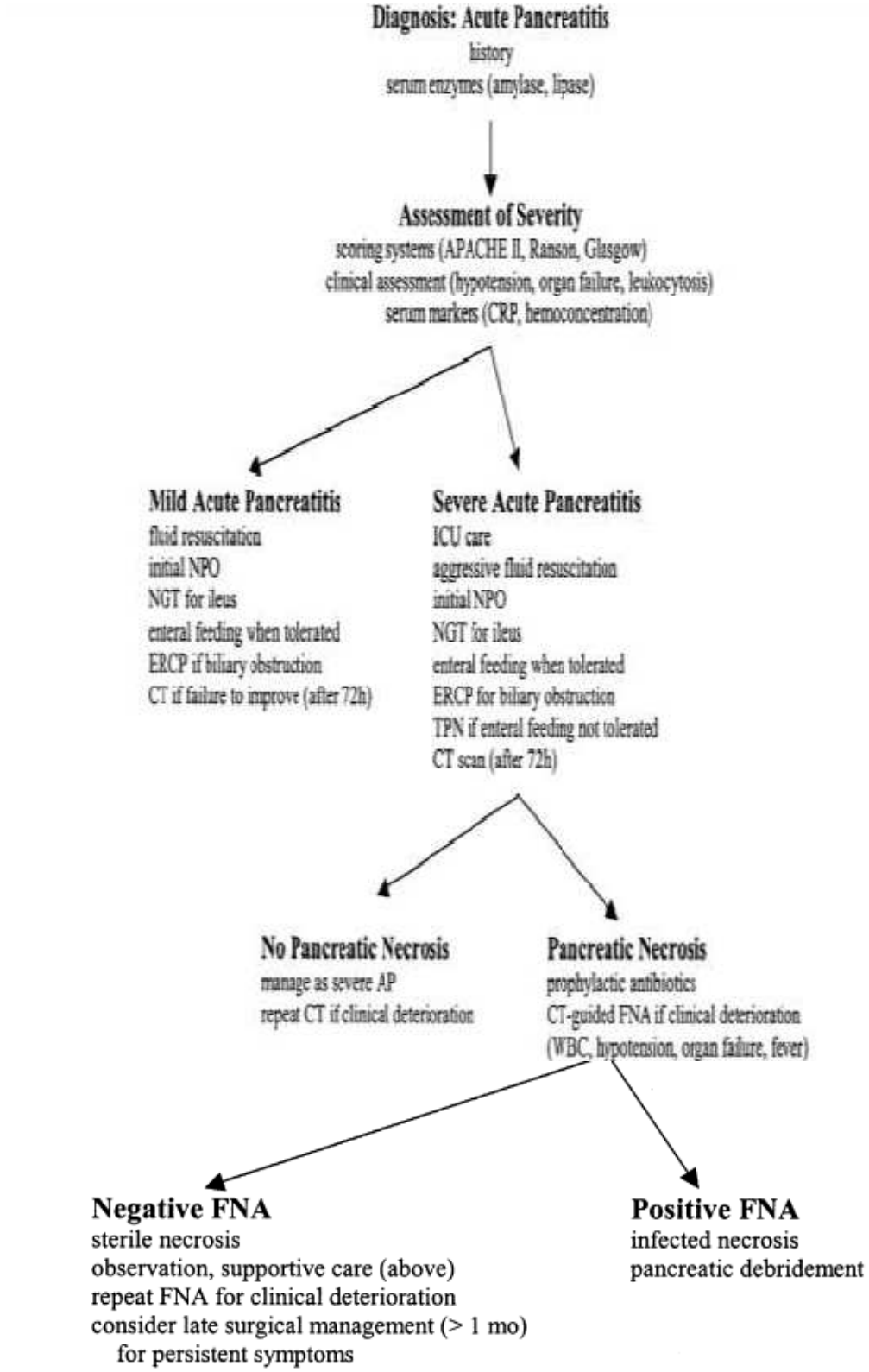
Miscellaneous

- Subcutaneous fat necrosis
- Arthralgia

ARDS, acute respiratory distress syndrome; DIC, disseminated intravascularcoagulation.

Schematic representation of Acute Pancreatitis Complications





Schematic representation of acute pancreatitis and its management

TREATMENT OF EARLY SYSTEMIC COMPLICATIONS OF PANCREATITIS

The pathogenesis and management of the cardiovascular collapse,respiratory failure, renal failure, metabolic encephalopathy, gastrointestinalbleeding, and disseminated intravascular coagulation that complicate severepancreatitis appear to be identical to those involved when these processes are superimposed on other disease states that are characterized by peritonitis andhypovolemia.

Cardiovascular collapse is largely caused by hypovolemia, and itsmanagement requires aggressive fluid and electrolyte repletion.The pulmonary manifestations of pancreatitis include atelectasis and acutelung injury. The latter appears to be similar to the acute lung injury caused by othersystemic processes, including septic shock, ischemia and reperfusion, and massiveblood transfusion. Management includes good pulmonary toilet combined withclose monitoring of pulmonary function. For many patients, intubation andrespiratory support may be required.Renal failure in pancreatitis is usually prerenal and is associated with a poorprognosis. In severe cases, dialysis, usually hemodialysis, may be required.Stress-induced gastro duodenal erosions account for most of thegastrointestinal bleeding, prophylaxis with antacids, H2-receptor antagonists, orproton pump inhibitors may be appropriate.Rarely, massive bleeding can result from injury to peripancreatic vascularstructures, leading to hemorrhage into the retroperitoneum.

The peripancreaticinflammatory process can also cause thrombosis of major gastrointestinal vesselsand result in ischemic lesions involving the stomach, small intestine, or colon that can cause bleeding. Management of these complications of pancreatitis is similarto that involved when they occur in the absence of pancreatitis.Some patients with severe pancreatitis develop disseminated intravascularcoagulation, but it rarely causes bleeding, and prophylactic heparinization isusually not indicated.Removal of precipitating factors, such as drugs or alcohol, is appropriate.After the first week, local complications has to be treated appropriately.

An indication for operative intervention in acute pancreatitis is the drainageof an infected pancreatic necrosis. These patients require removal of as much aspossible of the infected necrosis and drainage for the remaining viable exocrinetissue. Current opinion is against debridement in sterile necrosis unless it isaccompanied by life threatening systemic complications.A pancreatic abscess occurs 2 to 6 weeks after an initial attack of acutepancreatitis, in contrast to infected necrosis which occurs in the first few hours ordays. Treatment consists of external drainage, either by surgical or percutaneous catheter based measures.

MANAGEMENT OF BILIARY PANCREATITIS

The presence of gallstones leading to choledocholithiasis is recognized as amajor etiological factor worldwide. Endoscopic retrograde cholangiopancreatography (ERCP) has both diagnostic and most therapeutic utility inpatients with biliary obstruction or cholangitis.Magnetic resonance cholangiopancreatography (MRCP) is an additional alternative to ERCP s a diagnostic tool that avoids the risk of post procedure pancreatitis.Cholecystectomy with intra-operative CBD exploration is probably the best option for otherwise healthy patients with obstructive pancreatitis.However, patients who are at high risk for surgical intervention are best treated by endoscopic sphincterotomy, with clearance of stones by ERCP.

SURGICAL MANAGEMENT: INDICATIONS AND TIMING

There are very limited indications for surgical intervention; specifically,intervention may be needed to address the etiology of pancreatitis or itscomplications. Interventions, either surgical or endoscopic, to prevent recurrentgallstone pancreatitis are recommended in any patient with suspectedcholedocholithiasis. Delayed surgery is also, rarely needed for the treatment oflocal complications like pseudocysts. Early surgical intervention can lead to significant hemorrhage from thepancreatic bed, which may difficult to control, due to the fact that endarteritisobliterans was incomplete and the delineation between viable & non-viable tissuemight not be clearly made out.

Table 36–3. Indications for Surgical Intervention in Necrotizing Pancreatitis
Diagnostic uncertainty
Intra-abdominal catastrophe unrelated to necrotizing pancreatitis such as perforated viscus
Infected necrosis documented by FNA or extraluminal gas on CT
Severe sterile necrosis
Symptomatic organized pancreatic necrosis

SCORING SYSTEMS IN ACUTE PANCREATITIS

Pancreatitis is a serious disease with high morbidity and mortality rates.Some 80% were mild attack which recovers rapidly with conservativemanagement. The rest of 20% were severe, with protracted course that needsintensive care and specialized management. Several predictors of severity arecommonly used for this purpose.Scoring systems can be used to predict mortality, severity of disease andintensity of its complications. Prognostic factor analysis found to helpful incomparing the results, in-between the series of patients under study.Several scoring scales exist that predict both mortality and morbidity inpatients with acute pancreatitis.

These systems include:

- Ranson’s criteria
- Balthazar computed tomography (CT) grading
- Imrie Glasgow coma score (GCS)
- Bank’s clinical Criteria Simplified acute physiology score(SAPS)
- Marshall Multiple organ failure (MOF) score and
- Acute physiology and chronic health evaluation (APACHE) I, II, III & O.

The GCS and Ranson's multiple scoring systems require 48 hours of datacollection; however, APACHE can be calculated at any time and shows prognosticcorrelation with acute pancreatitis, as increasing scores are associated with poorprognosis.Once the acute pancreatitis has been diagnosed, assessment of severity isextremely important for execution of appropriate measures, preferably in an ICUsetup with close monitoring.

1) RANSON'S CRITERIA:

In 1974, Ranson and Pasternak identified 11 parameters with prognosticsignificance. Mortality was related to the number of parameters present: 0-0.9% inpatients with less than three positive prognostic signs, 10-20% in those with threeto five positive signs, mortality increases to > 50% in those with > 7 positive signs.

Criteria for Pancreatitis not due to gall stones:

At admission or diagnosis:

- Age more than55 years
- WBC count > 16,000/mm3
- Blood sugar> 200 mg/dL
- Serum LDH> 350 IU/L
- AST > 250 U/dL

During initial 48 hours:

- Fall in hematocrit> 10 percentage points
- BUN elevation > 5 mg/dL
- Serum calcium level < 8 mg/dL
- Arterial Po2 less than 60 mm Hg
- Base deficit more than 4 meq/L
- Estimated fluid sequestration > 6 L

Gall stone induced pancreatitis:

Recently, the cutoff values of these signs were modified in biliarypancreatitis. This limits the use of early prognostic signs; it now requiresmemorization of 18 separate parameters and etiology is not always known.Therefore the revisions for biliary pancreatitis have not had wide acceptance, andthe original system is the one that is widely utilized.

***On admission or diagnosis*:**

- Age > 70 yrs
- WBC count > 18,000/mm3
- Blood sugar> 220 mg/dL
- Serum LDH> 400 IU/L
- AST > 250 U/dL

During initial 48 hours: Fall in hematocrit greater than 10 percentage points

- BUN elevation > 2 mg/dl
- Serum ca2+ level < 8 mg/dl
- Base deficit more than 5 meq/L
- Estimated fluid sequestration > 4 L

2) IMRIE'S PROGNOSTIC CRITERIA:*With in 48 hours of admission*

- WBC count> 15000/cu.mm
- Blood sugar > 180mg/dl
- Serum urea > 44mg/dl (no response to IV fluids)
- Po2 level < 60 mm Hg
- Serum ca2+ level < 2 mmol/L
- LDH> 600 IU/L
- AST / ALT>200U/L
- Serum albumin level < 32 g/L

Ranson's and Imrie's scores indicate the severity at the time of admissionand are not intended for monitoring the clinical course.

3) BANK'S CLINICAL CRITERIA:

- Cardiac: Shock, tachycardia, arrhythmia, ECG changes
- Pulmonary:Dyspnoea, basal rales, PO2< 60 mm Hg, ARDS
- Renal: Urine output < 50 ml/h, rising BUN& creatinine
- Metabolic:Low Ca2+&pH; albumin
- Haematological: decreased HCT, DIC
- Neurological: cerebral Irritation & confused state
- GIT: paralytic ileus, free fluid, haemorrhagic peritoneal tap
- If the score was ≥ 1, the disease was severe in intensity.

4) BALTHAZAR COMPUTED TOMOGRAPHY SEVERITY INDEX(CTSI):

PROGNOSTIC INDICATOR	POINTS	GRADE
Pancreatic inflammation		
Normal pancreas	0	**A**
Focal or diffuse enlargement of the pancreas	1	**B**
Intrinsic pancreatic abnormalities with inflammatory changes in peripancreatic fat	2	**C**
Single, ill-defined fluid collection or phlegmon	3	**D**
Two or more poorly defined collections or presence of gas in or adjacent to the pancreas	4	**E**
Pancreatic necrosis		
None	0	
≤ 30%	2	
> 30–50%	4	
> 50%	6	

5) MODIFIED GLASGOW CRITERIA:

Used in both biliary and alcoholic pancreatitis.

The score ≥ 3 indicates severe pancreatitis requires ICU care.

- P - PaO2 <8kPa or < 60 mmhg
- A - Age more than 55 years old
- N - Neutrophilia with WBC count>15000 cells/cu.mm.
- C - Ca2+<2mmol/L or < 8 mg/dl
- R - Renal function, Urea > 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

Table 2. Criteria for organ failure based on Marshall scoring system

Organ system	Score				
	0	1	2	3	4
Respiratory (P_aO_2/FO_2)	>400	301–400	201–300	101–200	<101
Renal (serum creatinine, mg/dl)	≤1.5	>1.5 to ≤1.9	>1.9 to ≤3.5	>3.5 to ≤5.0	>5.0
Cardiovascular (systolic blood pressure, mm Hg)	>90	<90, fluid responsive	<90, fluid unresponsive	<90, pH <7.3	<90, pH <7.2

7) THE APACHE (ACUTE PHYSIOLOGICAL AGE AND CHRONICHEALTH EVALUATION) SYSTEM

Knaus et al (1981) proposed a scoring system APS for classifying thepatients according to the disease severity. This was based on recording theabnormal physiological parameters. In consultation with a large number ofintensive care specialists, they devised a scale. That included an acutephysiological assessment, which examined abnormality among 34 possiblemeasurements obtained during the 1st day of admission to the intensive care units.A number from zero to four was assigned to each measurement according to howfar from normal the measurements vary.When multiple values for the same measurement were available, the worstwas chosen. The final score, which ranged from zero to 124, indicates how farfrom normal homeostasis a patient had strayed because of acute illness.The true APACHE score was more difficult to calculate because of practicalproblems like collection of large number of variables. Also under the rules ofAPACHE system any unmeasured variable was assumed to be normal andweighted as zero. This gave rise to questions about the model's generalapplicability. Another major criticism of original APACHE system was that thevariables were chosen by a group of physicians and there was a potential of bias.

APACHE II

In 1985, Knaus et al developed this scoring system based on 12physiological variables30. To calculate the score, 0 - 4 values were assigned to allthe 12 physiological and laboratory values with 0 being normal and 4 being the most abnormal. APACHE II did not strictlydepend on ICU setting only but it wasfound to be as reliable as APS outside the ICU settings. The age and chronic healthproblems were included in this score as they reflect the physiological reservestatus.

APACHE II Score as published by Knaus et al is composed of three parts:

1) Acute Physiological Score (0 to 60 points)

2) Age points (0 to 6 points)

3) Chronic health points (0 to 5 points).

Range of potential score is 0 to 71 but scores above 40 are uncommon. Scoreabove 30 areassociated with mortality rate of at least 70%. Roumen et al, in theirstudy on acute hemorrhagic necrotizing pancreatitis, concluded that of Ranson, Imrie, Multiple organ failure (MOF) and Sepsis sensitivity score (SSS), APACHEII is the best for grading the severity of disease on admission. It is well suited forstratification of patients and comparisons of treatment methods.

The advantages are:

- Objective determination of AP within few hours of admission, which might helpful in detection of cases for ICU care or for clinical trial.
- Use of routine laboratory tests available 24 hours a day.
- Ability to be recalculated daily.Sequential monitoring of APACHE IIenables determination of improvement or deterioration in the physiologicstatus of the patient. Over the initial 48 h, the score increase significantly inthose with severe disease (median increase three points) but decrease(median decrease one point) in patients with mild pancreatitis. Thus thismight be useful for follow up of the disease course and helps to assess thetherapeuticresponse.
- This score was used universally for all serious illnesses, thereby avoidingthe need for a separate grading for acute pancreatitis

Beginning : Date ---------- Time ---------- APACHI II patients study number Patients initial									
Acute Physiology and Chronic health evaluation									
A: Acute physiology score (12 variables)	High abnormal rage					Low abnormal range			
Physiological Variables	+4	+3	+2	+1	0	+1	+2	+3	+4
Temperature – rectal (°C)	≥41	39-40.9		38.5–38.9	36–38.4	34–35.9	32–33.9	30–31.9	≤29.0
Mean arterial pressure (mm Hg)	≥160	130–159	110–129		70–109		50–69		≤49
Heart rate-ventricular response	≥180	140–179	110–139		70–109		55–69	40–54	≤39
Respiratory rate non ventilated or ventilated	≥50	35–49		25–34	12–24	10–11	6–9		≤5
Oxygen: A – a DO or PaO_2 (mm Hg) $FiO^2 \geq 0.5$ record A – aDO2 $FiO_2 < 0.5$ record only PaO_2	≥500	350-499	200–349		<200 PO_2>70	$PO_2$61–70		$PO_2$55–60	PO_2<55
Arterial pH	≥7.7	7.6–7.69		7.5-7.59	7.33–7.49		7.25–7.32	7.15–7.24	<7.15
Serum HCO_3 – only if no ABGs	≥52	41.5–1.9		32–40.9	23–31.9		18–21.9	15–17.9	<15
Serum sodium (mmol/l)	180	160–179	155–159	50–154	130–149		120–129	111–119	≤110
Serum potassium (mmol/l)	≥7	6–6.9		5.5–5.9	3.5–5.4	3–3.4	2.5–2.9		<2.5
Serum creatinine (umol/l)	≥350	200–340	150–190		60–140		<60		
Haematocrt (%)	≥60		50–50.9	46–49.9	30–45.9		20–29.9		<20
White Blood cell court (x1000 /mm^3)	≥40		20–39.9	15–19.9	30–14.9		1–2.9		<1
Glasgow Coma Score (GCS)	Score = 15 minus actual GCS								

[Table/Fig-1]: The APACHE II chart for scoring

B. Age points

Age years	Points	History	Points for elective surgery	Points for emergency surgery and non-operative patients	Apache II score: sum of A + B + C
≥ 44	0	Liver: Biopsy proven cirrhosis and documented porta hypertension or prior episodes of hepatic failure	2	5	A: APS score
45–54	2	Cardiovascular NYHA class IV	2	5	B: Age Points score
55–64	3	Respiratory eg. Severe COPD, hypercapnia, home O2 pulmonary hypertension	2	5	C: Chronic health points score
65–74	5	Renal chronic dialysis	2	5	
≥ 75	6	Immunocompromised	2	5	Total apache II

[Table/Fig-2]: The APACHE II chart for scoring

8) BISAP (**The bedside index for severity in AP):**

The BISAP score includes:

- Blood urea nitrogen (BUN) >25 mg / dl.
- Impaired mental status (GCS < 15).
- SIRS.
- Age >60 years.
- Pleural effusion.

SIRS was defined by presence of two or more of the following criteria:

- Pulse rate > 90/min.
- Respiratory rate > 20/min or PaCO 2 < 32 mm Hg.
- Temperature >100.4 F or < 96.8 F / < 36 or > 38 ° C.
- WBC count >12,000 or < 4,000 cells/mm3, or presence of more than10% immature blasts.

(SIRS - Systemic Inflammatory Response Syndrome) One point will be given for eachvariable present for a total of 5, score ranges from0 to 5.The presence of a pleural effusion was determined by a CT scan, chestradiograph or abdominal ultrasound obtained within 24 of presentation.

A BISAP score of three or more has been found to have high mortality and havepredicted the necrosis and organ failure as well.

ADVANTAGES:

- Simple and easy to calculate, usually done at the time of admission or within24 hrs. Of hospitalization.
- The scores prediction ability was tested across 390 hospitals among largenumber (36,248) of populations, in contrast to other studies which werebased on small number patients.
- This predicts in-hospital mortality

DISADVANTAGES:

- The Glasgow Coma Scale used for evaluating mental status was subject toInterobserver variation.
- It could not discriminate transient from persistent organ failure within 24hrs. Of hospitalization.
- This could not predict the preventable complications of acute pancreatitislike any other scoring system.

OBSERVATION AND RESULTS

TABLE:1 AGE DISTRIBUTION

	Frequency	Percent
Upto 30 yrs	11	11.0
31 - 40 yrs	39	39.0
41 - 50 yrs	31	31.0
51 - 60 yrs	19	19.0
Total	100	100.0

The following figure illustrates the age distribution of the participants with mean age of 42.

FIGURE-1: Age distribution

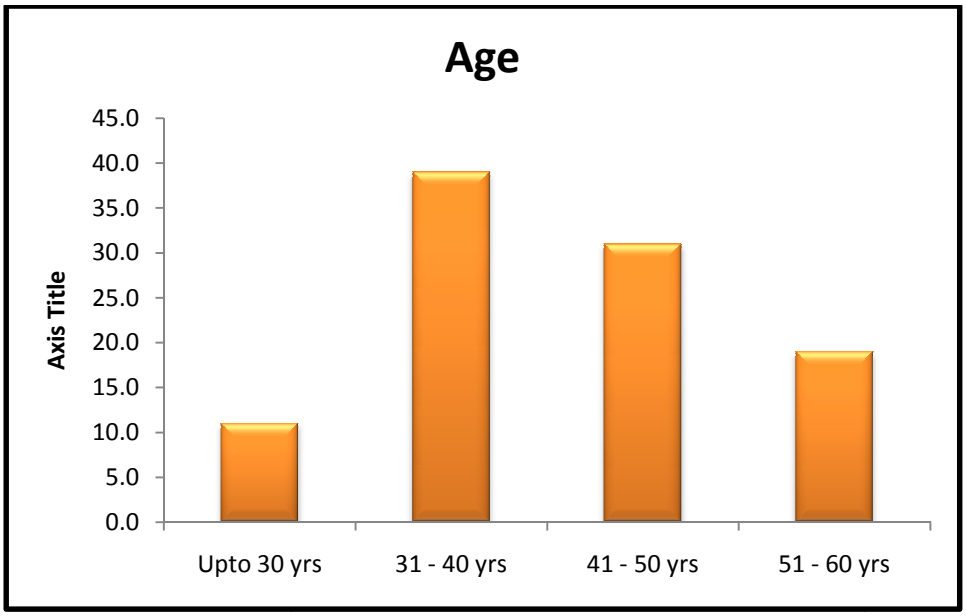


TABLE:2 GENDER DISTRIBUTION

	Frequency	Percent
Female	7	7.0
Male	93	93.0
Total	100	100.0

Majority of them were Males, the following figure illustrates the gender distribution of the sample.

FIGURE-2

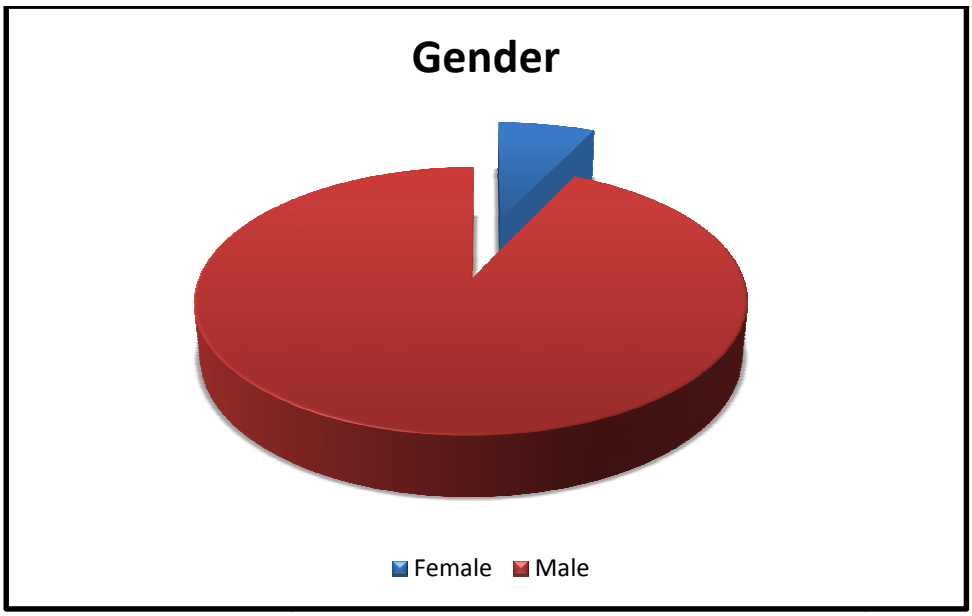


TABLE:3 CLINICAL FEATURES: ABD.PAIN

	Frequency	Percent
Present	100	100.0

	Abd.dist	Vomiting	Fever	Jaundice
Present	59	61	28	3
Absent	41	39	72	97

Abdominal pain, abdominal distention and vomiting were the common presentation in acute pancreatitis in this study. The Following figure illustrates the clinical features of the sample.

FIGURE-3

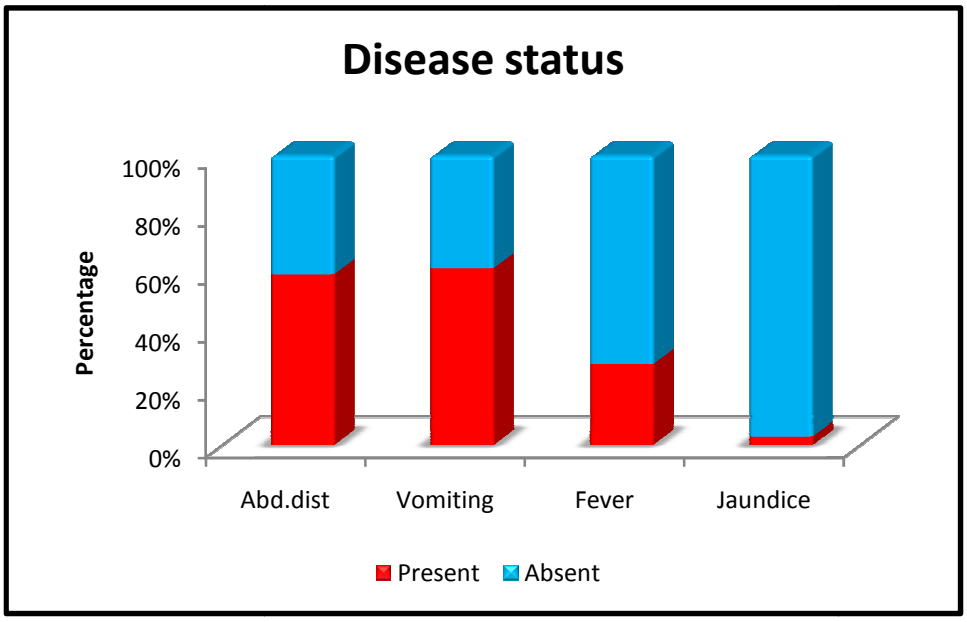


TABLE:4 APACHE II SCORE

	Frequency	Percent
SAP≥9	26	26.0
MAP≤9	74	74.0
Total	100	100.0

Based on APACHE II score 26 patients had severe acute pancreatitis of this sample.

TABLE:5 BISAP SCORE

	Frequency	Percent
SAP≥3	32	32.0
MAP≤2	68	68.0
Total	100	100.0

Based on BISAP score 32 patients had severe acute pancreatitis in this study.

TABLE:6 ATLANTA CLASSIFICATION

	Frequency	Percent
SAP	24	24.0
MAP	76	76.0
Total	**100**	**100.0**

Based on Atlanta classification 24 patients had severe acute pancreatitis in this study.

FIGURE-4 Outcomes of the sample.

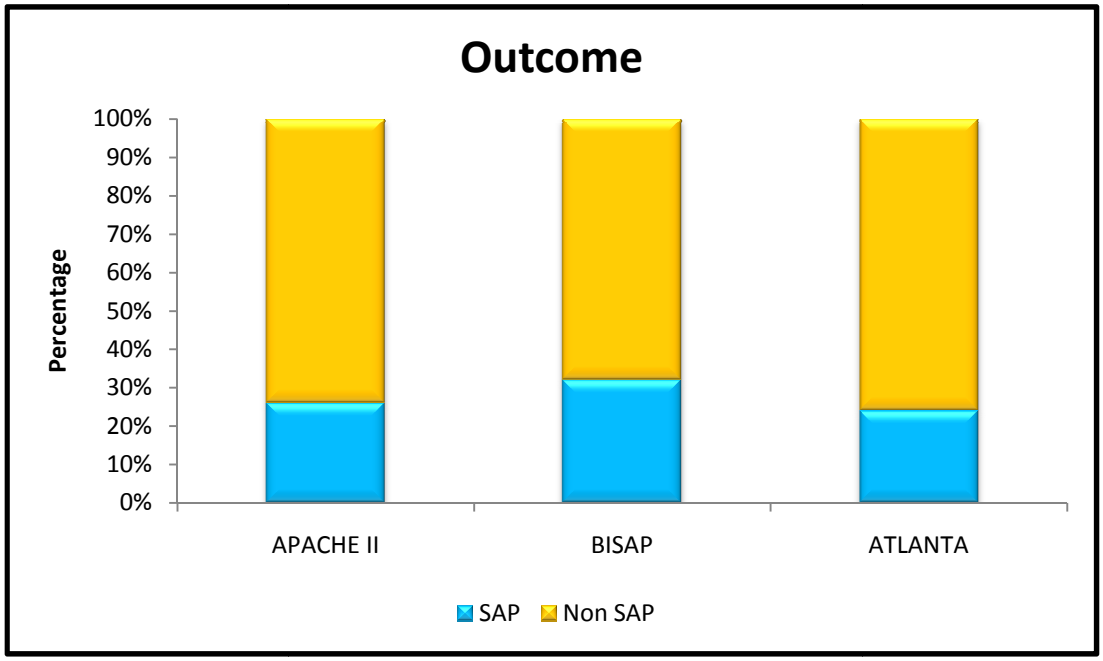


TABLE-7: ETIOLOGY

	Frequency	Percent
ALCOHOL	72	72.0
GSD	15	15.0
IDIOPATHIC	8	8.0
HYPERTRIGLYCERIDEMIA	3	3.0
TRAUMA	2	2.0
Total	100	100.0

Alcohol is the most common etiology factor in acute pancreatitis of the sample.

FIGURE-5 : Etiology in acute pancreatitis of the sample.

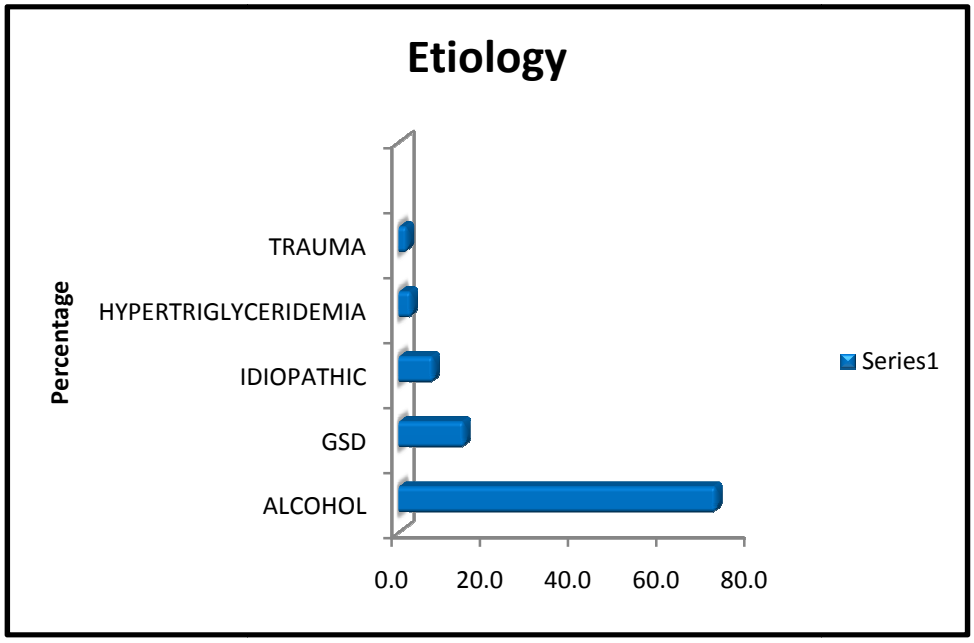


TABLE:8 COMPLICATIONS

	Frequency	Percent
MODS/SEPSIS	5	5.0
ARF	3	3.0
ARF/RF	3	3.0
HYPOCALCEMIA	1	1.0
PANCREATIC FISTULA WITH COLLECTION	2	2.0
PSEUDOCYST	4	4.0
PVT	1	1.0
NIL	86	86.0
Total	100	100.0

In this study 5 patients had multi organ dysfunction syndrome, 3 patients had acute renal failure, 3 patients had respiratory failure, 2 had peripancreatic collection with fistula, 4 patients had pseudocyst and 1 patient had portal vein thrombosis and 1 patient had Hypocalcemia.

FIGURE-6: COMPLICATIONS OF THE SAMPLE.

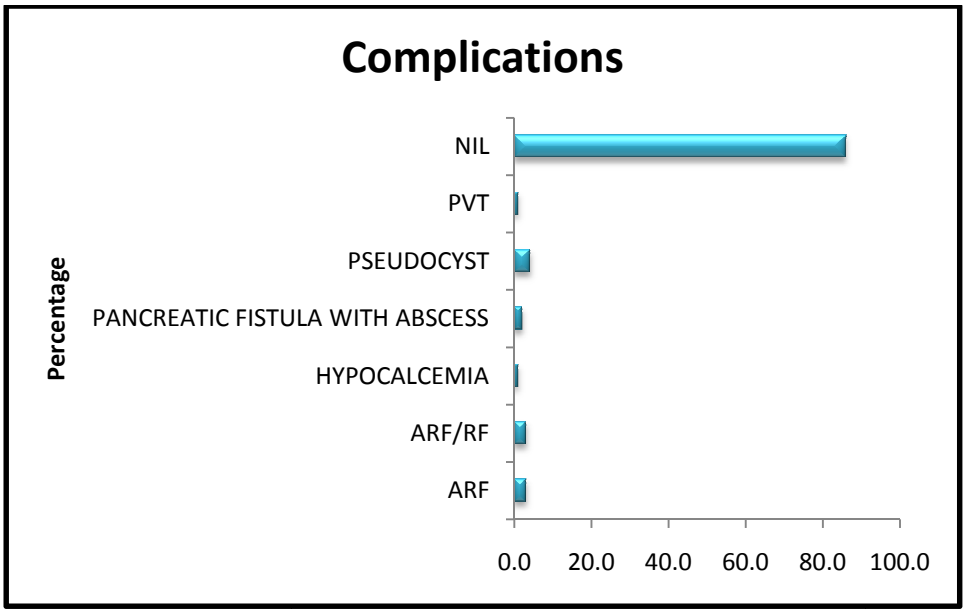


TABLE:9 SAMPLE OUTCOME

	Frequency	Percent
ALIVE	95	95.0
DEATH	5	5.0
Total	100	100.0

95 patients of the sample Alive and 5 patients were expired.In this study mortality rate 5%.

FIGURE-7: OUTCOME OF THE SAMPLE

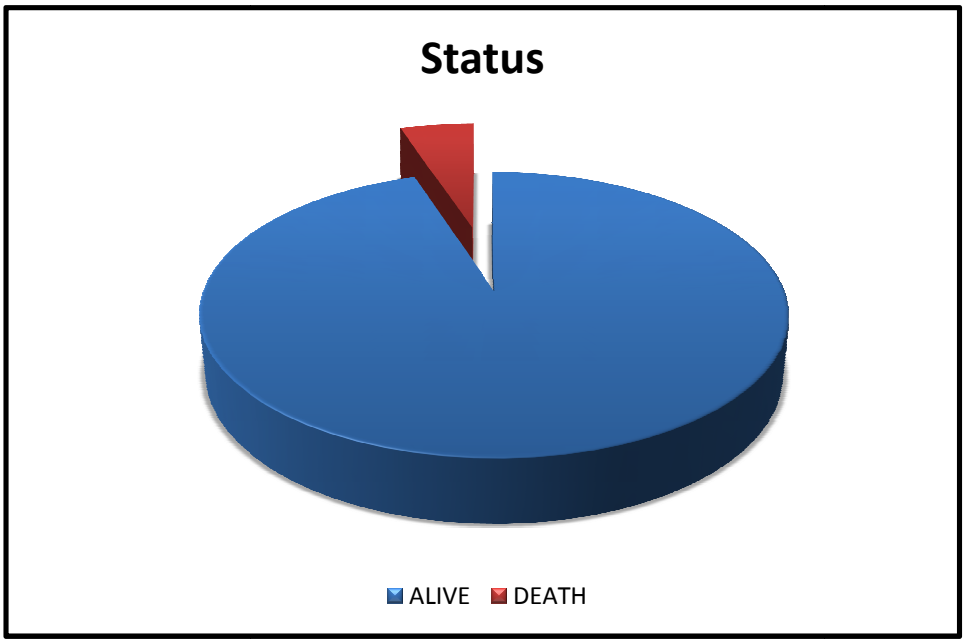


TABLE:10 AUC BASED ON SEVERITY

Area Under the Curve Based on SEVERITY				
Test Result Variable(s)	Area	P-value	95 % C.I	
			LB	UB
APACHE II SCORE	.822	0.0005 **	.713	.932
BISAP SCORE	.947	0.0005 **	.905	.990
** Highly Significant at P < 0.01 level				

TABLE:11 RESULTS OF SCORING SYSTEMS BASED ON ATLANTA CLASSIFICATION.

APACHE II SCORE with ATLANTA						
		ATLANTA		Total		
		SAP	MAP			
APACHE II	SAP	18	8	26	Sensitivity	75.00
	MAP	6	68	74	Specificity	89.47
Total		24	76	100	PPV	69.23
					NPV	91.89
BISAP SCORE with ATLANTA					Accuracy	82.24
		ATLANTA		Total		
		SAP	MAP			
BISAP	SAP	24	8	32	Sensitivity	100.00
	MAP	0	68	68	Specificity	89.47
Total		24	76	100	PPV	75.00
					NPV	100.00
					Accuracy	94.74

TABLE:12 AUC OF SCORING SYSTEMS BASED ON THE OUTCOME OF THE SAMPLE.

Area Under the Curve for Mortality				
Test Result Variable(s)	Area	P-value	95 % C.I	
			LB	UB
APACHE II	.679	0.178 #	.422	.936
BISAP	.858	0.007 **	.766	.950
** Highly Sig at P< 0.01 and # No Sig at P >0.05				

TABLE:13 RESULTS OF SCORING SYSTEMS WITH OUTCOME

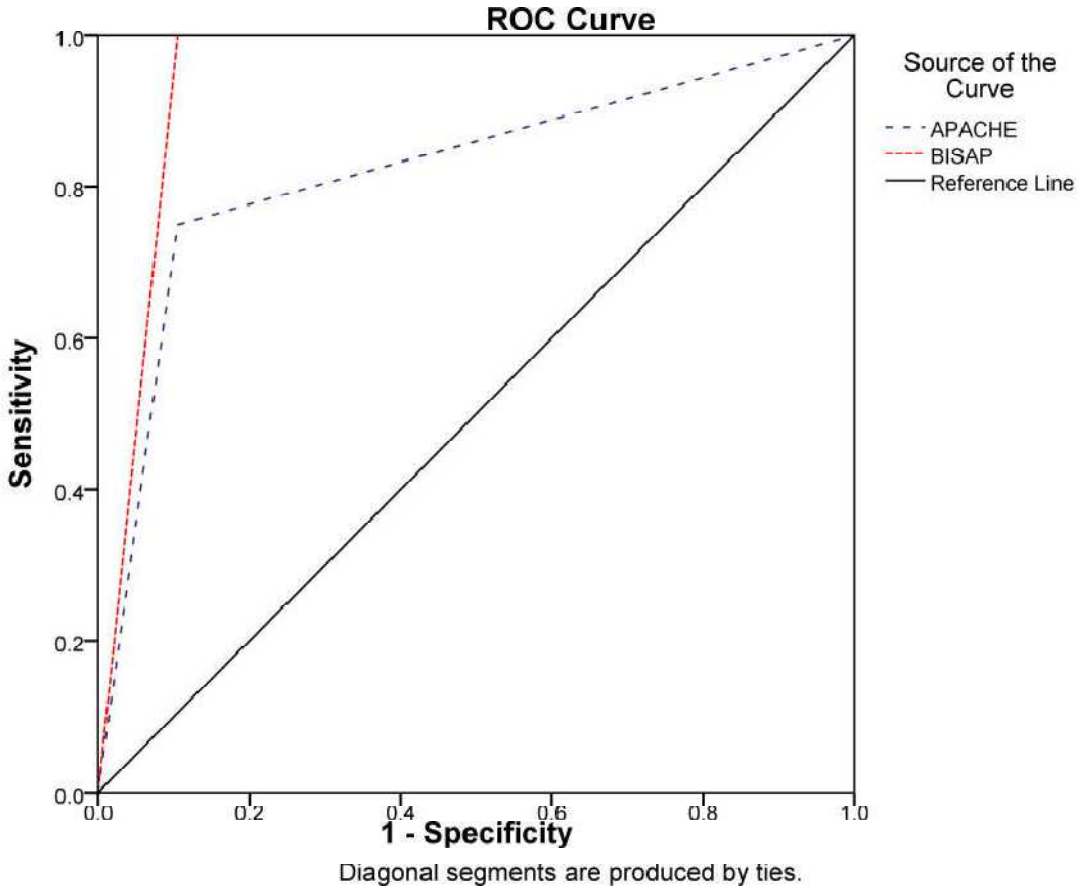
APACHE II SCORE with OUTCOME						
		OUTCOME		Total		
		DEATH	ALIVE			
APACHE II	SAP	3	23	26	Sensitivity	60.00
	MAP	2	72	74	Specificity	75.79
Total		5	95	100	PPV	11.54
					NPV	97.30
					Accuracy	67.89
BISAP SCORE with OUTCOME						
		OUTCOME		Total		
		DEATH	ALIVE			
BISAP SCORE	SAP	5	27	32	Sensitivity	100.00
	MAP	0	68	68	Specificity	71.58
Total		5	95	100	PPV	15.63
					NPV	100.00
					Accuracy	85.79

TABLE: 14 MEAN AGE AND LOH OF THE SAMPLE

Descriptive Statistics					
	N	Minimum	Maximum	Mean	S.D
AGE	100	25	60	42	9
LOH	100	2	24	11	5

Mean age was 42 and Mean Duration of Hospital Stay of this study was 11.

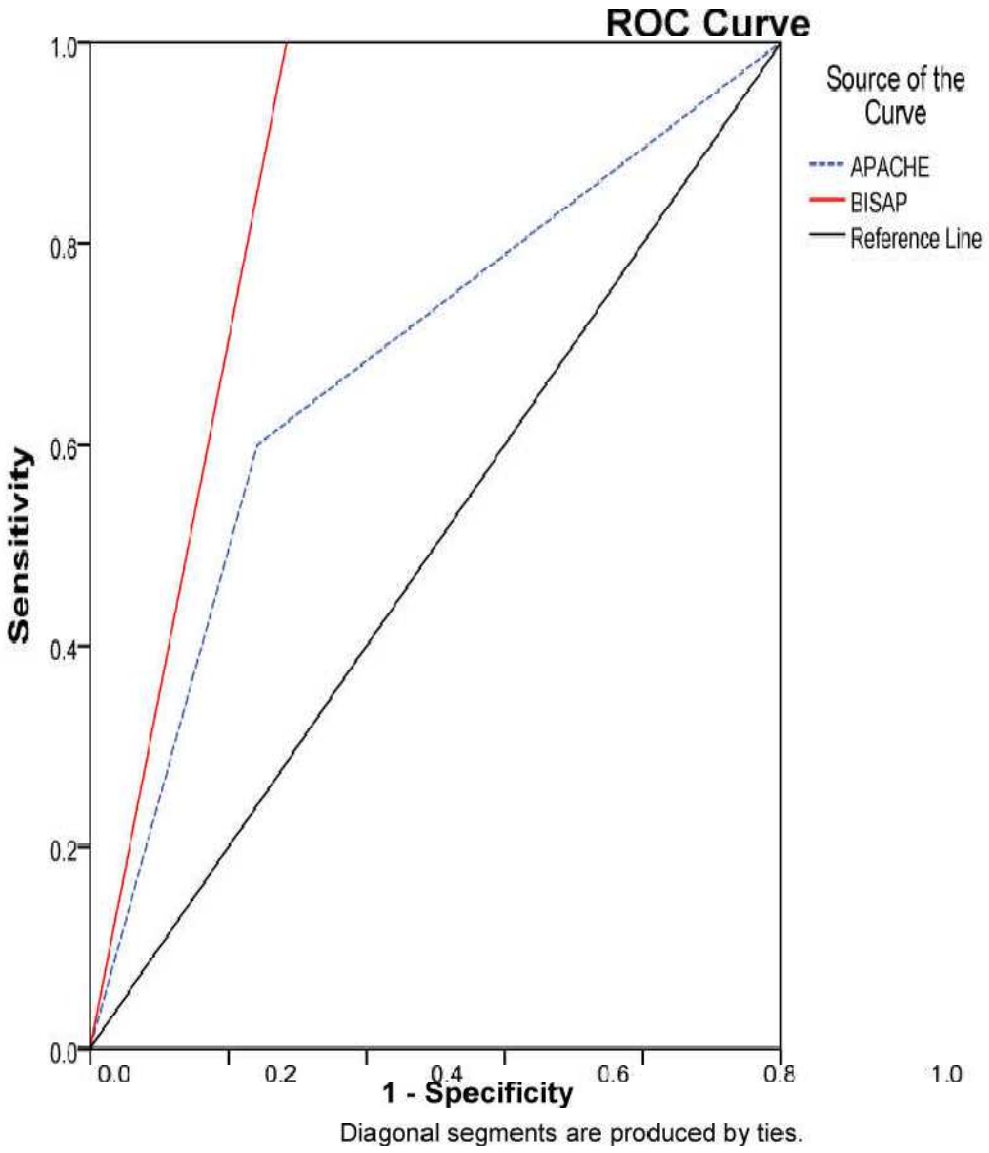
FIGURE-8: ROC CURVE ANALYSIS FOR SEVERITY IN ACUTE PANCREATITIS



AUC (Area Under Curve) for BISAP score 0.947

AUC for APACHE II score 0.822

FIGURE-9: ROC CURVE ANALYSIS FOR MORTALITY IN ACUTE PANCREATITIS



AUC (Area Under Curve) for BISAP score 0.858

AUC for APACHE II score 0.679

DISCUSSION

Severe acute pancreatitis having high morbidity and mortality rate. Early admission andintervention at time can provide favour results. In this study two clinical scoring systems were compared and analysed for their efficacy in predicting the severity and mortality of acutepancreatitis.

In this study acute pancreatitis more common in males. Male female ratio in our study is13.3:1.which didn't match with early study results, Vikesh K. Singh et al38 (6:1),Papachristou et al1 (5.1:1). Because alcohol is the most common etiological factor and it'smore common in males.

In this study, the mean age was 42 years which matches with the study ofSarath et al (40.8yrs), nearly matches with Vikesh K. Singh et al (49.6 yrs),Papachristou et al1 (51.7 yrs).

Risk of mortality also increased with age. Thus age is considered as a contributory factor toassess the outcome of severe acute pancreatitis.The most common etiological factor in thisstudy was alcohol (72%) which didn't correlate with results of with Bidarkundietal43(46.67%), Vikesh K. Singh et al38 (21.4%) andPapachristou et al1 (14%) wherein gallstonedisease was the most common cause.

In this study the mean length of hospital stay was 11 ± 5 days.

The most common presentation was predominantly abdominal pain (100%),followed by vomiting (60%), abdominal distention(59%)& other manifestations.In this study, 76 patients were diagnosed to have mild acute pancreatitis and24 patients found to have severe acute pancreatitis. All the 24 patients werecorrectly predicted by BISAP Score. The severity was assessed by correlating with Atlanta classification and mortality.

The ROC analysis to assess the severity in AP by BISAP score has AUC of 0.947, sensitivity 100%, specificity of 89.47%, PPV of 75%, NPV 100% and diagnostic accuracy of 94.74%;whereas APACHE II score has AUC 0.822, sensitivity of 75%, specificity of 89.47%, PPVof 69.23%, NPV of 91.89% and diagnostic accuracy of 82.24%.

This correlates well with the study by Papachristouet al1 where AUC (0.81, 0.78), specificity (92.4%, 71.9%), PPV (57.7%, 40%) and NPV (84.3%, 90.1%), forBISAP and APACHE II scores, respectively.

In this study, 6% underwent surgical intervention which comparable withSarath et al. In this study, 5 patients with severe acute pancreatitis were expired. All FIVE deaths were correctlypredicted by BISAP score. All five patients were expired due to MODS with septicemia.

The ROC analysis for prediction of mortality by BISAP and APACHE II score hasrespectively AUC (0.858,0.679), sensitivity (100%, 60%), specificity (71.58%, 75.79%), PPV (15.63%, 11.54%), NPV (100%, 97.30%)

and diagnostic accuracy (85.79%, 67.89%),This matches well with B U Wu et al41, Papachristou et al1, where specificity (87.6%,65.7%),PPV (15.4%, 10.8%), NPV (98.1%, 100%), for BISAP and APACHE II scores, respectively.

Thus by using Chi2 test, BISAP $\geq$ 3 was found to be significantly associated ($p < 0.007$) withhigh mortality than APACHE II score by ROC. It was found to have high sensitivity, PPVand NPV for mortality. This again matches well with previous study by Vikesh k. Singh etal38 and Papachristouet al1.

In this study, 3% patients developed acute renal failure, 5% patients developed MODS, 5%patients developed septicemia and 11% developed other complications like ARDS, UI bleed,Hypocalcemia etc. Remaining 86% of them not developed any complications. Thesecomplications were more likely seen in patients with BISAP $\geq$ 3 and APACHE $\geq$ 9 henceconcluded that these are the patients in high risk group, who requires intensive monitoringand probably early intervention if necessary.

BISAP score was found to have more sensitivity, specificity, positive and negative value, anddiagnostic accuracy than APACHE II score in predicting the severity of acute pancreatitis.Hence, BISAP is considered as better score in assessing theseverity than APACHE II score.

Limitations of this study are:

- No of patients in this study was less.
- Alcohol is the most common aetiology in this study which is different from worldwide accepted one.
- In the GCS score assessment may have inter-observer variation.
- Various factors associated with the disease may affect the physiological scoring assessments.
- Patient presenting timing variations may affect the scoring system.

CONCLUSION

- From this study, Alcohol (72%) was found to be the most common
- Aetiological factor for acute pancreatitis.
- Males were most commonly affected than female with a ratio of 13:1.
- The most common age groups of patients affected were in 4th decade of life.
- The overall mortality in patients with severe acute pancreatitis was 5%.
- The BISAP score predicted the mortality significantly over the APACHE II score in patients with severe acute pancreatitis.
- The BISAP score predicted the disease severity significantly over the APACHE II score in patients with acute pancreatitis.

From this study, we conclude that the BISAP score could be a simple andaccurate clinical scoring system for the evaluation of disease severity in acutepancreatitis.

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GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL,CHENNAI -01

INSTITUTIONAL ETHICS COMMITTEE

TITLE OF THE WORK : A COMPARATIVE STUDY BETWEEN BISAP SCORE AND APACHE II SCORE IN ASSESSING THE SEVERITY OF ACUTE PANCREATITIS BASED ON THE REVISED ATLANTA CLASSIFICATION.

PRINCIPAL INVESTIGATOR : DR. V. NEDUNCHEZHIAN,
DESIGNATION : PG IN MS GENERAL SURGERY
DEPARTMENT : DEPARTMENT OF GENERAL SURGERY,
GOVT. STANLEY MEDICAL COLLEGE.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 07.12.2018 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI

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482c4a7e-9033-4cf8-99da-26d0ac05ee98
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PLAGIARISM CERTIFICATE

This is to certify that this dissertation work titled **"A COMPARATIVE STUDY BETWEEN BISAP SCORE AND APACHE II SCORE IN ASSESSING THE SEVERITY OF ACUTE PANCREATITIS BASED ON THE REVISED ATLANTA CLASSIFICATION"** of the candidate **Dr. V. NEDUNCHEZHIAN** with **Registration Number 221711060** for the award of **M. S. Degree in the BRANCH-1 GENERAL SURGERY.** I Personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from Introduction to Conclusion pages and Results shows 1 percentage of plagiarism in the Dissertation.

GUIDE and SUPERVISOR SIGN WITH SEAL

PROFORMA

A COMPARATIVE STUDY BETWEEN BEDSIDE INDEX OF SEVERITY IN ACUTE PANCREATITIS (BISAP) SCORE AND ACUTE PHYSIOLOGICAL AGE AND CHRONIC HEALTH EVALUATION (APACHE II) SCORE IN ASSESSING THE SEVERITY OF ACUTE PANCREATITIS BASED ON THE REVISED ATLANTA CLASSIFICATION

- NAME :
- AGE/SEX :
- IP.NO :
- Date of admission:
- Date of surgery if any:
- Date of discharge:
- Address:

History of presenting illness

Past history

Personal history

Family history

GCS

VITALS

SYSTEMIC EXMINATION

ABDOMINAL EXAMINATION

DIAGNOSIS

BLOOD INVESTIGATIONS

Complete hemogram,

RFT,

LFT,

SERUM ELECTROLYTES,

Arterial Blood Gas (ABG) analysis:

serum amylase/lipase

CHEST XRAY,

ABDOMINAL XRAY

ECG,

USG ABDOMEN and CHEST

CECT ABDOMEN

- ETIOLOGY(gall stone disease/alcoholic/ideopathic):
- BISAP SCORING SYSTEM (0-5)

1) BUN(>25MG/DL)
2) IMPAIRED MENTAL STATUS(GCS SCORE<15)
3) SIRS

SIRS IS defined as two or more of the following

a.Temperature of <36 or > 38 degree Celsius.

b. Respiratory rate >20 breath/min or paco2<32mmHg.

c.Pulse>90 beats/min.

d.Wbc< 4000 or >12000 cells/mm3 or more than 10% immature blasts.

4)Age>60 yrs

5)Imaging study reveals pleural effusion

APACHE II SCORING SYSTEM(0-71)

ACUTE PHYSIOLOGICAL SCORE(0-60)
1)Body temperature rectal (*C)
2)MAP(mmhg)
3)HR(/min)
4)RR(/min)
5)oxygenation(mmhg)
6)Arterialph
7)NA+(mmol/l)
8)K+(mmol/l)
9)CREATININE(mg/dl)
10)Haematocrit(%)
11)total leucocyte count(in 1000/mm3)
12)glasgow coma scale
13)serum HCO3(IF NO ABG)

14)AGE(0-6)
<44years
45-54yrs
55-64yrs
65-74yrs
>75yrs

15)CHRONIC HEALTH POINTS(0-5)
a- For non operative or emergency postoperative patients
b-For elective post operative patients

According to revised ATLANTA CLASSIFICATION 2012

GRADES OF SEVERITY IN ACUTE PANCREATITIS

*MILD ACUTE PANCREATITIS

No organ failure

No local or systemic complications

*SEVERE ACUTE PANCREATITIS

Persisent organ failure >48hrs

With Local Complications

CONDITION ON DISCHARGE:

GOVT.STANLEY MEDICAL COLLEGE, CHENNAI- 600 001

INFORMED CONSENT

COMPARISON OF BISAP SCORE AND APACHEII SCORE IN ASSESSING SEVERITY OF ACUTE PANCREATITIS -SMC

- PLACE OF STUDY: GOVT. STANLEY MEDICAL COLLEGE, CHENNAI
- NAME AND ADDRESS OF PATIENT:
- I, _____ have been informed about the details of the study in my own language.
- I have completely understood the details of the study.
- I am aware of the possible risks and benefits, while taking part in the study.
- I understand that I can withdraw from the study at any point of time and even then, I will continue to receive the medical treatment as usual.
- I understand that I will not get any payment for taking part in this study.
- I will not object if the results of this study are getting published in any medical journal, provided my personal identity is not revealed.
- I know what I am supposed to do by taking part in this study and I assure that I would extend my full co-operation for this study.

Name and Address of the Participant:

Signature/Thumb impression of the Volunteer

Date:

Witnesses:

(Signature, Name & Address)

Date:

Name and signature of investigator

NO.	NAME	AGE	SEX	IP NO	AGE	TEMP	HR	RR	OXYG	MAP	Na+	K+	PH	CREATIN	PCV	WBC	GCS	Chronic	APACHE	BUN	GCS	SIRS	AGE	PE	BISAP	OUTCOME	MANAGEMENT	ORG.DYSFUNCTIO	NECROSIS	LOH	ETIOLOGY	ABD.PAIN	ABD.DIST	VOMITI	FEVER	JAUNDIC	COMPLICATI	ATLANTA
1	Baskaran	55	male	1839078	3	0	2	1	1	2	1	1	1	2	1	1	0	0	16	0	0	1	0	1	2	ALIVE	CONSERVATIVE	-	-	11	ALCOHOL	+	+	+	-	-		
2	Mariyappan	47	male	1839137	2	0	0	0	0	0	0	1	0	2	1	1	0	0	7	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	9	ALCOHOL	+	+	-	-	-		
3	Ramesh	35	male	1839574	0	0	0	0	0	2	0	0	0	0	1	1	0	0	4	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	5	IDIOPATHIC	+	-	-	-	-		
4	Kaveri	47	female	1837700	2	0	0	0	0	0	0	1	0	0	1	1	0	0	5	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	8	GSD	+	-	+	-	+		
5	rajendiran	57	male	1837489	3	0	2	1	2	2	0	1	1	2	1	2	0	0	17	1	0	1	0	0	2	ALIVE	CONSERVATIVE	-	-	9	ALCOHOL	+	+	+	-	-	PSEUDOCYST	
6	Navab	40	male	1837665	0	1	2	1	0	2	0	1	1	2	1	1	0	0	12	1	0	1	0	1	3	ALIVE	SURGERY	-	+	14	ALCOHOL	+	+	+	+	-		SAP
7	murugan	41	male	1837767	0	0	2	1	0	2	0	1	0	0	1	1	0	0	8	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	8	ALCOHOL	+	-	-	-	-		
8	Sundaram	47	Male	1842986	2	1	2	1	1	0	1	1	1	2	1	1	0	0	14	1	0	1	0	0	2	ALIVE	CONSERVATIVE	-	-	7	ALCOHOL	+	-	-	-	-		
9	Murugesan	58	Male	1840385	3	3	2	3	1	2	2	2	2	2	1	1	4	5	33	1	1	1	0	1	4	DEATH		SEPSIS/MODS	+	3	ALCOHOL	+	+	+	+	-	ARF/RF	SAP
10	Babu	47	Male	1842376	2	0	0	0	0	0	1	1	0	0	1	1	0	0	6	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	5	ALCOHOL	+	-	-	-	-		
11	Sekar	35	Male	1845129	0	0	0	0	0	0	0	0	0	0	1	1	0	0	2	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	5	TRAUMA	+	-	-	-	-		
12	Ponnambalam	50	Male	1847229	2	1	2	2	1	2	0	1	1	2	1	0	1	0	16	1	1	1	0	1	4	ALIVE	SURGERY	-	+	15	ALCOHOL	+	+	+	+	-	PANCREATIC	SAP
13	Kuppaiyan	50	Male	1847503	2	1	0	0	0	2	0	1	0	2	1	1	1	0	11	1	1	1	0	1	4	ALIVE	SURGERY	-	+	21	ALCOHOL	+	+	+	+	-	PANCREATIC	SAP
14	venkatesh	58	Male	1844477	3	0	0	0	0	0	0	0	0	0	1	0	0	0	4	1	0	1	0	0	2	ALIVE	CONSERVATIVE	-	-	18	IDIOPATHIC	+	+	+	-	-		
15	Kumar	56	Male	1848481	3	0	0	0	0	2	0	1	1	0	0	0	0	0	7	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	14	ALCOHOL	+	+	+	-	-		
16	venkatesan	34	Male	1843647	0	1	2	1	1	2	0	0	1	0	1	1	0	0	10	1	0	1	0	1	3	ALIVE	CONSERVATIVE	-	+	12	ALCOHOL	+	-	+	+	-		SAP
17	Sanjay	35	Male	1844417	0	1	2	0	0	0	0	0	0	0	0	0	0	0	3	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	11	ALCOHOL	+	-	+	+	-		
18	Veerabathran	50	Male	1850170	2	0	0	0	0	0	0	1	0	2	1	0	0	0	6	1	0	1	0	0	2	ALIVE	CONSERVATIVE	-	-	8	ALCOHOL	+	-	+	-	-		
19	Kuppammal	46	Female	1848150	2	0	1	0	0	0	0	0	0	0	0	0	0	0	3	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	9	GSD	+	+	-	-	-		
20	Ravichandiran	54	Male	1851308	2	0	0	0	0	0	0	0	0	0	1	0	0	0	3	1	0	1	0	0	2	ALIVE	CONSERVATIVE	-	-	6	ALCOHOL	+	-	+	-	-		
21	Suresh	36	Male	1850204	0	0	0	0	0	2	0	1	0	0	1	1	0	0	5	1	0	1	0	0	2	ALIVE	CONSERVATIVE	-	-	5	ALCOHOL	+	-	+	-	-		
22	Arokyaxavior	29	Male	1849484	0	1	2	1	1	2	0	0	0	0	1	1	0	0	7	1	0	1	0	1	3	ALIVE	CONSERVATIVE	-	+	7	IDIOPATHIC	+	-	+	+	-		SAP
23	Sakthivel	35	Male	1851178	0	1	0	0	0	0	0	0	0	0	1	0	0	0	2	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	9	IDIOPATHIC	+	-	-	+	-		
24	Akbarbabu	38	Male	1851732	0	0	0	0	0	0	0	0	0	0	1	1	0	0	2	1	0	1	0	0	2	ALIVE	CONSERVATIVE	-	-	7	ALCOHOL	+	-	-	-	-		
25	Jayaraman	60	Male	1853523	3	0	2	1	0	0	0	0	0	0	1	0	0	0	7	1	0	1	1	0	3	ALIVE	CONSERVATIVE	-	-	9	ALCOHOL	+	+	-	-	-	PSEUDOCYST	
26	Janakiraman	44	Male	1853435	0	0	2	1	0	0	0	0	0	0	1	0	0	0	4	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	16	ALCOHOL	+	+	+	-	-		
27	Dillibabu	35	Male	1851324	0	0	0	1	0	0	0	1	1	2	2	0	0	0	7	1	0	1	0	0	2	ALIVE	CONSERVATIVE	-	-	15	ALCOHOL	+	+	+	-	-		
28	Santhosh	27	Male	1853550	0	0	2	1	0	0	0	0	0	0	1	0	0	0	4	0	0	1	0	1	2	ALIVE	CONSERVATIVE	-	-	14	TRAUMA	+	+	+	-	-		
29	Chandran	44	Male	1852056	0	0	0	1	1	1	0	1	0	0	1	0	0	0	5	0	0	1	0	1	2	ALIVE	CONSERVATIVE	-	-	14	ALCOHOL	+	+	-	-	-		
30	Ramachandran	60	Male	1855355	3	0	2	1	0	0	0	0	0	2	1	0	0	0	9	0	0	1	1	1	3	ALIVE	CONSERVATIVE	-	-	16	ALCOHOL	+	+	+	-	-		
31	Vimalkumar	30	Male	1850762	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	5	HYPERTRIGLYCER	+	-	-	-	-		
32	Manikkam	48	Male	1851540	2	1	2	1	2	2	1	1	1	2	2	1	0	0	18	1	0	1	0	1	3	ALIVE	CONSERVATIVE	-	+	15	ALCOHOL	+	-	+	+	-		SAP
33	Parthiban	32	Male	1862657	0	1	2	1	2	2	0	1	2	2	1	1	0	0	15	1	0	1	0	1	3	ALIVE	CONSERVATIVE	AKI/PVT	+	24	ALCOHOL	+	+	+	+	-	PVT	SAP
34	Gopi	28	Male	1861935	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	0	0	0	0	ALIVE	CONSERVATIVE	-	-	5	IDIOPATHIC	+	-	-	-	-		
35	Dhanalakshmi	38	Female	1861976	0	0	2	0	0	0	0	0	0	1	0	1	0	0	4	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	7	GSD	+	+	+	-	-		
36	Boopathy	53	Male	1865728	2	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	7	ALCOHOL	+	-	+	-	-		
37	Radha	55	Female	1865830	3	0	2	1	2	2	0	1	1	0	1	0	0	0	13	0	0	1	0	1	2	ALIVE	CONSERVATIVE	-	-	11	ALCOHOL	+	+	+	-	-		
38	Vasu	48	Male	1865790	2	1	2	1	1	0	0	1	0	2	1	1	0	5	17	1	0	1	0	1	3	ALIVE	CONSERVATIVE	-	+	15	GSD	+	-	+	+	-		SAP
39	Rajesh	30	Male	1866452	0	0	0	0	0	0	0	1	0	0	0	0	0	0	1	0	0	0	0	0	0	ALIVE	CONSERVATIVE	-	-	5	ALCOHOL	+	-	-	-	-		
40	Saravanan	44	Male	1868648	0	0	2	0	1	0	0	0	0	0	1	0	0	0	4	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	8	GSD	+	-	+	-	-		
41	Srinivasan	48	Male	1866822	2	0	2	1	0	0	0	0	1	0	1	1	0	0	8	1	0	1	0	1	3	ALIVE	CONSERVATIVE	-	-	14	ALCOHOL	+	+	+	-	-		
42	Gajendirababu	52	Male	1864652	2	0	2	1	2	2	0	1	1	2	2	1	0	0	16	1	0	1	0	1	3	ALIVE	CONSERVATIVE	-	-	21	ALCOHOL	+	+	+	-	-		
43	Murugan	45	Male	1868296	2	0	0	0	0	0	0	1	0	0	1	0	0	0	4	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	13	GSD	+	-	+	-	+		
44	Ekambaram	51	Male	1872382	2	0	0	0	0	0	0	0	0	0	1	0	0	0	3	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	5	ALCOHOL	+	-	-	-	-		
45	Ramki	30	Male	1872686	0	0	2	0	1	0	1	1	0	0	0	0	0	0	5	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	20	ALCOHOL	+	+	+	-	-		
46	Akbar	38	Male	1876061	0	0	2	1	0	0	0	0	0	2	1	0	2	0	8	1	1	1	0	1	4	DEATH		SEPSIS/MODS	+	2	ALCOHOL	+	+	+	-	-	ARF	SAP
47	Balaji	32	Male	1879197	0	0	2	0	0	0	0	0	0	0	1	0	0	0	3	0	0	1	0	1	2	ALIVE	SURGERY	-	-	9	ALCOHOL	+	+	+	-	-		
48	Arichandran	44	Male	1879439	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	5	IDIOPATHIC	+	-	-	-	-		
49	Uthrapathi	43	Male	1876007	0	0	2	0	0	0	0	0	0	0	0	0	0	0	2	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	5	ALCOHOL	+	-	+	-	-		
50	Kalidhasan	46	Male	1878405	2	0	0	0	0	0	0	0	0	0	1	0	0	0	3	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	7	ALCOHOL	+	-	+	-	-		
51	Selvam	54	Male	1879311	2	0	2	0	1	2	0	0	0	0	1	1	0	0	9	0	0	1	0	1	2	ALIVE	CONSERVATIVE	-	-	19	ALCOHOL	+	+	+	-	-		
52	Sasikumar	36	Male	1880702	0	0	2	1	0	0	0	0	1	0	1	0	0	0	5	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	24	ALCOHOL	+	+	+	-	-		
53	Lalitha	38	Female	1880070	0	0	0	0	0	0	0	0	1	0	0	0	0	0	1	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	5	IDIOPATHIC	+	+	-	-	-		
54	Elangovan	35	Male	1885760	0	0	0	0	1	2	0	0	0	0	1	0	0	0	4	0	0	0	0	0	0	ALIVE	CONSERVATIVE	-	-	11	HYPERTRIGLYCER	+	+	+	-	-		
55	Solaiyappan	47	Male	1885126	2	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	7	ALCOHOL	+	+	+	-	-		
56	Saravanakumar	28	Male	1900144	0	1	2	1	1	2	0	1	1	0	1	1	0	0	11	1	0	1	0	1	3	ALIVE	SURGERY	AKI	+	12	ALCOHOL	+	+	+	+	-		SAP
57	Dharma	27	Male	1901211	0	1	2	0	2	2	0	0	1	0	1	0	0	0	9	1	0	1	0	1	3	ALIVE	CONSERVATIVE	-	+	10	ALCOHOL	+	+	-	+	-		SAP
58	Rajendiran	48	Male	1901406	2	0	0	0	0	0	0	0	0	0	1	0	0	0	3	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	7	GSD	+	-	+	-	-		
59	Natesan	55	Male	1889844	3	0	2	1	2	2	0	1	1	0	1	0	0	0	13	1	0	1	0	1	3	ALIVE	CONSERVATIVE	-	-	9	ALCOHOL	+	+	+	-	-		
60	Kamalakannan	47	Male	1900268	2	0	2	0	0	0	0	0	0	0	1	0	0	0	5	1	0	1	0	1	3	ALIVE	CONSERVATIVE	-	-	5	ALCOHOL	+	+	+	-	-		
61	Mani	40	Male	1900894	0	0	2	0	0	2	0	0	0	2	1	1	0	0	8	1	0	1	0	1	3	DEATH		SEPSIS/MODS	+	3	ALCOHOL	+	+	-	+	-	ARF	SAP

62	Sridhar	42	Male	1904342	0	1	0	1	0	2	0	0	1	0	1	1	0	0	7	1	0	1	0	1	3	ALIVE	CONSERVATIVE	-	+	9	ALCOHOL	+	+	+	+	-	PSEUDOCYST	SAP
63	Arul	31	Male	1904819	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	7	HYPERTRIGLYCER	+	-	+	-	-		
64	Khadhar moidhee	35	Male	1911331	0	0	0	0	1	0	0	0	0	0	1	0	0	0	2	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	5	ALCOHOL	+	+	-	-	-		
65	Sivaprakash	40	Male	1912879	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	7	ALCOHOL	+	+	-	-	-		
66	Johnprabakaran	44	Male	1913421	0	0	0	0	0	0	0	1	1	0	0	0	0	0	2	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	7	GSD	+	-	-	-	-		
67	Dineshkumar	28	Male	1913808	0	0	2	1	2	2	1	1	2	0	1	1	0	0	13	1	0	1	0	1	3	ALIVE	SURGERY	-	+	13	ALCOHOL	+	-	-	+	-	AKI/RF	SAP
68	Naresh	36	Male	1914638	0	0	0	0	0	0	0	0	1	0	1	0	0	0	2	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	9	ALCOHOL	+	-	-	-	-		
69	Desingh	58	Male	1914648	3	0	2	0	0	0	0	0	1	0	1	0	0	0	7	0	0	1	0	1	2	ALIVE	CONSERVATIVE	-	-	11	ALCOHOL	+	+	+	-	-		
70	Subramani	44	male	1914792	0	1	2	1	2	2	0	1	2	2	1	0	0	0	14	1	0	1	0	1	3	ALIVE	SURGERY	-	+	18	ALCOHOL	+	+	+	+	-		SAP
71	Parveen	45	Female	1916737	2	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	0	0	0	0	ALIVE	CONSERVATIVE	-	-	5	GSD	+	-	-	-	-		
72	Ravi	46	Male	1918953	2	1	0	0	0	0	0	1	1	0	1	0	0	5	11	1	0	1	0	1	3	ALIVE	CONSERVATIVE	-	+	13	GSD	+	-	-	+	-		SAP
73	Kamaraj	36	Male	1920252	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	15	ALCOHOL	+	-	+	-	-		
74	Ramesh	40	Male	1918048	0	0	2	0	0	0	0	0	1	0	1	0	0	0	4	1	0	1	0	0	2	ALIVE	CONSERVATIVE	-	-	22	ALCOHOL	+	+	+	-	-		
75	Sadaiyan	36	Male	1920146	0	0	0	0	1	0	0	1	1	0	1	0	0	0	4	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	17	ALCOHOL	+	+	-	-	-		
76	Krishnamoorthy	40	male	1920548	0	0	0	0	0	0	0	0	1	0	1	1	0	0	3	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	9	GSD	+	-	+	-	-		
77	Bharathi	36	Male	1920911	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	7	ALCOHOL	+	-	-	-	-		
78	Karthikeyan	34	Male	1920948	0	1	2	1	1	2	0	1	1	0	1	1	0	0	11	1	0	1	0	1	3	ALIVE	SURGERY	-	+	13	ALCOHOL	+	+	-	+	-		SAP
79	Anthony	42	Male	1920033	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	0	0	0	0	ALIVE	CONSERVATIVE	-	-	5	IDIOPATHIC	+	-	-	-	-		
80	Raja	40	Male	1922137	0	0	2	0	0	0	0	0	0	0	1	0	0	0	3	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	9	ALCOHOL	+	+	-	-	-		
81	Loordhusamy	32	Male	1922424	0	0	0	0	1	0	0	1	0	0	1	1	0	0	4	0	0	0	0	0	0	ALIVE	CONSERVATIVE	-	-	5	GSD	+	-	-	-	-		
82	Srinivasan	35	Male	1923166	0	0	2	1	0	0	0	0	1	0	0	0	0	0	4	0	0	1	0	1	2	ALIVE	CONSERVATIVE	-	-	11	ALCOHOL	+	+	-	-	-		
83	Ahammed	35	Male	1928417	0	0	2	0	0	0	0	1	1	0	1	1	0	0	6	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	18	ALCOHOL	+	+	+	-	-		
84	Immanuvel	26	Male	1928540	0	0	2	0	0	0	0	0	0	0	1	0	0	0	3	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	17	ALCOHOL	+	+	+	-	-	PSEUDOCYST	
85	Sundaram	32	Male	1925676	0	0	2	0	0	0	0	0	0	0	0	0	0	0	2	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	14	ALCOHOL	+	+	-	-	-		
86	Selvaraj	49	Male	1925767	2	1	2	1	2	2	0	1	1	0	2	1	0	0	16	1	0	1	0	1	3	ALIVE	SURGERY	AKI/RF	+	21	ALCOHOL	+	+	+	+	-		SAP
87	Jinnah	40	Male	1926779	0	0	0	0	0	0	0	0	0	0	1	0	0	0	1	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	5	GSD	+	-	+	+	-		
88	Sivasankar	34	Male	1928829	0	0	0	2	2	0	0	1	1	0	1	0	0	0	7	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	9	ALCOHOL	+	+	-	-	-		
89	Mahesh	32	Male	1929405	0	1	2	1	0	0	0	0	0	2	1	1	0	0	8	1	0	1	0	1	3	ALIVE	SURGERY	-	+	18	ALCOHOL	+	+	+	+	-		SAP
90	Robert	55	Male	1941909	3	1	2	1	1	2	0	1	1	2	1	1	0	0	16	1	0	1	0	1	3	ALIVE	SURGERY	-	+	22	ALCOHOL	+	+	+	+	-	HYPOCALCEM	SAP
91	Raji	52	Male	1941974	2	1	2	1	0	0	0	0	0	0	1	1	0	0	8	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	15	GSD	+	-	-	+	+		
92	Sukumar	43	Male	1947508	0	0	2	1	1	2	0	1	0	0	1	0	0	0	8	1	0	1	0	1	3	ALIVE	CONSERVATIVE	-	-	11	ALCOHOL	+	+	+	-	-		
93	Mohan	48	Male	1949460	2	0	2	1	0	0	0	0	0	0	1	1	0	0	7	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	19	ALCOHOL	+	+	+	-	-		
94	Sivagami	36	Female	1950714	0	0	2	1	0	0	0	0	0	0	1	0	0	0	4	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	5	GSD	+	-	+	-	-		
95	Shanmugam	34	Male	1957537	0	1	2	1	0	0	0	0	0	2	1	1	0	0	8	1	0	1	0	1	3	ALIVE	CONSERVATIVE	AKI	+	9	ALCOHOL	+	+	+	+	-		SAP
96	Ranganathan	33	Male	1948979	0	1	2	1	0	0	0	0	0	0	1	1	1	0	7	1	1	1	0	1	4	ALIVE	CONSERVATIVE	-	-	13	ALCOHOL	+	+	+	+	-		
97	Krishnan	55	Male	1950305	3	0	2	1	0	0	0	1	0	2	1	1	1	0	9	1	1	1	0	1	4	DEATH		SEPSIS/MODS	+	3	ALCOHOL	+	+	+	+	-	ARF	SAP
98	Sulaiman	54	Male	1955468	2	0	2	1	0	0	0	0	0	0	1	1	0	0	7	1	0	1	0	0	2	ALIVE	CONSERVATIVE	-	-	10	ALCOHOL	+	+	-	-	-		
99	Ramu	37	Male	1955764	0	1	2	1	2	2	0	1	1	3	1	2	2	0	18	1	1	1	0	1	4	DEATH		SEPSIS/MODS	+	2	ALCOHOL	+	+	-	+	-	ARF/RF	SAP
100	Ashokkumar	25	Male	1960227	0	0	2	1	0	0	0	1	0	0	1	0	0	0	5	0	0	1	0	0	1	ALIVE	CONSERVATIVE	-	-	11	ALCOHOL	+	+	-	-	-		

BUN-BLOOD UREA NITROGEN
SIRS-SYSTEMIC INFLAMMATORY RESPONSE SYNDROME
GCS-GLASGOW COMA SCALE
PE-PLEURAL EFFUSION

MODS-MULTI ORGAN DYSFUNCTION SYNDROME
AKI-ACUTE KIDNEY INJURY
ARF-ACUTE RENAL FAILURE
RF-RESPIRATORY FAILURE
NECROSIS-PANCREATIC NECROSIS

SAP-SEVERE ACUTE PANCREATITIS