"COMPARING THE EFFECTIVENESS OF BISAP SCORING SYSTEM WITH OTHER SCORING SYSTEMS IN PREDICTING THE OUTCOME IN ACUTE PANCREATITIS IN A TERTIARY HOSPITAL"

Dissertation Submitted to

THE TAMIL NADU DR.M.G.R.MEDICAL UNIVERSITY

In partial fulfilment of the regulations for the award of the degree of

M.S. BRANCH – GENERAL SURGERY

GOVERNMENT VELLORE MEDICAL COLLEGE

THE TAMIL NADU DR.M.G.R.MEDICAL UNIVERSITY

TAMILNADU, INDIA

APRIL 2020

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled "**COMPARING THE EFFECTIVENESS OF BISAP SCORING SYSTEM WITH OTHER SCORING SYSTEMS IN PREDICTING THE OUTCOME IN ACUTE PANCREATITIS** " is a bonafide work of Dr. M.A.HEMAKUMAR submitted to The Tamilnadu Dr. M.G.R Medical University in partial fulfilment of requirements for the award of the degree of M.S. BRANCH I (GENERAL SURGERY) examination to be held in MAY, 2020.

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Title of the Study	–	COMPARING THE EFFECTIVENESS OF BISAP SCORING SYSTEM WITH OTHER SCORING SYSTEMS IN PREDICTING THE OUTCOME IN ACUTE PANCREATITIS IN A TERTIARY CARE CENTRE
Principal Investigator	–	Dr. M.A. Hemakumar, I Year PG, MS General Surgery
Guide	–	Dr.R.Rajavelu,MS.,FRCS., Professor of General Surgery

The request for an approval from the Institutional Ethical and Scientific Committee (IEC) was considered on the IEC meeting held on 03.10.2018 at the Conference Hall, Govt. Vellore Medical College, Vellore-11.

The Convenor, Chairperson, Member Secretary and committee members decided to approve the proposed work mentioned above submitted by the Principal Investigator.

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This is to certify that the dissertation titled "**COMPARING THE EFFECTIVENESS OF BISAP SCORING SYSTEM WITH OTHER SCORING SYSTEMS IN PREDICTING THE OUTCOME IN ACUTE PANCREATITIS**" within General Surgery Department at Govt. Vellore Medical College Hospital. is a bonafide research work done by post graduate in M.S. General Surgery, Government Vellore Medical College & Hospital, Vellore-11 under my direct guidance and supervision in my satisfaction, in partial fulfilment of the requirements for the degree of M.S. General Surgery.

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– Vellore

CERTIFICATE II

This is to certify that this dissertation work titled **entitled "COMPARING THE EFFECTIVENESS OF BISAP SCORING SYSTEM WITH OTHER SCORING SYSTEMS IN PREDICTING THE OUTCOME IN ACUTE PANCREATITIS"**of the candidate **Dr.M.A.HEMAKUMAR** with registration Number 221711651 for the award of M.S. General Surgery in the branch of General Surgery. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows…19%… percentage of plagiarism in the dissertation.

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https://www.researchgate.net/publication/281197507_The_Value_of_BISAP_Score_for_Predicting_Mortality_and_Severity_in_Acute_Pancreatitis_A_Systematic_Review_and_Meta-Analysis
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https://www.ncbi.nlm.nih.gov/pubmed/29684670
https://www.researchgate.net/publication/273153421_Predicting_morbidity_and_mortality_in_acute_pancreatitis_in_an_Indian_population_a_comparative_study_of_the_BISAP_score_Ranson's_score_and_CT_severity_index
https://docplayer.net/134806270-Clinical-significance-of-serum-and-urinary-amylase-in-acute-pancreatitis.html
https://www.researchgate.net/publication/334121043_Accuracy_of_BISAP_score_in_prediction_of_severe_acute_pancreatitis
https://www.aafp.org/afp/2007/0515/p1513.html
https://www.ncbi.nlm.nih.gov/pubmed/29215536
https://www.science.gov/topicpages/p/pancreatitis+acute+pancreatitis
https://en.wikipedia.org/wiki/Pancreatic_necrosis
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DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled "**COMPARING THE EFFECTIVENESS OF BISAP SCORING SYSTEM WITH OTHER SCORING SYSTEMS IN PREDICTING THE OUTCOME IN ACUTE PANCREATITIS**". At Government Vellore Medical College Hospital., is a bonafide and genuine research work carried out by me in the Department of General Surgery, Government Vellore Medical and Hospital, Vellore-11, under the guidance of our Chief Prof. **Dr. R.RAJAVELU MS.,F.R.C.S.,** Government Vellore Medical College and Hospital.

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.

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ACKNOWLEDGEMENT

I am most thankful to **Prof. Dr. R.SELVI .M.D,** Dean, Vellore Medical College and Hospital for giving me the opportunity to conduct this study in the Department of General Surgery, Government Vellore Medical College Vellore-11.

I thank **Prof**. **Dr D.LOGANATHAN M.S,** Professor and Head of the department of General Surgery for his relentless care and concern that he has provided me to bring out this dissertation.

My deepest gratitude to my guide and mentor **Prof.R.RAJAVELU MS., F.R.C.S.,** Professor of the Department,, Department of General Surgery, Vellore Medical College, who has inspired me immeasurably during my training as a post graduate student.

I thank my Assistant professor **Dr.A.GOWRISHAKAR M.S,** . Guidance, supervision & encouragement throughout my career & for the preparation for this dissertation.

I thank my Assistant Professors **Dr. M.JAGADEESAN M.S., Dr. B.DANIRAJA D.A., M.S,** for their immense motivation and teaching attitude for shaping me.

I thank my fellow postgraduates and Interns for their support. Last but not the least I express my gratitude to all the patients not only for their consent and co-operation towards the preparation of this study but also for the privilege of Practicing our craft. I would like to thank my parents for making me who I am today and for supporting me in every deed of mine. I thank each and every person involved in making this manuscript from inception to publication.

CONTENTS

INTRODUCTION

Development of pancreas[11]

The term pancreas in Greek means 'all flesh'. Morphologically pancreas is made up of two distinct tissues, endocrine and exocrine derived from one simple epithelium. The morphological development is dictated by its two functions : producing digestive enzymes and regulation of blood chemistry.

The endocrine pancreas is organised as islets of Langerhans consisting five cell subtypes

- αcells – glucagon
- βcells - insulin
- δcells – somatostatin
- εcells – ghrelin
- PP cells – pancreatic polypeptide constitute 2% of the gland.

The exocrine pancreas , producing digestive enzymes, composed of acinar and ductal epithelial cells constitute 98% of adult pancreas mass.

Embryology of pancreas[12]

The pancreas is formed by the dorsal and ventral bud, originating from the endodermal lining of the caudal part of the primitive foregut tube.

Dorsal pancreatic bud forms the dorsal pancreas elongates into dorsal mesentery and ventral pancreatic duct elongates into ventral mesentery caudal to developing gall bladder forming the ventral pancreas and bile duct.

Branching of this bud occurs differently compared to the classic branching of other organs. Here a proliferating single-layered epithelium converted to multi layered and becomes stratified.

Followed by the formation of microlumen , that coalesce with epithelium to form branched lumens.

Groups of endocrine cells separate from epithelium to form islets staying peripheral to ductal and acinar cells.

By early 6th week the dorsal and ventral buds lie adjacent to each other in the plane of dorsal mesentery . later these two buds fuse to form the definitive pancreas.

Dorsal bud - **head , body and tail**

Ventral bud - **uncinate process.**

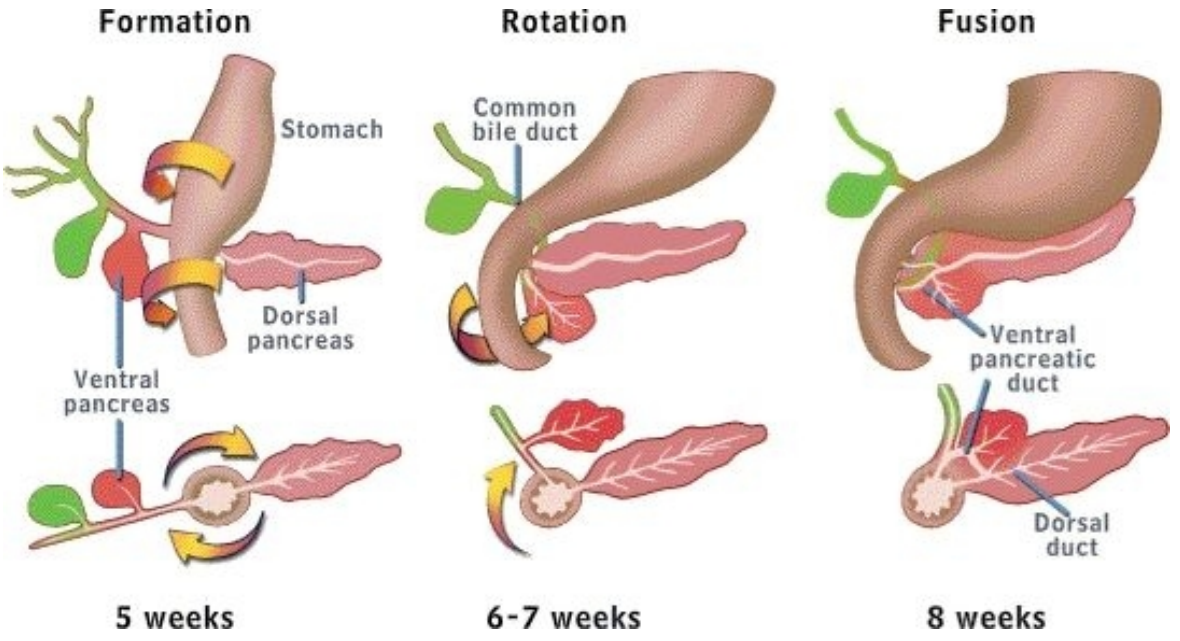
like the duodenum , pancreas attaches to the dorsal body wall becoming secondarily retroperitoneal.

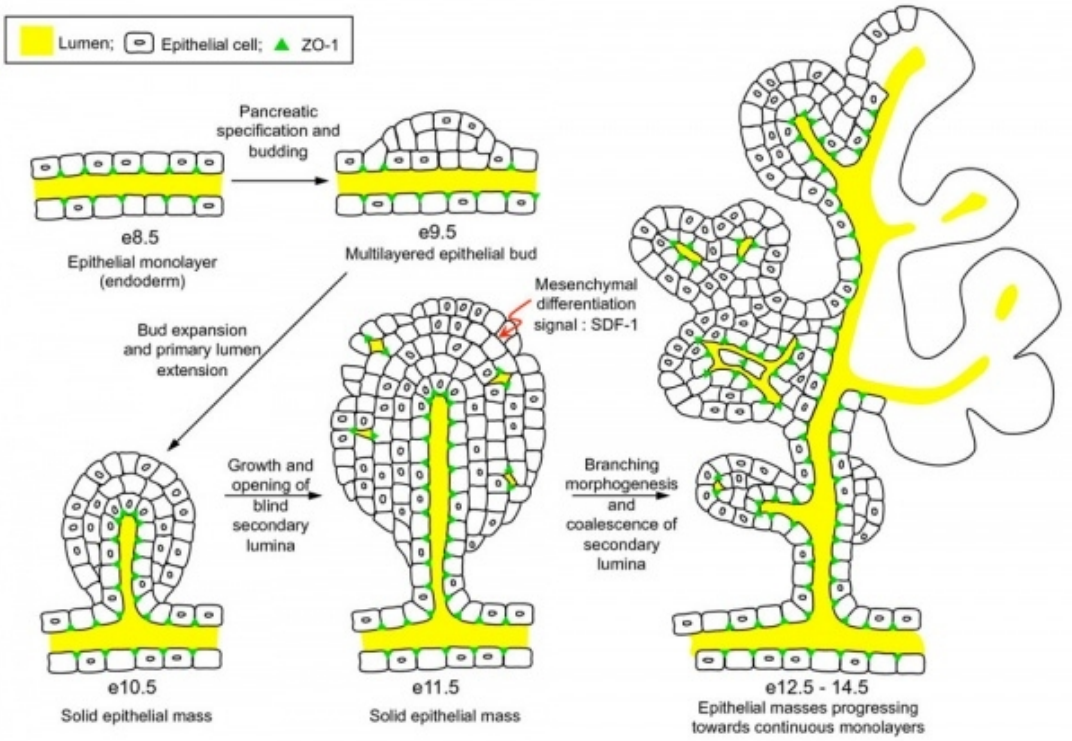
As the dorsal and ventral pancreatic buds fuse, their duct systems also become interconnected. The duct connecting the dorsal bud to duodenum usually degenerates leaving behind the duct of the ventral bud now called

the **main pancreatic duct** as the only channel for both dorsal and ventral pancreas into duodenum.

The main pancreatic duct and common bile duct join together empty into the duodenum as **ampulla of vater** at the **major duodenal papilla.**

In some patients the proximal dorsal pancreatic duct persists as an **accessory pancreatic duct** emptying into the duodenum at **minor duodenal papilla.**


Formation
Rotation
Fusion
Stomach
Dorsal pancreas
Ventral pancreas
Common bile duct
Ventral pancreatic duct
Dorsal duct
5 weeks
6-7 weeks
8 weeks


Lumen; Epithelial cell; ZO-1
Pancreatic specification and budding
e8.5
Epithelial monolayer (endoderm)
e9.5
Multilayered epithelial bud
Mesenchymal differentiation signal : SDF-1
Bud expansion and primary lumen extension
Growth and opening of blind secondary lumina
Branching morphogenesis and coalescence of secondary lumina
e10.5
Solid epithelial mass
e11.5
Solid epithelial mass
e12.5 - 14.5
Epithelial masses progressing towards continuous monolayers

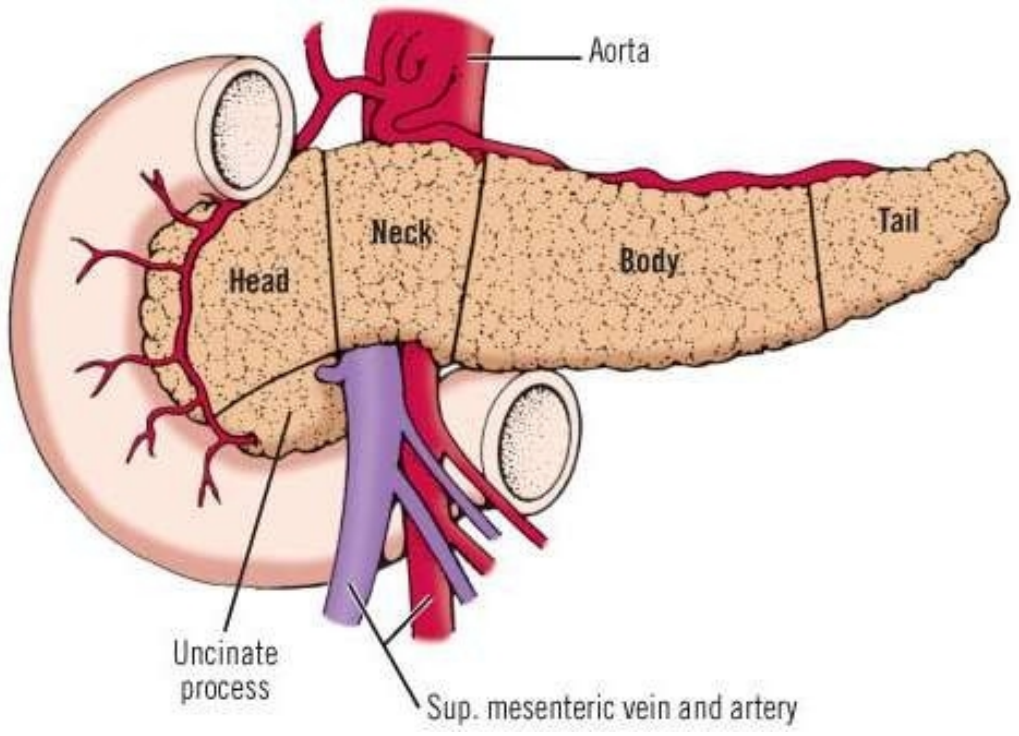
ANOMALIES OF THE PANCREAS[11]

- Aplasia
- Hypoplasia
- Hyperplasia
- Hypertrophy
- Dysplasia
- Variations and anomalies of the ducts
- Pancreas divisum
- Annular pancreas
- Pancreatic gall bladder
- Polycystic disease
- Congenital pancreatic cysts
- Cystic fibrosis
- vonHippel–Lindau syndrome
- Ectopic pancreatic tissue, accessory pancreas
- Choledochal cysts

ANATOMY OF PANCREAS[12]

The pancreas is both an exocrine and endocrine gland.

The exocrine part produces digestive juices for the digestion of food whilst endocrine part of the gland produces insulin and glucagon directly released into the blood stream.



PARTS OF PANCREAS :

- Head
- Body
- Uncinate process
- Tail

LOCATION:

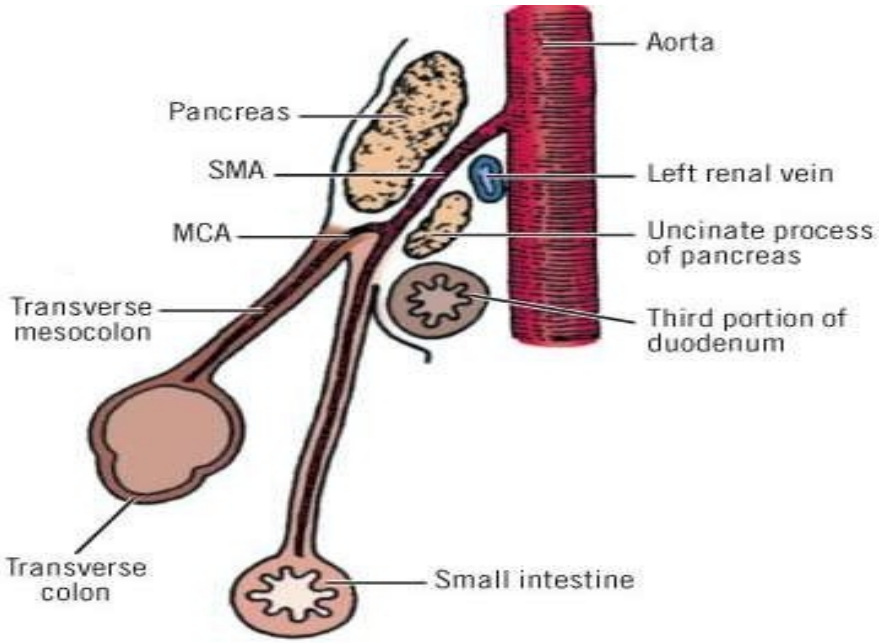
Epigastrium and left hypochondrium

Retroperitoneally at T12/L1-L3

Uncinate process :

Lies posterior to SMA and SMV

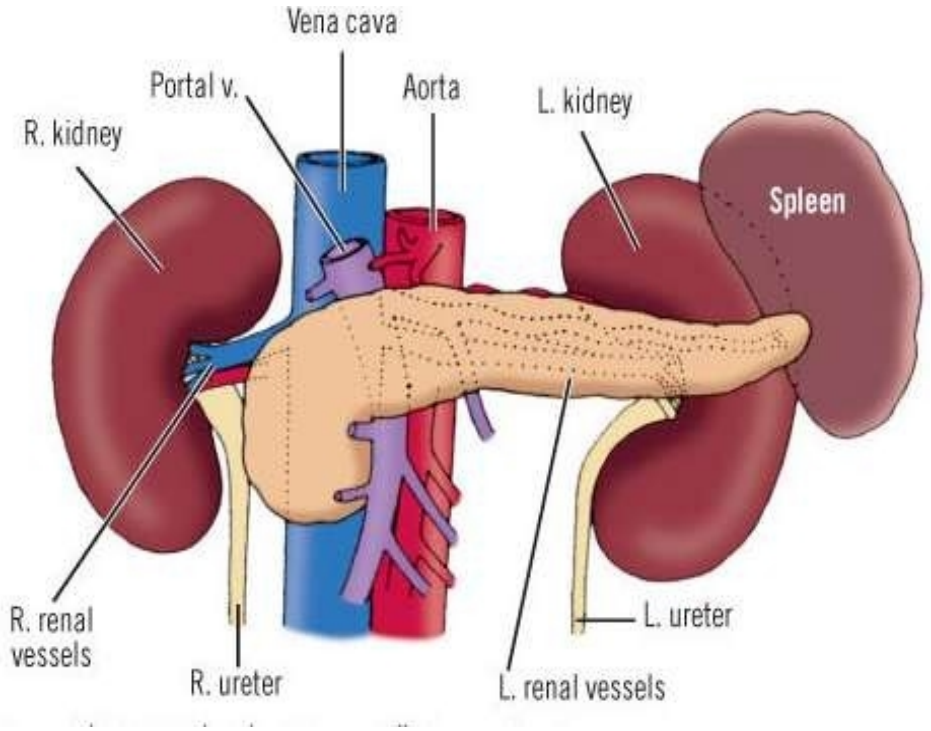
Lies anterior to aorta and IVC



THE NECK

*Lies anterior to superior mesnteric vessels and beginning of portal vein

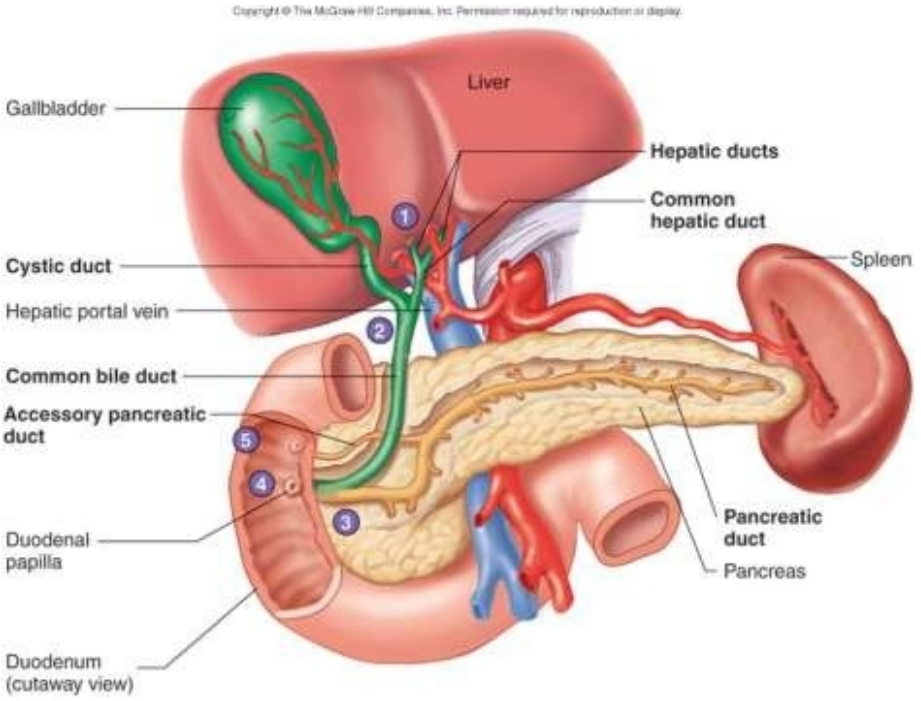
*Pylorus is just above



THE BODY

***** Related posteriorly to the aorta, the origin of the superior mesenteric artery. The splenic vein, the left kidney and its vessels, the left crus of diaphragm and the left adrenal gland.

* Celiac axis lies superior to body.



ARTERIAL SUPPLY

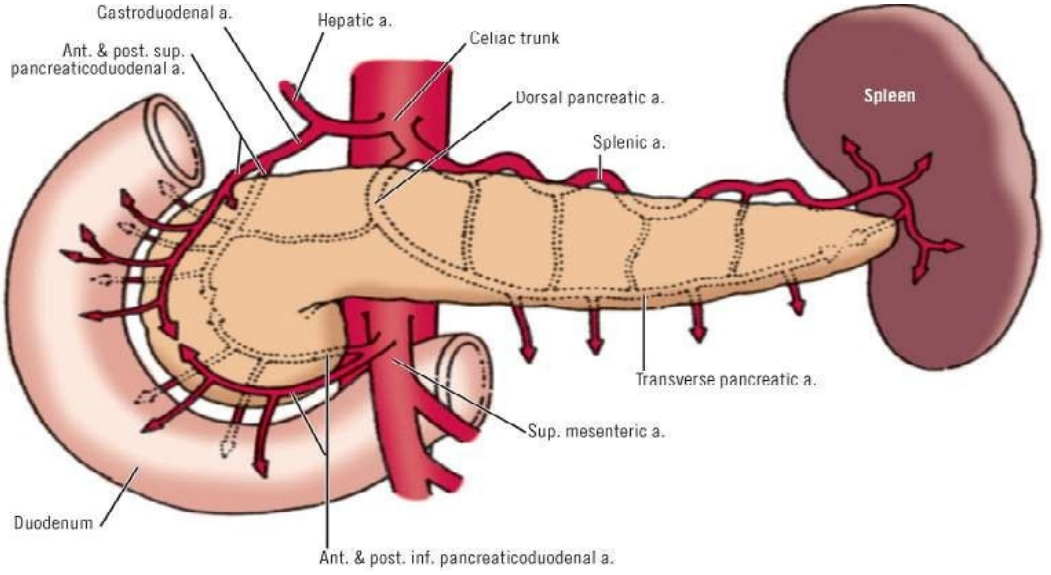
Head is supplied by

(i) Superior pancreatico duodenal artery

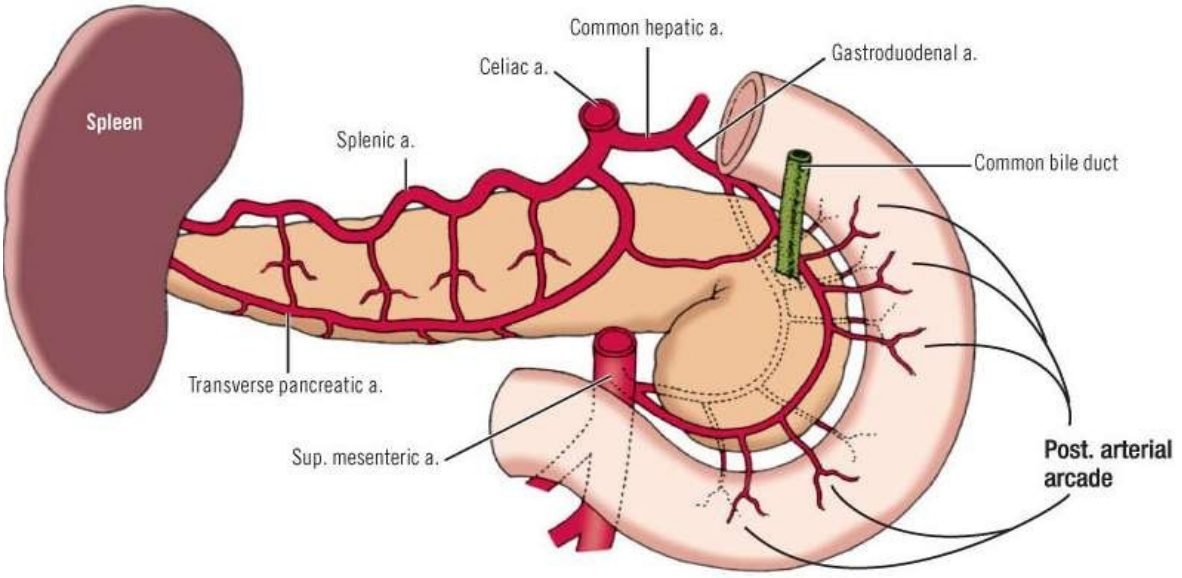
(ii) Inferior pancreatico duodenal artery

Body and tail are supplied by the branches of splenic artery

Arterial Supply: Anterior view



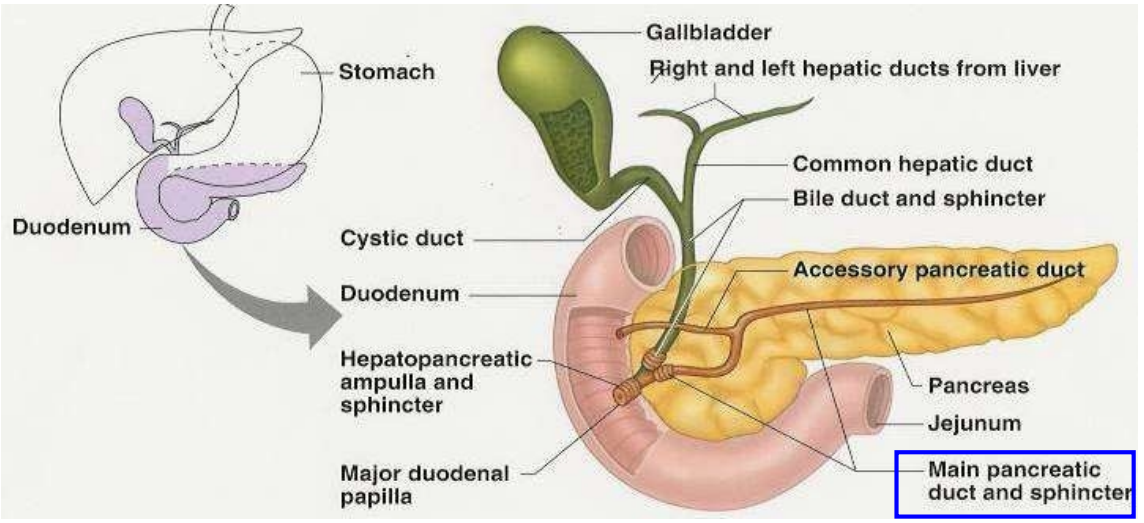
Arterial Supply: Posterior view



DUCTAL ANATOMY OF PANCREAS

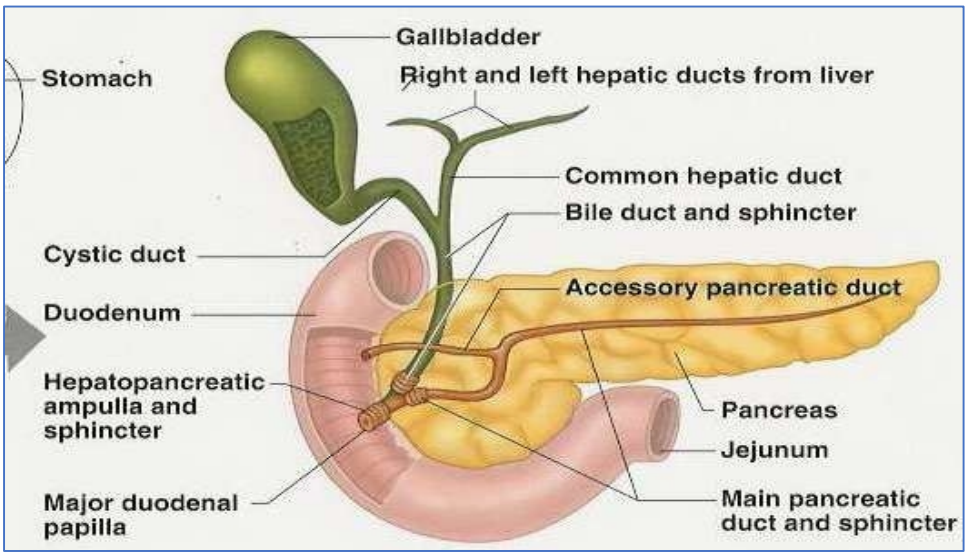
MAIN DUCT OF WIRSUNG

- Begins at tail
- Course is left to right
- Recieves numerous small ducts
- At the neck of pancreas ducts inferior, posterior and to right joins CBD at ampulla of vater 7-10cm below pylorus
- Duct diameter
    - (i) At the head of the pancreas (5mm)
    - (ii) At body (4mm)
    - (iii) At tail (3mm).



DUCT OF SANTORINI

- Accessory pancreatic duct
- Not universally identified
- Joins duodenum at minor duodenal papilla
- Part of duct from dorsal pancreas



The main pancreatic duct is 2 to 4 mm in diameter and has a ductal pressure ≈ 15 to 30 mm Hg. This is higher than the pressure in the common bile duct(7 to 17 mm Hg) thereby preventing reflux of bile into the pancreatic ductal system.

LYMPHATIC DRAINAGE

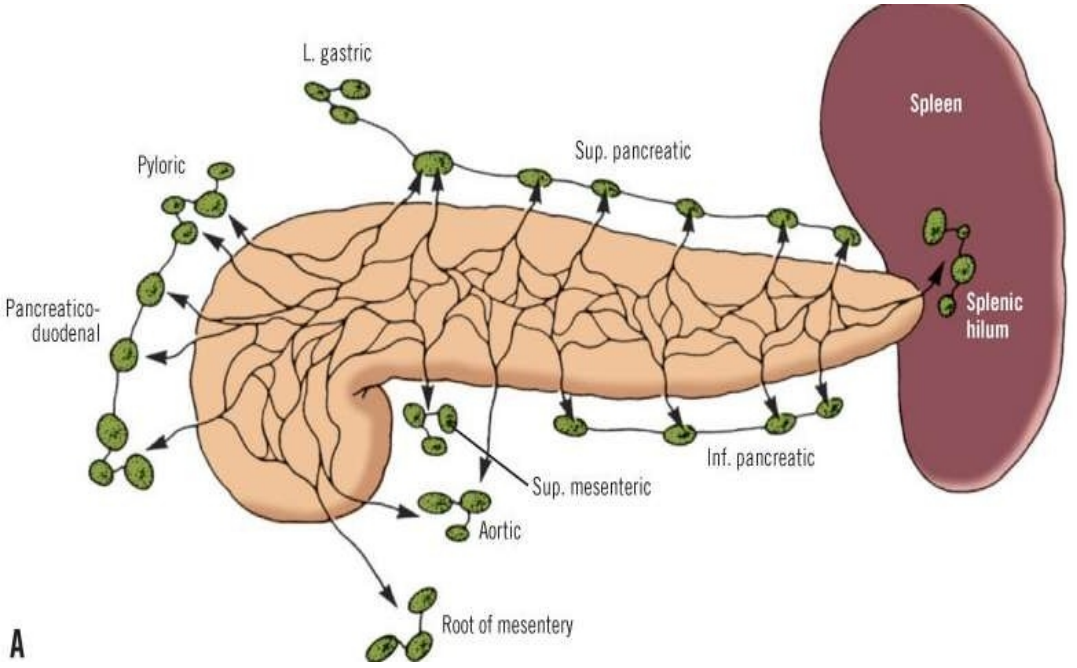
The pancreatic lymphatic vessels follow the blood vessels to:

(i) Pancreaticosplenic lymph nodes along splenic artery

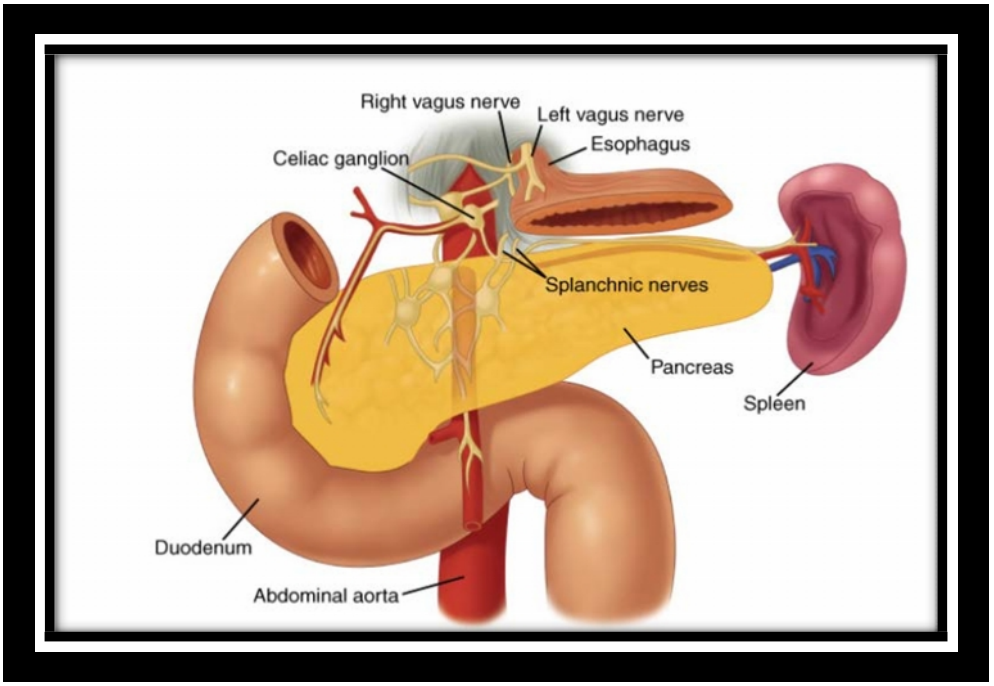
(ii) Pyloric lymph nodes

Efferent from these nodes drain into

1. Superior mesenteric lymph nodes or to
2. Celiac lymph nodes via hepatic lymph nodes



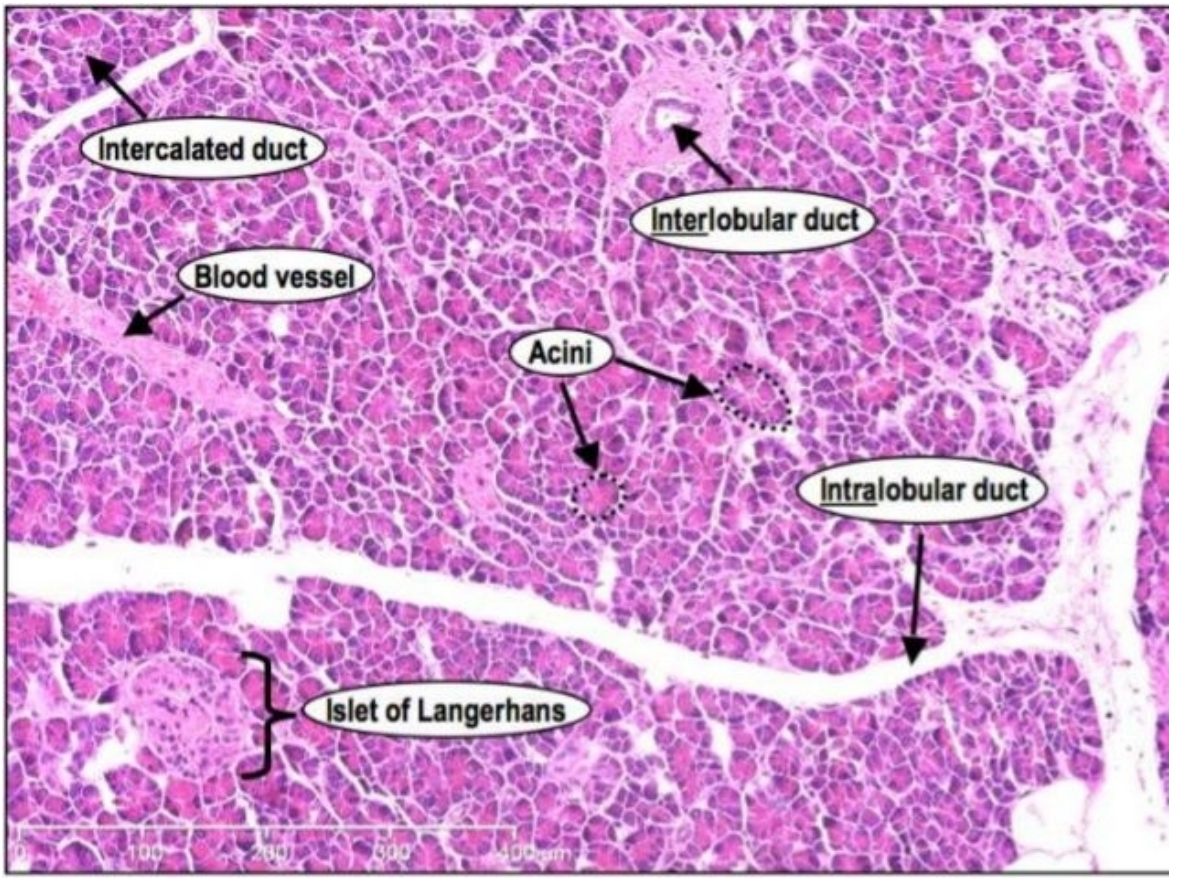
NERVE SUPPLY



Visceral efferent innervation → the vagi and the splanchnic nerves by way of the *hepatic* and *celiac* plexus.

The efferent fibers of the vagi, pass through these plexuses without synapsing. They end in parasympathetic ganglia in the interlobular septa of the pancreas. The postganglionic fibers innervate acini, islets, and the ducts.

The acinar cells which are responsible for exocrine secretion; the islet cells which are responsible for endocrine secretion; the islet vasculature, are innervated by both the systems.


Intercalated duct
Interlobular duct
Blood vessel
Acini
Intralobular duct
Islet of Langerhans

AIM

To compare the effectiveness of BISAP scoring system over other scoring systems used in risk assessment for acute pancreatitis.

OBJECTIVE

1.Evaluate BISAP score in prognosis of acute pancreatitis.

2.Comparing BISAP with other scoring systems on day of admission in predicting outcome.

STUDY DESIGN

Prospective observational study

SAMPLE SIZE

100 cases of acute pancreatitis

STUDY CENTRE

GVMCH,Tertiary care centre.

STUDY PERIOD

JUNE 2018-SEP 2019

MATERIALS AND METHODS

Demographic, clinical, and laboratory data of patients presenting within 2 weeks of onset were collected. BISAP, APACHE II scores on day of admission and RANSON's within 48 hrs were calculated after obtaining consent.

Area Under the Curve (AUC) was calculated for each scoring system for predicting SAP, mortality and ICU admission, obtaining optimal cutoff values from the receiver operating characteristic (ROC) curves

Multivariate analysis was used to identify predictors of outcome.

INCLUSION CRITERIA

Diagnosis of AP was made patients with clinical symptoms and elevated serum amylase/lipase (>3 times the upper limit of normal) or

characteristic findings on imaging Patients with AP presentedwithin 2 weeks of onset.

Post ERCP pancreatitis(new onset abdominal pain with serum amylase or lipase > 3 times upper limit of normal) older than 12 years and gave informed consent were included in the study.

EXCLUSION CRITERIA

Patients known to have chronic pancreatitis

and those who did not give consent were excluded from the study.

ANALYSIS PLAN

Statistical package for social sciences

INTRODUCTION

ACUTE PANCREATITIS :

Acute Pancreatitis is an inflammation of glandular parenchyma of the pancreas. Leading to injury or irreversible destruction of acinar components. The disease process could either result in a Self limited disease without any complications. Or it could end up in a auto-digestion of Gland resulting in systemic cytotoxic effects11.

Acute pancreatitis can be

(i) Mild / interstitial edematous pancreatitis

Affects majority , less mortality and organ dysfunction.

(ii) Severe/ necrotising pancreatitis

Seen in <10% of cases , associated with SIRS.

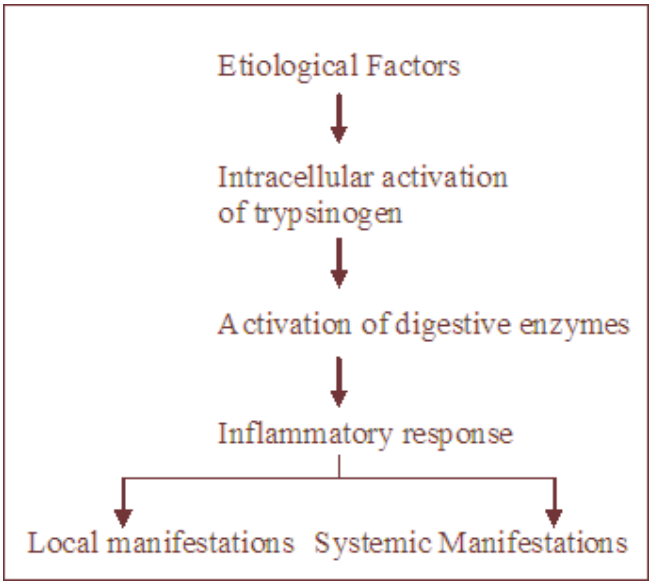
High mortality rates and organ dysfuction

ETIOLOGY[12]:

Common	Less common
Gallstones Alcohol Iatrogenic (post-ERCP, surgery) Idiopathic (<10%)	Trauma Infections, e.g. mumps, coxsackie B, HIV, adenovirus Pancreatic tumours Hereditary Congenital pancreatic abnormalities, e.g. pancreas divisum Metabolic, e.g. hypercalcaemia, hypertriglyceridaemia Venom, e.g. scorpion stings, spider Sphincter of Oddi dysfunction (see p. 493) Miscellaneous Drugs (see below)
Drugs	
Azathioprine/mercaptopurine Didanosine Oestrogens Antibiotics, e.g. tetracycline Valproic acid Furosemide Sulphonamides	Aminosalicylates Corticosteroids Metronidazole ACE inhibitors

PATHOGENESIS :

The exact mechanism of pathogenesis of pancreatitis remains to be unknown. The most commonly accepted mechanism is as follows.



some of the known concepts include as mentioned below

- *Autodigestion* by inappropriately activated pancreatic enzymes.
- *Trypsin* (+) digestive enzymes and prekallikrein → activation of clotting and Complement and clotting systems → microvascular thrombosis.
- Gallstones/alcohol concretions → increased intraductal pressure → accumulation of enzyme rich interstitial fluid → fat necrosis → inflammatory infilterate and cell injury

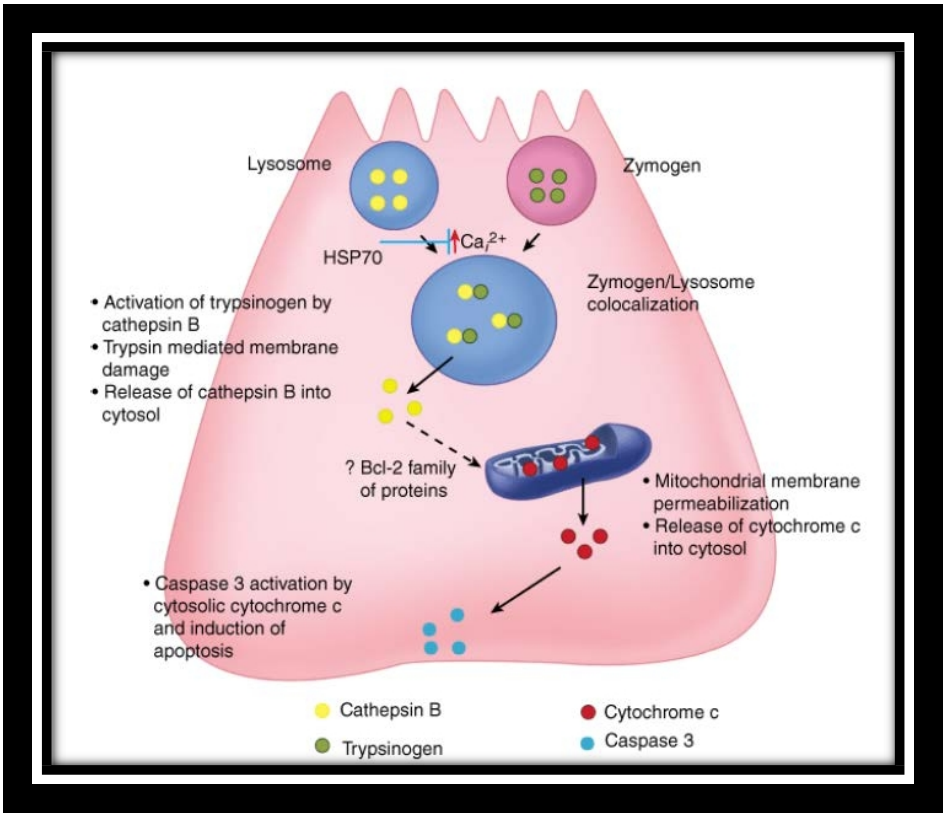
❖ Acinar cell injury by infection , drug , trauma ,shock. Premature release of pro enzyme and lysosomal hydrolases.

❖ Genetic factors identified in pathogenesis of acute pancreatitis are the following;

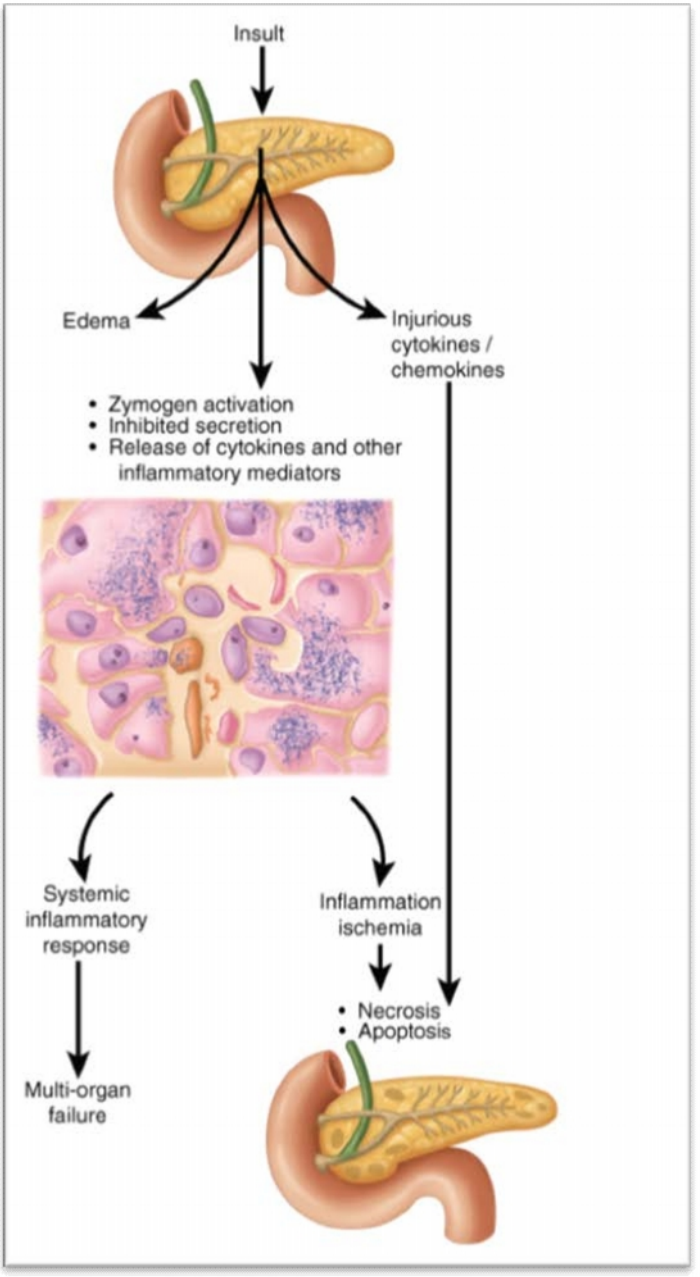
Cationic trypsinogen gene (*PRSS1*).

Cystic fibrosis transmembrane conductance regulator gene (*CFTR*).

Polymorphisms in SPINK1.



ACTIVATION OF ENZYMES



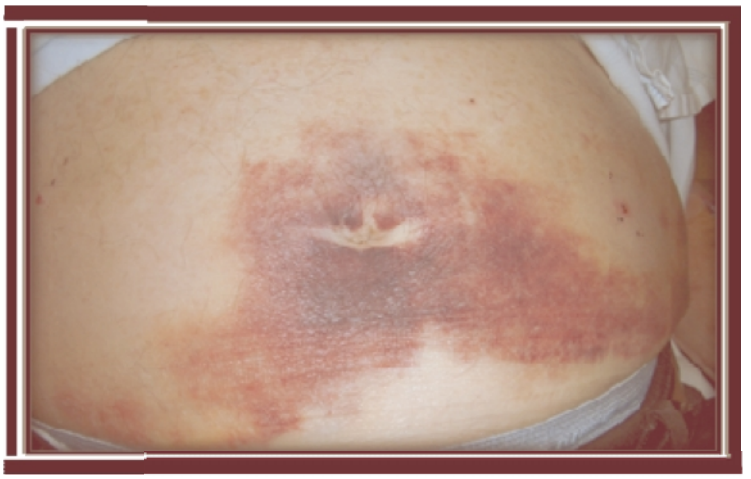
CLINICAL FEATURES :

- ❖ Pain
    - Cardinal symptom.
    - Epigastrium , can be generalised.
    - Stabbing type.
    - Radiating to back.
    - Relieved on leaning forwards.
    - Referred to shoulders
- ❖ Nausea ,vomiting,retching.

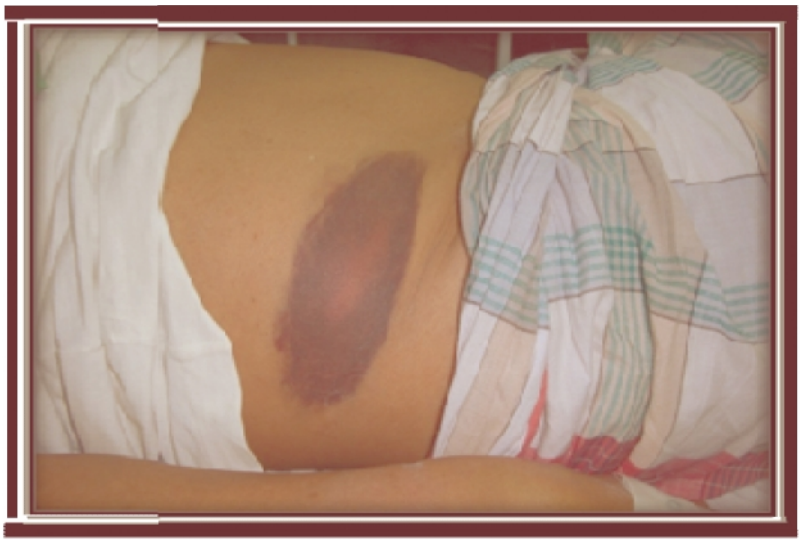
Per Abdomen:-

- ❖ Tenderness localized to epigastrium or diffuse.
- ❖ Guarding and rigidity.
- ❖ Absent bowel sounds due to ileus.
- ❖ Subcutaneous fat necrosis leading to subcutaneous tenderness and edema.
- ❖ Retroperitoneal haemorrhage leading to bluish discolouration

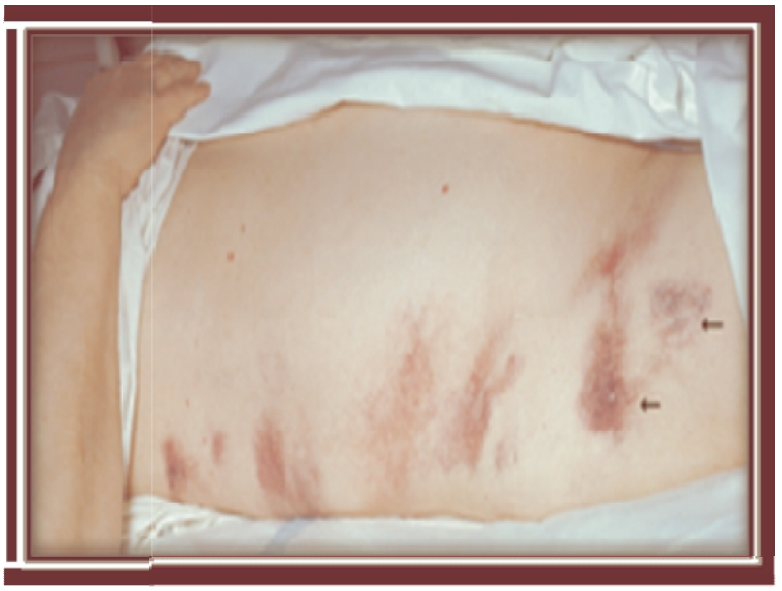
Umbilical area – Cullen's sign[12]



Flank - grey turner sign[12]



Groin-fox sign[12]



CLINICAL SIGNS[14]:

- Hyperthermia
- Hypotension / shock
- Tachycardia
- Tachypnea
- Toxicity
- Confusion
- Mild icterus (biliary obstruction in gall stone pancreatitis)
- Retroperitoneal hemorrhage
- Cullen's sign
- Grey turner's sign
- Fox's sign
- Shifting dullness (in case of ascites)
- Pleural effusion
- Acute swinging pyrexia (cholangitis)
- Small red tender nodules (subcutaneous fat necrosis)

DIAGNOSIS[15] :

Laboratory Test	Time of onset (Hours)	Purpose	Clinical observation /limitations
Alanine transaminase	12 to 24	Diagnosis .and etiology	Associated with gallstone pancreatitis; threefold elevation or greater in the presence of acute pancreatitis has a positive predictive value of 95 percent in diagnosing acute gallstone pancreatitis
Amylase	2 to 12	Diagnosis	Most accurate when at least twice the upper limit of normal; amylase levels and sensitivity decrease with time from onset of symptoms
C-reactive protien	24 to 48	Predictive of severity	Late marker; high levels associated with pancreatic necrosis
Interleukin-6	18 to 48	Predictive of severity	Early indication of severity
Interleukin-8	12 to 24	Predictive of severity	Early indication of severity
Lipase	4 to 8	Diagnosis	Increased sensitivity in alcohol−induced pancreatitis; more specific and sensitive than amylase for detecting acute pancreatitis

Phospholipase A_2	24	Predictive of severity	Associated with development of pancreatic necrosis and pulmonary failure
Procalcitonin	24 to 36	Predictive of severity	Early detection of severity; high concentrations in infected necrosis
Trypsinogen activation Peptide	Within a few hours	Diagnosis and predictive of severity	Early marker for acute pancreatitis and close correlation to severity

serum amylase and *lipase* are of diagnostic importance.

Serum amylase in AP is above threefold of the normal values. Levels are usually increased within a few hours of disease onset. Serum amylase usually remains elevated for 3–5 days in uncomplicated AP Specificity is <70 %

Urinary amylase and amylase to creatinine ratio distinguish pancreatitis from other causes of increased amylase.

Causes of Increased Serum Amylase Activity

- Pancreatic diseases
- Acute pancreatitis
- Pancreatic cancer
- Abdominal emergencies
- Acute cholecystitis
- Common bile duct obstruction
- Perforated viscous
- Intestinal ischemia
- Acute appendicitis
- Ruptured ectopic pregnancy and acute salpingitis
- Salivary gland diseases
- Renal insufficiency
- Macroamylasemia

IMAGING[12,13]

ULTRASONOGRAPHY

Advantages :

- widely available
- relatively inexpensive and safe.
- GB sludge/stone
- CBD size

Limitations:

- overlying bowel gas owing to ileus and peripancreatic edema.
- sensitivity and specificity is low.

COMPUTED TOMOGRAPHY SCAN :

The role of CT is both to document the findings that confirm the diagnosis of AP. To exclude other acute abdomen causes that may mimic pancreatitis. CT findings which suggest pancreatitis as a diagnosis include :

- diffuse or segmental enlargement of the pancreas.
- irregularity of the pancreatic contour.
- obliteration of the peripancreatic fat planes.
- areas of decreased density within the pancreas.
- ill-defined fluid collections in the pancreas or outside the gland

Computed Tomography Grading System[15]

- Grade A: Normal findings.
- Grade B: Focal or diffuse pancreatic enlargement.
- Grade C: Inflammation of the pancreas and pancreatic fat.
- Grade D: Peripancreatic fluid collection in single location.
- Grade E: Two or more fluid collections or the presence of peripancreatic gas.

ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY[12,13]

Endoscopic retrograde cholangiopancreatography (ERCP) has no role in diagnosing AP. Therapeutic role of ERCP in acute gallstone pancreatitis. has been to lower morbidity and mortality when compared to traditional medical treatment alone.

RISK STRATIFICATION IN ACUTE PANCREATITIS

Early evaluation of AP severity is essential . to allow the clinician to predict the patient's clinical course, estimate prognosis. It also helps in detecting patients needing intensive care.

ATLANTA CLASSIFICATION[12]

Box 15.4

Severity of acute pancreatitis: revised Atlanta criteria 2013

Mild

- Absence of organ failure
- Absence of local complication

Moderate/severe

- Local complication and/or transient organ failure (<48 h)

Severe

- Persistent organ failure (>48 h), defined by the modified Marshall Score for the evolution of organ failure (see ***Box 15.5***)

SCORING SYSTEMS USED FOR ASESSING SEVERITY IN ACUTE PANCREATITIS[11]

- Ranson
- Modified Glasgow system
- APACHE(Acute physiology and chronic health evaluation)
- BISAP(Bedside index in severity of acute pancreatitis)
- SAPS(simplified acute physiology score)
- SOFA(Sequential organ failure assessment score)
- MOD(Multiple organ dysfunction score)
- Modified marshall

VARIABLES OF THE RANSON CRITERIA AND MODIFIED GLASGOW SYSTEM[12]

Ranson Criteria

For Acute Non-Gallstone Pancreatitis

Ranson criteria in pancreatitis

Criteria	Measurement
At admission	
Age	>55 years
WBC	>16 × 10^9/L
Blood glucose	>11 mmol/L
Serum LDH	>600 U/L
Serum aminotransferase	>250 U/L
Within 48 h	
Haematocrit fall	>10%
Serum aminotransferase	>200 U/L
Serum calcium	<2 mmol/L
Serum urea increase	>1.8 mmol/L
Base deficit	>4 Meq/L
P_aO_2	<8.0 kPa (60 mmHg)

LDH, lactate dehydrogenase; WBC, white blood cell count.

For Acute Gallstone Pancreatitis

Upon admission:

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

Within 48 hours:

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

Modified Glasgow System

Glasgow prognostic criteria in acute pancreatitis[a]

Criteria	Measurement
Age	>55 years
WBC	>15 × 10^9/L
Blood glucose	>10 mmol/L
Serum urea	>16 mmol/L
Serum albumin	<30 g/L
Serum aminotransferase	>200 U/L
Serum calcium	<2 mmol/L
Serum LDH	>600 U/L
P_aO_2	<8.0 kPa (60 mmHg)

BISAP Score

Parameters	Score 0	Score 1
Blood urea nitrogen	<25 mg/dl	>25 mg/dl
Impaired mental status	Absent	Present
SIRS	Absent	Present
Age	<60 years	>60 years
Pleural effusion	Absent	Present

SIRS (Systemic Inflammatory Response Syndrome) is diagnosed by presence of any two of criteria:

1) Temperature (<36c or >38c),
2) Pulse > 90/min,
3) Respiratory Rate >20 or PaCO2 <32mmHg, and
4) WBC >12,000/mm^3 or <4,000/mm^3 or >10% bands.

Each point on BISAP score is worth 1 point. There is steady increase in risk for mortality with the increasing number of points. BISAP score is an uncomplicated, quick and reasonably reliable for assessment of disease severity on admission.

ADVANTAGES:

Simple and easy to calculate, usually done at the time of admission or within 24 hrs of hospitalization.

The scores prediction ability was tested across 400 hospitals among large number (36,000) of populations, in contrast to other studies which were based on small number patients.

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

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

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

APACHE II SCORING

It is abbreviated as Acute Physiology and Chronic Health Evaluation (APACHE II) score[11].

It is probably the most widely studied scoring system in acute pancreatitis. It has good negative predictive value. Having a modest positive predictive value, in predicting severity of AP and can be performed daily. Decreasing values during the first 48 hours will suggest a mild attack, whereas increasing values suggest a severe attack. Studies suggest that mortality is less than 4% with a score < 8 and is 11 to 18% with a score > 8.

APACHE II provides a general measure of the severity of disease. It is based on the patient's age, previous health status, and 12 routine physiologic measurements. An APACHE II score of 8 or more, defines severe pancreatitis. It has the advantage of be used on a daily basis. It has similar positive and negative predictive values as the Ranson score at 48 hours.

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

The Acute Physiology and Chronic Health Evaluation (APACHE) II scoring system[a]

Physiological

- Temperature
- Heart rate
- Respiratory rate
- Mean arterial pressure
- Glasgow Coma Scale score
- Presence of acute kidney injury
- Age
- Organ insufficiency
- Immunocompromise

Laboratory

- Oxygenation (P_aO_2)
- Arterial pH
- Serum:
  - Sodium
  - Potassium
  - Creatinine
  - Haematocrit
  - White blood cell count

[a]APACHE is applied within 24 h of admission. Score can range from 0–71 (normal to abnormal). Higher scores correspond to more severe disease and a higher risk of death. Body mass index (BMI) is an additional parameter that can be added specifically in assessing severity of acute pancreatitis.

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

The laboratory tests which are required are simple, routine and readily available.

APACHE-II[11] scores on admission and within 48 hours help distinguish mild from severe pancreatitis and to predict death. Most patients survive if APACHE-II scores are 9 or less during the first 48 hours. Patients with APACHE-II scores of 13 or more have a high likelihood of dying.

It takes into account all the major risk factors that influence the outcome from the disease including the acute physiological derangements, as well as the patient's ability to recover which may be diminished by advancing age or chronic disease.

The range of the APACHE II score is wide, providing a better spread between the mild and severe attacks because varying weights are assigned to increasingly abnormal values, rather than all or no judgements.

At admission, sensitivity is 33% to 71%, and specificity is 75% to 97%.

At 48 hours, sensitivity remains less than 52%, but specificity is close to 91% to a Score of ≥ 2 indicates presence of organ failure. These scores were calculated within 72 hours of hospitalisation. The organ failure was classified as[34]: Transient (less than 48 hrs.) Persistent (more than 48 hrs.)

CT Severity Index

Pancreatic inflammation		**points**
	Normal pancreas	**0**
	Enlargement of the pancreas	**1**
	Peripancreatic inflammation	**2**
	1 acute peripancreatic fluid collection	**3**
	⩾ 2 acute peripancreatic fluid collections	**4**
Pancreatic necrosis	None	**0**
	< 30%	**2**
	30% - 50%	**4**
	> 50%	**6**

Maximum 10 points

TREATMENT OF ACUTE PANCREATITIS

TABLE 88-2 Treatment of Acute Pancreatitis in Various Clinical Scenarios

Clinical Situation	Advice	Exception
	WEEKS 1-2	
Predicted severe pancreatitis	Fluid supplementation based on urine production, enteral nutrition, adequate pain control. Not useful: routine antibiotic prophylaxis antioxidants, and oral probiotics.	
Abdominal compartment syndrome	Decompression laparotomy without accessing the retroperitoneum	Large amounts of intraabdominal fluid. In these cases percutaneous catheter drainage may be used but should lead to immediate clinical improvement.
Sterile necrosis (collections) and multiple organ failure	Treat organ failure. No evidence that necrosectomy and/or drainage of collections will improve outcome. There is evidence that drainage will increase the risk of infection.	Abdominal compartment syndrome, bowel ischemia, bleeding
	WEEK 3 AND THEREAFTER	
Infected necrosis (collections) without or with only partial encapsulation	If possible, postpone intervention using antibiotics	Rapid deterioration without treatable cause
Infected walled-off necrosis (collections)	Intervention according to the "step-up" approach, starting with (retroperitoneal) catheter drainage. If needed, followed by (minimally invasive) necrosectomy.	Lack of experience; if so, transfer the patient to a more experienced center

Surgical Management

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

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

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

Indications for surgery in case of acute pancreatitis[12]

Surgical approach to the treatment of pancreatic necrosis	
Open surgery approaches	Minimally invasive approaches
Pancreatic resection	Laparoscopic necrosectomy
Necrosectomy + wide tube drainage	Laparoscopic assisted percutaneous drainage
Necrosectomy + relaparotomy (staged reexploration)	Laparoscopic transgastric necrosectomy
Necrosectomy + laparostomy± open packing	Percutaneous necrosectomy and sinus tract endoscopy
Necrosectomy + drainage + closed continuous lavage	MRI−radiologically assisted necrosectomy
	Video-assisted retroperitoneal debridement

Surgical options for pancreatic necrosis

COMPLICATIONS OF ACUTE PANCREATIS[12]

Complications can be divided into local and systemic

LOCAL

- ❖ Fluid collections
- ❖ Pancreatic ascites/pleural effusion
- ❖ Pancreatic pseudocyst
- ❖ Pancreatic necrosis
- ❖ Infected pancreatic abscess
- ❖ Hemorrhage/pseudo aneurysm

SYSTEMIC

A. PULMONARY

1. Pneumonitis, basal atelectasis
2. ARDS
3. Pleural effusion (L)

B. CARDIOVASCULAR

1. Hypotension
2. Hypovolemia
3. Sudden arrest &death
4. Nonspecific ECG (ST-T wave) changes
5. Pericardial effusion

C. HEMATOLOGIC

1. Hemoconcentration

2. Disseminated intravascular coagulopathy

D. GI hemorrhage

1. Acid peptic disease

2. Gastric erosion

3. Portal/splenic vein thrombosis with variceal bleed

E. RENAL

1. Oliguria

2. Azotemia

3. Renal vessel thrombosis

F. METABOLIC

1. Hyperglycemic state

2. Hypocalcemic state

3. Hyperlipidemia (triglyceridemia)

4. Metabolic encephalopathy

5. Sudden loss of vision (Purtscher's retinopathy)

G. CENTRAL NERVOUS SYSTEM

1. Acute psychosis

2. Fat embolism occlusion

3. Alcohol withdrawal syndrome (AWS)

H. FAT NECROSIS

1. Intra-abdominal saponification

2. Subcutaneous tissue necrosis

BOX 58-7 Complications of Acute Pancreatitis

Local
- Pseudocyst
- Sterile necrosis
- Infected necrosis
- Abscess
- GI bleeding
  - Pancreatitis-related
    - Splenic artery or splenic artery pseudoaneurysm rupture
    - Splenic vein rupture
    - Portal vein rupture
    - Splenic vein thrombosis leading to gastroesophageal variceal bleeding
    - Pseudocyst or abscess hemorrhage
    - Postnecrosectomy bleeding
  - Nonpancreatitis-related
    - Mallory-Weiss tear
    - Alcoholic gastropathy
    - Stress-related mucosal gastropathy
- Splenic complications
  - Infarction
  - Rupture
  - Hematoma
  - Splenic vein thrombosis
- Fistulization to or obstruction of the small intestine or colon
- Hydronephrosis

Systemic
- Respiratory failure
- Renal failure
- Shock
- Hyperglycemia
- Hypocalcemia
- DIC
- Fat necrosis (subcutaneous nodules)
- Retinopathy

Psychosis

REVIEW OF LITERATURE

Hagjer S et al[8] prospective observational study of 60 patients. Presenting with acute pancreatitis was done Medical College and Hospital. From July 2015 to June 2016. BISAP, APACHE-II, Ranson criteria, and CT severity index (CTSI) of all patients were calculated.Of the 60 patients, 14 developed SAP.11 Organ failure. 21 pancreatic necrosis and 7 died.The BISAP predicts severity, organ failure and death, very well.It is as good as APACHE-II but better than Ranson criteria

Zheng J et al [1]total of 114 cases of AP the scores of BISAP, acute physiology and chronic health evaluation (APACHE II), Ranson and computed tomography severity index (CTSI) were obtained .With rising BISAP scores, both severity and mortality increased in acute pancreatitis better predictive value for AP.

Arif A et al [110] cross sectional study total of 206 patients were included. subjected to investigations for Ranson's and BISAP scoring. On the basis of sensitivity, Ranson's scores predicted SAP more accurately than BISAP scores. Regarding specificity, both scores predicted SAP almost equally10 .

Vasudevan S, et al[7] 343 patients included,APACHEII BISAP marshall SCORE scores were calculated along with crp in predicting severity.Both BISAP and APACHE II are comparable in predicting

outcome. BISAP predicted all 3 outcomes with the same cutoff and hence is a robust scoring system.

Senapati D et al [9]9 246 patients included. 207 patients had no organ failure.Remaining 39 developed organ failure. 17 patients had persistent organ failure, 16 of those with BISAP score ≥3. 13 patients died, out of which 12 patients had BISAP score ≥3.The BISAP score is a simple and accurate method. For the early identification of patients at increased risk.

Chen Let al[4] Clinical data for 497 patients with AP were analyzed retrospectively. to compare BISAP with other scores in predicting the severity of AP . Detecting the occurrence of pancreatic necrosis, mortality, and organ failure. 396 had mild AP and 101 had SAP. BISAP performed similarly to other scoring systems in predicting SAP. It also detected well pancreatic necrosis, mortality, and organ failure in SAP patients. BISAP score is valuable in predicting the severity of AP and prognoses of SAP.

Shabbir S et al[9] studied total of 80 patients All patients were scored according to both Ranson's score and BISAP score. The number of patients with a BISAP score of ≥ 3 was 15. Those with Ranson's score ≥ 3 was 25.The newly proposed BISAP score is a simple and accurate tool. Was found to be equally effective in severity stratification and frequency of severity.

Yang L et al[2], studied a total of 326 diagnosed hyperlipidemic acute pancreatitis patients. With in study period from August 2006 to July 2015 retrospectively. Ranson did not have significant advantage in predicting severity and prognosis. APACHE II was the best in predicting severity of HLAP. APACHEII had shortcoming in predicting local complications whichb was overcome by MCTSI .But MCTSI was poor in predicting severity . BISAP score had high accuracy in assessment of severity, local complications, and mortality of HLAP

Chandra S et al[6] included twelve studies. Data-synthesis and methodology quality assessment was performed for 10. Studies using revised Atlanta classification in defining SAP had a pooled AUC of 0.92 . But heterogeneity persisted, $I2$ 67%. Subgroup analysis based on rate of SAP did not eliminate the heterogeneity. The BISAP has very good predictive performance for SAP across different patient population and etiologies

Ye JF et al studied 302 patients with AP according to single-factor logistic regression analysis. It was found that BISAP, MEWS and serum Ca2+ are prediction indexes of the severity of AP . Whereas RDW is not a prediction index of AP severity . The multi-factor logistic regression analysis shows BISAP and serum Ca2+ are independent prediction indexes of AP severity . MEWS is not an independent prediction index of AP severity .There is remarkable statistical significance for the predictive ability for

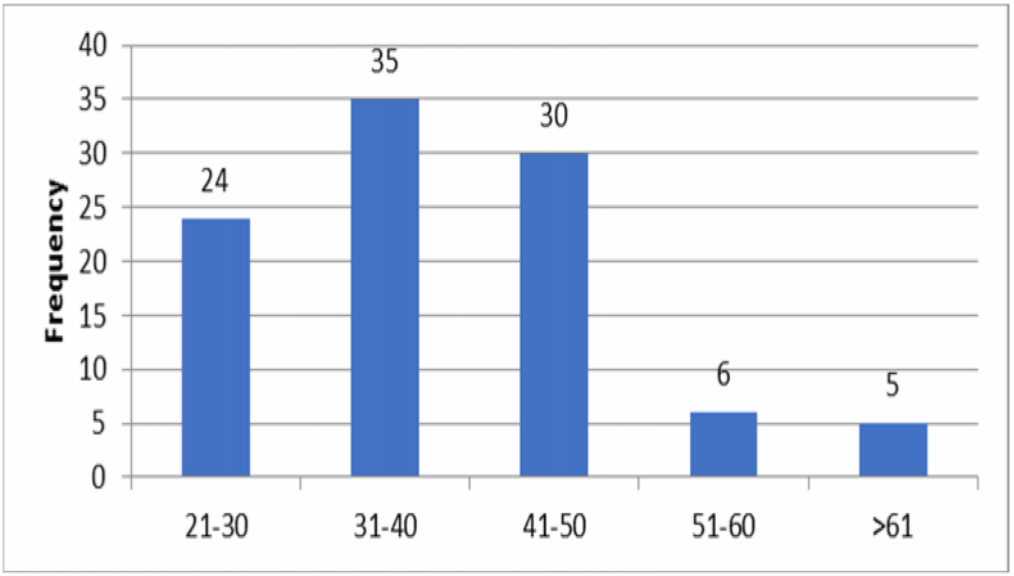
BISAP and serum Ca2+ individually. It was concluded that BISAP and serum Ca2+ have high predictive value for the severity of AP.

Park JY et al [5]analyzed 303 patients with acute pancreatitis based on BISAP, APACHE-II, Ranson criteria and CTSI . The BISAP predicts severity, death, and especially organ failure in acute pancreatitis as APACHE-II does. But it was found to be better than Ranson criteria, CTSI.

B U Wu et al, used classification and regression tree (CART) analysis. It is a clinical scoring system . It was developed for prediction of in hospital mortality in acute pancreatitis. The scoring system was derived on data collected from around 18000 cases of acute pancreatitis. The BISAP scoring system was validated on data collected from 18,246 acute pancreatitis cases .The accuracy of the BISAP was measured by the area under the AUC. BISAP is a simple and accurate method for the early identification of patients at increased risk for in- hospital mortality.

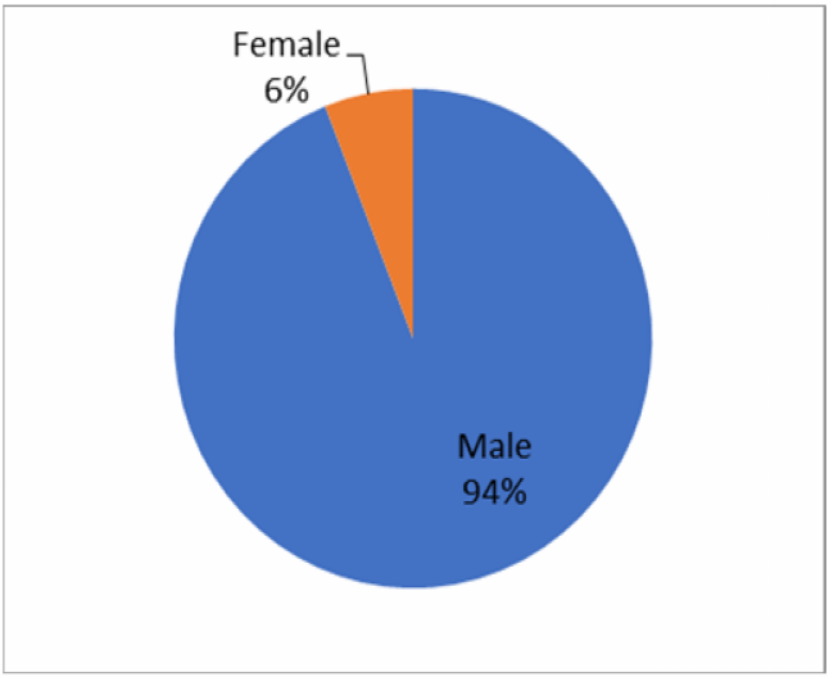
AGE WISE DISTRIBUTION

Age group	Frequency	Percent
21-30	24	24.0%
31-40	35	35.0%
41-50	30	30.0%
51-60	6	6.0%
>61	5	5.0%
Total	100	100.0%



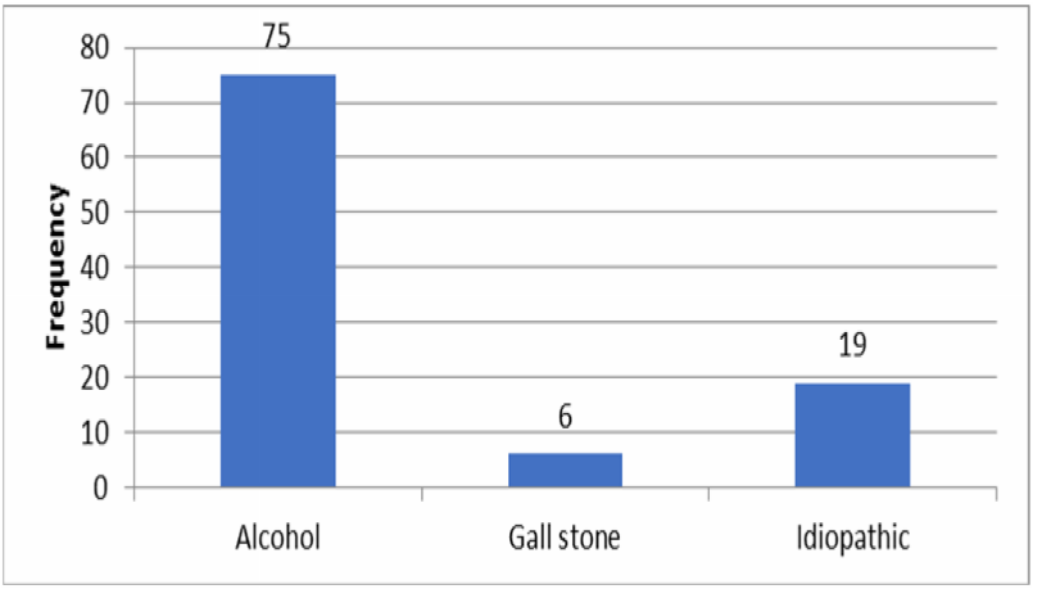
GENDER WISE DISTRIBUTION

Gender	Frequency	Percent
Male	94	94.0%
Female	6	6.0%
Total	100	100.0%



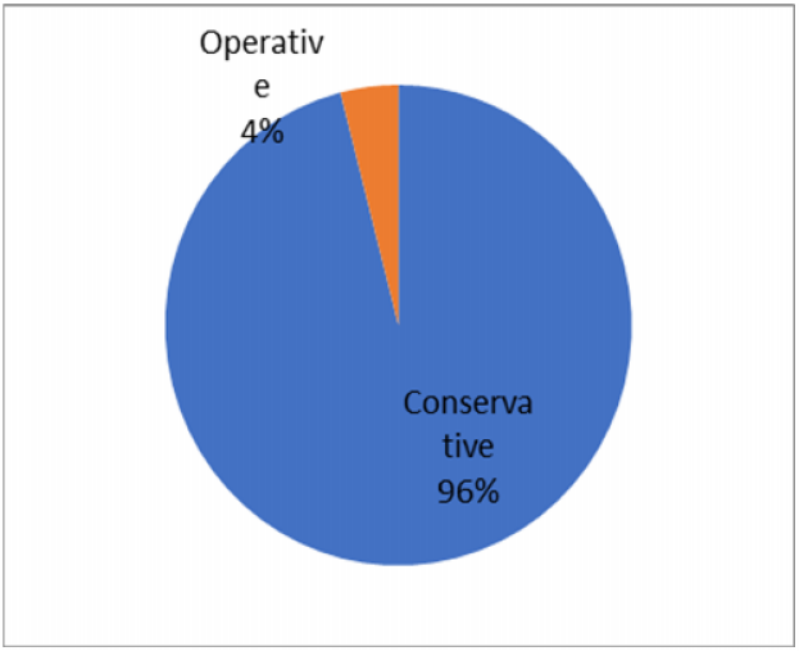
ETIOLOGY

Etiology	Frequency	Percent
Alcohol	75	75.0%
Gall stone	6	6.0%
Idiopathic	19	19.0%
Total	100	100.0%



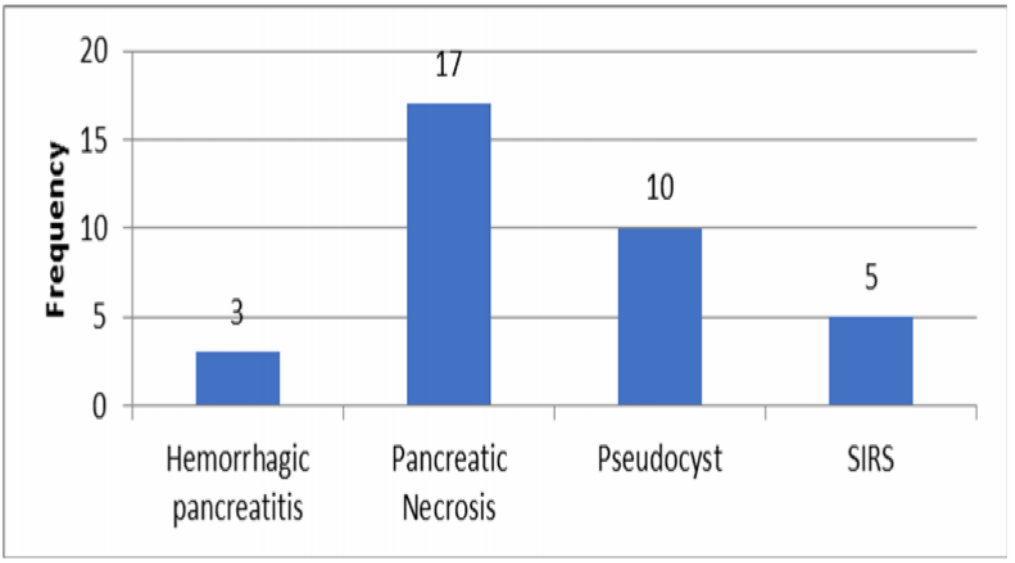
TREATMENT

Treatment	Frequency	Percent
Conservative	96	96.0%
Operative	4	4.0%
Total	100	100.0%



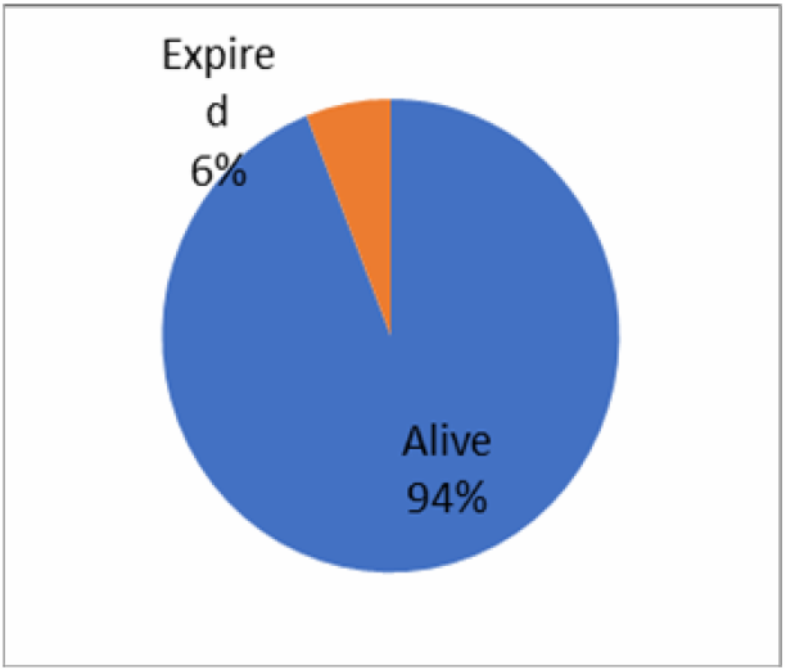
COMPLICATIONS

Complications	Frequency	Percent
Hemorrhagic pancreatitis	3	8.6%
Pancreatic Necrosis	17	48.6%
Pseudocyst	10	28.6%
SIRS	5	14.3%
Total	35	100.0%



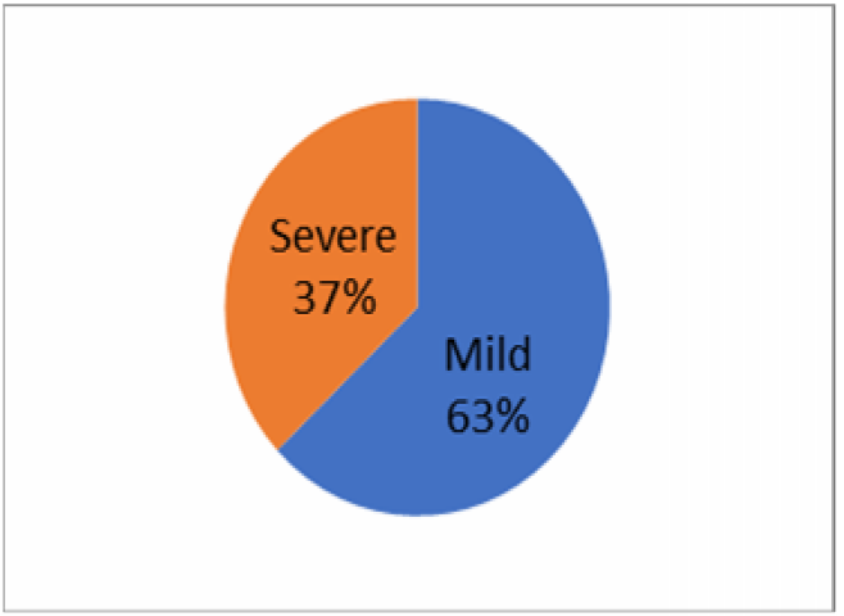
MORTALITY

Mortality	Frequency	Percent
Alive	94	94.0%
Expired	6	6.0%
Total	100	100.0%



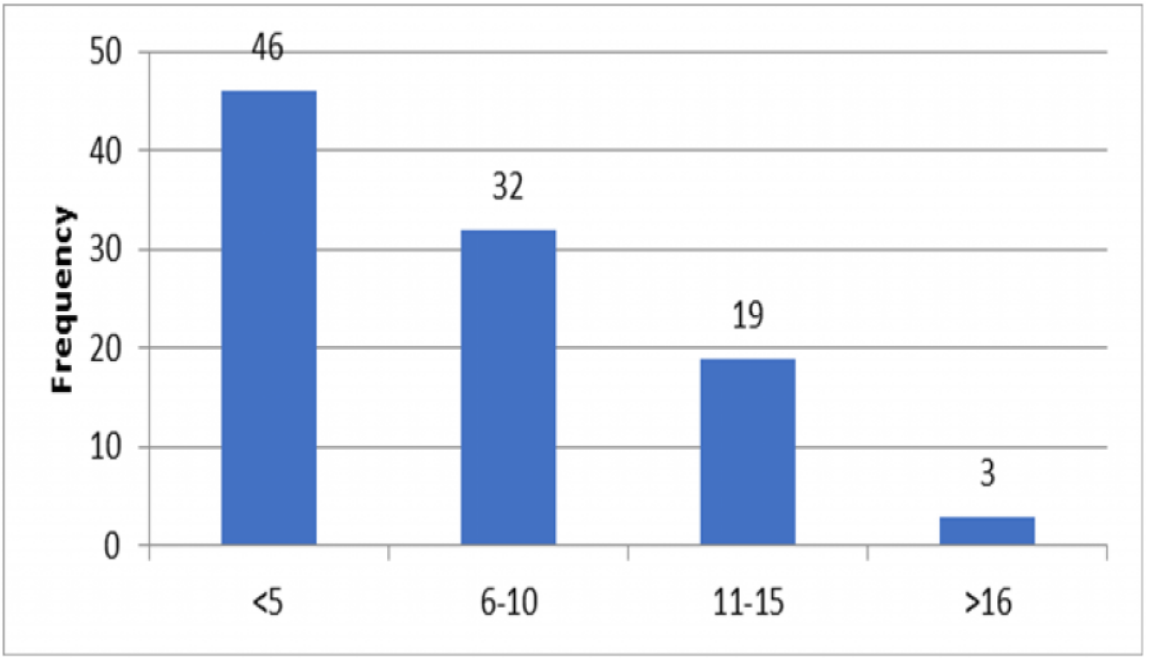
BASED ON ATLANTA CLASSIFICATION

Atlanta Classification	Frequency	Percent
Mild	63	63.0%
Severe	37	37.0%
Total	100	100.0%



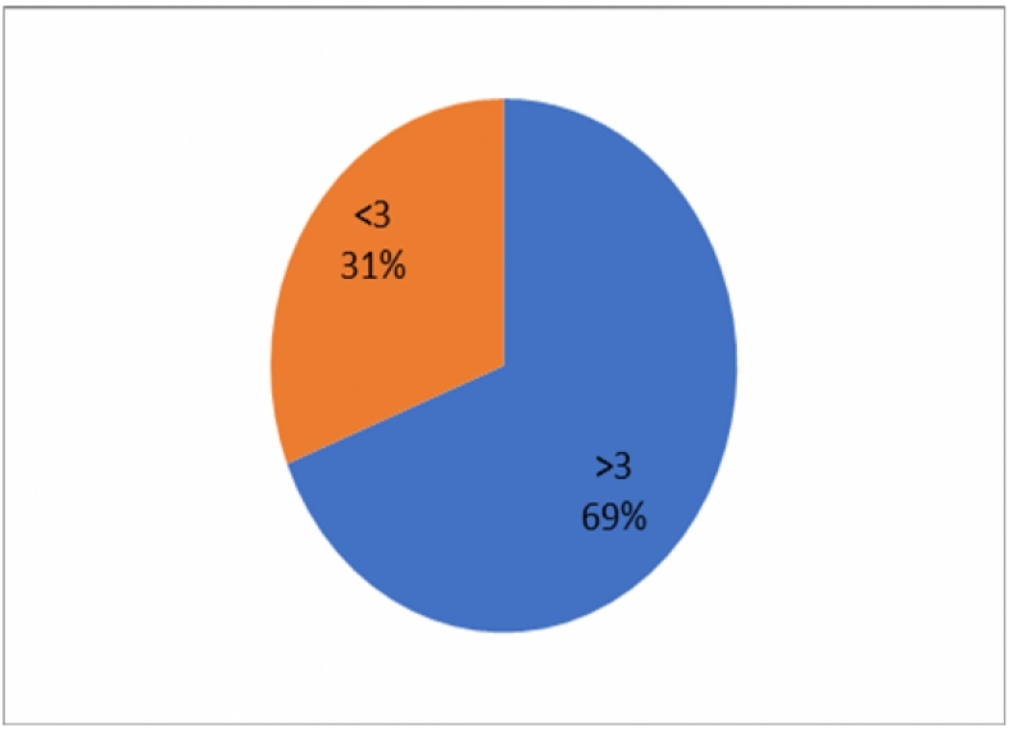
DURATION OF STAY

Duration Of Hospital Stay In Days	Frequency	Percent
<5	46	46.0%
6-10	32	32.0%
11-15	19	19.0%
>16	3	3.0%
Total	100	100.0%

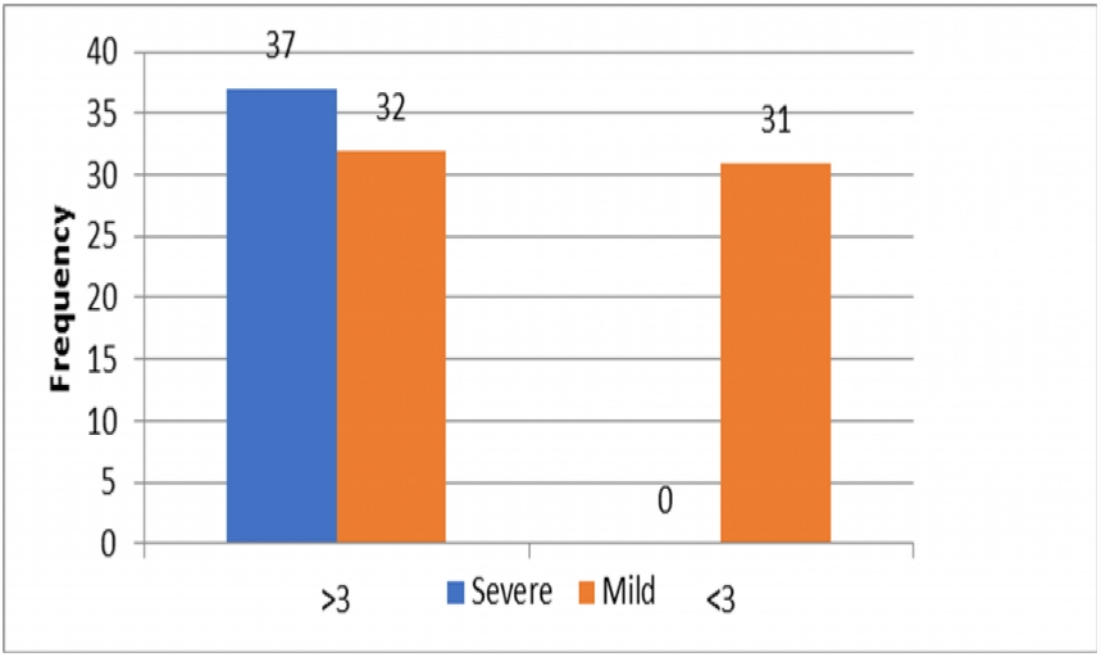


RANSON SCORE

Ranson Score	Total
>3	69
<3	31
Total	100

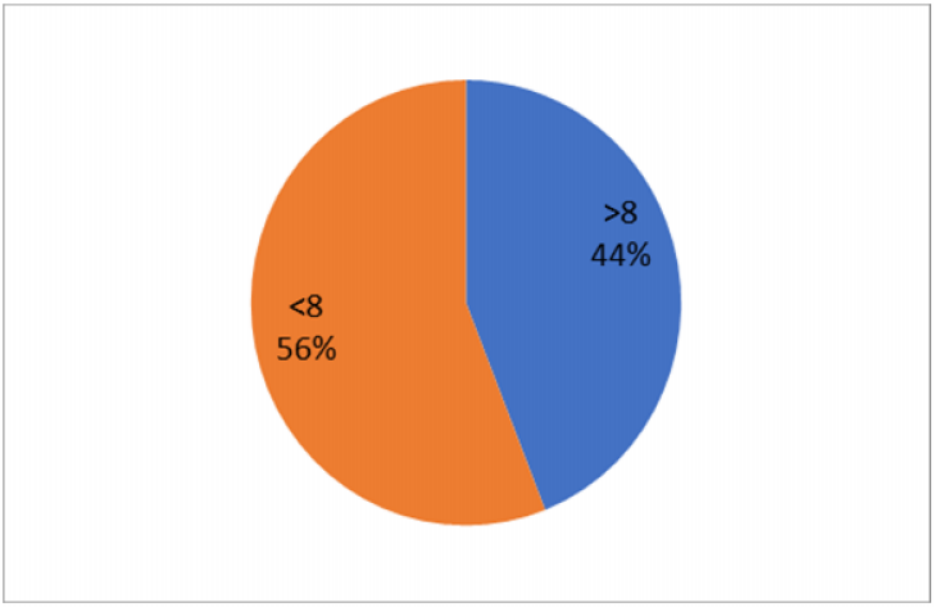


Ranson Score	Atlanta Classification		Total	P value
	Severe	Mild		
>3	37	32	69	<0.0001
<3	0	31	31	
Total	37	63	100	
Sensitivity	**Specificity**	**PPV**	**NPV**	**Accuracy**
100.00%	**49.21%**	**53.62%**	**100.00%**	**68.00%**

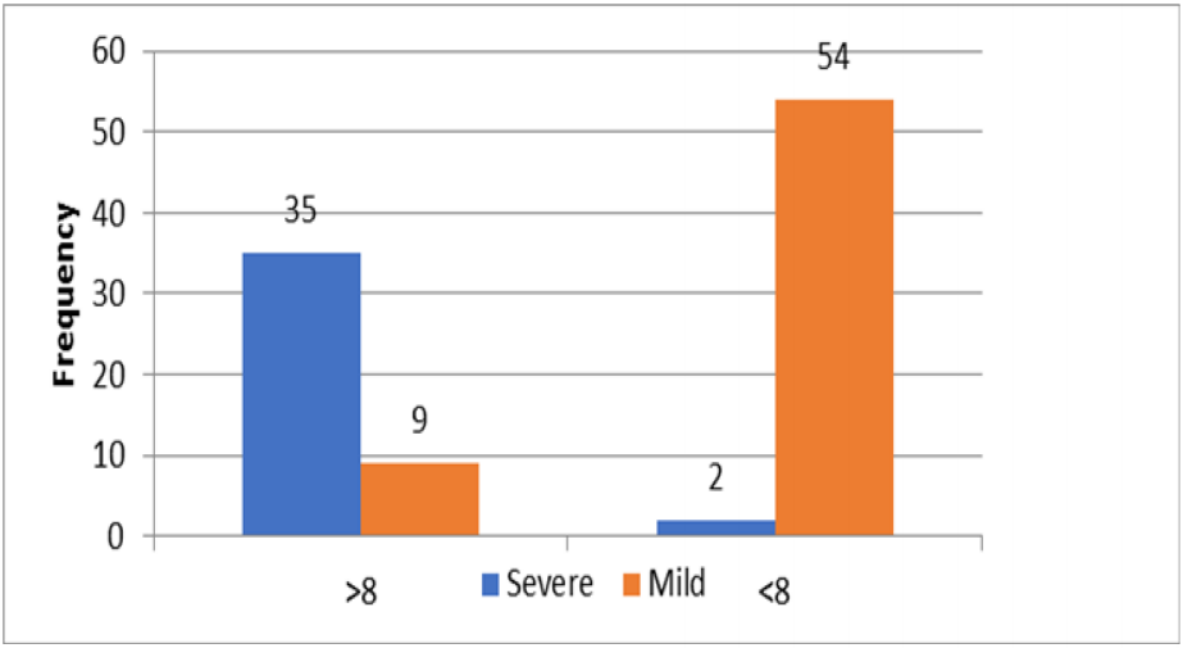


APACHE II SCORE

APACHE Score	Total
>8	44
<8	56
Total	100

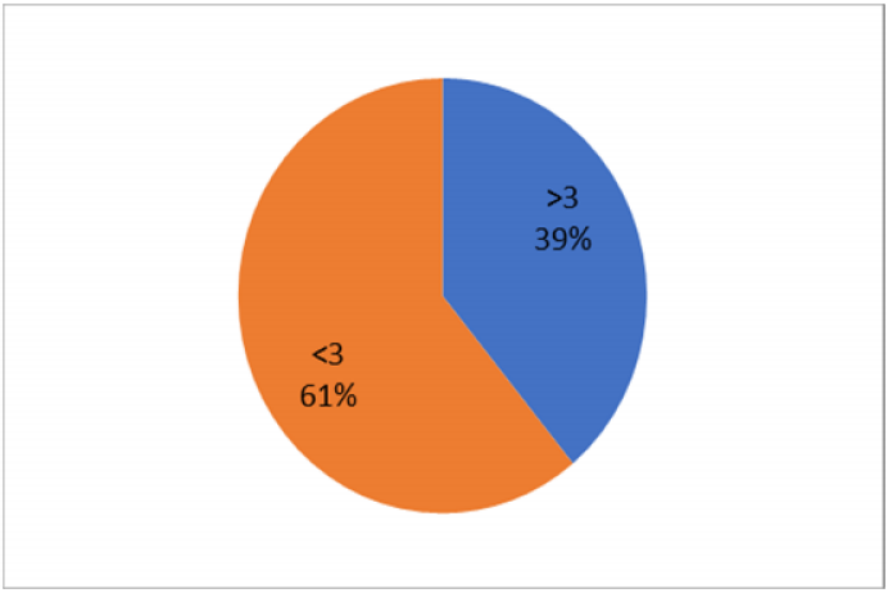


APACHE Score	Atlanta Classification		Total	P value
	Severe	Mild		
>8	35	9	44	0.065
<8	2	54	56	
Total	37	63	100	
Sensitivity	**Specificity**	**PPV**	**NPV**	**Accuracy**
94.59%	**85.71%**	**79.55%**	**96.43%**	**89.00%**

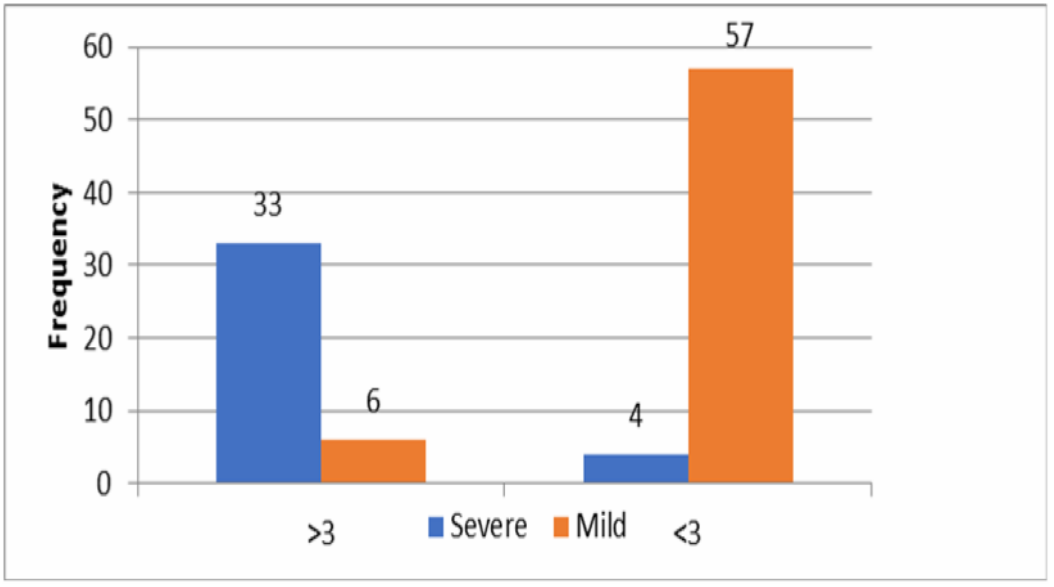


BISAP SCORE

BISAP Score	Total
>3	39
<3	61
Total	100



BISAP Score	Atlanta Classification		Total	P value
	Severe	Mild		
>3	33	6	39	0.754
<3	4	57	61	
Total	37	63	100	
Sensitivity	**Specificity**	**PPV**	**NPV**	**Accuracy**
89.19%	**90.48%**	**84.62%**	**93.44%**	**90.00%**



COMPARISION OF RANSON APACHE AND BISAP SCORES

	RANSON	APACHEII	BISAP
SENSITIVITY	100%	94.59%	89.19%
SPECIFICITY	49.21%	85.71%	90.48%
PPV	53.62%	79.55%	84.62%
NPV	100%	96.43%	93.44%
ACCURACY	68.00%	89.00%	90.00%

DISCUSSION

Acute pancreatitis being a disease of varying severity ranging from mild to moderate and sever disease. The in hospital mortality of the disease raises a concern. For the need of a simple assessment tool for evaluation of severity of pancreatitis. Many different scoring systems have been devised for the assessment of severity of acute pancreatitis, which are divided into two types : The first type attempts to correlate laboratory and clinical markers specific to pancreatitis with subsequent outcome and disease severity. The most widely used in this group is Ranson's Score. The second type of scoring system is the application of non specific physiological scoring system. Which was originally created for use in general population of critically ill patients like APACHE II scores. While bisap uses simple bedside indices which can readily identify patient's severity of disease at the the time of admission.

Ideal predicting criteria should be simple, non-invasive, accurate and quantitative. The assessment tests should be readily available at the time of diagnosis.In this study we compare the classical Ranson's scoring system with the more cumbersome APACHE II scoring system with the simple BISAP scoring system. We have classified the severity of acute pancreatitis in this study based on the Atlanta criteria.

Based on results of the study

- Acute pancreatitis was found to be 9 times more common in males cvompared to females.
- Age group 31-40 had (35 patients) more number of patients.
- Alcohol was found to be the most common cause 0f acute pancreatitis affecting 75 patients followed by gall stones.
- Most of the patients were conservatively managed only a few
- requiring surgical interventions.
- The most common complication of acute pancreatitis was
- pancreatic necrosis(48.6%) followed by pseudocyst (28.6%) and finally SIRS (14.3%).

CONCLUSION

- Of all the three test RANSON's was found to be the most
- sensitive but with the lowest specificity but BISAP showing a good sensitivity of 89.19%.
- Comparing the three scoring systems BISAP was found to be the most specific with a specificity of around 90%.
- On calculating the predictive values BISAP had a better negative predictive value of 93.44% compared to others..
- While calculating the accuracy of each test BISAP stands out to be the most accurate of all at 90% which makes it a valuable tool in assessing organ failure and mortality in acute pancreatitis.
- As RANSON's and APACHEII score needs to be calculated both at the time of admission and at 48 and 24 hrs respectively and is cumbersome while BISAP being a simple easy and involves simple lab investigation with overall better sensivity specificity and accuracy could be used as stratification tool in cases of acute pancreatitis.

LIMITATIONS

- Smaller sample size.(100)
- The most common etiology of this study was found to be alcohol which contraindicates with the common etiology mentioned in standard texts. Hence it might not be appropriate to compare both.
- The glasgow coma scale used for assessing the mental status of the patient has a limitation due to inter observer variation.
- Pancreatitis has a variable disease progression which does may affect the predictability of scoring systems used.
- Since the three scoring systems employs different variables in predicting the outcome of disease there could not be a standardised scoring system developed.
- Patients with acute pancreatitis present to the hospital at varied time with varied presentation. Hence calculation of different scores at different time of presentation might alter the score and outcome the disease.

BIBILIOGRAPHY

1. Zheng Y,Zhou Z., Li H.,et al .A multicenter study on etiology of acute pancreatitis in Beijing during 5 years .Journal of pancreas .2015;44(3):409-414.
2. Lixin Yang,Jing Liu,Yun Xing ,Lichuan Du,Jing Chen ,Xin Liu,Jianyu Hao Comparison of BISAP,Ranson,MCTSI,and APACHE II in prediction severity and prognoses of hyperlipidemic acute pancreatitis in chinese patients.Gastroentrol Res Pract 2016.
3. Sidra Shabbir,Shabana Jamal,Tanwir Khaliq,Zainab Mahsal Khan.Comparison of BISAP Score with Ranson s score in determining the severity of acute pancreatitis.Journal of the college of physicians and surgeons Pakistan 2015 ,Vol 25(5)328-331.
4. Chen L,Lu G ,Zhou Q,Zhan Q.Evaluation of the BISAP score in predicting severity and prognoses of acute pancreatitis in chinese patients.Int Surg 2013;98:6-12.
5. Park JY ,Jeon TJ,Ha TH,Bedside index for severity in acute pancreatitis :comparison wity other scoring systems in predicting severity and organ filure.Hepatobiliary Pancreas Dis Int .2013;12:645-650.
6. Subash Chandra ,Aravind Murali,Adrian Holm.,The bedside index for severity in Acute pancreatitis :a systemic review of prospective studies to determine predictive performance.J Community Hosp Intern Med Perspect.2017 Oct ;7(4);208-213.

7. Vasudevan S,Goswami P,Sonika U,ThakurB,Sreenivas V,Saraya A.Comparison of various scoring systems and biochemical markers in predictiong the outcome in acute pancreatitis.Pancreas .2018 Jan 47(1):65 - 71.

8. Hagjer S ,Kumar N .Evaluation of the BISAP scoring system in prognostication of acute pancreatitis-A prospective observational study.Int J Surg 2018 Jun ;5476-81.

9. Senapati D,Debata PK,Jenasamant SS,Nayak AK,Gowda S M,Swain NN.A prospective stud of the bedside index for severity in acute pancreatitis (BISAP) score in acute pancreatitis:an Indian perspective.Pancreatology 2014 sept –oct ;14(5):335-9.

10. Arif A,Jaleel F ,Rashid K .Accuracy of BISAP score in prediction of severe acute pancreatitis.Pak J Med Sci .2019;35(4):1008-1012.

11. Bailey and Loves Short practice of surgery 27th edition .

12. Sabiston Textbook of surgery 20th edition.

13. Juan Enrique Dominguez Clinical Pancreatology for practicing gastroenterologists and surgeons.

14. Sleisenger and Fordtrans Gastrointestinal and liver disease 10th edition.

15. Netters gastroenterology by Martin H.Floch 3rd edition.

PROFORMA

NAME :

DATE OF ADMISSION :

AGE :

DATE OF DISCHARGE :

SEX :

ADDRESS :

IP NO. :

OCCUPATIO :

CHIEF COMPLAINTS :

HISTORY OF ALCOHOL

INTAKE :

HISTORY OF DRUG INTAKE

PAST HISTORY :

GENERAL EXAMINATION:

PULSE :

BP :

RESPIRATORY RATE :

EXAMINATION OF ABDOMEN

INVESTIGATIONS :

CBC :

TC :

DC :

RANDOM BLOOD SUGAR

BLOOD UREA :

SERUM CREATININE :

SERUM AMYLASE :

SERUM LIPASE :

TOTAL BILIRUBIN

DIRECT :

INDIRECT :

SGOT :

SGPT :

CHEST XRAY :

USG ABDOMEN :

Pao2

FiO2

PaO2/FiO2 :

MASTER CHART

S. No.	Ip No.	Name	Age	Gender	Etiology	Treatment	Duration Of Hospital Stay In Days	Complications	Mortality	Atlanta Classification	Ranson Score	APACHE Score	BISAP Score
1	92667	Baskaran	36	male	Alcohol	Conservative	6		Alive	Mild	2	1	2
2	92541	Egambaram	45	male	Gall stone	Conservative	3		Alive	Mild	3	6	2
3	92592	Pooja	40	female	Alcohol	Conservative	7	Pseudocyst	Alive	Severe	4	14	3
4	499	Thirumal	26	male	Alcohol	Conservative	6	Hemorrhagic pancreatitis	Alive	Severe	5	14	3
5	1454	Vijai	30	male	Alcohol	Conservative	3		Alive	Mild	2	1	1
6	1866	Ajith	30	male	Idiopathic	Conservative	4	Pseudocyst	Alive	Severe	4	9	2
7	2004	Kumar	65	male	Gall stone	Conservative	5	SIRS	Expired	Severe	5	17	5
8	2301	Vadivel	22	male	Idiopathic	Conservative	6		Alive	Mild	2	6	1
9	2615	Ashok kumar	62	male	Alcohol	Conservative	6	Pseudocyst	Alive	Severe	5	11	2
10	2938	Munisami	25	male	Alcohol	Conservative	24	Pancreatic Necrosis	Alive	Severe	5	13	4

11	2427	Baskar	40	male	Alcohol	Conservative	10	Pancreatic Necrosis	Alive	Severe	5	14	3
12	3182	Krishnamurti	47	male	Alcohol	Conservative	7		Alive	Mild	3	8	1
13	2416	Murugan	44	male	Idiopathic	Conservative	3		Alive	Mild	3	6	2
14	1977	Parvathammal	38	female	Idiopathic	Conservative	2		Alive	Mild	3	5	2
15	3366	Murugan	27	male	Alcohol	Conservative	3		Alive	Mild	3	4	1
16	3737	Prabhakaran	27	male	Alcohol	Conservative	10		Alive	Mild	2	4	2
17	3140	Ushmann	50	male	Gall stone	Conservative	14		Alive	Mild	2	6	2
18	3876	Senthil kumar	60	male	Idiopathic	Conservative	13	Pancreatic Necrosis	Alive	Severe	5	10	4
19	1193	Rajeshwari	50	female	Idiopathic	Conservative	5		Alive	Mild	3	6	1
20	5224	Gnanaprakash	32	male	Alcohol	Conservative	7		Alive	Mild	3	4	2
21	5079	Gnanapathi	28	male	Alcohol	Conservative	10		Alive	Mild	4	9	2
22	5818	Narayanan	25	male	Alcohol	Conservative	8		Alive	Mild	1	3	2
23	4954	Dhinakaran	26	male	Alcohol	Conservative	24	Pseudocyst	Alive	Severe	3	8	2
24	6163	Raja	48	male	Alcohol	Conservative	4		Alive	Mild	1	3	1
25	6880	Arjunan	35	male	Alcohol	Conservative	2	Pancreatic Necrosis	Alive	Severe	5	12	3

26	6014	Loganathan	35	male	Alcohol	Conservative	5		Alive	Mild	3	7	1
27	7698	Saravanan	34	male	Alcohol	Conservative	13		Alive	Mild	2	4	2
28	9640	Parandhaman	40	male	Alcohol	Operative	10	Hemorrhagic pancreatitis	Alive	Severe	4	11	3
29	10099	Srinivasan	45	male	Idiopathic	Conservative	3		Alive	Mild	3	7	1
30	11295	Mani	37	male	Alcohol	Conservative	11	Pancreatic Necrosis	Alive	Severe	4	12	3
31	12203	Sathish	38	male	Alcohol	Conservative	3		Alive	Mild	3	6	2
32	12813	Paari	27	male	Alcohol	Conservative	9	Pancreatic Necrosis	Alive	Severe	3	7	3
33	12708	Venkatesan	27	male	Idiopathic	Conservative	4		Alive	Mild	2	4	2
34	15443	Settu	50	male	Gall stone	Operative	12	SIRS	Alive	Severe	3	8	3
35	15374	Iyyapan	59	male	Alcohol	Conservative	3		Alive	Mild	1	4	2
36	15534	Velu	50	male	Alcohol	Conservative	1	Pancreatic Necrosis	Expired	Severe	3	8	4
37	15262	Tirumal	32	male	Alcohol	Conservative	8		Alive	Mild	3	5	2
38	16441	Nithyakumar	28	male	Alcohol	Conservative	7		Alive	Mild	4	9	2
39	17636	Mumtaj	25	female	Idiopathic	Conservative	8		Alive	Mild	2	5	3

40	18115	Ganeshkumar	26	male	Alcohol	Conservative	22	Pseudocyst	Alive	Severe	3	8	2
41	18043	Lakshmi narayanan	48	male	Alcohol	Conservative	5		Alive	Mild	2	4	2
42	20009	Janakiraman	60	male	Alcohol	Conservative	7	Pancreatic Necrosis	Alive	Severe	5	12	4
43	19438	Srinivasan	35	male	Alcohol	Conservative	4		Alive	Mild	3	8	3
44	20592	Perumal	34	male	Alcohol	Conservative	13		Alive	Mild	3	5	2
45	22938	Venkatesan	42	male	Alcohol	Conservative	11	Pseudocyst	Alive	Severe	4	11	4
46	23172	Lakshmi narayanan	45	male	Idiopathic	Conservative	2		Alive	Mild	4	6	1
47	26949	Muraludharan	37	male	Alcohol	Conservative	12	Pancreatic Necrosis	Alive	Severe	4	11	3
48	27159	Mohanraj	38	male	Alcohol	Conservative	4		Alive	Mild	3	5	2
49	26856	Sabari	32	male	Alcohol	Conservative	3		Alive	Mild	3	4	2
50	21530	Aruldoss	27	male	Alcohol	Conservative	12	Pancreatic Necrosis	Expired	Severe	4	11	4
51	27583	Ramu	39	male	Alcohol	Conservative	4		Alive	Mild	2	6	2
52	28232	Muraludharan	50	male	Idiopathic	Conservative	13	SIRS	Alive	Severe	5	11	3
53	27911	Vimal	45	male	Gall	Operative	15		Alive	Mild	3	7	3

					stone								
54	29413	Murugan	36	male	Alcohol	Conservative	6		Alive	Mild	3	5	2
55	30529	Kumaran	29	male	Alcohol	Conservative	13	Pancreatic Necrosis	Alive	Severe	4	9	4
56	30335	Iyyapan	40	male	Alcohol	Conservative	7		Alive	Mild	2	5	2
57	30241	Mani	32	male	Alcohol	Conservative	11		Alive	Severe	3	8	3
58	31091	Iyyapan	44	male	Alcohol	Conservative	4		Alive	Mild	2	5	1
59	32898	Akbar ali	35	male	Alcohol	Conservative	12	Pancreatic Necrosis	Alive	Severe	5	11	4
60	32207	Kowsalya	37	female	Idiopathic	Conservative	5		Alive	Mild	3	7	1
61	33583	Thangavel	30	male	Alcohol	Conservative	1	Pancreatic Necrosis	Expired	Severe	4	12	4
62	33727	PARTHBAN	41	male	Alcohol	Conservative	14	pseudocyst	Alive	Severe	4	11	3
63	36903	SANJAY	49	male	Idiopathic	Conservative	3		Alive	Mild	3	8	2
64	36467	BALAGANESH	35	male	Alcohol	Conservative	13		Alive	Severe	4	10	3
65	36467	BALA GANESH	38	male	Alcohol	Conservative	2		Alive	Mild	2	7	1
66	40609	BASKAR	28	male	Idiopathic	Conservative	5		Alive	Mild	3	6	2
67	41432	SENDHIL KUMAR	50	male	Alcohol	Conservative	4		Alive	Mild	2	5	2

68	41459	MUNISAMY	65	male	Alcohol	Conservative	5		Alive	Mild	1	6	2
69	41608	RAJESH	42	male	Idiopathic	Conservative	6		Alive	Mild	2	7	3
70	43467	SAKTHIVEL	60	male	Alcohol	Operative	8	Hemorrhagic pancreatitis	Alive	Severe	3	9	4
71	44386	GANGADHARAN	32	male	Alcohol	Conservative	6		Alive	Mild	2	5	2
72	45205	SATHISH KUMAR	29	male	Alcohol	Conservative	7		Alive	Mild	3	6	2
73	43330	PREMKUMAR	30	male	Alcohol	Conservative	14	Pancreatic necrosis	Alive	Severe	3	8	3
74	45589	KOWSALYA	41	F	Gall stones	Conservative	10		Alive	Mild	3	8	2
75	44303	MOHAN	40	male	Alcohol	Conservative	12	Pancreatic necrosis	Alive	Severe	4	12	4
76	46737	BABU	38	male	Alcohol	Conservative	3		Alive	Mild	2	5	2
77	47537	MURUGAN	50	male	Alcohol	Conservative	5		Alive	Mild	3	5	2
78	50062	SELVAM	62	male	Alcohol	Conservative	8	pseudocyst	Alive	Severe	3	7	4
79	50129	ANANDHAN	34	male	Alcohol	Conservative	3		Alive	Mild	2	8	3
80	50744	SHALU AMEETH	35	male	Idiopathic	Conservative	5		Alive	Mild	1	7	2

81	50987	VINAYAGAM	45	male	Alcohol	Conservative	6		Alive	Mild	2	6	1
82	49604	SEKAR	62	male	Alcohol	Conservative	9	Pancreatic Necrosis	Alive	Severe	4	12	5
83	52612	RAMESH	45	male	Alcohol	Conservative	10	Pancreatic Necrosis	Alive	Severe	3	9	4
84	54076	SEKAR	38	male	Alcohol	Conservative	4		Alive	Mild	2	6	2
85	55537	RAJENDIRAN	60	male	Idiopathic	Conservative	3		Alive	Mild	2	5	1
86	56280	MURUGAN	54	male	Idiopathic	Conservative	4		Alive	Mild	3	6	2
87	55340	MANI	32	male	Alcohol	Conservative	5		Alive	Mild	2	4	2
88	55588	RAJENDHIRAN	30	male	Alcohol	Conservative	7		Alive	Mild	3	7	2
89	56883	THIRUNAVUKARASU	28	male	Alcohol	Conservative	8		Alive	Mild	3	8	2
90	57155	SANKAR	42	male	Alcohol	Conservative	3		Alive	Mild	2	6	1
91	57891	SATHISHKUMAR	45	male	Alcohol	Conservative	10	Pseudocyst	Alive	Severe	3	13	3
92	57589	ELANGOVAN	37	male	Alcohol	Conservative	5		Alive	Mild	2	5	1
93	59041	VENKATESAN	39	male	Alcohol	Conservative	6		Alive	Mild	3	8	3
94	57343	BABU	50	male	Alcohol	Conservative	11	Pseudocyst	Alive	Severe	4	11	4

95	48013	ASHOK KUMAR	47	male	Alcohol	Conservative	4		Alive	Mild	3	6	2
96	57645	INBARASAN	44	male	Alcohol	Conservative	5		Alive	Mild	3	4	1
97	57444	KUMAR	28	male	Idiopathic	Conservative	3		Alive	Mild	2	5	1
98	59541	PRABHAKARAN	40	male	Alcohol	Conservative	3		Alive	Mild	1	4	1
99	60654	ABDUL SAMATH	35	male	Alcoholic	Conservative	1	SIRS	Expired	Severe	4	12	4
100	65464	PALANI	45	male	Alcoholic	Conservative	1	SIRS	Expired	Severe	4	11	4

CONSENT FORM

நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)

ஆய்வு செய்யப்படும் தலைப்பு:
பங்கு பெறுவரின் பெயர்:
பங்கு பெறுவரின் வயது:

		பங்கு பெறுவர் இதனை ✓ குறிக்கவும்
1.	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	☐
2.	நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	☐
3.	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்காள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	☐
4.	இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	☐
5.	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்ட அறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்கிறேன்.	☐

பங்கேற்பவரின் கையொப்பம் / ..... இடம் .....
கட்டைவிரல் ரேகை
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
ஆய்வாளரின் கையொப்பம் /..... இடம் .....
ஆய்வாளரின் பெயர் .....
மையம் .....
கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை
சாட்சியின் கையொப்பம் /..... இடம் .....
பெயர் மற்றும் விலாசம் ....