“COMPARATIVE STUDY OF VARIOUS SCORES IN PREDICTING COMPLICATIONS AND MORTALITY IN ACUTE PANCREATITIS”

DISSERTATION SUBMITTED FOR

BRANCH-I M.S (GENERAL

SURGERY) APRIL 2020

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY

CHENNAI

DECLARATION BY THE CANDIDATE

I **DR.R,RAJARAJAN** here by solemnly declare that this dissertation entitled **"COMPARATIVE STUDY OF VARIOUS SCORES IN PREDICTING COMPLICATIONS AND MORTALITY IN ACUTE PANCREATITIS"** is a bonafide and genuine research work carried out by me. This is submitted to The Tamil Nadu Dr. M.G.R Medical University, Chennai in partial fulfillment of the regulations for the award of MS degree (Branch I) General surgery.

PLACE: Chennai

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DR.R.RAJARAJAN

DECLARATION BY THE GUIDE

This is to certify that this dissertation entitled **"COMPARATIVE STUDY OF VARIOUS SCORES IN PREDICTING COMPLICATIONS AND MORTALITY IN ACUTE PANCREATITIS"**is a bonafide original work of Dr.R.RAJARAJAN in partial fulfillment of the requirement for M.S. Branch-I (General Surgery) examination of the Tamilnadu Dr. M.G.R. Medical University to be held in April 2020. The period of study is from April 2019 to September 2019.

PLACE: Chennai
DATE

Dr.S. VIJAYALAKSHMI, M.S.DGO.,
Department of General Surgery
Kilpauk Medical College,
Chennai- 600010

BONAFIDE CERTIFICATE

Certified that this is the bonafide dissertation done by **DR.R.RAJARAJAN a**nd submitted in fulfillment of the requirements for the Degree of M.S. General Surgery, Branch I of Tamilnadu Dr. M.G.R Medical University, Chennai

Date: Unit Chief

Date: Professor and Head
Department of Surgery

Date: Dean
Kilpauk Medical College

CERTIFICATE – II

This is to certify that this dissertation work titled **"COMPARATIVE STUDY OF VARIOUS SCORES IN PREDICTING COMPLICATIONS AND MORTALITY IN ACUTE PANCREATITIS"** of the candidate DR.R.RAJARAJAN with registration number **221711159** for the award of Masters in Surgery in the branch of Branch I- General Surgery. I personally verified the www.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 **7%** of plagiarism in the dissertation.

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Dr.S. VIJAYALAKSHMI, M.S.DGO.,
Department of General Surgery
Kilpauk Medical College,
Chennai- 600010

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Place: Chennai
Date:

DR.R.RAJARAJAN
Post Graduate Student,
Dept. of General Surgery, KMC, Chennai

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INTRODUCTION

Acute pancreatitis is a typical scenario experienced during routine careful practice and it represents an incredible challenge to the treating specialist. "Acute pancreatitis is characterized as a pancreatic inflammatory procedure, with peripancreatic and multi-organ contribution causing multi-organ dysfunction syndrome (MODS), with increased death rate". Following articulation grossly abridges its consequences.

Acute pancreatitis is a condition in which there is sudden inflammation in pancreas. It ranges from self limiting mild inflammation to life threatening severe disease, the severe acute pancreatitis. Severe acute pancreatitis has been a challenge to surgeons worldwide because of its association with many complications and high mortality rate. The overall mortality in acute pancreatitis is noted in range of five to ten percent. Among them eighty to ninety percent of cases are of mild pancreatitis with a good outcome. The remaining ten to twenty percent of patients are of severe acute pancreatitis and noted to have mortality rate of up to 40%. So, early identification of patients with severe disease is beneficial to anticipate prognosis and complications so patients at risk can be provided adequate monitoring and care.

Many scoring systems has been described over the time to identify severe cases and patients at risk to develop complications and higher mortality by using multiple hematological, biochemical , radiological, clinical and hemodynamic criteria. Three of the commonly used scoring systems are:

1)BISAP (Bedside Index of Severity in Acute Pancreatitis) which considers 5 criteria- blood urea nitrogen (BUN) level, mental status, evidence of Systemic inflammatory response syndrome (SIRS), age, and evidence of pleural effusion ;

2) Ranson's score, which considers 11 criteria- age, blood glucose level, white blood counts, serum lactate dehydrogenase(LDH) levels, serum aspartate aminotransferase(AST) level, decrease in hematocrit levels, increase in blood urea nitrogen, serum calcium levels, partial oxygen pressure(PaO2) levels, base deficit, fluid sequestration;

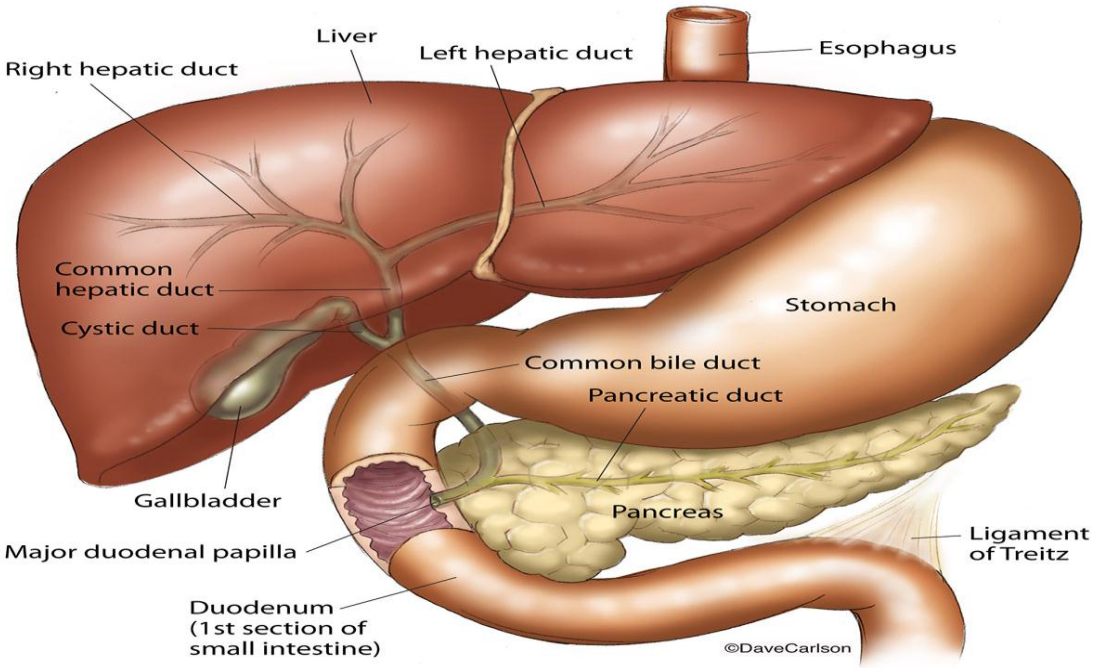
3) Modified CTSI (Computed Tomography Severity Index) which considers 3 aspects of CT findings- pancreatic inflammation, pancreatic necrosis and extra pancreatic complications. Each scoring system has its distinct method of evaluating severity, BISAP scoring system 2 focuses on inflammatory response and its effect, RANSON'S scoring assesses biochemical changes through 48 hour of time period, modified CTSI score assesses severity by anatomical changes like inflammation , pancreatic necrosis. These three scoring system are being compared in present study to find out which of these score is better screening method and accurately predicts mortality , complication such as renal failure, respiratory failure, multiorgan failure, pancreatic necrosis accurately

REVIEW OF LITERATURE

SURGICAL ANATOMY OF PANCREAS:

The **pancreas** is a long, muscular organ, which lies in close proximity with the duodenum. It is enclosed with a thin capsular connective tissue that extends inside as a septa, dividing the gland into lobules. Even though pancreas is mainly an exocrine gland, which secrets range of digestive enzymes, the pancreas also has an endocrine function. Its **pancreatic islets**—clusters of cells which was formerly known as the islets of Langerhans, produce the hormones, insulin, somatostatin, glucagon and pancreatic polypeptide .

Pancreas in relation to the stomach and duodenum.



Parts of pancreas

Head

The head is the most stretched out piece of the pancreas. The head of the pancreas is found in the right side of gut, settled in the bend of the duodenum.

Unicate process

The uncinate is the part of the head of pancreas that bend towards the back of the abdomen. The uncinate snares around two significant veins, the superior mesenteric artery and superior mesenteric vein.

Neck

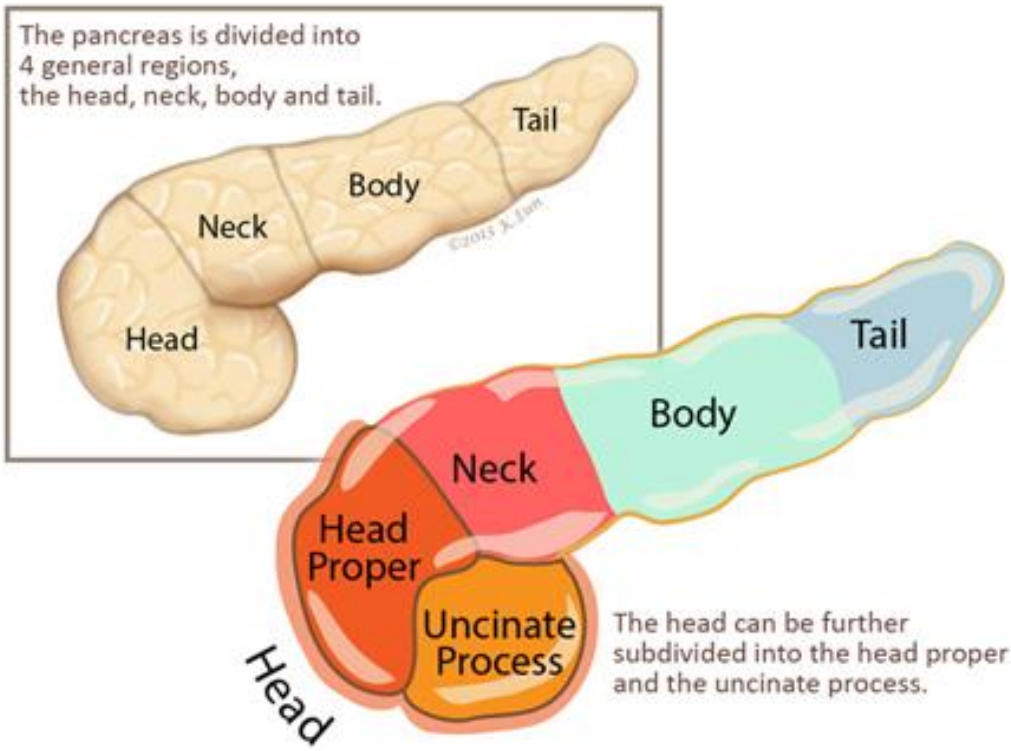
The neck is the thin area of the organ between the head and the body of the pancreas. It is continuation of the head of the pancreas and is arranged anterior to the portal vein formation for example at the intersection of the SMV and splenic vein at the L1 vertebra level. It is the intersection in the middle of the head and body. The neck of the pancreas has close proximation with few significant vessels posteriorly including SMV-portal vein, IVC and aorta

Body

The body is the center part of the pancreas linking the neck and the tail. The superior mesenteric artery and vein run behind this part of the pancreas. This lies behind the distal part of the stomach in between the neck and the tail

Tail

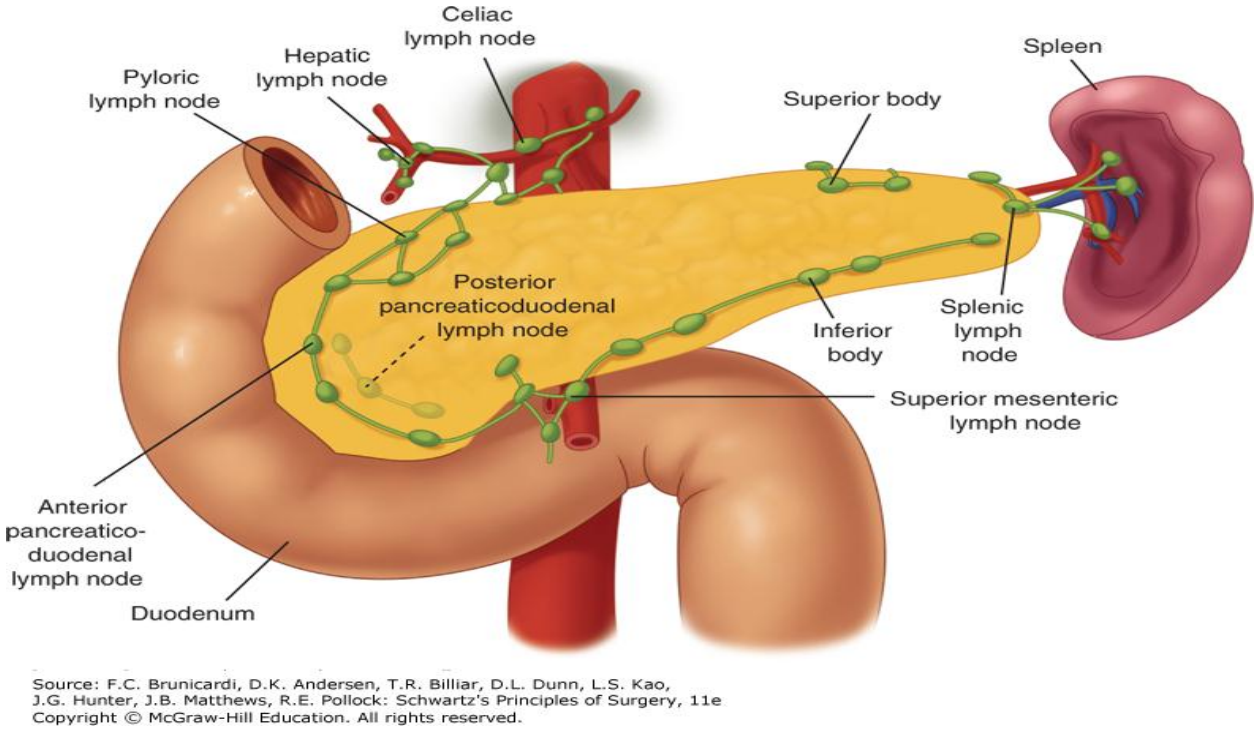
The tail is the slender tip of the pancreas in the left half of the abdomen, in nearness with the spleen. It is situated near the splenic hilum.



Pancreas

The exocrine function of the pancreatic gland which involves the acinar cells secreting digestive enzymes, which is transported to the small intestine via pancreatic duct. Its endocrine functions includes the secretion of insulin which is produced by the beta cells and glucagon which is secreted by alpha cells

within the pancreatic islets . These hormones help to regulate the rate of glucose metabolism in the body.

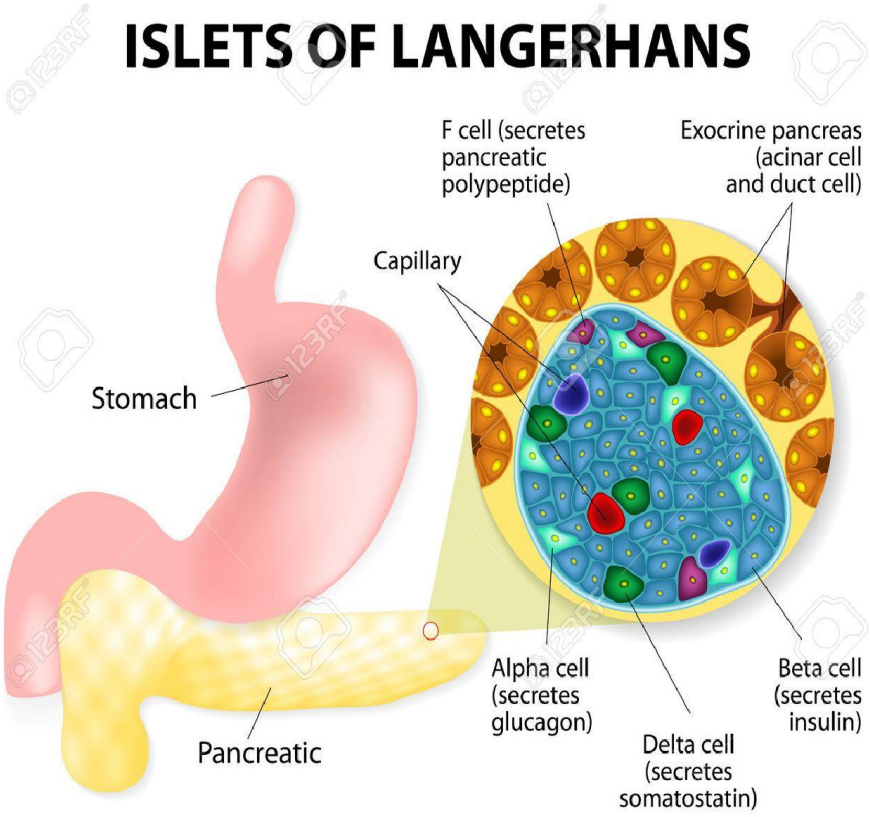


Secretions of the Pancreatic Islets

Pancreatic Islets have four type of cells, which has its own function and importance:

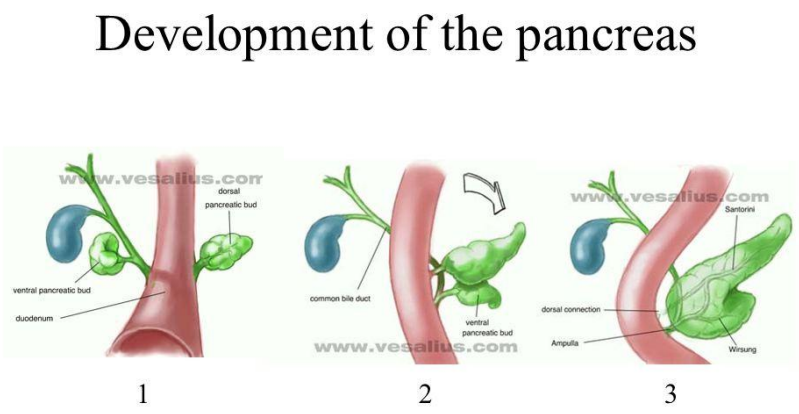
- The alpha cell- secretes the hormone glucagon .his hormones plays an important role in glucose regulation of blood. Glucagon is stimulated by the low level of glucose in the blood.
- The beta cell secretes the hormone insulin. Release of insulin in the blood is stimulated by elevated blood glucose in the blood stream.

- The delta cell secretes the hormone somatostatin which is a peptide. This hormone is an inhibitory hormone, which inhibits the release of both glucagon and insulin.
- The pancreatic polypeptide cell secretes the pancreatic polypeptide hormone. This hormone plays an important role in the appetite, as well as in the maintainace of pancreatic endocrine and exocrine secretions.

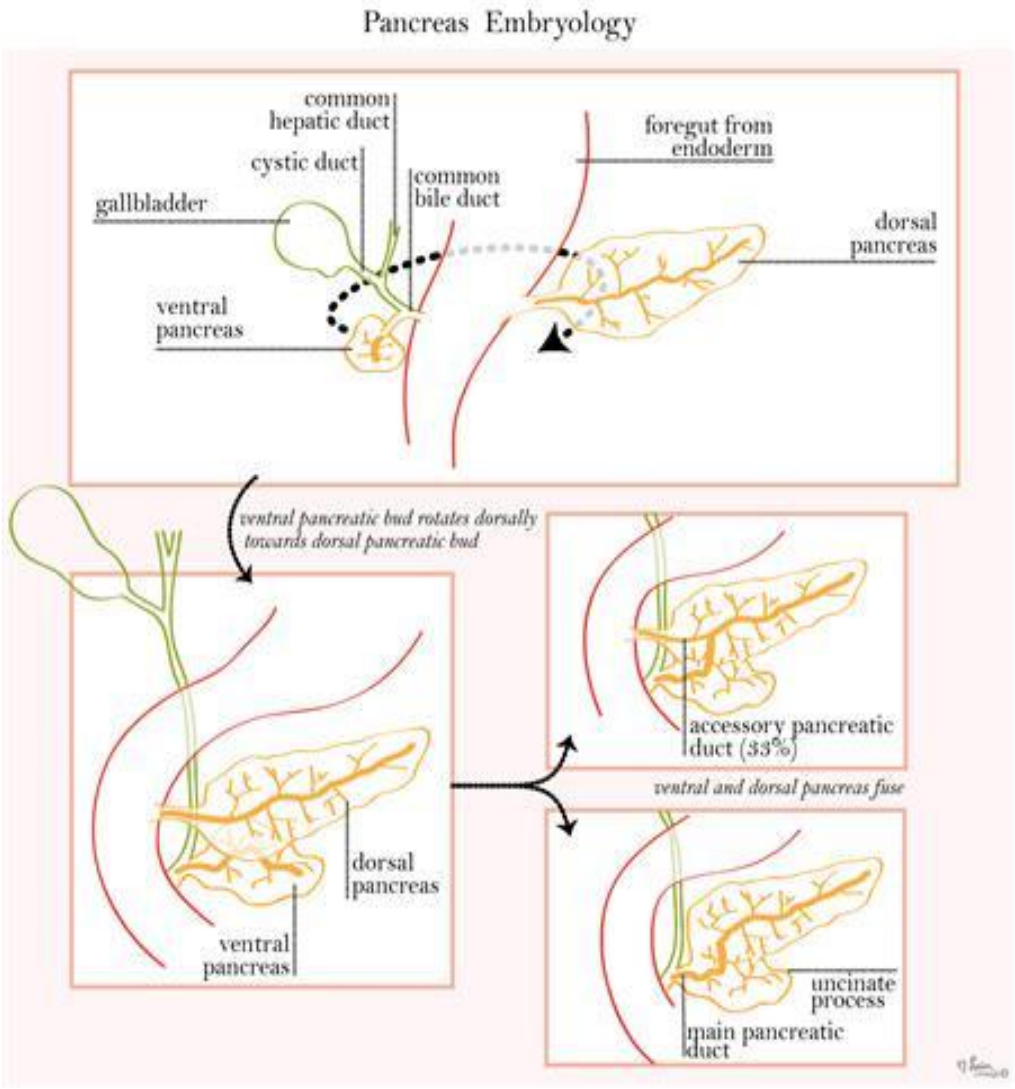


Development of pancreas

During the embryonic development, the pancreas is formed as two buds from the foregut, which is an embryonic tube and is a precursor to the gastrointestinal tract. .Development of Pancreas starts with the development of a dorsal and ventral pancreatic bud. Both the bud joins with the foregut via duct. The dorsal part of the pancreatic bud forms the body,neck and tail of the developed pancreas, however the head and uncinate process is formed by the ventral pancreatic bud.



Stages of the development of the pancreas



Blood supply

Blood supply of pancreas is very rich, with vessels originating as branches of both the coeliac artery and superior mesenteric artery. The splenic artery travel along the upper surface of the pancreas, and give blood supply to the left part of the body and the tail of the pancreas via its pancreatic branches, the longest branch is called the greater pancreatic artery. The superior & inferior pancreaticoduodenal arteries travel along the anterior and posterior margin of

the pancreatic head at its border with the duodenum, which supply the head of the pancreas. These vessels originate and join together in the middle.

The head, neck and body and of the pancreas drain into the the superior mesenteric and portal veins and splenic vein respectively.

ARTERIAL SUPPLY OF PANCREAS:

Major visceral arteries:

- Celiac
- Hepatic
- Splenic
- Gastroduodenal
- Superior mesenteric

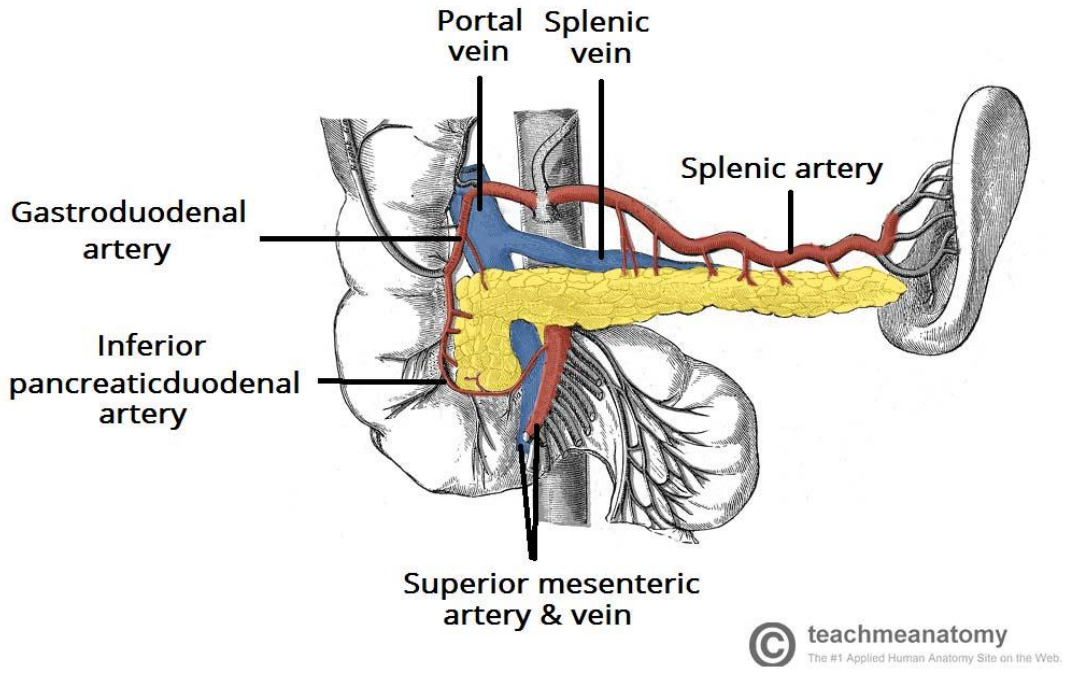
Other pancreatic arteries:

- Dorsal pancreatic
- Right branch of the dorsal pancreatic
- Caudal pancreatic
- Transverse pancreatic

- Anterior and posterior arcade

- Inferior pancreaticoduodenal

ARTERIAL SUPPLY OF PANCREAS



VENOUS DRAINAGE OF PANCREAS:

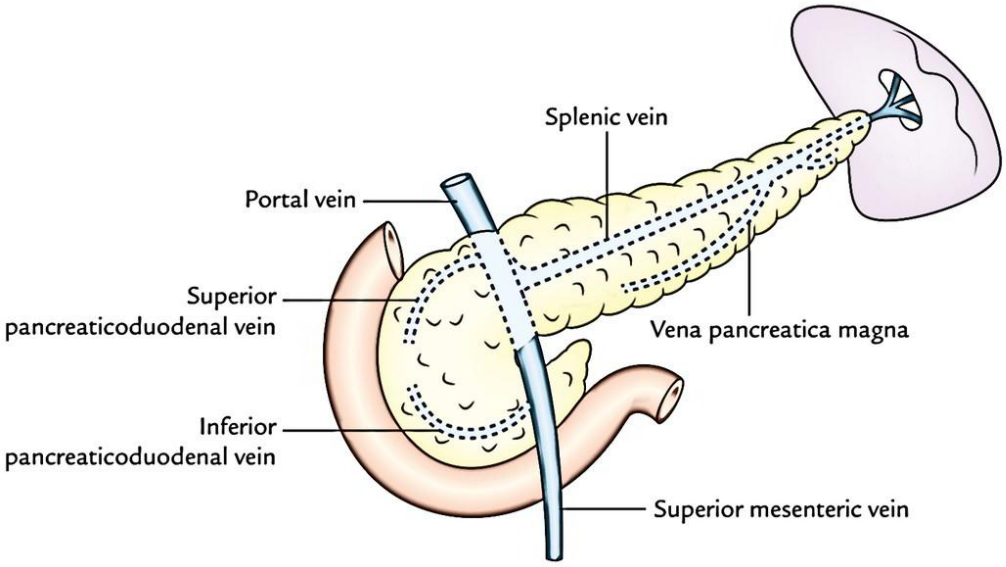
- The body and neck: splenic vein

- The head: superior mesenteric and portal veins

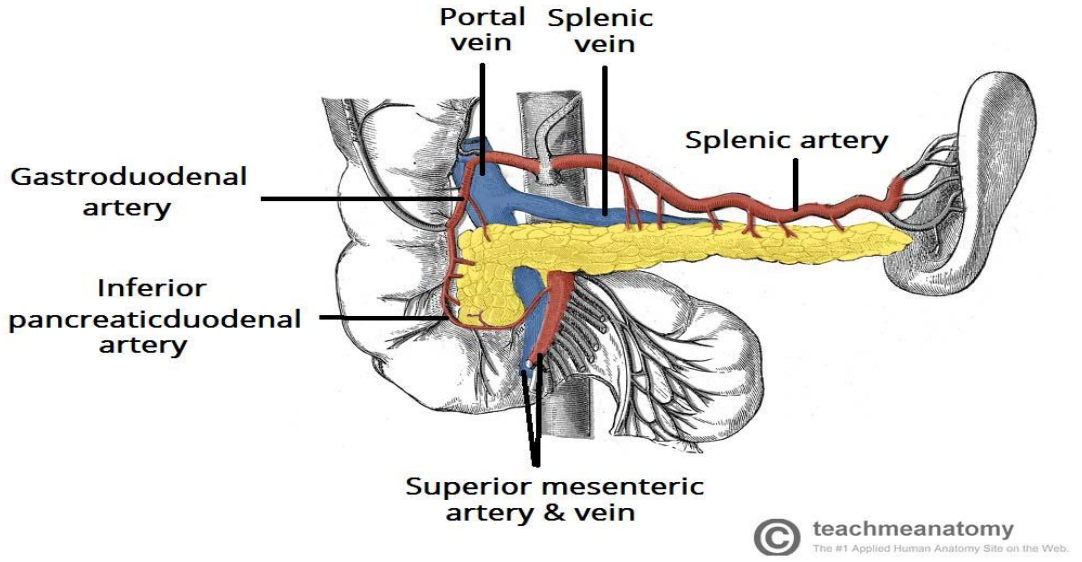
LYMPHATIC DRAINAGE OF PANCREAS:

- Splenic lymph nodes
- Celiac Lymph Nodes
- Superior Mesenteric Lymph Nodes

VENOUS DRAINAGE OF PANCREAS



LYMPHATIC DRAINAGE OF PANCREAS:



Mechanism of Regulation of Blood Glucose Levels by Insulin and Glucagon

Exocrine secretion of pancreas takes place in the time of interdigestive state and digestive state. The stages of secretion that takes place in time of digestive state found to be similar in stomach and pancreas.

During cephalic phase, vagal stimulation of the pancreas occurs by odour or vision of meal. In this stage, acetylcholine, which induce release of enzymes from acinar cell is secreted from terminal endings of postganglionic fibers. 20% to 25% of pancreatic secretion occurs during this stage.

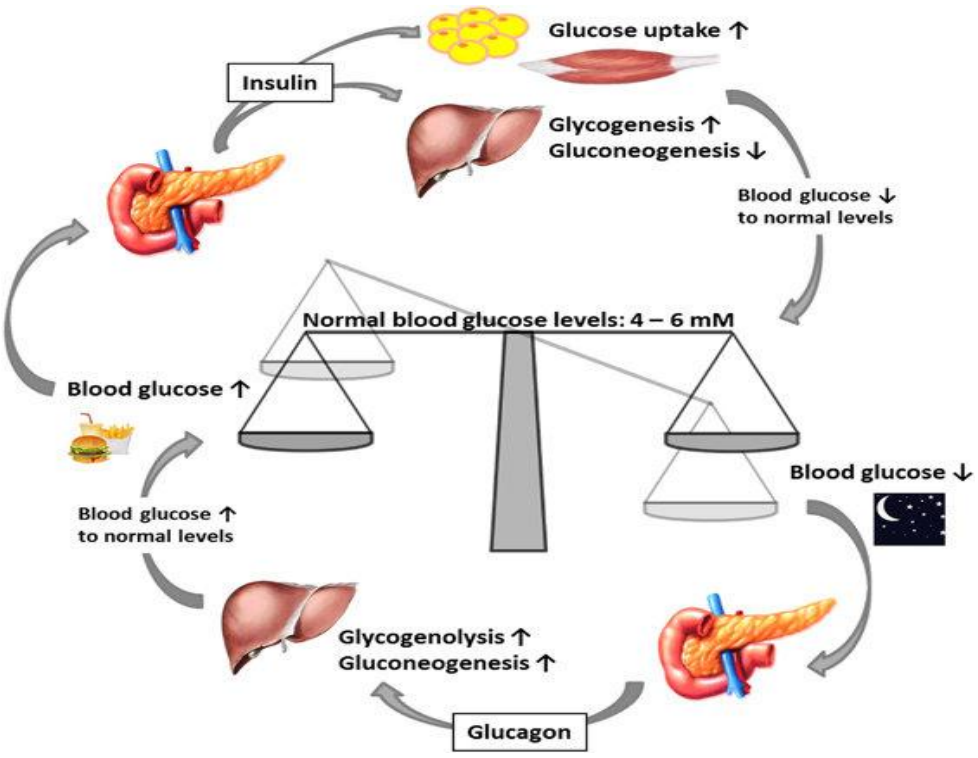
The gastric phase is second phase. In this phase distention of stomach by food causes vasovagal reflex which results in acinar cell secretion. 10% of secretion is because of this phase.

The intestinal phase is third phase, which is responsible for 65% to 70% of pancreatic secretion. It is mediated by secretin and cholecystokinin (CCK**).**

The pancreatic receptors detect the fall in blood glucose levels, in varying situations like during the periods of strenuous exercise or fasting. In response to that alpha cells of the pancreas produce the hormone glucagon which has following actions:

- Glucagon stimulates the liver and it start the process of glycogenolysis , which means liver convert its stores of glycogen back into glucose. Then the glucose is released in the blood streams for use by body cells.
- Glucagon also stimulates the liver for the intake of amino acids from the blood stream and convert them into glucose. This process is known as gluconeogenesis.
- Glucagon stimulates lipolysis, which breaks the stored triglycerides into free fatty acids and glycerol. Free glycerol is released into the bloodstream and is transported to the liver, which is then converted to glucose. This process is also known as gluconeogenesis.

These action together increase the blood glucose levels. Negative feedback mechanism; for the glucagon is the elevated blood glucose levels .



History of acute pancreatitis

In recent publication it has been suggested that Alexander the Great died of acute pancreatitis. History has many such examples to offer that might have been a case of acute pancreatitis. The first clear description if the disease was a published by a Dutch physician and anatomist in 1652. The first systematic analysis of acute pancreatitis was presented by the Virchow in 1889 by the title "Acute pancreatitis is a consideration of, hemorrhagic, suppurative, pancreatic hemorrhage and gangrenous pancreatitis, and of disseminated fat-necrosis" ,

which included details of clinical characteristics of 53 patients. Fitz's stated that "an operation … in the early stages of this disease, is extremely hazardous".This statement was discarded by Fitz in 1903. During the 20th century there were varing theories regarding whether to prefer surgery or conservative treatment. In the 1930s conservative approach was the most preferred approach due to high mortality rates after surgical interventions. During the 1960s and 1970s surgery again generally became more popular, including blunt necrosectomy for necrotizing pancreatitis.

Course of Acute pancreatitis

Acute pancreatitis is the condition of inflammatory changes of the pancreas, with marked involvement of peripancreatic tissues and remote organs. In majority of the cases the disease is mild to moderate, with only interstitial edema, which can be recovered within days or few weeks. Severe pancreatitis is characterized by systemic complications, which have grave co morbidities and even death, in around 15-20% of the patients. Persistent systemic inflammatory response syndrome (SIRS) can lead to death within first week of the disease, symptoms can also include including, tachycardia, pyrexia, leucocytosis and tachypnea with single or multiple organ dysfunction. Late mortality is mostly due to organ dysfunction or systemic infections.

Acute onset of upper abdominal pain with radiation to the back, nausea and vomiting, local peritonitis located in the epigastrium and sometimes an effect on the circulatory system, in combination with elevated pancreatic enzymes in blood or urine, are the typical findings in acute pancreatitis. Upper abdominal pain is, however, characteristic of several other acute disorders such as gastric and duodenal ulcers, cholecystitis, cholangitis, ruptured aortic aneurysm, ileus, and even pneumonia and myocardial infarction. Even if elevated serum pancreatic amylase has a high sensitivity and specificity for acute pancreatitis, a slight rise in serum pancreatic amylase can be seen in the other abdominal conditions mentioned.

Pathogenesis of acute pancreatitis

Primary events

Pancreatic acinar cells produce and discharge digestive enzyme precursors in inactive state into the duodenum. Inactive digestive enzyme precursors like chymotrypsinogen, trypsinogen, procarboxypeptidases A and B, proelastase and prophospholipase A2. Zymogens are produced in the endoplasmic reticulum and then stored in the secretory granules. After the stimulation of the acinar cell, these granules discharge its contents by exocytosis into the acinar lumen and pass through the pancreatic ductal system in the duodenum, where the inactive trypsinogen is converted to trypsin and this is catalyzed by enterokinase . Trypsin is the key enzyme for quick activation of all the proenzymes. There are

two isoenzymes of trypsinogen: trypsinogen-1 and trypsinogen-2.

Among healthy individuals, the ratio of trypsinogen-1 to trypsinogen-2 in pancreatic fluid is fourfold. Trypsinogen activation peptide activates trypsinogen. Owing to their strong proteolytic and lipolytic functions, these secretory enzymes holds a powerful autodigestive capacity.

Secondary events

Secondary events comprise of the release of various inflammatory mediators in the blood stream. A proinflammatory cytokine flow follows acinar cell injury. Local inflammation is the body's first physiological protective response. Later on there is an excessive uninhibited activation of inflammatory cells and mediators, which is clinically recognized as SIRS. Complication of SIRS includes organ system dysfunction, shock, renal failure, acute lung injury and MODS.

AETIOLOGY OF ACUTE PANCREATITIS

Acute pancreatitis has many aetiologies, though majority of all cases are due to either gallstones or alcohol. Etiology of acute pancreatitis varies from different geographical locations like in United Kingdom and Asia the most common cause are gallstones , however in USA and Finland alcohol is the most common etiological factor. The idiopathic causes comprises 10-30% of all cases. Lately, biliary sludge has been the topic of interest, which was found to be in 70% of

patients of acute pancreatitis. Additionally more than 85 drugs have been reported to cause acute pancreatitis. Rarely acute pancreatitis can be brought about by a change in the trypsinogen-1 quality permitting untimely enactment of trypsinogen causing autodigestion of acinar cell.

Etiology of pancreatitis

Well-established causes
- Gallstones
- Alcoholism
- Hypertriglyceridemia
- Post–endoscopic retrograde cholangiopancreatography
- Drug induced
- Autoimmune
- Genetic
- Abdominal trauma
- Postoperative
- Ischemia
- Infections
- Hypercalcemia and hyperparathyroidism
- Posterior penetrating ulcer
- Scorpion venom
- Pancreas divisum with ductular narrowing on pancreatogram
- Idiopathic

Controversial causes
- Sphincter of Oddi dysfunction
- Pancreas divisum without ductal narrowing on pancreatogram
- Microlithiasis/sludge

- **Gallstones**

  Gallstones cause about 40% of instances of pancreatitis . Proposed systems incorporate reflux of poisonous bile into the pancreatic course from transient square of the ampulla during gallstone segment and

pancreatic ductal hypertension from either a stone obstruction at the ampulla or ampullary injury brought about by stone section.

- **Obstructive reasons for pancreatitis**

  Obstructive causes of pancreatitis, in addition to gallstones, incorporate pancreas divisum, sphincter of Oddi stenosis, periampullary tumors, pancreatic malignant growth, parasites, and clumps. Pancreatic malignant growth at times can cause temporary duct obstruction by clots within the pancreatic duct and sometimes can copy constant pancreatitis in light of the fact that chronic malignant obstruction of the pancreatic duct. Intraductal papillary mucinous neoplasm is a rare pancreatic tumor characterized by intraductal proliferation of mucin-producing cells that secrete mucin into the. This tumor regularly gives intermittent episode of acute pancreatitis caused by temporary pancreatic duct obstruction by the excreted highly viscous mucus.

- **Alcoholism**

  Alcoholism is liable for about 35% of instances of intense pancreatitis. The pathophysiology might be multifactorial. Proposed components incorporate sphincter of Oddi fit, precipitation of insoluble protein plugs that block the pancreatic pancreatic ductules, actuation of pancreatic proteases, overstimulation of pancreatic secretion by cholecystokinin.

Alcoholic pancreatitis for the most part requires consuming more than 8 alcoholic drinks/day (100 g/d) for more than 5 years.

- **Hypertriglyceridemia**[47]

  Hypertriglyceridemia causes about 2% of cases of acute pancreatitis . A serum triglyceride level greater than 1000 mg/dL suggests this possible cause, and a triglyceride level greater than 2000 mg/dL is diagnostic . Alcoholic pancreatitis sometimes is associated with an elevated serum triglyceride level caused by acute alcoholism, but this elevation generally is mild and rarely is higher than 1000 mg/dL . The triglyceride level should be measured early after clinical presentation with pancreatitis, because this level tends to decline rapidly during the hospitalization due to fasting, insulin therapy, and restoration of fluid and electrolyte balance. The serum in patients who have hypertriglyceridemia may be opalescent because of increased very low density lipoprotein or milky because of hyperchylomicronemia.

Following criteria to classify serum Triglyceride levels:

- Normal (<150 mg/dl)
- Mild HTG (150-199 mg/dl),
- Moderate HTG (200-999 mg/dl);

- Severe HTG (1000-1999 mg/dl);

- Very severe HTG (≥2000 mg/dl).

- **Drug-induced pancreatitis**

Drugs are liable for about 2% of the pancreatitis. Commonly concerned drugs responsible for the pancreatitis are as follows.

Aminosalicylic acid/sulfasalazine

Azathioprine

Valproic acid

Didanosine

Metronidazole

Isoretinoin

Mercaptopurine

Tamoxifen

Tetracycline

Toxic metabolite Pentamidine

Drug-induced hypertriglyceridemia Thiazides

Overdose reaction Acetaminophen

Erythromycin

- **Iatrogenic:**

  Post-ERCP pancreatitis estimates range from 1.6% to 7% in various studies41,42. young age, biliary sphincter balloon dilation in intact papilla, pancreatic duct contrast injection, normal bilirubin, precut sphincterotomy or pancreatic sphincterotomy, and suspected sphincter of Oddi dysfunction are considered as risk factors for post-ERCP pancreatitis.

Classification of acute pancreatitis

Based on revised Atlanta classification of acute pancreatitis

Mild acute pancreatitis

- No organ failure, local or systemic complications

Moderately severe acute pancreatitis

- Organ failure that resolves within 48 h and/or
- Local or systemic complications without persistent organ failure

Severe acute pancreatitis

- Persistent organ failure > 48 h

Interstitial edematous acute pancreatitis

- Acute inflammation of the pancreatic parenchyma and peri-pancreatic tissues, but without recognizable tissue necrosis

Necrotizing acute pancreatitis

- Inflammation associated with pancreatic parenchymal necrosis and/or peri-pancreatic necrosis

Organ failure and systemic complications of acute pancreatitis

- Respiratory: $Pa_{O_2}/Fi_{O_2} \leq 300$
- Cardiovascular: systolic blood pressure < 90 mm Hg (off ionotropic support), not fluid responsive, or pH < 7.3
- Renal: serum creatinine ≥ 170 μmol/L

<u>Scoring system for acute pancreatitis</u>

- BISAP score
- Ransons score
- Glasgow score
- APACHE-II score
- CT severity Index
- Modified CT severity Index

- HAPS score
- Revised Atlanta Classification

Acute pancreatitis signs and symptoms

- Acute pancreatitis signs and symptoms include:
- Upper abdominal pain that radiates to your back.

  Upper stomach steady pain may transmit to the back and-might be serious. Pain is exasperated by the food intake or by a beverage of liquor. Pain is impervious to analgesics. Patient accept of different poses in order to get some relief from pain.
- Nausea and vomiting.

  Vomiting is usually of low volume and non projectile and it contains gastric and duodenal content..
- Abdominal pain that feels worse after eating.
- Fever.
- Rapid pulse.

Diagnosis of acute pancreatitis

- Evaluation of signs and symptoms
- Physical examination
- Patient is restless.
- Rapid respiratory rate and pulse.

- Hypotension

- Abdomen- epigastric dullness with moderately distended abdomen. Tenderness mainly in the upper part of abdomen.

- Abdominal muscle spasmof moderate intensity is present.

- **GREY TURNERS SIGN**

  Grey green staining of the flank in individuals with peripancreatic heamorrhage

- **CULLEN' S SIGN** - bluish staining of periumbilical region

- **Extra abdominal manifestations**

- Pleural effusion in left side

- Acute pulmonary failure as a result of which tachypnoea, dyspnoea is seen,

- Cyanosis - due to

a) Phospholipase found in circulation

b) Lipolysis leading to circulation of free fatty acids from triglycerides

c) pulmonary capillary leakage leading to volume overload

- **Central Nervous System manifestations-**

  Central Nervous System manifestations leasing to psychosis, confusion and coma. This is mainly because of hypoperfusion, hyperosmolarity, cerebral fat embolism, hypoxia, disseminated intravascular coagulation.

- **<u>Blood tests</u>:-**
- Haematocrit- high.
- Leucocytosis
- High lipase and amylase levels
- Serum Amylase

  1. Elevated in majority of the patients with Acute pancreatitis. But this is not reliable marker for diagnosis since it is elevated in other conditions such as –

     Peptic ulcer

     Intestinal obstruction

     Biliary lithiasis

     Salivary gland diseases

     Mesenteric infarction.

  2. Patients with acute pancreatitis can have normal levels of serum amylase in due to-

     Triglyceridemia

     Destroyed glandular tissues- in previous attack

     Massive destruction of glands- in present attack

Serum amylase in acute Pancreatitis is elevated within 24 hrs of onset of symptoms and returns to normal in 2-3 days.

- **SERUM LIPASE:**

  Serum lipase is solely of pancreatic origin hence serum lipase level is more specific than amylase. Recent development of an enzyme immuno assay of lipase is reliable and is of great value in Acute Pancreatitis. Duration of Hyper lipasemia exceeds hyperamylassemia.

- **Trypsinogen**

Trypsinogen has two major isoenzymes, trypsinogen-1 and trypsinogen-2 , trypsinogen 2 is elevated in acute pancreatitis.

Biochemical markers of acute pancreatitis are amylase, lipase, and the proenzyme trypsinogen. serum amylase is the most commonly used of these in clinical practice .viding greater sensitivity in patients with a delayed presentation.

3. **PLEURAL AND PERITONEAL FLUID AMYLASE**

   In pancreatitis, pleural fluid effusion show higher levels of amylolytic activity.

- Lipid profile
- **Stool tests- to detect** fat malabsorption.
- **Imaging Parameters in Acute Pancreatitis**

Ultrasound is the first imaging methodology in quite a while for the adaptation of the conclusion of intense pancreatitis and managing out of different reasons for intense stomach area, since it is speedy and simple to perform, it is repeatable, free of radiation and can be done at bedside. The benefit of US in the early period is that it permits to assess the nerve bladder and biliary tract, and to distinguish gallstones and dilatation of the bile pipes. In 30% of cases, pancreatic extension and diminished parenchymal echogenicity because of interstitial edema might be seen . Central not well characterized hypo/hyperechoic territories (edema/drain), which perhaps saw in parenchyma. Obscuring of the pancreatic forms because of edema of the encompassing fat tissue and the liquid gathering in the peripancreatic district, particularly in the lesser sac and the left front pararenal space might be seen. Ultrasound is utilized in portrayal of the substance of the liquid accumulations and the pseudocysts

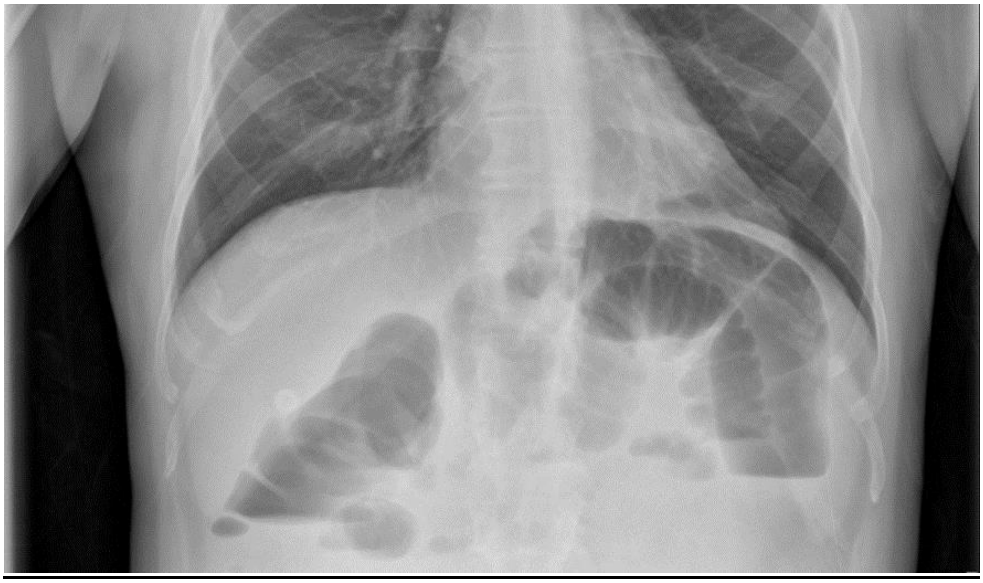
Plain x-rays

- **Ultrasound**
- **Computed tomography**
- **Magnetic resonance cholangiopancreatography (MRCP)**
- **Endoscopic ultrasound**
- **Pancreatic Function Test**

Plain x-rays

- "SENTINEL LOOP' in the left upper quadrant. – that means segmental small bowel ileus
- "COLON CUT OFF SIGN"- that means dilatation of the transverse colon –
- Enhanced epigastric soft tissue bulk
- Psoas muscle margins- Obscured.
- Gall stones - Present
- Pancreatic calcification

Fig- **COLON CUT OFF SIGN- XRAY**



ii). Plain x-ray chest

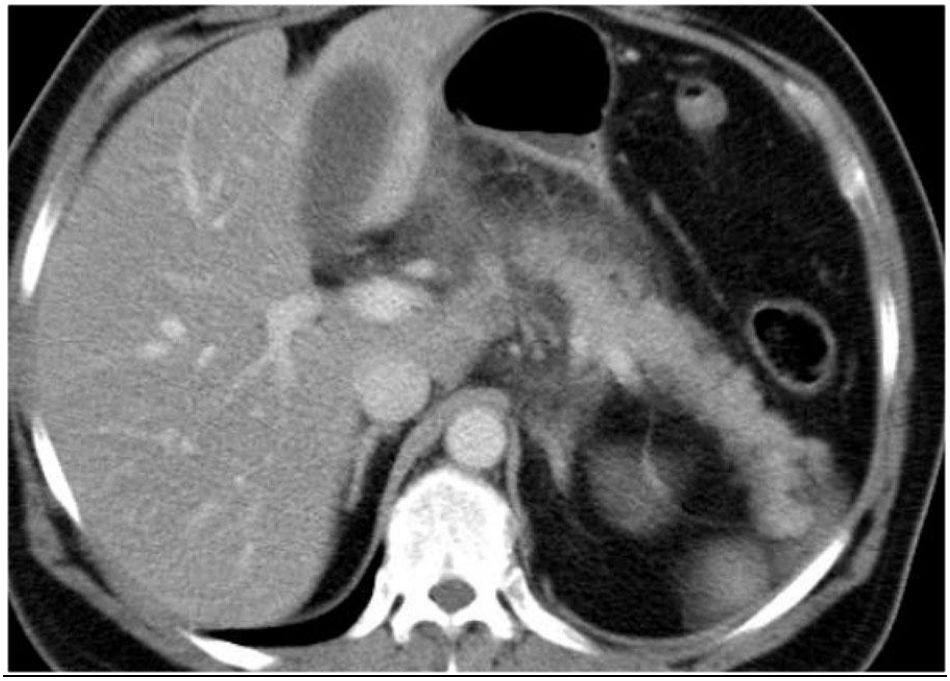
- Pleural effusion
- Atelectasis
- Pneumonia
- Pulmonary edema

Ultrasound

- edema and enlargement of pancreas
- Pancreatic Pseudocysts
- Pancreatic abscess
- Widening of Bile duct and existence of stone in gall bladder and common vile duct

Computed tomography

Pancreatic necrosis is characterized by the non enhancement in the contrast CT scan.



Endoscopic ultrasound

- In order to find biliary pancreatitis.
- Utilized for papillotomy and destruction of stones impacted at the ampulla of Vater.

MRI

Comprehensive evaluation of acute pancreatitis, requires to evaluate the pancreatic parenchyma, vasculature and the peripancreatic tissues . MRI for acute pancreatitis needs the collective use of T1-weighted imaging like the fast spin-echo image with numerous breath-hold acquisitions or else single-breath-hold gradient echo imaging and T2-weighted imaging like the dearly recovery and spin-echo or single-shot fast spin-echo (SSFSE) imaging and the MRCP.

The T1-weighted imaging with fat inhibition improves the definition of pancreas and pancreatic borders and, and it helps to evaluate the pancreatic hemorrhage and complications of acute pancreatitis.

Local complications of acute pancreatitis

- Acute peripancreatic fluid collections
- Pseudocysts in pancreas
- Acute necrotic
- Pancreatic necrosis

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

RANSON'S SCORE

Ranson (alcoholic etiology or other)	Ranson (biliar etiology)
At admission Age > 55 years Leukocytes > 16 000/mm3 LDH > 350 U/l AST > 250 U/l Glicemia > 200 mg/dl	**At admission** Age > 70 years Leukocytes > 18 000/mm3 LDH > 250 U/l AST > 250 U/l Glicemia > 220 mg/dl
After 48 hours Reduction in hematocrit > 10% Increase in BUN > 5 mg/dl Calcium < 8 mg/dl PO2 < 60 mmHg Base excess> 4 mEq/l Fluid leakage > 6L	**After 48 hours** Reduction in hematocrit > 10% Increase in BUN > 2 mg/dl Calcium < 8 mg/dl PO2 < 60 mmHg Base excess > 5 mEq/l Fluid leakage > 4L

CT SEVERITY INDEX SCORE

Grade of Acute pancreatitis		Points
A	NORMAL PANCREAS	0
B	PANCREATIC ENLARGMENT ALONE FOCAL OR DIFFUSE WITH CONTOUR IRREGULARITIES AND INHOMOGENOUS ATTENUATION	1
C	B+PERIPANCREATIC INFLAMMATION	2
D	C+ONE PERIPANCREATIC FLUID COLLECTION	3
E	D+TWO OR MORE PERIPANCREATIC OR RETROPERITONEAL FLUID COLLECTION OR GAS COLLECTION	4
DEGREE OF PANCREATIC NECROSIS		
1	NO – NECROSIS	0
2	NECROSIS OF <33% PANCREASE	2
3	NECROSIS OF 33%-50% OF PANCREASE	4
4	NECROSIS OF > 50% OF PANCREASE	6
CT SEVERITY INDEX (CT SI)BALTHAZAR SCORE+NECROSIS SCORE		
CT GRADE + NECROSIS GRADE (0 - 4) + (0 – 6) → (0 – 10)		

Management of acute Pancreatitis

- Endoscopic retrograde cholangiopancreatography (ERCP)
- In patients with acute gallstone pancreatitis associated with bile duct obstruction or cholangitis. - ERCP should be performed within 24–48 h
- In unstable patients with severe acute gallstone pancreatitis and associated bile duct obstruction or cholangitis, placement of a percutaneous transhepatic gallbladder drainage tube should be considered if ERCP is not safely feasible.

- In patients with alcohol induced pancreatitis-

A) We put the patient nil per oral, insert ryle's tube

B) Patient is started on antibiotics, somatostatin analogues and vitamin k ,

C) Amylase level are repeated after 72 hrs

D) If amylase levels are still high then **c**holecystectomy is done

Studies

- Parimalaetal Compared BISAP score with RANSON score in order to predict severity of acute pancreatitis. 60 patients were incorporated in the study and BISAP score and Ranson's score was assessed in all the

patients on the basis of data obtained within 48 hours of hospitalization. Results of the study showed that according to Atlanta Revised criteria, thirty patients had mild pancreatitis, twenty patients had modestly to serious pancreatitis, ten patients had extreme pancreatitis. Of the sixty patients, thirty seven patients had Ranson's score not exactly or equivalent to 3. 23 patients had a score of more than three .Out of the sixty patients, thirty nine patients had a BISAP score not exactly or equivalent to 3, 21 patients had a score more than three.

- YadavJ etal Predicted morbidity and mortality in acute pancreatitis in an Indian population**.** Of the 119 cases, 42 (35.2%) created organ failure and were delegated serious acute pancreatitis (SAP), 39.5% created PNec, and 10.1% died. Ranson's score showed a to some degree lower exactness for foreseeing SAP and mortality . CTSI was the most exact in anticipating PNec, with an AUC of 0.958. The affectability and identity of BISAP score, with a cut-off of ≥3 in foreseeing mortality, were 100% and 69.2%, independently.

- Wu BUet al, developed a clinical scoring system in acute pancreatitis patient using (CART) analysis, to predict hospital mortality. Data was collected for 18,256 acute pancreatitis cases in 177 hospitals within 2004-2005 on which the BISAP score was validated. Area under the Receiver

operating characteristic curve (AUC) was used to measure the accuracy of the BISAP score to predict mortality in the hospitals. On comparison with the APACHE II score the credibility of BISAP score was further established. Hence it was concluded, that BISAP score was a simple and accurate scoring system for indentifying acute pancreatitis patient who are at risk for in- hospital mortality.

- Kumar AH etal studied assessment of BISAP, APACHE II, Ranson's score and modified CTSI in order to predict the severity of acute pancreatitis**.** Results showed that APACHE II was a useful prognostic scoring framework for surveying the seriousness of intense pancreatitis and can go about as a pivotal guide in unequivocal the gathering of patients that have a more prominent possibility of requirement for tertiary consideration over the span of their sickness and consequently need early revival and brief referral, particularly in creating nations.

- Singh VKet al ,published a study in which BISAP score was evaluated for 397 acute pancreatitis cases admitted in their hospital to analyse the ability of BISAP score to predict mortality. Within 24 hours of presentation, the BISAP score was analysed. They observed that out of 397 patients, 3.5% (14 patients) died. As the BISAP score increased, the mortality also increases ($p<0.0001$). Thus, they concluded that if BISAP score is calculated within 24 hours of presentation, it is an easiest and an

accurate method to evaluate patients at the risk of increased mortality and to develop intermediate markers of severity.

- Khanna AKetal looked at Ranson, Glasgow, MOSS, SIRS, BISAP, APACHE-II, CTSI Scores, IL-6, CRP, and Procalcitonin. Results indicated thatIL-6 and CRP demonstrated a promising outcome in early distinguishing proof of seriousness and pancreatic corruption though APACHE-II and Ranson score in foreseeing AP related mortality in this examination.

- Mounzer Ret al published a comparative study in acute pancreatitis patient discussing the prediction of organ failure in the existing clinical scoring systems. He observed that the Glasgow score was an excellent classifier for evaluating the severity. All the other predicting systems depicted moderate accuracy.

- Fabre A et al did a study on 48 children diagnosed with acute pancreatitis. In that study, Ranson's, CTSI, and Glasgow scoring were calculated for the patients. For predictive severity, the affectability and explicitness of Ranson's score was 56% and 85% individually contrasted with the CTSI which had affectability and particularity of 80% and 86% separately. In his examination he inferred that for assessing the seriousness of intense pancreatitis in kids CT seriousness file was superior to the next scoring frameworks.

- Papachristou GI etal compared BISAP score, Ranson's score, APACHE-II score, and CTSI score in predicting mortality in acute pancreatitis. Results showed that the quantity of patients with a BISAP score of > or =3 was 26; Ranson's> or =3 was 47, APACHE-II > or =8 was 66, and CTSI > or =3 was 59. Of the seven patients that passed on, one had a BISAP score of 1, two had a score of 2, and four had a score of 3. AUCs for BISAP, Ranson's, APACHE-II, and CTSI in foreseeing SAP are 0.81 and 0.84, separately. We confirmed that the BISAP score is an exact strategies for chance stratification in patients with AP. Its fragments are clinically material and easy to get. The prognostic precision of BISAP resembles those of the other scoring systems. We reason that direct scoring systems may have landed at their maximal utility and novel models are relied upon to further improve insightful definite

- Zhang WW et al did a comparative study between CT pancreatic inflammatory infiltration degree of severe acute pancreatitis (SAP) and the clinical disease severity. The study included 83 patients. In that study, the concluded that, the score for extra pancreatic inflammation spread is better among all the CT severity indices.

AIMS AND OBJECTIVES

1. To assess accuracy of BISAP score, RANSON'S score and MODIFIED CTSI score for predicting severity in acute pancreatitis.

2. To compare efficiency of BISAP score, RANSON'S score and MODIFIED CTSI score for predicting mortality and complications of acute pancreatitis such as renal failure, respiratory failure, MODS and pancreatic necrosis.

3. To evaluate demography of patients of acute pancreatitis admitted in GOVT ROYAPETTAH HOSPITAL between April 2019 to september 2019.

MATERIALS AND METHOD

STUDY DESIGN

Cross sectional observational study

DURATION OF STUDY

April 2019 to October 2019.

PLACE OF STUDY

This study was conducted in department of general surgery, at Govt Royapettah Hospital, Chennai.

STUDY POPULATION

Study conducted among the patients attending the department of general surgery, at Govt Royapettah Hospital, Chennai.

SELECTION OF PATIENTS

Inclusion Criteria

- Patients diagnosed with acute pancreatitis and admitted in DEPARTMENT OF GENERAL SURGERY, GOVT ROYAPETTAH HOSPITAL, CHENNAI by following methods.

- Clinical examination.
- Serum amylase > 250mg/dl
- Radiological (CT Scan/ USG) findings suggestive of acute pancreatitis.

Exclusion Criteria

- Patients of pediatric age group.
- Patients with known co-morbidity which can interfere with criteria involved in scoring systems included in the study (CKD, bronchitis, liver cirrhosis).
- Chronic pancreatitis.

SAMPLE SIZE

Sample size was determined based on

The prevalence of acute pancreatitis was 70%.

Description:

• The confidence level is estimated at 95%

• with a z value of 1.96

• the confidence interval or margin of error is estimated at +/-5

• Assuming p% =70% and q%=30%

$n = p\% \times q\% \times [z/e\%]^2$

n= 68

Final minimum sample size adjusted to losses = 68

SAMPLING PROCEDURE

Convenience sampling procedure

DATA COLLECTION

Study tools

A semi structured questionnaire was developed to record the medical history and examination details

Study procedure

- Patients fulfilling the inclusion criteria were enrolled into the study and evaluation and recording in a preformed proforma of the following were done after getting a written informed consent.
- Patients details were collected
- Clinical and examinations findings are recorded

BISAP score calculated as soon as possible after admission. For which BUN, and WBC counts assessed by blood investigation; pleural effusion assessed by chest X ray; rest parameters assessed clinically.

Cases in which CT scan was suggestive of mild pleural effusion but could not be appreciated on chest x ray were considered as 0 score in pleural effusion criteria of BISAP score, but the same cases were given 2 score in extrapancreatic complications criteria of MODIFIED CTSI score. To avoid bias, first x ray chest was taken for all cases and pleural effusion score of BISAP score was decided than only CT scan was taken for MODIFIED CTSI.

Ranson's score calculated on admission and after 48 hours of admission. For which WBC count, blood glucose level, LDH, AST, hematocrit, BUN, base deficit, PaO2, calcium levels assessed by blood investigations, requirement of fluid replacement assessed by strict input and output monitoring.

Modified CTSI score obtained by Contrast enhanced CT scan. Within 24 hours of admission and score assessment was done by radiologist.

Consideration of complications:

Renal failure was considered present by presence of any of the following:[70]

- Urine output less than400ml/day
- Serum creatinine >4mg/dl
- Need forhemodialysis

Pulmonary failure was considered present by presence of any of the following:[71]

- PaO2< 60mmhg
- Need for mechanical ventilation.

MODS were considered by signs of 2 or more organ failure.

Pancreatic necrosis was assessed by radiologist while evaluating MODIFIED CTSI score.

STATISTICAL ANALYSIS

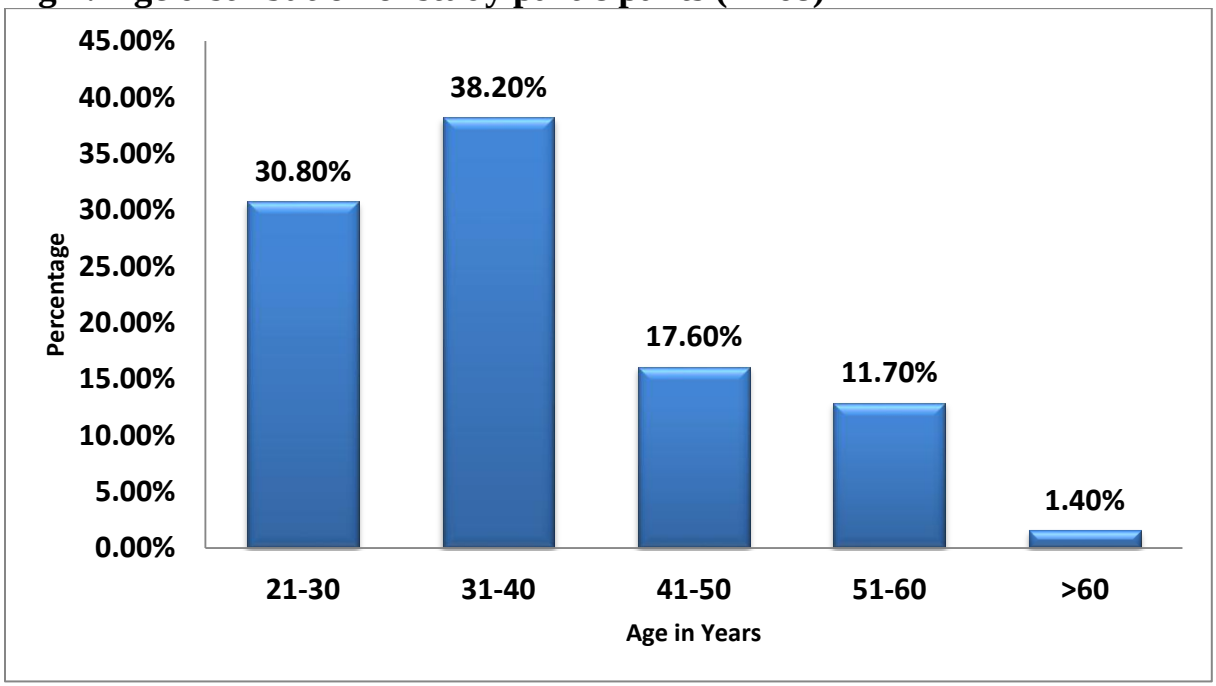
Data was entered into Microsoft excel data sheet and was analyzed using SPSS 22 version software. Categorical data was represented in the form of Frequencies and proportions. p value (Probability that the result is true) of <0.05 was considered as statistically significant after assuming all the rules of statistical tests. ROC curve was used to depict the sensitivity and specificity. Statistical software: MS Excel**,** *SPSS* for Windows Inc. Version 22. *Chicago*, *Illinois*was used to analyze data.Graphical representation of data:MS Excel and MS word was used to obtain various types of graphs such as bar diagram and Pie diagram.

RESULTS

Table 1: Age distribution of study participants (N=68)

Sl no	Age (in years)	Frequency	Percentage
1	21-30	21	30.8
2	31-40	26	38.2
3	41-50	12	17.6
4	51-60	8	11.7
5	>60	1	1.4
Mean=34±11.01	Total	68	100

Fig 1: Age distribution of study participants (N=68)

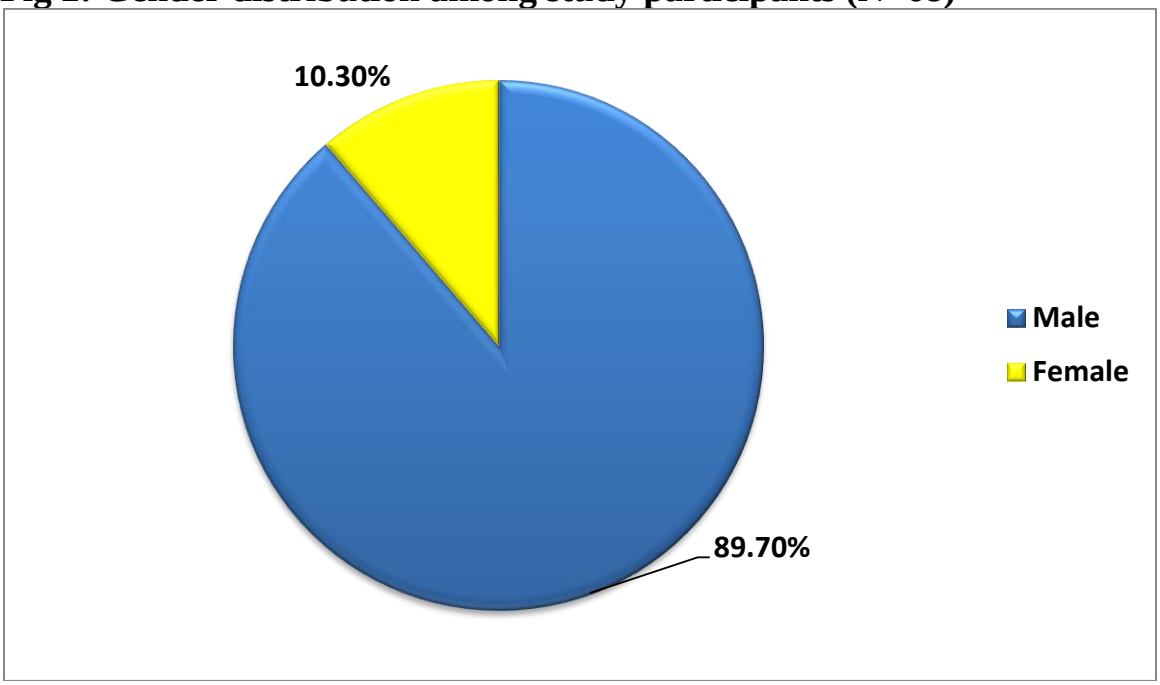


Among the subjects majority of them are in the age group of 31-40 (38.2%) followed by 21-30 yrs (30.8%), 41-50 yrs (17.6%) and more than 60 years (1.4%).

Table 2: Gender distribution among study participants (N=68)

Slno	Gender	Frequency	Percentage
1	Male	61	89.7
2	Female	7	10.3
	Total	68	100

Fig 2: Gender distribution among study participants (N=68)

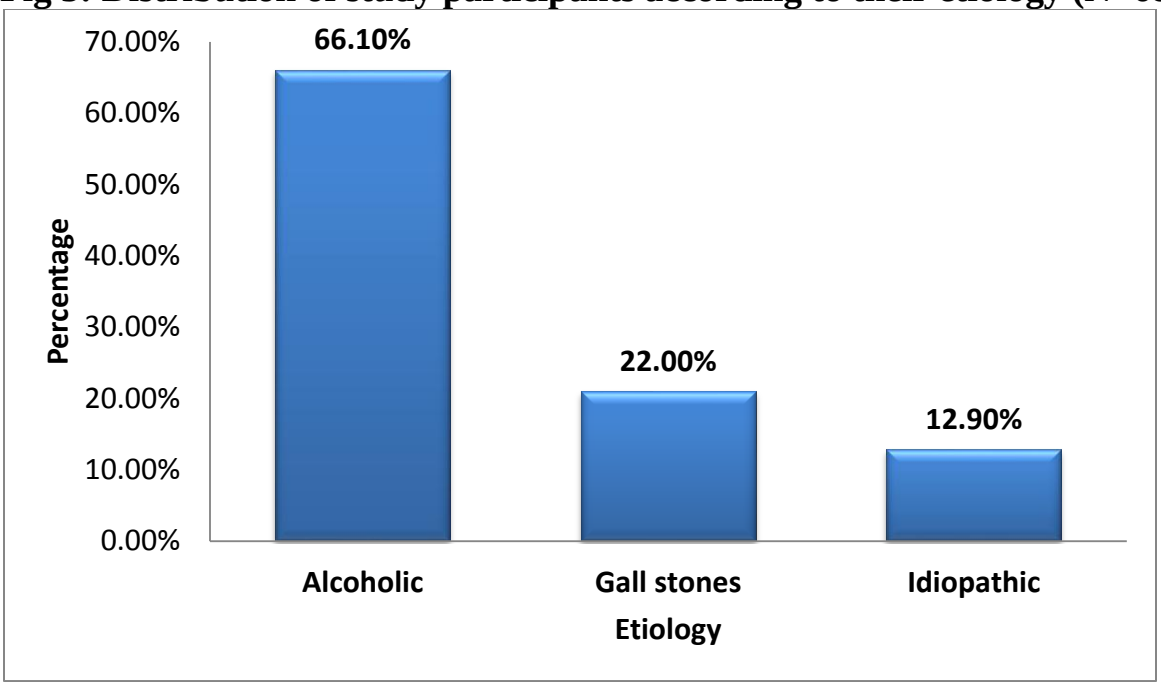


Male preponderance is seen in our study (89.7%).

Table 3: Distribution of etiology among study participants (N=68)

Slno	Etiology	Frequency	Percentage
1	Alcoholic	45	66.1
2	Gall stones	15	22.0
3	Idiopathic	8	12.9
	Total	68	100

Fig 3: Distribution of study participants according to their etiology (N=68)



Majority of the participants were alcoholic (66.1%) followed by having gall stones (22%) and few were idiopathic in nature (12.9%).

Table 4: Distribution of study participants based on their outcome (N=68)

	BISAP		**RANSON**		**Modified CTSI**	
	≤2	**≥3**	**< 3**	**≥3**	**< 8**	**≥8**
Number of patients	50	18	48	20	47	21
Renal failure	9	14	10	14	16	5
Respiratory failure	10	13	10	14	16	5
MODS	6	13	6	12	18	2
Pancreatic necrosis	25	16	16	14	24	16

Total number of patients having BISAP score ≤2 was fifty and ≥3 were eighteen. Out of which those having BISAP scores ≤2, nine were having renal failure, 10 were having respiratory failure, 6 of them with MODS and 25 with pancreatic necrosis. Among those with BISAP scores ≥3, 14 were having renal failure, 13 were having respiratory failure, 13 of them with MODS and 16 with pancreatic necrosis.Out of 48 subjects with RANSON scores ≤3, 48 were having renal failure, 10 were having respiratory failure, 10 of them with MODS and 16 with pancreatic necrosis. Among those with RANSON scores ≥3, 14 were having renal failure, 14 were having respiratory failure, 12 of them with MODS and 14 with pancreatic necrosis.Out of 47 subjects with CTSI scores ≤8,

16 were having renal failure, 16 were having respiratory failure, 18 of them with MODS and 24 with pancreatic necrosis. Among those with RANSON scores ≥8, 5 were having renal failure, 5 were having respiratory failure, 2 of them with MODS and 16 with pancreatic necrosis.

Table 5: Distribution of study participants based on their mortality (N=68)

	Survived	**Expired**
BISAP		
≥ 3	5	16
≤ 2	1	46
RANSON		
≥ 3	3	21
< 3	1	43
Modified CTSI		
≥ 8	4	18
< 8	2	44

Table 6: Analysis of BISAP score in predicting mortality (N=68)

BISAP SCORE	Expired	Survived	Total
≥ 3	5	16	**21**
≤ 2	1	46	**47**
Total	**6**	**62**	**68**

Statistics	Estimate	Lower- Upper 95% CI
Sensitivity	83.33%	35.88% to 99.58%
Specificity	74.19 %	61.50% to 84.47%
Disease prevalence	8.82%	3.31% to 18.22%
Positive Predictive Value	23.81%	15.23% to 35.21%
Negative Predictive Value	97.87 %	88.43% to 99.64%
Accuracy	75.00%	63.02% to 84.71%

Table 7: Analysis of RANSON score in predicting mortality (N=68)

RANSON SCORE	Expired	Survived	Total
≥ 3	3	21	**24**
<3	1	43	**44**
Total	**4**	**64**	**68**

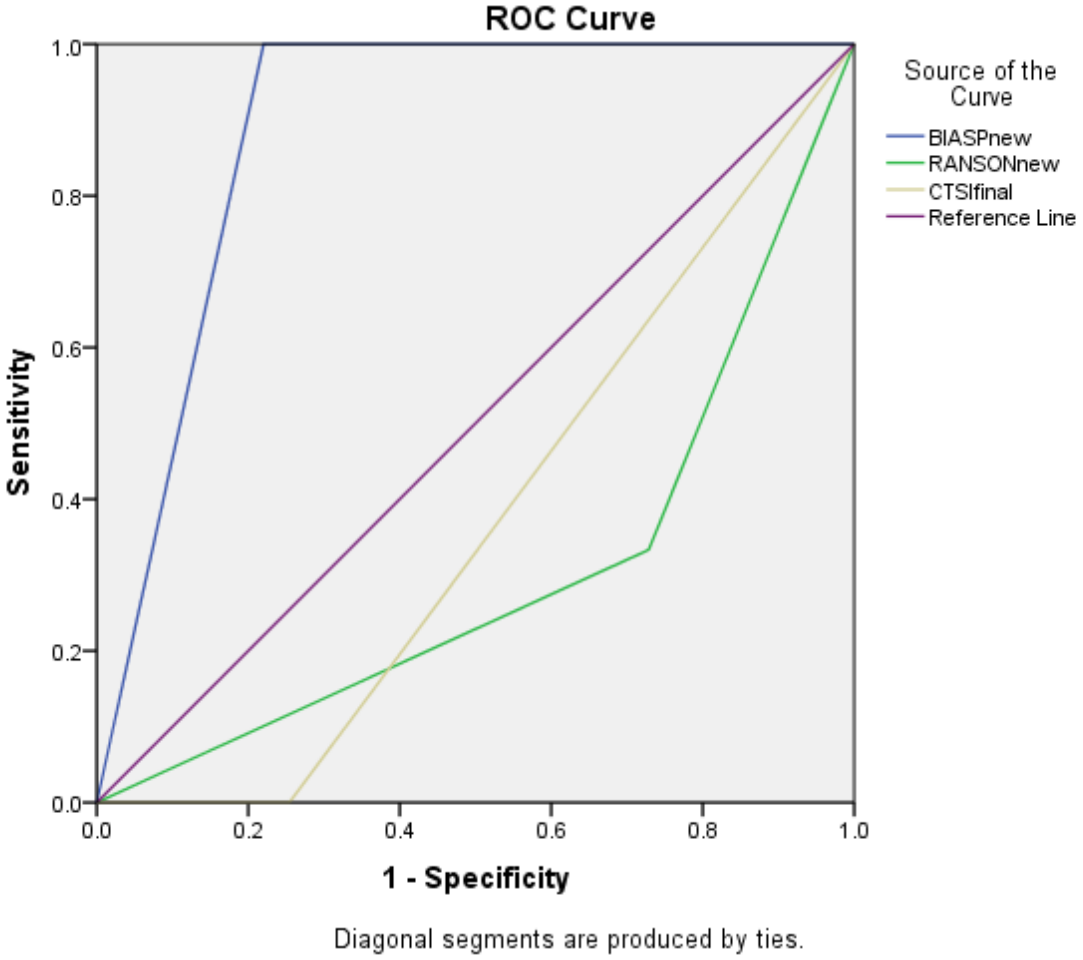
Statistics	Estimate	Lower- Upper 95% CI
Sensitivity	75.00%	19.41% to 99.37%
Specificity	67.19 %	54.31% to 78.41%
Disease prevalence	5.88%	1.63% to 14.38%
Positive Predictive Value	12.50%	6.84% to 21.75%
Negative Predictive Value	97.73 %	88.65% to 99.58%
Accuracy	67.65%	55.21% to 78.49%

Table 8: Analysis of Modified CTSI score in mortality (N=68)

CTSI SCORE	Survived	Expired	Total
≥ 8	4	18	**22**
< 8	2	44	**46**
Total	**6**	**62**	**68**

Statistics	Estimate	Lower- Upper 95% CI
Sensitivity	66.67%	22.28% to 95.67%
Specificity	70.97 %	58.05% to 81.80%
Disease prevalence	8.82%	3.31% to 18.22%
Positive Predictive Value	18.18%	10.06% to 30.63%
Negative Predictive Value	95.65 %	87.53% to 98.57%
Accuracy	70.59%	58.29% to 81.02%

ROC Curve comparing the scoring system predicting the mortality



Test Result Variable(s)	Area	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
BISAP	.890	.024	.790	.989
RANSON	.302	.251	.000	.620
CTSI	.373	.460	.112	.634

The ROC curve finds out the score which predicts the mortality. So this clearly suggests that BISAP score is better when compared to the other score.

Table 9: Analysis of BISAP score in predicting pancreatic necrosis (N=68)

BISAP SCORE	Pancreatic necrosis present	Pancreatic necrosis absent	Total
≥ 3	16	2	**18**
≤ 2	25	25	**50**
Total	**41**	**27**	**68**

Statistics	Estimate	Lower- Upper 95% CI
Sensitivity	39.02%	24.20% to 55.50%
Specificity	92.59 %	75.71% to 99.09%
Disease prevalence	60.29%	47.70% to 71.97%
Positive Predictive Value	88.89%	66.64% to 96.97%
Negative Predictive Value	50.00 %	43.36% to 56.64%
Accuracy	60.29%	47.70% to 71.97%

Table 10: Analysis of RANSON score in predicting pancreatic necrosis (N=68)

RANSON SCORE	Pancreatic necrosis present	Pancreatic necrosis absent	Total
≥ 3	14	6	**20**
≤ 3	16	32	**48**
Total	**30**	**38**	**68**

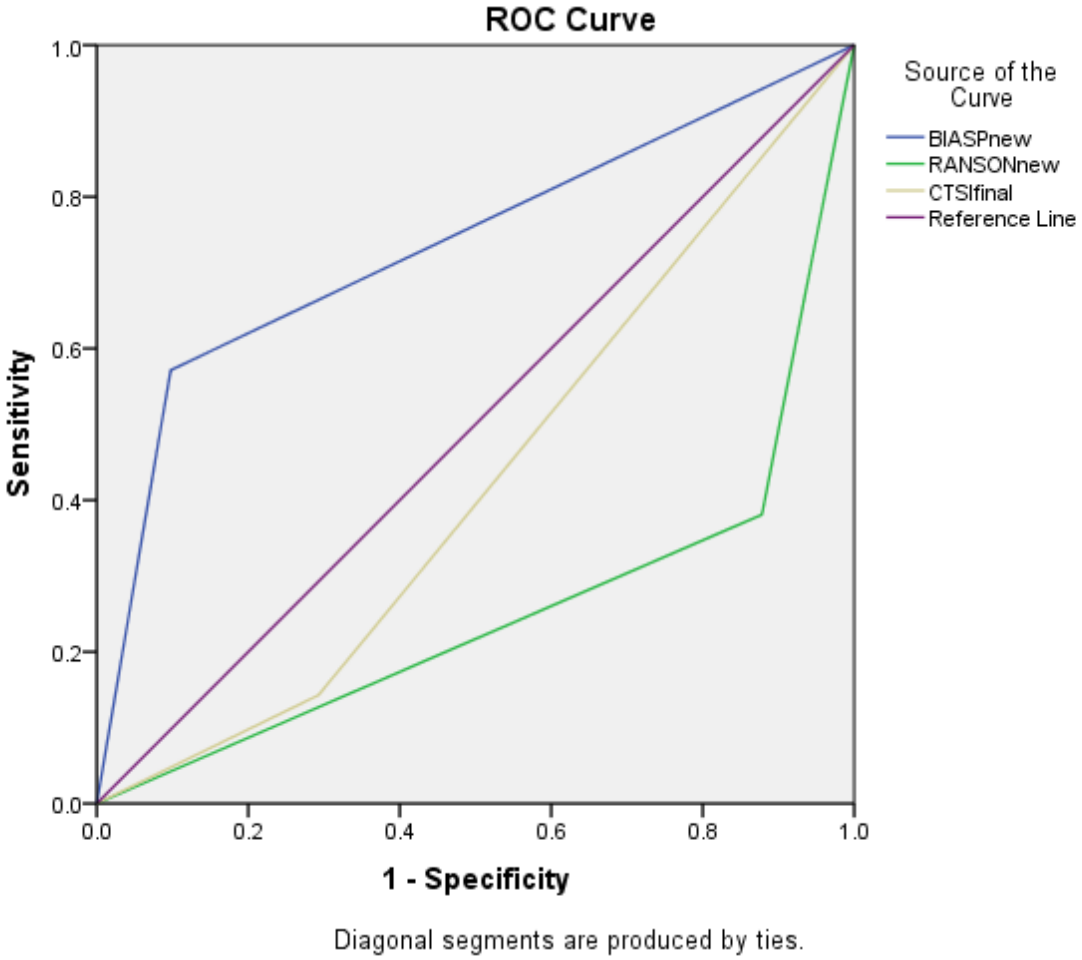
Statistics	Estimate	Lower- Upper 95% CI
Sensitivity	46.67%	28.34% to 65.67%
Specificity	84.21 %	68.75% to 93.98%
Disease prevalence	44.12%	32.08% to 56.68%
Positive Predictive Value	70.00%	50.48% to 84.23%
Negative Predictive Value	66.67 %	58.21% to 74.18%
Accuracy	67.65%	55.21% to 78.49%

Table 11: Analysis of Modified CTSI score in pancreatic necrosis (N=68)

CTSI SCORE	Pancreatic necrosis present	Pancreatic necrosis absent	Total
≥ 8	16	5	**21**
≤ 8	24	23	**47**
Total	**40**	**28**	**68**

Statistics	Estimate	Lower- Upper 95% CI
Sensitivity	40.00%	24.86% to 56.67%
Specificity	82.14 %	63.11% to 93.94%
Disease prevalence	58.82%	46.23% to 70.63%
Positive Predictive Value	76.19%	57.02% to 88.53%
Negative Predictive Value	48.94 %	41.36% to 56.56%
Accuracy	57.35%	44.77% to 69.28%

ROC Curve comparing the scoring system predicting the pancreatic necrosis



The ROC curve finds out the score which predicts the pancreatic necrosis. So this clearly suggests that RANSONS score is better when compared to the other score.

Table 12: Analysis of BISAP score in predicting MODS (N=68)

BISAP SCORE	MODS present	MODS absent	Total
≥ 3	13	5	**18**
≤ 2	6	44	**50**
Total	**19**	**49**	**68**

Statistics	Estimate	Lower- Upper 95% CI
Sensitivity	68.42%	43.45% to 87.42%
Specificity	89.80 %	77.77% to 96.60%
Disease prevalence	27.94%	17.73% to 40.15%
Positive Predictive Value	72.22%	51.76% to 86.30%
Negative Predictive Value	88.00 %	78.98% to 93.47%
Accuracy	83.82%	72.90% to 91.64%

Table 13: Analysis of RANSON score in predicting MODS (N=68)

RANSON SCORE	MODS present	MODS absent	Total
≥ 3	12	8	**20**
≤ 3	6	42	**48**
Total	**18**	**50**	**68**

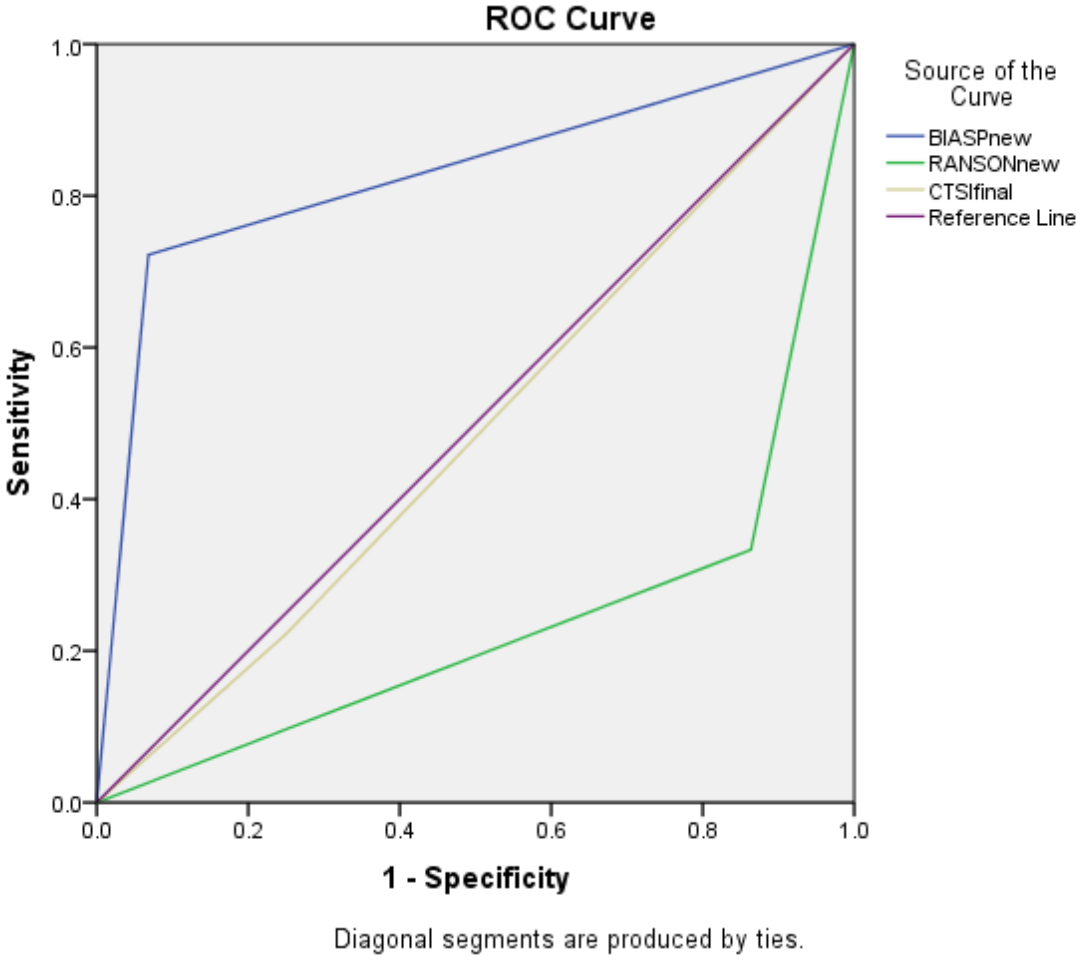
Statistics	Estimate	Lower- Upper 95% CI
Sensitivity	66.67%	40.99% to 86.66%
Specificity	84.00 %	70.89% to 92.83%
Disease prevalence	26.47%	16.50% to 38.57%
Positive Predictive Value	60.00%	42.34% to 75.39%
Negative Predictive Value	87.50 %	78.27% to 93.15%
Accuracy	79.41%	67.88% to 88.26%

Table 14: Analysis of Modified CTSI score in MODS (N=68)

CTSI SCORE	MODS present	MODS absent	Total
≥ 8	16	5	**21**
< 8	24	23	**47**
Total	**30**	**28**	**68**

Statistics	Estimate	Lower- Upper 95% CI
Sensitivity	40.00%	24.86% to 56.67%
Specificity	82.14 %	63.11% to 93.94%
Disease prevalence	58.82%	46.23% to 70.63%
Positive Predictive Value	76.19%	57.02% to 88.53%
Negative Predictive Value	48.94 %	41.36% to 56.56%
Accuracy	57.35%	44.77% to 69.28%

ROC Curve comparing the scoring system predicting the MODS



Test Result Variable(s)	Area	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
BIASP	.827	.000	.695	.959
RANSON	.235	.001	.092	.378
CTSI	.486	.865	.328	.645

The ROC curve finds out the score which predicts the MODS. So this clearly suggests that BISAP score is better when compared to the other score.

Table 15: Analysis of BISAP score in predicting respiratory failure (N=68)

BISAP SCORE	Respiratory failure present	Respiratory failure absent	Total
≥ 3	13	5	**18**
≤ 2	10	40	**50**
Total	**23**	**45**	**68**

Statistics	Estimate	Lower- Upper 95% CI
Sensitivity	56.52%	34.49% to 76.81%
Specificity	88.89 %	75.95% to 96.29%
Disease prevalence	33.82%	22.79% to 46.32%
Positive Predictive Value	72.22%	51.37% to 86.49%
Negative Predictive Value	80.00 %	71.28% to 86.57%
Accuracy	77.94%	66.24% to 87.10%

Table 16: Analysis of RANSON score in predicting respiratory failure

(N=68)

RANSON SCORE	Respiratory failure present	Respiratory failure absent	Total
≥ 3	14	6	**20**
< 3	10	38	**48**
Total	**24**	**44**	**68**

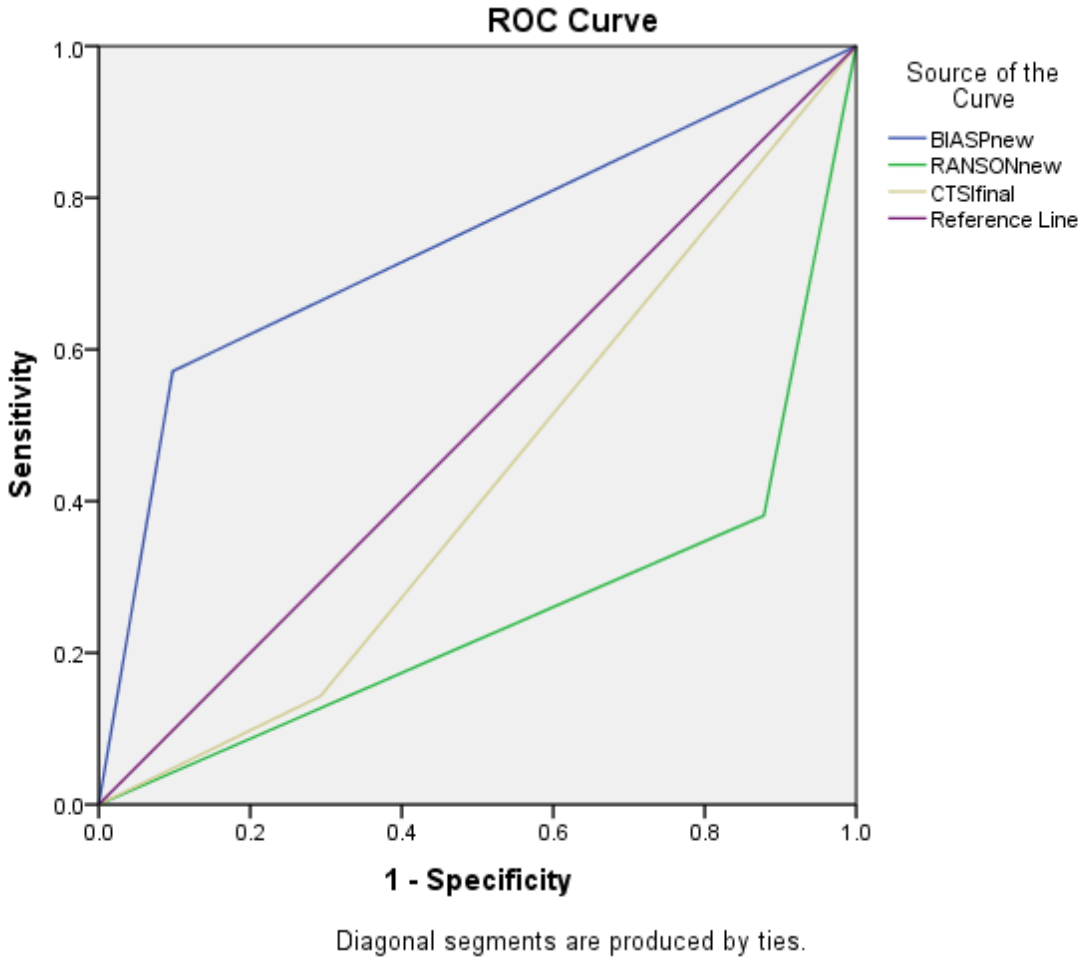
Statistics	Estimate	Lower- Upper 95% CI
Sensitivity	58.33%	36.64% to 77.89%
Specificity	86.36 %	72.65% to 94.83%
Disease prevalence	35.29%	24.08% to 47.83%
Positive Predictive Value	70.00%	50.76% to 84.08%
Negative Predictive Value	79.17 %	70.00% to 86.09%
Accuracy	76.47%	64.62% to 85.91%

Table 17: Analysis of Modified CTSI score in predicting respiratory failure (N=68)

CTSI SCORE	Respiratory failure present	Respiratory failure absent	Total
≥ 8	5	16	**21**
< 8	16	31	**47**
Total	**21**	**47**	**68**

Statistics	Estimate	Lower- Upper 95% CI
Sensitivity	23.81%	8.22% to 47.17%
Specificity	65.96 %	50.69% to 79.14%
Disease prevalence	30.88%	20.24% to 43.26%
Positive Predictive Value	23.81%	11.65% to 42.54%
Negative Predictive Value	65.96 %	58.57% to 72.64%
Accuracy	52.94%	40.45% to 65.17%

ROC Curve comparing the scoring system predicting the respiratory failure



Test Result Variable(s)	Area	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
BIASP	.737	.002	.594	.880
RANSON	.251	.001	.112	.391
CTSI final	.425	.337	.278	.572

The ROC curve finds out the score which predicts the respiratory failure.

So this clearly suggests that BISAP score is better when compared to the other score.

Table 18: Analysis of BISAP score in predicting renal failure (N=68)

BISAP SCORE	Renal failure present	Renal failure absent	Total
≥ 3	14	4	**18**
≤ 2	9	41	**50**
Total	**23**	**45**	**68**

Statistics	Estimate	Lower- Upper 95% CI
Sensitivity	60.87%	38.54% to 80.29%
Specificity	91.11 %	78.78% to 97.52%
Disease prevalence	33.82%	22.79% to 46.32%
Positive Predictive Value	77.78%	56.50% to 90.41%
Negative Predictive Value	82.00 %	73.08% to 88.43%
Accuracy	80.88%	69.53% to 89.41%

Table 19: Analysis of RANSON score in predicting renal failure (N=68)

RANSON SCORE	Renal failure present	Renal failure absent	Total
≥ 3	14	6	**20**
< 3	10	38	**48**
Total	**24**	**44**	**68**

Statistics	Estimate	Lower- Upper 95% CI
Sensitivity	58.33%	36.64% to 77.89%
Specificity	86.36 %	72.65% to 94.83%
Disease prevalence	35.29%	24.08% to 47.83%
Positive Predictive Value	70.00%	50.76% to 84.08%
Negative Predictive Value	79.17 %	70.00% to 86.09%
Accuracy	76.47%	64.62% to 85.91%

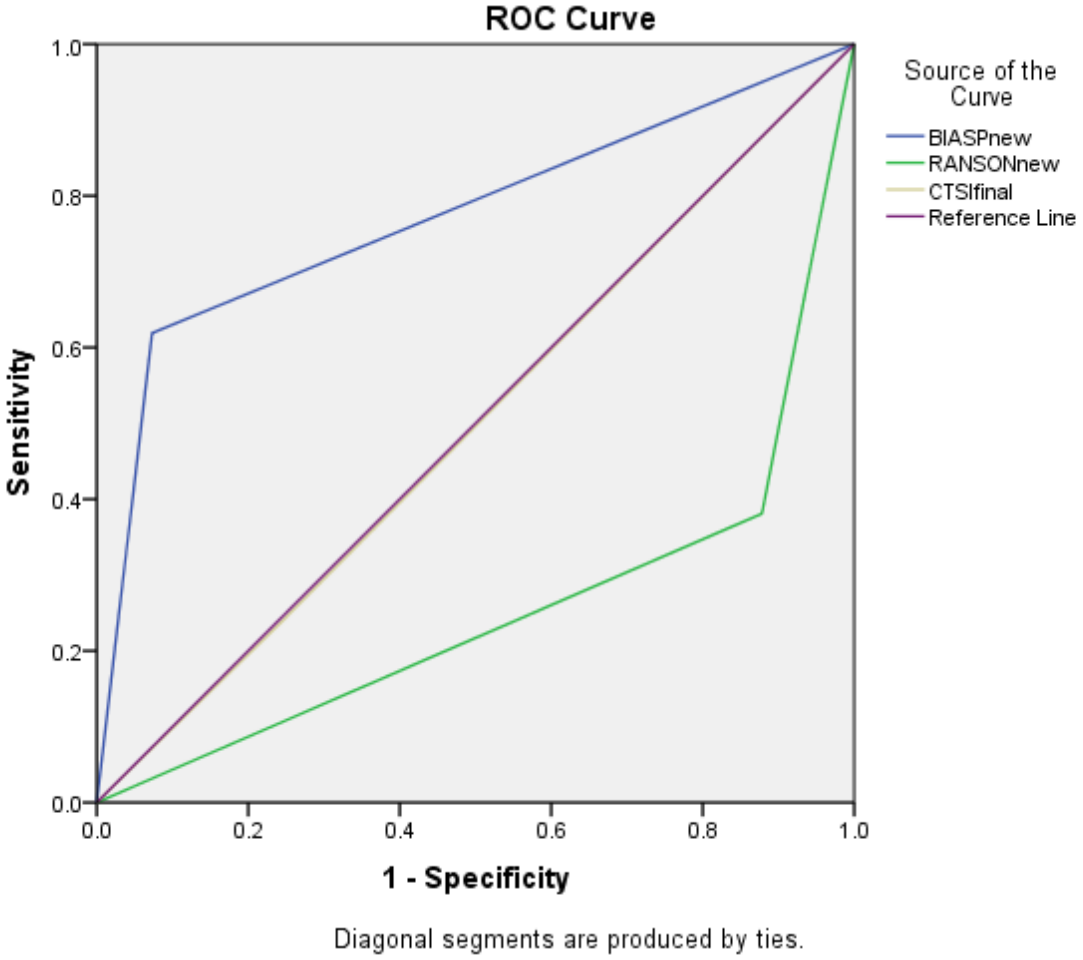
Table 20: Analysis of Modified CTSI score in predicting renal failure

(N=68)

CTSI SCORE	Renal failure present	Renal failure absent	Total
≥ 8	5	16	**21**
< 8	16	31	**47**
Total	**21**	**47**	**68**

Statistics	Estimate	Lower- Upper 95% CI
Sensitivity	23.81%	8.22% to 47.17%
Specificity	65.96 %	50.69% to 79.14%
Disease prevalence	30.88%	20.24% to 43.26%
Positive Predictive Value	23.81%	11.65% to 42.54%
Negative Predictive Value	65.96 %	58.57% to 72.64%
Accuracy	52.94%	40.45% to 65.17%

ROC Curve comparing the scoring system predicting the renal failure



Test Result Variable(s)	Area	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
			Lower Bound	Upper Bound
BIASP	.773	.000	.635	.911
RANSON	.251	.001	.112	.391
CTSI final	.497	.970	.344	.650

The ROC curve finds out the score which predicts the renal failure. So this clearly suggests that BISAP score is better when compared to the other score.

Age comparison

Slno	Study	Mean age
1	PRESENT STUDY	34±11.01
2	VIKESH SINGH et al	52±16.24
3	GEORGIOS et al	51±14.23
4	ANUBHAV KUMAR et al	48.42±11.75
5	AJAY K KHANNA et al	40.2±9.53

- In our study the total mean age calculated was 34±11.01 years and majority of them were in the age group of 21-30 years.
- Vikhesh singh and group found mean age of their study group to be 52 years.
- Georgios et al had conducted study on around one hundred and eighty five patients where the mean age of the participants calculated were 51.7 years.
- Similarly in a study conducted by Anubhav kumar et al studied fifty patients with acute pancreatitis and their mean age calculated was 48.42 years.
- Ajay k khannaet studied on seventy two patients and their mean age calculated was 40.5 year.

Gender

- In the current study there was male preponderance. There were around sixty one males and seven females. Higher percentage of males in our study reveals the higher prevalence of alcoholic pancreatitis.

Slno	Study	Males	Females
1	PRESENT STUDY	61	7
2	VIKESH SINGH et al	210	185
3	GEORGIOS et al	95	90
4	ANUBHAV KUMAR et al	17	33
5	AJAY K KHANNA et al	35	37

Etiology

- Majority of the participants were alcoholic (66.1%) followed by having gall stones (22%) and few were idiopathic in nature (12.9%).
- These findings are consistent with some studies and contradicting to some in regard of highest etiological cause being alcohol, but findings are consistent in view of alcohol and gallstone combined being highest etiology.

Slno	Study	ALCOHOL	GALLSTONE
1	PRESENT STUDY	66.1%	22%
2	VIKESH SINGH et al	54%	30%
3	GEORGIOS et al	45%	27%
4	ANUBHAV KUMAR et al	18%	74%
5	AJAY K KHANNA et al	13%	64%

SCREENING OF MORBIDITY AND ITS COMPLICATIONS

- In our current studythe total number of patients having BISAP score ≤2 was fifty and ≥3 were eighteen. Out of which those having BISAP scores ≤2, nine were having renal failure, 10 were having respiratory failure, 6 of them with MODS and 25 with pancreatic necrosis.
- Among those with BISAP scores ≥3, 14 were having renal failure, 13 were having respiratory failure, 13 of them with MODS and 16 with pancreatic necrosis.

BISAP (PRESENT STUDY)	SENSITIVITY	SPECIFCITY
RENAL FAILURE	60.97%	91.11%
MODS	68.42%	89.80%
RESPIRATORY FAILURE	56.25%	88.89%
PANCREATIC NECROSIS	39.02%	92.59%

- Out of 48 subjects with RANSON scores ≤3, 48 were having renal failure, 10 were having respiratory failure, 10 of them with MODS and 16 with pancreatic necrosis.
- Among those with RANSON scores ≥3, 14 were having renal failure, 14 were having respiratory failure, 12 of them with MODS and 14 with pancreatic necrosis.

RANSON (PRESENT STUDY)	SENSITIVITY	SPECIFCITY
RENAL FAILURE	58.33%	86.36%
MODS	66.07%	84.00%
RESPIRATORY FAILURE	58.33%	86.36%
PANCREATIC NECROSIS	46.67%	84.21%

- Out of 47 subjects with CTSI scores ≤8, 16 were having renal failure, 16 were having respiratory failure, 18 of them with MODS and 24 with pancreatic necrosis.
- Among those with CTSI scores ≥8, 5 were having renal failure, 5 were having respiratory failure, 2 of them with MODS and 16 with pancreatic necrosis.

MODIFIED CTSI (PRESENT STUDY)	**SENSITIVITY**	**SPECIFCITY**
RENAL FAILURE	23.81%	65.96%
MODS	40%	82.14%
RESPIRATORY FAILURE	23.81%	65.96%
PANCREATIC NECROSIS	40%	82.14%

- Rawasmounzer et al in study found sensitivity and specificity of BISAP for renal failure is 61% and 84% respectively. Similarly sensitivity and specificity of RANSON's for renal failure is 66% and 88% respectively.

MORTALITY COMPARISON AMONG DIFFERENT STUDIES OF BISAP SCORES

S.NO	STUDY	SENSITIVITY%	SPECIFICITY %
1	PRESENTSTUDY	83.33%	74.19
2	VIKESH SINGH et al	71	83
3	GEORGIOS et al	57.1	87.6

CONCLUSION

- Majority of them are in the age group of 21-30 (38.2%) followed by 31-40 yrs (30.8%).
- Male preponderance is seen in our study (89.7%).
- Majority of the participants were alcoholic (66.1%) followed by having gall stones (22%) and few were idiopathic in nature (12.9%).
- BISAP score is the better screening system and accurate predicting system for mortality and MODS when compared to other scores.
- RANSON'S score is found to be better screening system for respiratory failure. But BISAP score is more accurate predicting method having higher PPV than RANSON'S score.
- RANSON'S score and BISAP score are equally better screening method for renal failure but BISAP is better predicting method than other two.
- Overall BISAP is better screening method and accurate predicting method for mortality and complications of acute pancreatitis than RANSON'S score and MODIFIED CTSI score.

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CONSENT FORM

I …….. hereby give my consent to be included as a subject in this study “ COMPARATIVE STUDY OF VARIOUS SCORES IN PREDICTING COMPLICATIONS AND MORTALITY IN ACUTE PANCREATITIS” in the department of general surgery, Government royapettah hospital, Government kilpauk medical college, Chennai-10. All the details regarding the study have been explained to me by Dr.R. Rajarajan, the principal investigator. I wholeheartedly agree without any compulsion to disclose the true data regarding my illness and treatments taken by me to Dr.R. Rajarajan on request. The maintenance of confidentiality of the details has been assured. I agree to participate in this study and understand that no cost will be incurred on my side. I also know that I can withdraw from the study at anytime and my withdrawal from the study would not affect the treatment given to me.

Name of the investigator:

Dr.R. Rajarajan
Postgraduate student
Department of general surgery
Govt.Royapettah hospital
Govt.kilpauk medical college

Name of the patient:

Signature and date:

Signature of the investigator:

Name and signature of witness:

சுய ஒப்புதல் படிவம்:

ஆய்வு செய்யப்படும் தலைப்பு‘‘

- COMPARATIVE STUDY OF VARIOUS SCORES IN PREDICTING COMPLICATIONS AND MORTALITY IN ACUTE PANCREATITIS ‘, Department of General Surgery,GRH, KMCH.
- பங்கு பெறுபவரின் பெயர் :
- பங்கு பெறுபவரின் வயது :
- பங்கு பெறுபவரின் எண் :
- மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு விளக்கப்பட்டது. நான் இவ்வாய்வில் தன்னிச்சையாக பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த சட்டசிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக் கொள்ளல்லாம் என்றும் அறிந்து கொண்டேன். இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்கொள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்கமாட்டேன்.
- இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன். இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன்.
- பங்கேற்பவரின் கையொப்பம்:
- இடம் :
- தேதி :
- ஆய்வாளரின் கையொப்பம்:

URKUND

Urkund Analysis Result

Analysed Document:	urkund.docx (D58009385)
Submitted:	10/31/2019 6:13:00 PM
Submitted By:	drrajarajanmd@gmail.com
Significance:	7 %

Sources included in the report:

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Instances where selected sources appear:

28

GOVT. KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Protocol ID. No.203/2019 Meeting held on 16/07/2019

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "COMPARATIVE STUDY OF VARIOUS SCORES IN PREDICTING COMPLICATIONS AND MORTALITY IN ACUTE PANCREATITIS" submitted by Dr. R. Rajarajan, 2nd year, M.S Post Graduate, Dept of General Surgery, Government Kilpauk Medical College, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

18.10.2019.

DEAN
Govt. Kilpauk Medical College,
Chennai-10.

18.10.19

NO	NAME	AGE	SEX	BUN>25mg/dl	GCS<15	SIRS	PLEURAL EFFUSION(+)	BISAP SCORE	GALLSTONE OR AGE>70 FOR GALLSTONE	BLOOD GLUCOSE >200	WBC>16000 FOR NON GALLSTONE OR >18000 FOR GALLSTONE	LDH>350 FOR NON GALLSTONE OR>400 FOR GALLSTONE	AST>250	DECREASE>10%	SERUM CALCIUM <8mg/dl	BASE DEFICIT>4mEq/l FOR NON GALLSTONE OR >5mEq/L FOR GALLSTONE	BUN INCREASE >5 mg/dl FOR NON GALLSTONE OR >2 mg/dl FOR GALLSTNE	FLUID REQUIREMENT >6L FOR NON GALLSTONE OR >4L FOR GALLSTONE	PaO2<60 mm Hg	RANSON'S SCORE	INFLAMMATION	PANCREATIC NECROSIS	EXTRAPANCREATIC COMPLICATIONS	MODIFIED CTSI SCORE	ETIOLOGY	MORTALITY	RENAL FAILURE	RESPIRATORY FAILURE	MODS
1	VENKATESAN	26	M	0	0	1	0	1	0	0	1	0	1	0	0	0	0	0	0	2	4	2	2	8	A	0	0	0	0
2	MURUGAN	28	M	0	0	1	0	1	0	0	1	0	0	0	0	0	0	0	0	1	2	0	0	2	A	0	1	0	0
3	DHANASEKAR	31	M	0	0	1	0	1	0	0	1	0	0	0	0	0	0	0	0	1	4	2	2	8	I	0	0	0	0
4	PURUSOTHAMA	55	M	0	0	1	1	2	0	0	0	0	0	0	0	0	0	0	0	1	1	2	0	2	A	0	0	1	1
5	VENKATESAN	39	M	0	0	0	1	1	0	1	0	0	0	0	1	0	0	0	0	2	4	2	2	8	A	0	0	0	0
6	KARPAGAM	43	F	1	1	1	1	4	0	0	1	1	0	1	0	0	1	1	0	5	4	2	2	8	G	0	1	1	1
7	GOWTHAM	23	M	1	0	1	1	3	0	0	1	0	0	0	0	1	1	0	1	4	2	2	2	6	I	0	1	1	1
8	DAVID KUMAR	21	M	0	0	1	0	1	0	0	0	1	0	0	0	0	0	0	0	1	2	4	0	6	A	0	0	0	0
9	GANESAN	52	M	0	0	1	1	2	0	0	1	0	0	0	0	0	0	0	0	1	4	2	2	8	A	0	0	0	0
10	MANIKANDAN	29	M	1	0	0	0	1	0	0	0	0	0	1	0	0	0	0	0	1	0	0	0	0	A	0	0	1	0
11	KANIYAPPAN	45	M	0	1	1	0	2	0	0	1	0	0	0	0	1	0	0	0	2	2	0	2	4	G	0	0	0	0
12	VENKATESAN	29	M	0	0	1	0	1	0	1	0	1	1	0	1	0	0	0	0	4	2	0	0	2	A	0	0	1	0
13	VIJAYKUMAR	42	M	0	1	1	1	3	0	0	1	0	0	0	0	0	1	0	1	3	2	2	2	6	A	0	1	1	1
14	SELVAM	40	M	0	0	1	0	1	0	0	1	0	0	0	0	0	0	0	0	1	0	2	0	2	I	0	0	0	0
15	RAGENDRAN	48	M	0	0	0	1	1	0	0	0	0	0	0	0	0	1	0	0	1	2	2	2	6	A	0	1	0	0
16	ANBARASU	30	M	1	0	0	0	1	0	0	0	0	1	0	0	0	1	0	0	2	2	4	2	8	A	0	0	0	0
17	SURESH	30	M	0	0	1	1	2	0	0	1	0	0	0	0	0	0	0	0	1	0	0	2	2	A	0	0	0	0
18	MARAGATHAM	43	F	0	1	0	0	1	0	1	0	0	0	0	0	0	0	0	0	1	2	2	0	4	G	0	0	1	0
19	SHAHUL AHME	45	M	1	0	1	1	3	0	0	0	1	0	1	1	0	1	1	1	6	2	2	2	6	G	0	1	1	1
20	RAVI	28	M	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2	0	0	2	A	0	0	1	1
21	MUTHUKUMAR	27	M	0	0	1	0	1	0	0	1	0	0	0	0	0	0	0	0	1	4	0	0	4	A	0	0	0	0
22	ARUMUGAM	56	M	1	1	1	0	3	0	0	1		0	0	0	1	0	0	0	2	2	2	2	6	I	0	1	0	0
23	SELVARAJ	47	M	1	0	0	0	1	0	0	0	0	0	0	0	0	0	1	0	0	1	0	2	0	A	0	0	0	0
24	RAMAMOOTHY	32	M	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	1	2	0	0	2	A	0	1	1	1
25	MURUGAN	34	M	0	0	1	0	1	0	0	1	0	0	0	0	0	0	0	0	1	2	0	0	2	G	0	1	0	0
26	SUBULAXMI	26	F	0	0	1	1	2	1	0	1	0	0	0	0	0	0	0	0	2	2	2	2	6	G	0	0	0	0
27	SANTAKUMAR	47	M	0	0	0	1	1	0	0	0	1	1	0	0	0	0	1	1	4	2	4	2	8	A	0	0	0	0
28	SHANKAR	28	M	0	1	0	0	1	0	0	1	0	0	0	0	0	0	0	0	1	4	2	2	8	A	0	0	0	0
29	SADIK ALI	56	M	1	1	1	1	4	0	1	1	1	0	1	1	1	1	1	1	0	8	4	4	2	10	A	1	1	1
30	SUNDARAMOO	34	M	1	1	1	0	3	0	0	0	0	0	0	1	0	0	1	1	3	4	2	0	6	G	1	1	1	1

31	VIJAYKUMAR	21	M	0	0	1	0	1	0	0	1	0	0	0	0	0	0	0	0	1	2	0	0	2	A	0	0	0	0
32	SHIVA	38	M	1	0	0	1	2	0	0	0	0	0	0	0	0	0	0	1	1	4	2	2	8	G	0	0	0	0
33	PAVITHRA	25	F	0	0	1	0	1	0	0	1	0	0	0	0	0	0	0	0	1	4	2	2	2	G	0	0	0	0
34	SELVAM	40	M	1	1	1	1	4	0	0	1	1	0	0	1	0	0	1	1	5	2	0	2	4	A	0	0	0	1
35	MUTHUKUMAR	27	M	1	1	0	1	3	0		0	1	1	1	1	1	1	1	0	7	2	2	2	6	A	0	1	0	0
36	JEBASUNDAR	37	M	0	0	1	1	2	0	0	1	0	0	0	0	0	1	0	0	2	4	2	2	6	A	0	0	0	0
37	MURUGAN	52	M	0	1	1	1	3	0	0	0	0	0	0	0	0	1	0	1	2	2	2	2	6	A	1	1	1	1
38	SANTHAKUMAR	47	M	1	1	1	0	3	0	1	0	1	0	1	0	1	0	1	0	5	4	4	0	8	G	0	0	0	0
39	VASANTHI	26	F	0	0	0	1	1	0	0	1	0	0	0	0	0	0	0	0	1	2	0	2	4	G	0	0	0	0
40	ARNOLDALEX	28	M	0	0	1	0	1	0	0	1	0	0	0	0	0	0	0	0	1	4	2	2	8	A	0	0	0	0
41	MURUGAPPAN	36	M	0	0	1	1	2	0	0	1	0	0	0	0	0	0	0	0	1	4	2	2	6	A	0	0	0	0
42	ANBUMANI	25	M	0	0	1	0	1	0	0	1	0	1	0	0	0	0	0	0	2	0	0	0	2	I	0	0	0	0
43	DEVRAJ	40	M	0	1	0	0	1	0	0	0	0	0	0	0	1	1	0	0	2	2	4	2	8	A	0	0	0	0
44	PREMABALAN	24	M	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	1	1	2	0	2	4	A	0	0	0	0
45	MAHENDRAJ	33	M	0	0	1	1	2	0	1	0	0	1	0	1	0	0	0	1	3	0	2	2	4	I	0	1	1	1
46	JOHNSUNDAR	37	M	1	0	0	0	1	0	0	1	0	0	0	0	0	0	0	0	1	0	0	0	0	A	0	0	0	0
47	SUBATHRA	64	F	1	0	1	1	4	0	0	1	0	0	1	0	1	1	1	1	6	2	2	2	6	I	0	1	1	1
48	RAMAMOOTHY	60	M	0	0	0	1	2	0	0	0	1	0	0	0	0	0	0	0	1	0	0	2	2	A	0	0	0	0
49	RAMADURAI	52	M	0	1	0	0	1	0	0	0	0	0	0	1	0	0	0	0	1	2	0	0	2	G	0	0	0	0
50	ARJUN	30	M	1	0	0	0	1	0	0	0	0	1	0	0	0	0	0	0	1	2	0	0	2	A	0	0	1	1
51	RAJESH	48	M	0	1	1	0	2	0	0	1	0	0	0	0	1	0	0	0	2	0	4	2	6	A	0	0	0	0
52	JESIKUMAR	24	M	0	1	1	1	3	0	0	1	1	0	1	0	0	0	0	0	3	2	2	2	6	A	0	1	1	1
53	PERIYASAMI	25	M	0	0	0	1	1	0	0	0	0	1	0	0	0	0	0	0	1	2	4	2	8	A	0	1	0	0
54	BALAJI	52	M	0	0	1	0	1	0	0	1	0	0	0	0	0	0	0	0	1	2	0	0	2	A	0	1	0	0
55	BAKTAVACHAL	33	M	0	0	1	0	1	0	0	1	0	0	0	0	1	0	0	0	2	4	2	2	0	A	0	0	0	0
56	ARUNKUMAR	21	M	1	1	1	1	4	0	0	1	0	0	1	1	0	0	1	1	5	2	2	2	6	A	0	1	1	1
57	GOPINATH	39	M	0	0	1	1	2	0	0	0	0	1	0	0	0	0	0	0	1	2	0	2	4	A	0	0	0	0
58	DURAIRAJ	33	M	0	1	0	0	1	0	0	0	1	0	1	0	1	0	0	0	3	2	4	2	8	I	0	0	1	0
59	SANKAR	37	M	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	1	2	4	2	8	A	0	0	0	0
60	INDHUMATHI	30	F	1	0	1	1	3	0	0	1	0	0	0	0	0	0	0	1	2	2	2	2	6	G	0	0	1	1
61	GOKUL	40	M	0	0	0	1	1	0	1	0	0	1	0	0	0	0	0	1	3	2	0	2	4	A	0	1	0	0
62	MURUGAN	34	M	1	1	1	1	4	0	0	1	0	0	1	0	0	1	1	0	4	2	2	2	6	A	0	1	1	1
63	SUBATHRA	64	F	1	0	1	1	4	0	0	1	0	0	1	0	1	1	1	1	6	2	2	2	6	I	0	1	1	1
64	RAMAMOOTHY	60	M	0	0	0	1	2	0	0	0	1	0	0	0	0	0	0	0	1	0	0	2	2	A	0	0	0	0
65	RAMADURAI	52	M	0	1	0	0	1	0	0	0	0	0	0	1	0	0	0	0	1	2	0	0	2	G	0	0	0	0
66	ARJUN	30	M	1	0	0	0	1	0	0	0	0	1	0	0	0	0	0	0	1	2	0	0	2	A	0	0	1	1
67	RAJESH	48	M	0	1	1	0	2	0	0	1	0	0	0	0	1	0	0	0	2	0	4	2	6	A	0	0	0	0
68	JESIKUMAR	24	M	0	1	1	1	3	0	0	1	1	0	1	0	0	0	0	0	3	2	2	2	6	A	0	1	1	1