"A COMPARATIVE EVALUATION OF RADIOLOGIC AND CLINICAL SCORING SYSTEM IN THE EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS"

A DISSERTATION SUBMITTED TO

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CERTIFICATE

This is to certify that the dissertation titled **"A COMPARATIVE EVALUATION OF RADIOLOGIC AND CLINICAL SCORING SYSTEM IN THE EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS "** is the bonafide work done by **DR .K.ASHOK KUMAR** Post Graduate student (2013 – 2016) in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under my direct guidance and supervision, in partial fulfillment of the regulations of The Tamilnadu Dr. M.G.R. Medical University, Chennai for M.S., Degree (General Surgery) Branch - I, Examination to be held in April 2016.

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DECLARATION

I, **DR.K.ASHOK KUMAR** solemnly declare that this dissertation titled "**A COMPARATIVE EVALUATION OF RADIOLOGICAL AND CLINICAL SCORING SYSTEMS IN THE EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS**" is a bonafide work done by me in the Department of General Surgery, Government Stanley Medical College and Hospital, Chennai under the guidance and supervision of my unit chief.

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Place: Chennai

Date: September 2015   DR..K.ASHOK KUMAR

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CONTENTS

INTRODUCTION

Acute Pancreatitis is a common disorder due to development of acute inflammation of normally existing Pancreas. Acute Pancreatitis includes varying type of diseases from mild self-limiting symptoms to fulminant multi organ failure and high mortality. The overall mortality rate is 3-10%, where in 11-30% of cases with severe disease manifested as pancreatic necrosis.

Acute pancreatitis refers to an acute inflammatory process of the pancreas, usually accompanied by abdominal pain and elevations of serum pancreatic enzymes.

This syndrome is usually a discrete episode, which may cause varying degrees of injury to the pancreas, and adjacent and distant organs. The incidence of acute pancreatitis has wide variability within populations, with about 1–5 cases per 10,000 population per year. Eighty percent of the cases of acute pancreatitis in the are related to alcohol use or biliary stones.

Pancreatitis may be classified as mild, moderate, or severe based on physiological findings, laboratory values, and radiological imaging.

Mild disease is not associated with complications or organ dysfunction and recovery is uneventful. In contrast, severe pancreatitis is characterized by pancreatic dysfunction, local and systemic complications, and a complicated recovery. In addition, pancreatitis may be further classified into acute interstitial and acute hemorrhagic disease . In the first type, the gland architecture is preserved but is edematous. Inflammatory cells and interstitial edema are prominent within the parenchyma. Hemorrhagic disease is characterized by marked necrosis, hemorrhage of the tissue, and fat necrosis. There is marked pancreatic necrosis along with vascular inflammation and thrombosis.

Acute Pancreatitis was diagnosed when two of the three following criteria were met:

1. Elevated Amylase/Lipase defined as three times the upper limits of normal,

2.Radiological evidence of pancreatitis,

3. Abdominal pain.

In 1879 ,Reginaald Fitz described the classic clinico pathological features of acute pancreatitis and discussed in detail about the ineffectiveness and hazards of early operative intervention.

The reason behind the assessment of severity is mainly for practical purpose, where mild pancreatitis responds to supportive treatment very well but severe acute pancreatitis needs some intensive monitoring of numerous parameters ,specific therapeutic interventions and it has very good prognosis.

Since 1963 , several scoring systems have been created clinically and radiologically for this purpose,, including Bedside index for severity in Acute pancreatitis (BISAP) score, Acute physiology and chronic health evaluation (APACHE,1 11,111 & O) score, Medical Research Council Sepsis Scoring (MRCS) , Modified Glasgow score (IMRIE'S), Balthazar computed tomography (CT) grading , Marshall Scoring system for Organ failure. The Ranson's and Modified Glasgow score (IMRIE'S) contain's data which are not routinely collected during hospitalization. Both these study require 48 hrs to complete there by reducing the most necessary early therapeutic window period.

An ideal prognostic method should be able to differentiate between patient's with mild & severe disease, easy to use and widely available and should be accurate , and should have low inter observer variability. It should also be able to apply early in disease process so that patient who could prone to develop potential complications will be closely monitored and treated if possible empirically.

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REVIEW OF LITERATURE

HISTORY OF THE PANCREAS :

The Pancreas was generally ignored in the past, both as an organ and as a seat of disease.

The Pancreas was first discovered by Herophilus, a Greek anatomist cum surgeon ,born in 336 BC on the Asiatic side in Chalkedion.

The word pancreas was first mentioned in the writings of E ristators (310-250 B.C.). The Four hundred years later , Rufus , (1st or 2nd Century AD), an anatomist cum surgeon of Ephesus , gave the name " pancreas". Written in Greek language, the word meant " pan:all,kreas : flesh".

Galen (Claudius Galenus 138-201 AD), "Physician to the Gladiators" of Rome & the Roman Emperor,taught that the pancreas serves as a cushion to protect the large blood vessels lying behind it.

In March ,1654 , a German emigres , johann Georg Wirsung, discovered the pancreatic duct at San Francisco Monastery in Padua, Italy. But it was named by his colleague as "The Duct of Wirsung". Whereas papilla, the enlargement of that duct at its junction with the common bile duct (CBD) which projects into the second part of

duodenum, were first described bt Vater in 1724 . Santorini , in 1735 described the accessory duct that bear his name.

In 1870, Paul Langerhans (" Junior") , a student of the famous Berlin Institute of Pathology , headed by the Eminent Professor Rudolph Virchow , described the islets of the pancreas that was subsequently known as the " islets of Langerhans", an endocrine system which lies with in the pancreas .This was the first good histologic description of pancreas.

In 1893, Laguessen suggested that the islet cells produce a hormone. In 1909 Jean de mayor suggested the name " insulin " for this hormone. Eugenne Lindssay (1874-1971) was able to show the association between diabetes and failure of the islet cells and in 1901, proposed his "common channel" hypothesis.

In 1908 ,Julius Wohlgermuth , of berlin , devised a method for measuring the concentration of serum amylase (:diastase"). Which was found to be most useful for diagnosing the acute pancreatitis prior to laprotomy or autopsy.

Since 1898 , many surgeons undertook various steps for the resection of tumors of Ampulla and Head of pancreas . Allen O.Whipple (1881-1963), son of american missionaries in persia , was recognised as the

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" Father of Pancreatic Surgery" for his successful single stage surgery in pancreatic head tumors.

In 1963 , the first Marseilles Symposium favoured the development of classification system for pancreatitis. This was revised in 1984 ; at the second Marseilles Symphosium.

Finally, at the Atlanta Symphosium , in 1992 , clinically oriented Classification. In the upcoming years, we may expect further refinements in classification systems with the availability of MRI and other newer innovative technologies.

Although the disease now classified as acute pancreatitis has been known from the past , not untill the mid – 19th century did the importance of pancreas and its severity became evident . In 1889 , Fitz presented a succinct clinical and pathologic feature of acute pancreatitis . Moynihan in 1925 described " the most terrible of all calamities which occur in relation with the abdominal viscera " as acute pancreatitis ".

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GROSS ANATOMY

The pancreas is a elongated retroperitoneal organ which lies obliquely from the C- loop of the duodenum to the hilum of spleen.

The pancreas lies posterior to the stomach , roughly in the Transpyloric plane. The gland weights approximately 80 g , varying from 75-125 g and measures 15- 22 cm length in adults & lies against L1-L2 vertebra.

The pancreas has four parts:

1. The head (which includes the uncinate process),
2. The neck,
3. The body and
4. Tail
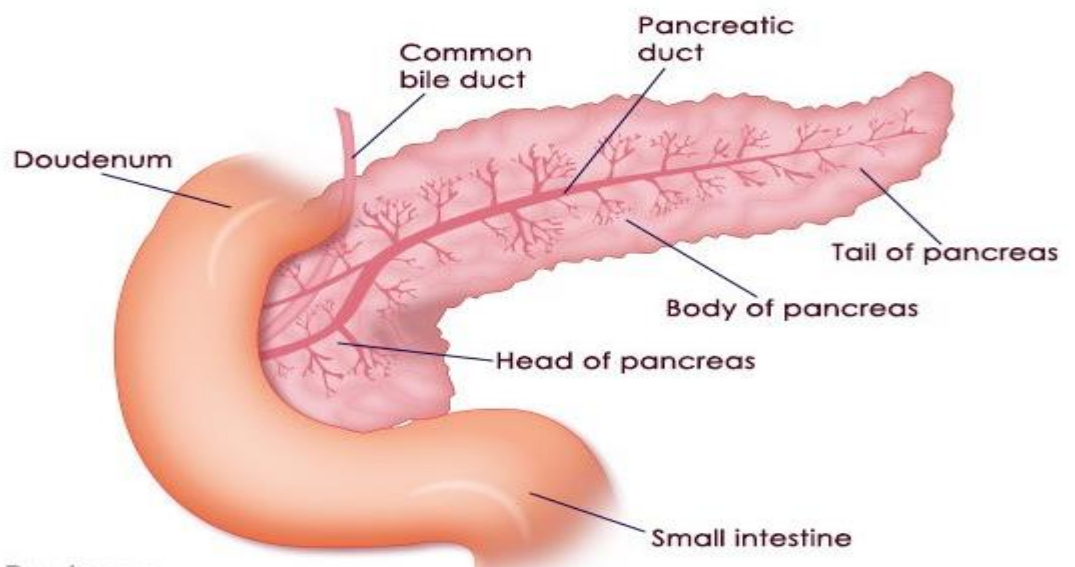
The head lies in the C- loop of the duodenum overlying the body of L2 vertebra and the inferior venacava,with the aorta beneath the neck of the gland , more medially and posterior to transverse mesocolon. The right renal artery and both renal veins lie posterior to the head. Coming off the side of the pancreatic head and passing to the left and behind the superior mesentric vein is the pancreatic uncinate process.

The neck of pancreas lie directly anterior to the portal vein . Behind the neck of pancreas , the superior mesentric vein joins the splenic vein and continues as the portal vein. The inferior mesentric vein drains into the splenic vein near its confluence with portal vein & superior mesentric vein. Inferior mesentric vein merges with the superior mesentric portal venous junction and forms a trifurcation. The common bileduct lies with in a groove in head of the gland or embedded within it, untill joining the main pancreatic duct at ampulla of vater and opens into the 2nd part of duodenum.

The body and tail of pancreas related posteriorly to splenic artery and its vein. The splenic vein lies in a groove on the posterior surface of the pancreas and draining multiple fragile pancreatic venous branches. The splenic artery which has torturous course runs parallel and just superior to the vein along the posterior superior edge of the body and

tail of pancreas, The peritoneum covers the anterior surface of the pancreatic body once the gastrocolic omentum was divided,the body and tail of pancreas can be seen along the floor of the lesser sac ,just posterior to the stomach. Pancreatic pseudocysts commonly develop in this area and the posterior aspect of the stomach can form the anterior wall of the pseudocyst , allowing drainage into the stomach.

The body of pancreas overlies the aorta at the origin of the superior mesentric artery. The neck of the pancreas overlies the vertebral body of L1 & L2 and blunt antero-posterior trauma can compress the neck of pancreas against the spine , causing parenchymal and ductal injury. The neck divides the pancreas into approximately two equal halves.

The tail is the small portion of the pancreas that lies in front of the left kidney and was nested in splenic hilum near the splenic flexure of colon.

Pancreatic Ductal Anatomy:

The common variations in the pancreatic can be appreciated by understanding the embryology . The pancreas is formed by the fusion of ventral and dorsal bud.

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1. The duct from the smaller ventral bud , which arises from the hepatic diverticulum,connects directly to the common bile duct

2. The duct from the larger dorsal bud , which arises from the duodenum , drains directly into the duodenum.

The duct of ventral anlage becomes the duct of wrisung , and from the dorsal anlage becomes the duct of santorini. The ducts from each anlage usually fuse together in the pancreatic head such that most of the pancreas drains through the Wrisung , or main pancreatic duct (MPD) , into the common channel formed from the CBD and MPD.

The length of common channel is often variable . In about one third of patients , the CBD and MPD remains distinct from the papilla : the two ducts may merge at the papilla in another third , and in the remaining   third a true common channels will be there for few millimeters.

Commonly , the duct from the dorsal anlage , the duct of santorini, persist as the lesser pancreatic duct , and sometimes drain directly into the duodenum through the lesser papilla just proximal to the major papilla. In approximately 30 % of patients , the duct of santorini ends as a blind accessory duct. In 10% of patients , the ducts of Wrisung and Santorini fail to fuse with each other. This ends up with the majority of

drainage via the duct of santorini and lesser papilla ,while the inferior part of the pancreatic head and uncinate drains via the duct of wrisung and major papilla. This normal anatomic variant , which occurs in 10 % of patients is referred as Pancreatic Divisum. The MPD is normally 2 to 3 mm in diameter and lies between the superior and inferior borders and closer to the posterior surface..

The MPD pressure inside is about twice that of in the CBD ,thus said to prevent bile reflux into the pancreatic duct.

The muscle fibre which lies around the ampulla forms the sphincter of oddi that controls the flow of biliary and pancreatic secretion into the 2nd part of duodenum

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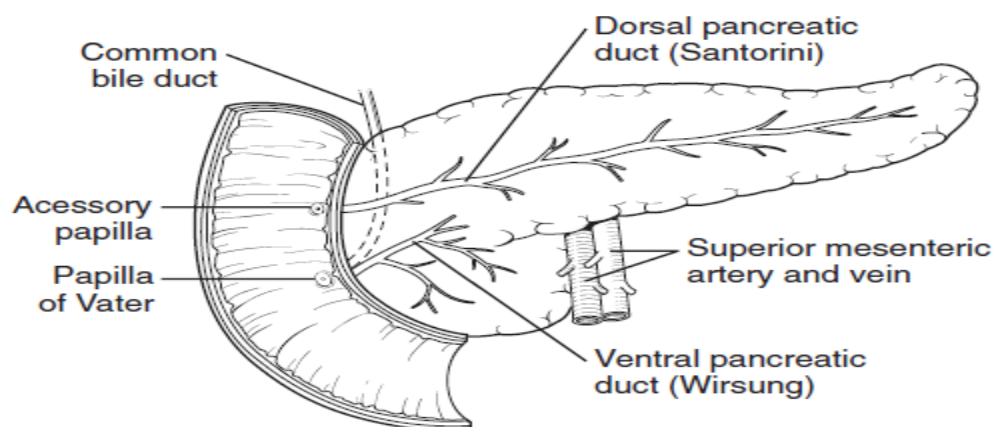
ARTERIAL SUPPLY :

The pancreatic blood supply comes mainly from the celiac axis and the superior mesentric artery.

The coeliac axis gives the common hepatic artery which in turn gives rise to gastroduodenal artery. The gastroduodenal trunk becomes superior pancreaticoduodenal artery as it passes behind the first portion of duodenum and branches into the anterior and posterior divisions. The superior mesentric artery while passes behind the neck of pancreas, it gives off the pancreaticoduodenal artery, and divides into the anterior and posterior divisions. The superior and inferior pancreaticoduodenal arteries anastomose within the parenchyma of head of pancreas along the medial aspect of C-loop of duodenum to form arcades that give off numerous branches to duodenum and head of pancreas. The body and tail are supplied by multiple branches from the splenic artery. Three vessels runs perpendicular to the axis of the pancreatic body and tail and connect the splenic artery and inferior pancreatic artery. They are from medial to lateral , the dorsal (AKA the transverse pancreatic artery) , great and caudal pancreatic arteries.These arteries form arcades within body and tail of pancreas , and account for rich blood supply to the organ.

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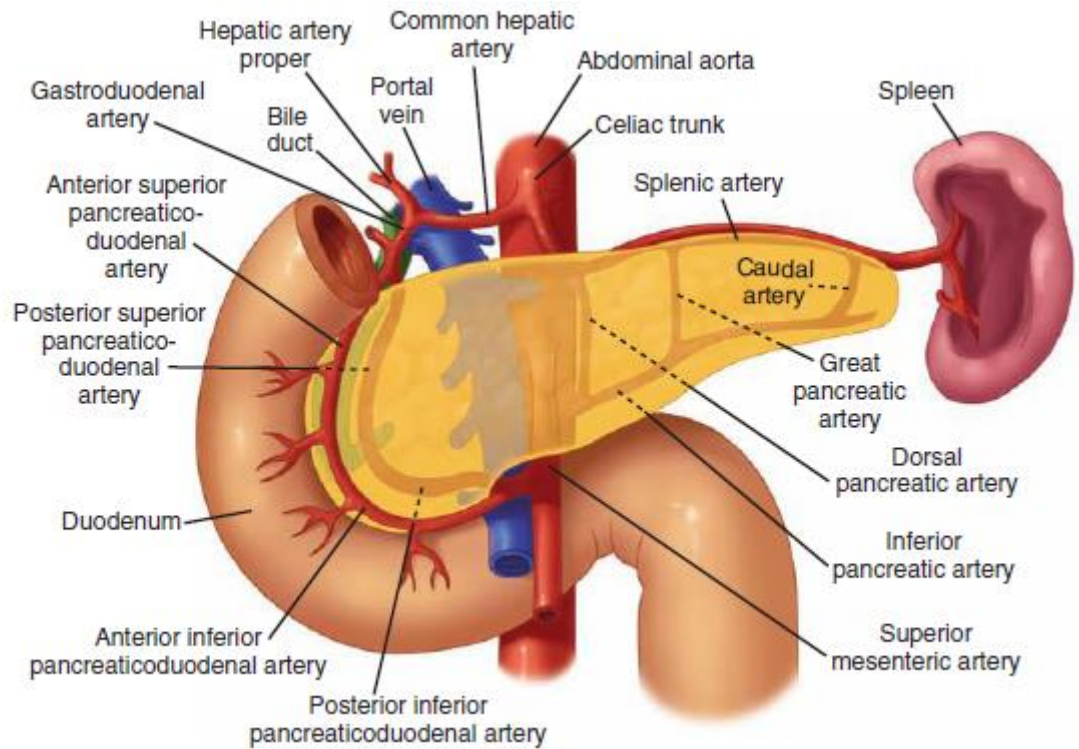
VENOUS DRAINAGE :

The venous drainage of the pancreas follow the arterial supply. The veins are superficial to the arteries within the parenchyma of the pancreas. There is an anterior and posterior arcade with in the pancreatic head. The superior vein drains directly into the portal vein and posterior inferior arcade veins directly into the inferior mesentric veins. The anterior inferior pancreaticoduodenal vein joins the right gasrtoepiploic vein and the middle colic vein and forms a common venous trunk that drains into the (SMV) superior mesentric vein, Traction on the

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transverse colon during colectomy can tear these fragile veins , which then retract into the parenchyma of the pancreas , making control tedious.There are also numerous small venous branches coming from the pancreatic parenchyma directly into the lateral and posterior aspect of portal vein. Venous return from the body and tail of pancreas drain into the splenic vein.
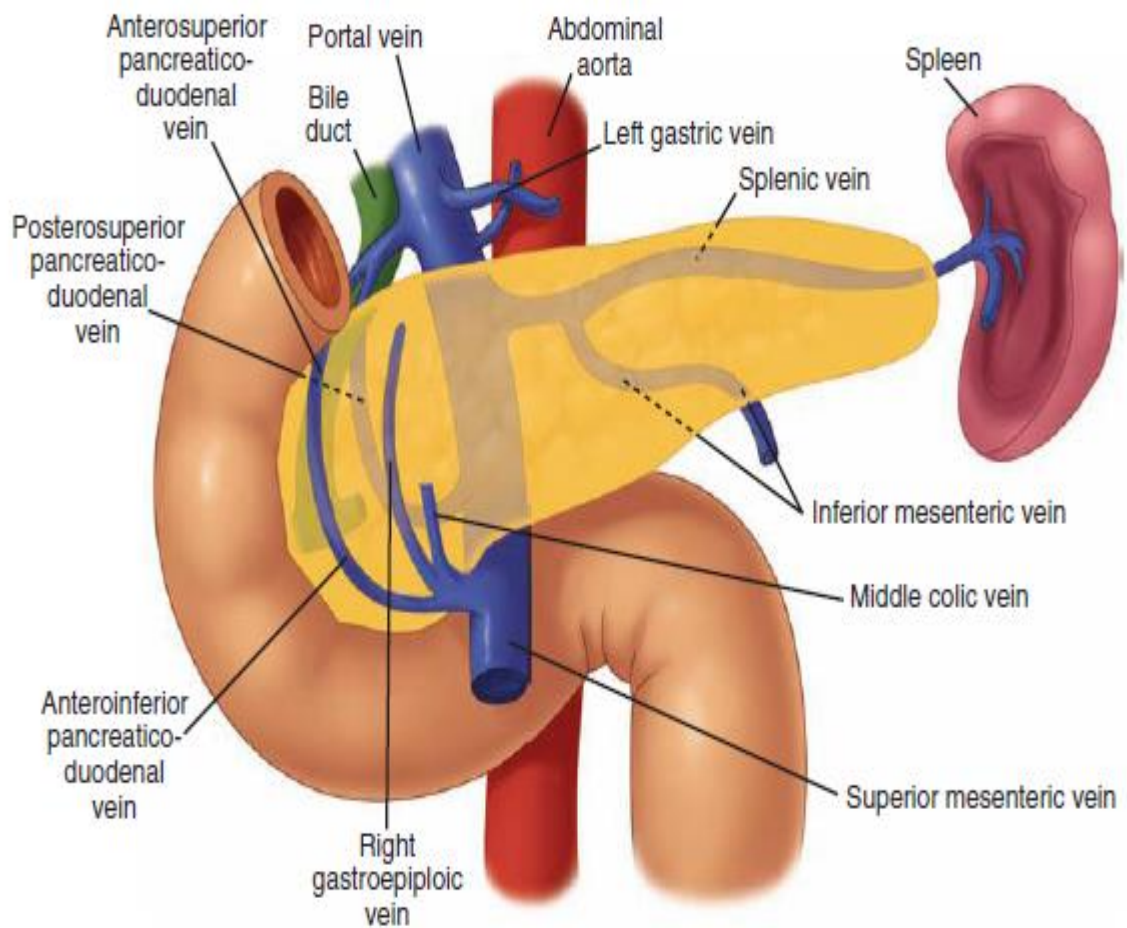
LYMPHATIC DRAINAGE :

The pancreas has rich lymphatic drainage and follows venous drainage in all directions . This diffuse lymphatic drainage contributes to the fact that pancreatic cancer often presents with positive lymphnodes and a high incidence of local recurrence after resection. Lymphnodes can be palpated along the posterior aspect of head in the pancreatic head in the pancreaticoduodenal groove , where the mesentric vein passes under the pancreatic neck , along inferior border of the pancreas , along the hepatic artery ascending into the portahepatis and along the splenic artery and vein, The pancreatic lymphatics also communicate with lymphnodes in the transverse mesocolon and mesentry of the proximal jejunum. Tumors in the body and tail often metastasize to

these   nodes   .

Celiac
lymph node
Hepatic
lymph node
Pyloric
lymph node
Spleen
Superior body
Posterior
pancreaticoduodenal
lymph node
Inferior
body
Splenic
lymph
node
Superior mesenteric
lymph node
Anterior
pancreatico-
duodenal
lymph node
Duodenum
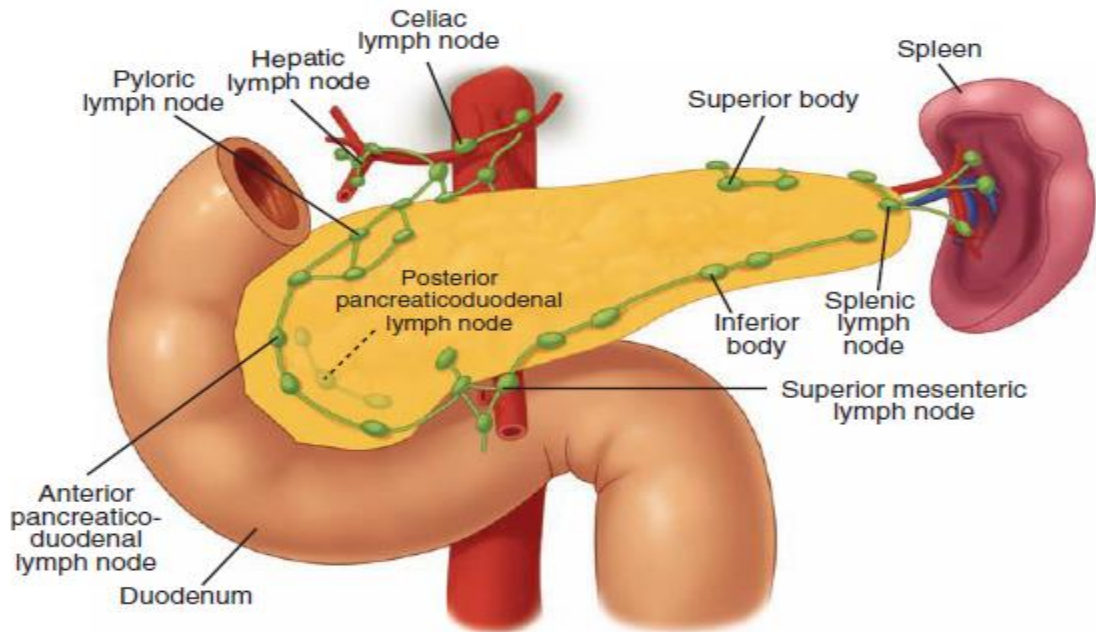
NERVE SUPPLY :

The pancreas is innervated by both sympathetic via splanchnic nerve & parasympathetic via vagus nerve . The acinar cells responsible for exocrine secretion , the islet cells responsible for endocrine secretion, and islet vasculature are innervated by both systems . The parasympathetic system stimulates endocrine and exocrine secretion and the sympathetic system inhibit secretion . The pancreas is also innervated by neurons that secrete amines and peptides , such as somatostatin , calcitonin gene – related peptide (CGRP) , vasoactive intestinal peptide (VIP) , and galanin . The exact physiological role of these neurons is not certain , may appear to affect both exocrine and

endocrine function. The pancreas also has a rich supply of afferent sensory fibres , which are responsible for the intense pain associated with advanced pancreatic cancer , as well as acute and chronic pancreatitis . These somatic fibres travel superiorly to the celiac ganglia. Interruption of these somatic fibres can stop transmission of pain sensation in pancreatic disease.
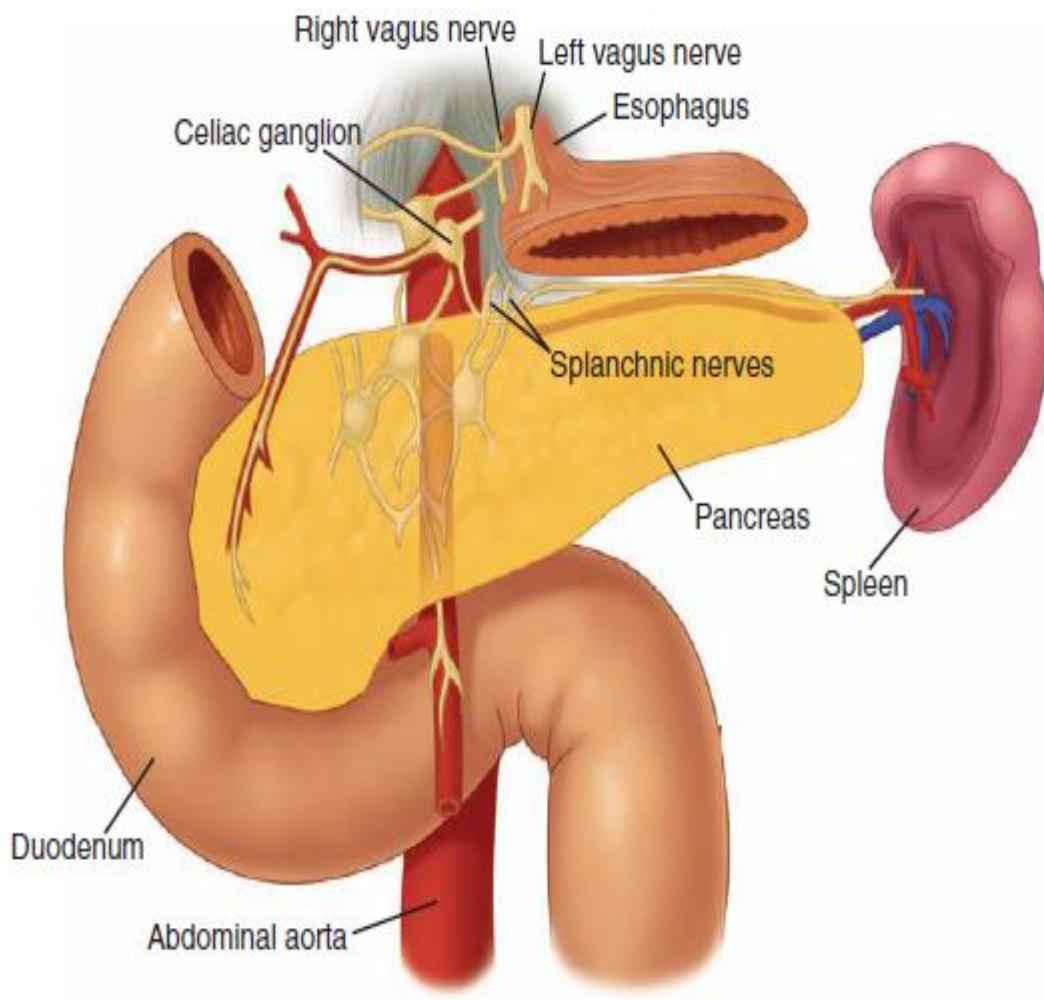
HISTOLOGY :

Pancreas has exocrine and the endocrine glandular tissues . The exocrine pancreas consists of acinar glands where as the endocrine part consists of islets of Langerhans.The pancreas contains 85% exocrine gland , 10 % extracellular matrix , and 4 % blood vessel & the major ducts , and only 2 % endocrine tissue . Thus the endocrine and exocrine pancreas is thought to be functioning separately , but coordinated well for regulating the feedback system of digestive enzyme and hormone secretion.

The acinar cells , so named because they are clustered like grapes on the stem of a vine are organised into lobules .The main duct ramifies into intralobular and interlobular ducts , ductules and finally acini , that secretes into a centrally located acinar space that communicates with the main pancreatic duct. Histologically ,acinar cells have a high content of endoplasmic reticulum and an abudance of apically located eosinophilic zymogen granules . The cells lining the main pancreatic duct are tall columnar cells and many contain mucin granules . With progression from the large ducts to the smaller intralobular and interlobular ducts ,

the lining cells become flatter , assuming a cuboidal configuration ,and mucin granules are no longer seen. Centroacinar cells, located at the junction between ducts and acini resemble acinar cells in size and shape but lack zymogen granules.

The islets of Langerhans are distributed throughout the pancreas . Within an islet , the B cells form an inner core surrounded by the other cells . Capillaries draining the islet cells drain into the portal vein forming a pancreatic portal system.

SURGICAL PHYSIOLOGY :

In response to a meal, the pancreas secrete digestive enzymes in an alkaline (Ph 8.4) bicarbonate rich fluid . The duodenal mucosa releases the hormone secretin which evokes a bicarbonate rich fluid. Cholecystokinin – pancreozymin (CCK) is released from the duodenal mucosa in response to food : CCK produces no increase in the volume of secretion , but is responsible for enzyme secretion. Vagal stimulation increases volume . Approximately 6-20 g of digestive enzymes enter the duodenum each day.

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EXOCRINE **PANCREAS :**

The pancreas secrete about 500 – 800 mL of colourless , odourless , isoosmotic , alkaline , pancreatic juice daily . Pancreatic juice is made up of secretions from ductal and acinar cells . The acinar cells secrete the enzymes that are responsible for digestion of carbohydrate , protein and fatty foods.

Pancreatic amylase is the only enzyme secreted in active form and all other enzymes are secreted in proenzymes form which requires further activation for their action . Familial pancreatitis is a condition , where there is no expression of normal trypsinogen inhibitors , like SP1NK1 or pancreatic secretory trypsin inhibitor (PSTI) .Trypsinogen is expressed in several isoform and a missense mutation on the cationic trypsinogen .

This   account

Pancreatic enzymes		
ENZYME	SUBSTRATE	PRODUCT
Carbohydrate Amylase (active)	Starch, glycogen	Glucose, maltose, maltotriose, dextrins

Protein
  Endopeptidases
    Trypsinogen (inactive) $\xrightarrow{\text{Enterokinase}}$ Trypsin (active)
    Chymotrypsinogen (inactive) $\xrightarrow[\text{Trypsin}]{\text{Enterokinase}}$ Chymotrypsin (active)
    Proelastase (inactive) $\xrightarrow[\text{Trypsin}]{\text{Enterokinase}}$ Elastase (active)
  Exopeptidases
    Procarboxy peptidase A&B (inactive) $\xrightarrow{\text{Enterokinase}}$
      Carboxypeptidase A&B (active)
	Cleave bonds between amino acids
	
	Cleave amino acids from end
	  of peptide chains
		Amino acids, dipeptides
		
		—

Fat
  Pancreatic lipase (active)
  Phospholipase A2 (inactive) $\xrightarrow{\text{Trypsin}}$ Phospholipase A2 (active)
  Cholesterol esterase
	Triglycerides
	Phospholipase
	
	Neutral lipids
		2-Monoglycerides, fatty acids
		—
		
		—

At the time of secretion from the pancreatic acini , the proteolytic enzymes are in an inactive form , the maintenance of which is important in preventing pancreatitis.

ENDOCRINE PANCREAS :

There are about 1 million pancreatic islet cells present in adults normally . The size varies from 40 – 900mm . Largest cells lie close to major arterioles and smaller cells are embedded more deeply in the parenchyma . Most islets contain five major tyoes of cells :

1. Alpha Cells – secrete glucagon (20%)

2. Beta   Cells – secretes insulin(75%)

3. D Cells – secretes somatostatin

4. G Cells –secretes ghrelin and

5. PP cells – secretes pancreatic polypeptide.

ACUTE PANCREATITIS

Definition :

Acute pancreatitis is " an inflammatory disease , associated with little or no fibrosis of the pancreas ". There are several initiating factors , which include gallstones , alcohol , trauma and infections and very rarely hereditary.

Etiology of acute pancreatitis :

There are so many different factors have been implicated in the causation of this disease . On the basis of worldwide data , the most common cause are gall stones , account for 45 % of cases. Alcoholism is the second most common cause ,in about 35 percent of cases . In a study done in New Delhi , gall stones and alcoholism were found to be the cause in 49.5% and 23.6% cases ,respectively.

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The disease occurs at higher rate in younger men and older women, Females are more to have gall stone pancreatitis and males are more prone to have alcohol induced pancreatitis.

CAUSES OF ACUTE PANCREATITIS :

Alcohol

Biliary diseases

Obstructive causes :

1. Choledocholithiasis

2. Ampullary carcinoma or pancreatic malignancy

3. Papillary obstruction by worms / foreign bodies

4. Pancreatic divisum with minor duct obstruction

5. Choledochocoel

6. Duodenal diverticula at periampullary region

7. Spasm of sphincter of oddi

Toxins or Drugs :

1. Toxins :- ethanol/methanol , scorpion sting, organophosphorous compounds

2. Drugs:- Definite cause

1. 5-Aminosalicyclic acid (ASA)

2. 6-Mercaptopurine (6-MP)

3. Azathiopurine

4. Cytosine arabinoside (cytarabine)

5. Didanosine

6. Diuretic agents

7. Estrogens , etc.

Probable cause :-

1. Acetaminophen

2. Methyldopa

3. L- Asparaginase

4. Isoniazid (INH)

5. Phenformin , etc.

Trauma :-

1. External /surgical traumatic injury to the abdomen.

2. Iatrogenic injury – postoperative trauma , post ERCP ,post

   sphincterotomy and manometry of sphincter of oddi

Metabolic abnormalities :

    1. Hypercalcemiea, 2. Hypertriglyceridemia

Inherited conditions

Infections :

1. Parasitic :- ascariasis , Clonorchis sinensis

2. Viral :- mumps , rubella , hepatitis A,B, non -A , non –B , coxsackie B ,echo virus ,adenovirus ,CMV,varicella,EBV,HIV.

3. Bacterial – mycoplasma pneumoniae ,campylobacter jejuni , Myco. Tuberculosis ,MAC, legionella pneumophilia , leptospiral infection.

Vascular causes :-

1. Hypoperfusion causing ischemia (e .g.,after major cardiac vascular surgery)

2. Athero-embolism

3. Vasculitis –SLE ,PAN , malignant hypertension

Miscellaneous causes :

1. Peptic ulcer penetration

2. Cystic fibrosis

3. Crohn's disease

4. Reye's syndrome

5. Hypothermia

Idiopathic causes

GALL STONES:

Gall stones are the leading causes of acute pancreatitis in most series (30-60%). Women are more commonly affected than man , and the peak incidence is 50 – 60 years of age.

In 1901 , Opie , at the Johns Hopkin's hospital in Balitmore documented impaction of the gall stone in ampulla of vater during the autopsy of a patient (operated on by Halsted) who had died due to gall stone pancreatitis and there by first to describe the pathogenic mechanism of gallstone induced pancreatitis .

He suggested that stone might have caused the outflow obstruction from a common ' biliopancreatic channel'. This led him to propose the " common- channel hypothesis" in which a blockage below the junction of biliary and pancreatic ducts would cause bile to flow into the pancreas , which could then be damaged by the detergent action of bile salts. Although this bile reflux theory was originally flavoured , most observers now believe that it is stone –induced pancreatic duct obstruction and ductal hypertension, rather than bile reflux triggers acute pancreatitis.

Microlithiasis (occult gall stone / biliary sludge) is a well known cause of acute pancreatitis . The diagnosis of microlithiasis should be ruled out before labeling the disease as idiopathic pancreatitis . Biliary microscopy & endosonography are recommended now a days to diagnose microlithiasis.

ALCOHOL :

The second most common etiological agent, alcohol is responsible for 30 % of all cases. In a patient with history of exposure to alcohol with absence of all other possible causes, even when the first attack of pancreatitis is considered to be related to alcoholic pancreatitis . However it is possible that first attack of alcohol – related pancreatitis

in the typical long standing alcohol user is really the first manifestation of chronic pancreatitis. The disease can recur with continuous abuse of alcoholism. The nature of alcohol that was consumed (i.e.,beer,wine,or hard liquor) is less significant than a daily intake of between 100 and 150 gm of ethanol.

Various theories have been put forward .

1. Alcohol consumption can alter the lipid metabolism and a transient hyperlipidemic state that causes hypertriglyceridemia and the generation of fatty acids as well as their ethyl ester metabolites ,that can injure the pancreas.

2. Alcohol consumption causes intrapancreatic generation of oxygen free radicals, which can injure the pancreas.

3. It promotes the secretion of pancreatic juice that is high in proteolytic enzyme content but low in enzyme inhibitor content. Enzyme activation can theoretically occur in these conditions and cause pancreatic injury.

4. The **" secretion with blockage "** mechanism is possible because ethanol causes sphincter of Oddi , leading to ductal hypotension and more important , ethanol is a metabolic toxin to pancreatic

acinar cells , where it can interfere with enzyme synthesis and secretion.

5. Secretion of enzyme- rich fluid , deficient in enzyme inhibitors could also lead to precipitation of protein and calcium with in this protein matrix , causing multiple ductal obstructions , while continued secretion can cause pressure to buildup and the formation of intraductal plugs ,which cause ductal obstruction and ductal hypertension.

6. Ethanol causes focal ischemic injury to the gland ,there by transiently decreases pancreatic blood flow.

HYPERLIPIDEMIA :

It s responsible for 1.5 – 4 % of cases. Triglyceride level > 1000 mg/dl increases the likelihood of developing pancreatitis . It is hyperlipidemia type I . IV OR V that causes pancreatitis . It has been suggested that lipase can liberate large amounts of toxic fatty acids into the pancreatic microcirculation . This could lead to endothelial injury , sludging of blood cells , and consequent ischemic states..

HYPERCALCEMIA

Hypercalcemia secondary to hyperparathyroidism or any other cause acute pancreatitis . The mechanism most likely involves hyper secretion and the formation of calcified stones intraductally.

Iatrogenic Pancreatitis

Acute pancreatitis can be associated with a number of surgical procedures , most commonly those performed on or close to pancreas , such as pancreatic biopsy , biliary duct exploration ,distal gastrectomy and splenectomy. Acute pancreatitis is associated post operatively with Billroth 11 gastrectomy and jejunostomy, in which increased intraduodenal pressure can cause backflow of activate enzymes into pancreas. However , pancreatitis also can occur in association with surgery that uses low systemic perfusion , such as cardiopulmonary bypass and cardiac transplantation . Acute pancreatitis has been reported to be associated with severe hypothermia and hypothermia associated cardiopulmonary bypass may be similarly causative . It also is possible that atheromatous emboli or ischemia may cause pancreatic injury . Most commonly , endoscopic retrograde cholangio pancreatography (ERCP) results in pancreatitis in 2 % to 10%of patients , due to direct injury and/or intraductal hypertension . Similarly manometry of sphincter of Oddi is associated with increased risk for AP.

TUMOURS

About 1 % to 2% of patients with acute pancreatitis may have pancreatic malignancy , in which an episode of acute pancreatitis could be the first sign of a periampullary tumour . In both conditions , the pancreatitis occurs probably due to blockade of pancreatic secretion and its upcoming consequences.

Drugs

For practical purposes ,it often is difficult to implicate a drug as the cause of acute pancreatitis . Many drugs can produce hyper amylasemia and /or abdominal pain , and a drug is considered to be a cause if the pancreatitis like illness resolves with its discontinuation.

Infections

Though Mumps , coxsackie virus , and Mycoplasma pneumoniae are believed to be capable of inducing acute pancreatitis by infecting the acinar cells , none of these agents has been isolated from a diseased pancreas. The antibody titres to mumps and coxsackie virus are elevated

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in about 30 % of cases with acute pancreatitis with no other identified cause . However , this elevation may be an amanestic or nonspecific response to pancreatitis.

Pathophysiology

Acute pancreatitis occurs in varying degrees of severity , the determinants of which are multifactorial . It is generally believed that acute pancreatitis is triggered by digestive enzymes which got activated inside acinar cells . This was thought to be counter acted by endogenously secreted pancreatic enzyme inhibitor . The ultimate severity depends upon the event that subsequently occurs following the acinar cell injury .

There are three reasons for this theory :

1 . The pancreas is digestible by the activated enzymes of duodenum

2. Activated digestive enzymes are found within pancreas during pancreatitis.

3.The histology of pancreas is suggestive of coagulative necrosis

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

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According to " **colocalization hypothesis** " digestive enzymes are localised in cytoplasmic vacuoles which also contain lysosomal hydrolase Cathepsin B , which is known to activate trypsinogen . Recent studies suggest that Cathepsin B activity inhibition by highly specific inhibitor , CA -074me , protects against intracellular activation of trypsinogen and hence pancreatitis . These findings suggest that the trypsinogen is activated because it erroneously colocalises in cytoplasmic vacuoles with cathepsin B.

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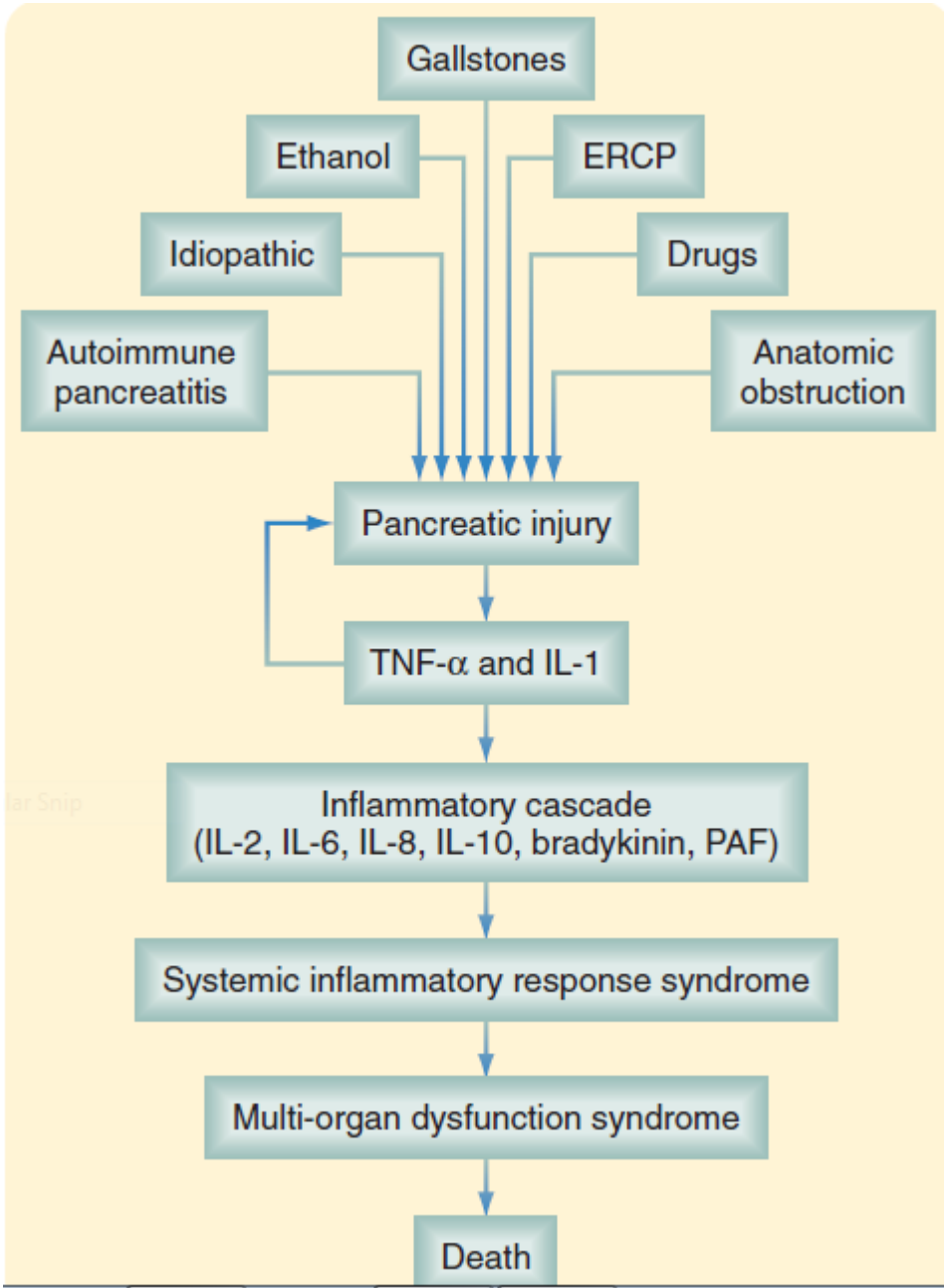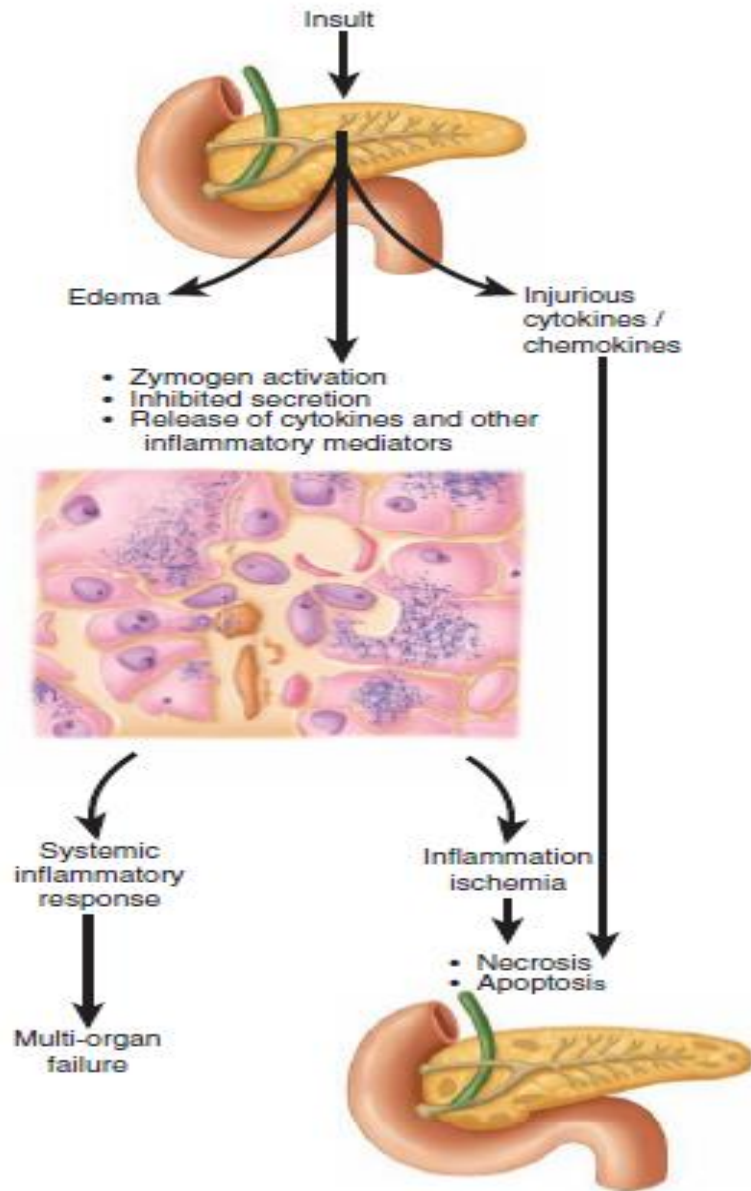
Recent studies suggest that trypsin ,once activated inside the colocalized vacuoles (appears similar to autophagic vacuoles) ,mediates the permeability of these organelles and release of their contents into the cytosol . Cathepsin B is one of the enzymes released into the cytosol during pancreatitis . Once inside the cytosol, it initiates apoptotic cell death by permeabilizing mitochondrial membranes , which allows

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cytochrome C to be released into the cytosol . This initiates the apoptotic cascade and ultimately the apoptotic death of acinar cells.
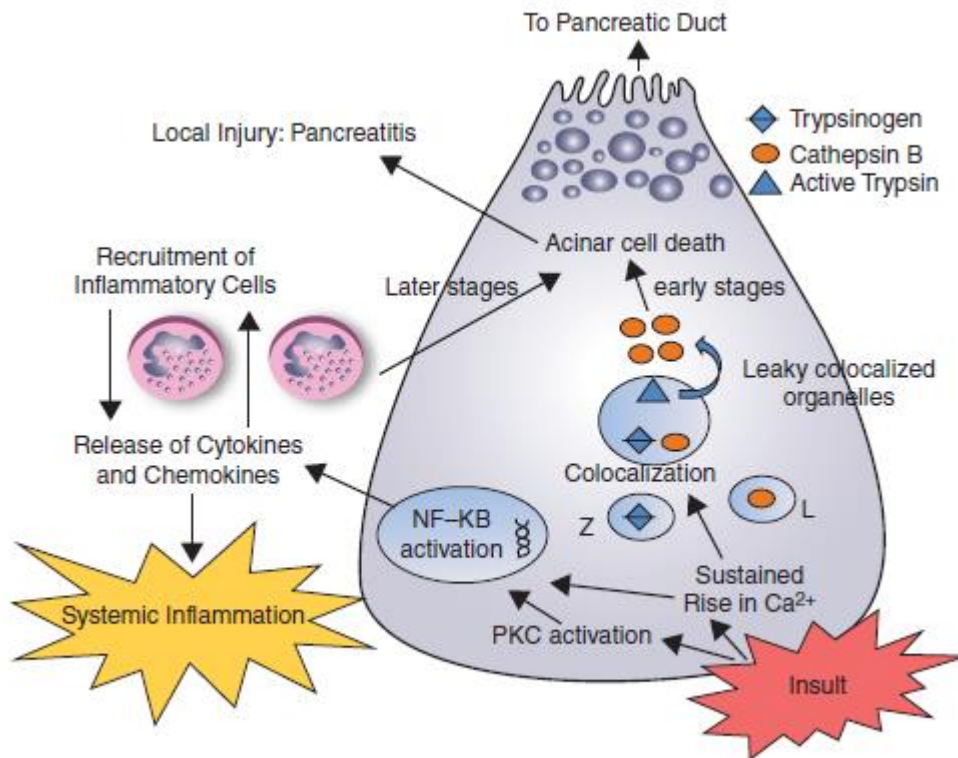
FACTORS DETERMINING THE SEVERITY OF ACUTE PANCREATITIS :

The severity of acute pancreatitis varies significantly . some may have mild form of the disease that is self limiting , while others suffer a more severe and some times lethal attack. The factors determining the severity of acute pancreatitis are multifactorial , but their identification

is of considerable therapeutic importance , because their manipulation may decrease the morbidity and mortality associated with the disease .

In addition to the cells of the immune system like neutrophils , the pancreatic acinar cells are also a source of inflammatory mediators during pancreatitis .

The list of factors associated with pancreatitis and associated lung injury include : tumour necrosis factor alpha , monocyte chemotactic protein -1 , Mob1 , Interleukin -1B ,platelet activating factor , substance p , adhesion molecules (intercellular adhesion molecule -1 ICAM -1 and selectins) ,IL-6,8,10,C5a ,the CCR1 receptor and its ligands , granulocyte –macrophage colony –stimulating factor (GMCSF) , macrophage migratory inhibiting factor , COX2 ,prostaglandin E1 ,nitric oxide (NO) and reactive oxygen species . The heat shock proteins are found to be protective in acute pancreatitis . The ultimate severity of pancreatitis and associated lung injury depends on the balance between the pro inflammatory and anti – inflammatory factors .

CLINICAL PRESENTATION :

The clinical presentation , diagnosis and management of an acute attack of pancreatitis are similar regardless of whether that attack is acute or chronic pancreatitis . The acute pancreatitis can mimic like acute abdomen and should never be excluded in differential diagnosis.

Abdominal pain , nausea and vomiting are the pre dominant symptoms . Each episode begins with severe pain , following a substantial amount of meal . The cardinal symptom is usually epigastric pain , but can occur anywhere in the lower abdomen or lower chest . The pain was described as " knifing " or " boring through " to the back , and might be relieved by leaning forward (**Mohmadian prayer position**) . Pain can occur in starts 12 – 48 hours after a bout of alcohol or after a large meal in case of gall stone pancreatitis. Pain became generalized once peritonitis has been sets in .

Peritoneal dialysis , post operative situations , legionnarie's disease are well known for the occurance of uncommon painless pancreatitis.

If patient develops generalised paralytic ileus ,abdominal distention and vomiting can occur . The vomiting may lead to gastroesophageal tears (i.e., Mallory –Weiss syndrome) and upper gastrointestinal

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bleeding. Vomiting is more intense in necrotising pancreatitis than in edematous pancreatitis . Although vomiting and retching may be relieved by passage of a nasogastric tube , the pain usually persists even after gastric decompression.

Fever is an important sign . Fever in the first week is due to acute inflammation mediated by cytokines .Fever in the second or third week is due to infected pancreatic necrosis. Fever in gall- stone induced pancreatitis , may be due to cholangitis and mandates prompt biliary decompression.

Physical findings :

On examination , the patient may be tachypnoeic , hypotensive and hyperthermic and have tachycardia . The temperature was mildly elevated in uncomplicated pancreatitis . Voluntary and involuntary guarding may present over the epigastric region . The bowel sounds may be decreased or absent . There is usually no palpable swelling or masses. The abdomen may be distended with free intraperitoneal fluid , may associated with pleural effusion , particularly on the left side.

With increasing severity , there are sequestrations of fluid in the retro peritoneum that leads to life threatening intravascular fluid loss . This

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leads to haemo concentration. There might be bleeding into retroperitoneum or peritoneal cavity which may dissect via the soft tissues and appears as a bluish discolouration around the umbilicus (Cullen's sign) or in the flanks (Grey Turner's sign) and the inguinal region (Fox's sign). Neither sign is pathognomonic of AP :actually cullen's sign was first described with ruptured ectopic gestation.

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

Diagnosis :

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

Serum pancreatic enzymes:

Serum pancreatic enzyme estimation is the gold standard for diagnosis . The reason is pancreatic acinar cell synthesize , store , and secrete a large amount of digestive enzymes (e.g., amylase , lipase , trypsinogen , and elastase), the levels of which are elevated in the serum of most patients.

Because of the ease of measurement , serum amylase levels are measured most often . Serum amylase concentration will increases immediately reaches the peak value with in several hours after the onset of disease and remains elevated for 3 to 5 days before returns back to normal. There was no significant correlation between the magnitude of serum amylase raise and severity of acute pancreatitis . But there are many nonpancreatic causes for hyperamylasemia (e.g., biliary tract disease , intestinal obstruction ,mesentric ischaemia , acute appendicitis ,mumps,parotitis ,impaired amylase excretion etc .) , that make the interpretation of this marker difficult . In contrast , a patient with acute pancreatitis may have a normal serum amylase level , which could be due to several reasons like patients with hyperlipidemia ; values might appear to be normal because of interference by lipids with chemical determination of serum amylase. The urinary amylase clearance from the circulation increases during pancreatitis ; therefore the urinary amylase levels might be more sensitive than serum levels . For these reasons ,it is recommended to measure the urinary amylase concentrations , which usually remain elevated for several days after serum amylase levels have returned back to normal . In patients with severe pancreatitis associated with significant necrotic damage , the pancreas may not release large amounts of enzymes into the circulation .

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It is important to recognize that , in patients with severe pancreatitis , frequent measurements of serum enzymes is not needed . Patients with alcoholic pancreatitis , in general have a smaller increase in serum amylase levels . Because hyperamylasemia can be observed in many extra pancreatic diseases , measuring pancreatic specific amylase (p-amylase) rather than total amylase , which also includes salivary amylase , makes the diagnosis more specific (88- 93%).

The serum lipase estimation has been found to have high sensitivity and specificity in the diagnosis as there are no other sources of lipase. Total amylase is having a sensitivity of 84% , the serum p-amylase has 95% and lipase has 93%. Specificities for amylase , P –amylase and lipase 88%,93%,96% respectively. Thus P –amylase is the enzyme with the higher diagnostic value.

IMAGING:

In general , the plain chest and abdominal radiographs can be useful in the management by identifying other causes for the patient's symptoms (e.g., pneumonia , perforated hollow viscous , mechanical bowel obstruction). Plain abdominal x- ray findings are either generalised or local ileus (known as sentinel loop) ,colon " cut off "

sign or "renal halo" sign. A chest radiograph may show left pleural effusion , elevated left hemi diaphragm or basal atelectasis.

Ultrasonography:

Abdominal ultrasound (US) examination is the gold standard for confirmation of gallstone pancreatitis . It is also helpful to detect extrapancreatic ductal dilatations and pancreatic edema , swelling , free peritoneal fluid and peri pancreatic acute fluid collections (PFCs) . It may not be sensitive in 20 % of cases .
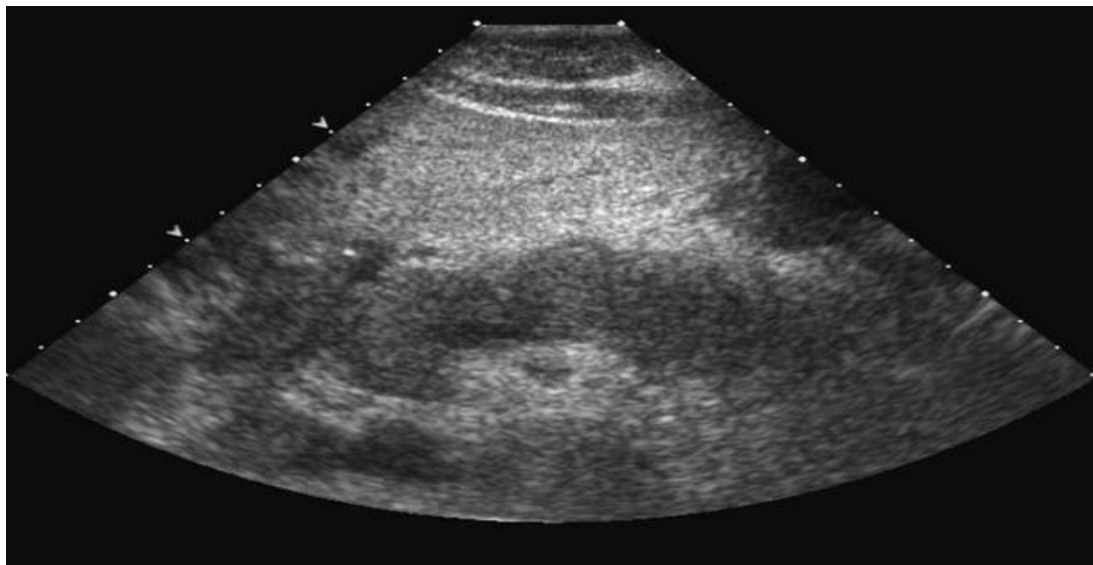
CT SCAN :

The contrast enhanced computed tomography (CECT) , has become gold standard for

1. Diagnosis

2. Assessing the severity

3. Detection of complication of acute pancreatitis.
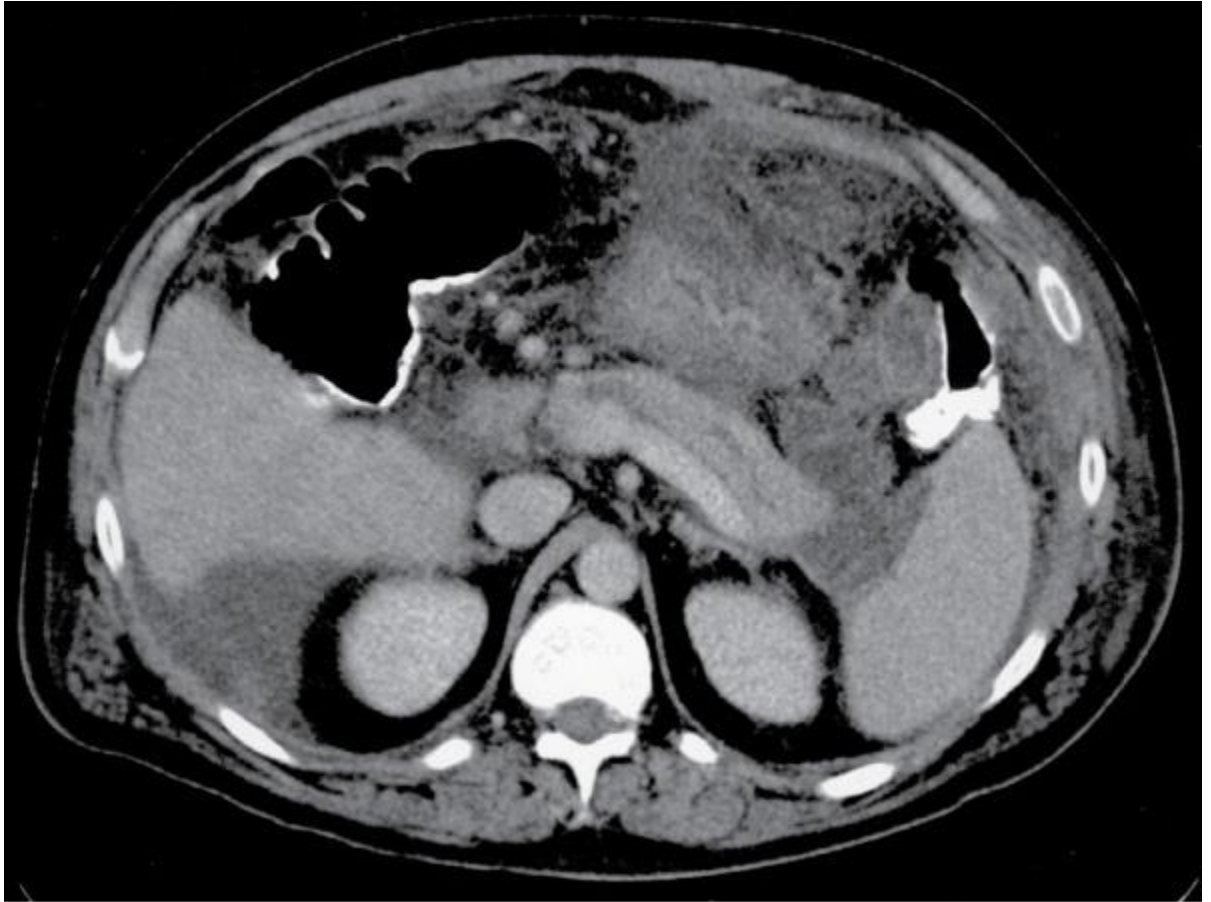
The Balthazar scoring system and other similar grading systems have incorporated various CT findings such as inflammation and fluid collections in & around the pancreas to correlate radiographic appearance with morbidity and mortality.

Early CT scans often fail to detect evolving necrosis , which become well demarcated by 2 to 3 days after the onset of symptoms , The CT scans are not useful in diagnosing necrosis or predicting the severity within 24 hours of onset of illness. The sensitivity for identifying pancreatic necrosis and image guided aspiration of necrosis , when patient not improving clinically or who experience clinical decline. In the patient with moderate renal impairment or allergy to intravenous contrast material , magnetic resonance imagining (MRI) may be useful. MRI has been found to have sensitivity and specificity similar to contrast enhanced CT for detecting severe acute pancreatitis.

ERCP should be done with acute pancreatitis, whose clinical course fails to improve despite full intensive care support , and in whom ampullary or common bileduct stone impaction is suspected , based on ultrasonography ,or clinical .biochemical signs of cholangitis. It may also be helpful in patients with recurrent attack of acute pancreatitis , without any obvious cause . It is useful in correcting potentially correctable lesions such as CBD stones with impaction , pancreas divisum , ampullary stenosis , pancreatic duct stenosis etc.,

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Assessment of severity:

An early interpretation between mild and severe necrotising pancreatitis is the most important thing for providing optimal care to the patient. There are so many predictors available for assessing the severity of acute pancreatitis which includes early prognostication signs , serum markers and CT .

Scoring system in acute pancreatitis :

The various prognostic scoring systems for assessing the severity will be discussed in detail later .

UK guidelines for management of AP :

1. The correct diagnosis has to be made with in 48 hours of admission

2. The etiology has to be determined in 80% of cases atleast and idiopathic cause should not exceed 20 %.

3. The serum lipase assay has been preferred over serum amylase for diagnosing acute pancreatitis.

4. The contrast enhanced computed tomography has to be preferred over USG for detection of presence or absence of pancreatitis.

TREATMENT:

There are two phases in evolution of acute attack of pancreatitis . Both the phases overlap each other

The initial phase , which lasts for 1 to 2 weeks involves an acute inflammatory and autodigestive phase that takes place within and around pancreas . It may have systemic phase as well.

The second phase which may last weeks or months is primarily characterised by the development of local complications that are, themselves ,the results of necrosis ,infection and pancreatic duct rupture.

The initial management of patients with acute pancreatitis focuses on early establishment of diagnosis , assessing the severity , treating the major symptoms and haltering the disease progression. The treatment for acute pancreatitis is largely supportive . since 15-30% develop severe pancreatitis , so each and every patient should be treated aggressively. The main aim of treatment is ' allowing rest to gland ' by oral and fluid restriction . The goal of initial management consist if adequate fluid management ,correction of electrolyte abnormalities , nutritional support and prevention of local and systemic complications.

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Management of pain :

Good analgesics should be given to these patients as the pain can be severe in intensity . Most patients require narcotics analgesics like Meperidine . Meperidine is preferred as morphine induces spasm of sphincter of Oddi , which can atleast theoretically worsen biliary pancreatitis.

Fluid and Electrolyte Management :

Aggressive fluid resuscitation is important to replenish extravascular or " third space" , fluid losses , which may be considerable. The fluid resuscitation is of utmost importance to prevent systemic complication , mainly acute renal insufficiency ,that may occur with hypovolemia . Transudation of the fluid from intravascular space into areas of inflammation (i.e., peripancreatic , retroperitoneum and into pulmonary parenchyma and soft tissues elsewhere in the body) is the principle cause of hypovolemia .Furthermore studies have shown that inadequate resuscitation may add upon as a significant risk that leads to pancreatic injury .

During the first several days of a several attack , circulating levels of many pro inflammatory factors , including cytokines and chemokines are elevated . This so- called **" cytokine storm ",** in many cases triggers the systemic immune response system and as a result of haemodynamic parameters of these patient may resemble those of sepsis associated with other disease states . Heart rate , cardiac output and cardiac index usually rise and total peripheral resistance falls **Nasogastric Decompression :**

The nausea and vomiting of pancreatitis can result in significant fluid as well as electrolyte losses and retching can lead to esophageal mucosal tears and result in upper gastrointestinal bleeding (Mallory – Weiss syndrome) . For symptomatic relied and to increase patient comfort , nasogastric decompression may be needed although the institution of nasogastric drainage does not show to alter the eventual outcome of an attack .

Prophylactic Antibiotics :

Infection is a serious complication of acute pancreatitis and it is the most common cause of death . It is mostly caused by enteric bacteria and was seen commonly in necrotising pancreatitis . Local infection were common with larger amounts of pancreatic necrosis and this

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increases in incidence as time progresses for at least three weeks in the course of the disease . Aerobic and anaerobic gastrointestinal floras are the primary organisms involved and infections may be either mono or polymicrobial in nature . The predominant microbes seen were E . coli (35 %),klebsiella pneumoniae (25 %) , streptococcus (25%) , staphylococcus (15%) , pseudomonas (10%) . The association of high mortality with pancreatic infection has been rationale behind the use of prophylatic antibiotics widely in patient with pancreatic necrosis . In severe pancreatitis beneficial effects have been observed with regimens that included imipenem alone , imipenem with cilastatin , metronidazole and third generation cephalosporins (cefuroxime). Because candida species are common inhabitants of upper GI tract , candida sepsis and secondary fungal infection of pancreatic necrosis is a risk in severe disease and many surgeons advocate empirical therapy with flucanazole in severe acute pancreatitis.

Nutritional support :

Classically speaking , the enteral feeding should be limited , thereby pancreatic stimulation and further pancreatic injury by the release of proteolytic enzymes can be avoided . Recent data suggests that such limitations of enteral nutrition may have been unnecessary.Most of the

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severe pancreatitis patients found to have prolonged course of illness with hypercatabolic states and ileus that led to a generous use off parenteral nutrition in them.

Treatments of Limited or Unproven value :

In patients who develop severe disease , other modalities may be tried . The antiproteases like gabexate/aprotinin , antisecretory agents like octretide and antiinflammatory drugs or PAF antagonists like Lexipafant were found to be less useful.

Treatment of Early Systemic Complications of Pancreatitis :

The pathogenesis and management of cardiovascular collapse , respiratory failure, renal failure, metabolic encephalopathy, gastrointestinal bleeding and Disseminated intravascular coagulation that complicate severe pancreatitis appear to be identical to those involved when these processes are superimposed on other disease states that are characterised by peritonitis and hypovolemia.

Cardiovascular collapse is largely caused by hypovolemia and its management requires aggressive fluid and electrolyte repletion.

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The pulmonary manifestations of pancreatitis include atelectasis and acute lung injury . The latter appears to be similar to the acute lung injury caused by other systemic processes , including septic shock , ischemia and reperfusion and massive blood transfusion . Management includes good pulmonary toilet combined with close monitoring of pulmonary function . For many patients intubation and respiratory support may be needed .

Renal failure in pancreatitis is usually prerenal and is associated with poor prognosis. In severe cases, dialysis ,usually haemodialysis may be required.

Stress induced gastric duodenal erosions account for most of the gastrointestinal bleeding, prophylaxis with antacids, H_2 receptor antagonists , or protonpump inhibitors may be appropriate.

Some patients with severe pancreatitis develop disseminated intravascular coagulation , but it rarely causes bleeding and prophylactic heparinisation is usually not indicated.

Removal of precipitating factors ,such as drugs or alcohol is appropriate . Once the acute phase has been survived, usually by the end of first week and major organ failure is under control , then local

complications become pre – eminent in the management of these patients.

Treatment of Biliary Pancreatitis :

The presence of Gall stones leading to Choledocholithiasis is recognized as a major etiological factor worldwide. Endoscopic retrograde cholangio pancreatography (ERCP) has both diagnostic and therapeutic utility in patients with biliary obstruction and cholangitis .By randomizing patients with AP to early ERCP versus no ERCP ,both Neoptolemos and colleagues , and Fan & colleagues have showed significant decrease in morbidity but there was no significant improvement in mortality with routine use of ERCP . A metacentric randomized control study in ERCP group by Folisch and colleagues recently have demonstrated increased complications and mortality rate , after excluding the patients with biliary sepsis or obstruction . It therefore , found that early ERCP may be harmful even in the absence of ongoing biliary obstruction . Magnetic resonance cholangiopancreatography (MRCP) is an additional alternative to ERCP as a diagnostic tool that avoids the risk of post procedure pancreatitis.

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In general either early intervention (Cholecystectomy) with in first 48 to 72 hours of admission , or briefly delayed intervention (after 72 hours , but during the initial period of hospitalisation) may be favoured. Cholecystectomy with intraoperative CBD exploration is probably best option for otherwise healthy patients with obstructive pancreatitis .However patients who are at high risk for surgical intervention are best treated by endoscopic sphincterotomy , with clearance of stones by ERCP.

SURGICAL MANAGEMENT : INDICATIONS AND TIMING

There are very limited indications for surgical intervention ; specifically ,intervention may be needed to address the etiology of pancreatitis or its complication . Interventions , either surgical or endoscopic ,to prevent gallstone pancreatitis are recommended in any patient with suspected choledocholithiasis . Delayed surgery is also , rarely needed for the treatment of local complications like pseudocysts.

Indications for Surgical Intervention in Necrotising Pancreatitis
Diagnostic uncertainty
Intra –abdominal catastrophe unrelated to necrotising pancreatitis such as perforated viscus

Infected necrosis documented by FNA or extraluminal gas on CT
Severe sterile necrosis
Symptomatic organised pancreatic necrosis

Diagnosis: Acute Pancreatitis
History
Serum enzymes (amylase, lipase)

Assessment of Severity
Scoring systems (APACHE II, Ranson, Glasgow)
Clinical assessment (hypotention, organ failure, leukocytosis)
Serum markers (CRP, hemoconcentration)

Mild Acute Pancreatitis
Fluid resuscitation
Initial NPO
NGT for ileus
Enteral feeding when tolerated
ERCP if biliary obstruction
CT if failure to improve (after 72 h)

Severe Acute Pancreatitis
ICU care
Aggressive fluid resuscitation
Initial NPO
NGT for ileus
Enteral feeding when tolerated
ERCP if biliary obstruction
TPN if enteral feeding not tolerated
CT scan (after 72 h)

No Pancreatic Necrosis
Manage as severe AP
Repeat CT if clinical deterioration

Pancreatic Necrosis
Prophylactic antibiotics
CT-guided FNA if clinical deterioration
(WBC, hypotention, organ failure, fever)

Negative FNA
Sterile necrosis
Observation, supportive care (above)
Repeat FNA for clinical deterioration
Consider late surgical management (>1 mo)
   for persistent symptoms

Positive FNA
Infected necrosis
Pancreatic debridement

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Early surgical intervention can lead to significant haemorrhage from the pancreatic bed , which may difficult to control , due to the fact that endarteritis obliterans was incomplete and the delineation between viable & non-viable tissue might not be clearly made out.

COMPLICATIONS :

1. **LOCAL :**

Fluid collections
Pancreatic ascites /Pleural effusion
Pancreatic pseudocyst
Pancreatic necrosis
Infected pancreatic abscess/Hemorrhage /
Pseudoaneurysm

2. **REGIONAL:**

Venous thrombosis
Paralytic ileus
Intestinal obstruction
Intestinal ischemia / necrosis
Cholestasis

3. SYSTEMIC :

A . Pulmonary
1. Pneumonitis , basal atelectasis
2. ARDS
3.Pleural effusion
B .Cardiovascular
1.Hypotension
2.Hypovolemia
3.Cardiopulmonary arrest
4.Nonspecific ECG (ST-T) changes
5.Percardial 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

SCORING SYSTEM IN ACUTE PANCREATITIS

Pancreatitis is a serious disease with high morbidity and mortality rates .Some 80% were mid attack which recovers rapidly with conservative management . The rest of 20 % were severe , with protracted course that needs intensive care and specialized management. Several predictors of severity are commonly used for this purpose.

Scoring system can be used to predict mortality , severity of disease and intensity of complications. Prognostic factor analysis found to helpful in comparing the results , in-between the series of patients under study.

Several scoring scales exist that predict both mortality and morbidity in patients with acute pancreatitis.

These system include :

1. Ranson's criteria

2. Balthazar computed tomography (CT) grading

3. Imrie Glasgow coma scale (GCS)

4. Bank's clinical criteria

5. Simplified acute physiology score (SAPS)

6. Acute physiology and chronic health health evaluation evaluation (APACHE) I,II, III & O

7. Modified CT severity index (MCTSI)

The GCS and Ranson's multiple scoring systems require 48 hours of data collection ; however , APACHE can be calculated at any time and shows prognostic correlation with acute pancreatitis , as increasing scores are associated with poor prognosis.

Once the acute pancreatitis has been diagnosed , assessment of severity is extremely important for execution of appropriate measures , Preferably in an ICU setup with close monitoring.

1. RANSON'S CRITERIA :

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

Gall stone induced pancreatitis :

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

Ranson's prognostic signs of pancreatitis	
Criteria for acute pancreatitis not due to gallstones	
At admission	During the initial 48 h
Age >55 y	Hematocrit fall >10 points
WBC >16,000/mm^3	BUN elevation >5 mg/dL
Blood glucose >200 mg/dL	Serum calcium <8 mg/dL
Serum LDH >350 IU/L	Arterial PO$_2$ <60 mm Hg
Serum AST >250 U/dL	Base deficit >4 mEq/L
	Estimated fluid sequestration >6 L
Criteria for acute gallstone pancreatitis	
At admission	During the initial 48 h
Age >70 y	Hematocrit fall >10 points
WBC >18,000/mm^3	BUN elevation >2 mg/dL
Blood glucose >220 mg/dL	Serum calcium <8 mg/dL
Serum LDH >400 IU/L	Base deficit >5 mEq/L
Serum AST >250 U/dL	Estimated fluid sequestration >4 L

2. IMRIE'S PROGNOSTIC CRITERIA :

During initial 48 hours

WBC count >15000/mm3

Blood sugar >10mmol/L

Serum Urea > 16mmol/L (no response to IV fluids)

Po2 level < 60 mmhg

Serum ca level < 2mmol/L

Lactic dehydrogenase >600 IU/L

AST/ALT >200

Serum albumin < 32 g/L

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

3.BANK'S CLINICAL CRITERIA :

Cardiac	Shock,Tachycardia,Arrhythmia,ECG changes
Pulmonary	Dyspnoea,basal rales ,PO2<60mmHG,ARDS
Renal	Urineoutput<50ml/h,rising BUN&creatinine
Metabolic	Low Ca2+&Ph : Decreased Albumin
Hematological	Decreased HCT,DIC

Neurological	Cerebral Irritation & Confused state
GIT	Paralytic ileus , free fluid , hemorrhagic tap

If the score is >1 the disease was severe in intensity.

4.BALTHAZAR COMPUTED TOMOGRAPHY SEVERITY INDEX:CTSI

Emil j. Balthazar et al , developed CTSI a grading system used to determine the acute pancreatitis severity

Grade	Balthazar Score	Points
A	Normal pancreas	0
B	Pancreatic enlargement	1
C	Pancreatic inflammation and/or peripancreatic fat	2
D	Single peripancreatic fluid collection	3
E	Two or more fluid collections and/or retroperitoneal air	4
	Percentage necrosis	
	0	0
	< 30	2
	30 – 50	4
	> 50	6
	CT Severity Index	
	Low degree	0 – 3
	Middle degree (6% mortality)	4 – 6
	High degree (17% mortality)	7 - 10

MODIFIED CT SEVERITY INDEX:

Prognostic Indicator	Points
Pancreatic inflammation	
Normal pancreas	0
Intrinsic pancreatic abnormalities with or without inflammatory changes in peripancreatic fat	2
Pancreatic or peripancreatic fluid collection or peripancreatic fat necrosis	4
Pancreatic necrosis	
None	0
≤ 30%	2
> 30%	4
Extrapancreatic complications (one or more of pleural effusion, ascites, vascular complications, parenchymal complications, or gastrointestinal tract involvement)	2

Modified CT severity score

1.score >/= 8 is considered as a severe pancreatitis

2.Score < 8 is considered as mild pancreatitis

5.MODIFIED GLASCOW SCORE :

This one was useful in both alcoholic and biliary pancreatitis .

The score more than 3 means severe disease requires ICU care.

PaO$_2$	<8kPa or < 60 mmHG

Age	>55 years
Neutrophils	>15 x 10/L
Ca$_2$+	<2mmol/L or < 8 mg/dl
Urea	>16mmol/L or > 45mg/dl
LDH/AST	>600IU/L / >200IU/L
Albumin	<3.2g/dl
Sugar	>10mmol or >180 mg/dl

Modified Marshall Scoring System

Organ System	0	1	2	3	4
Respiratory (Pao_2/Fio_2)	>400	301-400	201-300	101-200	<101
Cardiovascular (systolic blood pressure [mm Hg])	>90	<90; fluid responsive	<90; not fluid responsive	<90; pH <7.3	<90; pH <7.2
Renal					
Serum creatinine (µmol/L)	<134	134-169	170-310	311-439	>439
Serum creatinine (mg/dL)	<1.4	1.4-1.8	1.9-3.6	3.6-4.9	>4.9

5. **CRITERIA FOR ORGAN FAILURE BASED ON MARSHALL SCORING SYSTEM :**

The APACHE II Score

Physiologic Variable	High Abnormal Range				0	Low Abnormal Range			
	+4	+3	+2	+1	0	+1	+2	+3	+4
Rectal Temp (°C)	≥41	39-40.9		38.5-38.9	36-38.4	34-35.9	32-33.9	30-31.9	≤29.9
Mean Arterial Pressure (mmHg)	≥160	130-159	110-129		70-109		50-69		≤49
Heart Rate	≥100	140-179	110-139		70-109		50-69	40-54	≤39
Respiratory Rate	≥50	35-49		25-34	12-24	10-11	6-9		≤5
Oxygenatation a) FIO_2≥0.5 record A-aDO_2 b) FIO_2<0.5 record PaO_2	≥500	350-499	200-349		<200 PO_2>70	PO_2 61-70		PO_2 55-60	PO_2<55
Arterial pH	≥7.7	7.6-7.69		7.5-7.59	7.33-7.49		7.25-7.32	7.15-7.24	<7.15
HCO_3 (mEq/l)	≥52	41-51.9		32-40.9	22-31.9		18-21.9	15-17.9	<15
K (mEq/l)	≥7	6-6.9		5.5-5.9	3.5-5.4	3-3.4	2.5-2.9		<2.5
Na (mEq/l)	≥100	160-179	155-159	150-154	130-149		120-129	111-119	≤110
S. Creat (mqm/dl)	≥3.5	2-3.4	1.5-1.9		0.6-1.4		<0.6		
Hematocrit (%)	≥60		50-59.9	46-49.9	30-45.9		20-29.9		<20
TLC (10^3/cc)	≥40		20-39.9	15-19.9	3-14.9		1-2.9		<1
GCS									

Age -score
<44 → 0
45-54 → 2
55-64 → 3
65-74 → 5
≥75 → 6

GCS:
15 → 0   14 → 1   13 → 2
12 → 3   11 → 4   10 → 5
9 → 6   8 → 7   7 → 8
6 → 9   5 → 10   4 → 11
3 → 12

JAMA 1993;270(24):2957-2963

8.BISAP (The Bedside Index for Severity in AP):

This new scoring system has been developed recently for early detection of patients with risk of in hospital mortality.

The BISAP score has been developed and validated retro respectively

1

On a large population based study , done by Cardinal Health Clinical Outcomes Research Database , Marlborough ,USA .

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

The BISAP includes :

1.Blood urea nitrogen (BUN)>25mg/dl

2.Impaired mental status (GCS<15)

3. SIRS

4.Age>60 yrs

5.Pleural effusion

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

1.Pulse rate >90/min

2.Respiratory rate >20/min or $PaCO_2$ <32mmHg.

3.Temperature>100.4F or <96.8F/<36 or >38

4. WBC count >12,000 0r < 4,000cells/mm or presence of more than 10 % immature blasts.

(SIRS – Systemic Inflammatory Response Syndrome)

One point will be given for each variable present for a total of 5 score ranges from 0 to 5.

The presence of pleural effusion was determined by a CT scan. Chest radiograph or abdominal ultra sound obtained within 24 hours of presentation . Imaging obtained within24 hours of presentation at the hospital of origin for transferred patients was also collected and reviewed.

A BISAP score or more has been found to have high mortality and have predicted the necrosis and organ failure very well .

ADVANTAGES:

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

2.The scores prediction ability was tested across 390 hospitals among large number (36,248) of populations ,in contrast to other studies which were based on small number of patients.

3. This predicts in- hospital mortality

1

DISADVANTAGES :

1. The Glasgow Coma Scale used for evaluating mental status was subject to inter observer variation.

2. It could not discriminate transient from persistent organ failure within 24 hrs of hospitalization.

3. This could not predict the preventable complications of acute pancreatitis like any other scoring system.

AIMS AND OBJECTIVES OF THE STUDY

Acute pancreatitis is a catastrophic condition with many complications and poses a great challenge to the treating surgeon. 10 - 20% of the patients who develop complications will not recover with simple supportive therapy. Hence the early identification of clinically severe acute pancreatitis is critical for the triage and treatment of patients.

The aim of this study was to compare the accuracy of computed tomography(CT) and clinical scoring systems for predicting the severity of acute pancreatitis on admission and to correlate the outcome of the study with the scores observed ,in terms of disease severity and mortality.

MATERIALS & METHODS

MATERIALS AND METHODS

Study Design : Comparative Analytical study

Setting : Department of General Surgery ,Govt Stanley Medical College and Hospital , Chennai. The study was conducted after obtaining the Institutional Ethical Committee approval **(annexure 2)**

INCLUSION CRITERIA :

- Characteristic abdominal pain.

- Serum amylase/lipase (>3 times of its normal value).

- Presents with in 24 hours of onset of symptoms

- Age : 30 to 70

- Chest x – ray and Abdominal x- ray taken

EXCLUSION CRITERIA:

- Pancreatic abscess

- Pancreatic pseudocyst

- Pancreatic necrosis

- Co morbidities: copd ,bronchial asthma,DM,HT,CAD

1

- Patients presenting more than 24 hours of onset of pain

- CKD and renal failures patients

- CVA patients

- Salivary gland disease, bowel obstruction, myocardial infarction, cholecystitis, perforation.

METHODS :

First 50 patients attending the surgical emergency ward with clinical features of Acute Pancreatitis are evaluated clinically and subjected to laboratory and radiological investigations as per the designed proforma **(annexure 1).** Data pertinent to the scoring systems will be recorded with in 24 hours af admission to the hospital.

Once diagnosis is established the patient disease severity will be assessed by following two scoring system.

Statistical Analysis : Appropriate statistical tools.

For each of 50 patients included in the study , BISAP and MCTSI scores were calculated by the manner described by Knaus et al and Cardinal Health Database system .

1

Patients were classified to have mild or severe acute pancreatitis according to the definitions set by Atlanta Classification guidelines (1992).

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

Biliary Pancreatitis was presence of gall stones /biliary sludge in the gallbladder or bileduct , which was documented by any radiological methods. Alcoholic Pancreatitis was considered ,when the patient found to have regular high intake of alcohol daily , or if there was binge of alcohol consumption prior to the onset of illness and has no signs of other etiologies present . Idiopathic pancreatitis was the one with no identifiable etiological factor based on history , or after initial investigations.

Patient were observed prospectively until discharge or death.

BISAP score more than or equal to 3 and MCTSI score more than or equal to 8 were expected to predict severe Acute Pancreatitis.

1

OBSERVATION & RESULTS

OBSERVATION & RESULTS

This study was conducted in the department of general surgery , Govt Stanley Medical College & Hospital , Chennai for a period of one year . The 50 persons with features of acute pancreatitis were enrolled in this study after obtaining an informed consent.

Table : 1 Age distribution

Age distribution

	31-40	41-50	51-60	61-70	Total
No. of Patients	10	15	7	18	50
Percentage(%)	20	30	14	36	100
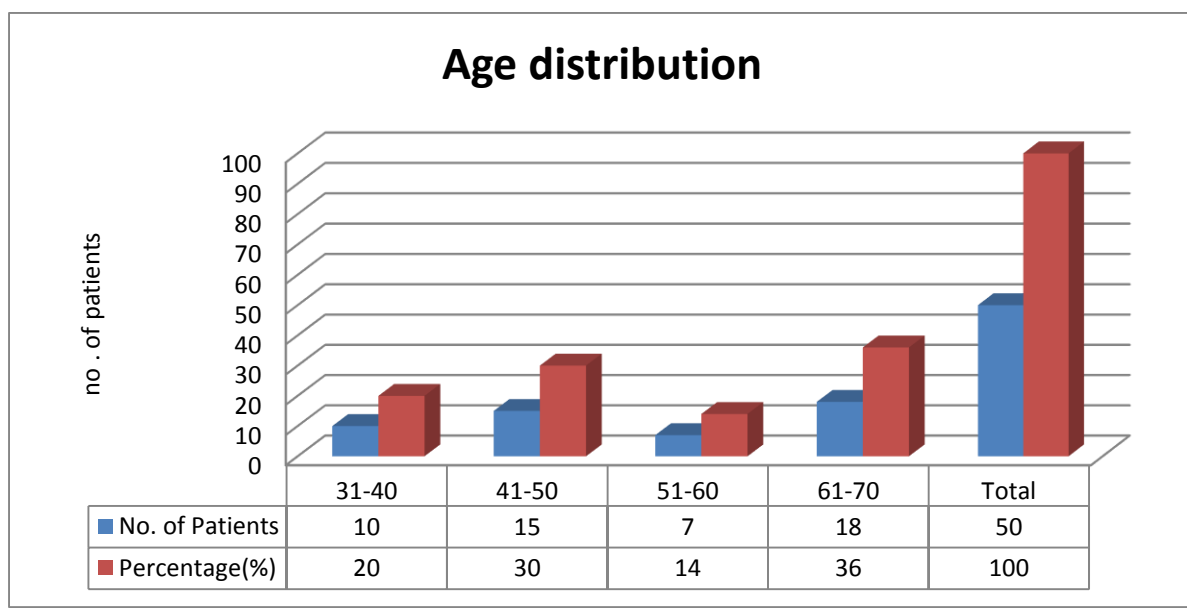
The age group of patients enrolled in this study ranges from 30 to 70 yrs. The peak incidence of the disease was noted in the 6th decade of life.

Table :2 Gender Distributions:

Out of 50 patients enrolled in this study there were 45 male and 5 female patients.

GENDER DISTRIBUTION

	MALE	FEMALE	TOTAL
No. of patients	45	5	50
Percentage(%)	90	10	100
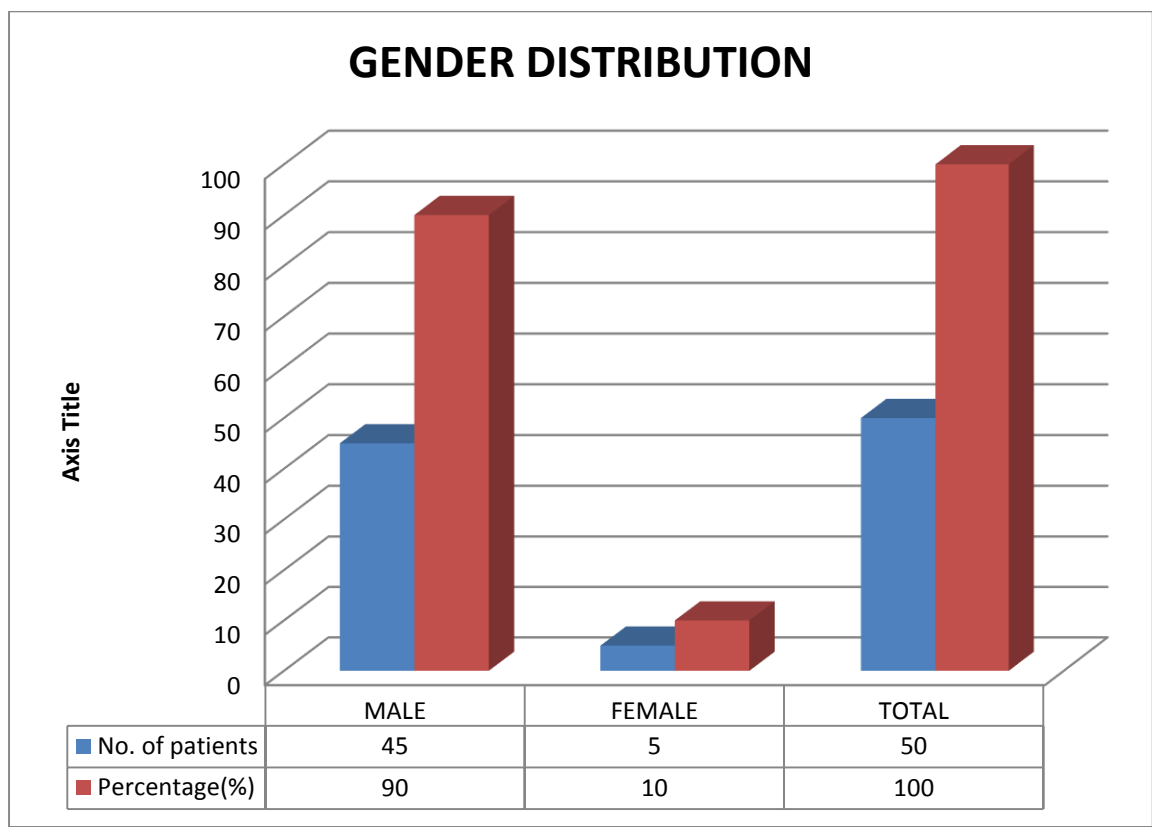
Table 3 :Length of hospital stay:

Length of Hospital Stay

	1-7 days	8-14 days	15-21 days	22-28 days	>28 days	total
No. of Patient's	31	10	3	3	3	50
Percentage(%)	62	20	6	6	6	100
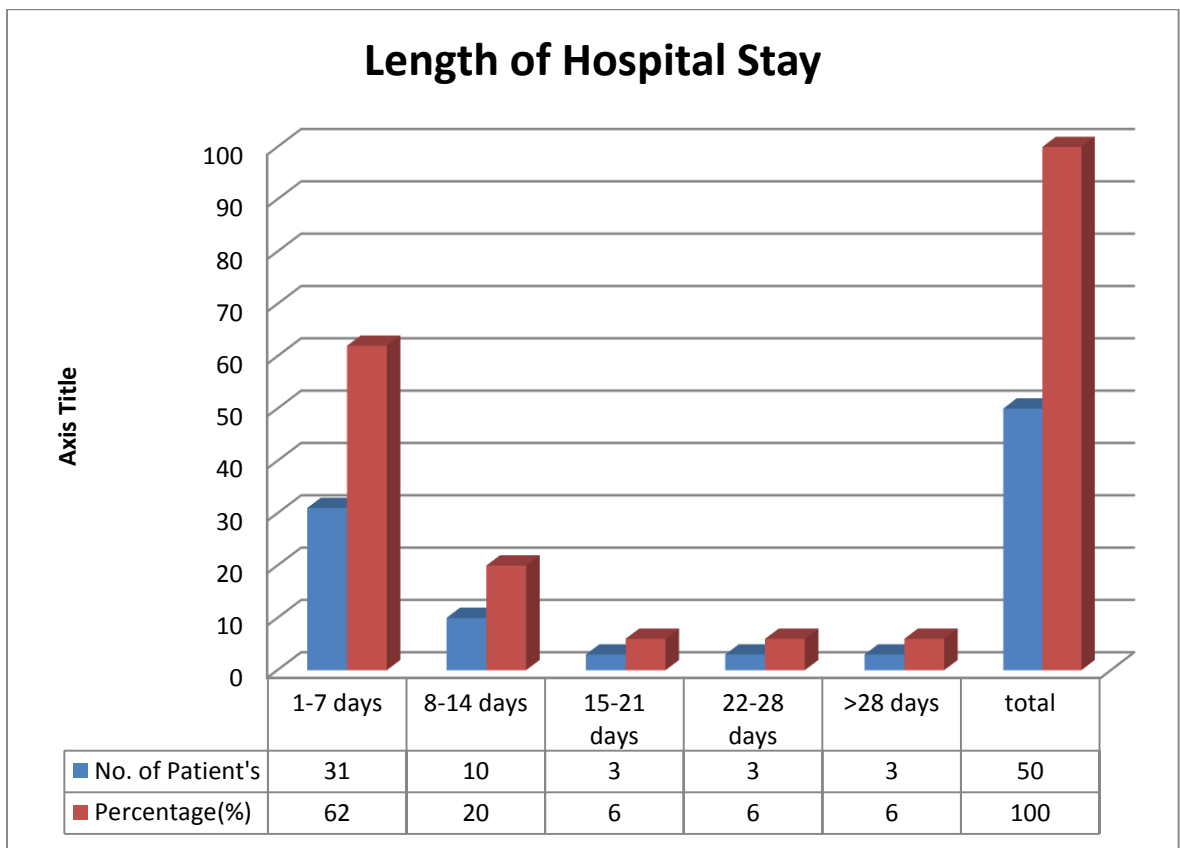
The length of hospital stay ranges from 1 day to 30 days.

The Mean length of hospital stay was 8.32+/- 7.742 days in this study , increasing BISAP & MCTSI scores was correlated well with the duration of hospital stay.

1

The most common presentation was predominantly abdominal pain(100%),vomiting (74%),fever (64%),jaundice (18%). Other manifestations.

clinical feature's

- pain — 100%
- vomiting — 74%
- fever — 60%
- jaundice — 18%
- amylase — 100%

ETIOLOGY

- Alcohol — 68%
- Gsd — 20%
- Triglycerides — 2%
- Idiopathic — 8%
- Drug induced — 2%
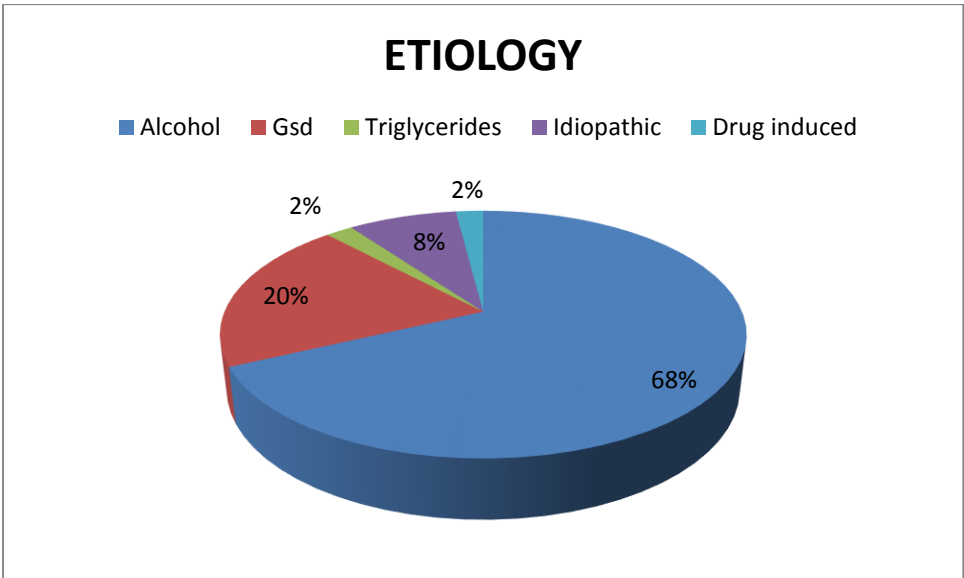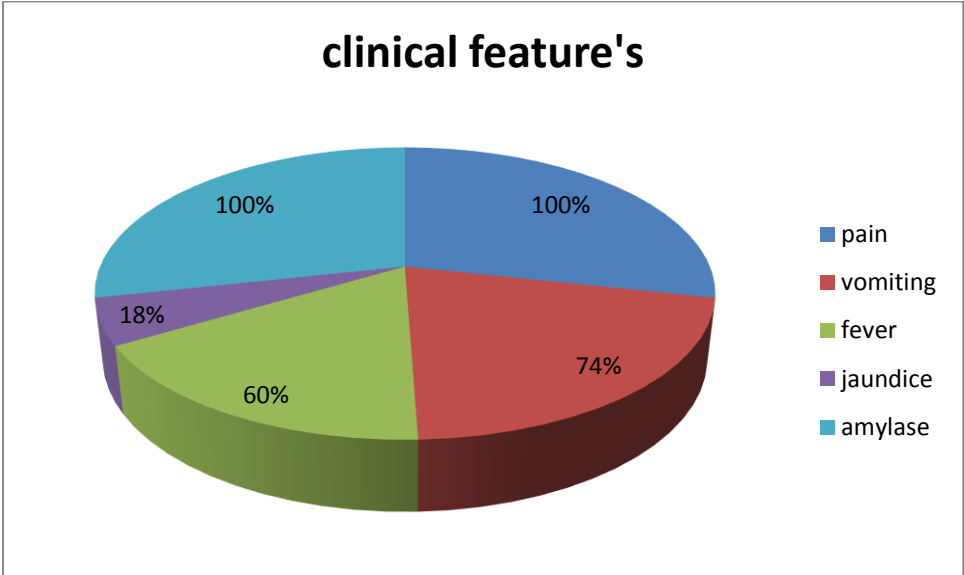
AGE

Descriptive Statistics

	N	Minimum	Maximum	Mean	Std. Deviation
AGE	50	35	68	52.26	10.222
Valid N (listwise)	50				

AGE GROUP

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	30-45	16	32.0	32.0	32.0
	46-60	16	32.0	32.0	64.0
	>60	18	36.0	36.0	100.0
	Total	50	100.0	100.0	

AGE GROUP2

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1.00	11	22.0	22.0	22.0
	2.00	14	28.0	28.0	50.0
	3.00	7	14.0	14.0	64.0
	4.00	18	36.0	36.0	100.0
	Total	50	100.0	100.0	

Correlations

		BISAP	MCTSI
BISAP	Pearson Correlation	1	.904**
	Sig. (2-tailed)		.000
	N	50	50

1

MCTSI	Pearson Correlation	.904**	1	
MCTSI	Sig. (2-tailed)		.000	
	N		50	50

**. Correlation is significant at the 0.01 level (2-tailed).

Case Processing Summary

OUTCOME	Valid N (listwise)
Positive[a]	6
Negative	44

Larger values of the test result variable(s) indicate stronger evidence for a positive actual state.

a. The positive actual state is DEAD.

Coordinates of the Curve

ROC MORTALITY

ROC Curve
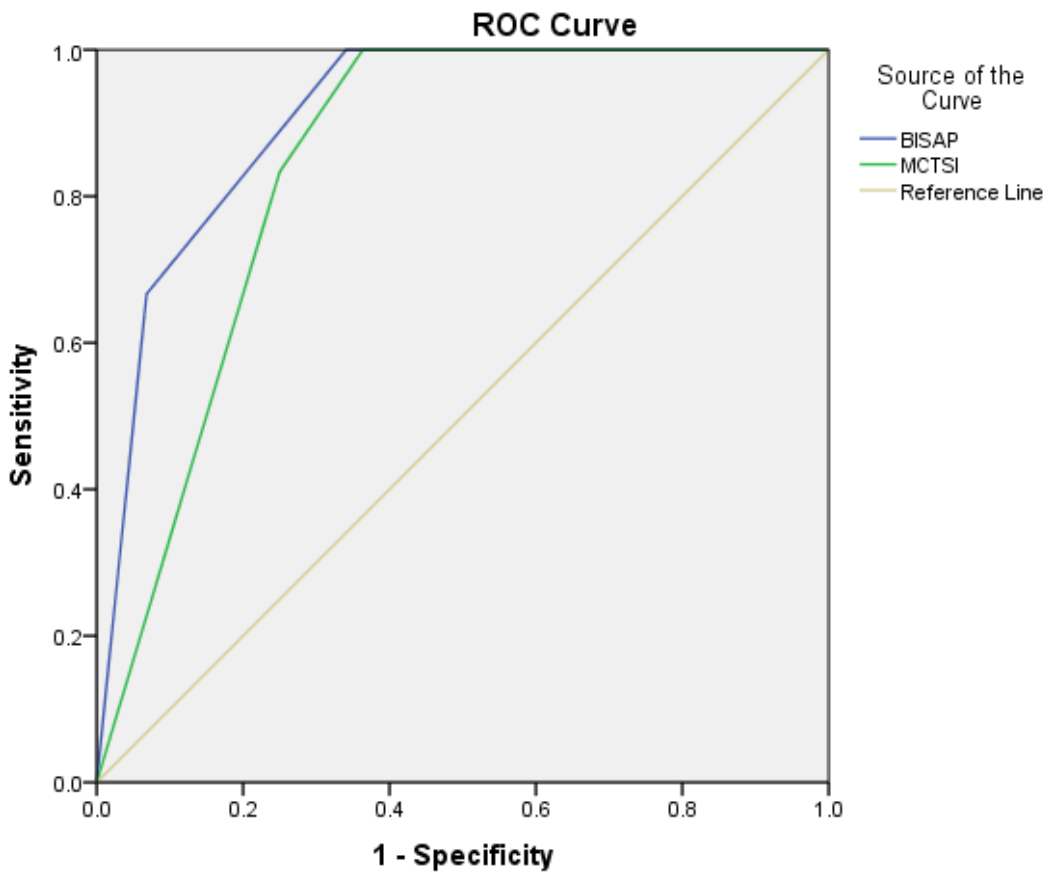
Diagonal segments are produced by ties.

BISAP GROUP * OUTCOME Crosstabulation

1

| | | OUTCOME | | Total |
		ALIVE	DEAD	
BISAP GROUP	**1.00** Count	20	0	20
	% within BISAP GROUP	100.0%	0.0%	100.0%
	% within OUTCOME	45.5%	0.0%	40.0%
	% of Total	40.0%	0.0%	40.0%
	2.00 Count	24	6	30
	% within BISAP GROUP	80.0%	20.0%	100.0%
	% within OUTCOME	54.5%	100.0%	60.0%
	% of Total	48.0%	12.0%	60.0%
Total	Count	44	6	50
	% within BISAP GROUP	88.0%	12.0%	100.0%
	% within OUTCOME	100.0%	100.0%	100.0%
	% of Total	88.0%	12.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	4.545[a]	1	.033		
Continuity Correction[b]	2.849	1	.091		
Likelihood Ratio	6.668	1	.010		
Fisher's Exact Test				.069	.037
N of Valid Cases	50				

a. 2 cells (50.0%) have expected count less than 5. The minimum expected count is 2.40.

b. Computed only for a 2x2 table

ROC COMPLICATIONS

Case Processing Summary

COMPLICATIONS	Valid N (listwise)
Positive[a]	25
Negative	25

1

Larger values of the test result variable(s) indicate stronger evidence for a positive actual state.

a. The positive actual state is PRESENT.

ROC Curve
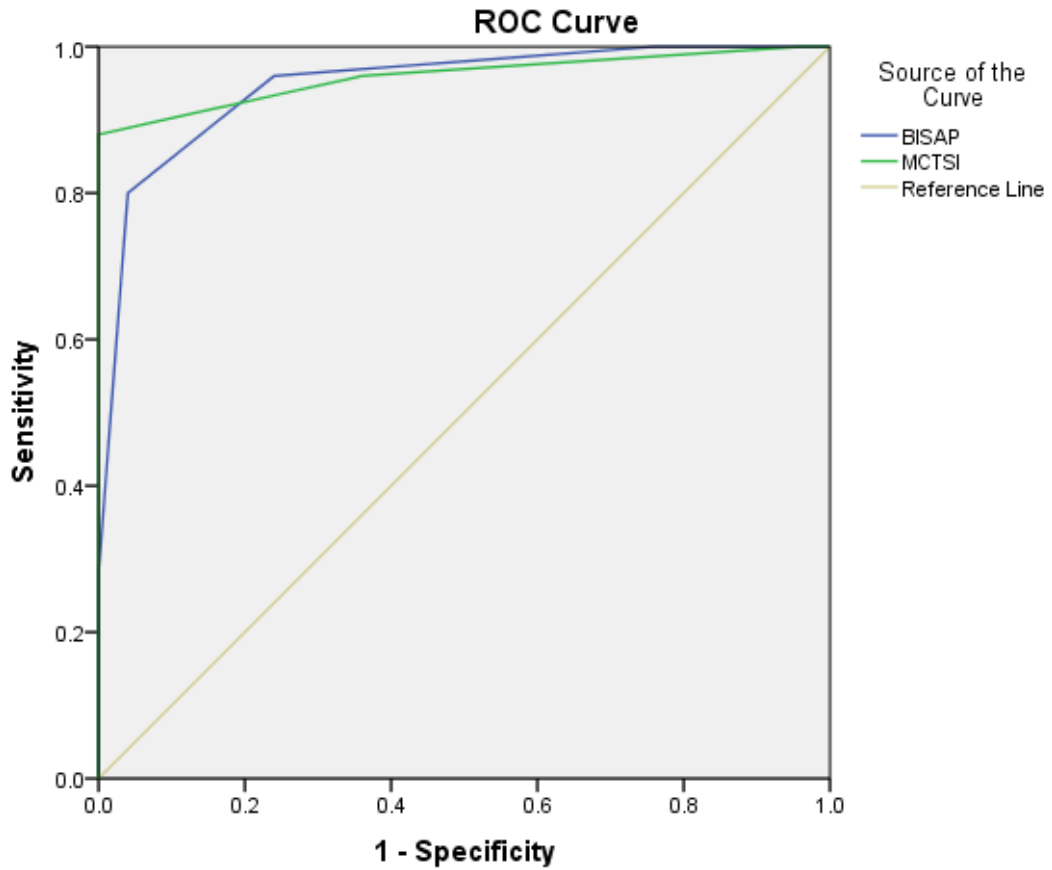
Diagonal segments are produced by ties.

Coordinates of the Curve

Test Result Variable(s)	Positive if Greater Than or Equal To[a]	Sensitivity	1 – Specificity
BISAP	.00	1.000	1.000
	1.50	1.000	.760
	2.50	.960	.240
	3.50	.800	.040
	4.50	.280	.000
	6.00	.000	.000

1

MCTSI	-1.00	1.000	1.000
	1.00	1.000	.960
	3.00	.960	.360
	5.00	.880	.000
	7.00	.640	.000
	9.00	.000	.000

The test result variable(s): BISAP, MCTSI has at least one tie between the positive actual state group and the negative actual state group.

a. The smallest cutoff value is the minimum observed test value minus 1, and the largest cutoff value is the maximum observed test value plus 1. All the other cutoff values are the averages of two consecutive ordered observed test values.

MCTSI GROUP * OUTCOME Crosstabulation

			OUTCOME		Total
			ALIVE	DEAD	
MCTSI GROUP	1.00	Count	33	1	34
		% within MCTSI GROUP	97.1%	2.9%	100.0%
		% within OUTCOME	75.0%	16.7%	68.0%
		% of Total	66.0%	2.0%	68.0%
	2.00	Count	11	5	16
		% within MCTSI GROUP	68.8%	31.2%	100.0%
		% within OUTCOME	25.0%	83.3%	32.0%
		% of Total	22.0%	10.0%	32.0%
Total		Count	44	6	50
		% within MCTSI GROUP	88.0%	12.0%	100.0%
		% within OUTCOME	100.0%	100.0%	100.0%
		% of Total	88.0%	12.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	8.257[a]	1	.004		
Continuity Correction[b]	5.794	1	.016		
Likelihood Ratio	7.795	1	.005		

				.010	.010
Fisher's Exact Test					
N of Valid Cases	50				

a. 2 cells (50.0%) have expected count less than 5. The minimum expected count is 1.92.

b. Computed only for a 2x2 table

COMPLICATIONS

V18

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid		25	50.0	50.0	50.0
	abscess	1	2.0	2.0	52.0
	ABSCESS	1	2.0	2.0	54.0
	ARDS	1	2.0	2.0	56.0
	ARDS/DIC	1	2.0	2.0	58.0
	ARF	6	12.0	12.0	70.0
	ARF/DIC	1	2.0	2.0	72.0
	ARF/ME	1	2.0	2.0	74.0
	ARF/UGIB	1	2.0	2.0	76.0
	fistula	1	2.0	2.0	78.0
	ME	1	2.0	2.0	80.0
	ME/ARF	1	2.0	2.0	82.0
	MODS	1	2.0	2.0	84.0
	pseudocyst	3	6.0	6.0	90.0
	RF	1	2.0	2.0	92.0
	SEPSIS	3	6.0	6.0	98.0
	UGB	1	2.0	2.0	100.0
	Total	50	100.0	100.0	

COMPLICATIONS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	ABSENT	25	50.0	50.0	50.0
	PRESENT	25	50.0	50.0	100.0
	Total	50	100.0	100.0	

1

SEX DISTRIBUTION

SEX

		Frequency	Percent	Valid Percent	Cumulative Percent
	f	5	10.0	10.0	10.0
Valid	m	45	90.0	90.0	100.0
	Total	50	100.0	100.0	

LOH STAY

LOH NEW

		Frequency	Percent	Valid Percent	Cumulative Percent
	1.00	31	62.0	62.0	62.0
	2.00	10	20.0	20.0	82.0
Valid	3.00	3	6.0	6.0	88.0
	4.00	6	12.0	12.0	100.0
	Total	50	100.0	100.0	

SYMPTOMS

FEVER

		Frequency	Percent	Valid Percent	Cumulative Percent
	_	15	30.0	30.0	30.0
Valid	-	5	10.0	10.0	40.0
	+	30	60.0	60.0	100.0
	Total	50	100.0	100.0	

VOMITING

		Frequency	Percent	Valid Percent	Cumulative Percent
	-	13	26.0	26.0	26.0
Valid	+	37	74.0	74.0	100.0
	Total	50	100.0	100.0	

1

JAUNDICE

		Frequency	Percent	Valid Percent	Cumulative Percent
	-	41	82.0	82.0	82.0
Valid	+	9	18.0	18.0	100.0
	Total	50	100.0	100.0	

ABDPAIN

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	+	50	100.0	100.0	100.0

ETIOLOGY

		Frequency	Percent	Valid Percent	Cumulative Percent
	Alcohol	34	68.0	68.0	68.0
	drug induced	1	2.0	2.0	70.0
	GSD	10	20.0	20.0	90.0
Valid	Idiopathic	3	6.0	6.0	96.0
	Idipathic	1	2.0	2.0	98.0
	Triglyceride	1	2.0	2.0	100.0
	Total	50	100.0	100.0	

BISAP & MCTSI CORRELATION WITH ORGAN FAILURE

% OF PATIENT'S

ORGAN FAILURE, 22

BISAP<3   BISAP>/=3   MCTSI<8   MCTSI>/=8
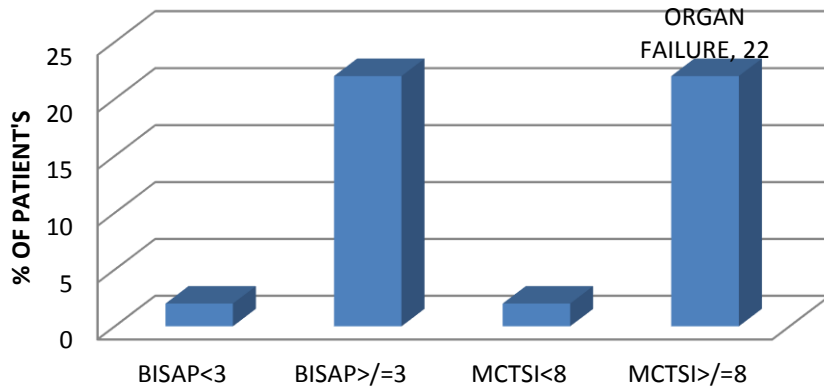
BISAP & MCTSI CORRELATION WITH OTHER COMPLICATION'S

% OF PATIENT'Se

■ Other complication's

BISAP<3   BISAP>/=3   MCTSI<8   MCTSI>8/=

SCORES

BISAP & MCTSI CORRELATION WITH MORTALITY
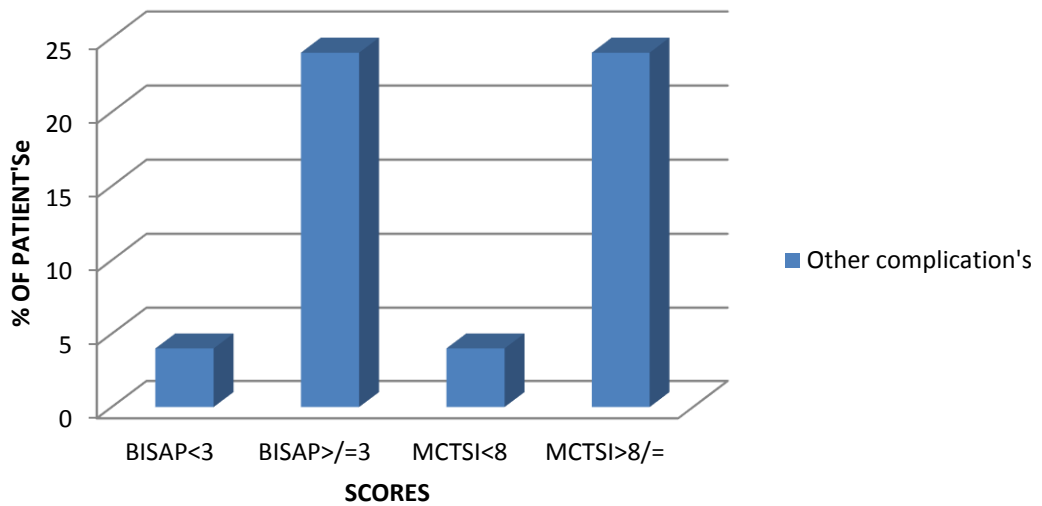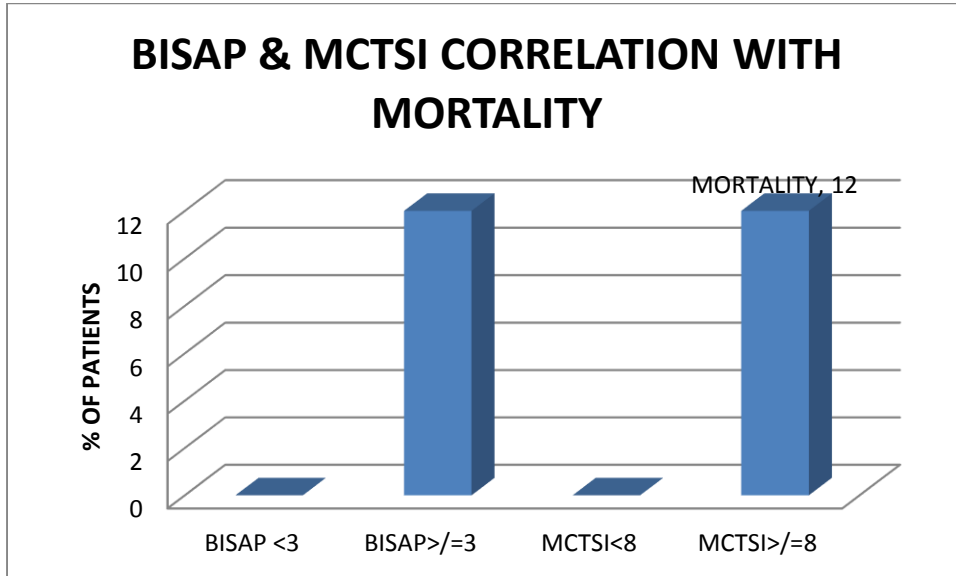
DISCUSSION

Acute pancreatitis is a common disorder with wide spectrum of illnesses. Severe pancreatitis having high morbidity and mortality rate , multiple interventions have been tried to prevent this. Early hospitalization may be beneficial to identify thosw who reqire aggressive interventions to prevent the severe attack of AP .

In this study , the two different scoring systems (BISAP and MCTSI) were compared and analzed to assess the severity in patients with acute pancreatitis . An attempt also made to compare this study with previous similar studies done by others.

Acute pancreatitis found 10 times more common in males than females in this study.This could be explained by the fact that , in this

study alcohol has found to be most common etiological factor and it's more common in males.

Patient's less than 30 years were excluded from this study because the normal values of heart rate and respiratory rate are higher at younger age group. So , if these patients had been included in this study , they could have got higher scores incorrectly and could have predicted incorrectly as at risk for developing severe pancreatitis , even with mild disease.

In this study the mean age was 52.6 years.

The mean age of non- survivors in this study was found to be 61 years as compared to survivors 43.33 years.Taking 70 years as cut off value , increasing age was found to be correlated well with increasing incidence of mortality . Thus age is considered as a significant contributory factor in predicting the outcome of severe acute pancreatitis.

The most common etiological factor in this study was alcohol and gall stones as second most common cause 68% and 20 % respectively. The mean length of hospital was 8.32 +/- 7.742.

BISAP & MCTSI is correlated well for mortality with high positive value of 0.904 which is highly significant (0.01).

1

The ROC analysis for Mortality showed BISAP score has AUC of 0.904 , P value (0.001) which is more than MCTSI score which has AUC of 0.845, P value (0.007). So BISAP is HIGHLY ACCURATE with P value (0.001) & confidence interval of 0.873.

BISAP score is highly sensitive (100 %) ,specificity (60%) at score more than 3.5 & MCTSI score sensitivity (85 %) ,specificity (77 %) at score more than 7.

Crosstabulation test for OUTCOME shows Chi –Square value of 4.545 with degree of freedom of 1 & P value 0.33 for BISAP scoring which is HIGHLY SIGNIFICANT when compared to MCTSI which has Chi – Square Value of 8.352 with degree of freedom 1 & P value 0.04.

The ROC curve for complications showed BISAP score AUC (0.903) with P value (0.001) and MCTSI score AUC (0.850) with P value (0.008), So BISAP is HIGHLY ACCURATE in detecting complications when compared to the MCTSI score.

BISAP score of more than 2 has high sensitivity 96 % & specificity 76% And MCTSI score of more than 3 has sensitivity 96% & specificity 64 % in detecting complications.

1

BISAP score was found to have more sensitivity , specificity and Diagnostic accuracy than MCTSI score in prediction of assessing the severity of acute pancreatitis.Hence BISAP score found to predict more number of patients and likelihood of progressing to severe disease .Larven et al stated the same in their study 42. Hence BISAP is considered as better available score for assessing the severity than MCTSI score

Limitations of this study are :

1. Small number of patients in this study.

2. The etiology in this study were found to be different from world wide accepted one , hence might not be correct to compare with other studies

3. The GCS score used to assess the mental status of the patient got admitted were subject to inter observer variation.

4. Various factors associated with the disease like cholangitis , alcohol withdrawal may interfere with the assessment of physiological scores ,which may leads to difference in the results.

1

5. Recently , it has been suggested that severe acute pancreatitis may have variable disease progression : therefore the lack of predictability might be associated with this disease variability.

6. Variation in timing of patients to the hospital after onset of symptoms may interfere with assessment of the scoring systems..

CONCLUSION AND SUMMARY

CONCLUSION AND SUMMARY

➢ From this study , Alcohol (68 %) was found to be the most common etiological factor for acute pancreatitis .

➢ Males were most commonly affected than female with a ratio of 10 :1

➢ The most common age group of patients affected were in 6 th decade

➢ The over all mortality in patients with severe acute pancreatitis was 12%

➢ The BISAP score predicted the Mortality significantly over the MCTSI score in patients with severe acute pancreatitis.

➢ The BISAP score predicted the disease severity significantly over the MCTSI score in patients with acute pancreatitis .

From this study , we conclude that BISAP score could be simple and accurate clinical scoring system for the evaluation of disease severity in acute pancreatitis, so CT needed not be taken in first 24 hours of admission.

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PROFORMA

A COMPARATIVE EVALUATION OF RADIOLOGIC AND CLINICAL SCORING SYSTEM IN THE EARLY PREDICTION OF SEVERITY IN ACUTE PANCREATITIS

Patient Details :

Name :   IP:NO:

Age :   Sex :

Hospital No: Date of Admission:

Date of surgery(if any)   Address :

Date of discharge :

History :

    Abdominal pain :

- Duration
- Onset
- Progression
- Nature of pain
- Radiation
- Aggravation/relieving factors

Vomiting:

- Duration
- Episodes
- Nature of vomitus
- Hematemesis

1

Fever :

- Duration

- Grade

- Associated with chills/rigor

Trauma

Prolonged drug intake

Jaundice

Malena

Loss of appetite/loss of weight

Breathlessness

Past h/o:

Previous Surgical illness

DM/HT/TB/BA/COPD/IHD/EPILEPSY/BA

Personal h/o:

Occupation: socioeconomic status :

Smoking: Alcoholism:

Drug addiction: Tobacco/Betel nut chewing:

Family h/o   :

General Examination:

GCS :E V M

Vitals : PR:

          BP:

          RR:

          Temperature:

BMI:

Systemic Examination:

Abdominal Examination:

Cardiovascular Examination:

Respiratory Examination:

Diagnosis :

Investigations:

Complete Hemogram:

Hb: Tc: Dc :

Esr :   Pcv: Platelet :

Blood sugar :

Blood urea: Sr. Creatinine :

Sr. electrolytes :   Na+   k+   cl-   HCO3-

Liver function test :

ABG analysis :

 pH   PaCO2   PaO2

Sr. amylase /Sr. lipase

Chest X- ray

Abdomen X- ray

ECG

USG Abdomen & chest :

CT scan abdomen :

BISAP SCORING SYSTEM

1.Blood urea nitrogen (BUN)>25mg/dl

2.Impaired mental status (GCS<15)

3. SIRS (defined as two or more of the following)

1.Pulse rate >90/min

2.Respiratory rate >20/min or PaCO2 <32mmHg.

3.Temperature>100.4F or <96.8F/<36 or >38

4. WBC count >12,000 0r < 4,000cells/mm or presence of more than 10 % immature blasts.

4.Age>60 yrs

5.Pleural effusion

TOTAL SCORE :

BISAP ,Bedside Index for Severity in Acute Pancreatitis ;

SIRS . Systemic Immune Response Syndrome

One point is assigned for each variable within 24 hours of presentation and added for a composite score (0-5)

MCTSI SCORING SYSTEM

Prognostic Indicator	Points
Pancreatic inflammation	
Normal pancreas	0
Intrinsic pancreatic abnormalities with or without inflammatory changes in peripancreatic fat	2
Pancreatic or peripancreatic fluid collection or peripancreatic fat necrosis	4
Pancreatic necrosis	
None	0
≤ 30%	2
> 30%	4
Extrapancreatic complications (one or more of pleural effusion, ascites, vascular complications, parenchymal complications, or gastrointestinal tract involvement)	2

Total score :

Risk Stratification Score

BISAP :

    Score :   Severity status :

 MCTSI :

    Score: Severity status

Course of Hospital stay.

INSTITUTIONAL ETHICAL COMMITTEE, STANLEY MEDICAL COLLEGE, CHENNAI-1

Title of the Work : A comparative evaluation of radiologic and clinical scoring system in the early prediction of severity in acute pancreatitis .

Principal Investigator : Dr. K Ashok Kumar

Designation : PG MS (General Surgery)

Department : Department of General Surgery
Government Stanley Medical College,
Chennai-01

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 13.01.2015 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

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

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

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

MEMBER SECRETARY,
IEC, SMC, CHENNAI

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MASTER CHART

NAME	IP NO	AGE	SEX	BUN	GCS	SIRS	AGE	PE	BISAP	PI	PN	EPC	MCTSI	OUTCOME	MANAGEMENT	COMPL	ETIOLOGY	LOH	Abdominal	jaundice	vomiting	lipase/am	fever
sankar	1541053	45	m	0	1	1	0	0	2	2	0	0	2	Alive	Conservative		Alcohol	3	+	-	-	+	+
jegan	1541067	62	m	1	0	1	1	1	4	4	2	2	8	Death	Conservative	MODS	Alcohol	8	+	-	-	+	+
murugesan	1541090	59	m	0	0	1	0	0	1	2	0	0	2	Alive	Conservative		Alcohol	3	+	-	+	+	-
rajendran	320561	50	m	0	0	1	0	0	1	2	0	0	2	Alive	Conservative		Triglyceric	9	+	-	-	+	+
lenin	350675	48	m	0	0	1	0	0	1	2	0	0	2	Alive	Conservative		Alcohol	2	+	-	+	+	-
muzaffer	350678	56	m	0	0	1	0	0	1	2	0	0	2	Alive	Conservative		Alcohol	3	+	-	+	+	-
kannan	1542001	38	m	1	0	1	0	0	2	2	0	0	2	Alive	Conservative	ARF	Alcohol	4	+	-	+	+	-
muniyapp	1543901	62	m	1	1	1	1	1	5	4	2	2	8	Alive	Conservative	ARF	Alcohol	15	+	-	+	+	+
chinnan	362356	68	m	1	0	1	1	1	4	4	2	2	8	Alive	Conservative	UGB	Alcohol	9	+	-	-	+	+
ramesh	353765	38	m	0	0	1	0	0	1	0	0	0	0	Alive	Conservative		Alcohol	3	+	-	-	+	-
retheesh	1545321	35	m	0	1	1	0	0	2	2	0	0	2	Alive	Conservative		Alcohol	4	+	-	+	+	-
sivamani	324568	48	m	0	1	1	0	0	2	2	0	0	2	Alive	Conservative		GSD	2	+	+	-	+	-
manikand	1526651	61	m	1	1	1	1	0	4	4	0	0	4	Alive	Conservative		Alcohol	5	+	-	+	+	+
soorya	1523573	63	m	1	0	1	1	1	4	4	2	2	8	Alive	Conservative	ARF	Alcohol	23	+	-	+	+	+
vishal	362672	42	m	0	1	1	0	0	2	2	0	0	2	Alive	Conservative		GSD	5	+	+	+	+	-
jeyakandh	1587732	58	m	1	0	1	0	1	3	2	0	2	4	Alive	Conservative	SEPSIS	drug induc	6	+	-	+	+	+
parvathya	1576436	60	m	1	0	1	1	1	4	4	0	2	6	Alive	surgery	pseudocys	Alcohol	8	+	-	+	+	+
selvaraj	1576473	40	m	1	0	1	0	1	3	2	0	2	4	Alive	Conservative	ME	Alcohol	4	+	-	-	+	+
sivaji	376687	65	m	1	1	1	1	1	5	4	2	2	8	Death	Conservative	ARF	Alcohol	2	+	-	-	+	+
manikand	1576267	61	m	1	0	1	1	0	3	4	0	0	4	Alive	Conservative		Idiopathic	4	+	-	+	+	+
varun kun	1524356	37	m	1	1	1	0	0	3	4	0	2	6	Alive	Conservative	SEPSIS	Alcohol	8	+	-	+	+	-
karthick	1528238	45	m	1	1	1	0	1	4	4	0	2	6	Alive	Conservative	pseudocys	Alcohol	10	+	-	+	+	+
sudharsan	379797	43	m	0	1	1	0	0	2	2	0	0	2	Alive	Conservative		Alcohol	2	+	-	+	+	+
bharath	384848	50	m	0	0	1	0	1	2	2	0	2	4	Alive	Conservative		GSD	9	+	+	+	+	

1

#	Name	ID	Age	Sex										Total	Status	Treatment	Complication	Etiology						
26	sarath kur	1534341	61	m	1	1	1	1	1	5	4	2	2	8	Death	Conservative	RF	Alcohol	2	+	-	+	+	_
27	shiraj	1583221	65	m	1	1	1	1	1	5	4	2	2	8	Death	Conservative	ARDS	GSD	3	+	+	+	+	+
28	preetham	376787	48	f	1	0	1	0	0	2	2	0	0	2	Alive	Conservative		Idiopathi	5	+	-	+	+	_
29	neeraj	1578882	37	m	0	0	1	0	0	1	2	0	0	2	Alive	Conservative		Alcohol	6	+	-	+	+	+
30	danajeyar	389979	62	m	1	1	1	1	1	5	4	2	2	8	Alive	Conservative	ARF	Alcohol	15	+	-	-	+	+
31	raj kumar	1590982	56	m	1	1	1	0	1	4	4	2	2	8	Alive	Conservative	ARF	Alcohol	12	+	-	+	+	+
32	kumarave	1598791	61	m	1	0	1	1	1	4	4	0	2	6	Alive	Conservative	fistula	Alcohol	2	+	-	+	+	+
33	rathinavel	308772	38	m	0	0	1	0	1	2	2	0	2	4	Alive	Conservative		Alcohol	7	+	-	+	+	+
34	asha	150988	45	f	1	0	1	0	1	3	2	0	2	4	Alive	Conservative		GSD	24	+	+	+	+	_
35	prabavath	1543421	61	f	1	1	1	1	1	5	4	2	2	6	Death	Conservative	ARDS/DIC	GSD	9	+	+	+	+	+
36	malaichan	356571	68	m	1	1	1	1	1	5	4	2	2	8	Alive	Conservative	ARF/UGIB	Alcohol	11	+	-	+	+	+
37	pandithur	1576621	48	m	1	0	1	0	1	3	2	0	2	4	Alive	Conservative		Alcohol	6	+	-	-	+	_
38	mayandi	1546542	63	m	1	0	1	1	1	4	4	2	2	8	Alive	Conservative	ARF/DIC	Alcohol	18	+	-	+	+	+
39	rajendra p	1568721	50	m	0	0	1	0	1	2	2	0	2	4	Alive	Conservative		GSD	5	+	-	+	+	-
40	karuthapa	388726	38	m	1	0	1	0	0	2	2	0	0	2	Alive	Conservative		Alcohol	1	+	-	+	+	-
41	sivakumar	1566621	64	m	1	0	1	1	1	4	4	2	2	8	Alive	Conservative	ME/ARF	GSD	29	+	+	+	+	+
42	karthikeyan	1545332	61	m	1	0	1	1	0	3	4	0	0	6	Alive	Conservative	pseudocy	Alcohol	4	+	-	+	+	+
43	prabhu	355241	40	m	1	0	1	1	0	3	4	0	0	4	Alive	Conservative		Alcohol	7	+	-	+	+	+
44	moses	1544421	65	m	1	0	1	1	1	4	4	2	2	8	Alive	Conservative	SEPSIS	Alcohol	23	+	-	+	+	+
45	rajamani	1532271	48	m	1	1	1	0	1	4	4	2	2	8	Alive	Conservative	ABSCESS	GSD	30	+	-	-	+	+
46	priya	309809	40	f	1	1	1	0	1	4	4	2	2	8	Death	Conservative	ARF/ME	Idipathic	1	+	-	+	+	_
47	arokya ma	1587687	38	f	1	0	1	0	0	2	2	0	0	2	Alive	Conservative		GSD	5	+	-	-	+	+
48	velumani	384848	64	m	1	0	1	0	0	2	2	0	0	2	Alive	Conservative		Alcohol	2	+	-	+	+	_
49	naduthura	1577651	48	m	1	0	1	0	0	2	2	0	0	2	Alive	Conservative		Alcohol	4	+	-	-	+	_
50	pasupathy	354455	56	m	1	0	1	0	1	3	2	0	2	4	Alive	Conservative		Idiopathic	5	+	+	+	+	-

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A Comparative Evaluation of Radiologic and Clinical scoring systems in the Early

BY 221311082.MS GENERAL SURGERY K .ASHOK KUMAR

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ABSTRACT

AIM OF THE STUDY:

The aim of this study is to compare the accuracy of clinical scoring and Radiologic scoring for predicting the severity of acute pancreatitis at the time of admission.

MATERIALS AND METHODS:

Demographic, clinical and laboratory data of all consecutive patients with a primary diagnosis of acute pancreatitis during the study period are prospectively collected for this study. Analysis of abdominal ct″'s are

PAGE: 1 OF 115

Text-Only Report

1

அரசு ஸ்டான்லி மருத்துவக் கல்லூரி, சென்னை - 600 001.

பங்கு பெறுபவரின் ஒப்பம்

ஆராய்ச்சியின் தலைப்பு	:	கடுமையான கணைய அழற்சி தீவிரத்தை மருத்துவ மற்றும் கதிரியக்க மதிப்பெண் மூலம் ஆரம்ப நிலையில் ஒப்பிட்டு மதிப்பிடுதல்
ஆராய்ச்சி நடை பெறும் இடம்	:	அரசு ஸ்டான்லி மருத்துவக் கல்லூரி, சென்னை - 1.

பங்கு பெறுபவரின்
பெயரும் முகவரியும்   :

நான்,...இந்த ஆராய்ச்சியின் விவரங்களை எனது சொந்த மொழியில் கூற அறிந்து கொண்டேன்.

இந்த ஆராய்ச்சியின் முழு விவரங்களையும் நான் அறிந்து கொண்டேன். இந்த ஆராய்ச்சியில் நான் பங்கு பெறும் போது எனக்கு ஏற்படும் நன்மை தீமைகளை முழுவதுமாக அறிந்து கொண்டேன்.

இந்த ஆராய்ச்சியின் போது எப்போது வேண்டுமானாலும் நான் விலகிக் கொள்ளலாம் என்பதையும், அதனால் எனக்கு கிடைக்கும் மருத்துவத்தில் எந்தவித மாற்றமோ பாதிப்போ இருக்காது என்றும் அறிவேன். இந்த ஆராய்ச்சியில் நான் பங்கு பெறுவதற்காக நான் எந்தவித சன்மானமும் (பணமாகவோ, பொருளாகவோ) வாங்கமாட்டேன். இந்த ஆராய்ச்சியின் முடிவுகளை, என் அடையாளங்களை குறிப்பிடாமல் மருத்துவ இதழ்களில் வெளியிட எனக்கு எந்த ஆட்சேபனையும் இல்லை. இந்த ஆராய்ச்சியில் என் பங்கு என்ன என்பதை அறிவேன். இந்த ஆராய்ச்சிக்கு எனது முழு ஒத்துழைப்பையும் தருவேன் என்று உறுதி அளிக்கிறேன்.

பங்கு பெறுபவரின் பெயரும் முகவரியும்:

பங்கு பெறுபவரின் கையொப்பம் / விரல்ரேகை :

தேதி:

சாட்சி :

(சாட்சியின் பெயர், முகவரி, கையொப்பத்துடன்)

ஆராய்ச்சி செய்பவரின் பெயரும் கையொப்பமும் :

அசோக்குமார். K

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