DISSERTATION ON

A CLINICAL STUDY OF ACUTE PANCREATITIS

Dissertation submitted to

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

In partial fulfilment of the requirements for the degree of

M.S. DEGREE EXAMINATION

BRANCH I

(GENERAL SURGERY)

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI

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

CHENNAI-TAMILNADU

APRIL 2017

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This is to certify that this dissertation entitled "**A CLINICAL STUDY OF ACUTE PANCREATITIS"**is the bonafide original work of **Dr.B.UMA** postgraduate M.S student in department of general surgery,Tirunelveli medical college&hospital Tirunelveli, in partial fulfilment of the requirements for M.S Branch -I (General Surgery) Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in APRIL - 2017. The period of study was from FEBRUARY 2015-AUGUST 2016

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Dear , Dr. B.Uma, MBBS., The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during the IEC meeting held on 09.02.15.

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1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of the Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
14.	Clinical Trials Registry-India (CTRI) Registration	☐

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1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
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7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
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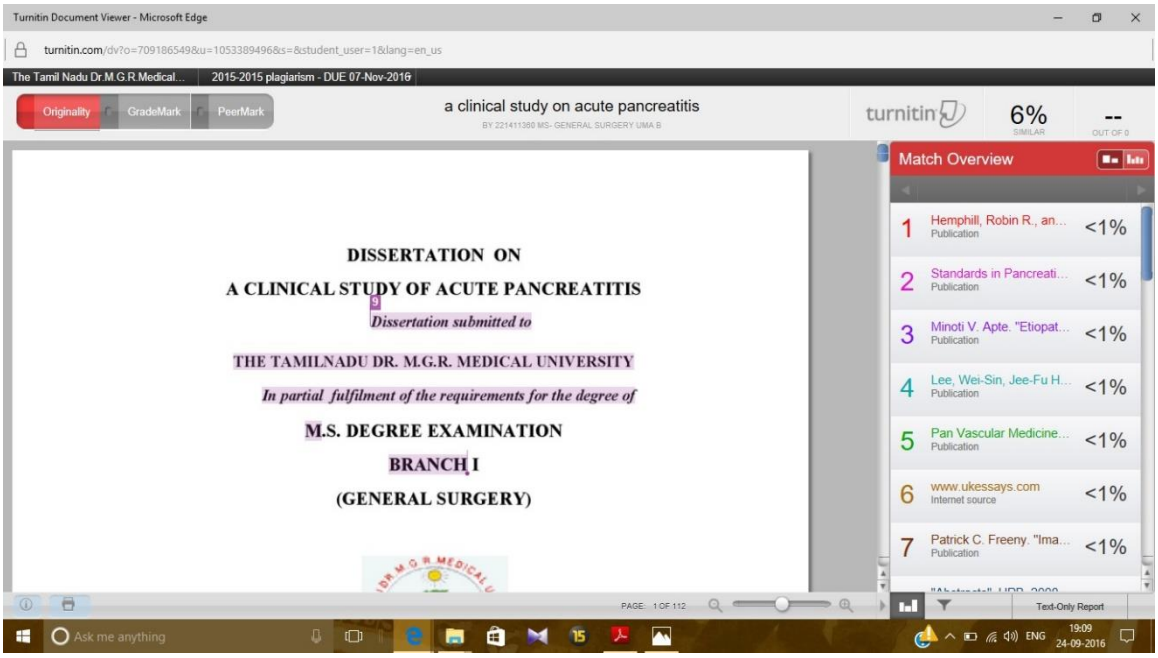
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INTRODUCTION

INTRODUCTION

Acute pancreatitis is one of the important causes of acute abdomen.Patients of acute pancreatitis usually get admitted in the surgical emergency units. Generally chronic alcoholism is the important cause of acute pancreatitis in men &biliary tract pathology is the common etiology in women. Various epidemiological studies are concluded that most cases of acute pancreatitis are attributed to chronic alcoholism. The incidence and the prevalence of it are varying in different countries. Lankishet al. reported from twenty studies of acute pancreatitis from various countries of Europe that the biliary tract disease contributes to the majority of cases of acute pancreatitis.

The significant variability in data indicates that the accurate measurement of the magnitude of problem is very difficult for the researchers. This is because of the following factors. 1)Lack of standardization in diagnosing acute pancreatitis with the laboratory and clinical background 2)variation in the inclusion and exclusion criteria's in different studies 3)regarding tools used for the consumption of alcohol 4)confusion between acute and chronic forms.

The interesting fact is that the incidence of alcohol induced pancreatitis is increasing over the past decade. A study in United Kingdom shows that alcoholic pancreatitis incidence has increased from 14.5 per lakh population during 1989-1990 to 20.7 per lakh population in the year 1999-2002. Clinically acute pancreatitis occurs only in five percent of heavy alcoholics.

About 3-8 % patients with symptomatic biliary calculus can develop acute pancreatitis. The relative risk of developing acute pancreatitis in patients with the biliary stones is 35 times higher than the normal population.Biliary stone pancreatitis commonly occurs in women& tends to occur in older age group.

The development of pancreatitis is related to the stone size and its number. Patient can develop acute pancreatitis even with small but multiple gall stones. It indicates migration of gall stones into the bile duct. High level of mucin in bile in patients with pancreatitis revealed that mucin enhances stone formation. In a study of 528 patients with biliary stones, acute pancreatitis more prevalent in patients with smaller stones than obstructive stones. Some study shows about 80 % of idiopathic pancreatitis were due to biliary stones.

Most of the acute pancreatitis patients had only milder forms, which can be managed conservatively and they recovered completely. The severe forms occurred only in 15% of patients. In recent studies the mortality rate is greatly

decreased (from 35-85%-10-20%). Severe acute pancreatitis has two stages of the disease. Initial inflammation and necrotic changes of the parenchyma is followed by signs of systemic inflammatory response syndrome leads to multi organ failure within 7-10 days. About half of patients with severe forms of pancreatitis died in the first week due to complications like ARDS/MODS. The mortality rate in patients with MODS stage varies from 30 % to maximum 100 %. These patients need early resuscitation, intensive care to prevent multi organ/respiratory failure. Some patients need surgical interventions like retroperitoneal drainage and necrosectomy in pancreatic necrosis and infected necrosis. To conclude the purpose of my study is to diagnose and assess the severity in patients with acute pancreatitis with clinical manifestations, labarotary investigations and imaging studies in our institution.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

BACKGROUND

Eristratos(310-250 BC) mentioned the word pancreas in his writing. Later Rufus of Ephesus gave the name as pancreas at 100 A.D. In Greek language, pan meansall (kreasmeans flesh) as the organ has no cartilage or bone.

EMBRYOLOGY

Pancreas developed from two endodermal buds which originate from duodenum at 4 th week of intra uterine life,one is dorsal pancreatic bud, another one is ventral pancreatic bud. Most part of the head, body and tail of pancreas developed from the dorsal bud.Uncinate process and inferior part of the head of pancreas formed from the ventral bud. Both the pancreatic buds fuse at 37 th day of intra uterine life and form pancreas.

ANATOMY

Pancreas is a retroperitoneal organ which occupies the epigastric region. Pancreas is situated behind the stomach and the omental bursa. It extends from the medial edge of the second part of duodenum to the splenic hilum. Portal vein is formed behind the neck of pancreas. Abdominal aorta, inferior vena cava are situated behind the pancreas.

Pancreas has head, uncinate process, neck, body and tail. Uncinate process is situated between the superior mesenteric vessels anteriorly and the aorta posteriorly. Root of the transverse mesocolonis attached to the anterior surface of thebody of pancreas.

PANCREATIC DUCT

Ductal system of the pancreas usually has high variability. Wirsung described the main pancreatic duct in the year 1642. The fusion of the pancreatic duct with the bile duct and opening into the duodenal papilla are described by Vater at 1720. Santorini first described the accessory pancreatic duct. Main pancreatic duct is 0.3 cm in diameter. It courses midway between the upper and lower border of the pancreas. It joins with the bile duct in the head of pancreas and empties at the ampulla of Vater, which is situated on medial part of the second part of the duodenum. Sphincter of Oddi is formed by the muscle fibers around the ampulla, which is used for controlling the flow of bile and pancreatic juice into the duodenum.

Accessory pancreatic duct starts at the lower portion of the pancreatic head

cm proximal and just anterior to the major papilla.

SPHINCTER OF ODDI

The terminal end of the common bile duct and pancreatic duct is encircled bysphincteric smooth muscle fibers called the Sphincter of Oddi. The sphincteric fibers are having a separate choledochal part. Only thirty percent of persons are having the pancreatic sphincter. At the end of pancreatic duct there is a zone of high pressure. The secretin hormone relaxes this high pressure zone and enhances the pancreatic secretions to flow into ampulla. In 75 % individuals, the biliary and pancreatic ducts unite and form the common duct before they enter into the duodenum. The sphincters are having the regulatory action in the rate of flow into duodenum and prevent back flow of duodenal contents into duct. The duct passes obliquely into the duodenal wall and the valves of mucosa also preventthe reflux.

Sphincter of Oddiis controlled by the neural pathways. During feeding it reduces the sphincter pressure. Cholecystokinin causes relaxation of the sphincter after food intake. Some kind of biliary and pancreatic pathology may be caused by the sphincteric stenosis and dyskinesia.

VASCULAR SUPPLY

Arterial supply is from superior pancreatico duodenal artery, a branch of gastro duodenal artery and inferior pancreatico duodenal artery, a branch of superior mesenteric artery.

Branches from the splenic artery supply body and tail of pancreas.Veins from the pancreas drain into splenic and superior mesenteric, portal veins.

LYMPHATIC DRAINAGE

Different parts of the pancreas drained by different lymphatic groups. Pancreatic head and uncinate process mainly drained by the infra pyloric group and also drained by the periportal,mesenteric, mesocolic, pre and para aortic lymph nodes.

Body of pancreas mainly drains into coeliac group of lymph nodes and also drained by mesenteric and aortic group of lymph nodes.

Tail portion of pancreas drains mainly into celiac groups and also into splenic hilar nodes

NERVE SUPPLY

Pancreas is supplied by two types of autonomic nerve system, one is sympathetic nervous system and another one is parasympathetic nerve system.

The ascending tract for pain from any pancreatic pathology is through the nociceptive nerve fibers which are arising from the pancreas itself. These tracts of fibers ascend and pass through the coeliac ganglionic part and they form three splanchnic nerves which are

1) Greater splanchnic nerve

2) Lesser splanchnic nerve

3) Least splanchnic nerve

These nerve fibers finally end into nerve cell bodies of sympathetic chain in thoracic level. Descending pathway is also supplied to pancreas by sympathetic and parasympathetic components of autonomic nerve system as like ascending pathway. The parasympathetic part is originated from the nucleus of vagusnerve. From the nerve cell bodies of the vagus, nerve fibers to pancreas pass via right vagusnerve and pass to celiac plexus of nerves. From there post ganglionic fibers form which innervate islets of Langerhans and vessels and exocrine parts. Generally the pathways of nerves followthe arterial supply to the pancreas.

Histology

The developed pancreas is made up of two components. One is endocrine portion and another part is exocrine pancreas.

1) Endocrine part: endocrine part constitutes only 2% of the whole pancreatic tissue. Endocrine portion of the pancreas is having islets of Langerhans cells. About 10 lakhs Langerhans islets present in matured normal pancreas. There are five major types of cells present in the endocrine portion.

1) Alpha cells

2) Beta cells

3) Delta cells

4) Epsilon cells

5) Pancreatic polypeptide cells

Alpha cells secrete glucagon hormone, which is involved in glucose metabolism. Beta cells secrete insulin hormone. Insulin is released into the blood stream in response to high levelsof glucose in blood. It reduces blood glucose level by converting into glycogen and storing in liver and muscles.

Delta cells secrete somatostatin hormone. It is involved in reducing gastro intestinal secretions. It is also involved in inhibition of secretion and functions of other gastro intestinal hormones.

Ghrelin hormone is secreted by epsilon cell groups. Its function is to reduce the level of insulin release and block actions of insulin. Pancreatic polypeptide is secreted from PP cells. It blocks exocrine secretions of pancreas.

EXOCRINE PART

Exocrine part of pancreas constitutes about more than three fourth of pancreas. Extra cellular part constitutes 10 % of the mass and remaining part comprises blood vessels and ductal system. Exocrine pancreas comprises two types of cells which are acinar cells and ductal cells.

Acinar cell is named because it looks like a cluster of grapes. These acinarcells secrete into central space which drain into the main pancreatic ductal system through the communications. In histopathological examination these cells are having the large amount of endoplasmic reticulum and eosinophilic zymogen granules. The main pancreatic ductal system is lined by columnar cells of tall variant which are having mucinous granules. Intra and inter lobar ductal system cell lining has cuboidal type of configuration which has no mucinous granules. Centroacinarlining epithelium is similar to acinar cells, but it contains no zymogen granules.

CONGENITAL ANOMALIES OF PANCREAS

1) **Agenesis of pancreas** :

In this condition, pancreas is totally absent. It is usually associated with other severe congenital abnormalities. This condition is seen in mutation in long arm of chromosome 13. Patient cannot survive with this condition.

2.Divisum of pancreas:

This is one of the common developmental pancreatic anomalies. This is due to failure of fusion of primordial dorsal and ventral pancreatic ductal system in intra uterine life. The main pancreatic duct is usually very short and drains few portions of pancreatic head. Dorsal part of the pancreas drains into lesser papilla through the Santorini ductal system. Ventral pancreatic ductal system drains via main ampulla of Vater opening. This anomaly ultimately predispose to chronic pancreatitis

3) Annular pancreas:

In this condition the second part of the duodenum is encircled by a band of normal pancreatic tissue which is partial or complete and extends into the pancreatic head. It is also having small duct which communicates with the main pancreatic duct. The etiology of annular pancreas is not clear. It may due to absence of ventral pancreatic bud in clockwise rotation during development or extension of pancreatic tissue in duodenal wall. It causes duodenal obstruction either partially or completely. This condition is usually associated with other system congenital malformations. Pancreatitis can be developed from the abnormal segment of pancreas. It is treated by diversion rather than segment resection by duodenojejunostomy.

4) Congenital cysts of pancreas:

These are usually solitary cysts which is a rare condition. But most commonly occurred cysts are multiple in numbers. Multiple cysts lined by cuboidal epithelial cells. This condition is usually associated with cysts of other intra-abdominal solid organs like liver and kidney. It is commonly seen in Von Hippel Landau syndrome. Congenital cysts are usually asymptomatic. No specific treatment is required unless they become symptomatic.

5) Ectopic pancreas:

This condition is seen in two percent of the post mortem cases. Most common sites are stomach and the proximal part of duodenum. The other sites include jejunal loops, meckel's diverticulum and distalileal segments. On histopathological examination it shows normal tissues of the exocrine part of pancreas like acinar part and ducts. Very rarely Langerhans islet cells also present. Usually these are asymptomatic conditions which are diagnosed as incidental findings. It can cause pain due to localized inflammatory process or bleeding manifestations. Very rarely tumours of Langerhans cells arise from this ectopic pancreas.

PHYSIOLOGY

Pancreatic secretion:

Pancreas has two types of secretions. One is exocrine part which secretes enzymes for digestion. Another part is endocrine part which synthesizes hormones for carbohydrate metabolism and gastro intestinal hormonal regulation.

Exocrine pancreas:

Adult pancreatic tissue secretes approximately two to two thousand five hundred milliliters of pancreatic juice. This secretion is characteristically clear, odorless, with pH is of more than eight. It has high amount of bicarbonate. It is rich in protein. Pancreatic juice is released from bothacinar and ductal cells of pancreas. It is the second most protein rich fluid after mammary glands. Pancreatic acinar cells secrete amylase,lipase, proteindigestion enzymes. Amylase is responsible for the digestion of carbohydrates. Lipase is involved in fat digestion. Protein digestion is mediated by proteases.

Amylase

Amylase is the only digestive enzyme synthesized in the active form in the intra pancreatic part. It completes digestion of saccharides, after salivary amylase is involved in initiation of carbohydrate metabolism. The salivary amylase breaks polysaccharides into disaccharides (starch into maltose) inside the oral cavity itself. The pancreatic amylase enzyme breaks remaining polysaccharides like glycogen and starch into disaccharides like maltose,

sucrose, glucose. The glucose absorbed into intestinal mucosal cells by transport mechanism.

Enzyme synthesis:

The digestive enzymes account for about eighty to eighty five percent of protein synthesis in pancreas. Greater parts of the enzymes are synthesized in an inactive form called proenzymes or zymogens which are secreted from acinar cells. Some enzymes are synthesized in an active form since they does not need any process of activation for its target action. These are amylase, lipase and ribonuclease. Acinar cells also synthesize some kind of proteins only for the purpose of using within the cell itself. These are structural proteins and hydrolytic enzymes.

Process of secretion:

The freshly synthesized enzymes are accumulated into endoplasmic reticular organelles. After that enzymes are transported into the Golgi apparatus. These are packed in vacuoles inside the cells and formed into granules which are called as zymogen granules. Finally these zymogen granules migrated into the acinar cell membranes. The zymogen granules released into the lumen of acinar cells by the mechanism of fusion and fission.

Synthesis of enzymes in acinar cells is in an orderly fashion. Low level or basal secretion occurs during the resting state. After the neuronal and hormonal stimulation due to external signals the rate of secretion is increased into a marked amount. Pancreatic acini is having some receptors for neurotransmitters from cholinergic nervous system, and receptors for other gastro intestinal hormones like kinins, secretin, vasoactive peptides. Stimulation for secretory process is initiated by neurotransmitters or kinin causes activation of intra cellular transmitters like phospholipase C, creation of phosphorylated inositol (ITP) and diacyl glycerol, then leads to increased ionized calcium inside the cells. The rate of release of enzymes from acinar cells will be by an unexplained regulatory process

Electrolyte secretion:

Pancreatic secretion is a highly alkaline solution because of its abundant content of bicarbonate ions. Acinar cells secrete minimal level of serous fluid. But major part of fluid and bicarbonate rich secretion by ductal epithelial cells. Initially blood carbon di oxide gas diffuses into ductal epithelium. Carbon di oxide is converted into carbonic acid by the enzyme carbonic anhydrase. Carbonic acid further breaks down into hydrogen ions and bicarbonate ions.

The hydrogen ions efflux out of cells and enter into the blood stream. But the ions of bicarbonate are sequestered inside the cells. The enzyme secretin stimulates secretion of fluid and electrolytes. It acts through mediator like cyclic AMP interns result in secretion of chloride ions in the surface of acinar cells. This is regulated by cystic fibrosis membrane conductance regulator, acts in channels for chloride. The secreted chloride ions again reabsorbed into ductal cells interns into exchange of bicarbonate ions via the mechanism of chloride-bicarbonate exchanging. Final event of the process is the fluid which is rich in bicarbonate secreted into the lumen of ductal system. Pancreatic juice contains only plasma like fluid if the secretin stimulation is absent because there is little or no activation of ductal epithelium for bicarbonate exchange.

Activation of pancreatic enzymes:

The digestive enzymes are released as pro enzymes. These need activation by some enzymes, to act on target sites. Trypsinogen is secreted in an inactive form and converted into an active form trypsin. This is activated by enzyme called entero kinase which is secreted from lumen of intestine. Intern trypsin activates other inactive digestive enzymes of pancreas.

Trypsinogen is in inactivated form inside the pancreas, because there is some inhibitors of trypsinogen synthesized inside the acinar cells. Fail to express this inhibitors of trypsinogen will lead onto familial type of pancreatitis. The trypsinogen activated in the lumen of duodenum. Premature or intra pancreatic activation causes pancreatitis in one condition which is mutation in cationic trypsinogen. This condition constitutes about sixty percent of familial pancreatitis.

Chymotrypsinogenactivated into the active form called chymotrypsin. Trypsin also activates the enzymes like elastase, phospholipase, carboxy peptidases type A and B.

Protein digestion:

These activated enzymes act on particular amino acids of peptide chains and cleave in between the amino acids. Carboxy peptidases break the amino acids at their peptide chain ends. The amino acids and di peptides are absorbed into dipeptides are absorbed into intestinal epithelial cells by activated transport system.

Fat digestion:

Triglycerides hydrolyzed into fatty acids and glycerol by the action of lipase. Trypsin also activates phospholipase A2 which is secreted as pro enzyme. This enzyme hydrolyzes the phospholipids. Lipases act on lipids only after action of bile salts on lipids by reducing its surface tension. Cholesterol esters, fat soluble vitamins like vitamin A,D,E,K and triglycerides are hydrolyzed by enzymes like carboxylic ester hydrolases and cholesterol esterase. The micelles formed transported into epithelium of intestinal mucosa. Chylomicrons transported through the lymph ducts finally enter into the blood circulation.

Phases of pancreatic secretion:

The amount of pancreatic secretion during resting phase of digestive tract is very minimal when compared with activated stage. These are three stages of pancreatic secretion.

1) **Cephalic stage**:

   This phase constitutes only twelve to sixteen percent of food induced secretion of pancreatic juice. This is the phase which is in response to smelling or chewing the food lead onto secretion of pancreatic juice. It is considered to be due to the peripheral

stimulation of cholinergic nerve fibers which induces secretion of hydro chloric acid in stomach and fluid and electrolytes rich pancreatic secretion. This intern led onto acidic duodenal contents which cause secretin release.

2) **Gastric phase** :

This stage also constitutes only minimal portion of food stimulated pancreatic secretion. This part is due to distension of stomach when food particles enter into stomach. The entry of food particles produces gastrin release and vagal nerve stimulation.

3) **Intestinal phase** :

The food particles and gastric juice enters into the duodenum which intern stimulate pancreas to secrete pancreatic juice. The lipids and proteins, their partially digested products stimulate cholecystokinin release from mucosa of duodenum. This enzyme results in release of enzyme rich secretion from pancreas. This stage constitutes about three fourth of total pancreatic secretion.

Feedback mechanism:

The duodenal mucosa secretes factor for release of cholecystokinin, pancreas secretes a peptide for monitoring. Trypsin proteolysis these two factors. In the

presence of high protein diet inside the duodenum, releasing factor stimulates cholecystokinin which again lead onto stimulation of pancreatic secretion.

In the absence of food particle inside the duodenum, trypsin causes lysis of the releasing factor which will lead onto decrease in pancreatic secretion. In pancreatitis, there is insufficient pancreatic secretion which causes insufficient duodenal proteolysis. So the level of cholecystokinin release is increased .probably this is the reason for pain in chronic pancreatitis. It can be reduced by administration of exogenous pancreatic enzymes to reduce pancreatic stimulation.

Endocrine secretion :

Endocrine part of pancreas constitutes only less than two percent of total pancreas. The islets of Langerhans cells having 5 types of cells which is already discussed.

Insulin:

Insulin hormone which is discovered by Frederick and best in 1920. Insulin is a peptide hormone which contains fifty one amino acids. It has two chains which are alpha and beta. They are connected together by sulphur bonds and a connecting peptide called C- peptide. Insulin formed as pro insulin in the endoplasmic reticulum and passed into golgi apparatus. In the golgi apparatus insulin in packed into granules .The cleavage of C peptide occurs at this stage. Secretion of insulin occurs at two stages. During the first stage insulin released from stored granules. This stage occurs about five to six minutes after glucose challenging. In the second stage of secretion it is prolonged and sustained release due to new insulin produced continuously. Blood sugar levels and nerve signals regulate and influence beta cells to produce insulin. Glucose tolerance test is used for the diagnosis of impaired fasting glucose and diabetes mellitus. Oral glucose stimulates the secretion of gastro intestinal hormoneslike gastric inhibitory peptides,kinins which increase the insulin secretion, so these hormones called as incretin. So oral glucose will stimulate the insulin secretion more than intravenous glucose.

Insulin secretion from beta cells also depends on plasma level of amino acids and fatty acids. Glucagon also increases the insulin secretion. In contrst, somatostatin, pancreatostatin decrease insulin secretion. Acetyl choline fibers,

beta adrenergic nerves stimulate insulin secretion whereas alpha adrenergic nerves decrease insulin secretion.

Functions of insulin:

Insulin decrease the blood glucose level by the following mechanism. 1) It arrests glucose production from lipids and amino acids in liver. 2) It stops the breakdown of glycogen into glucose in liver and muscles. 3) It also stops the breakdown of lipids and ketone body formation 4) It enhances the deposit of lipids into adipose tissue and protein synthesis from amino acids.

Diabetes mellitus:

There are two main types of diabetes mellitus. Type 1 diabetes mellitus is due to insulin deficiency. Since there is insulin deficiency the receptors for insulin is up regulated which results in oversensitivity to insulin. Type 2 diabetes mellitus patients will have decreased insulin sensitivity, in other words insulin resistance is an important component of type 2 diabetes mellitus. It is leading onto overproduction of insulin and result in hyperinsulinemia.

Glucagon:

Glucagon secreted from alpha cells of islet of Langerhans. It is also involved in carbohydrate metabolism. It is a peptide hormone made up of twenty nine amino acids. It increases the blood glucose production from lipids and amino acids in liver. It enhances the breakdown of glycogen into glucose in liver and muscles. Glucose inhibits the secretion of glucagon. Arginine, an amino acid enhances glucagon release. GASTRIC INHIBITORY peptide, cholinergic nerves, and beta adrenergic nerves stimulate insulin release. Insulin, glucagon, alpha adrenergic nerves decrease the glucagon secretion.

Somatostatin:

Initially somatostatin was identified from hypothalamic region, but now it is found to be distributed in various parts of the human body. It is seen in pancreas, intestines and other parts of the human body. It is involved in regulatory mechanism of the metabolic processes of the body. Two types of biologically active product of somatostatin present which is coded by a single gene. These two types of somatostatin are tissue specific. It is a peptide type hormone. It is usually involved in regulation of exocrine and endocrine secretory process and neural transmission, intestinal peristalsis, gastro intestinal vascular tone and cell division. Five types of receptors for

somatostatin are identified. These receptors are G- protein coupled type, which will have intra cellular transmitters. The somatostatin analogues bind only to type 2 , 3 ,5 receptors. Their potent inhibitory action used for the treatment of exocrine and endocrine pancreatic disorders. Octrotide has effect on decreasing the output in entero cutaneous fistula and pancreatic fistula.

Food intake stimulates somatostatin release. Fat in the lumen of intestine is the most powerful stimulant of somatostatin release. Gastric acidity and acidity in duodenal lumen stimulates somatostatin release. Stimulation of para sympathetic fibers will lead onto decrease in somatostatin release.

Pancreatic polypeptide:

During purification of insulin Kimmel discovered pancreatic polypeptide. It is a peptide which has straight chain. This polypeptide is released mainly in response to proteins. Lipids and carbohydrates are having weak effect on pancreatic polypeptide secretion. Hypoglycemia potentially stimulates pancreatic polypeptide release. Glucagon and somatostatin hormones mainly having inhibitory effect on polypeptide release.

Stimulation of parasympathetic nerves to pancreas is having the pre dominant regulatory effect on pancreatic polypeptide secretion. The raised level of this pancreatic polypeptide hormone after meal can be eliminated by

truncalvagotomy. So this can be used as a diagnostic tool to diagnose whether the vagotomy has been performed completely or not and also to diagnose the autonomic neuropathy.

Function of pancreatic polypeptide:

Pancreatic poly peptide decreases biliary secretion and contraction of gall bladder. It has inhibitory action on pancreatic exocrine secretion. From recent trials it is clear that pancreatic polypeptide has an important role in regulation of blood sugar via regulation of expression of genes for insulin receptor in liver. There is reduced insulin sensitivity in liver due to inadequate secretion of pancreatic polypeptide in the conditions like post proximal pancreatectomy and chronic pancreatitis. This condition can be treated by pancreatic polypeptide administration.

Other hormones:

1) **Ghrelin:**

   It is secreted from epsilon cells. These cells are present in higher levels in the fundus of stomach. It stimulates secretion of growth hormone through its growth hormone release hormone (GHRH) release property from the pituitary gland. It is the potent stimulator of appetite so called

orexigenic. In obese individuals the level of hormone is high in plasma samples. Ghrelin hormone inhibits effect of insulin in hepatic parenchyma. It also inhibits the effect of incretins and sugar on pancreatic beta cells.

2) **Amylins:**

It is secreted by beta cells. It is stored in granules along with insulin hormone. Its action is to modulate insulin release and its target action.

ACUTE PANCREATITIS

Definition

Acute pancreatitis is characterized by the sudden onset of abdominal symptoms in a previously normal persons and disappearance of these symptoms once the attack got resolved. It is an inflammatory process of pancreatic parenchyma,which has little or absence of fibrosis of gland.

Etiology

Acute pancreatitis has different types of etiological factors. In western countries biliary tract disease like calculus is the leading cause of acute pancreatitis. Alcoholism is the important cause of pancreatitis in developing countries. Both alcoholism and biliary tract calculus are responsible for seventy five to eighty five percent of acute pancreatitis. About ten percent of cases of acute pancreatitis had no identifiable etiology which is considered as idiopathic pancreatitis. Hereditary pancreatitis associated with mutations in genes of trypsin inhibitors. In hereditary pancreatitis patients will be having frequent attacks, finally develops calcification of entire pancreas and development of diabetes. Other causes are pancreatic duct obstruction due to congenital, biliary tract pathology, periampullarytumours, endoscopic procedures.

Alcohol:

Acute pancreatitis is commonly occurs in chronic alcoholics of at least more than two years. But it can happen even in a single time alcohol user. It may possible that the initial attack in chronic alcoholics is actually the first symptom of chronic pancreatitis. In alcoholics, it is the recurrent attacks ultimately lead to chronic pancreatitis. The daily intake of more than or equal to 150 grams of alcohol has significant percent of developing acute pancreatitis. Similar proportion of patients can be affected by cirrhotic changes of liver.

Alcohol causes increased tone of sphincter of oddi which lead on to blockage of secretions. Metabolites of alcohol are toxic to acinar cells which will affect enzyme synthesis and release. There is increased protein content with enzymes initially which is deposited inside the wall of ductal system of pancreas. Calcium precipitates proteins in the ductal system which will lead on to blockage of ductal system. All these ultimately result in increase in ductal pressure. Alcohol also causes increase in permeability of pancreatic ducts. It leads to extravasation of partially activated enzymes into parenchyma of pancreas. And also ethanol itself activates trypsin inside the pancreas. Alcohol reduces pancreatic arterial supply which causing localized ischemic

damage in the parenchyma. Alcohol also causes alteration in fat metabolism which in turn leads on to hyperlipidemia.

Biliary diseases:

Acute pancreatitis is commonly associated with biliary tract calculi or acalculus disease. The possible pathology of development of pancreatitis in biliary stones is uncertain. There may be blockage of duct below the confluence of biliary and pancreatic ducts which leads to bile inflow into the pancreatic duct. Then the damage of pancreas will be caused by the bile salts which is having the detergent action.

Another mechanism is when the gall stones pass through the sphincter of oddi, they will make the sphincter to become incompetent. After that the contents of duodenum can reflux in to the pancreatic duct which causes damage to the pancreas ultimately result in acute pancreatitis.

The obstruction of pancreatic duct leads to increased ductal pressure result in disruption of small ductal system which in turn lead on to leakage of secretion into the parenchymal tissues. In the interstitial site the pH is less when compared to the intra ductal pH which is more alkaline. When enzyme activation occur, it leads to protease enzyme activation causing injury to parenchyma. The mechanism of extravasation and increased intra ductal

pressure which causes pancreatitis is still unclear. Normally inactive zymogens and lysosomal enzymes are placed in separate organelles. In ductal hypertension or obstruction, colocalization of substances occur in vacuoles of acinar cells. This will ultimately result in activation of trypsin and further activation of other enzymes. Finally the activated zymogens cause auto digestion of pancreas result in pancreatitis.

Neoplasms:

About two percent of patients with pancreatitis will be having malignancy in pancreas and periampullary growth. An attack of acute pancreatitis can be the initial manifestation of periampullary growth. Possible mechanism of pancreatitis in this condition will be ductal obstruction with blockage of secretions.

Medications:

Some drugs are capable of producing acute pancreatitis. But in practical aspect, some drugs cause increased serum amylase and abdominal pain which mimic acute pancreatitis. All these symptoms will subside once the medicine is stopped. Drugs commonly associated with pancreatitis are diuretics (thiazide group and loop diuretics), hormones like oral contraceptive pills, immune suppressants like azathioprine, cancer chemo therapeutic agents like

alkylating agents and antimetabolites, some anti microbials and anti epileptics,anti-retroviral drugs(didanosine).

Hyperlipidemia:

In altered lipid metabolism, there is high levels of toxic fatty acids enter into pancreatic capillary circulation by the action of lipase enzyme. These can lead to endothelial damage, increased viscosity of blood ultimately result in ischaemic damage to the pancreas. Patients with familial hyper lipoproteinemias and hypertriglyceridemias will be having recurrent attacks of pancreatitis by the above mentioned mechanism. The attack rate of pancreatitis can be prevented by dietary modulation and reduction in the level of saturated food intake.

Infections:

Viral infections like mumps and coxsackie B virus can cause acute pancreatitis by infecting acinar cells. Mycoplasma can also cause pancreatitis. In one third of patients with pancreatitis the antibody titers for mumps and coxsackie virus are increased when the cause in not defined. Cytomegalo virus causes pancreatitis in HIV infected individuals. About half of patients of HIV having signs of acute pancreatitis.

Obstruction

Duodenal pathologies like duodenal ulcers, inflammatory bowel disease affecting the duodenum, periampullary carcinomas can cause pancreatitis. Periampullary diverticulosis can cause pancreatitis because the diverticulum filled with food debris. Most of the patients with obstruction will present with chronic pancreatitis. This type affects only the obstructed part and cam be treated with the removal of obstructed portion of pancreas. Post traumatic strictures can cause obstruction which leads to pancreatitis. Round worm and biliary flukes can cause obstruction which may lead on to pancreatitis. Congenital conditions like pancreatic divisum can cause pancreatitis

Hereditary type:

Normally some kind of activation of trypsinogen occurs inside of pancreas. But it is protected by presence of trypsinogen activation inhibitors. SPINK1 gene codes for the trypsin inhibitors. Any mutation which results in decreased or defective synthesis of trypsinogen activation inhibitors will result in auto digestion of pancreas. In hereditary pancreatitis the pathological process starts in the early part of life which causes chronic changes like calcification and

fibrosis. Apart from chronic pancreatitis these conditions are also having high risk of pancreatic malignancies.

Auto immune type:

This condition is frequently associated with other auto immune diseases like primary sclerosing cholangitis, primary biliary cirrhosis, sjogren's syndrome. This condition is characterized by high degree of sclerosis and lymphocyte infiltration. This is usually results in biliary and pancreatic strictures. Laboratory reports will reveal high levels of circulating antibodies (Ig G). This condition may present as mass in the head of pancreas and ductal strictures and is often confused with pancreatic malignancies. Steroid use may be of immense value in this condition.

Miscellaneous causes:

1)**Trauma:**

Blunt injury of abdomen may result in disruption of ductal system of pancreas and may result in pancreatitis. Contusions, lacerations and small ductal disruptions will initiate the inflammatory process only to end up with pancreatitis.

2)**postoperative :**

Surgical procedures done within the pancreas or near to pancreas like open heart surgeries, heart transplantation, kidney transplantation will result in hypo perfusion of the pancreas to end up with pancreatitis.

3)**endocrine abnormalities**:

Hypercalcemia due to hyperparathyroidism is an important endocrine cause of pancreatitis we should keep in our mind. The mechanism of hypercalcemia induced pancreatitis is enhanced activation of zymogens intrapancreatically. By treating the underlying hyperparathyroidism we can prevent the development of pancreatitis

4)**scorpion sting:**

Scorpion venom contains stimulants which will increase the pancreatic secretion and result in pancreatitis.

5**)ERCP**:

Pancreatitis is one of the expected iatrogenic complications in one to twelve percent of patients. In fifty percent of patients who undergone ERCP asymptomatic elevation of serum amylase is identified. The possible mechanism is direct injury to the ductal epithelium during ERCP.

Idiopathic:

For about fifteen to twenty percent of patients with pancreatitis will have no identifiable pathology in them. Some of them may have sludge in the gall bladder and micro crystals. The subsequent attack can be prevented by cholecystectomy or endoscopic sphincterotomy. Mutation in cystic fibrosis gene will also result in pancreatitis. This mutation causes fibrosis of the pancreas and deficiency of both exocrine and endocrine hormones due to blockage of duct with secretions.

Pathology:

Macroscopic appearance:

The macroscopic appearance depends on the severity of the disease. It ranges from mild inflammation with edema of pancreas to severe necrosis of parenchyma and hemorrhagic areas. The alterations include,

1) edema due to leakage from capillaries

2) fat necrosis resulting from the action of lipolytic enzymes

3) acute inflammation

4) destruction of parenchyma due to the action of proteolytic enzymes

5) vessels disruption with haemorrhagic areas

Pathogenesis:

The exact mechanism of development of pancreatitis is unclear. The most popular theory is the intra pancreatic activation of zymogens result in damage to acinar cells. Recent studies suggest that the severity of pancreatitis can be determined by events following the acinar cell injury. These are sequestration

of inflammatory cells and their activation,production of cytokines and chemical substances involved in inflammation.

Initial events:

Under normal conditions, pancreas produces a huge amount of proteins in which the majority of them will be digestive enzymes. In the active form they are injurious to the pancreas. But the auto digestion is prevented by their inactivated forms with which they are stored intracellularly known as zymogens. These inactive forms are transported intracellularly and secreted out of the cells by exocytosis mechanism. In duodenum their activation is initiated by brush border enzyme called entero kinase. Enterokinase converts trypsinogen into active form trypsin. Trypsin will subsequently activates other enzymes.

The zymogens are segregated from the cytoplasmic space within the acinar cells by the membrane bound organelles called zymogen granules. One more major amount of proteins also stored along with the inactivated forms of enzymes. They are trypsin inhibitors. They are transported along with the inactive zymogen granules. These also inhibit the action of prematurely activated trypsinogen within acinar cells. Theoretically pancreatitis occurs

when these zymogens are enormously activated. The reasons are 1) pancreas can be digested with activated enzymes in duodenum. 2)during pancreatitis the activated enzymes present within the pancreas 3)histopathological examination shows features of coagulative necrosis. The mechanism of enormous activation is not clearly defined.

Recent studies regarding the exact etiology of mechanism of activation reveal the synthesis and transport of zymogens are unaffected during pancreatitis. But the secretion into the lumen is grossly reduced. In the early stage the enzymes are stored in vacuoles which also contains hydrolase cathepsin B. Cathepsin B activates trypsinogen. This is done by colocalization mechanism. Further support of this theory is based on inhibition of cathepsin B by its specific inhibitors(CA-074 me), protect acinar cells from trypsinogen activation. The activated trypsin in vacuoles mediates the permeability of these organelles and causes release of these enzymes into the cytoplasm. It initiates cell death by apoptotic mechanism evidenced by increased permeability of mitochondrial membranes and release of cytochrome C into the cytoplasm. These events finally initiate apoptotic cascade events.

Inflammatory events:

The activation of trypsin will lead onto other zymogen activation like phospholipase and pro elastase. These activated enzymes disintegrate fat cells and rupture the elastic tissues of small blood vessels. Trypsin also causes activation of kallikrein system, complement and coagulation system. These events result in thrombus formation in small vessels and congestion and rupture of blood vessels.

Increased vascular permeability will result in edematous pancreas due to protein rich fluid in the interstitial space.

Leucocyte infiltration is mediated by chemical mediators which include selectins, antibodies, integrins and glycoproteins.

Selectins:

L selectin(CD62L),E selectin (CD 62 E), P selectinare involved in binding of poly morpho nuclear cells to cytokine activated endothelium.

Integrins:

These proteins enhance interactions between inter cellular and intra cellular matrix.

Morphology:

In mild forms of pancreatitis – interstitial edema, foci of fat necrosis within the pancreas and peri- pancreatic fat, saponification of fatty acids with calcium are seen.

In severe forms of pancreatitis also called necrotizing pancreatitis – parenchymal hemorrhage along with foci of chalky white fat necrosis are seen. Fat necrosis is also seen in extra pancreatic sites like omentum and mesentry of small and large bowel.

In most severe forms of pancreatitis – diffuse necrosis of parenchyma along with extensive intra parenchymal haemorrhage are seen.

Clinical manifestations:

Abdominal pain located in the epigastric region and also in the hypochondrial region which is referred to back is the characteristic nature of pain we will get in acute pancreatitis. The pain is usually continuous and pt usually feels better in the leaning forward position. Other symptoms are nausea, vomiting.

Physical findings:

Patient moves all around to search the comfortable position. Fever, tachycardia, tachypnea, cardio vascular instability, basal atelectasis of lungs,

left sided pleural effusion is seen in most of the patients. ARDS, altered mental status are seen in severe form of pancreatitis.

Varying degrees of jaundice is usually seen in biliary stone disease induced pancreatitis. Paralytic ileus, rebound tenderness, guarding of abdomen, epigastric mass (edematous pancreas with surrounding tissues) are also common in them. Ecchymosis of the flank region called, **grey turner's sign** and ecchymosis of the peri umbilical region called **cullen's sign** present in severe forms of acute pancreatitis due to retro peritoneal haemorrhage. Subcutaneous fat necrosis may present as erythema nodosum.

Diagnosis of acute pancreatitis:

Clinical:

The characteristic type of pain with the associated history of alcoholism or biliary tract stone disease will make us to think in terms of acute pancreatitis. But still it is our duty to exclude the other conditions which mimic pancreatitis like hollow viscous perforation, acid peptic disease, small bowel obstruction, cholecystitis.

Biochemical:

The serum amylase level starts increasing 2-12 hours after the attack of acute pancreatitis and returns to normal level within 3-5 days. But the urinary amylase level remains elevated than the serum level. Serum amylase is not getting elevated in all the cases of pancreatitis. The amylase level does not correlate with the severity of pancreatitis. In 10% of patients with severe pancreatitis the amylase level found to be normal. In necrotizing pancreatitis amylase is not entering the systemic circulation. So in severe pancreatitis it seems that the amylase measurement is not much of a value. Persistent elevation of serum amylase indicates the occurrence of complications like pseudo cyst formation, pancreatic ascites, abscess formation. Serum amylase also elevated in other conditions like perforated small bowel, small intestinal obstruction. Measurement of pancreas specific amylase (P-Amylase) makes the diagnosis more specific.

Serum lipase is more specific than amylase in diagnosing acute pancreatitis. The rise is usually parallels the serum amylase elevation.

C-Reactive Protein:

it is mainly used for prognostic implications. If the level of CRP goes more than 1.3 mg/100 ml the possibility of developing complications is also high. It is 85% sensitive in the first 3 days of onset of symptoms.

Trypsinogen and its activation peptides:

Urinary concentration of this peptide level at the time of admission is greatly correlated with the severity of pancreatitis.

Pro calcitonin:

It is used to assess the severity and prognosis of pancreatitis. During the inflammatory process the inflammatory cytokines and the bacterial antigens induce the production of procalcitonin. Important aspect of this test is its resistance to infections which has high correlation with the severity. High levels of procalcitonin will result in vasodilatation and extravasation of fluid. It leads to complications like organ failure.

Analysis in 2006 study revealed the procalcitonin level is having high specificity in assessing the severity of pancreatitis. But the sensitivity is only moderate.

Elevated hematocrit level is the common finding due to vomiting, hypovolemia and capillary leak. Hemoglobin, BUN, serum creatinine levels to be measured. Serum albumin level will be decreased. Vomiting will lead onto hypochloremic alkalosis. WBC count will be elevated. Serum bilirubin level may also get elevated due to biliary stones or edematous pancreas causing compression of bile duct. Hypertriglyceridemia is also seen in some pancreatitis patients. Hypocalcemia is associated with poor prognosis. In severe pancreatitis features of DIC may also be present.

Radiological diagnosis:

Plain X ray:

X ray of abdomen and chest is usually indicated to rule out hollow viscous perforation or intestinal obstruction. CXR may also reveal basal atelectasis and left sided pleural effusion. X ray abdomen may also reveal paralytic ileus, bowel gas in duodenal loop, abrupt narrowing of gas shadow in splenic flexure called, **colon cut off sign.**

Ultrasonography:

It is useful in diagnosing gall stone pancreatitis, extra pancreatic dilatation of bile duct, pancreatic edema, free fluid in the peritoneal cavity.

Computerized tomography:

CT scan with intravenous contrast is the gold standard investigation to detect acute pancreatitis. It is also more helpful in assessing the severity of acute pancreatitis. In mild forms of pancreatitis when the micro vasculature is intact there is uniform enhancement of contrast material throughout the gland. In severe pancreatitis it detects the pancreatic necrosis and fluid collection inside the pancreas and the peripancreatic region. The contrast enhancement is markedly decreased in necrotizing pancreatitis.

MRI:

MRI is also having the same sensitivity and specificity similar to CT in diagnosing acute pancreatitis. MRI is having the advantage of avoiding radiation and nephrotoxic contrast material. It will identify the vessel thrombosis and pseudo aneurysm.

Assessment of severity:

Acute pancreatitis has challenging clinical course in the prediction of severity and prognosis. According to most of the case reports the severe cases contributes to 15-25%. In the following ways we can assess the severity.

Approach:

We can assess the severity from history, symptoms and signs, laboratory findings, radiological imaging. In the initial periods the serum amylase is the only investigation used to diagnose acute pancreatitis. In 1970, John Ranson attempted to define the objective criterias to assess the severity of acute pancreatitis. During the early phase of 1980, the severity of pancreatitis is assessed only intra operatively after seeing the amount of necrosis and the presence of signs of infection in necrotic tissues. After the introduction of CT the severity assessment of acute pancreatitis becomes an uncomplicated one. In initial days the morphological criteria is used. Nowadays the presence of multi organ failure dominates the clinical picture.

Clinical factors:

Age and comorbid illness:

Advanced age & the presence of other comorbid conditions like diabetes and cardiac diseases associated with poor prognosis.

Clinical signs:

Presence of fever, cardio vascular instability, respiratory distress, altered sensorium, mass abdomen, paralytic ileus increases the mortality to 25 % in operated patients and 70% in conservatively managed patients.

Atlanta classification:

This classification differentiates between mild and severe types of acute pancreatitis. It depends on the clinical and morphological findings.Mild forms otherwise called edematous pancreatitis will have minimal organ dysfunction that will lead onto good outcome. Morphology will reveal edema in interstitium and microscopic fat necrosis. The other form severe or necrotizing pancreatitis will have features of multi organ dysfunction, pancreatic necrosis, abscess, pseudocyst formation. Morphology reveals extensive fat necrosis and hemorrhagic areas in pancreas or peri pancreatic fat.

Multi parameter scoring ystem:

Ranson and Clement Imrie proposed the analysis of multiple clinical and biochemical parameters which contributes to complications and death. Their system paved the way for the development of multiple newer scoring systems.

Ranson system:

This system analyzes the severity from the clinical and the biochemical parameters during and 48 hours after admission. A total of 11 points were taken. Five from the time of admission and the remaining from the next assessment after 48 hours from the abnormal values were taken. Since the accuracy and the positive predictive values are low in case of biliary disease induced pancreatitis this scoring system undergone some changes for biliary pancreatitis.

Imrie and Glasgow scoring system:

This system is based on an interventional study in acute pancreatitis. It includes age of the patient at the time of admission and physiological and biochemical parameters obtained during the first two days of admission. There are some changes to improve the performance in biliary stone pancreatitis. Even after modification the sensitivity and the specificity remains only to a

moderate level. The overall sensitivity is less than 70% and the positive predictive value is 70%.

APACHE II system:

The Acute Physiology And Chronic Health Evaluation developed by intensive care research unit in United States. There are some changes from the initial system which reduces the variables from 35 to 11 which is called APACHE II. This system evaluates the patient in 24 hours and at 48 hours. The performance of APACHE scoring system is the same during hospitalization and at 24 hours. The results may vary depending upon the etiology of the disease and intensive care set up. The advantage of APACHE system is in its speed and easiness in application. There is also a possibility that recalculation is possible at any period throughout the disease for monitoring. The Atlanta classification proposed that score of 8 or more of APACHE II scoring system indicates severe attack.

Organ Failure scoring system:

This scoring system is applied in some studies to assess organ failure and prognosis. The MOF scoring system is mainly concentrating the prediction of survival based on organ failure. The cardio respiratory system, renal system, haematological, hepato biliary, neurological and alimentary system is

monitored in this scoring system. The Marshall and SOFA are the newer system of organ failure scoring systems which describe the individual and multi organ failure. In both systems, six major systems are considered which include respiratory, cardiac, hepato-biliary, renal, neurological and haematological system. In SOFA scoring system specific parameters like ventilatory support and inotropic supports are added.

MOF scoring system:

This system is based on the presence or absence of multi organ failure. This scoring system poorly assesses the complications like necrosis. The prediction of nonsurvival when the score is more than 3 is having high sensitivity and specificity.

Marshall scoring system:

Marshall scoring system is a modified one because it excludes the liver function. Other systems are monitored. The parameters closely correspond to Atlanta classification. There are two scores assessed in first 3 days of admission. Marshall scoring system is having comparable results with APACHE II scoring system in prediction of mortality in acute pancreatitis.

SOFA scoring system:

The SOFA scoring system gives the advantages like very easy calculations in therapeutic requirements and can compare acute pancreatitis with other diseases which are in need of critical care.

Hematocrit:

More than 44% during admission is related to complications like necrosis or multi organ dysfunction.

BUN & Serum creatinine:

Renal failure (serum creatinine> 2 mgs%) is an important predictor of mortality.

Blood glucose:

At the time of admission if the blood sugar value is more than 250 mgs there is high chance of development of pancreatic necrosis, multi organ failure and mortality.

Trypsin activation peptide &carboxy peptide B activation peptide: According to atlanta scoring system the measurement of CPAP has both diagnostic and prognostic significance. It is also correlated with the severity

of the disease. The limitation of this parameter is the requirement of RIA for its measurement.

Cytokines:

IL-6 & IL-8 measurements are having importance in assessing the severity of the disease. IL-6 level is a good indicator of severity. It is elevated enormously in complicated acute pancreatitis. The rise in CRP occurs 24-36 hours earlier than CRP. IL-8 is found to be the early marker in the prediction of severity in the first 24 hours of attack. It decreases rapidly over the next 72-96 hours. Its rise is in parallel line with IL-6. It is proved to be the best marker for monitoring the fatal complications.

Pro calcitonin:

This is one of the best biochemical parameter the level of which correlate well with the occurrence of infection and sepsis. Infection occurs in the setting of necrotic tissues which is having a major impact on survival and also alters the mode of treatment.

Radiological assessment:

Balthazar classification and severity index:

Balthazar classifies the severity of acute pancreatitis into 3 different stages mild, moderate, severe.

Stage A (0): About 15-28 % of patients with acute pancreatitis are showing normal CT images. This indicates that the disease is very mild so that there is no fluid collection and no parenchymal changes. In this state the diagnosis is very challenging.

Stage B: Alteration of parenchymal contour and heterogenous attenuation of the parenchyma are the imaging findings in this state.

Stage C: Along with the stage B changes there will be presence of streaky densities in the peri pancreatic region.

Stage D: It includes stage C changes and a single focus of fluid collection in the peri pancreatic region.

Stage E: There are multiple focus fluid collections in the peri pancreatic tissues along with the retro peritoneal air which indicates presence of infection.

Mortele modification of severity index:

This is the modification of Balthazar severity index. It gives a score of 0 to the normal pancreas. Score of 2 indicates intra pancreatic abnormalities along with the presence or absence of peri pancreatic inflammatory process. Score of 4 denotes the presence of fluid collection in pancreas and/or the peri pancreatic region or the presence of pancreatic necrosis. Additional points are given depending upon the extent of pancreatic necrosis. Additional 2 points are also added if there are extra pancreatic complications like pleural effusion, free fluid in the abdomen, vascular involvement. Severity score of 0-3 indicates mild variant, 4-6 indicates moderate type, 7-10 shows severe pancreatitis.

Correlation of modified CTSI with other parameters:

1) Length of hospitalization is increased in patients with severe forms.
2) The percentage of patients undergoing surgical procedure is also increased in severe forms of pancreatitis.
3) Infections are more common in severe forms.
4) Organ dysfunction is common in severe forms when compared to the mild ones.

Treatment

Initial management:

The management principles include early diagnosis, to assess the severity, to identify the complications and manage the patient in a multi disciplinary approach. When the abdominal exploration is planned the diagnosis should be thought of and better to be confirmed or excluded because the incidence of morbidity and mortality is very high in postoperative patients.

Management of pain:

Meperidine analogues are the mainstay of analgesics used in the treatment of pancreatitis. Morphine is relatively contra indicated as it may increase the sphincter of oddi spasm.

Fluid therapy:

Intensive fluid therapy with electrolyte replacement is the mainstay of treatment in acute pancreatitis due to third space loss of fluid in retro peritoneum. This will also lead onto increased hematocrit and hypotension. Vomiting will lead onto metabolic alkalosis whereas hypotension will lead on to metabolic acidosis. Hypoalbuminemiaiscaused by both chronic alcoholism

and the acute attack of pancreatitis. Hypocalcemiacan be treated by intra venous calcium administration.

Ryle's tube aspiration will not alter the course and the prognosis of the disease.

Role of antibiotics:

Prophylactic antibiotic may be of immense value in severe pancreatitis with infective complications.

Feeding:

Early oral feeding is advisable if the patient is able to tolerate it because it will prevent bacterial translocation and infectious complications.

Endoscopic stone retraction:

Early endoscopic stone retraction in biliary stone pancreatitis reduces the incidence of further attacks and infective complications.

Complications:

1) **pancreatic fluid collection:**

This condition is observed in about half of patients with the initial stage of pancreatitis. There is absence of wall of fibrous tissue. Some collections are within the pancreas. Some are in the peri pancreatic tissues. More than 50% of the collections will resolve over time. Unresolved collections will turn into pseudocyst of pancreas. This condition is easily diagnosed by serial ultra sound or CT scan of the abdomen. The fluid may get infected which can be identified by the presence of air bubbles. This condition is managed by either surgical or percutaneous drainage.

2) **Pseudo cyst formation**:

pseudocyst formed from unresolved pancreatic fluid collections in edematous pancreatitis. The contents are pancreatic secretions with large amount of enzymes which are covered with fibrous wall. It occurs not only within the pancreas but also in omental bursa. Approximately 50% pseudocyst of pancreas resolves spontaneously by rupture either into the pancreatic duct or into the other parts of the alimentary tract. It may lead onto further complications like obstruction to the pancreatic duct or intra pancreatic portion of bile duct and duodenum. It may also erode into adjacent organs. Bleeding into the cyst either from the artery or vein, infection of the cyst are

also the other complications. Infected pseudocyst of pancreas can be drained either percutaneously or by surgical drainage.

3) **Post necrotic fluid collection**:

In necrotising pancreatitis, necrosis and disruption of pancreatic duct will lead onto leakage of secretions into the necrotic portion of the pancreas. It is often confussed with acute pancreatitis.

4) **Vascular involvement**:

Vessels involvement in acute pancreatitis is characterized by erosion of pancreatic vessels which will lead onto bleeding and subsequent pseudoaneurysm formation. Splenic vein thrombosis is also one more complication. CECT of the abdomen and MRI will be useful in diagnosing this condition. Bleeding will lead onto death of the patient if not controlled in the right time. Bleeding from the pseudo aneurysm is suspected when there is a sudden drop in the hematocrit level during acute attack. It can be easily controlled with angiography followed by transcatheter embolization.

5) **Biliary tract involvement**:

Obstruction of bile duct due to edema of pancreatic head or compression by pseudocyst of pancreas. In necrotizing disease, the periductal inflammation

also causes obstruction. Pseudocyst of pancreas can encroach directly into the duct and produces erosion of intra hepatic bile ducts.

6) **Gastro intestinal involvement:**

Post necrotic fluid collections can traverse through the transverse meso colon, mesentry of jejunum and also into the retro peritoneal structures. Stomach and duodenum are also involved when the collections are present in the omental bursa or in the perinephric region.A rare condition called groove pancreatitis in which the inflammation occurs at the level of pancreatic head within the groove that separates the duodenum and the pancreas.

7) **Splenic vein thrombosis and the formation of gastric varices:**

This complication occurs in severe forms of pancreatitis. It may lead onto gastric varices formation. Since bleeding from these varices is rare, there is no requirement of prophylactic treatment. If bleeding occurs it can be controlled by splenectomy.

8) **Pancreatic ascites:**

Secretions into the peritoneal cavity due to rupture of ducts or pseudocyst. It can be diagnosed by measurement of amylase level in free fluid. This condition is initially treated with conservative line of management like nil per oral, Ryle's tube aspiration and attempt to reduce the secretions by somatostatin analogues. 50% of patients will respond to this type of

conservative management. It is usually getting resolved within 14-21 days. Recurrent ascites is treated by stenting of the pancreatic duct endoscopically. ERCP can be used to locate the site of disruption. If the site of disruption is in the distal part resection will be the ideal treatment. If it is in the proximal part, Roux –en-Y drainage can be done.

9) **Pancreatic fistula:** Pancreatico pleural fistula can occur when the disruption of duct occurs into retro peritoneal pancreatic secretions which will finally enter into the pleural space. It is a rare condition

10**)Systemic complications:**

Organ dysfunction is common in the first week. Sometimes it can be transient& it will not be associated with fatal outcome. If it is persistent it may lead onto fatal outcome.

AIMS OF THE STUDY

Aims of the study:

1) To analyze the etiologies and the varying clinical presentations of acute pancreatitis.
2) To validate the clinical, biochemical and radiological signs in diagnosing acute pancreatitis.
3) To correlate the severity of acute pancreatitis with the clinical, biochemical and radiological signs.
4) To correlate the prognosis of the disease with the above mentioned parameters.

MATERIALS AND METHODS

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PATIENTS:

Total of sixty patients include both male and female of adult age group who got admitted in the emergency surgical ward with the symptoms of acute pancreatitis and evaluation confirmed the presence of this serious illness.

PLACE OF THE STUDY:

Tirunelveli medical college hospital.

PERIOD OF THE STUDY:

From February 2015 to august 2016.

INCLUSION CRITERIA:

Patients got admitted in the emergency surgical ward with the symptoms of acute pancreatitis in which the biochemical and radiological evaluation confirmed the presence of acute pancreatitis.Patients who had recurrent attacks of acute pancreatitis also included in this study.

EXCLUSION CRITERIA:

Patients with chronic pancreatitis are excluded from this study.

Patients with known co morbid conditions like diabetes mellitus, systemic hypertension, coronary artery disease, chronic obstructive pulmonary disease, chronic kidney disease are excluded from this study.

Paediatric age group not included in this study.

PARAMETERS USED:

Clinical manifestations: Symptoms and signs &Ranson's clinical scoring system

Biochemical parameters: Serum amylase and lipase.

Radiological: CT abdomen plain and contrast.

DIAGNOSTIC CRITERIA:

1) Clinical signs and symptoms of acute pancreatitis as described in review of literature.

2) Elevated serum amylase and lipase level

3) CT with contrast confirming the presence of acute pancreatitis.

SEVERITY ASSESSMENT:

Based on clinical (Ranson's criteria), biochemical (serum amylase and lipase) and radiological (contrast enhanced CT abdomen-Balthazar scoring) findings.

RANSON'S PROGNOSTIC SCORING SYSTEM

A) NON GALL STONE ETIOLOGY

During admission	Within 48 hours
Age >55 years	hematocrit fall >10%
TLC >16000/mm^2	elevation of BUN > 5mg%
Blood sugar >200 mg%	serum Ca^{++} < 8 mg%
LDH >350 IU/L	PaO_2< 55 mmHg
AST >250 U/ dl	base deficit >4 mEg/ L
	Fluid sequestration >6 litres

B) GALL STONE ETIOLOGY

Age > 70 years	hematocrit fall > 10%
TLC > 18000/mm^3	elevation of BUN > 2 mg%
Blood sugar >220 mg%	serum Ca^{++} < 8 mg%
LDH >400 IU/L	base deficit >5 mEg/ L
AST >250 U/ dl	Fluid sequestration >4 litres

TLC-Total leucocyte count , LDH-Lactate dehydrogenase

AST-Aspartate transaminase., BUN-Blood urea nitrogen.

RANSON'S SCORE 3 or Above- Defines severe pancreatitis

CT SCORING : (BALTHAZAR)

GRADING	APPEARANCE	SCORE
A	Normal appearance	0
B	Focal or diffuse enlargement of pancreas	1
C	Peripancreatic inflammation	2
D	Intra / extra pancreatic fluid collection	3
E	2 or more fluid collection / air in pancreas or retroperitoneum.	4

CT NECROSIS INDEX:

This is based on necrosis seen in the contrast enhanced CT scan.

Percentage of necrosis	scoring
0% of necrosed pancreas	0
<33% of necrosed pancreas	2
33 – 50 % of necrosed pancreas	4
>50% of necrosed pancreas	6

CT severity index = CT Balthazar score + Necrosis index.

Score 0 – 3 ---------- mild acute pancreatitis.

Score 4 -6 ---------- moderate acute pancreatitis

Score 7 or above------ severe acute pancreatitis

OBSERVATION AND DISCUSSION

OBSERVATIONS& DISCUSSION

1. CLINICAL MANIFESTATIONS & THE SEVERITY OF THE DISEASE:

In this study, various clinical symptoms and signs were observed. The following clinical manifestations were commonly encountered.

S.NO.	CLINICAL MANIFESTATIONS	PERCENTAGE
1.	Abdominal pain	100
2.	Abdominal distension	42
3.	Vomiting	72
4.	Fever	26
5.	Shock	6
6.	Guarding / rigidity	4
7.	Ascites	12

From this result, the presence of hemodynamic instability is an important factor associated with poor prognosis. The presence of massive ascites is also having poor outcome. Those two patients who presented with significant guarding and rigidity had bad outcome in terms of mortality.

2. SERUM AMYLASE AND LIPASE AND THE SEVERITY OF THE DISEASE:

In this study, serum amylase and lipase levels were elevated enormously in recurrent attacks eventhough they were milder forms. In case of severe pancreatitis, the levels of amylase and lipase show only mild to moderate elevation. Hence it is obvious that there is no correlation between the levels of serum amylase and lipase and the severity of the disease.

3. ETIOLOGY & THE SEVERITY OF THE DISEASE:

In this study of 60 patients, most of the patients were alcoholic. The various etiologies are listed below.

S.NO	ETIOLOGY	NO. OF PATIENTS	PERCENTAGE
1.	Alcoholism	55	90
2.	Biliary stones	2	4
3.	Malignancy	1	2
4.	Idiopathic	2	4

Almost all severe forms of pancreatitis were due to alcoholism. The two cases with biliary stone as the etiologic factor had milder form of acute pancreatitis. Of these sixty patients, only one patient developed pancreatitsin malignancy.

RANSON'S SCORE AND THE SEVERITY OF THE DISEASE:

Most of the patients hadRanson's score of 1 and 2. Out of 60 patients,8 patients were expired. All of them had Ranson's score of 3. From this we can conclude that the severity of the disease correlate well with the Ranson's score.

Ranson's score	Number of patients
0	5
1	42
2	9
3	4

5. BALTHAZAR SCORE AND THE SEVERITY OF THE DISEASE:

The following table shows analysis of different CT scoring.

CT GRADING	NO OF MALES	NO OF FEMALES
A	0	2
B	46	3
C	4	0
D	3	1
E	4	0

From this study, we can conclude that most of the attacks of acute pancreatitis were milder forms. In one patient the CT abdomen showed normal pancreas

associated with elevated serum amylase and lipase levels. Grades A and B accounted for more than 75% of the patients.

SEX DIFFERENCE AND THE SEVERITY OF DISEASE:

Most forms of severe pancreatitis occurred in male patients. The severity between the sex groups cannot be assessed correctly as the number of female patients in this study is insufficient.

COMPARISON BETWEEN RANSON'S SCORING AND CT SCORING IN SEVERITY OF DISEASE:

CT GRADING	AVERAGE RANSON'S NUMBER
A	1
B	1
C	1
D	2
E	2.5

Both CT grading and Ranson's number are correlating directly with the severity of the disease. From this study we can conclude that there is a linear progression between the Ranson's scoring and CT scoring in assessing the severity of the disease as shown in the line diagram.

DURATION OF THE HOSPITAL STAY AND THE SEVERITY OF THE DISEASE:

CT GRADE	DURATION OF THE HOSPITAL STAY
A	10 days
B	7.7 days
C	10 days
D	6.5 days
E	11.5 days

The duration of the hospital stay does not correlate with the CT or Ranson's grading as the patients with the higher grade had shorter hospital stay due to the death of the patients which is because of the high mortality associated with the higher grades.

RANSON'S SCORE AND THE MORTALITY RATE:

The eight patients who died had Ranson's score of 2 or 3. Thus the Ranson's score is surely having an influence on the prognosis of the patient.

Ranson's score	No of deaths
0	0
1	0
2	2
3	6

MORTALITY IN DIFFERENT CT GRADES:

CT grades	Number of deaths
A	0
B	0
C	0
D	3
E	5

From this study CT gradings A, B & C show better prognosis when compared to grades D & E. CT grades D & E show bad outcome in terms of mortality. In grade D the mortality rate is 50 % (3 out of 6 patients were expired). In grade E the mortality rate is 75 % (5 out of 6 were expired).

FOLLOW – UP:

In this study, out of the 60 patients, 8 patients were died. In the remaining 52 patients, only 20 patients came for the follow – up. Of these 20 patients, 6 male patients had recurrent pancreatitis and one male patient had recurrent attack. He developed pseudocyst of the tail of the pancreas. He underwent cystogastrostomy. Patient improved symptomatically after surgical intervention.

INTERNATIONAL TRIALS :

Hill et al in 1983, conducted a study in which he studied a total of 83 patients. None of the patients in grade A and Grade B died. But 17 % of grade D and 61% of grade E developed infected fluid collections and died.

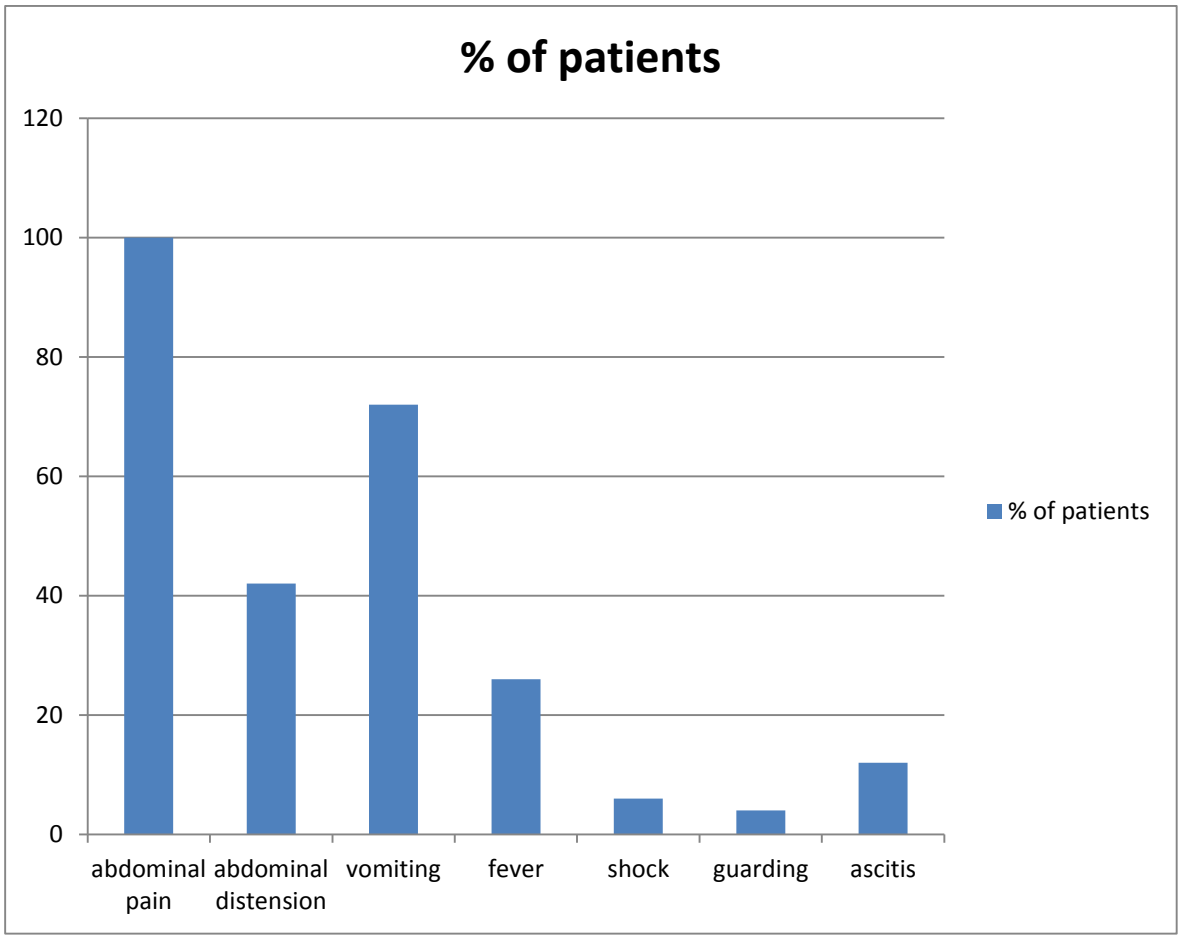
- Claviens et al conducted a study for evaluation of 176 patients. From this study he found that grade D and grade E had a protracted clinical course and development of most of the complications.
- In 1980 and 83, balthazar had conducted two separate studies. He found that CT scan is the most sensitive and specific in assessing the severity of the disease, the scoring is named after him.

CONCLUSION

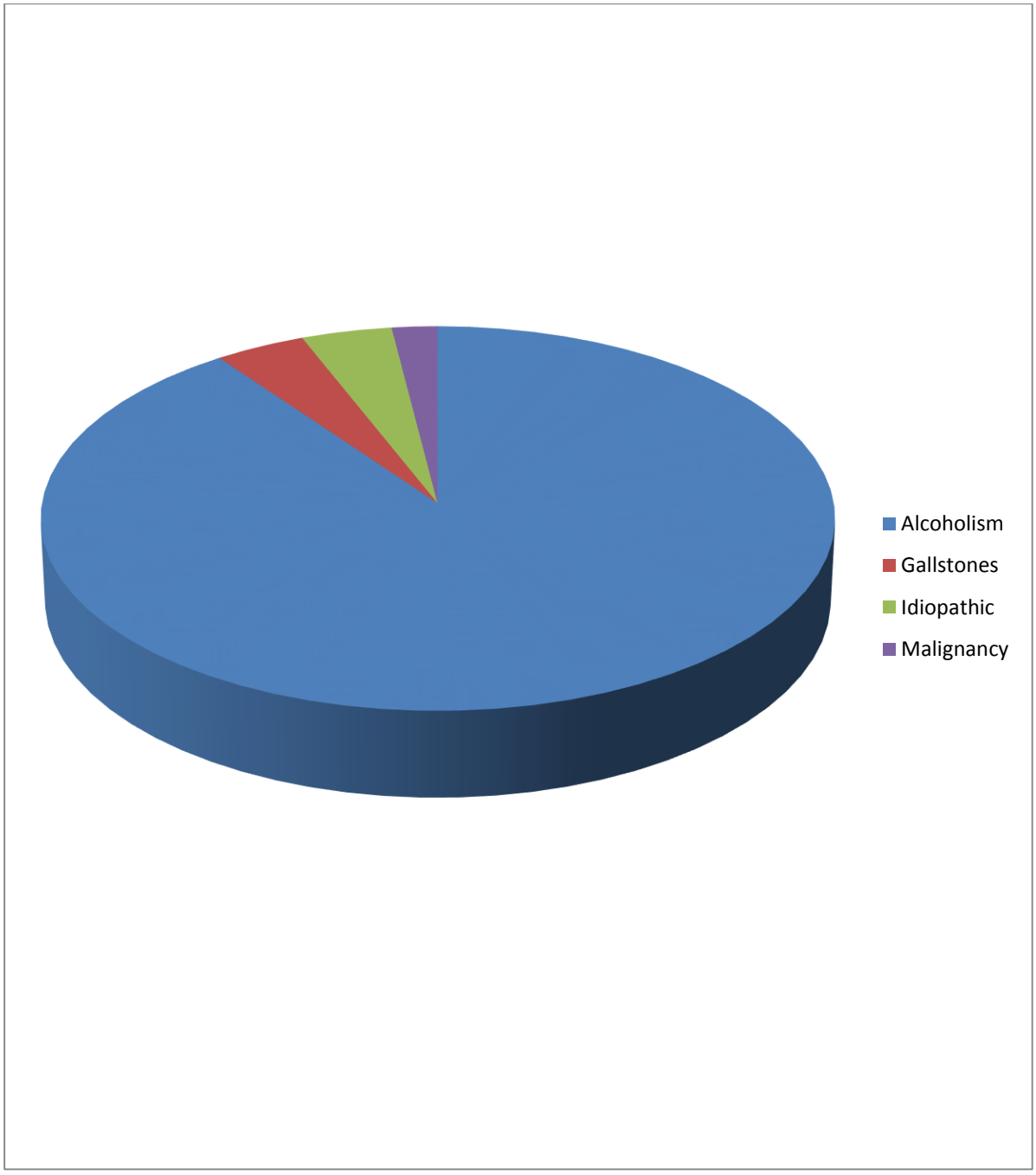
CONCLUSION

1. Serum amylase and lipase has only role in diagnosis of acute pancreatitis. so the rise of serum amylase and lipase level can not be used as a parameter to assess the severity of acute pancreatitis.
2. Ranson's scoring has important role in assessing the severity of acute pancreatitis.
3. CT scan plays a very important role in both diagnosis and assessment of severity of the disease.

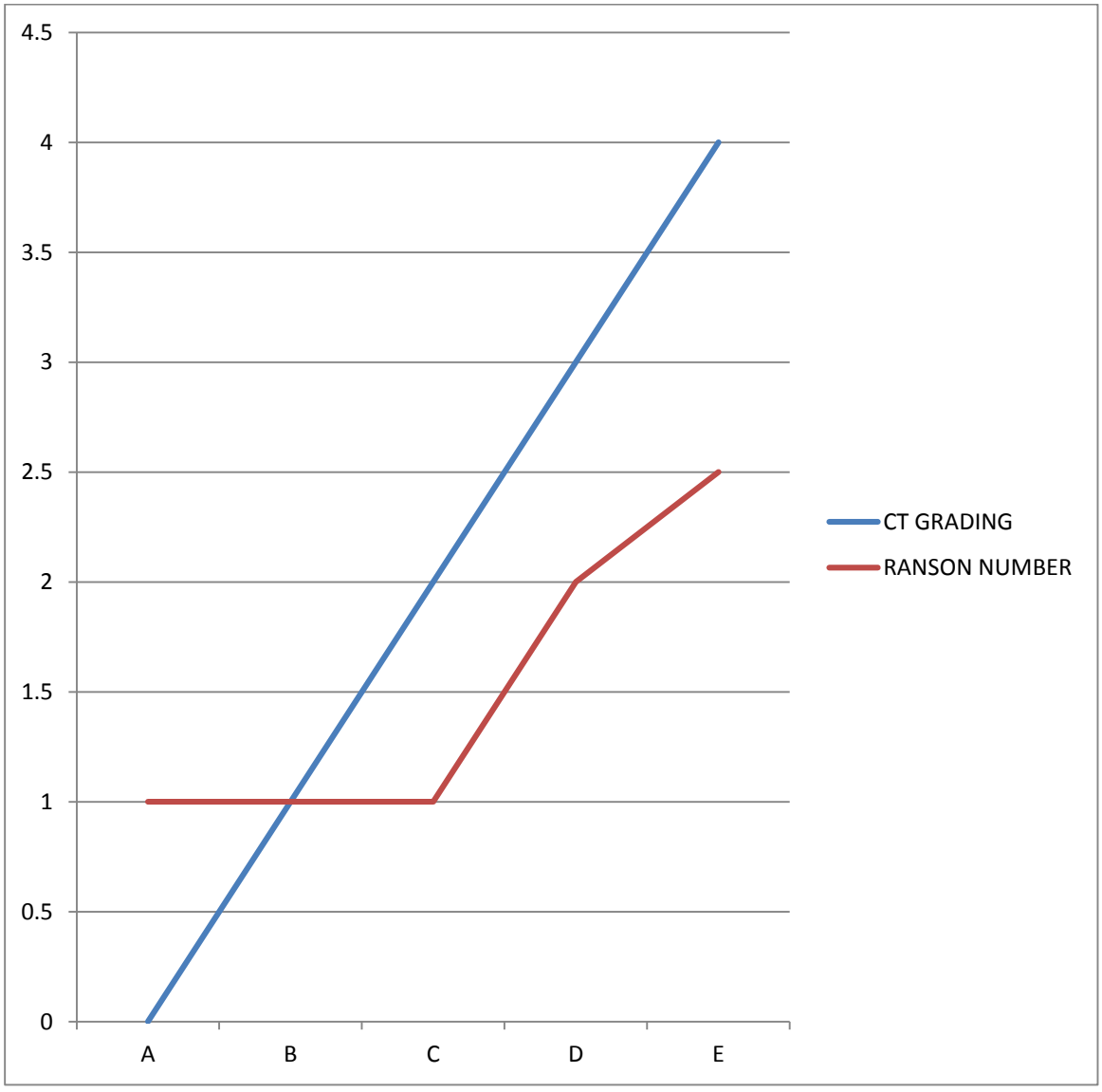
CLINICAL MANIFESTATIONS OF ACUTE PANCREATITIS



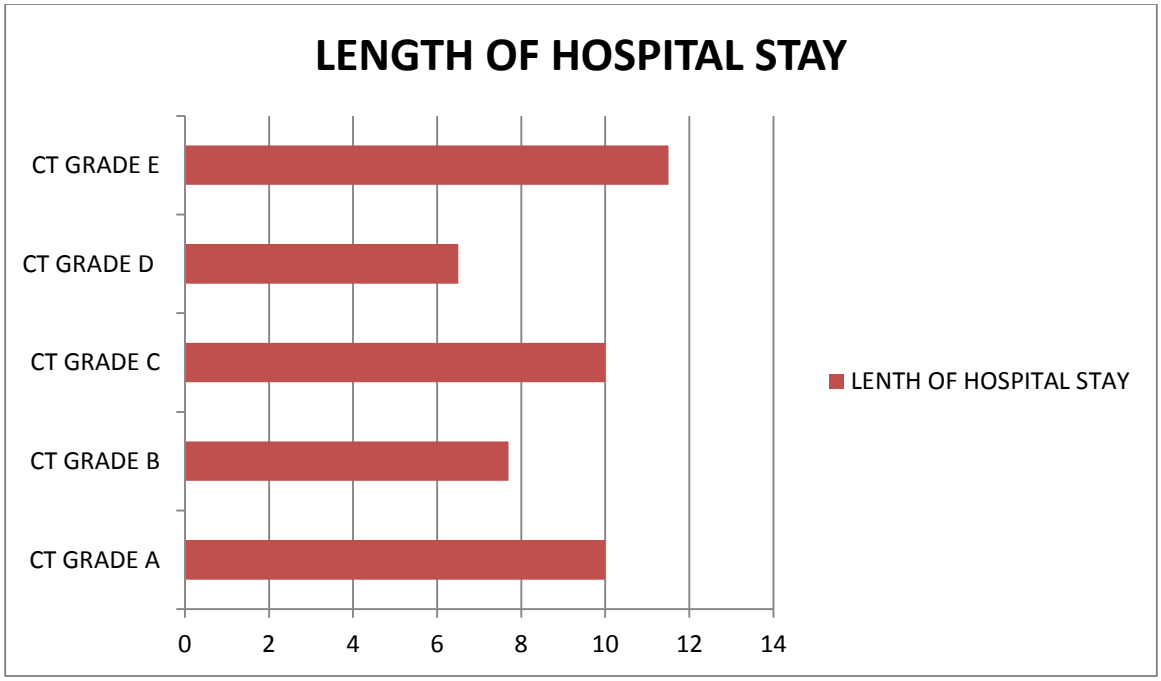
ETIOLOGY OF ACUTE PANCREATITIS



CORRELATION BETWEEN CT SCORING AND RANSON NUMBER



LENGTH OF HOSPITAL STAY



CASE PROFORMA

NAME:

AGE/SEX:

IP NUMBER/ UNIT:

DATE OF ADMISSION & DATE OF DISCHARGE/ DEATH:

COMPLAINTS & HISTORY:

GENERAL EXAMINATION:

VITAL SIGNS:

PR: RR: TEMPERATURE:

BP: SPO2:

SYSTEMIC EXAMINATION:

CVS: RS: PER ABDOMEN:

CNS: PER RECTAL:

INVESTIGATIONS:

SERUM AMYLASE: SERUM LIPASE:

RANSON CRITERIA:

AT ADMISSION:	CRITERIA	SCORE
	AGE	
	BLOOD GLUCOSE	
	WBC	
	LDH	
	SGOT	
48 HRS AFTER ADMISSION:		
	DECREASE IN HEMATOCRIT	
	INCREASE IN SERUM AMYLASE	
	SERUM CALCIUM	
	FLUID SEQUESTRATION	
	SPO2	
	BASE DEFICIT	

CT ABDOMEN:

UNENHANCED CT SCORE: GRADE SCORE

NECROSIS INDEX:

SEVERITY INDEX:

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41.karne S,Gorelick F, ETIOPATHOGENESIS OF ACUTE PANCREATITIS

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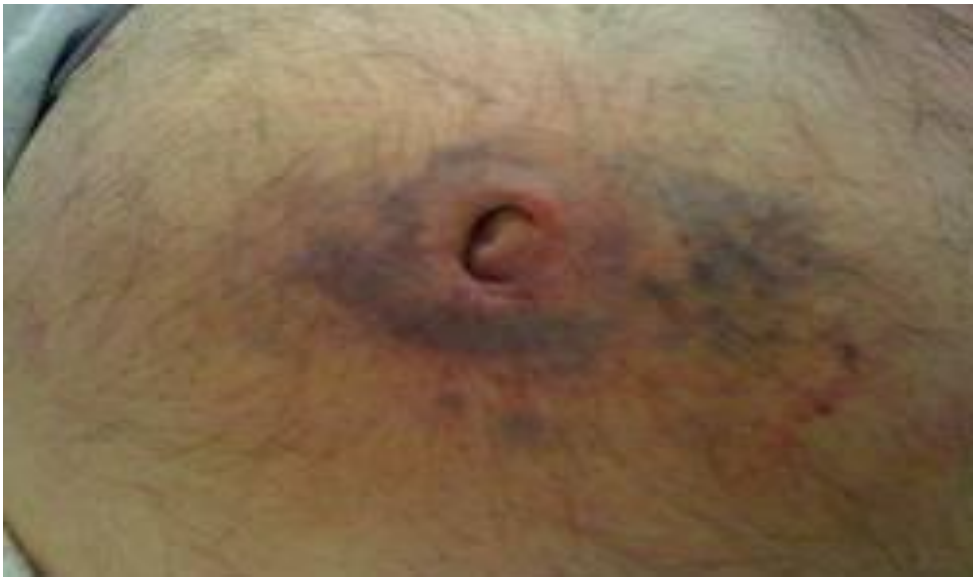
47.Comfort M, Steinberg A.Pedigree of a family with hereditary chronic

Relapsing pancreatitis

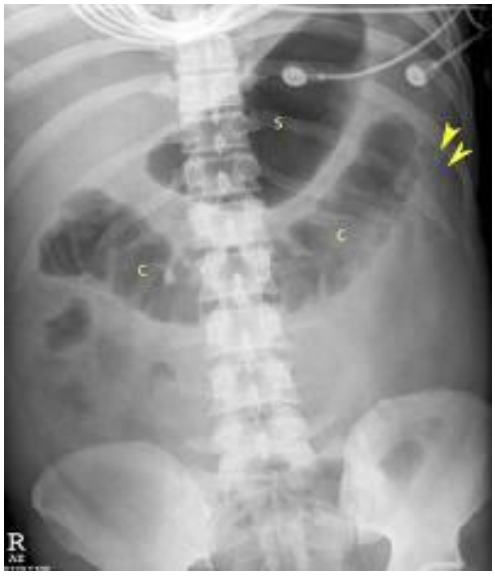
48.Baron T,Morgan D. Acute necrotizing pancreatitis

49.Balthazar E.

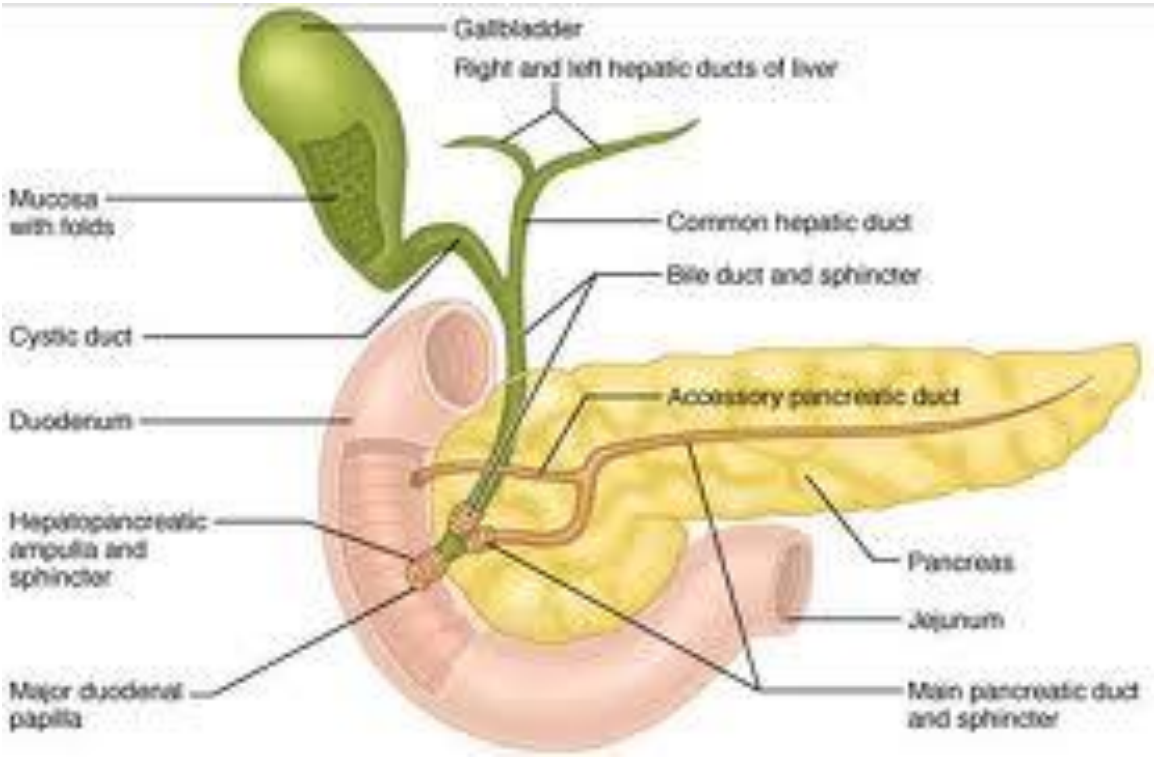
CULLEN'S SIGN



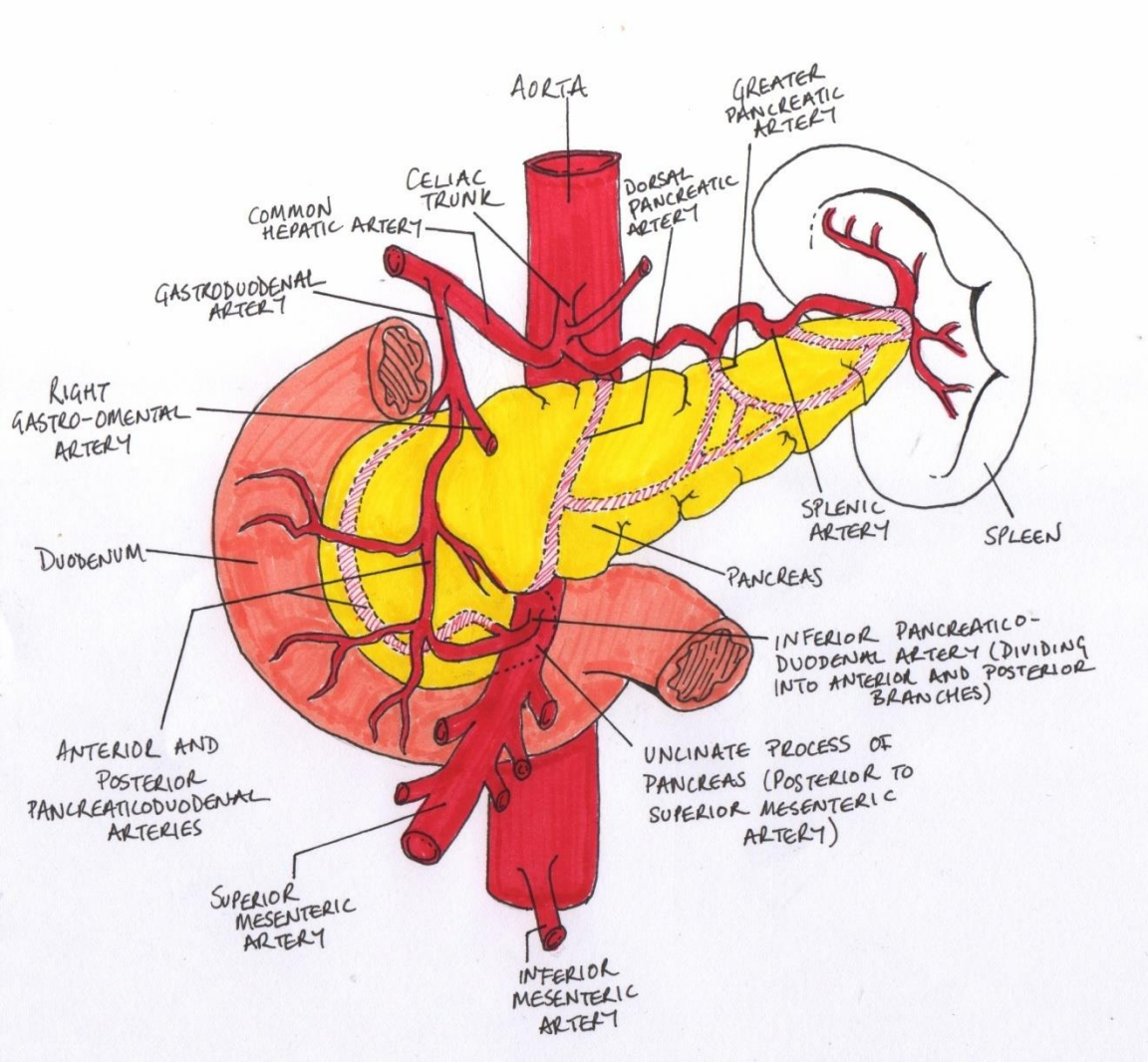
COLON CUT - OFF SIGN

h 

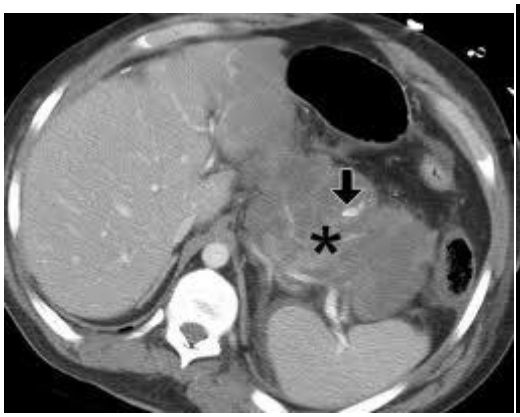
ANATOMY OF PANCREATIC DUCT.



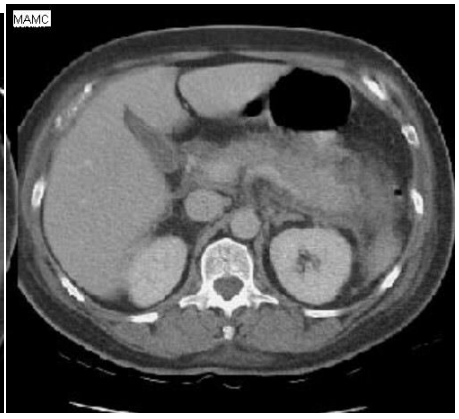
BLOOD SUPPLY



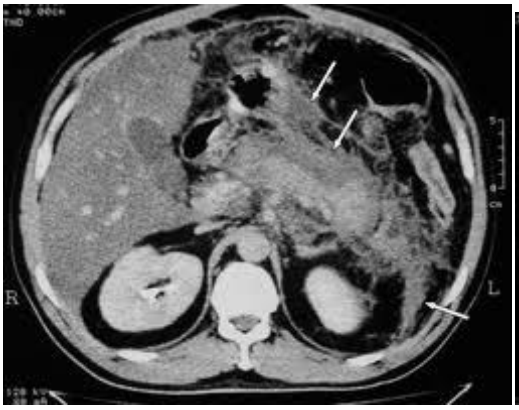
ACUTE HAEMORRHAGIC PANCREATITIS



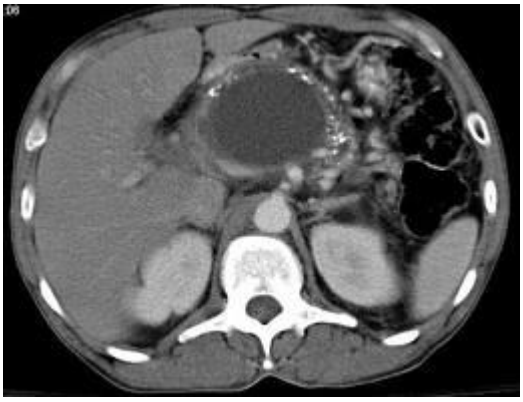
ACUTE OEDEMATOUS PANCREATITIS



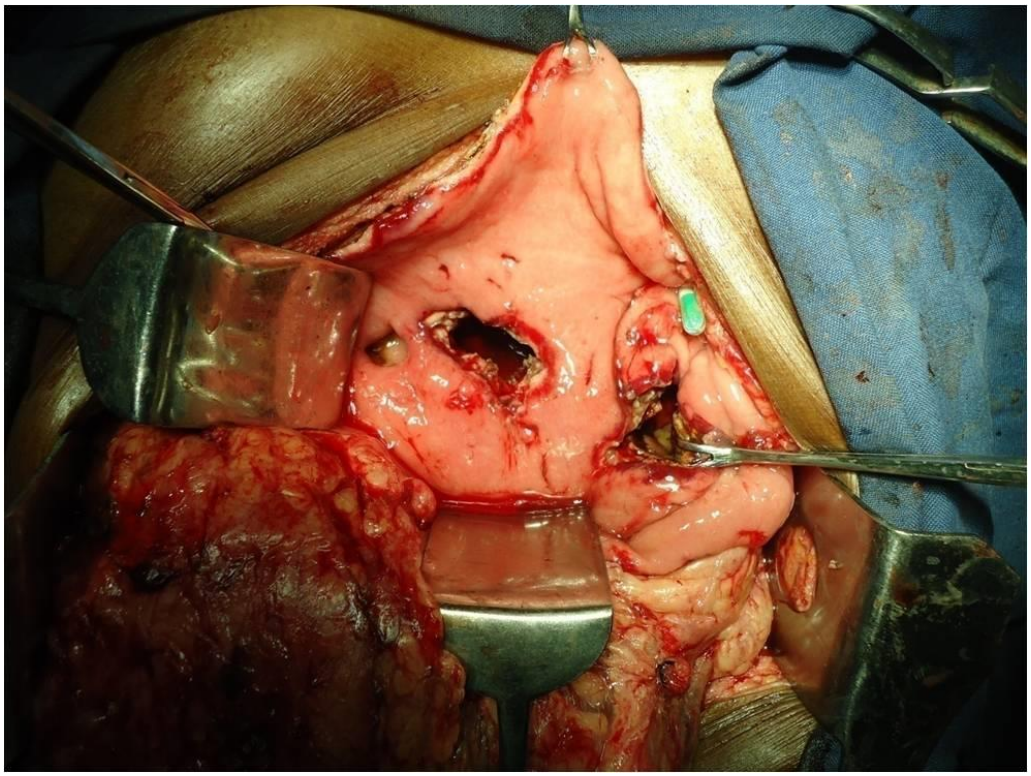
ACUTE NECROTISING PANCREATITIS



PSEUDOCYST OF PANCREAS



CYSTOGASTROSTOMY



S.no	Name	Age/Sex	IP Number	DOA	DODi/DODe	R.S	Amylase : lipase	CT Grade	Necrosis index	Severity index
1	Gurusamy	45/M	46713	2.2.15	24.2.15E	3	A-409/L-680	D(3)	2	5
2	Kottaisamy	40/M	68036	15.2.15	28.2.15D	1	A-789/L-990	B(1)	0	1
3	Murugan	58/M	68563	8.2.15	19.2.15D	1	A-3370/L-2940	B(1)	0	1
4	Pavunkani	24/F	70697	20.2.15	3.3.15D	2	A-1180/L-3210	B(1)	0	1
5	Vijiyan	36/M	25754	12.3.15	28.3.15D	3	A-488/L-622	D(3)	2	5
6	Thangamani	25/M	72042	5.3.15	21.3.15D	2	A-922/L-628	E(4)	2	6
7	Kumari	23/F	5798	11.3.15	24.3.15E	3	A-328/L-400	E(4)	2	6
8	Minnal	65/M	74175	15.3.15	28.3.15D	1	A-300/L-928	D(3)	0	3
9	Kottaisamy	56/M	74182	2.4.15	18.4.15D	0	A-228/L-498	B(1)	0	1
10	Arumugam	28/M	74144	3.4.15	15.4.15D	1	A-770/L-848	B(1)	0	1
11	Paramasivan	29/M	76516	3.4.15	17.4.15D	2	A-1048/L-728	B(1)	0	1
12	Tharmaraj	61/M	76751	21.4.15	12.5.15D	1	A-236/L-486	B(1)	0	1
13	Shanmugam	39/M	77133	27.4.15	13.5.15D	1	A-428/L-364	D(3)	2	5
14	Shankar	39/M	77191	11.5.15	24.5.15D	1	A-3210/L-2678	B(1)	0	1
15	Kanthaiyah	65/M	77550	29.5.15	12.6.15D	2	A-218/L-482	B(1)	0	1
16	Rathnamala	34/F	77609	30.5.15	9.6.15D	1	A-348/L-482	B(1)	0	1
17	Subramaniyan61	61/M	13249	9.6.15	19.6.15D	0	A-746/L-825	B(1)	0	1
18	Sankar	39/M	71179	22.6.15	29.6.15D	2	A-1313/L-348	B(1)	0	1
19	Nagammal	42/F	67290	15.7.15	28.7.15D	1	A-378/L-624	B(1)	0	1
20	Ranjith kumar	35/M	72276	18.8.15	27.8.15D	1	A-114/L-128	C(2)	0	2
21	Antony dhilip	22/M	16721	22.8.15	28.8.15D	1	A-348/L-1128	B(1)	0	1
22	Sivakumar	35/M	24253	23.8.15	28.8.15E	3	A-141/L-348	D(3)	0	6
23	Shankar	39/M	77191	25.8.15	12.9.15E	3	A-90/L-132	D(3)	0	5
24	Karuppan	44/M	73421	27.8.15	10.8.15D	1	A-748/L-928	B(1)	0	1
25	Kannan	43/M	77435	4.9..15	20.9.15D	1	A-340/L-920	B(1)	0	1
26	Marisingadurai	41/M	25308	11.9.15	23.9.15E	3	A-340/L-820	E(4)	0	5
27	Kaliyamoorthy	28/M	25663	15.9.15	30.9.15E	3	A-389/L-440	D(3)	0	6
28	Sujatha	35/F	43206	18.9.15	27.9.15E	3	A-287/L-390	D(3)	0	5
29	Pitchaiya	38/M	11264	30.9.15	3.10.15D	1	A-143/L-213	C(2)	0	1
30	Sreenivasan	55/M	21994	2.10.15	12.10.15D	1	A-388/L-564	D(3)	0	2

31	Paramasivan	36/M	18943	14.10.15	27.10.15D	2	A-489/L-211	C(2)	0	1
32	Munniyasamy	42/M	45323	21.10.15	29.10.15D	1	A-342/L-167	D(3)	0	2
33	Subramaniyan	39/M	34452	3.11.15	16.11.15D	2	A-155/L-545	C(2)	0	2
34	Isaacpandiyan	48?M	17725	29.11.15	15.12.15D	1	A-110/L-173	D(3)	0	1
35	Chandran	62/M	18920	13.12.15	24.12.15D	1	A-145/L-322	D(3)	0	2
36	Velmani	29/M	12987	19.12.15	29.12.15D	2	A-211/L-254	C(2)	0	2
37	Ramasubbu	43/M	19873	25.12.15	3.12.15D	2	A-190/L-233	D(3)	0	1
38	Viji	19/F	316	5.1.16	28.1.16D	1	A-748/L-1384	C(2)	0	2
39	Krishnan	38/M	966	12.1.16	19.1.16D	2	A-288/L-443	B(1)	0	1
40	Udayakumar	27/M	967	21.2.16	5.3.16D	1	A-1444/L-848	B(1)	0	1
41	Kannan	33/M	1403	27.2.16	11.3.16D	0	A-349/L-121	C(2)	0	2
42	Karuppasamy	39/M	1534	18.3.16	29.3.16D	0	A-110/L-173	B(1)	0	1
43	Kulomin nag	35/M	3078	19.3.16	28.3.16D	2	A-300/L-369	B(1)	0	1
44	Muthumanikam	53/M	5097	22.3.16	30.3.16D	1	A-140/L-103	B(1)	0	1
45	Mariyappan	45/M	10960	26.3.16	11.4.16D	1	A-410/L-784	B(1)	0	1
46	Sudalaikani	35/M	12038	27.3.16	12.4.16D	2	A-310/L-740	B(1)	0	1
47	Peratchi	53/M	13805	2.4.16	13.4.16D	2	A-770/L-884	E(4)	2	6
48	Muthukumar	29/M	14231	7.4.16	18.4.16D	3	A-410/L-588	B(1)	0	1
49	Kumar	35/M	17596	15.4.16	28.4.16D	0	A-220/L-200	C(2)	2	4
50	Siva	25/M	18815	22.4.16	31.4.16D	1	A-1141/L-1320	B(1)	0	1
51	Murgan	40/M	19554	28.4.16	5.5.16D	1	A-422/L-624	B(1)	0	1
52	Thiruvarankam	43/M	17283	4.5.16	13.5.16D	1	A-675/L-778	B(1)	0	1
53	Ashok kumar	36/M	17298	12.5.16	21.5.16D	1	A-400/L-324	B(1)	0	1
54	Muthumanikam	53/M	20193	23.5.16	29.5.16D	1	A-2148/L-3788	B(1)	0	1
55	Ganesan	33/M	21298	26.5.16	4.6.16D	1	A-399/L-874	A(0)	0	0
56	Muthukrishnan	55/M	22543	27.6.16	11.7.16D	1	A-548/L-740	B(1)	0	1
57	Rajavel	37/M	23419	1.7.16	16.7.16D	1	A-599/L-1102	E(4)	2	6
58	Sivaraman	41/M	12987	13.7.16	22.7.16E	2	A-448/L-384	B(1)	0	5
59	Manisamy	42/M	43851	17.8.16	30.8.16D	1	A-1200/L-948	D(3)	2	5
60	Kannan	32/M	13987	21.8.16	29.8.16D	1	A-348/L-625	B(1)	0	1