A DISSERTATION ON

"A COMPARATIVE STUDY ON THE EFFECTS OF EARLY ENTERAL FEEDING AND PARENTERAL FEEDING IN ACUTE PANCREATITIS"

Dissertation submitted to

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for the Award of the degree

M.S. [General Surgery]

Branch – I

DEPARTMENT OF GENERAL SURGERY,

STANLEY MEDICAL COLLEGE ,

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MAY-2018

CERTIFICATE

This is to certify that the dissertation entitled **"A COMPARATIVE STUDY ON THE EFFECTS OF EARLY ENTERAL FEEDING AND PARENTERAL FEEDING IN ACUTE PANCREATITIS"** is a bonafide original work of **Dr.EZHIL.P** , in partial fulfilment of the requirements for M.S.Branch–I (General Surgery) Examination of the Tamil Nadu Dr. M.G.R. Medical University to be held in APRIL 2018 under my guidance and supervision in 2017-18.

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DECLARATION

I, **Dr. M . EZHIL.P** solemnly declare that dissertation titled, **"A COMPARATIVE STUDY ON THE EFFECTS OF EARLY ENTERAL FEEDING AND PARENTERAL FEEDING IN ACUTE PANCREATITIS"** is a bonafide work done by me at Govt. Stanley Medical College & Hospital during 2015-2018 under the guidance and supervision of my Unit Chief. **Prof.DR.G.UTHIRA KUMAR.M.S**Professor of Surgery. The dissertation is submitted to Tamil Nadu Dr. M.G.R. Medical University, towards partial fulfilment of requirement for the award of **M.S. Degree (Branch – I) in General Surgery,** Examination to be held in April 2018.

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Principal Investigator : Dr. P Ezhil

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The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 25.10.2016 at the Council Hall, Stanley Medical College, Chennai-1 at 2PM

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Dr.EZHIL.P

INRODUCTION

The treatment of acute pancreatitis is purely symptomatic because there is no effective therapy to prevent the activation of inflammatory and proteolytic cascades. This vicious cycle of cell signalling is believed to be triggered by bacterial infection, predominately Gram-negative strains. The most likely hypothetical source of the bacterial infection is the gastrointestinal tract. Bacterial translocation is caused by increased permeability in the gut and a consequent migration of macromolecules such as bacteria,endotoxins and antigens from the gastrointestinal tract to the portal system, mesenteric lymph nodes, liver, spleen and pancreas. This process leads to the stimulation of macrophages, circulatory neutrophils and granulocytes, and then the release of pro-inflammatory cytokine causes an inflammatory response. If the inflammatory response, which is initially part of the defence mechanisms of the host, is over-activated, it may turn into a self-destructive process. The unbalanced production of inflammatory mediators might lead to the development of systemic inflammatory response syndrome (SIRS), infectious pancreatic necrosis and ultimately multi-organ failure (MOF)[1]

Severe acute pancreatitis (SAP) represents a typical model of septic syndrome due to a failure of the gut barrier .Hence, one of the main therapeutic goals in AP is to maintain gut integrity to prevent bacterial and endotoxin translocation

AIMS AND OBJECTIVES

a) To assess the occurrence of infective & non infective complications in both types of feeding
b) To assess the average duration of hospital stay in both groups
c) To compare the effects of early enteral feeding with parenteral feeding in acute pancreatitis

PLACE OF STUDY

Department of general surgery

Stanley medical college

Chennai

Duration

DURATION

1 year

INCLUSION CRITERIA

1. Age > 18 years and < 70 years
2. Mild and moderate in severity

EXCLUSION CRITERIA

1. Age < 18 years and >70 years
2. Severe acute pancreatitis
3. signs of shock at the time of presentation

ETHICAL COMMITTIE APPROVAL

Obtained

POPULATION

Cases - 30

Controls - 30

METHODOLOGY

1. all the patients who are admitted in the ward with upper abdominal pain are evaluated
2. the diagnosis of pancreatitis will be made clinically,biochemically & radiologically
3. severity will be assessed by BISAP scoring system & CT SEVERITY INDEX
4. early enteral feeding will be started in cases within 24 to 48 hours of admission
5. patients who will be on nil per mouth and on parenteral feeding is considered as controls
6. incidence of complications & course of hospital stay will be observed in both cases & controls
7. datas will be collected & analysed

REVIEW OF LITERATURE

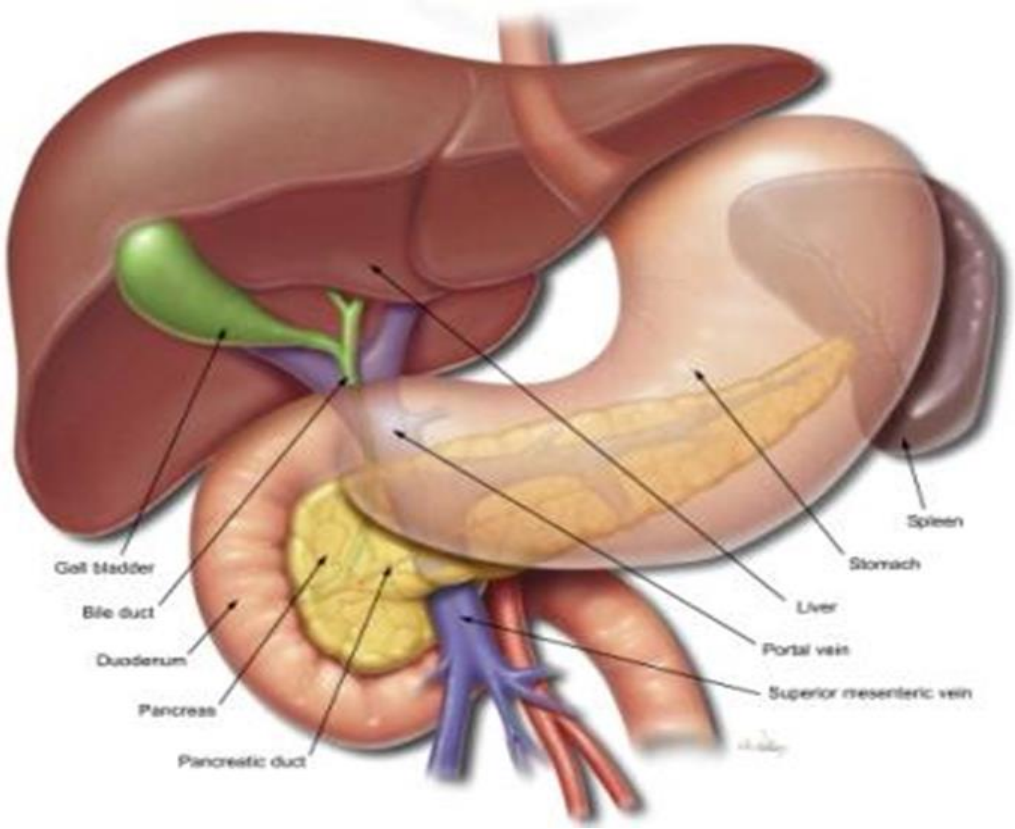
PANCREAS

The pancreas is an important organ situated in the deep centre of the abdomen.it is called as unforgiveful organ because the minor manipulation or minor trauma can leads to the morbitidy.

GROSS ANATOMY

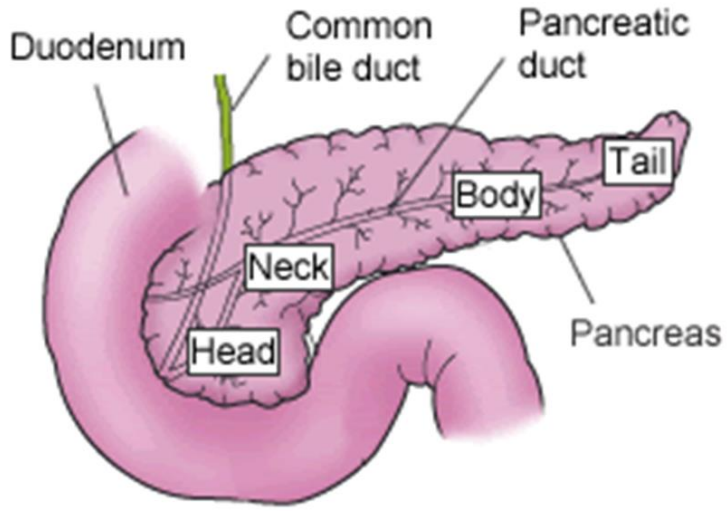
The pancreas is a composite gland having exocrine acini which discharge their secretions into the duodenum to assist in digestion, and groups of endocrine cells, the islets of Langerhans, whose role is in carbohydrate metabolism. In shape the gland resembles the upper end of a thick walking-stick or hook, lying sideways with the handle or hook on the right and turned downwards . Its length is about 15 cm. The gland is of firm consistency, and its surface is finely lobulated. Its big head on the right is connected by a short neck to the body, which crosses the midline and tapers to a narrow tail on the left. The head and tail incline towards the paravertebral gutters, while the neck and body are curved boldly forward over the inferior vena cava and aorta in front of the first lumbar vertebra. The gland lies somewhat obliquely, sloping from the head upwards towards the tail behind the peritoneum of the posterior abdominal wall. The transpyloric plane (L1) is the guide to the surface marking; the neck lies on the plane, which passes across the head and body, and below the tail.

LOCATION OF PANCREAS



- ✓ Pancreas is an elongated, accessory digestive gland that lies retroperitoneally
- ✓ Transversely across the posterior abdominal wall posterior to the stomach between duodenum on the right and the spleen on the left

PARTS OF PANCREAS



NECK

T he neck is best defined as the narrow band of pancreatic tissue that lies in front of thecommencement of the portal vein, continuous to the right with the head and to the left with the body

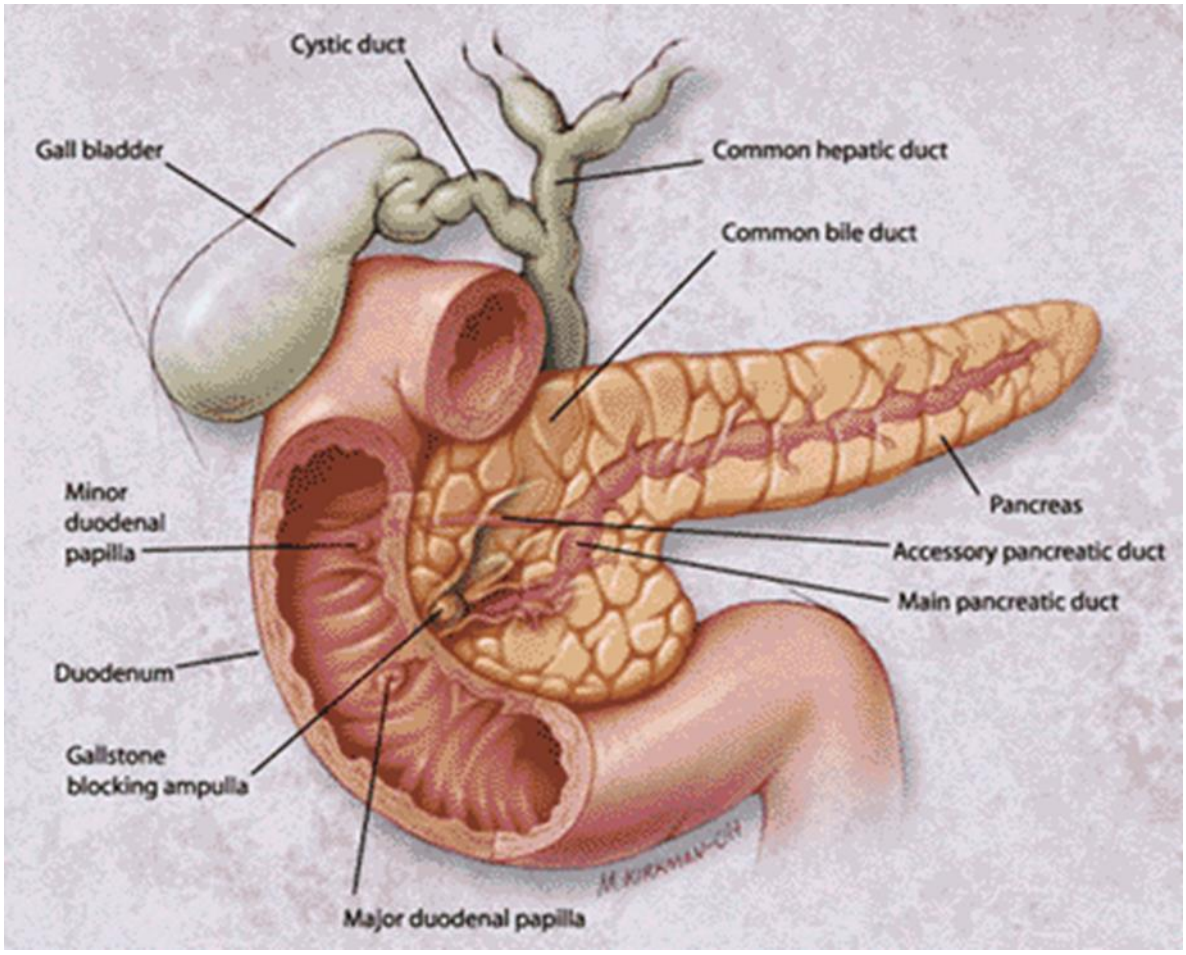
HEAD

The head, the broadest part of the pancreas, is moulded to the C-shaped concavity of the duodenum, which it completely fills. It lies over the inferior vena cava and the right and left renal veins, mainly at the level of L2 vertebra

BODY

The body of the pancreas passes from the neck to the left, sloping upwards across the left renal veinand aorta, left crus of the diaphragm, left psoas muscle and lower part of left suprarenal gland, to the hilum of the left kidney.

THE PANCREATIC DUCTS



The pancreatic duct (of Wirsung) is a continuous tube running from the tail to the head, gradually increasing in diameter as it receives tributaries. At the hepatopancreatic ampulla . it is joined at an angle of about 60° by the bile duct

and the manner of their joint opening into the duodenum . In intubation of the ampulla for endoscopic retrograde cholangiopancreatography (ERCP), the catheter preferentially enters the pancreatic duct. [2]

NEUROANATOMY

Parasympathetic vagal fibres, which are capable of stimulating exocrine secretion, reach the glandmainly from the posterior vagal trunk and coeliac plexus, but, as with the gallbladder, hormonal control is more important than the neural. Sympathetic vasoconstrictor impulses are derived from spinal cord segments T6–10 via splanchnic nerves and the coeliac plexus, the postganglionic fibres running to the gland with its blood vessels. As with other viscera, pain fibres accompany the sympathetic supply, so that pancreatic pain may radiate in the distribution of thoracic dermatomes 6–10

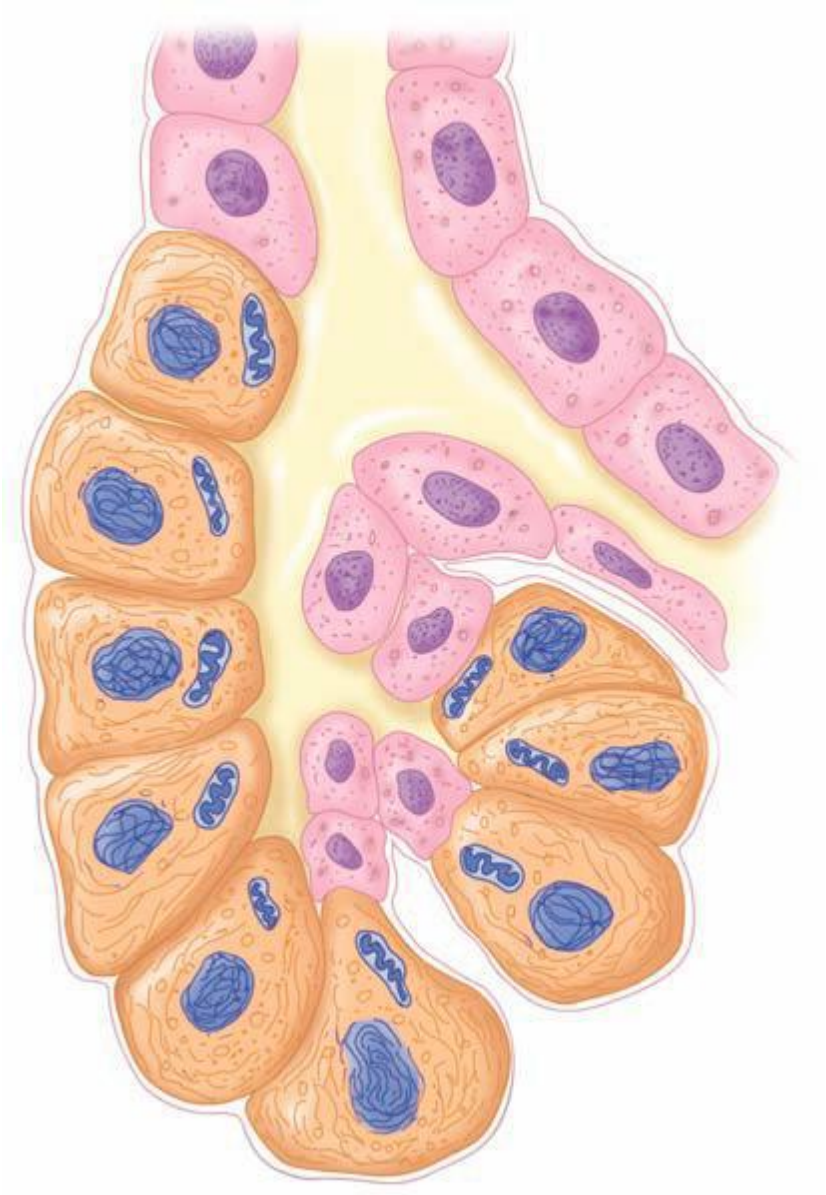
HISTOLOGY & PHSIOLOGY

In response to a meal, the pancreas secretes digestive enzymes in an alkaline (pH 8.4) bicarbonate-rich fluid. Spontaneous secretion is minimal; the hormone secretin, which is released from the duodenal mucosa, evokes a bicarbonate-rich fluid. Cholecystokinin (CCK) (synonym: pancreozymin) is released from the duodenal mucosa in response to food. CCK is responsible for enzyme release. Vagal stimulation increases the volume of secretion. Protein is synthesised at a greater rate (per gram of tissue) in the pancreas than in any other tissue, with the possible exception of the lactating mammary gland. About 90 per cent of this

protein is exported from the acinar cells as a variety of digestive enzymes. Approximately 6–20 g of digestive enzymes enter the duodenum each day. Nascent proteins are synthesised as preproteins and undergo modification in a sequence of steps. The proteins move from the rough endothelial endoplasmic reticulum to the Golgi complex, where lysosomes and mature zymogen storage granules containing proteases are stored, and then to the ductal surface of the cell, from which they are extruded by exocytosis. During this phase, the proteolytic enzymes are in an inactive form, the maintenance of which is important in preventing pancreatitis.[3]

EXOCRINE PANCREAS

The pancreas secreting 500 to 800 mL per day of colorless, odorless, alkaline, isosmotic pancreatic juice. Pancreatic juice is the combination of acinar cell and duct cell secretions. The acinar cells secrete amylase,proteases, and lipases, enzymes and which are responsible for the digestion of all three food types carbohydrate, protein, and fat. The acinar cells are pyramid shaped, and their apices facing the lumen of acinus. Near the apex of the each cell are numerous enzyme-containing zymogen granules that fuse with apical cell membrane . Unlike the endocrine pancreas, inhich islet cells specialize in the secretion of one hormone type, individual acinar cells are secreting all types of enzymes. However, the ratio of different enzymes which is adjusted to the composition of digested food through nonparallel regulation of the secretion



STRUCTURE OF THE ACINAR CELL

The amylase from pancreas is secreted in its active form and completes the digestive process already begun by thesalivary amylase. Amylase is only pancreatic enzyme secreted in its active form, and it hydrolyzes starch and the glycogen to glucose, maltose, maltotriose, and dextrins. These simple sugars are transported across brush border of the intestinal epithelial cells by active transport mechanisms. Gastric hydrolysis of protein yields peptides that enter t intestine and stimulate intestinal endocrine cells to release cholecystokinin (CCK)-releasing peptide, CCK, and secretin, and that stimulate the pancreas to secrete enzymes and bicarbonate into the intestine.

The proteolytic enzymes are secreted as proenzymes that require activation. Trypsinogen is converted to its active form, trypsin, by enterokinase, which is produced by the duodenal mucosal cells. Trypsin, activates the other proteolytic enzymes. Trypsinogen activation within the pancreas is prevented by the presence of trypsinogen activation inhibitors that are also secreted by the acinar cells. A failure to express a normal trypsinogen activation inhibitor, pancreatic secretory trypsin inhibitor (PSTI) is also known as serine protease inhibitor Kazal type 1 , is a cause of familial pancreatitis. Inhibition of trypsinogen activation ensures that the enzymes within the pancreas remain in the inactive precursor state and are activated only after entering into the duodenum. Hereditary pancreatitis occurs because of the the missense mutation of cationic trpsinogen.[4]

Chymotrypsinogen is activated to form thechymotrypsin .Elastase, carboxypeptidase A and B, and phospholipase are also activated by the trypsin. Trypsin, chymotrypsin, and elastase are cleaving bonds between amino acids within the target peptide chain, and carboxypeptidase A and B cleave amino acids at the end of the peptide chains. Individual amino acids and small dipeptides are then actively transported into the intestinal epithelial cells. Pancreatic lipase hydrolyzes the triglycerides to 2-monoglyceride and fatty acid. Pancreatic lipase is secreted in an active form. Colipase is also secreted by pancreas and binds to lipase, changing its molecular configuration and increasing its activity.

Pancreas secretes phospholipase A2 a proenzyme that becomes activated by trypsin. Phospholipase A2 hydrolyzes the phospholipids and, as with all lipases, requires bile salts for its action. Carboxylic ester hydrolase and the cholesterol esterase hydrolyze the neutral lipid substrates like esters of cholesterol,

fat-soluble vitamins, a triglycerides. The hydrolyzed fat is then packaged into micelles for transport into intestinal epithelial cells, where the fatty acids are reassembled and packaged inside chylomicrons for the transport through the lymphatic system into the bloodstream. The centroacinar and intercalated duct cells are secreting the water and electrolytes present in the pancreatic juice.

About 40 acinar cells were arranged into a spherical unit called an acinus. Centroacinar cells were located near the center of the acinus and were responsible for fluid and electrolyte secretion. These cells contain the enzyme carbonic anhydrase, which is needed for the bicarbonate secretion. The amount of bicarbonate secreted varies with the pancreatic secretory rate, with greater concentrations of the bicarbonate was secreted as the pancreatic secretory rate increases. Chloride secretion varies inversely with the bicarbonate secretion such that the sum of these two remains constant. In contrast, sodium and potassium concentrations are kept constant throughout the spectrum of the secretory rates . The duodenal mucosal cells are releasing secretin in response to acidic chyme passing through the pylorus into the duodenum. Secretin is the major stimulant for the bicarbonate secretion, which buffers the acidic fluid entering the duodenum from the stomach. CCK stimulated bicarbonate secretion, but to a much lesser extent than secretin. CCK is potentiating secretin-stimulated bicarbonate secretion. Gastrin and acetylcholine, both are the stimulants of gastric acid secretion, are also weak stimulants of the pancreatic bicarbonate secretion. Truncal vagotomy is producing a myriad of complex effects on the downstream digestive tract, but the sum effect on the exocrine pancreas is the reduction in bicarbonate and fluid secretion. The endocrine pancreas is influencing the adjacent exocrine pancreatic secretions. Somatostatin, pancreatic polypeptide (PP), and glucagon are all thought to inhibit exocrine secretion. The acinar cells release pancreatic enzymes from

theirzymogen granules into the lumen of the acinus, and these proteins combine with the water and bicarbonate secretions of the centroacinar cells. The pancreatic juice then travels into the small intercalated ducts. Several small intercalated ducts join to form the interlobular duct. Cells in the interlobular ducts contribute fluid and electrolytes to adjust the final concentrations of the pancreatic fluid. Interlobular ducts are joining to form about 20 secondary ducts that empty into the main pancreatic duct. Destruction of the branching ductal tree from recurrent inflammation, scarring, and deposition of stones eventually contributing to the destruction of the exocrine pancreas and the exocrine pancreatic insufficiency.[5]

ACUTE PANCREATITIS

The definition of acute pancreatitis is “acute condition presenting with abdominal pain and is usually associated with the raised pancreatic enzyme levels in the blood or urine as a result of pancreatic inflammation”. Acute pancreatitis may recur.

The mechanism of acute pancreatitis is thought to be premature activation of pancreatic enzymes within the pancreas, leading to a process of autodigestion. Anything that injures the acinar cell and impairs the secretion of zymogen granules, or damages the duct epithelium and thus delays enzymatic

secretion, can trigger acute pancreatitis. Once cellular injury has been initiated, the inflammatory process can lead to pancreatic oedema, haemorrhage and, eventually, necrosis[6]

Etiologies of acute pancreatitis

A. Alcohol
B. Biliary tract disease
C. Hyperlipidemia
D. Hereditary
E. Hypercalcemia
F. Trauma External

Surgical

Endoscopic retrograde cholangiopancreatography

Hypoperfusion

Atheroembolic

G. Vasculitis

H. Pancreatic duct obstruction

I. Neoplasms

J. Pancreas divisum

K. Ampullary and duodenal lesions

L. Infections

M. Venom

N. Drugs

O. Idiopathic

GALL STONES

There are several school of thoughts

1.The mechanism by which the small gallstones are passing down the common bile duct and past the pancreatic duct junction and into the duodenum, causing acute pancreatitis

2. Another school of thought is that transient incompetence occurs by the passage of a stone through the sphincter may allow the back flow of duodenal

fluid and bile into the pancreatic duct, but this is refuting by the usual absence of acute pancreatitis after endoscopic sphincterotomy or surgicalsphinteroplasty.

3.A third school of thought is that acute pancreatitis is due to a gallstone obstructing the pancreatic duct that leading to ductal hypertension. It is being postulated that this backpressure might lead to the minor ductal disruption, extravasation of pancreatic juice into the less alkaline interstitium of the the pancreas, and promotion of enzyme activation.

ALCOHOL

There are several mechanisms by which ethanol causes acute pancreatitis.

1.Ethanol is a toxin to pancreatic acinar cells and it causes a brief secretory increase followed by inhibition. The secretory increase coupled with spasm of the sphincter of Oddi is causing incite acute pancreatitis.

2.Ethanol is inducing ductal permeability, that allow premature activation of enzymes that leads to the pancreatic damage

3.Ethanol is increasing the protein content of pancreatic juice, decreasing the bicarbonate levels, and the trypsin inhibitor concentration. Ethonal induces The formation of protein plugs may also lead an obstructive element to pancreatic outflow.

IATROGENIC

1.Many procedures can lead to acute pancreatitis, CBD exploration and exploration ampulla of Vater, distal gastrectomy, splenectomy, colectomy, nephrectomy, aortic aneurysmorraphy, and retroperitoneal lymphadenectomy.

2. cardio-pulmonary bypass or cardiac transplant can lead to ischaemic pancreatitis due to hypoperfusion

3.Aute pancreatitis may occur after ERCP. The risk of post-ERCP acute pancreatitis is increased if the contrast agent is infused repeatedly under high pressure and the patients with sphincter of Oddi dysfunction

HEREDITARY PANCREATITIS

Hereditary pancreatitis (HP) is an inflammation of the pancreas, attributed to genetic causes. It was first described in 1952 by Comfort and Steinbergbut it was not until 1996 that Whitcomb et alisolated the first responsible mutation in the trypsinogen gene (PRSS1) on the long arm of chromosome seven (7q35).these mutations are rarely identified in general screens of patients with idiopathic disease and the phenotype of p.R122H and p.N29I is now well characterised with the p.A16V mutation recently characterised for the first time. There are many other rare mutations or polymorphisms of PRSS1 which remain less well understoodand not all HP families have had the responsible genetic mutation identified.

TUMOURS

Pancreatic tumours may produce acute pancreatitis . acute pancreatitis can be the first clinical manifestation of the tumor. If the etiology can not be identified in acute pancreatitis ,cross sectional imaging should be done to rule out pancreatic tumours

HYPERLIPIDEMIA

Patients with types I and V hyperlipoproteinemia are frequently developing acute pancreatitis and often it is occurring in association with the marked hypertriglyceridaemia. Toxic fatty acids have been released into the circulation because of lipases leads to the impairement in the microcirculation can cause acute pancreatitis

OTHERS

1. several drugs are causing acutepancreatitis. These include the thiazide diuretics, furosemide,estrogens, azathioprine, l-asparaginase, 6-mercaptopurine, methyldopa, the sulfonamides, tetracycline, pentamidine, procainamide,

nitrofurantoin, dideoxyinosine, valproic acid, andacetylcholinesterase inhibitors. lipid-based intravenous drugs and solutions, such as propofol, can also cause acute pancreatitis.

2.hyperparathyroidism causes hypersecretion and the formation of calcified stones in the duct and parenchyma of the pancreas can produce both the acute & chronic pancreatitis

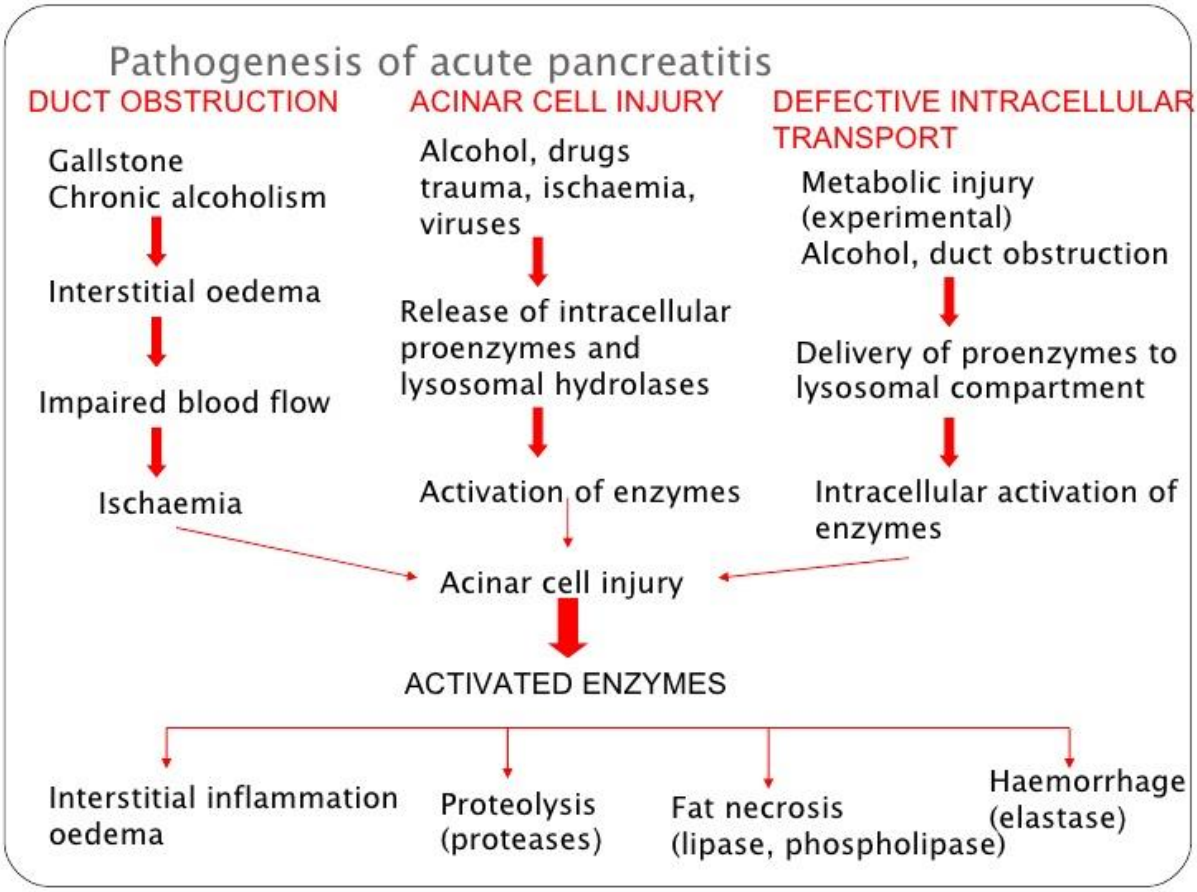
3. Ascaris lumbricoides and the liver fluke Clonorchis sinensis infestations are causing Oriental cholangitis, which is associated with cholangiocarcinoma which leads to the obstruction the pancreatic duct.

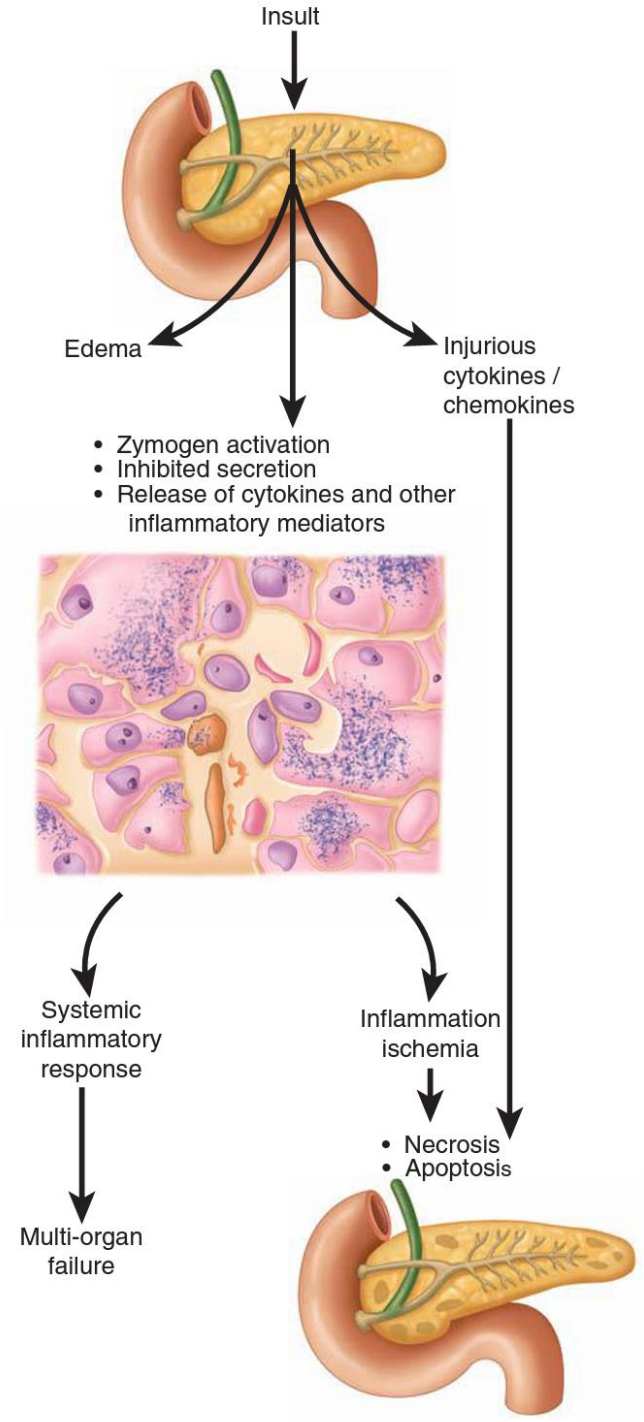
4.azotemia, vasculitis, and the sting of the Trinidadian scorpion Tityus trinitatis. massive production of pancreatic juice occurs because of the This scorpion's venom that causesneurotransmitter discharge from cholinergic nerve terminals, leading to pancreatitis. Antiacetylcholinesterase poisoning also causing the same effect laeding to the acute pancreatitis.if no cause has been identified in the course of evaluation it will be called as idiopathic pancreatitis.

PATHOPHSIOLOGY

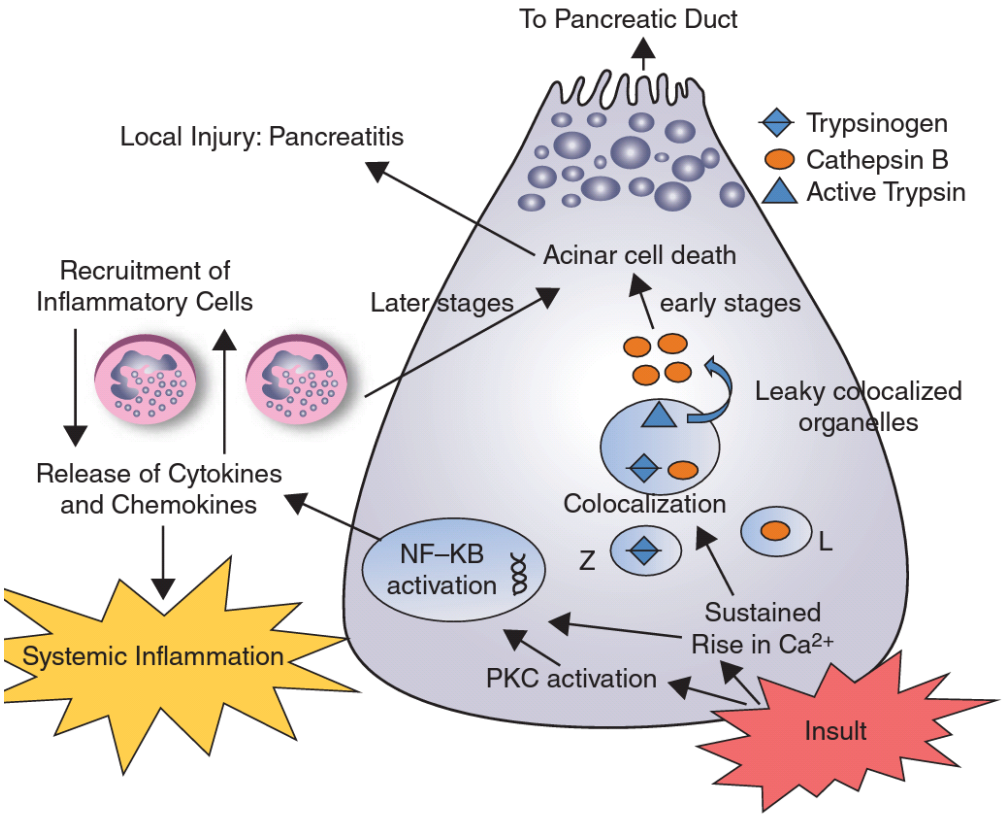
Pancreatitis begins with the activation of digestive zymogens inside acinar cells, which cause acinar cell injury.severity of pancreatitis may be determined by the Inflammatory cell recruitment and activation, as well as generation and release of cytokines and other chemical mediators of inflammation

Precipitating Initial Event





PATHOPHYSIOLOGY OF ACUTE PANCREATITIS



ACINAR CELL EVENTS

SYSTEMIC EVENTS

Acute pancreatitis (AP) is an inflammatory condition of the pancreas that involves peripancreatic tissue and remote organs. Excessive systemic inflammatory response syndrome (SIRS) in AP leads to distant organ damage and multiple organ dysfunction syndrome (MODS), which is the primary cause of morbidity and mortality in this condition. Mild AP is self limiting but up to 25% of the patients suffer a severe attack and around 30% of these will die. [9]

Approximately half of the deaths in AP occur within the first 2 wk of illness and are generally attributed to organ failure. The rest of the deaths occur weeks to months later, characterized by extensive retroperitoneal pancreatic necrosis and septicemia. AP involves a complex cascade of events initializing in pancreatic acinar cells. An unknown trigger within the pancreas leads to conversion of digestive proenzymes into their active form, initiating auto digestion of the gland causing hemorrhage, necrosis, edema and complete destruction of pancreatic parenchyma. Intrapancreatic activation of trypsinogen by lysosomal hydrolases is an early triggering event in AP.

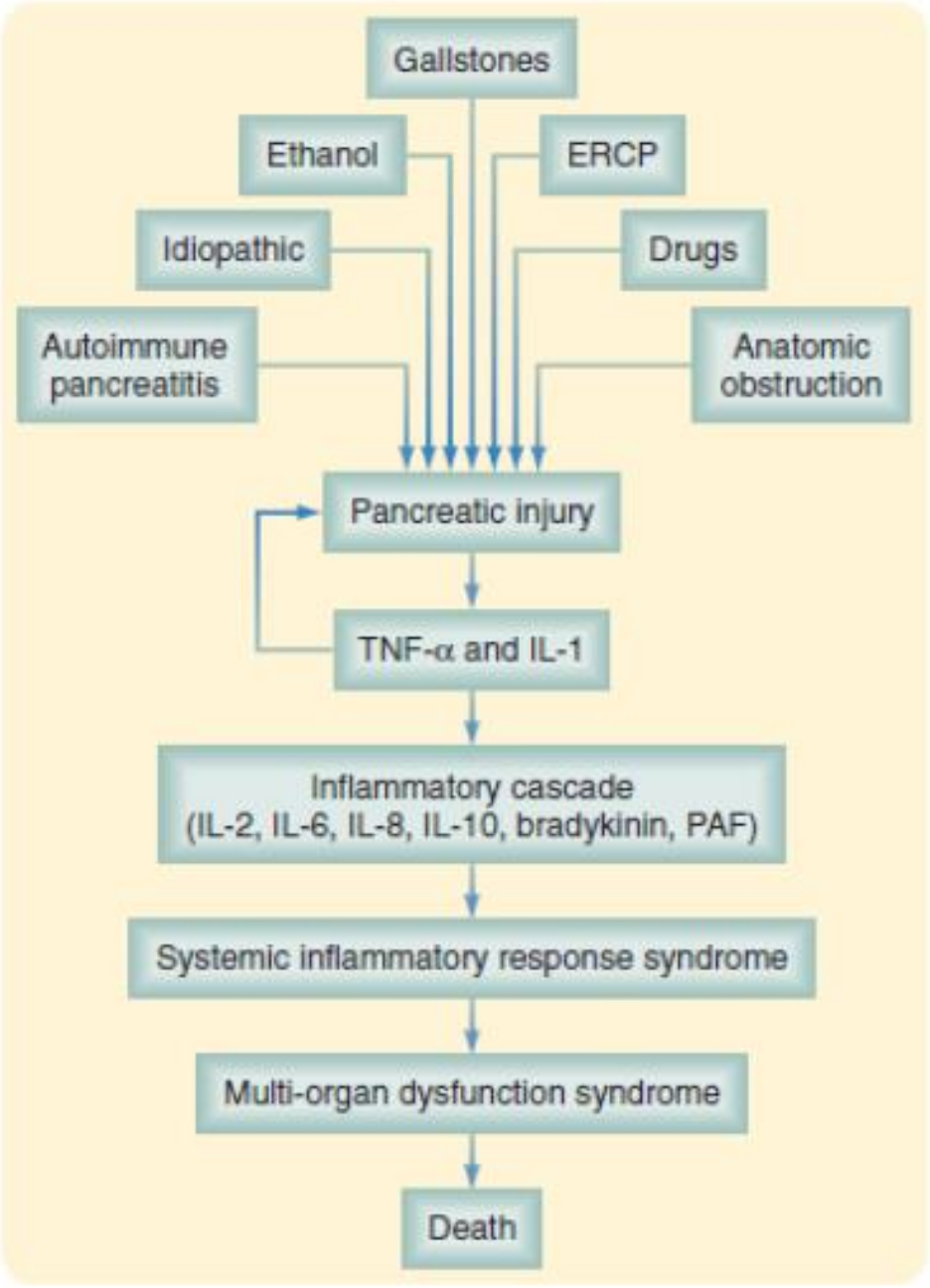
Interestingly both pharmacological and genetic deletion of lysosomal hydrolases like cathepsin B can reduce the severity of pancreatitis[7]. Other pharmacological agents which block trypsinogen activation can also modulate the outcome of AP.

Immune cells involved in elaborating the inflammatory mediators in AP are the pancreatic acinar cells, endothelial cells, neutrophils, lymphocytes, monocytes and macrophages. Inflammatory mediators believed to participate in the pathophysiology of this condition include: tumor necrosis factor-α (TNF-α), interleukin-1 (IL-1β), interleukin-6 (IL-6), platelet activating factor (PAF), ICAM-1, IL-8, growth related oncogene-a/cytokine-induced neutrophils chemo attractant (GRO-α/CINC), monocyte chemotactic protein-1 (MCP-1), IL-10, complement component C5a, substance P (SP), hydrogen sulfide (H2S), and neutral endopeptidase (NEP)[10].

In recent years, it has become clear that the signaling molecule nuclear factor κB (NF-κB) plays a central role in the initiation and progression of AP[11]. The emerging body of evidence suggest that blocking NF-κB activation can markedly reduce the severity of AP[12,13]. These findings have opened a window of opportunity for the use of selective NF-κB inhibitors in regulating the inflammatory process in AP. [11]

The expression levels of various proinflammatory mediators like TNF-α and IL-1β in AP are positively regulated by NF-κB[14,15]. Systemic amplification of AP is associated with excessive release of these inflammatory mediators from local tissue and systemically.

This systemic amplification is responsible for most of the mortality associated with AP[16]. Studies indicate that both pancreatic and extra pancreatic (lung, liver, monocytes, macrophages and endothelial cells) activation of NF-κB is associated with development of MODS in AP[17,18].



SYSTEMIC EVENTS IN ACUTE PANCREATITIS

DIAGNOSIS

CRITERIAS

- ✓ acute onset of a severe constant epigastric pain which often radiates through to the mid back)
- ✓ elevation of serum amylase or lipase (>3 times upper limit of normal).
- ✓ Imaging (usually by contrast enhanced CT scanning) is only required for the diagnosis of acute pancreatitis when these diagnostic criteria are met

The serum amylase concentration increases almost immediately with the onset of disease and peaks within several hours. It remains elevated for 3 to 5 days before returning to normal. There is no significant correlation between the magnitude of serum amylase elevation and severity of pancreatitis; in fact, a milder form of acute pancreatitis is often associated with higher levels of serum amylase compared with that in a more severe form of the disease.

severe form of the disease.

Hyperamylasemia can also occur as a result of conditions not involving pancreatitis. For example, hyperamylasemia can occur in a patient with small bowel obstruction, perforated duodenal ulcer, or other intra-abdominal inflammatory conditions. In contrast, a patient with acute pancreatitis may have a normal serum amylase level, which could be due to several reasons. In patients with hyperlipidemia, values might appear to be normal because of

interference by lipids with chemical determination of serum amylase. In many cases, urinary clearance of pancreatic enzymes from the circulation increases during pancreatitis; therefore, urinary levels may be more sensitive than serum levels. For these reasons, it is recommended that amylase concentrations also be measured in the urine. Urinary amylase levels usually remain elevated for several days after serum levels have returned to normal. In patients with severe pancreatitis associated with significant necrotic damage, the pancreas may not release large amounts of enzymes into the circulation. With increasing severity of disease, the intravascular fluid loss may become life-threatening as a result of sequestration of edematous fluid in the retroperitoneum. Hemoconcentration then results in an elevated hematocrit. However, there also may be bleeding into the retroperitoneum or the peritoneal cavity[86]. In some patients (about 1%), the blood from necrotizing pancreatitis may dissect through the soft tissues and manifest itself as a bluish discoloration around the umbilicus (Cullen's sign) or in the flanks(Grey Turner's sign)[14]. The severe fluid loss may lead to prerenal azotemia with elevated blood urea nitrogen and creatinine levels. There also may be hyperglycemia, hypoalbuminemia, and hypocalcemia sufficient in some cases to produce tetany

SEVERITY ASSESSMENT

Predicting acute pancreatitis severity is important in making triage and in making decisions about fluid therapy, whether an ERCP is indicated, and other issues. the most widely used being the Ranson's criteria or modified Glasgow criteria. Both use clinical and biochemical parameters scored over the first 48 hours of admission. When there are 3 or more positive criteria, the disease is considered"predicted severe." There are many other approaches to predicting severity. At 24 hours after admission an APACHE II score of 8 or more or a serum C-reactive protein level of >150mg/dl has a similar accuracy in predicting severity as Ranson's criteria. The more recently proposed Bedside Index for Severity of Acute Pancreatitis (BISAP) is calculated from blood urea nitrogen (> 25 mg/dl), impaired mental status (GCS <15), presence of systemic inflammatory response syndrome (SIRS), age >60 years, and pleural effusion.

Another approach has been taken in seeking to predict those with 'harmless' acute pancreatitis using three factors that can be determined on admission; absence of rebound tenderness or guarding, normal hematocrit, and normal serum creatinine. The accuracy of this approach appears to be over 90%, and triages most patients with acute pancreatitis away from intensive care.

CLASSIFICATION OF SEVERITY OF ACUTE PANCREATITIS

A four-category classification of severity

- ✓ mild,
- ✓ moderate,
- ✓ severe,
- ✓ critical) .

The key determinants of severity are local complications

- ✓ absent,
- ✓ sterile or
- ✓ infected)

and systemic complications (absent, transient organ failure, or persistent organ failure).

FOUR CATEGORIES OF ACUTE PANCREATITIS BASED ON ORGAN FAILURE & LOCAL COMPLICATIONS

DETERMINANTS	NO LOCAL COMPLICATIONS	STERILE LOCAL COMPLICATIONS	INFECTED LOCAL COMPLICATIONS
NO ORGAN FAILURE	MILD	MODERATE	SEVERE
TRANSIENT ORGAN FAILURE	MODERATE	MODERATE	SEVERE
PERSISTENT ORGAN FAILURE	SEVERE	SEVERE	CRITICAL

RANSON'S SCORE FOR NON GALLSTONE PANCREATITIS

BOX 56-1 Ranson's Prognostic Criteria for Non gallstone Pancreatitis
At presentation • Age >55 yr • Blood glucose level >200 mg/dL • WBC >16,000 cells/mm3 • Lactate dehydrogenase level >350 IU/L • Aspartate aminotransferase >250 IU/L After 48 hours of admission • Hematocrit*: Decrease >10% • S erum calcium level <8 mg/dL • Base deficit >4 mEq/L • Blood urea nitrogen level†: Increase >5 mg/dL • Fluid requirement >6 liters • Pao2 <60 mm Hg
Ranson score ≥3 defines severe pancreatitis.*As compared with admission value.

RANSON'S SCORE FOR GALLSTONE PANCREATITIS

At presentation • Age >70 yr • Blood glucose level >220 mg/dL • WBC >18,000 cells/mm3 • Lactate dehydrogenase level >400 IU/liter • Aspartate aminotransferase level >250 IU/liter After 48 hours of admission • Hematocrit*: Decrease >10% • S erum calcium level <8 mg/dL • Base deficit >5 mEq/L • Blood urea nitrogen level†: Increase >2 mg/dL • Fluid requirement >4 liters • Pao2: Not available
Ranson score ≥3 defines severe pancreatitis.

APACHE II SCORING SYSTEM

AP severity can also be addressed using the Acute Physiology and Chronic Health Evaluation (APACHE II) score. Based on the patient's age, previous health status, and 12 routine physiologic measurements, APACHE II provides a general measure of the severity of disease. An APACHE II score of 8 or higher defines severe pancreatitis. The main advantage is that it can be used on admission and repeated at any time. However, it is complex, not specific for AP, and based on the patient's age, which easily upgrades the AP severity score.

ATLANTA'S CRITERIA FOR ACUTE PANCREATITIS

Organ Failure As Defined Shock (systolic blood pressure <90 mm Hg) Pulmonary insufficiency (PaO2 <60 mm Hg) Renal failure (creatinine level >2 mg/dL after fluid resuscitation) GI bleeding (>500 mL/24 hr) Systemic Complications Disseminated intravascular coagulation (platelet count ≤100,000) Fibrinogen <1 gr/L Fibrin split products >80 μg/dL Metabolic disturbance (calcium level ≤7.5 mg/dL) Local Complications Necrosis Abscess Pseudocyst
Severe pancreatitis is defined by the presence of any evidence of organ failure or a local complication.

BISAP SCORE

the Bedside Index of Severity in Acute Pancreatitis incorporates five clinical and laboratory parameters obtained within the first 24 hours of hospitalization . Presence of three or more of these factors was associated with substantially increased risk for in-hospital mortality among patients with acute pancreatitis.

PARAMETERS	**SCORE**	
	1	**0**
Blood Urea Nitrogen	>25 mg%	<25 mg%
Impared mental status	present	Absent
Age	>60	<60
SIRS	2/4 present	Absent
Pleural effusion	present	Absent

CRITERIA FOR **SIRS**

• Temperature > 38 0 C or <36 • Pulse > 90/minute • Tachypnea > 24/minute • WBC > 12000/mm
Any two of four will be significant if present simultaneously. A score of > 3 will indicate pancreatitis (early organ failure/ pancreatic necrosis)

CT SEVERITY INDEX

FEATURE	POINTS
PANCREATIC INFLAMMATION	
Normal pancreas	0
Focal or diffuse pancreatic enlargement	1
Intrinsic pancreatic alterations ith peripancreatic fat inflammatory changes	2

Single fluid collection / or phlegmon	3
Two or more fluid collections or gas ,in or adjacent to the pancreas	4
Pancreatic necrosis	
None	0
Less than 30 %	2
30 to 50 %	4
>50 %	6

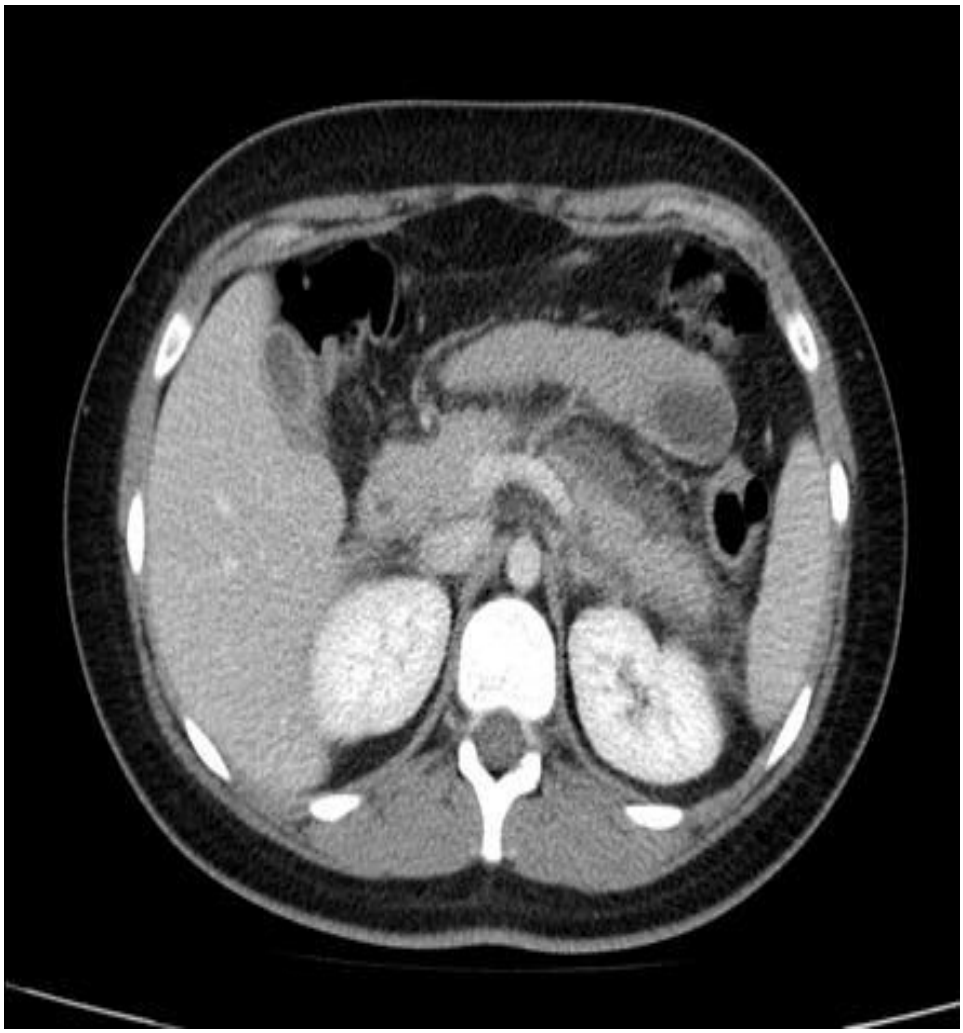
INTERPRETATION

Points	severity	morbitidy	Mortality
0 to 3	mild	3 %	8 %
4 to 6	moderate	6%	35%
7 to 10	severe	17%	92%

COMPLICATIONS

Sterile and Infected Peripancreatic Fluid Collections

The presence of acute abdominal fluid during an episode of AP has been described in 30% to 57% of patients.3 In contrast to pseudocysts and cystic neoplasias of the pancreas, fluid collections are not surrounded or encased by epithelium or fibrotic capsule. The presence of fever, elevated white blood cell (WBC) count, and abdominal pain suggest infection of this fluid and percutaneous aspiration is confirmatory. Percutaneous drainage and IV administration of antibiotics should be instituted if infection is present

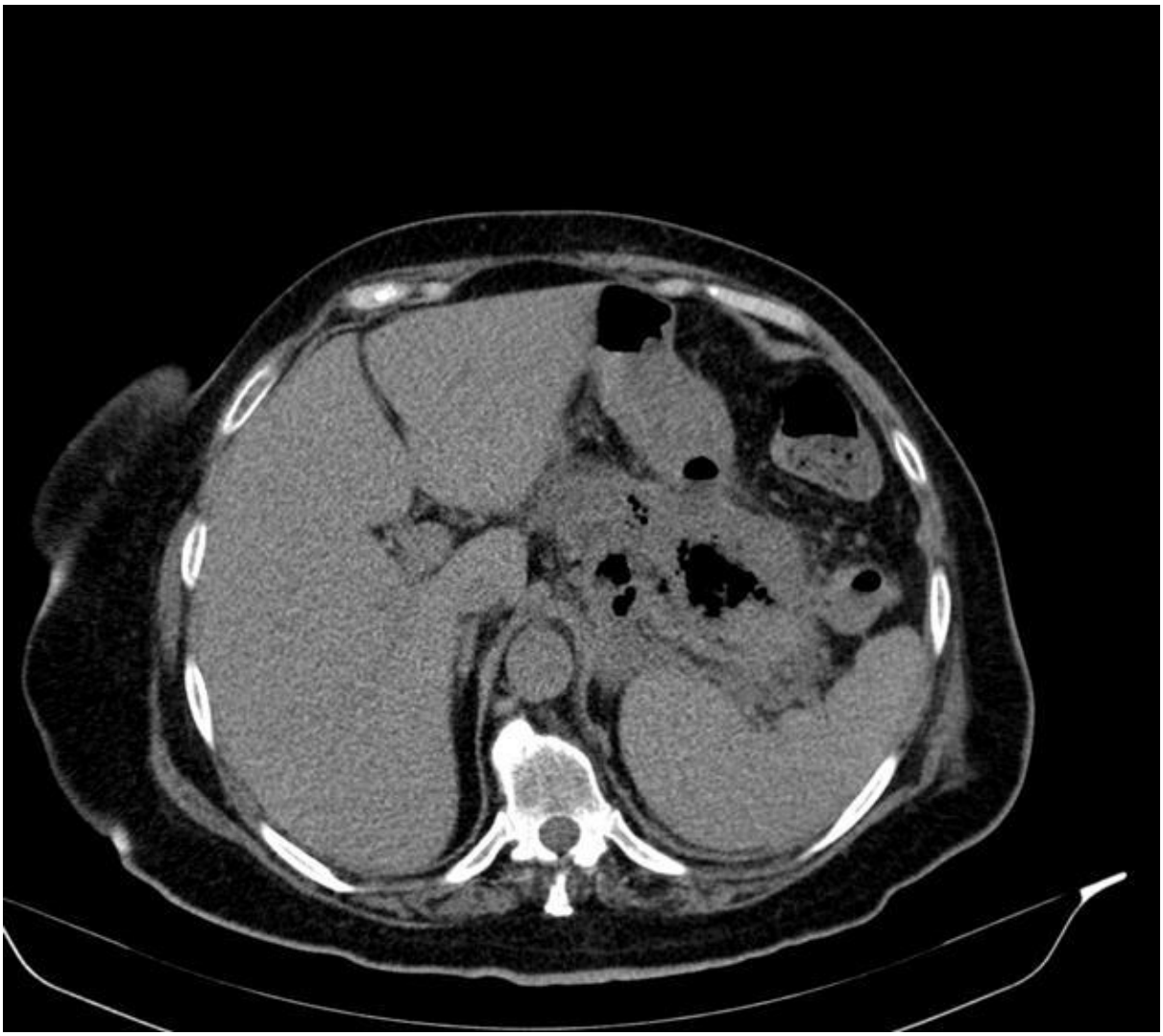


Peripancreatic fluid collection

Pancreatic Necrosis and Infected Necrosis

Pancreatic necrosis is the presence of nonviable pancreatic parenchyma or peripancreatic fat; it can present as a focal area or diffuse involvement of the gland. Contrast-enhanced CT is the most reliable technique to diagnose pancreatic necrosis. It istypically seen as areas of low attenuation (<40 to 50 HU) after the injection of IV contrast. Normal parenchyma usually has a density of 100 to 150 HU.9 Up to 20% of patients with AP develop pancreatic necrosis. It is important to identify and provide proper treatment of this complication because most who develop multiorgan failure have necrotizing pancreatitis; pancreatic necrosis has been documented in up to 80% of the autopsies of patients who died after an episode of AP.4 The main complication of pancreatic necrosis is infection. The risk is directly related to the amount of necrosis; in patients with pancreatic necrosis involving less than 30% of the gland, the risk of infection is 22%. The risk is 37% for patients with pancreatic necrosis that involves 30% to 50% of the gland and up to 46% if more than 70% of the gland is affected.4 This complication is associated with bacterial translocation usually involving enteric flora, such as gram-negative rods (e.g., Escherichia coli, Klebsiella and Pseudomonas spp.) and Enterococcus spp. Infected pancreatic necrosis should be suspected in patients with prolonged fever, elevated WBC count, or progressive clinical deterioration. Evidence of air within the pancreatic necrosis seen on a CT scan confirms the diagnosis but is a rare finding. If infected necrosis is suspected, fine-needle aspiration (FNA)

should be performed. Once infection has been demonstrated, IV antibiotics should be given. Because of their penetration into the pancreas and spectrum coverage, carbapenems are the first option of treatment. Alternative therapy includes quinolones, metronidazole,third-generation cephalosporins, and piperacillin. Definitive treatment for infected pancreatic necrosis is surgical débridement with necrosectomy, closed continuous irrigation,
and open packaging .



Acute pancreatitis with infected necrosis

Pancreatic Pseudocysts

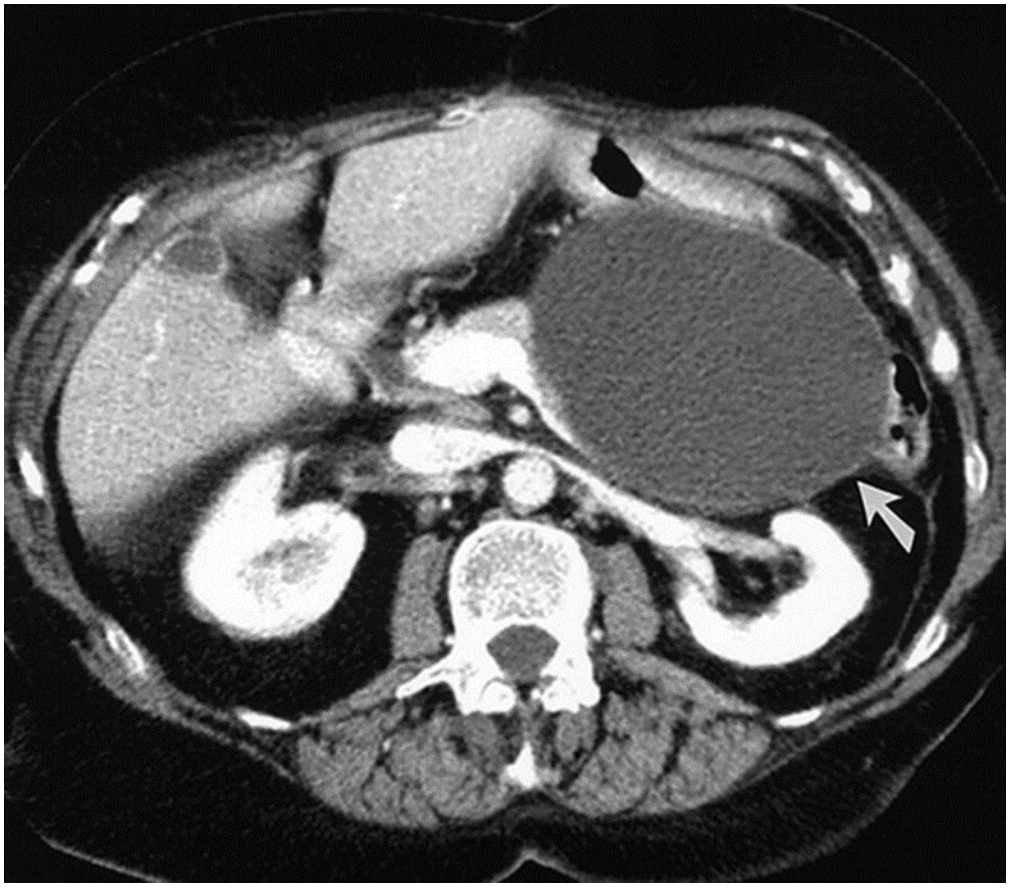
Pancreatic pseudocysts occur in 5% to 15% of patients who have peripancreatic fluid collections after AP. By definition, the capsule of a pseudocyst is composed of collagen and granulation tissue and it is not lined by epithelium.The fibrotic reactiontypically requires at least 4 to 8 weeks to develop. Up to 50% of patients with pancreatic pseudocysts will develop symptoms. The presence of persistent pain, early satiety, nausea, weight loss, and elevated pancreatic enzyme levels in plasma suggest this diagnosis. The diagnosis is corroborated with by CT or MRI. EUS with FNA is indicated for patients in whom the diagnosis of pancreatic pseudocyst is not clear. Characteristic features of pancreatic pseudocysts include high amylase levels associated with the absence of mucin and low carcinoembryonic antigen (CEA) levels

Observation is indicated for asymptomatic patients because spontaneous regression has been documented in up to 70% of cases; this is particularly true for patients with pseudocysts smaller than 4 cm in diameter, located in the tail, and no evidence of pancreatic duct obstruction or communication with the main pancreatic duct.Invasive therapies are indicated for symptomatic patients or when the differentiation between a cystic neoplasm and pseudocyst is not possible. Because most patients are treated with decompressive procedures and not with resection, it is imperative to have a pathologic diagnosis. Surgical drainage has been the traditional approach for pancreatic pseudocysts.[16]

Transgatric and transduodenal endoscopic drainage are safe and effective approaches for patients with pancreatic pseudocysts in close contact (defined as <1 cm) with the stomach and duodenum, respectively. In addition,transpapillary drainage can be attempted in pancreatic pseudocysts communicating with the main pancreatic duct. For patients in whom a pancreatic duct stricture is associated with a pancreatic pseudocyst, endoscopic dilation and stent placement are indicated

Surgical drainage is indicated for patients with pancreatic pseudocysts that cannot be treated with endoscopic techniques and patients who fail endoscopic treatment. Definitive treatment depends on the location of the cyst. Pancreatic pseudocysts closely attached to the stomach should be treated with a cystgastrostomy. Pancreatic pseudocysts located in the head of the pancreas that are in close contact with the duodenum are treated with a cystoduodenostomy. Finally, some pseudocysts are not in contact with the stomach or duodenum. The surgical treatment for these patients is a Roux-en-Y cystojejunostomy.

Complications of pancreatic pseudocysts include bleeding and pancreaticopleural fistula secondary to vascular and pleural erosion, respectively, bile duct and duodenal obstruction, rupture into the abdominal cavity, and infection. Percutaneous drainageis only indicated for septic patients secondary to pseudocyst infection because it has a high incidence of external fistula.

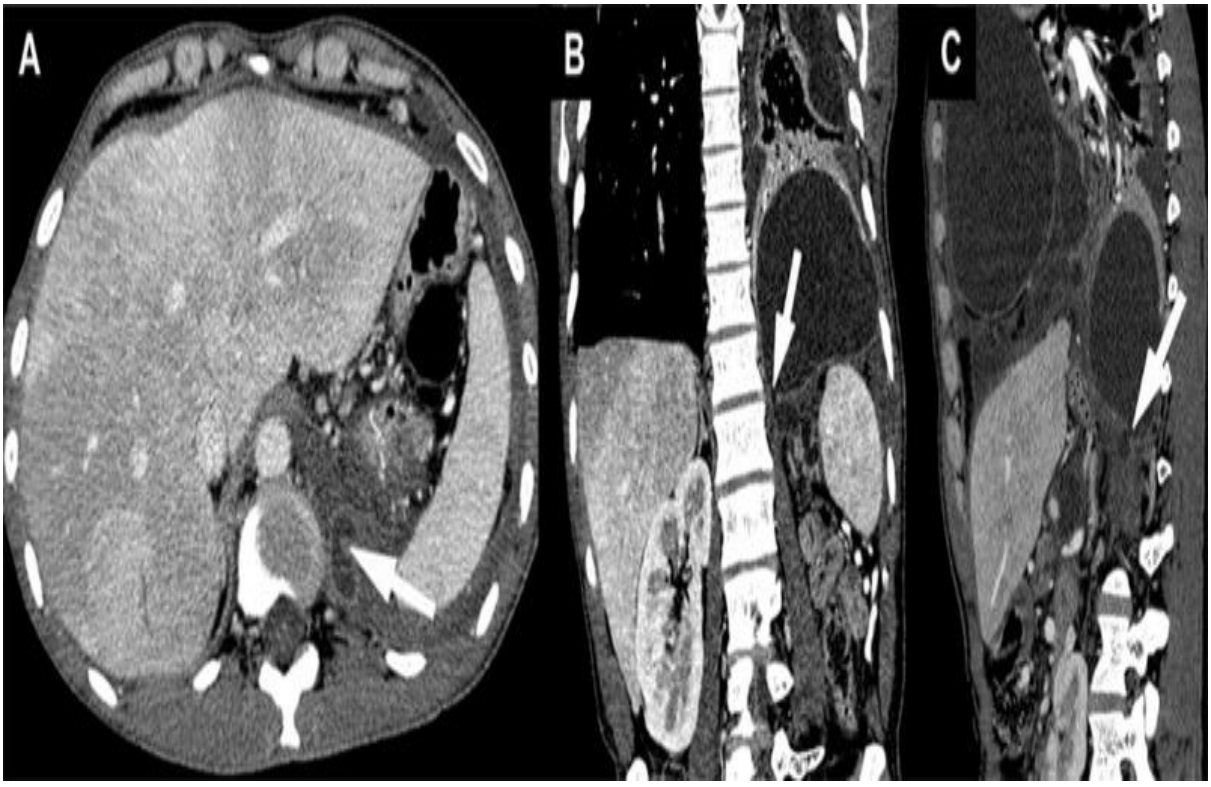


PANCREATIC PSEUDOCYST

Pancreatic Ascites and Pancreaticopleural Fistulas

complete disruption of the pancreatic duct can lead to significant accumulation of, fluid. This condition should be suspected in patients who have an episode of AP, develop significant abdominal distention, and have free intraabdominal fluid. Diagnostic paracentesis typically demonstrates elevated amylase and lipase levels. Treatment consists of abdominal drainage combined with

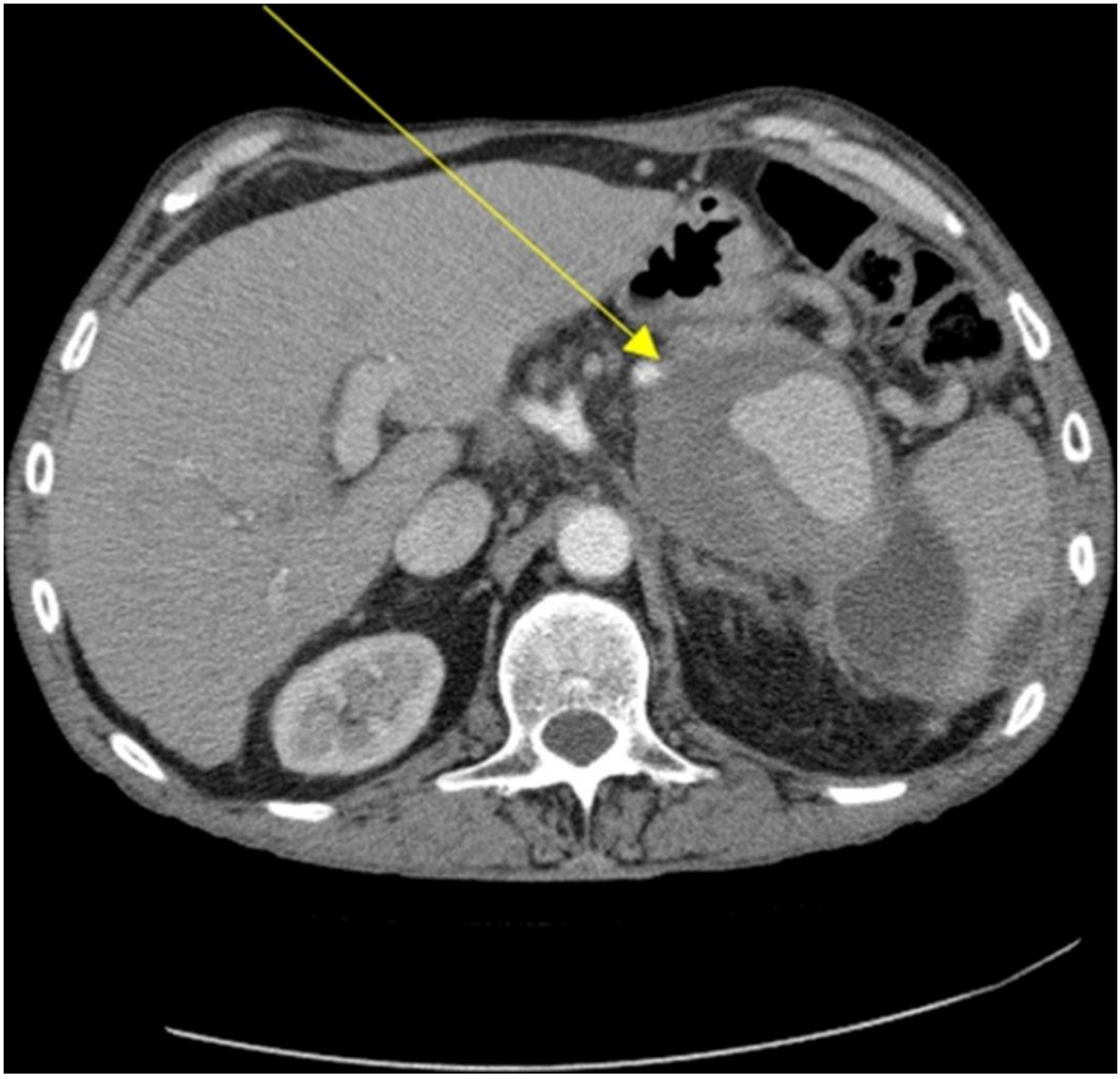
endoscopic placement of a pancreatic stent across the disruption. Failure of this therapy requires surgical treatment; it consists of distal resection and closure of the proximal stump. Pancreatico pleural fistula is rare. Symptoms that suggest this condition include dyspnea, abdominal pain, cough, and chest pain. The diagnosis is confirmed with chest x-ray, thoracentesis, and CT scan. Amylase levels above 50,000 IU in the pleural fluid confirm the diagnosis. It is more common after alcoholic pancreatitis and, in 70% of patients, is associated with pancreatic pseudocysts. Initial treatment requires chest drainage, parenteral nutritional support, and administration of octreotide. Up to 60% of patients respond to this therapy. Persistent drainage should also be treated with endoscopic sphincterotomy and stent placement. Patients who do not respond to these measures require surgical treatment, similar to that described for pancreatic ascites.[17]



Pancreaticopleural fistula (arrows) detected with the second CT examination, which was performed after the thoracocentesis: (A) axial image, (B) coronal reconstruction, (C) sagittal reconstructionS

Vascular Complications

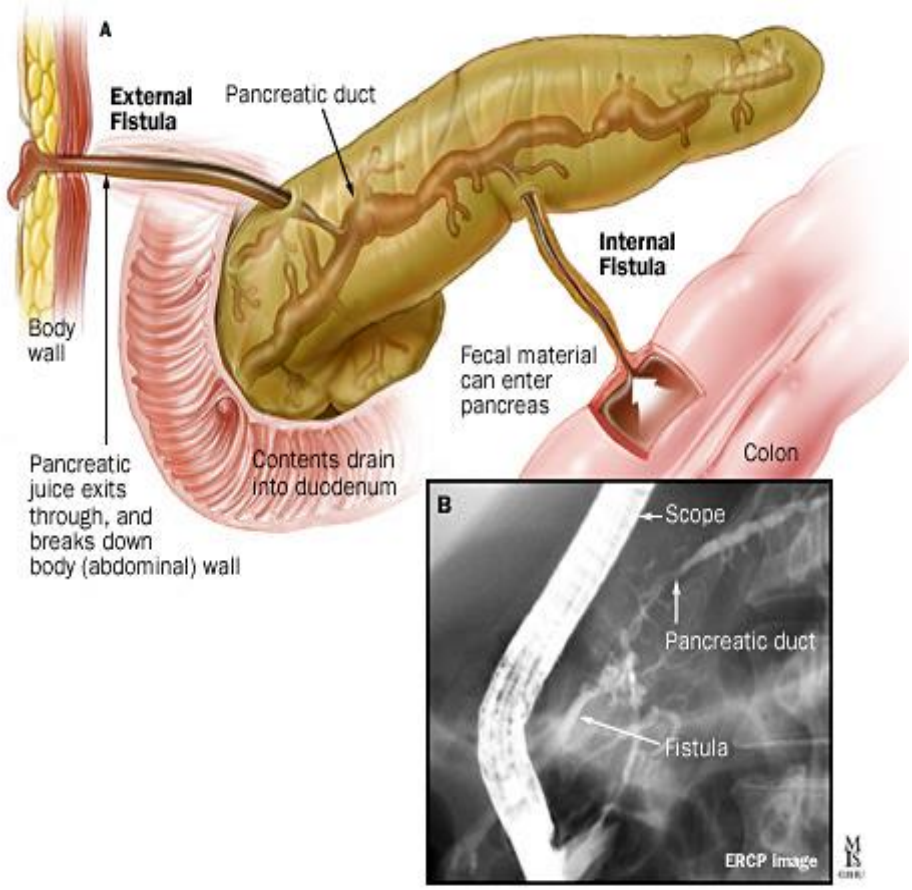
Acute pancreatitis is rarely associated with arterial vascular complications. The most common vessel affected is the splenic artery, but the superior mesenteric, cystic, and gastroduodenal arteries have also been found to be affected. It has been proposed that pancreatic elastase damages the vessels, leading to pseudoaneurysm formation. Spontaneous rupture results in massive bleeding. Clinical manifestations include sudden onset of abdominal pain, tachycardia, and hypotension. If possible, arterial embolization should be attempted to control the bleeding. Refractory cases require ligation of the vessel affected. The mortality ranges from 28% to 56%. Pancreatic inflammation can also produce vascular thrombosis; the vessel usually affected is the splenic vein but, in severe cases, it can extend into the portal venous system.



CT angiography depicting an 8.6-cm partially thrombosed splenic artery pseudoaneurysm at the pancreatic tail and a subcapsular splenic fluid collection.

Pancreatocutaneous Fistula

The frequency of pancreatic fistulas is rare However, the incidence of these complications increases in patients with other complications after AP in patients with pancreatic pseudocysts and in patients with infected necrosis after surgical débridement.Treatment is conservative for most patients.



PANCREATICO CUTANEOUS & PANCREATICO ENTERIC FIST

OBSERVATION AND DISCUSSION

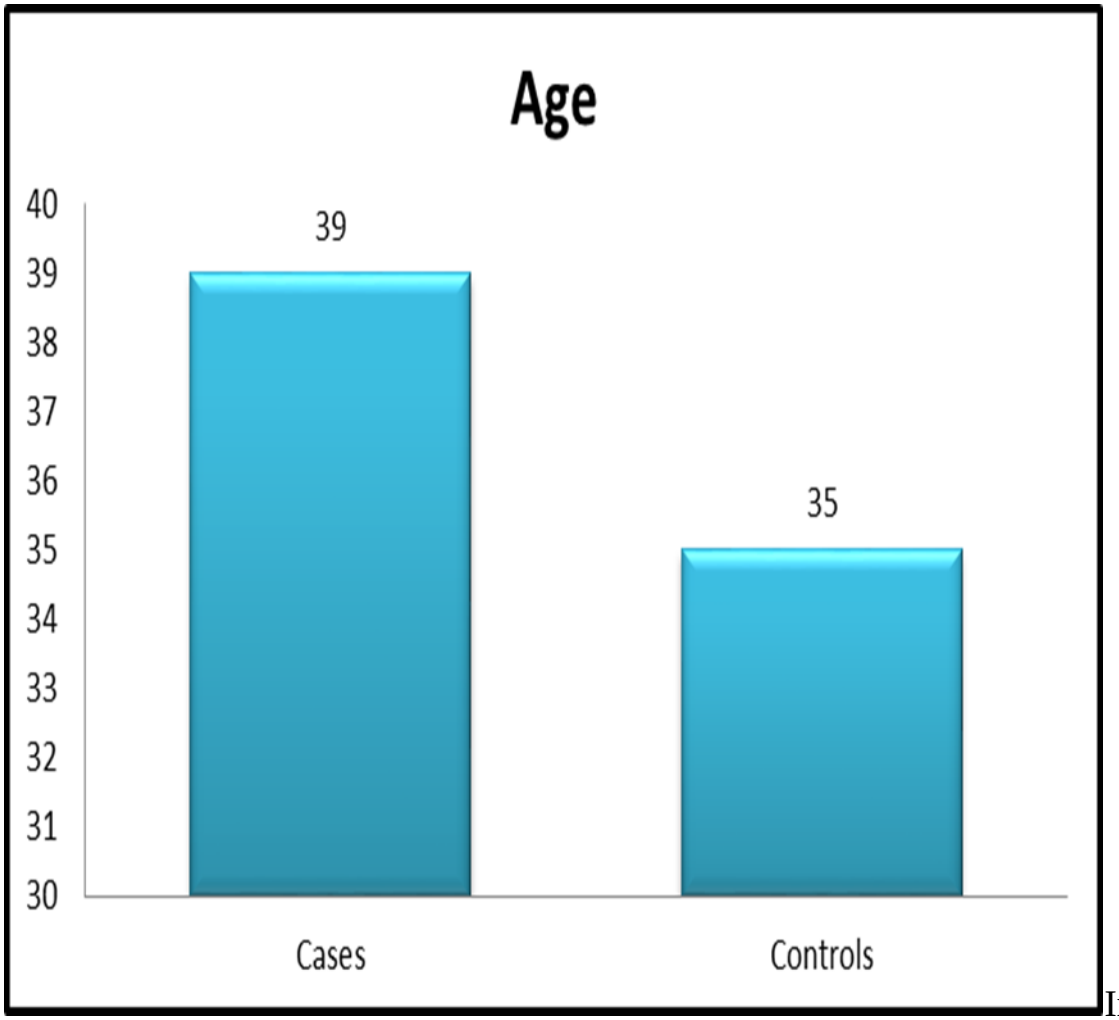
- ✓ it is a randamised control study

- ✓ this review describes the beneficial effects of early enteral feeding in acute pancreatitis

- ✓ relevant literature analysed in the view points of early enteral vs parenteral feeding in acute pancreatitis

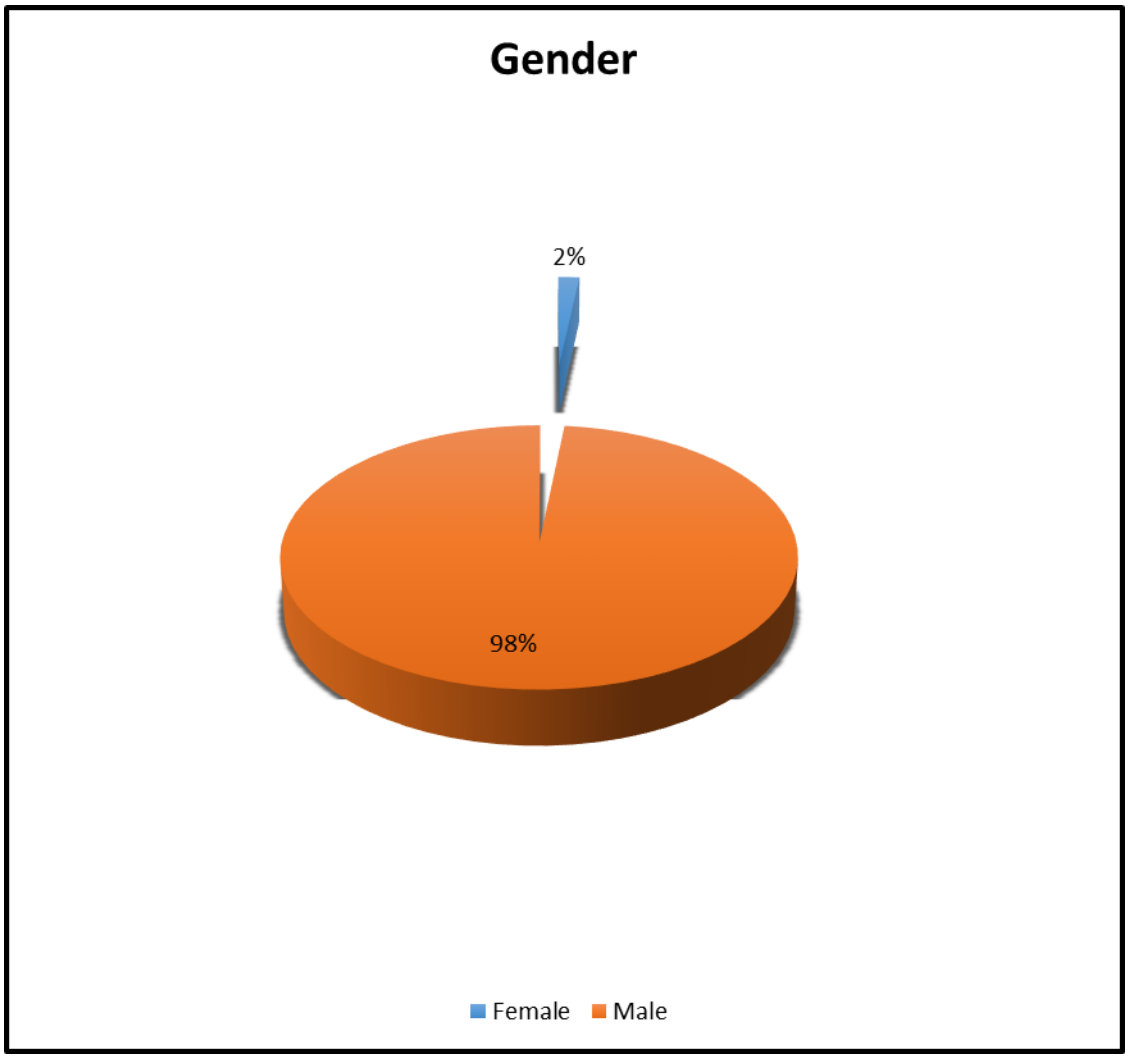
- ✓ latest meta analyses suggest that enteral nutrition significantly reduces the morbitidies and mortality in acute pancreatitis compared to parentral feeding

- ✓ to maintain the gut barrier function and to prevent the early bacterial translocation,early enteral feeding commenced with in 24-48 hours of admission



In st

- ✓ IN MY STUDY MOST COMMONEST AGE GROUP IS 31 – 40 YEARS,WHICH IS 61.7 % OF MY POPULATION
- ✓ *LEAST COMMONEST AGE GROUP IS ABOVE 50,WHICH IS 6.7 % OF MY POPULATION*



✓ IN MY STUDY THE MOST COMMONEST SEX AFFECTED IS MALE (98.3 %)

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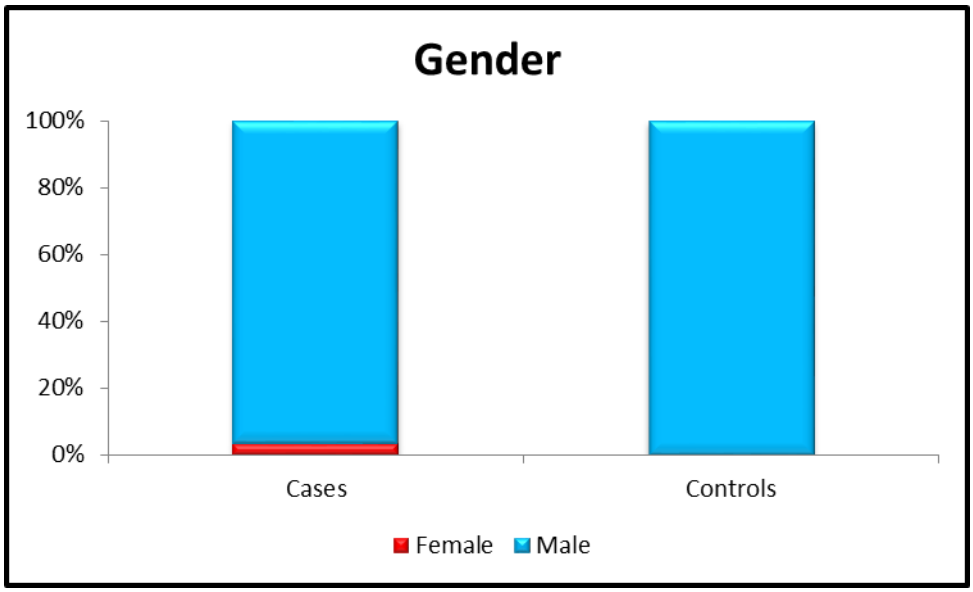

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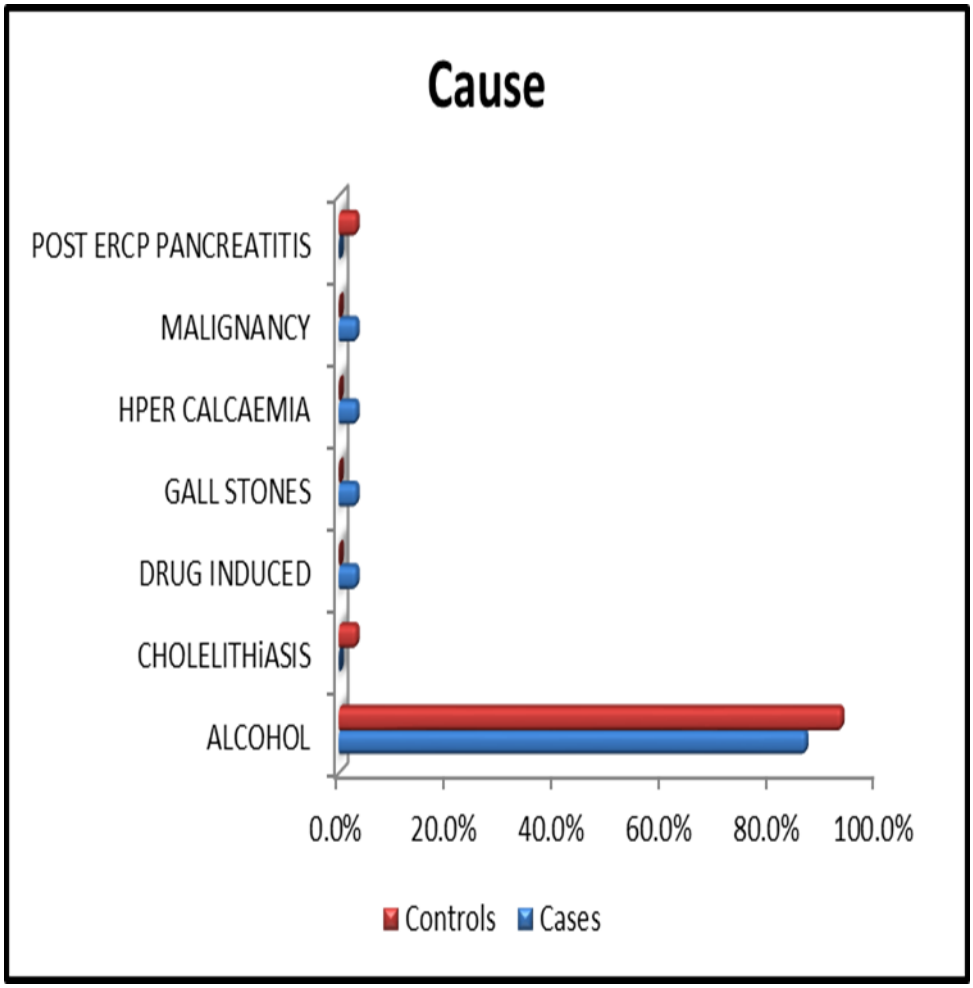
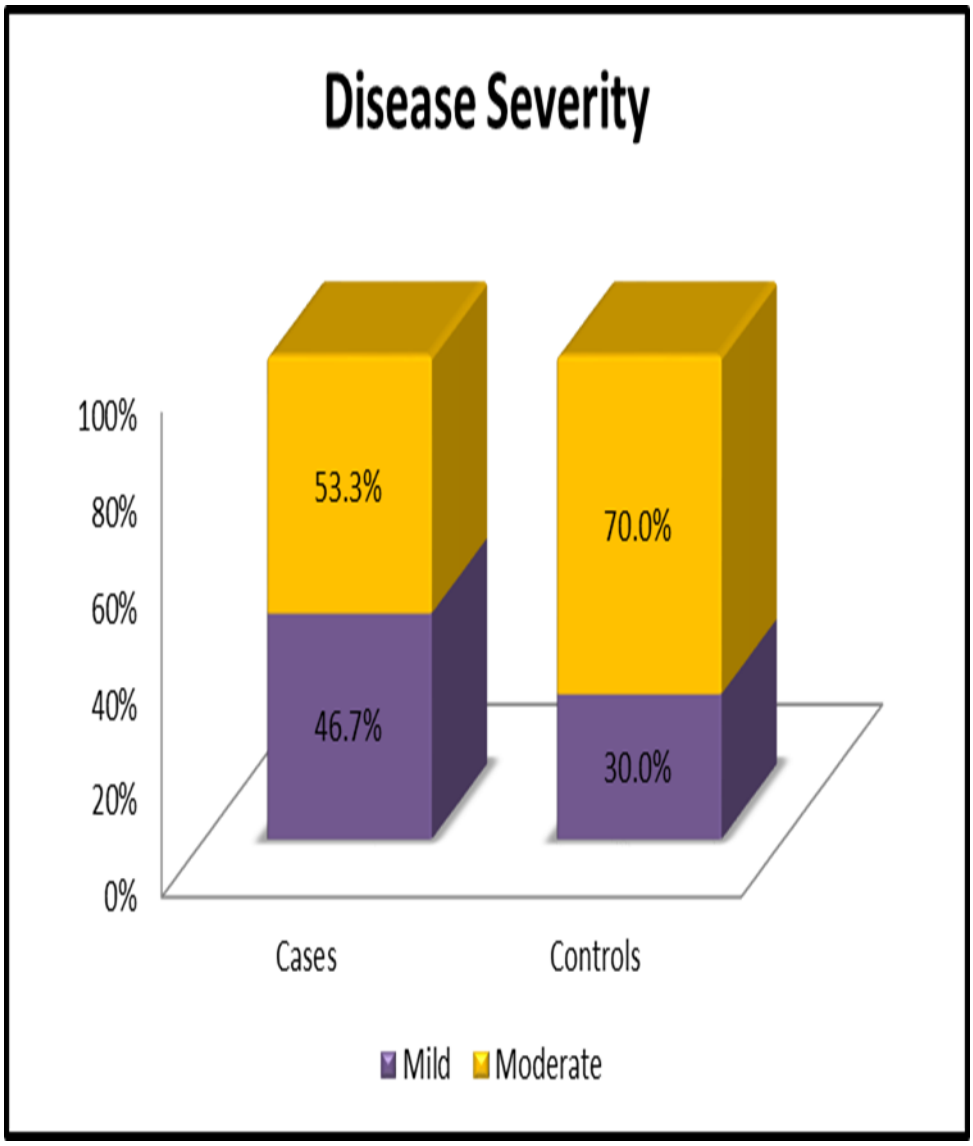
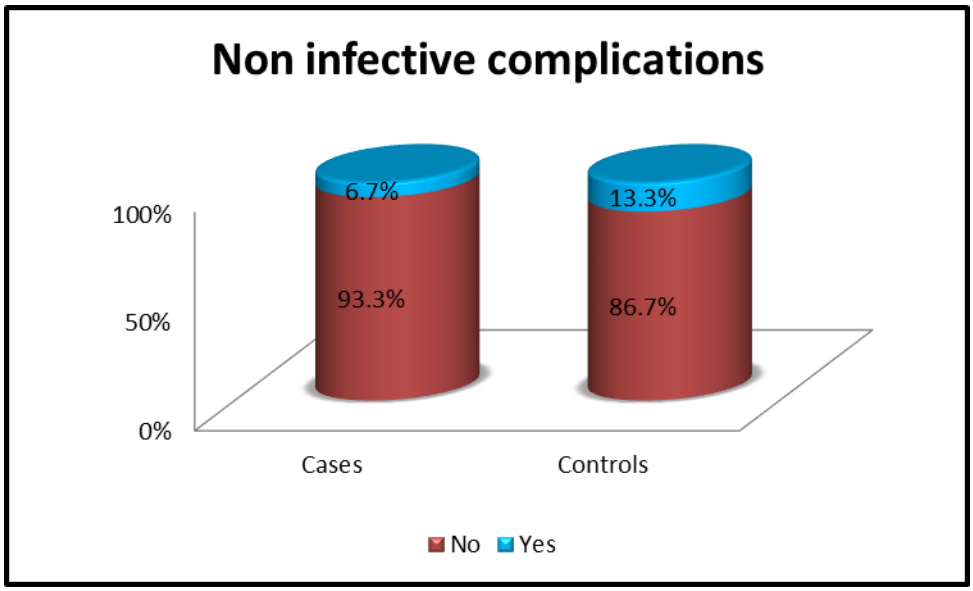


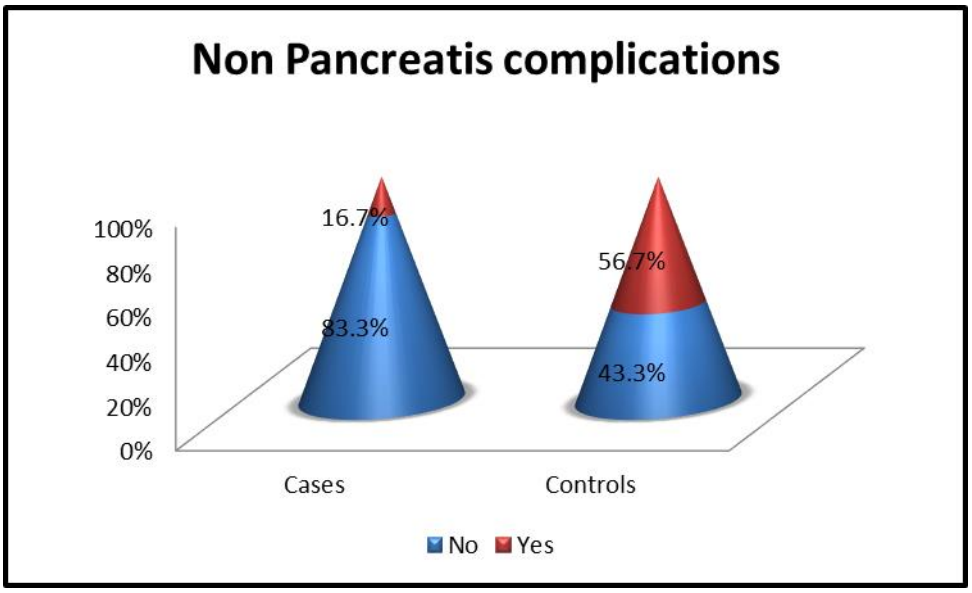
- ✓ THE MOST COMMONEST CAUSE OF PANCREATITIS IN MY STUDY POPULATION IS ALCOHOL (90%)



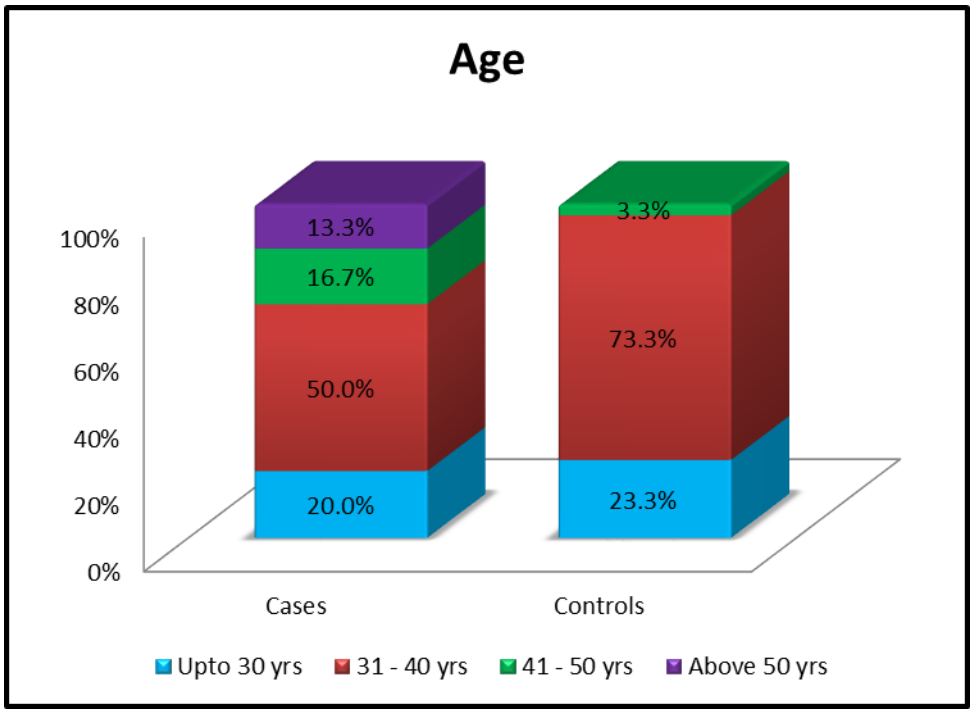
- ✓ Total no of mild severity cases in my study population are 23 (38.3%),in which the cases are 14 (46.7% of mild cases) controls 9 (30% of mild cases).Total no of moderate cases in my study population are 37 (61.7 %).inwhich the cases are 16 (53.3 %) and controls are 21 (70 %)



- Totally 6 cases are affected by non infective complications (10% of total population).in which 2 cases (6.7 % of cases) & 4 controls (13.3 % of controls)

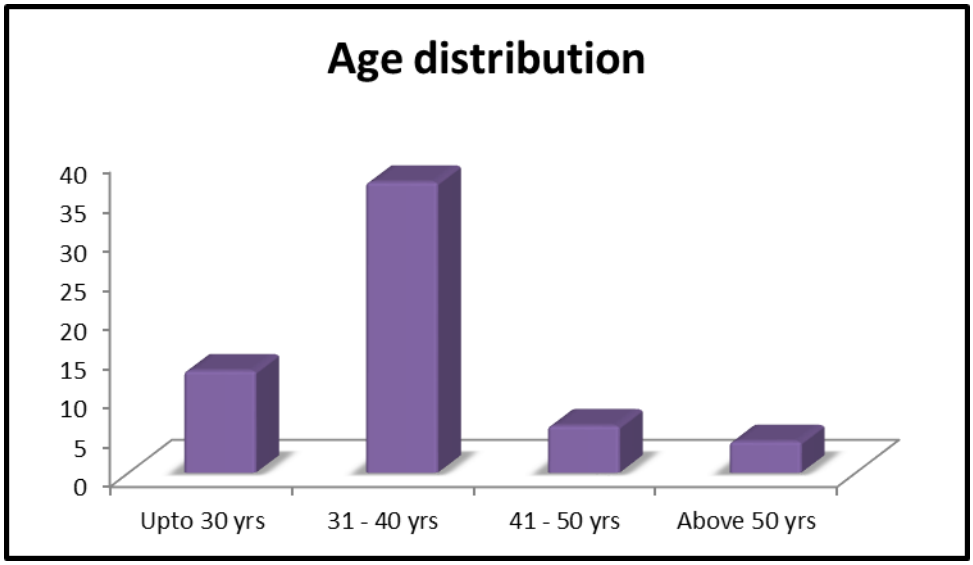


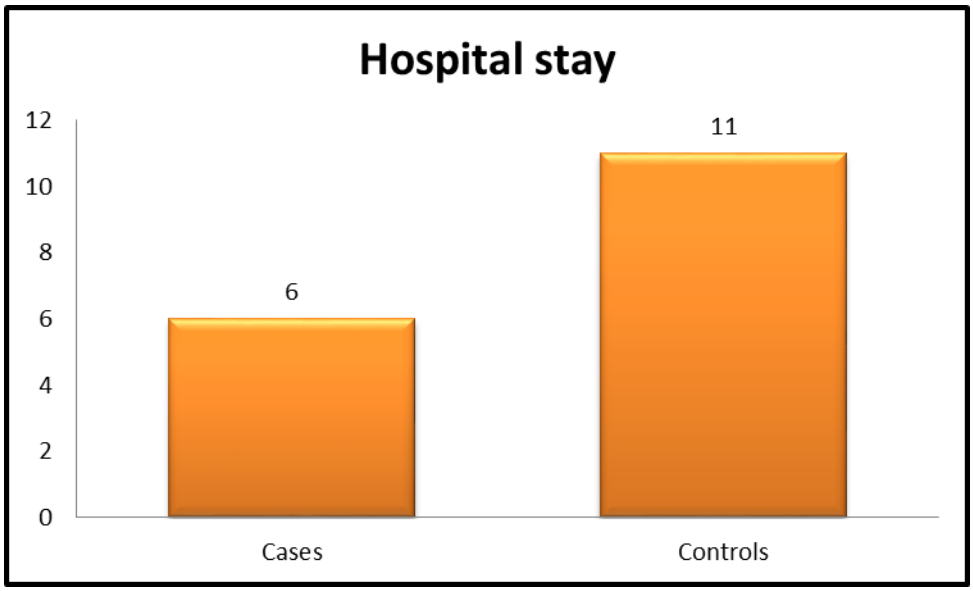
- **Totally 22 cases were affected by non pancreatitis complications.in which 5 cases (16.7 % of cases),17 controls (56.7 % of controls)**



- **IN MY STUDY MOST COMMONEST AGE GROUP IS 31 – 40 YEARS,WHICH IS 61.7 % OF MY POPULATION**
- **LEAST COMMONEST AGE GROUP IS ABOVE 50,WHICH IS 6.7 % OF MY POPULATION**

✓


Age distribution
40
35
30
25
20
15
10
5
0
Upto 30 yrs
31 - 40 yrs
41 - 50 yrs
Above 50 yrs



- ✓ The average length of hospital stay in cases -6.43 days

The average length of hospital stay in controls- 10.48 days

T-Test

CC		N	Mean	Std. Deviation	Std. Error Mean
AGE	Cases	30	39	9.380	1.712
	Controls	30	35	4.056	.741
STAY	Cases	30	6	2.873	.525
	Controls	30	11	3.199	.584

Independent Samples Test

	t-test for Equality of Means		
	t	df	Sig. (2-tailed)
AGE	2.162	58	.035
STAY	-5.562	58	.000

			CC		
			Cases	Controls	Total
SEX	F	Count	1	0	1
		% within CC	3.3%	0.0%	1.7%
	M	Count	29	30	59
		% within CC	96.7%	100.0%	98.3%
Total		Count	30	30	60
		% within CC	100.0%	100.0%	100.0%

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.017[a]	1	.313		
Continuity Correction[b]	0.000	1	1.000		
Likelihood Ratio	1.403	1	.236		
Fisher's Exact Test				1.000	.500
N of Valid Cases	60				

a. 2 cells (50.0%) have expected count less than 5. The minimum expected count is .50.

			CC		
			Cases	Controls	Total
CAUSE	ALCOHOL	Count	26	28	54
		% within CC	86.7%	93.3%	90.0%
	CHOLELITHiASIS	Count	0	1	1
		% within CC	0.0%	3.3%	1.7%
	DRUG INDUCED	Count	1	0	1
		% within CC	3.3%	0.0%	1.7%
	GALL STONES	Count	1	0	1
		% within CC	3.3%	0.0%	1.7%
	HPER CALCAEMIA	Count	1	0	1
		% within CC	3.3%	0.0%	1.7%
	MALIGNANCY	Count	1	0	1
		% within CC	3.3%	0.0%	1.7%
	POST ERCP PANCREATITIS	Count	0	1	1
		% within CC	0.0%	3.3%	1.7%
Total		Count	30	30	60
		% within CC	100.0%	100.0%	100.0%

	Cases	Controls
ALCOHOL	86.7%	93.3%
CHOLELITHiASIS	0.0%	3.3%
DRUG INDUCED	3.3%	0.0%
GALL STONES	3.3%	0.0%
HPER CALCAEMIA	3.3%	0.0%
MALIGNANCY	3.3%	0.0%
POST ERCP PANCREATITIS	0.0%	3.3%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	6.074[a]	6	.415
Likelihood Ratio	8.392	6	.211
N of Valid Cases	60		

a. 12 cells (85.7%) have expected count less than 5. The minimum expected count is .50.

			CC		
			Cases	Controls	Total
DISEASE SEVERITY	MILD	Count	14	9	23
		% within CC	46.7%	30.0%	38.3%
	MODERATE	Count	16	21	37
		% within CC	53.3%	70.0%	61.7%
Total		Count	30	30	60
		% within CC	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.763[a]	1	.184		
Continuity Correction[b]	1.128	1	.288		
Likelihood Ratio	1.773	1	.183		
Fisher's Exact Test				.288	.144
N of Valid Cases	60				

			CC		
			Cases	Controls	Total
NON INFECTIVE COMPLICATIONS	NO	Count	28	26	54
		% within CC	93.3%	86.7%	90.0%
	YES	Count	2	4	6
		% within CC	6.7%	13.3%	10.0%
Total		Count	30	30	60
		% within CC	100.0%	100.0%	100.0%

	Cases	Controls
No	93.3%	86.7%
Yes	6.7%	13.3%

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.741[a]	1	.389		
Continuity Correction[b]	.185	1	.667		
Likelihood Ratio	.754	1	.385		
Fisher's Exact Test				.671	.335
N of Valid Cases	60				

	CASES	CONTROLS
NO	83.3 %	43.3 %
YES	16.7 %	66.7 %

			CC		
			Cases	Controls	Total
NON PANCREATITIS COMPLICATIONS	NO	Count	25	13	38
		% within CC	83.3%	43.3%	63.3%
	YES	Count	5	17	22
		% within CC	16.7%	56.7%	36.7%
Total		Count	30	30	60
		% within CC	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	10.335[a]	1	.001		
Continuity Correction[b]	8.684	1	.003		
Likelihood Ratio	10.771	1	.001		
Fisher's Exact Test				.003	.001
N of Valid Cases	60				

	Cases	Controls
Upto 30 yrs	20.0%	23.3%
31 - 40 yrs	50.0%	73.3%
41 - 50 yrs	16.7%	3.3%
Above 50 yrs	13.3%	

Crosstab

			CC		
			Cases	Controls	Total
AGE	Upto 30 yrs	Count	6	7	13
		% within CC	20.0%	23.3%	21.7%
	31 - 40 yrs	Count	15	22	37
		% within CC	50.0%	73.3%	61.7%
	41 - 50 yrs	Count	5	1	6
		% within CC	16.7%	3.3%	10.0%
	Above 50 yrs	Count	4	0	4
		% within CC	13.3%	0.0%	6.7%
Total		Count	30	30	60
		% within CC	100.0%	100.0%	100.0%

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	8.068[a]	3	.045
Likelihood Ratio	9.866	3	.020
Linear-by-Linear Association	4.750	1	.029
N of Valid Cases	60		

RESULTS & CONCLUSIONS

AGE

- In my study , the total population is 60
- Cases – 30 & controls – 30. The mean age of case group is 39 & the mean age of control group is 35.
- Regarding distribution 13 patients are upto 30 years of age (21.7 % of population).in which 6 cases (20 % of cases) & 7 controls (23.3 % of controls) Confined to this group
- 37 patients (61.7 %) of study population fall between 31 – 40 years .inwhich 15 cases (50 % of cases) & 22 controls (73.3 % of controls) confined to this group
- 6 patients fall between 41 – 50 years (10 % of population).in which 5 cases (16.7 % of cases) & 1 control (3.3% of controls) confined to this group
- 4 (6.7% of population)patients fall in above 50 group.in which 4 controls (13.3 % of controls) confined to this group
- **IN MY STUDY MOST COMMONEST AGE GROUP IS 31 – 40 YEARS,WHICH IS 61.7 % OF MY POPULATION**
- **LEAST COMMONEST AGE GROUP IS ABOVE 50,WHICH IS 6.7 % OF MY POPULATION**

SEX

- In the total population of 60, only one case was female , females in my study is 1.7 % & males are 98.3 %

- **IN MY STUDY THE MOST COMMONEST SEX AFFECTED IS MALE (98.3 %)**

CAUSES

- Total no of patients affected by alcohol in my study population are 54 & it is 90 % of my population , followed by cholelithiasis (3.3 %) ,hpercalcaemia (1.7 %),malignancy (1.7 %)& post ERCP (1.7 %).

- **THE MOST COMMONEST CAUSE OF PANCREATITIS IN MY STUDY POPULATION IS ALCOHOL (90%)**

DISEASE SEVERITY

- Total no of mild severity cases in my study population are 23 (38.3%),in which the cases are 14 (46.7% of mild cases) controls 9 (30% of mild cases).Total no of moderate cases in my study population are 37 (61.7 %).inwhich the cases are 16 (53.3 %) and controls are 21 (70 %)

- **THERE IS NO STATISTICALLY SIGNIFICANT DIFFERENCE BETWEEN CASES & CONTROLS IN DISEASE SEVERITY**

NON INFECTIVE COMPLICATIONS

- Totally 6 cases are affected by non infective complications (10% of total population).in which 2 cases (6.7 % of cases) & 4 controls (13.3 % of controls)

- **INCIDENCE OF NON-INFECTIVE COMPLICATIONS IS HIGER IN CONTROL GROUPS THAN CASES**

- **THERE IS NO STATISTICALLY SIGNIFICANT DIFFERENCE IN INCIDENCENCE OF NON INFECTIVE COMPLICATIONS BETWEEN CASES & CONTROLS**

NON PANCREATITIS COMPLICATIONS

- Totally 22 cases were affected by non pancreatitis complications.in which 5 cases (16.7 % of cases),17 controls (56.7 % of controls)

- **INCIDENCE OF NON PANCREATITIS COMPLICATIONS IS HIGHER IN CONTROL GROUP THAN THE CASE**
- **THERE IS A STATISTICALLY SIGNIFICANT DIFFERENCE IN NON PANCREATITIS COMPLICATIONS BETWEEN CASES & CONTROLS**

HOSPITAL STAY

The average length of hospital stay in cases -6.43 days

The average length of hospital stay in controls- 10.48 days

- **THERE IS A STATISTICALLY SIGNIFICANT DIFFERENCE IN LENGTH OF HOSPITAL STAY BETWEEN CASES & CONTROLS**

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