"EVALUATION OF ACUTE PANCREATITIS AND ITS CORRELATION WITH CLINICAL OUTCOME USING MODIFIED COMPUTED TOMOGRAPHY SEVERITY INDEX IN A TERTIARY CARE HOSPITAL - AN OBSERVATIONAL STUDY"

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Endorsement

This is to certify that the dissertation entitled **"EVALUATION OF ACUTE PANCREATITIS AND ITS CORRELATION WITH CLINICAL OUTCOME USING MODIFIED COMPUTED TOMOGRAPHY SEVERITY INDEX IN A TERTIARY CARE HOSPITAL - AN OBSERVATIONAL STUDY"** is a bonafide research work done by **REG NO. BH0119001.**

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LIST OF ABBREVIATIONS

KAHER	–	KLE Academy of Higher Education and Research
KLES	–	Karnataka Lingayat Education Society
AP	_	Acute Pancreatitis
CP	_	Chronic Pancreatitis
CECT	_	Contrast Enhanced Computed Tomography
USG	_	Ultrasonography
DB	_	Dorsal Bud
VB	_	Ventral Bud
IUL	_	Intra uterine life
CBD	_	Common bile Duct
PD	-	Pancreatic duct
SMA	-	Superior mesenteric artery
PDA	-	Pancreatico duodenal artery
GDA	-	Gastro duodenal artery
SMV	-	Superior mesenteric Vein
PV	-	Portal vein
VIP	-	Vasoactive intestinal peptide
ER	-	Endoplasmic Reticulum
ABP	-	Acute biliary Pancreatitis
AAP	_	Acute alcoholic Pancreatitis
GP	-	Glycoprotein
ERCP	-	Endoscopic retrograde cholangiopancreatography
MRCP	-	Magnetic resonance cholangiopancreatography

CRP	-	C-reactive Protein
SD	-	Standard deviation
LOHS	-	Length of hospital stays
AUC	-	Area under curve
APACHE	-	Acute physiological assessment and chronic health evaluation
CTSI	-	Computed tomography severity index
MCTSI	-	Modified computed tomography severity index
MODS	-	Multi organ dysfunction syndrome

ABSTRACT

Background- One of the maximum causes presenting to casualty as acute pain abdomen is acute pancreatitis (AP)[1.] AP can be classified as [2] - Mild - self limiting disease (edematous) and severe - necrosis (MODS and death). Simple radiographs are not useful for diagnosing AP, but can rule out differential diagnosis that mimic AP such as perforated duodenal ulcers. After 48 to 72 hours of acute pain abdomen, CECT abdomen of pelvis gives increased chances of diagnosing acute pancreatitis[17]. Balthazar introduced a classification scheme for AP to forecast the severity of the disease. Presence of organ failure, extra pancreatic complications such as ascites, pleural effusion, vascular complications such as splenic vein thrombosis were not included in Balthazar scoring system. [1,2,3,14,22,23] Due limitations in Balthazar scoring system, Mortele introduced another system using CT severity index to measure the accurate clinical scoring and outcome including the occurrence of infections organ failure length of hospital stay and death [1,2,8,6,22,23]

Aims - To evaluate acute pancreatitis and its correlation with the clinical outcome using MCTSI

Methodology - A Hospital based one year cross sectional study was conducted in the Department of General surgery , KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi from 1st January 2020 to 31st December 2020 and required data was collected from 60 patient who were clinically diagnosed to have acute pancreatitis is admitted and subjected to contrast enhanced CT scan of abdomen and pelvis and further assessed with Modified CT Severity index scoring and the length of hospital stay.

Results – 60 patients were assessed in our study. On analyzing patients with respect to severity, 11-mild score, 33-moderate score, 16-severe score. Mean duration of hospital stay is 10.17 ± 7.64 ranging from minimum stay of 1 day to maximum stay of 77 days. Mean duration of hospital stay in mild group was 10.82 days, moderate group was 8.97 days and severe group was 12.19 days. The p value of variance (ANOVA) for average number of

hospital days is 0.3725 (NS). It was found that there was no significant difference in the number of hospital days.

Conclusion – From our observations; the duration of hospitalisation is not influenced by MCTSI. Duration of Hospitalisation differs from patient to patient and their response to treatment and does not follow the MCTSI grading as such mild patients stay for lesser time and severe patients stay for longer time. We conclude that MCTSI scoring does not influence clinical outcome (length of hospital stay).

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INTRODUCTION

One of the maximum causes presenting to casualty as acute pain abdomen is acute pancreatitis (AP)[1].

AP is an inflammation of the pancreas - Initially edema occurs followed by necrosis when severe.

AP can be classified as [2]

a) Mild - self-limiting disease (oedematous)

b) Severe - necrosis (MODS and death)

Common causes of pancreatitis in India is cholelithiasis with choledocholithiasis, alcohol abuse, ERCP induced such as in choledocholithiasis, drug-induced such as sulfonamides, thiazides, furosemide, propofol, oestrogen therapy, metabolic diseases such as hypercalcemia and hyperlipidemia.[12]

For classifying and grading - Acute pancreatitis, there are several clinical and radiological scoring systems such as Ranson's criteria, Apache II scoring system and CT severity index. [1,2,3]

Three-fold increase in pancreatic enzymes such as serum amylase and lipase inside the plasma verifies the diagnosis.

T half of serum amylase is shorter hence after 24 to 48 hours of symptoms serum lipase is the most sensitive indicator to establish the diagnosis. [9]

Usage of plain or simple radiographs are not useful for diagnosing AP but can rule out differential diagnosis that mimic AP such as perforated duodenal ulcers.

Usage of ultrasonography in AP is limited by abdominal fat and ileus (increased intestinal gas). But USG is the research of choice investigation for pancreatitis induced by gallstones. The investigation of choice for AP is CECT of abdomen and it is gold standard.[17]

After 48 to 72 hours of acute pain abdomen doing CECT abdomen gives increased chances of diagnosing acute pancreatitis. [17]

Usage of multidetector CT scan involving contrast phase i.e. the portal venous phase after injecting contrast material, in 65 to 70 seconds leads to the assessment of viability of pancreatic parenchyma for detecting current pancreatic inflammation, collections, and free air intra-abdominally. [17,5.] Peak enhancement of normal pancreas proven is 50-80 HU. [14,15]

The clinical characteristics of AP in CECT is [18]

1. focal or diffuse enlargement of pancreas,
2. peripancreatic fat stranding
3. peripancreatic fascial thickening and
4. fluid collection

Survey of interest in AP - CECT (Sensitivity - 87%, Specificity - 90%) [19]

Balthazar introduced a classification scheme for AP to forecast the severity of the disease. Edematous pancreatitis and pancreatic necrosis were not correlated with the clinical result in the Balthazar classification system. Presence of organ failure, extra pancreatic complications such as ascites, pleural effusion, vascular complications such as splenic vein thrombosis were not included in Balthazar scoring system.[14,22,23]

Due to many limitations in the Balthazar scoring system, Mortele introduced another system using CT severity index to measure the accurate clinical scoring and outcome including the occurrence of secondary infections, organ failure, length of hospital stays and death [1,2,8,6,22,23].

OBJECTIVE

To evaluate acute pancreatitis and its correlation with the clinical outcome using MCTSI.

REVIEW OF LITERATURE

PANCREAS

Pan (all) + Kreas (flesh) derivative of Greek word. It is a retroperitoneal organ. Situated along the level of L1. Weighing around 75 to 125 grams and 10 to 20 CM in length. [10]

Pancreas is divided into[10]

1. Head – positioned in C shaped loop of duodenum, cited posteriorly to transverse mesocolon.
2. Neck - divides pancreas (two halves)
3. Body and tail – sprawls anterior to the splenic artery, vein. Peritoneum covers the anterior surface of the pancreas.

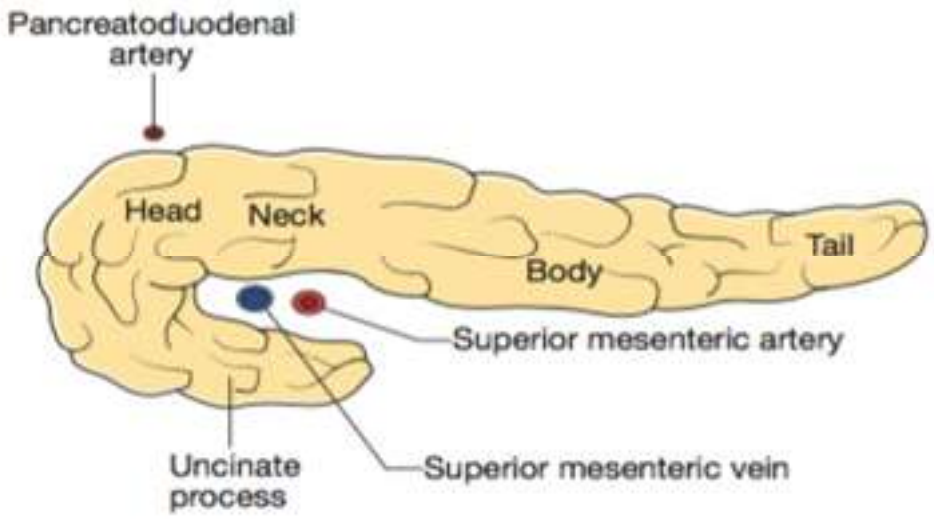


Fig 1. Division Of Pancreas

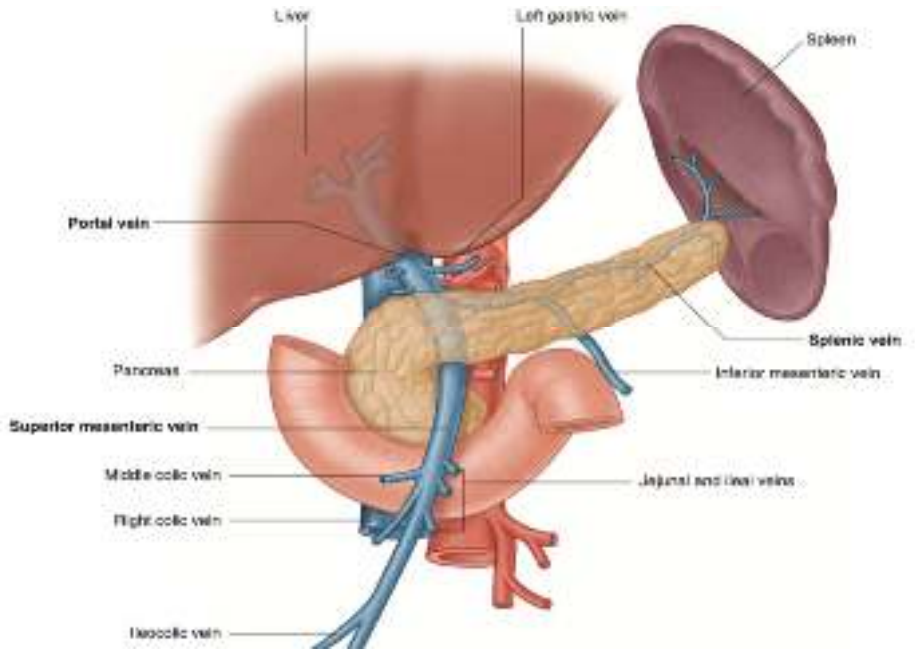


Fig 2. Anatomy Of Pancreas

Embryology [10]

Development begins at the 4th week of gestation.

Pancreas is endodermal in origin. Primitive endoderm gives rise to dorsal bud (DB) and ventral bud (VB). DB appears first.

1. DB blossoms into – Body, tail, neck, head of the pancreas.
2. VB is a part of hepatic diverticulum. Biliary tree formation and communication is maintained by the VB.

Around 4th to 8th week of the intrauterine life (IUL) VB rotates and fuses with the DB posteriorly in the clockwise fashion. At the completion of the 8th week of the intrauterine life both the dorsal and the ventral buds are fused together. Larger DB duct which arises from the duodenum forms the duct of Santorini or the minor pancreatic duct (PD). Small Ventral PD which arises from the hepatic diverticulum joints the CBD thus forming the Duct of wirsung or the main / major PD.

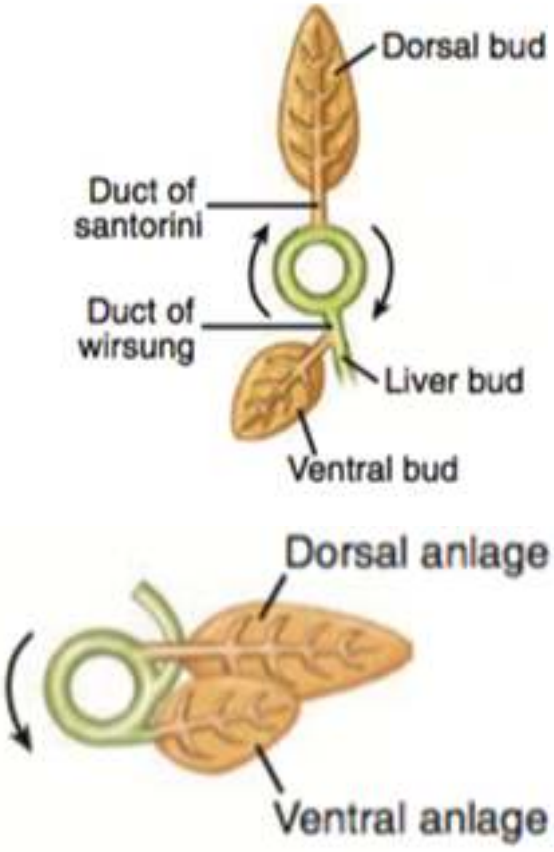


Fig 3. Embryology of pancreas

VB + CBD - Major PD. The preponderance of the pancreas drains through the main PD. Accessory PD - drains body and tail. Major pancreatic duct and the CBD empties to the ampulla of vater or major papilla located in the second part of the duodenum in the medial border. Pancreatic duct pressure is more - twice the CBD which prevents the reflex of bile juice into the pancreatic duct. The flow of the pancreatic juice and the biliary secretions into the duodenum are controlled by the muscle fibers around the major papilla that is sphincter of Oddi.

Accessory PD channels into the 2nd part of the duodenum - 2 cms proximate to the ampulla of vater.

Blood supply [10]

Celiac trunk and Superior mesenteric artery (SMA).

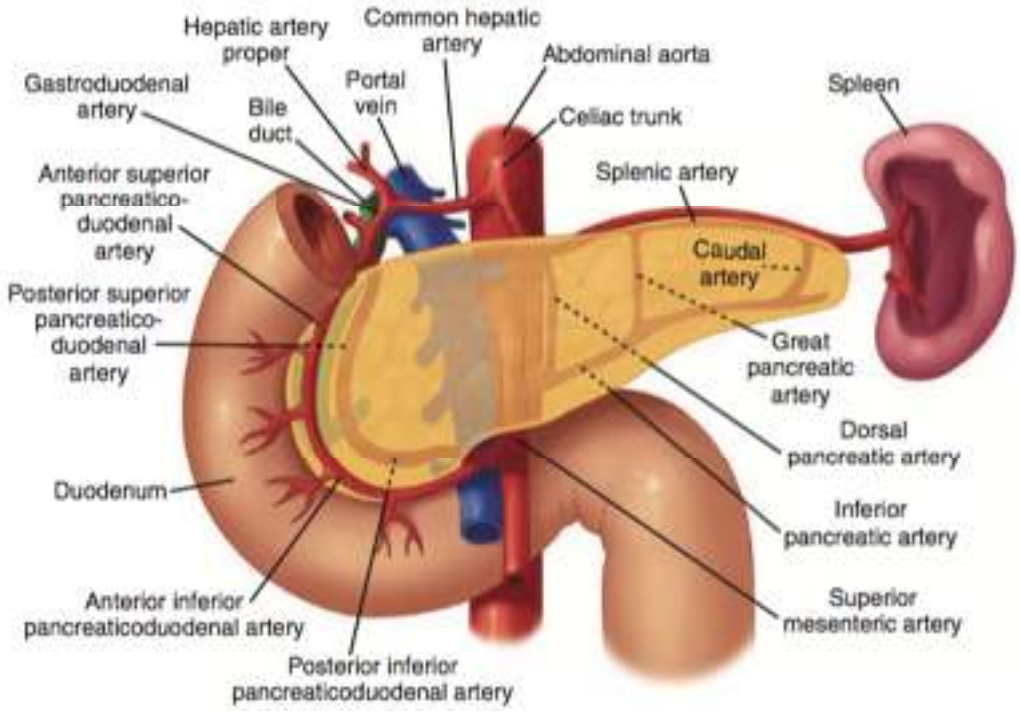


Fig 4. Blood supply of pancreas

1. Anterosuperior PDA (pancreaticoduodenal artery) - bestows blood to superior surface of the head, a divaricate of gastroduodenal artery (GDA)
2. Anteroinferior PDA - a divaricate of SMA, supplies anteroinferior surface.
3. Posterosuperior PDA - bestows superior posterior surface, a divaricate of GDA
4. Posteroinferior PDA - supplies inferior posterior surface, a branch of SMA
5. Splenic artery - Supplies body and tail
6. Superior PDA + inferior PDA - joins to complete the arcade.

Venous Drainage [10]

Main venous supply - Portal vein (PV) + superior mesenteric vein (SMV).

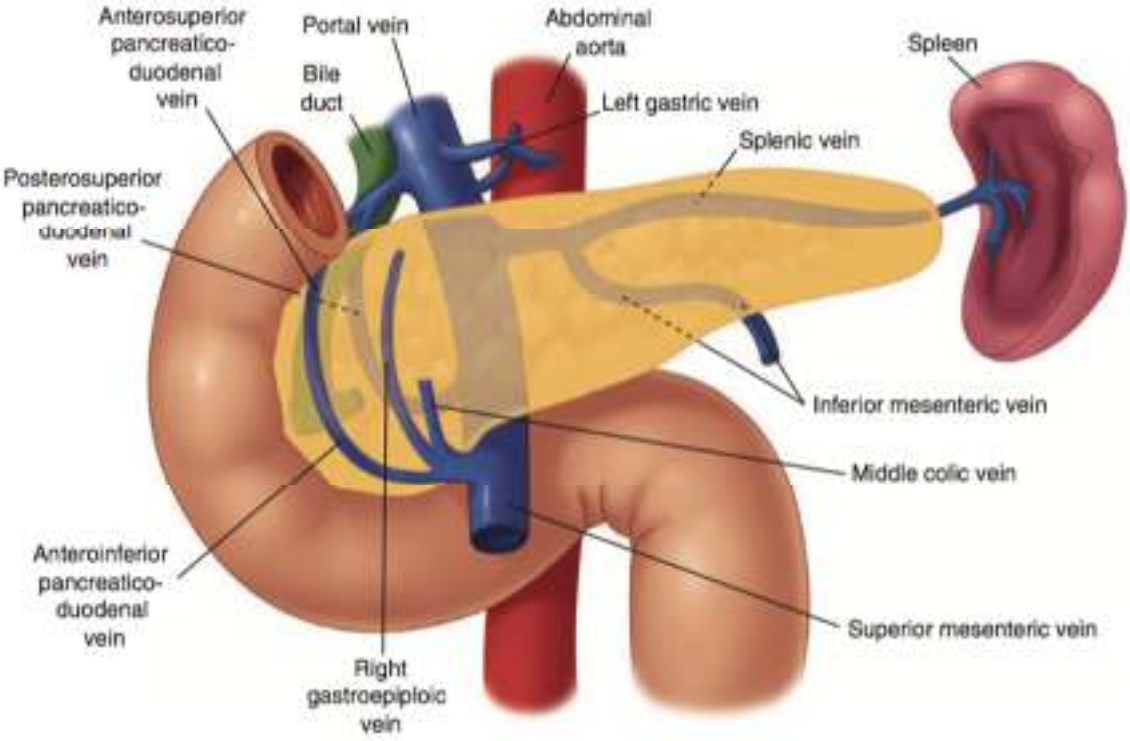


Fig 5. Venous drainage of pancreas

Anterior surface of Head

1. Antero-superior PD vein which further drains into PV
2. Antero-inferior PD vein + right gastroepiploic vein+ middle colic vein which further drains into the SMV.

Posterior Surface, Body and tail of the pancreas = inferior mesenteric vein and splenic vein drains into the PV.

Lymphatic drainage [10]

Widespread and diffuse.

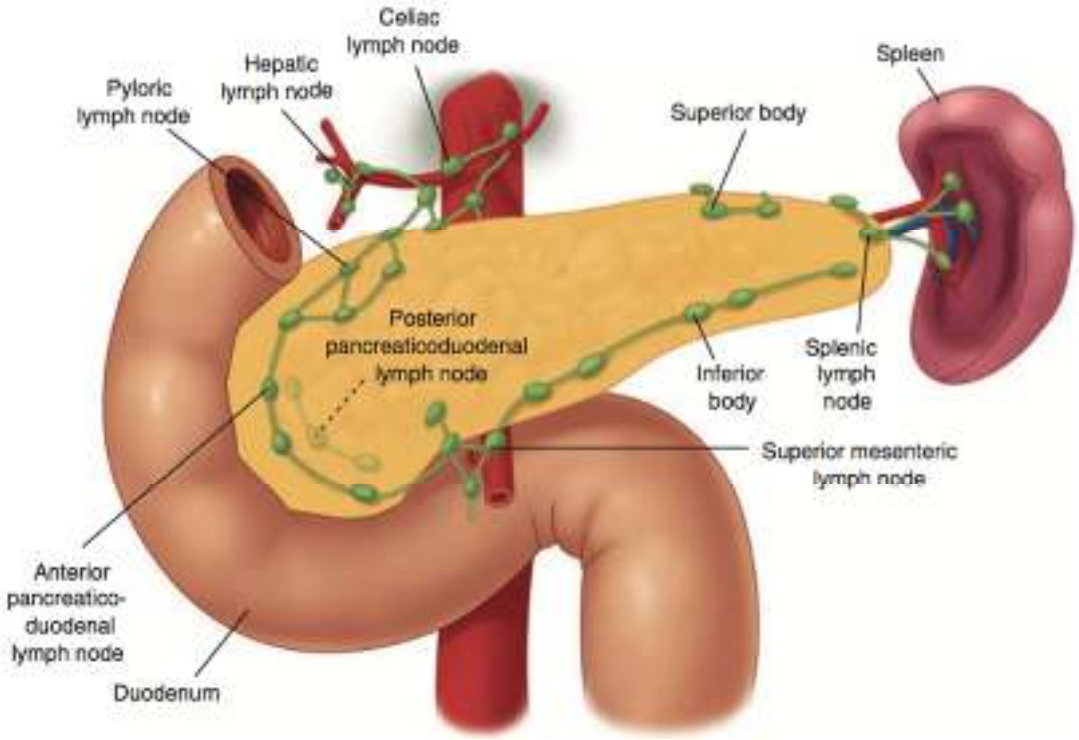


Fig 6. Lymphatic drainage of pancreas

Neuroanatomy [10]

- Sympathetic & parasympathetic nervous system.
- Sympathetic - inhibits the secretions
- Parasympathetic - stimulates the secretions.

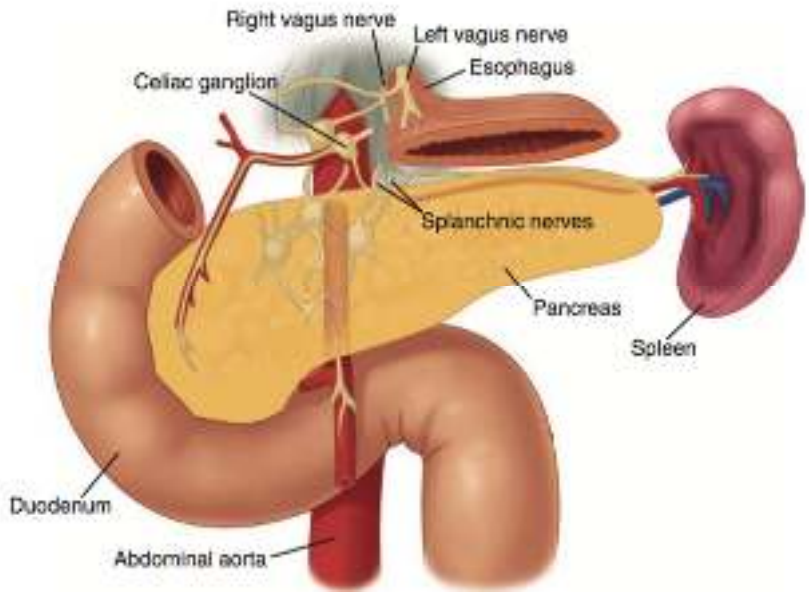


Fig 7. Neuroanatomy of pancreas

Physiology of pancreas

Exocrine function [36, 9,10]

- Colourless and odourless fluid secretion.
- 1500 to 2500 ml daily.
- Alkaline pH -8.0.
- Secreted by centriacinar and ductal cells.
- Pancreatic enzymes are influenced by parasympathetic supply.

Pancreatic secretion has two components [36, 9,10]

1. Water and the electrolyte secretion –
    a. The main ion is the bicarbonate.
    b. Synchronised by secretin - hormone which is liberated from duodenal mucosa.
    c. The stream of Pancreatic juice - stimulated by secretin.
    d. Rich in bicarbonate
    e. Vasoactive intestinal peptide (VIP) also synchronises water and bicarbonate secretion in the pancreas.
    f. VIP controls cyclic AMP which in turn stimulates bicarbonate secretion.
    g. Inhibited by - Glucagon, somatostatin and antidiuretic hormone.

2. Secretion of enzymes- Digestive enzymes synthesized in polysomes. Endoplasmic reticulum (ER) binds digestive enzymes. Ribosomes form the secretory proteins. These proteins are transferred to rough ER. These enzymes constitute the active and inactive forms. Inactive forms are chymotrypsinogen A and B, carboxypeptidase A and B, trypsin. Active forms are amylase, lipase, deoxyribonuclease and ribonuclease. These enzymes are activated in the duodenum. Trypsinogen is converted to trypsin by enterokinase. Trypsin in turn activates carboxypeptidase and chymotrypsinogen. Inside intestine proteins break down into peptides and amino acids by pancreatic proteolytic enzymes, fat is broken into glycerol and fatty acids by lipase, starch is degraded to disaccharides by amylase. [36, 9,10]

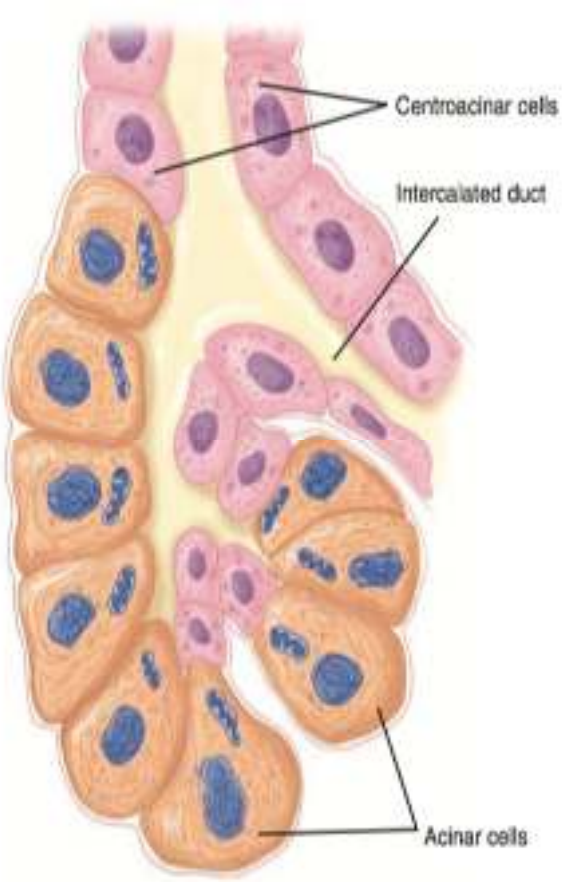


Fig 8. Histology of Pancreas

Stimulants of pancreatic secretion.[36, 9,10]

a. Pancreozymin,
b. Cholecystokinin,
c. Gastrin
d. Vagus nerves

Exocrine secretion of the pancreas[36, 9,10]

It has 4 phases

1. Cephalic phase - smell and sight of food initiates this stage by vagal stimulation resulting in secretion of enzyme rich juice in low volume.

2. Gastric phase - when meal reaches the stomach and causes gastric distension causes release of gastrin, which in turn stimulates the enzyme secretion by pancreas.
3. Intestinal phase - Synchronises secretin which controls pancreatic juice and bicarbonate secretion.
4. Postcibal phase - inhibition of pancreatic secretion.

Endocrine secretion of pancreas [36, 9,10]

Endocrine secretions of pancreas are mainly derived from islet cells of langerhans. There are about 1 million islet cells approximately.

Islet cells consists of

1. Alpha cells (20%) - constitute the outermost layer and secretes glucagon.
2. Beta cells (75%) - constitute the central layer and are the source of insulin.
3. Delta cells (5%) - constitute the intermediate layer – produces somatostatin and gastrin.
4. Pancreatic Polypeptide cells
    - Small in number
    - Scattered all over the pancreas.
    - They are responsible for secretion of VIP, pancreatic and gastrointestinal inhibitor polypeptide

ACUTE PANCREATITIS

Pancreatitis - Inflammation of pancreatic glandular parenchyma leading to injury / irreversible destruction of acinar components [9].

It is classified into Acute Pancreatitis (AP) or Chronic pancreatitis (CP)[9]

CP is attributed by [36, 9,10]

i. Fibrosis
ii. Calcification of the pancreatic gland.

AP completely resolves with no morphological, functional, or symptomatic sequelae. In the event of necrotizing pancreatitis, it further leads to scarring-strictures and wreckage of pancreatic function [36, 9,10].

ETIOLOGIES OF ACUTE PANCREATITIS
ALCOHOL
BILIARY TRACT DISEASE
HYPERLIPIDEMIA
HEREDITARY
HYPERCALCEMIA
TRAUMA • EXTERNAL • SURGICAL • ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATICOGRAPHY
ISCHAEMIA • HYPOPERFUSION • ATHEROEMBOLIC • VASCULITIS
PANCREATIC DUCT OBSTRUCTION • NEOPLASM • PANCREATIC DIVISUM • AMPULLARY AND DUODENAL LESIONS
INFECTION
VENOM
DRUGS
IDIOPATHIC

Table1. Etiology of Acute pancreatitis.

GALLSTONES [9]

Exploitation of alcohol and gallstones together add up to 60 to 80 percentage of AP.

All over the world, woman is more prone to biliary pancreatitis and men are more prone to develop alcoholic pancreatitis.

Acute biliary pancreatitis top cause is

1. Gallstones (4 to 8%)
2. Migratory gallstones.

ABP is dissimilar from acute alcoholic pancreatitis such as after betterment, the pancreatic gland is histologically normal after clinical recovery with return of normal exocrine and endocrine functions.

Pathogenesis of biliary pancreatitis is multifaceted such as due to obstruction of the ampulla of vater, reflection of bile into the PD and genetics. Gallstone ampullary obstruction aggravates biliary pancreatitis.

In absence of stone causing obstruction in the ampulla, inflamed ampulla leads to local edema or spasm of the ampulla which further leads to PD obstruction.

Obstruction is either mechanical or due to inflammation - leads to increased pressure in the PD, which causes alkaline pancreatic juice extravasation into the interstitium causing injury to the pancreatic gland. Meal intensifies the injury of the gland.

After a meal,

- there is increased secretion of pancreatic juice.
- In case of obstruction – there is further raise in pressure in PD leading to increased destruction of the gland.
- Pressure in PD – threefold > CBD.

The most common risk factors for ABP are

- Gallstones less than 5 mm in diameter
- Cystic duct size greater than 5 mm and
- Number of gallstones greater than 20.

ALCOHOL [9]

Acute alcoholic pancreatitis (AAP)

- Men are mostly affected
- Middle aged men (40 to 60 years)
- Average alcohol consumption - 100 to 150 gram per day in AP.

Alcohol ingestion is not directly proportional to the AAP. Recent studies shows that only less than half of heavy drinkers developed AAP.

Alcohol intake alone doesn't bring about to AAP but it proposes that it should be accompanied by genetic and other environmental factors.

Alcohol only sensitizes the pancreas while pancreatitis is initiated by general and environmental factors.

Excess alcohol intake forms intraductal calculi by increasing GP 2 and lithostathine production. Precipitation of GP2 and lithostathine forms calculi.

Smoking plays an important role in environmental factors.

HYPERTRIGLYCERIDEMIA [9]

1 to 10 % cause.

Pancreatic lipase hydrolyses triglycerides and releases free fatty acids inside pancreatic microvasculature in high concentrations. This accumulation promotes capillary injury and acinar cell injury leading to ischemia. Ischemia further leads to inflammation and necrosis.

HYPERCALCEMIA [9]

Rare cause – 1- 4 %

Hypercalcemia secondary to increased parathormone production leads to accumulation of calcium in the pancreatic duct which leads to stone formation and obstruction of PD.

Acute hypercalcemia is directly proportional to permeability of the PD which cause a leak and injury to the gland.

INBORN ERRORS OF METABOLISM [9]

Top Cause in neonates and paediatrics.

Familial disorders –

a. branched chain amino acid degradation,
b. glycogen storage disorders (Von Gierke),
c. hemolytic disorders such as acute intermittent porphyria, homocystinuria
d. several amino acid transporter defects.

AP is rare in these diseases and it is poorly understood.

DIALYSIS INDUCED AP IN CHRONIC RENAL FAILURE [9]

In chronic renal failure AP is seen with end-stage renal disease. Renal dysfunction confounds the diagnosis of AP by contributing pancreatic gland damage and by altering the levels of serum amylase and serum lipase estimation as a result of dialysis and uremia. 60 % of long-term dialysis patients shows pancreatic abnormalities on post-mortem studies.

AP incidence - Peritoneal dialysis > Hemodialysis.

Toxic substances in peritoneal dialysis dialysate alters parathormone and calcium levels for the leading to PD stone formation.

DRUG-INDUCED PANCREATITIS [9]

Rare - 0.1 to 2% of AP cases.

Mostly due to toxic metabolites released from drugs, which leads to accumulation in pancreas, constriction of pancreatic duct, localized angioedema and intra pancreatic thrombosis.

INFECTIOUS CAUSES [9]

Bacterial

- Ascending infection from the biliary tree
- Ascending infection from gastrointestinal tract,
- Lymphatic spread
- Hematogenous spread.

Organisms

- Mycoplasma - Most common and due to pancreas specific toxin production and auto immune-mediated response.
- Salmonella typhi
- Campylobacter jejuni
- Leptospira interrogans causes sporadic pancreatitis.

Viral

- Mumps infection - Due to MMR vaccination AP due to mumps is rarely reported.

- Hepatitis B - Associates with fulminant liver failure. Fulminant liver failure causes AP as a complication. AP is uncommon in non-fulminant viral hepatitis.

Fungal and parasitic causes

- Fungal - rare (aspergillus)
- Parasitic pancreatitis (ascaris lumbricoides) – Causes obstruction to the pancreatic duct.

IATROGENIC OR TRAUMATIC PANCREATITIS [9]

- Uncommon - due to retroperitoneal location.
- Incidence of blunt traumatic pancreatitis is less than 2 %. Incidence in penetrating pancreatitis is 12 to 30 %.
- Triad of abdominal pain, leucocytosis and elevated serum amylase levels.

Post ERCP pancreatitis is defined as increased abdominal pain which is clinically significant and similar to AP with elevation of pancreatic enzymes three times the normal limit within 24 hours after the procedure.

PEP is most common in 1 to 3% in patients undergoing diagnostic ERCP and 2 to 5 % in patients undergoing therapeutic ERCP.

CONGENITAL CAUSES [9]

Pancreatic divisum – Top congenital cause for pancreatitis. Due to defect in fusion of the ductal system of the pancreas, major pancreatic duct fuses with the minor papillae. Due to restricted flow in the minor papilla, there is pancreatic obstruction and hypertension leading to injury to pancreas, further causing pancreatitis.

TUMOUR [9]

Pancreatobiliary and periampullary tumours – AP is the index presentation. AP associated with loss of weight and loss of appetite and new onset diabetes mellitus above 40 years should be considered for tumour etiology.

Most common tumours to cause AP are mucinous cystic neoplasm, IPMN, islet cell tumour, ampullary tumours and pancreatic adenocarcinoma.

Benign tumours such as adenoma, lipoma arising near the major papilla can cause pancreatitis by ductal obstruction

GENETIC CAUSES [9]

Hereditary pancreatitis is caused by trypsinogen mutations (gain-of-function). Mutations such as SPINK1, anionic trypsinogen, PRSS1, CFTR, CTRC can cause AP by a variety of mechanisms causing hyperlipidemia and hypercalcemia.

IDIOPATHIC [9]

Pathological cause for AP is unidentifiable in 30 % of patients even after complete radiological and laboratory investigations. Such cases are grouped under idiopathic acute pancreatitis.

ASSESSMENT OF ACUTE PANCREATITIS [9,10,36]

- Clinical assessment
- Laboratory Investigations
- Radiological Imaging Modalities.

Clinical assessment [9,10,36]

1. Abdominal Pain (85% – 100%)
2. Nausea (90- 100 %)
3. Vomiting (90- 100 %)
4. Restlessness
5. Tachycardia
6. Tachypnea
7. Hypotension
8. Epigastric and Right hypochondriac Guarding
9. Grey Turner's sign
10. Cullen's sign

Laboratory Investigations [9,10,36]

1. Complete blood count
2. Urine analysis
3. Blood urea, creatinine
4. Random blood sugar
5. Triglycerides level
6. Arterial blood gases

Special indicators [9,10,36]

1. SERUM AMYLASE –

- Boosts within hours of inflammation
- Back to normal after 5 days
- Increased more than 3 times of normal.
- Nonspecific to pancreatitis.

Also increased in

- Perforated viscus,
- Acute appendicitis,
- Intestinal obstruction
- Mesenteric ischemia
- Biliary lithiasis.

Serum iso-amylase P has higher Specificity for AP.

2. SERUM LIPASE –

It is an exclusive enzyme of Pancreas. Hence more specific than amylase. It is reliable and is of great value in detecting AP.

3. LIVER FUNCTION TESTS –

Slight lift up in ALP and AST with elevation in total bilirubin can be seen. Marked elevation of AST and ALT are seen within 48 hours within onset differentiates biliary from non-biliary pancreatitis.

Radiological Imaging Modalities.

1. Plain X rays
2. USG Abdomen.
3. Computed tomography.
4. ERCP.
5. Endoscopic USG.
6. MRI.
7. MRCP.

Plain X ray Abdomen.

- Sentinel Loop Sign
- Colon cut off sign

Plain X ray Chest

- Pleural effusion
- Pneumonia
- Atelectasis
- Pulmonary edema

Trans abdominal ultrasonogram [9,10,36]

1. USG detects enlarged edematous pancreas, echogenicity of pancreas and free fluid cyst.
2. Detects - Pseudocyst and delineates abscess
3. To rule out Gall stones and choledocholithiasis.
4. Disadvantage - Difficulty in diagnosing obese patients and also due to bowel gases.

Computed Tomography of abdomen and pelvis[7,5,8,9]

Indication

a. Uncertain Diagnosis

b. Complication Assessment

In early stages, CT scan identifies

a. Early phase inflammation

b. Interstitial Edema

c. Fluid collection.

In later Stages, CT scan identifies

a. Pseudocyst of Pancreas

b. Acute necrotic Collection and Walled of Necrosis

Method of CT scan [1,2,7,8,9,10]

Unenhanced CT - detects extent of pancreatic and peri-pancreatic changes.

Disadvantage - unable to detect necrosis

Contrast enhanced CT - detects the extent of Necrosis.

Investigation of Choice in AP - CECT (contrast enhanced CT) [19].

Sensitivity - 87%

Specificity - 90%.

MRI of abdomen and pelvis

This test is not routinely done. It is done in certain cases to confirm diagnosis and also to detect complication of AP.

ERCP:

It used to detect microlithiasis, pancreatic duct strictures. Urgent ERCP is needed in biliary cases like biliary sepsis i.e., severe persistent jaundice, severe pancreatitis with obstruction and increased pain. Advantage –can be used in jaundice patients[10].

MRCP

a. It is a newer non-invasive technique - Also called as pancreatogram.
b. Advantage - It is error free in diagnosing severity of pancreatitis identifying pancreatic necrosis. It can also detect pancreatic and peri-pancreatic cyst [9,10].

TREATMENT OF AP [9,10,36]

1. MEDICAL MANGEMENT
2. SURGICAL MANGEMENT

MEDICAL MANAGEMENT

1. Fluid therapy – Mainstay in treatment of AP. Lack of fluid resuscitation leads to complications. 5-10ml/kg/hr. 3:1 (Crystalloids / colloids ratio). Adequate urine output indicates proper fluid therapy. To maintain haematocrit >35%.

2. Pain management –
    a. Opioid analgesics.
    b. Non-steroidal analgesics
    c. Thoracic epidural analgesics are used.

3. Nutrition- AP is a catabolic state. Nutrition requirement is high. Preferred is enteral route.
4. Antibiotics – Controversial. It is used to prevent secondary infections and abscess formation. Imipenam, Piperazillin tazobactum, 3rd generation cephalosporins are most commonly preferred as it has more affinity to cross blood-pancreatic barrier.
5. Inhibition of pancreatic secretions –
    a. Nasogastric suction,
    b. antacids,
    c. somatostatin analogues

SURGICAL MANAGEMENT [9,10]

- In gallstone Pancreatitis –
    - Early endoscopic papillotomy,
    - Interval Cholecystectomy,
    - CBD drainage.
- In Pancreatic Necrosis –

- Resection
- Pancreatic debridement
- Percutaneous necrosectomy

Table 2.- Complications of Acute Pancreatitis

THE COMPLICATIONS OF ACUTE PANCREATITIS	
LOCAL	FLUID COLLECTIONS
	PANCREATIC ASCITES/PLEURAL EFFUSION
	PANCREATIC PSEUDOCYST
	PANCREATIC NECROSIS
	INFECTED PANCREATIC ABSCESS
	HEMORRHAGE PSEUDOANEURYSM
REGIONAL	VENOUS THROMBOSIS
	PARALYTIC ILEUS
	INTESTINAL OBSTRUCTION
	INTESTINAL ISCHAEMIA/ NECROSIS
	CHOLESTASIS
SYSTEMIC	SYSTEMIC INFLAMMATORY RESPONSE SYNDROME
	MULTI-ORGAN-DYSFUNCTION SYNDROME
	ARDS/PULMONARY FAILURE
	RENAL FAILURE
	CARDIOVASCULAR COMPLICATIONS
	HYPOCALCEMIA
	HYPERGLYCEMIA

PROGNOSTIC INDICATORS

Goal in treating acute pancreatitis [8,9,10,18,28]

a. Early spotting of high-risk cases and initiate treatment.
b. Transfer the patient to tertiary care centre.
c. Shift the patient to ICU to minimize complications
d. To start antibiotics and parenteral nutrition.

Importance of scoring system:

AP is a self-limiting condition in 80% and in 20% complications occur. Complications leads to morbidity and mortality. Scoring systems are designed to predict the prognostic and clinical course of AP [8,3,9,18,28].

TABLE 3. Revised Atlanta Classification

MILD	• NO ORGAN FAILURE. • NO LOCAL OR SYSTEMIC COMPLICATIONS.
MODERATE	• ORGAN FAILURE THAT RESOLVES WITHIN 48 HOURS • LOCAL OR SYSTEMIC COMPLICATIONS WITH PERSISTENT ORGAN FAILURE
SEVERE	• PERSISTENT ORGAN FAILURE > 48 HOURS

Disadvantage [9]

1. Difficulty to differentiate between predicted and actual severity.
2. Failed to identify organ failure.

RANSONS CRITERIA [9]

Most common system used in AP. From admission to 48 hours, 11 significant criteria are assessed. Criteria are given in the following table.

Table 4. Ranson's criteria

AT ADMISSION	**AT 48 HOURS**
Age >55 years	Hematocrit (decrease >10%)
WBC > 16000/ml	BUN (increase >5mg/dl)
GLUCOSE > 200mg/dl	Calcium (>8 mg/dl)
LDH > 350 IU/ml	Pao2 (>60mmhg)
AST > 250 IU/ml	Base deficit (>4 mEq/L)
	Fluid sequestration (>6 L)

Demerits – Prognosis can be assessed only after 48 hours and is accurate only at the extremes of the scale and less at intermediate scores.

BALTHAZAR SCORING - CT SEVERITY INDEX [6,7,9,14,22,23]

First radiological scoring system was invented in 1985 (using non contrast CT). 1990 CT severity index was developed.

CTSI = Balthazar Score + Evaluation of Pancreatic Necrosis

Table 5. Balthazar Scoring – CTSI scoring

PANCREATIC INFLAMMATION	
Normal pancreas	0
Focal or diffuse enlargement of pancreas	1
Intrinsic pancreatic abnormalities with inflammatory changes in peripancreatic fat	2
Single, ill-defined fluid collection or phlegmon	3
Two or more poorly defined collections or presence of gas in or adjacent to the pancreas	4
PANCREATIC NECROSIS	
None	0
Less than or equal to 30%	2
Greater than 30 and less than, or equal to 50%	4
Greater than 50%	6

CTSI	**SCORE**
MILD	0 to 3
MODERATE	4 to 6
SEVERE	7 to 10

Pancreatic necrosis is a part of disease severity criteria. CTSI is used to detect mortality. Extra pancreatic complications are not included in Balthazar – CTSI which is a marked limitation in assessing pancreatic disease severity and clinical outcome. [6,7,9]

MORTELE – MODIFIED CT SEVERITY INDEX [8,25,29]

MCTSI differs from CTSI by including extra pancreatic complications and grading the pancreatic and peri-pancreatic fluid collection grading. MCTSI is a better evaluator of prognosis of AP patients. MCTSI is a good prognostic indicator in patients with end organ failure and assessing hospital duration[1].

Main disadvantage of MCTSI - inability to predict the early surgical intervention[8]

Table 6. Mortele – MCTSI scoring

PROGNOSTIC INDICATOR		POINTS
PANCREATIC INFLAMMATION	Normal pancreas	0
	Intrinsic pancreatic abnormalities with or without inflammatory changes in peripancreatic fat	2
	Pancreatic or peri-pancreatic fluid collection or peri-pancreatic fat necrosis	4
PANCREATIC NECROSIS	None	0
	Less than or equal to 30%	2
	Greater than 30%	4
EXTRA PANCREATIC COMPLICATIONS	One or more of the following - pleural effusion, ascites, vascular complications, parenchymal complications or gastrointestinal tract involvement	2

MODIFIED CTSI	SCORE
MILD	0 to 2
MODERATE	4 to 6
SEVERE	8 to 10

OTHERS

- C-reactive protein (CRP) assays.
- APACHE II score.
- Trypsinogen -activating peptide (TAP) assays.

Emil J. Balthazar[40] et al, in 2001 did a review on "Acute pancreatitis: assessment of severity with clinical and CT evaluation". After thorough multiple studies, they evaluated that the assessment of the severity of AP is based on clinical and laboratory findings and contrast-enhanced CT imaging. Numeric systems (APACHE II, Ranson) are commonly used to help detect organ failure, and the attained data are used as indirect suggestion of disease severity, with a sensitivity-70%. Use of individual risk factors determined with laboratory tests (markers of pancreatic injury and markers of inflammatory response) has been anticipated to help expect clinical outcome. They concluded that further clinical studies are needed in which CT findings (CT severity index) combined with a numeric system or with one or several laboratory markers may be useful to identify the severity of disease in patients with acute pancreatitis.

Casas JD, Díaz R[5] et al, in 2003 did a study titled "Prognostic value of CT in the early assessment of patients with acute pancreatitis". They assessed the prognostic value of CT in AP with 148 patients to find out indicator of prognosis in pancreatic necrosis. Within 72 hours of onset of symptoms of AP, both unenhanced and contrast study was done. CT grade was divided into mild and severe. All severe grade patients had complications and death occurred. CT grade showed a sensitivity- 100% and specificity - 61.6% for predicting morbidity and for predicting mortality showed a sensitivity - 100% and specificity - 56.9%. Necrosis detection on early CT

showed sensitivity – 53.3%and specificity - 90.2% for predicting morbidity whereas 75% sensitivity and 83.8% specificity for mortality prediction. Early unenhanced CT alone - a good indicator of severity of acute pancreatitis. For outcome prediction of AP - CT grade was sensitive. Pancreatic necrosis - estimated on early CECT and observed only in patients who had severe disease, was a specific predictor of morbidity and mortality. Main limitation of the study is that CECT is done only in severe grade patients [5].

K J Mortele[8] et al, in 2004 did a study titled "A Modified CT Severity Index for Evaluating Acute Pancreatitis: Improved Correlation with Patient Outcome". 266 AP patients were assessed to correlate patient outcome with the MCTSI. The MCTSI includes pancreatic necrosis, infection along with extra-pancreatic complications. They evaluated that the modified CT severity index relates much closer with patient outcome measures than the CT severity index.[8]

Kaya E, Dervişoğlu A, Polat C[37] et al, in 2007 did a study titled "Evaluation of diagnostic findings and scoring systems in outcome prediction in acute pancreatitis ". In 199 AP patients they evaluated and compared the parameters (clinical, biochemical) with scoring systems (Ranson, Imrie and APACHE Ⅱ) to predict patient outcome. On analysis they found out that the APACHE Ⅱ score, LDH, base excess and CT severity index have prognostic value and CRP is a reliable marker for predicting both mortality and morbidity[37].

Mäkelä JT, Eila H[6] et al, in 2007 did a study titled "Computed tomography severity index and C-reactive protein values predicting mortality in emergency and intensive care units for patients with severe acute pancreatitis". They compared retrospectively 68 patients with CTSI and CRP values, also the length of ICU stay concluded that

CECT is the imaging of choice and a valuable predictor in detecting necrosis and complications in AP [6].

Bollen TL, Singh VK[2] et al, in 2011 did a study titled "Comparative evaluation of the modified CT severity index and CT severity index in assessing severity of acute pancreatitis". In 397 AP patients they compared the CTSI and MCTSI. MCTSI had better correlation with clinical severity and higher accuracy in detecting the pancreatic infection and intervention. Clinical scoring systems accurately correlate with systemic complications and mortality. But radiologic scoring systems, more accurately diagnose clinical severity of disease and better correlate with pancreatic infection and intervention needs. The main limitation is that not all patients who were diagnosed with AP got contrast- enhanced CT or MRI done within 1 week of the onset of symptoms which was shared by many radiologic AP studies [2].

Piero Alberti, Elizabeth Pando[38] et al, in 2012 did a study titled "Evaluation of the modified CT severity index (MCTSI) and CT severity index (CTSI) in predicting severity and clinical outcomes in acute pancreatitis". In 149 acute pancreatitis patients APACHE-II, MCTSI, and CTSI were calculated. Severity parameters included persistent multi-organ failure, length of hospital stay, the need for intensive-care, death and local complications. The area under the curve (AUC) was evaluated and the scoring systems undertook a likely comparison. Both CTSI and MCTSI were linked significantly with all the evaluated severity parameters and exhibited an association between image severity and bad clinical outcomes.[38]

Sharma Vishal[39] et al, in 2015 did a study titled "Clinical outcomes and prognostic significance of early vs. late computed tomography in acute pancreatitis". They analysed 219 patient records retrospectively with 2 groups - the early CT group (CT done at 4–5 days after pain onset) and the late CT group (CT done in days 6–14 of

pain onset). The two groups were linked for variances in clinical outcomes and prognostic information gained from CT, such as detection of pancreatic necrosis and local complications, and CT severity index. The mean CTSI was higher in the late CT group (6.6562.27 vs. 5.5262.7; P=0.005). The incidence of persistent organ failure in the early group did not differ that of the late group (38.6% vs. 49.4%; P= 0.143). Even though carrying out early CT does not unfavourably affect the result in AP, CT carried out more than 5 days after the onset of symptoms may detect more local complications[39].

Prem Chand, Rommel Singh[4] et al, in 2017 did a study titled "Evaluation on the outcome of acute pancreas by Ranson's criteria and modified CT severity index". They compared 30 AP patient's outcomes with Ranson's Criteria and MCTSI. Their observation revealed that both scores are good in predicting patient outcome and systemic complications. MCTSI score showed significant difference in predicting local complications [4].

Sahu B, Abbey P[3] et al**,** in 2020 did a study titled "Severity assessment of acute pancreatitis using CT severity index and modified CT severity index: Correlation with clinical outcomes and severity grading as per the Revised Atlanta Classification" they observed in 120 patients of 20-60 years age to compare APACHE II and MCTSI they found out that the diagnostic accuracy in APACHE II was 91.67 percent and in MCTSI is 100%. Thus, MCTSI has better sensitivity and specificity than APACHE II [3].

MATERIALS AND METHODS

The present study was done at KLES Dr. Prabhakar Kore and M.R.C, Belagavi, in the Department of General Surgery on patients attending General Surgery OPD and casualty with pain abdomen and clinically suspected to have AP, diagnosed and confirmed by a Contrast enhanced CT scan of abdomen and pelvis, and getting admitted for the treatment between January 2020 to December 2020.

Study- Design

A Cross-sectional Study.

Selection criteria

Inclusion criteria

All patients with clinical / laboratory / ultrasonography diagnosis of acute pancreatitis, who were willing to undergo Contrast enhanced computed tomography

Exclusion criteria

- Chronic pancreatitis
- Cases not willing to undergo Contrast study.
- Cases with known history of allergy to iodinated contrast agents.
- Cases with deranged Renal function test (serum creatinine> 1.5 mg/dl).
- Pregnant Women.

Sample size

By using the formula for sample size calculation and based on the study[1], the average number of days of stay of the patient with severe acute pancreatitis in the hospital was 14.2 days.

Taking the prevalence as 14.2 days and by using the formula for sample size calculation of

P = 14.2

q = 100-P

= 100-14.2

= 85.8

d = absolute error of 9

By using the formula for sample size calculation of

$$\frac{4Pq}{d^2}$$

Figure has been rounded to 60. Thus, the sample size is 60.

With the Ethical Clearance being obtained from the Institutional Ethics Committee, Jawaharlal Nehru Medical College, Belagavi. After sorting out according to selection criteria, informed written consent taken on paper (Annexure I).

Patients with complaints of pain abdomen, were subjected to clinical examination and suspected acute pancreatitis patients were included in this study. Patient particulars, history, weight, occupation, vitals, investigations were noted. Results were plotted in a proforma.

All selected patients underwent CECT of abdomen and pelvis.

Procedure

- Patient with suspected AP were admitted and date of admission was noted.

- Informed consent including risk factors obtained from the patient and attenders.
- Patients with normal renal parameters were subjected to CECT abdomen and pelvis after 48-72 hours of the onset of the symptoms.

Assessment score was

PROGNOSTIC INDICATOR		**POINTS**
PANCREATIC INFLAMMATION	Normal pancreas	0
	Intrinsic pancreatic abnormalities with or without inflammatory changes in peripancreatic fat	2
	Pancreatic or peri-pancreatic fluid collection or peri-pancreatic fat necrosis	4
PANCREATIC NECROSIS	None	0
	Less than or equal to 30%	2
	Greater than 30%	4
EXTRA PANCREATIC COMPLICATIONS	One or more of the following - pleural effusion, ascites, vascular complications, parenchymal complications or gastrointestinal tract involvement	2

MODIFIED CTSI	SCORES
MILD	0 to 2
MODERATE	4 to 6
SEVERE	8 to 10

- With the help of radiologists MCTSI scoring was done.
- Pancreatic necrosis was graded morphologically i.e. No necrosis, necrosis < 30% or necrosis >30%.
- Patient undergoing any intervention (Abscess drainage and debridement) during admission period was followed and noted.
- Patient was followed till the date of discharge.
- Total number of Hospital stay was calculated and noted in the proforma.

Safety Considerations

In each step, patient safety was the top priority. All procedures were done only with either patient's or attender's consent.

Quality Assurance

Strict care was taken to ensure that the study was done in the best quality.

Expected Outcome

This study was basically done to test the effectiveness of MCTSI, as a predicting tool in clinical outcome of AP.

Statistical methods:

One way analysis of variance and Chi-square tests were used in the data analysis.

RESULTS

Final survey encompasses sixty (60) subjects.

Table (7): Descriptive-analysis of Gender in research section

GENDER	NUMBER	PERCENTAGE
FEMALE	12	20
MALE	48	80
TOTAL	60	100

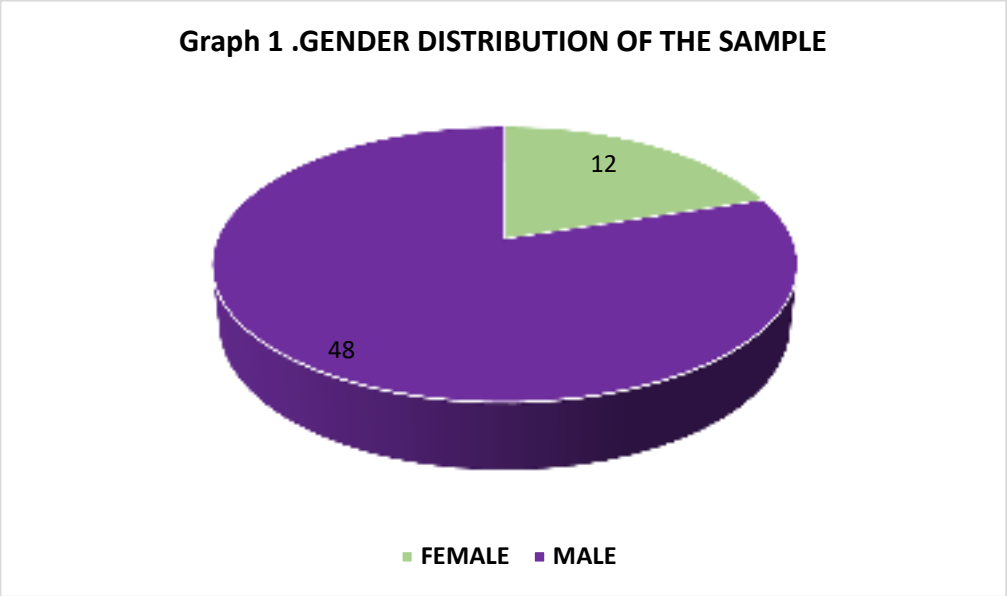


Table (8): Descriptive-analysis of gender with respect to severity in research section

	FEMALE	MALE	TOTAL
MILD	2	9	11
MODERATE	7	26	33
SEVERE	3	13	16
TOTAL	12	48	60

Majority of the patients were male, comprising 80% of overall patients. The Male: Female Ratio is 4:1.

Table (9): Descriptive-analysis of age (years) and number of hospital days in research section

	MEAN	S.D.	MINIMUM	MAXIMUM
AGE	39.88	15.39	16	77
NUMBER OF HOSPITAL DAYS	10.17	7.64	1	38
MCTSI -SCORE	5.23	2.33	2	10

The mean age was 39.88 ± 15.39 in the study population, ranging between 16 years to 77 years.

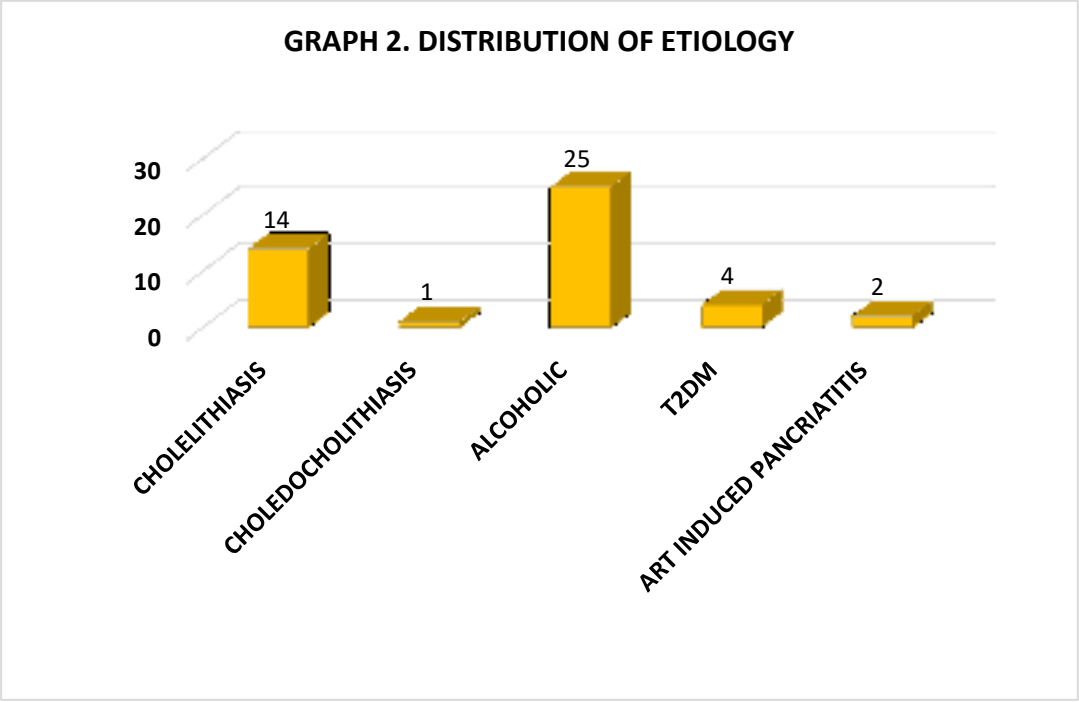
Table (10): Descriptive-analysis of age distribution and MCTSI scoring in the research section

	MILD	MODERATE	SEVERE
MEAN	43.09	39.70	38.06
S.D.	16.43	15.19	15.75
MINIMUM	16	16	19
MAXIMUM	77	76	72

Among the study population, mean age in mild score is 43.09 ± 16.43, moderate score is 39.40 ± 15.19, severe score is 38.06 ± 15.75. The p value is 0.7093 (NS). There is no significant difference with respect to age.

Table (11): Descriptive-analysis of Etiology

ETIOLOGY	NUMBER	%
CHOLELITHIASIS	14	23.33
CHOLEDHOCHOLITHIASIS	1	1.67
ALCOHOLIC	25	41.67
ART INDUCED PANCREATITIS	2	3.33



In our study, alcohol intake is the most common cause for AP with 25 patients (41.67). Followed by cholelithiasis 23.33%. ART induced pancreatitis was seen in 2 patients (3.33%), and choledocholithiasis is seen in 1 patient. It was found that cholelithiasis is the most common cause to AP in females.

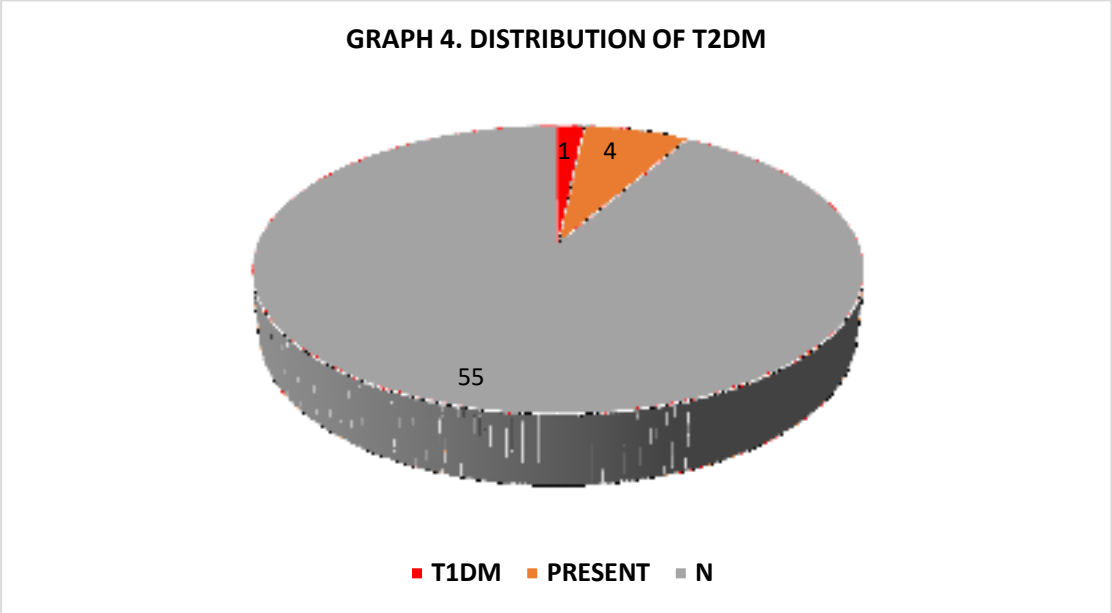
Table (12): Descriptive-analysis of Mortality

MORTALITY	NUMBER	%
SURVIVORS	58	96.67
EXPIRED	2	3.33
TOTAL	60	100.00

In our study two mortalities were observed due to ARDS (covid infection) and MODS. Both the mortality patients were in severe MCTSI score. Two COVID patients with AP were observed in our study during the pandemic. Mortality is 3.33% and survival rate is 96.67%.

Table (13): Descriptive-analysis of Diabetes Mellitus

T2DM	NUMBER	%
T1DM	1	1.67
PRESENT	4	6.67
N	55	91.67
TOTAL	60	100.00



In our study type 2 diabetes mellitus is observed in 4 patients (6.67%) and type 1 diabetes mellitus in 1 patient (1.67%).

Table (14): Descriptive-analysis of S. Amylase and MCTSI scoring in the research section

S. AMYLASE			
	MILD	**MODERATE**	**SEVERE**
MEAN	336.18	503.09	441.13
S.D.	356.62	510.94	452.14
MINIMUM	46	42	51
MAXIMUM	1026	1971	1305

The p value is 0.5933 (NS). There is no significant difference in serum amylase.

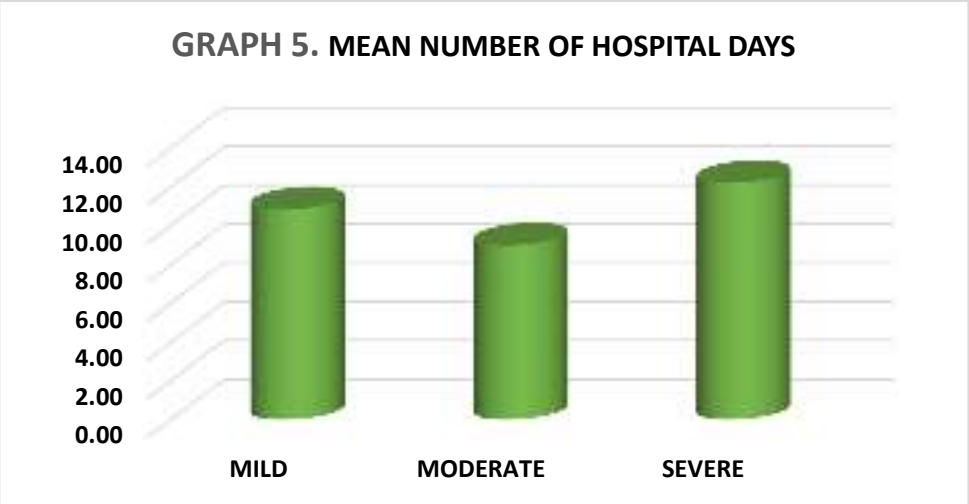
Table (15): Descriptive-analysis of S. Lipase and MCTSI scoring in the research section.

S. LIPASE			
	MILD	**MODERATE**	**SEVERE**
MEAN	404.55	639.42	373.56
S.D.	336.68	612.11	436.18
MINIMUM	26	12	33
MAXIMUM	1031	2355	1325

The p value is 0.1900 (NS). There is no significant difference in serum lipase.

Table (16): Descriptive-analysis of Number of hospital days and MCTSI scoring in the research section

NUMBER OF HOSPITAL DAYS			
	MILD	**MODERATE**	**SEVERE**
MEAN	10.82	8.97	12.19
S.D.	10.69	3.99	10.57
MINIMUM	4	2	1
MAXIMUM	36	19	38



On analysis with respect to severity, 11 patients are in mild score, 33 patients in moderate score, 16 patients in severe score. Mean duration of hospital stay is 10.17 ± 7.64 ranging from minimum stay of 1 day to maximum stay of 77 days. Mean duration of hospital stay in mild group is 10.82 days, moderate group is 8.97 days and severe group is 12.19 days. The p value of variance (ANOVA) for average number of hospital days is 0.3725 (NS). There is no significant difference in number of hospital days.

Table (17): Descriptive-analysis of complications in patients with AP.

PLUERAL EFFUSION	NUMBER	%
LEFT	2	3.33
RIGHT	1	1.67
BILATERAL	17	28.33
N	40	66.67
TOTAL	60	100.00

ASCITES	NUMBER	%
PRESENT	36	60.00
N	24	40.00
TOTAL	60	100.00

In our study, ascites was the most common complication present in 60% of the patients and pleural effusion was seen in 33.33% of the patients.

In our study, intervention in acute pancreatitis was not done in any patients.

DISCUSSION

In this study, MCTSI was assessed as a predictive tool for assessing the clinical outcome in acute pancreatitis patients.

It was conducted over a period of 12 months in Department of General Surgery, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi

The clinical diagnosis of AP is not only challenging but may mimic other conditions of acute abdomen as well, thereby making it risky to assess the prognosis and clinical outcome of the disease[3].

The important rationale for evaluating severity is clinical as mild patients respond well and severe patients has more guarded prognosis[8]. An ideal, easily applicable, widely available prognostic method is MCTSI that will help in severe patients to carefully monitor complications[8].

Among many prognostic indicators and criteria for AP, multiple studies showed MCTSI is a better scoring system in assessing clinical outcome for AP. Among the world-wide accepted scoring systems, presence of pancreatic inflammation and necrosis allows for grading of mild and severe pancreatitis and helps in evaluation of the prognosis[8].

Mortele[8] et al suggested a modified CTSI including extra pancreatic complications and proved MCTSI correlates more closely with patient outcome[8].

Bollen TL, Singh VK[2] et al observed 397 AP patients and compared CTSI and MCTSI. MCTSI had higher AUC in ICU stay and death. CTSI had higher AUC in organ failure, pancreatic infection and need for early intervention. MCTSI Showed better correlation with clinical severity and higher accuracy in detecting the pancreatic infection and intervention[2].

Prem Chand, Rommel Singh[4] et al compared 30 AP patient's outcomes with Ranson's Criteria and MCTSI and observed both scores are good in predicting patient outcome and systemic complications. Though many local complications were seen in higher Ranson's score but the values were statistically non-significant. MCTSI score showed significant difference in predicting local complications[4]. Hence MCTSI has been chosen as the method of choice in our study.

Banday IA, Gattoo I[1] et al compared MCTSI with clinical outcome such as development of infection, occurrence of organ failure, length of hospital stay and overall mortality. In our study, we have taken only the length of hospital stay as an indicator for clinical outcome[1,7,8]. In 50 patients showed average hospital stay in mild patients was 1.5 days, moderate patients were 6.9 days and severe patients was 14.2 days[1].

Raghuwanshi S, Gupta R[7] et all observed in 50 patients that mild patients is 9 days, moderate patients is 22 days and severe patients is 29 days[7].

Mortele KJ[8] et al observed in 266 patients that mild patients were 3 days, moderate patients were 09 days and severe patients was 11 days (p=0.035). However, using CTSI there is no difference in hospital duration between moderate and severe cases (p=0.15)[8].

The findings of above-mentioned studies[1,2,4,7,8] is comparable with our study.

In our study total of random 60 patients with pain abdomen and diagnosed to have acute pancreatitis were radiologically categorized as mild, moderate and severe using modified CT severity index and evaluated for clinical outcome with length of hospital stay. On analysis with respect to severity, 11 patients were in mild score, 33 patients in moderate score, 16 patients in severe score. Mean duration of hospital stay is 10.17 ± 7.64 ranging from minimum stay of 1 day to maximum stay of 77 days.

Mean duration of hospital stay in mild group was 10.82 days, moderate group was 8.97 days and severe group was 12.19 days (p=0.3725). Among 60 patients, no patients underwent any interventional procedures.

Our study differed from these studies. Other studies showed the grading of hospital stay as per scoring system and lacks the accuracy of the hospital stay. In our study, hospitalization is almost same in mild and severe patients. There is no difference in the hospitalization of either mild or severe cases. In our Study we have observed that, duration of hospitalization differs from patient to patient and patient's response to treatment and it does not follow the MCTSI grading as such mild patients stay for lesser time and severe patients stay for longer time[7].

No intervention was observed in our study.

Limitations - In our study, we observed only a small portion of total AP in a limited time period. This study was conducted in a tertiary care hospital, which may not be a complete representation of the overall population. Duration of stay is not differentiated into ICU stay and ward stay.

MCTSI is an excellent scoring system for AP, as it establishes the disease extent and severity scoring[1]. In our study, we did not find a statistical correlation of MCTSI with clinical outcome (length of hospital stay).

CONCLUSION

In this study on 60 patients to evaluate whether MCTSI can be utilized as an important aid in assessing clinical outcome of AP, it was observed that the duration of hospitalisation is not influenced by MCTSI. Duration of Hospitalisation differs from patient to patient and their response to treatment and does not follow the MCTSI grading as such mild patients stay for lesser time and severe patients stay for longer time. We conclude that the MCTSI scoring does not influence length of hospitalisation.

SUMMARY

Acute pancreatitis (AP), is an inflammatory disease which can be self-limiting edematous disease or a serious illness leading to such complications such as necrosis / abscess formation leading to multi-organ failure and mortality.

The clinical diagnosis of AP is not only challenging but may mimic other conditions of acute abdomen as well, thereby making it risky to assess the prognosis and clinical outcome of the disease

This study aimed to determine MCTSI as a reliable clinical tool in evaluating clinical outcome in a case of AP.

In the past year, cross sectional study was conducted in the Dept. of General Surgery, KLES Dr. P.K. and M.R.C, Belgaum. from January 2020 to December 2020.

A total of 60 patients with pain abdomen and diagnosed to have acute pancreatitis were radiologically categorized as mild, moderate and severe using modified CT severity index and evaluated for clinical outcome with length of hospital stay.

BIBLIOGRAPHY

1. Banday IA, Gattoo I, Khan AM, Javeed J, Gupta G, Latief M. Modified computed tomography severity index for evaluation of acute pancreatitis and its correlation with clinical outcome: a tertiary care hospital based observational study. Journal of clinical and diagnostic research: JCDR. 2015 Aug;9(8):TC01.
2. Bollen TL, Singh VK, Maurer R, Repas K, Van Es HW, Banks PA, Mortele KJ. Comparative evaluation of the modified CT severity index and CT severity index in assessing severity of acute pancreatitis. American Journal of Roentgenology. 2011 Aug;197(2):386-92.
3. Sahu B, Abbey P, Anand R, Kumar A, Tomer S, Malik E. Severity assessment of acute pancreatitis using CT severity index and modified CT severity index: Correlation with clinical outcomes and severity grading as per the Revised Atlanta Classification. The Indian journal of radiology & imaging. 2017 Apr;27(2):152.
4. Prem Chand, Rommel Singh, DP Singh, Nisha Rani. Evalua on of the outcome of acute pancreas by Ranson's criteria and modified CT severity index. International Journal of Contemporary Medicine Surgery and Radiology. 2017;2 (2):58-61.
5. Casas JD, Díaz R, Valderas G, Mariscal A, Cuadras P. Prognostic value of CT in the early assessment of patients with acute pancreatitis. American Journal of Roentgenology. 2004 Mar;182(3):569-74.
6. Mäkelä JT, Eila H, Kiviniemi H, Laurila J, Laitinen S. Computed tomography severity index and C-reactive protein values predicting mortality in emergency and intensive care units for patients with severe acute pancreatitis. The American journal of surgery. 2007 Jul 1;194(1):30-4.

7. Raghuwanshi S, Gupta R, Vyas MM, Sharma R. CT evaluation of acute pancreatitis and its prognostic correlation with CT severity index. Journal of clinical and diagnostic research: JCDR. 2016 Jun;10(6):TC06.
8. Mortele KJ, Wiesner W, Intriere L, Shankar S, Zou KH, Kalantari BN, Perez A, VanSonnenberg E, Ros PR, Banks PA, Silverman SG. A modified CT severity index for evaluating acute pancreatitis: improved correlation with patient outcome. American Journal of Roentgenology. 2004 Nov;183(5):1261-5.
9. Jarnagin, W.R.. (2016). Blumgart's Surgery of the Liver, Biliary Tract and Pancreas: Sixth Edition. 10.1016/C2013-1-19523-8.
10. *Schwartz's Principles of Surgery, 11e* Eds. F. Charles Brunicardi, et al. McGraw Hill,
2019, https://accesssurgery.mhmedical.com/content.aspx?bookid=2576&sectionid=208294867.
11. Whitcomb DC. Clinical practice. Acute pancreatitis. *N Engl J Med*. 2006;354(20):2142-50.
12. Lin A, Feller ER. Pancreatic carcinoma as a cause of unexplained pancreatitis: Report of ten cases. *Ann intern Med*. 1990;113:166-67.
13. Redman HC. Standard Radiological Diagnosis and CT scanning in pancreatic cancer. *Cancer*. 1981;47:1656-61.
14. Balthazar EJ, Robinson DL, Megibow AJ, et al. Acute pancreatitis: value of CT in establishing prognosis. *Radiology*. 1990;174(2):331-36.
15. Shankar S, van Sonnenberg E, Silverman SG, et al. Imaging and percutaneous management of acute complicated pancreatitis. *Cardio vasc Intervent Radiol*. 2004;27(6):567-80.
16. Banks PA, Freeman ML. Practice guidelines in acute pancreatitis. *Am J*

Gastroenterol. 2006;101(10):2379-400.

17. Isenmann R, Buchler M, Uh lW, et al. Pancreatic necrosis: an early Finding in severe acute pancreatitis. *Pancreas.* 1993;8(3):358-61.
18. Paspulati RM. Multidetector CT of pancreas. *Radiology clinics of North America.* 2005;43:999-1020.
19. Yassa NA, Agostini JT, Ralls PW. Accuracy of CT in estimating extent of pancreatic necrosis. *Clinical imaging*. 1997;21:407-10.
20. Alhajeri A, Erwin S. Acute pancreatitis: value and impact of CT severity index. *Abdom Imaging*. 2008;33:18-20.
21. Bastera G, Alvarez M, Marcaide A, et al Acute pancreatitis: Evaluation of the prognostic criteria of latest Balthazar tomographic classi cation. *Rev EspEnferm Dig.* 1999;91:433-38.
22. Balthazar EJ, Ranson JH, Naidich DP, et al. Acute pancreatitis: prognostic value of CT. *Radiology.* 1985;156(3):767-72.
23. Balthazar EJ, Freeny PC, Van Sonnenberg E. Imaging and intervention in acute pancreatitis. *Radiology*. 1994;193(2):297–306.
24. Lankisch PG, Struckmann K, Assmus C, et al. Do we need a computed tomography examination in all patients with acute pancreatitis within 72 h after admission to hospital for the detection of pancreatic necrosis *Scand J Gastroenterol*. 2001;36(4):432-36.
25. Mortele KJ, Mergo P, Taylor H, et al. Renal and perirenal space involvement in acute pancreatitis: state-of-the-art spiral CT findings. *Abdom Imaging.* 2000;25:272–78.

26. Wiesner W, Studler U, Kocher T, et al. Colonic involvement in non-necrotizing acute pancreatitis: correlation of CT findings with the clinical course of affected patients. *Eur Radiol.* 2003;13:897–902.

27. Chamisa I, Mokoena T, Luvhengo TE. Changing Pattern of Incidence, Aaetiology and Mortality from Acute Pancreatitis at Kalafong Hospital, Pretoria, South Africa, 1988-2007: A Retrospective Evaluation. *East and Central African Journal of Surgery.* 2010;15 (1):35-39

28. Lecesne R, Tourel P, Bret PM, et al. Acute pancreatitis: interobserver agreement and correlation of CT and MR cholangiopancreatography with outcome. *Radiology.* 1999;211(3):727–35.

29. Mortele KJ, Mergo PJ, Taylor HM, et al. Peripancreatic vascular abnormalities complicating acute pancreatitis: contrast-enhanced helical CT ndings. *Eur J* 15. *Radiol.* 2004;52(1):67-72

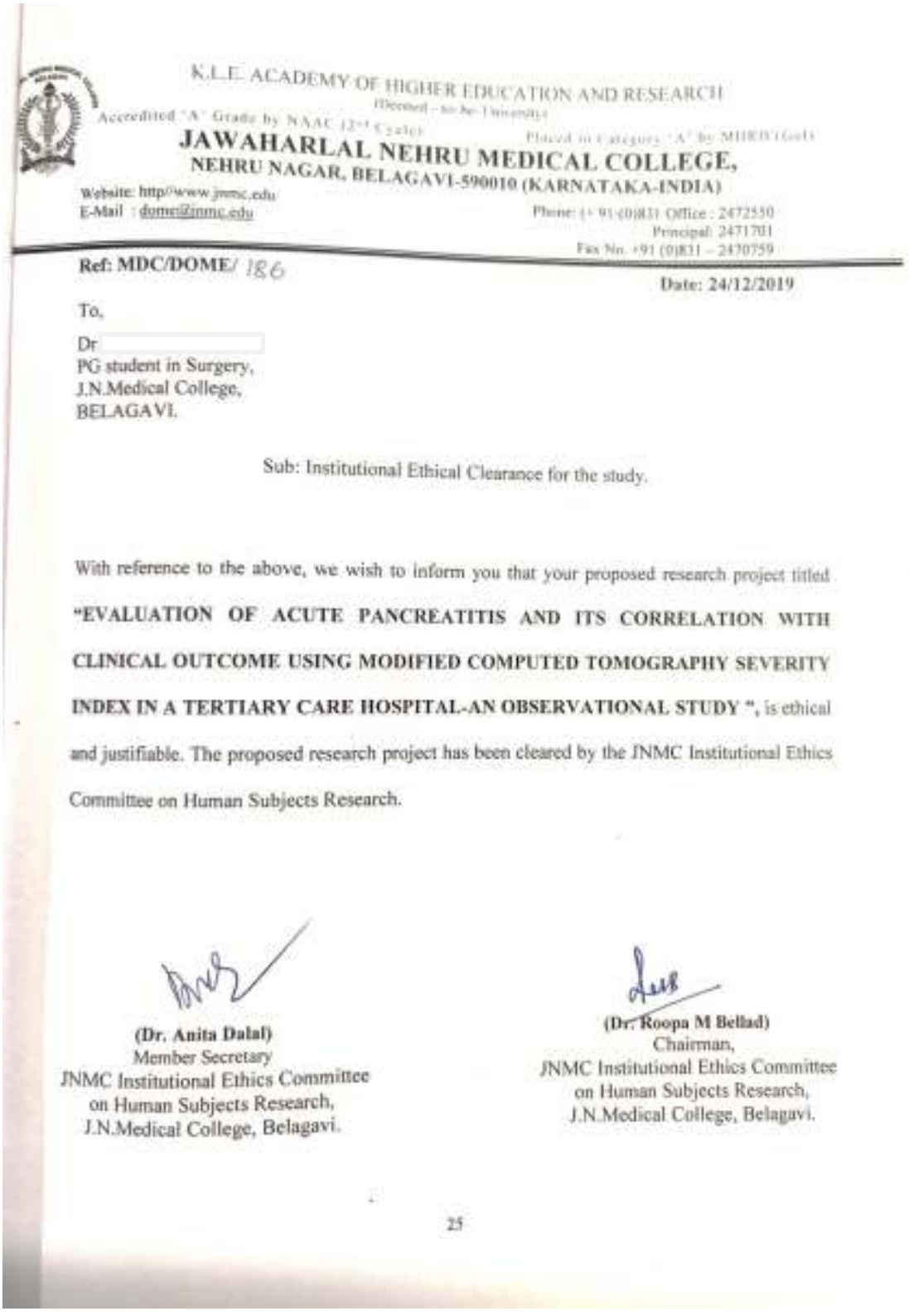
30. Triantopoulou C, Lytras D, Maniatis P, et al. Computed tomography versus Acute Physiology and Chronic Health Evaluation II score in predicting severity of acute pancreatitis: a prospective, comparative study with statistical evaluation. *Pancreas*. 2007;35(3):238-42.

31. Thomas BL, Singh VK, Maurer R, et al. Comparative evaluation of the modified CT severity index and CT severity index in assessing severity of acute pancreatitis. *AJR.* 2011;197:386–92.

32. De Waele JJ, Delrue L, Hoste EA, et al. Extrapancreatic inflammation on abdominal computed tomography as an early predictor of disease severity in acute pancreatitis: evaluation of a new scoring system. *Pancreas.* 2007;34(2):185-90.

33. Knoep i AS, Kinkel K, Berney T, et al. Prospective study of 310 patients: can early CT predict the severity of acute pancreatitis *Abdom Imaging*. 2007;32(1):111- 15
34. Munoz-Bongrand N, Panis Y, Soyer P, et al. Serial computed tomography is rarely necessary in patients with acute pancreatitis: a prospective study in 102 patients. *J Am Coll Surg*. 2001;193(2):146-52.
35. Ju S, Chen F, Liu S, Zheng K, et al. Value of CT and clinical criteria in assessment of patients with acute pancreatitis. *Eur J Radiol*. 2006;57(1):102-07.
36. S.Das ; A concise textbook of surgery (2020). ISBN : 9788190568128
37. Kaya E, Dervişoğlu A, Polat C. Evaluation of diagnostic findings and scoring systems in outcome prediction in acute pancreatitis. World journal of gastroenterology: WJG. 2007 Jun 14;13(22):3090.
38. Alberti P, Pando E, Mata R, Vidal L, Roson N, Mast R, Armario D, Merino X, Dopazo C, Blanco L, Caralt M. Evaluation of the modified computed tomography severity index (MCTSI) and computed tomography severity index (CTSI) in predicting severity and clinical outcomes in acute pancreatitis. Journal of Digestive Diseases. 2021 Jan;22(1):41-8.
39. Sharma V, Rana SS, Sharma RK, Gupta R, Bhasin DK. Clinical outcomes and prognostic significance of early vs. late computed tomography in acute pancreatitis. Gastroenterology report. 2015 May 1;3(2):144-7.
40. Balthazar EJ. Acute pancreatitis: assessment of severity with clinical and CT evaluation. Radiology. 2002 Jun;223(3):603-13.

ANNEXURE I. ETHICAL CLEARANCE.



K.L.E. ACADEMY OF HIGHER EDUCATION AND RESEARCH
(Deemed – to-be-University)
Accredited 'A' Grade by NAAC (2nd Cycle) Placed in Category 'A' by MHRD (GoI)
JAWAHARLAL NEHRU MEDICAL COLLEGE,
NEHRU NAGAR, BELAGAVI-590010 (KARNATAKA-INDIA)
Website: http://www.jnmc.edu
E-Mail : dome@jnmc.edu
Phone: (+ 91-(0)831 Office : 2472550
Principal: 2471701
Fax No. +91 (0)831 – 2470759

Ref: MDC/DOME/ 186

Date: 24/12/2019

To,

Dr
PG student in Surgery,
J.N.Medical College,
BELAGAVI.

Sub: Institutional Ethical Clearance for the study.

With reference to the above, we wish to inform you that your proposed research project titled **"EVALUATION OF ACUTE PANCREATITIS AND ITS CORRELATION WITH CLINICAL OUTCOME USING MODIFIED COMPUTED TOMOGRAPHY SEVERITY INDEX IN A TERTIARY CARE HOSPITAL-AN OBSERVATIONAL STUDY "**, is ethical and justifiable. The proposed research project has been cleared by the JNMC Institutional Ethics Committee on Human Subjects Research.

(Dr. Anita Dalal)
Member Secretary
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

(Dr. Roopa M Bellad)
Chairman,
JNMC Institutional Ethics Committee
on Human Subjects Research,
J.N.Medical College, Belagavi.

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ANNEXURE II

INFORMED CONSENT

• Objective/ Purpose of the study:

We are requesting you to enroll in study titled "EVALUATION OF ACUTE PANCREATITIS AND ITS CORRELATION WITH CLINICAL OUTCOME USING MODIFIED COMPUTED TOMOGRAPHY SEVERITY INDEX IN A TERTIARY CARE HOSPITAL AN OBSERVATIONAL STUDY"

This study will be conducted by DR._____, Post Graduate in M.S. General Surgery under the guidance of DR._____. Associate Producer, Department of General surgery, J.N. Medical College, KAHER, Belagavi.

Respected Sir/Madam, we request you to enroll yourself to participate in our study as you are eligible for the same. Your participation in this research is voluntary. Your decision whether or not to participate in the study will not affect your relationship with J. N. Medical College or KLEH. If you decide not to participate you are free to withdraw at any time.

• Procedure Involved:

Informed consent will be taken from all patients involving the study.

- The patient who have been diagnosed as Acute Pancreatitis should undergo contrast enhanced computed tomography and scored with Mortele severity index and correlated with the length of hospital stay.

Assessment:-

Prognostic Indicator		Points
Pancreatic Inflammation	Normal pancreas	0
	Intrinsic pancreatic abnormalties with or without inflammatory changes in peripancreatic fat.	2
	Pancreatic or peripancreatic fluid collection or peripancreatic fat necrosis	4
Pancreatic Necrosis	None	0
	≤ 30%	2
	> 30%	4
Extra Pancreatic Complications	One or more of following: Pleural Effusion, ascites, vascular complications, parenchymal complications, or gastrointestinal tract involvement.	2

[Table/Fig-2]: Mortele Modified CTSI Scoring (2004)

- BENEFITS: The benefits of the procedure under study is to assess the potency of the MCTSI in treatment of acute pancreatitis

- RISKS: There is no additional risk compared to the standard treatment.

• Alternatives:

Even if you decline your participation in the study, the course of your treatment will not be affected. You are free to withdraw from the study at any point of time. Everything about the study will be explained in detail to you before giving consent for the same.

• Privacy and Confidentiality:

The only people to know that you are a research subject are members of the research team. No information about you or information provided by you during the research will be disclosed to other without your written permission except:

1. In emergency to protect your rights and welfare.

2. If required by law.

• Institutional policy:

In the event of injury related to the study, treatment will be made available through KLES' Hospital & MRC, Belagavi. There is no compensation or payment for such medical treatment by law.

• Financial Incentives for participation:

No financial incentives are being offered to enrolled patients. It is purely being done with the idea of research and all the cost of the study will be borne by the investigator.

• Authorization to Publish Results:

When the results of the research are published or discussed, in a conference, no information will be displayed that would disclose your identity. Any information that is obtained in connection with this study and that can be identified with your identity remaining confidential.

Consent statement:

Mode of communication of consent form : Verbal / Written

Contents : Self Read/ Read out by Investigator

Participant's awareness regarding voluntary withdrawal from study : Yes / No

Investigators decision to remove participants from study : Yes / No

Awareness regarding voluntary participation: Yes/ No

Adequate time given to clarify any doubts about the study or rights a study participant: Yes/ No

n case they have any questions related to the study, in future or in case of study related injury or illness, they can contact DR._____, Department of General Surgery, KLES Hospital and MRC, Belagavi, Phone number- or DR. _____ M.S., Associate Proffesor, Dept. Of General Surgery, KLE'S Hospital and MRC, Belagavi Phone number:

If they have any queries about their rights as a study subject, they may call DR. ROOPA BELLADM.D., Chairman, and Ethical Committee for Human Subjects Research. Professor, Department of Paediatrics, J. N. Medical College, Belagavi, Phone number-9448113403.

Signature or left thumb print of participant or legally authorized representative

Participant's name. _____

Participant's signature/thumb print _____

Experimenters' name _____

Experimenters' signature _____

Witness' name_____

Witness' signature _____

Date _____

ANNEXURE III

PROFORMA

PROFORMA OF CLINICAL EXAMINATION OF INDIVIDUAL PATIENT

Name : Age :

Address : IP no.:

Sex : Religion:

Education: Date of admission:

Occupation: Date of discharge:

HISTORY

Clinical history:

Other associated illness:

GENERAL PHYSICAL EXAMINATION:

Built and Nourishment:

Weight:

Pallor/Icterus/Cyanosis/Clubbing/Edema/Lymphadenopathy

Vitals Signs: PR: /min BP: mmhg RR: /min Temp:

Systemic Examination

PA –

RS-

CVS –

CNS -Investigations

S. Amylase

S. Lipase

USG -

CECT Scan –

MODIFIED CT SEVERITY INDEX

	PROGNOSTIC INDICATOR	POINTS	SCORE OF THE PATIENT
PANCREATIC INFLAMMATION	NORMAL PANCREAS	0	
	INTRINSIC PANCREATIC ABNORMALITIES WITH OR WITHOUT INFLAMMATORY CHANGES IN PERIPANCREATIC FAT	2	
	PANCREATIC OR PERIPANCREATIC FLUID COLLECTION OR PERIPANCREATIC FAT NECROSIS	4	
PANCREATIC NECROSIS	NONE	0	
	<30%	2	
	>30%	4	
EXTRA PANCREATIC COMPLICATIONS	ONE OR MORE OF THE FOLLOWING – PLEURAL EFFUSION,ASCITES, VASCULAR COMPLICATIONS, PARENCHYMAL COMPLICATIONS OR GASTROINTESTINAL TRACT INVOLVEMENT	2	

Total Score -

Modified CTSI score – Mild / Moderate / Severe

DATE OF ADMISSION –

DATE OF DISCHARGE –

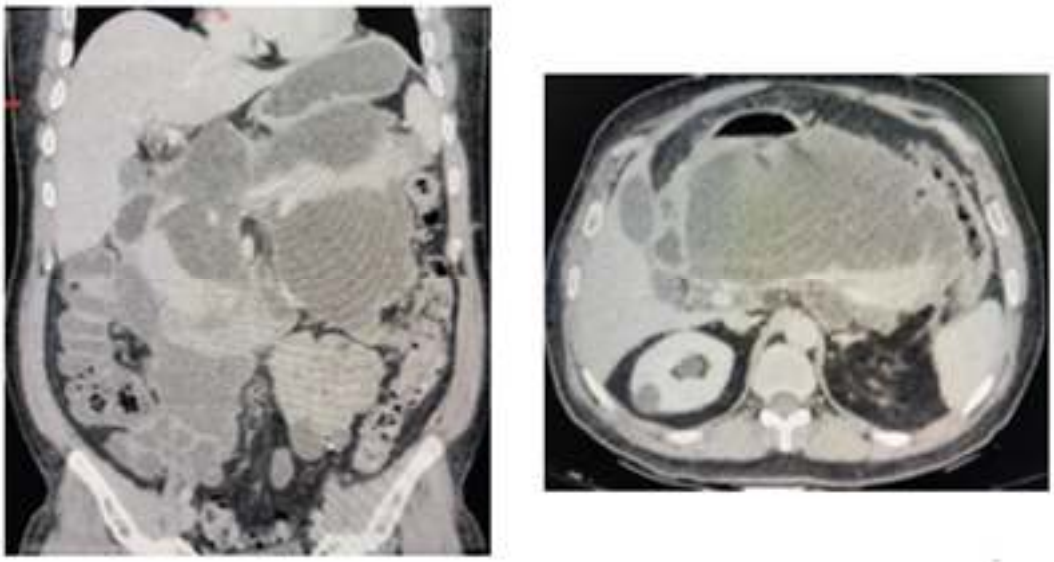
LENGTH OF HOSPITAL STAY –

MCTSI SCORE –

RESULT -

ANNEXURE IV – PHOTOGRAPH

Photograph 1. CT image of pancreatic collection.



Photograph 2. CT image of Walled off Necrosis