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"Assessment of Severity of Acute Pancreatitis Using Inflammatory Markers C Reactive Protein and Lactate Dehydrogenase A Crossectional Study in A Tertiary Care Centre"

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STRUCTURED ABSTRACT:

BACKGROUND:

Acute pancreatitis is defined as an acute condition presenting with abdominal pain, a threefold or greater rise in the serum levels of the pancreatic enzymes amylase or lipase, and/ or characteristic findings of pancreatic inflammation on contrast-enhanced CT. Acute pancreatitis may recur.

Acute pancreatitis may be categorized as mild (interstitial edematous pancreatitis) or severe (necrotising pancreatitis). The former is characterized by interstitial edema of the gland and minimal organ dysfunction. The majority of patients will have a mild attack of pancreatitis, the mortality from which is around 1%. Severe acute pancreatitis is seen in 5–10% of patients, and is characterized by pancreatic necrosis, a severe systemic inflammatory response and often multi-organ failure

This study aims at identification of variations in inflammatory markers c reactive protein and lactate dehydrogenase according to severity of acute pancreatitis using CT severity index and thus enabling to predict the mortality and morbidity

OBJECTIVES: To assess the variations in CRP and LDH levels according to severity of acute pancreatitis by CT severity index before any actual medical intervention done

METHODS:

In this study 91 patients admitted in Government medical college Thrissurwith acute pancreatitis were evaluated their blood samples for obtaining CRP quantitative ,LDH were sent within 48hrs of onset of symptoms and this values have been compared with CTSI obtained from ct imaging .Patient clinical history and values have been recorded in PROFORMA after attaining consent and analysed with chi square test to attain the significance

Results and Discussion : Among the total 91 patients admitted 86%was male population and rest was female,and most of the patients falls in between 30 to 60 years .On evaluating ct severity index 65.9% comes in mild category and 15.4% in severe category rest 18.7% in moderate category.

Then comparing c reactive protein value with chi square test to severity 55.9% had value less than 10mg/dl and around 44.1%had value less than 100 but more than 10 and 71.4% in severe category had values more than 200mg/dl,

Similarly comparing LDH values 81.4% mild category falls in to less than 300IU/L range while85.7% of severe category had values more than 600IU/l and both comparison had p value <.001.

CONCLUSION: From this study we could conclude that as as the CRP and LDH values increases beyond certain limits the severity also changes, CRP values more than 200 falls into severe category and LDH values more than 600 falls in severe category,so combining these parameters the prognosis can be easily predicted and early intervention can be done.

KEY WORDS: CRP,LDH,CTSI,ACUTE PANCREATITIS

I. INTRODUCTION

The most frequent, serious disease that necessitates hospitalisation in daily life is acute pancreatitis. About 25% of individuals experience severe acute pancreatitis, which can lead to problems like organ failure, necrosis, and pseudocysts in the long run. While the mortality rate for mild acute pancreatitis is only approximately 1%, that of severe acute pancreatitis ranges from 15 to 35%.

The majority of acute pancreatitis patients receive conservative care. Secondary infection or necrosis may develop in roughly 25–30% of patients, with a death rate that reaches 100% if untreated. Therefore, it's crucial to determine the disease's severity and begin therapy as soon as a patient with acute pancreatitis is admitted in order to stop complications from occurring.

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The severity of acute pancreatitis has been measured using a variety of scores, including as the RANSON, APACHE, and GLASGOW scores Several serum markers have recently emerged in the diagnosis of acute pancreatitis severity. The importance's of C-reactive protein and lactate dehydrogenase as markers in the prediction of severity of severe acute pancreatitis in comparison with ct severity index is emphasised in this study

AIMS AND OBJECTIVES

1. To study the level of CRP and LDH in patients admitted with acute pancreatitis.
2.To assess the relation of inflammatory markers with ctseverity index

BACKGROUND AND REVIEW OF LITERATURE

Acute pancreatitis is defined as an acute inflammatory process of the pancreas with little or no fibrosis, and it can range from mild self-limiting disease to critical disease with infected pancreatic necrosis and multiple organ failure. (3) The most important and common causes of acute pancreatitis are alcohol consumption and gallstones. ERCP, hyperlipidemia, hypertriglyceridemia, biliary tract operations, pancreatic neoplasms, parasitic infection of the biliary tree, sphincter of oddi dysfunction, and pancreas divisum are other causes of pancreatitis. The age at presentation and gender distribution are determined by the aetiology of acute pancreatitis.

Embryology:

Pancreas is an endodermally derived organ, having two distinct parts viz exocrine and endocrine pancreas. These two tissues present together inside the pancreas despite its morphological functions. Endocrine pancreas is called as Islets of Langerhans consisting of 5 subtypes of cells secreting glucagon, insulin, somatostatin, ghrelin and pancreatic polypeptide hormones and it constitutes only 2% of the total pancreas. The remaining 98% is formed by exocrine pancreas and it is composed of acinar and ductal epithelial cells[i]

Anatomy

The pancreas is a glandular organ with four major parts: the head, neck, body, and tail. It also has an accessory lobe, also known as an uncinate process. It is located retroperitoneally, obliquely with the head, and the uncinate process is in the curvature of the second part of the duodenum and the tail at the region of the spleen's hilum. It measures 12 to 15cm in length. The acini is formed by the accumulation of acinal cells, which secretes bicarbonates and digestive enzymes into centrally located acinar spaces that are linked by tiny ductal networks. These ducts connect to form the main pancreatic ducts, namely the duct of wirsung and the duct of santorini. These ducts drain into the duodenum via the ampulla of vater via the major and duodenal papillae.. The islet of langerhans are scattered through the pancreas and produces endocrine hormones. (1)

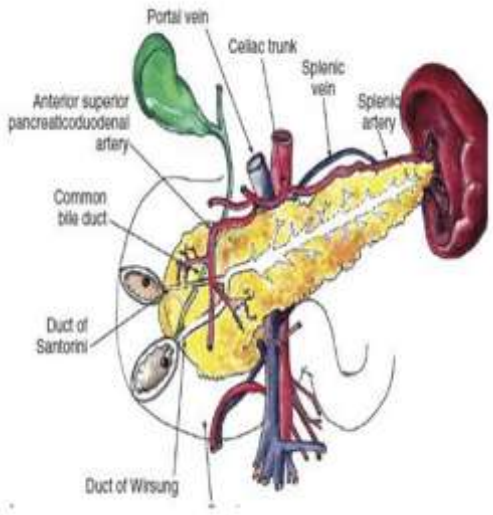

Fig 1

Blood supply:

The arterial supply of the pancreas is primarily provided by the celiac trunk and superior mesenteric arteries, which form arterial arcades within the pancreas. The celiac trunk gives rise to the splenic and common hepatic arteries. The dorsal and greater pancreatic arteries are derived from the splenic artery, and the gastroduodenal artery is derived from the common hepatic artery. Around the head of the pancreas, the gastroduodenal artery divides into anterior and posterior superiorpancreaticoduodenal arteries, which anastamose with anterior and posterior inferiorpancreaticoduodenalarteries, which are branches of the superior mesenteric artery

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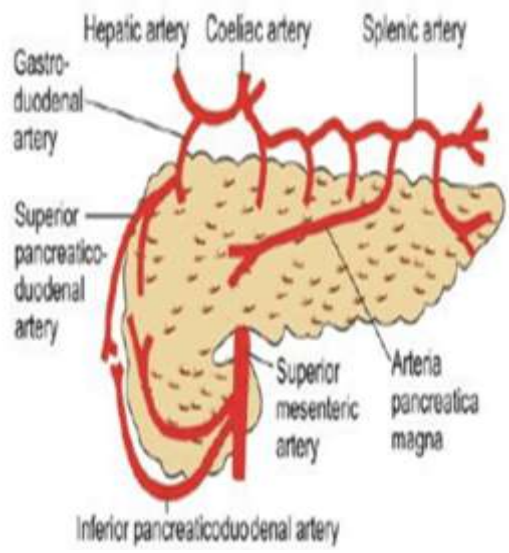
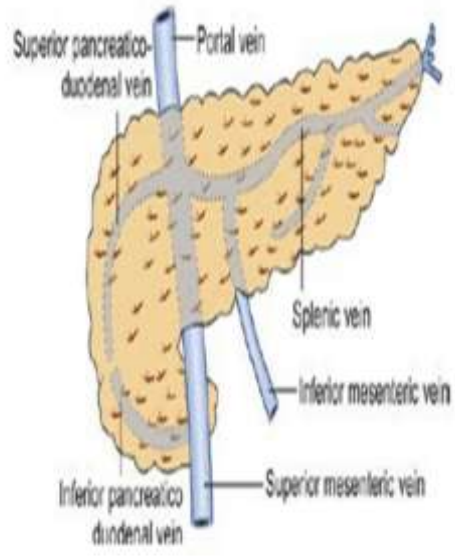


Fig 2.

Venous drainage:
The pancreas's head and neck drain into superior andinferior pancreaticoduodenal veins. The pancreas's body and tail drain into the splenic vein. Venous drainage drains primarily into the portal system.



Venous drainage mainly drains into the portal system.

Fig 3

LYMPHATIC DRAINAGE:

Body and tail drain into pancreaticosplenic nodes via lymphatic drainage. The pancreas's head and neck drain into lymph nodes located along the superior mesenteric artery, hepatic artery, and pancreaticoduodenal artery.

Nerve supply:
The pancreas gets its nerve supply from the autonomic nervous system, which includes both sympathetic and parasympathetic fibres. The vagus nerve is parasympathomimetic, and the sympathetic supply comes from the splanchnic nerves. Thus, celiac plexus block or ablation can be beneficial in the treatment of chronic pain caused by pancreatic tumours.

Physiology:
Normal pancreatic juice is a bicarbonate-rich fluid with 15 grammes of protein. The average pancreas secretes approximately 2.5 litres of pancreatic juice per day. It aids in the alkalinization of the duodenum and thus digestion. The pancreas secretes inactive proenzymes into the duodenum, where they are activated by trypsin. Lipase and Amylase are also secreted into the duodenum in active forms. Basal secretin of these enzymes are low in resting phase and increases during neural and hormonal stimulation. (3)Its secretion is controlled by cholecystokinin and secretin Acinar cells secrete the protein portion of pancreatic juice, while duct cells secrete fluid and electrolytes. At rest, pancreatic juice secretion is extremely low. During eating, the cephalic phase mediates 10% of pancreatic juice stimulation via acetylcholine, whereas the gastric phase mediates 15% of secretion via gastrin release and vagal stimulation. During the intestinal phase, 75% of the stimulation occurs through secretin as a result of duodenal acidification and the release of bile and cholecystokinin as a result of fat and protein entry into the duodenum.[(3)]

Serum amylase:

Normal level is 200-250units. Its level increases in acute pancreatitis. Half life of serum amylase is 24hours. [(3)] It is not very sensitive. It also increases in other conditions as follows: - - - - - -

- diseases of salivary gland
- Mesenteric ischaemia

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- Ruptured aortic aneurysm
- Intestinal obstruction
- Ectopic gestation
- Salpingitis
- Perforated duodenal ulcer

There are two kinds of amylase. Amylase-P levels are elevated in pancreatitis. Amylase-S levels are elevated in conditions other than pancreatitis. Amylase levels in ascitic fluid are also measured. It is highly significant for pancreatitis if ascitic fluid amylase is higher than serum amylase. Amylase levels are extremely high in pancreatic pseudocysts. It is not used to assess the severity of pancreatitis. [3] Persistent elevation indicates a pseudocyst, abscess, or ascitis. Serum amylase levels will be normal in 10% of cases of necrotising pancreatitis. The increase in amylase caused by pancreatic causes is greater than the increase caused by nonpancreatic causes. Amylase enters the lymphatics and circulation from the basal part of acinar cells, and weakened intercellular adhesions in pancreatitis allow amylase to seep into the circulation. Amylase inhibitors are present in the circulation sometimes which masks the serum amylase level. Occasionally amylase binds with albumin which cannot be cleared from the circulation normally leading to false rise in serum amylase level, which causes false positives in the absence of pancreatitis. It occurs in 0.2% of the population.

Pancreatitis:

Pancreatitis is defined as inflammation of the pancreas which may be acute, chronic or relapsing which may lead to various complications. Classification: Two types of classification are there. 1) Marseilles classification: based on clinical classification
2) Trapnell's classification: based on etiology
Marseilles classification:

- Acute pancreatitis
- Acute relapsing pancreatitis
- Chronic relapsing pancreatitis
- Chronic pancreatitis
- In acute pancreatitis, changes are reversible whereas in chronic pancreatitis, changes are irreversible.
- Trapnelle's classification:
- Biliary tract disease due to stones
- Alcohol
- Trauma
- After biliary, gastric, splenic surgeries,
- ERCP
- Hyperparathyroidism
- Hypercalcemia, hyperlipidemia
- Diabetes
- Porphyria
- Autoimmune conditions
- Vascular diseases
- Drugs like steroids, INH, diuretics, tetracycline, estrogens, septran, azathioprine, valproic acid, 5-aminosalicylic acid, etc.,
- Biliary ascariasis, clonorchis sinensis
- Viral infections Infectious mononucleosis
- Mycoplasma
- Pancreas divisum
- Idiopathic[3]

Acute pancreatitis

It is the most common gastrointestinal disease for which patients are acutely hospitalized and its incidence is rising. Around 80% of patients with acute pancreatitis have a mild disease course where symptoms usually resolve within 1 week.2,3 Approximately 20% of patients develop severe acute pancreatitis with organ failure and/or necrotizing pancreatitis. Necrotizing pancreatitis is defined by pancreatic parenchymal necrosis and/or peripancreatic fat necrosis.2 Those patients are at risk for a persistent systemic inflammatory response syndrome and/or (multiple) organ failure. Sterile pancreatic necrosis and sterile peripancreatic collections can usually be treated successfully with conservative measures. However, 30% of patients develop secondary infection of necrosis, most often 3 to 4 weeks after the onset of disease. When secondary infection of necrosis occurs, morbidity and mortality increase dramatically.5,6 Overall mortality in severe pancreatitis is high (15% to 30%) compared with mild pancreatitis (0% to 1%).[1]

Classification Of Acute Pancreatitis

The 1992 Atlanta Symposium attempted to offer a global consensus and a universally applicable classification system for acute pancreatitis. Due to improvements in diagnostic imaging and therapy, together with a better understanding of the pathophysiology of organ failure and pancreatitis, it was necessary to revise the Atlanta Classification. The aims of the 2012 Revised Atlanta Classification were to clarify terminology and stimulate the use of uniform

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definitions and standardized reporting in patients with acute pancreatitis. Three categories of acute pancreatitis were defined, based on the absence or presence of local complications and/or organ failure: mild, moderate, and severe . Based on local complications on diagnostic imaging, acute pancreatitis is divided into interstitial edematous or necrotizing pancreatitis. Four types of local complications can be defined: acute fluid collections, pseudocysts, acute necrotic collections (i.e., sterile or infected), and walled-off necrosis (i.e., sterile or infected)[1]

TABLE 91.1 Severity of Acute Pancreatitis as Defined in the 2012 Revised Atlanta Classification

	2012 REVISED ATLANTA CLASSIFICATION		
Complications	Mild	Moderate	Severe
Local complications	No	Yes	Yes
SYSTEMIC COMPLICATIONS			
Transient organ failure	No	Yes	Yes
Persistent organ failure	No	No	Yes
Exacerbation of preexisting comorbidity	No	Yes	Yes

Table1

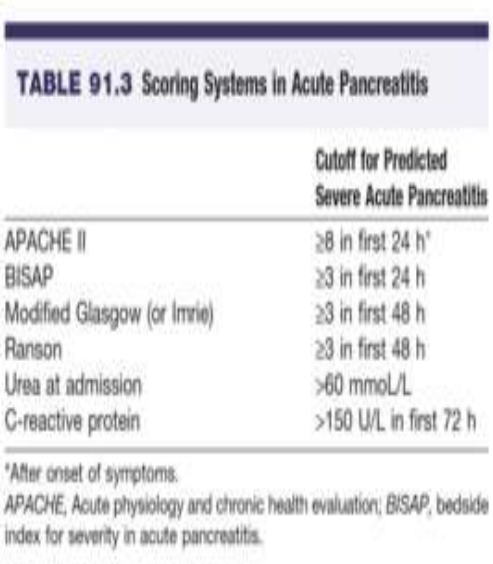

TABLE 91.3 Scoring Systems in Acute Pancreatitis

	Cutoff for Predicted Severe Acute Pancreatitis
APACHE II	≥8 in first 24 h*
BISAP	≥3 in first 24 h
Modified Glasgow (or Imrie)	≥3 in first 48 h
Ranson	≥3 in first 48 h
Urea at admission	>60 mmol/L
C-reactive protein	>150 U/L in first 72 h

*After onset of symptoms.
APACHE, Acute physiology and chronic health evaluation; *BISAP*, bedside index for severity in acute pancreatitis.

Table 2

TABLE 91.2 Collections as Defined in the 2012 Revised Atlanta Classification

Definition	Description*
Acute fluid collection (<4 wk after onset and edematous pancreatitis)	• Homogeneous fluid density • Confined by normal peripancreatic fascial planes • No definable wall encapsulating the collection • Adjacent to pancreas (not intrapancreatic)
Pseudocyst (rare, usually >4 wk after onset and edematous pancreatitis)	• Well circumscribed, usually round/ova • Homogeneous fluid density • Well-defined wall and completely encapsulated • Adjacent to pancreas (not intrapancreatic)
Acute necrotic collection (<4 wk after onset and necrotizing pancreatitis)	• Heterogeneous and nonliquid density • No definable wall encapsulating the collection • Location: intrapancreatic and/or extrapancreatic
Walled-off necrosis (usually >4 wk after onset and necrotizing pancreatitis)	• Heterogeneous and nonliquid density • Well-defined wall and completely encapsulated • Location: intrapancreatic and/or extrapancreatic

*Revised Atlanta Classification definitions of morphologic features of acute pancreatitis.

Table 3

PREDICTING DISEASE SEVERITY

Because of large variability in the clinical course of acute pancreatitis, a number of predictive scoring systems have been developed. These

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scoring systems are based on clinical and biochemical parameters: for example, the Ranson, APACHE-II, Imrie, or modified Glasgow scores. Blood levels of C-reactive protein and blood urea nitrogen are also often used in predicting severity at the time of hospital admission. All these scoring systems have their own strengths and limitations, and a system with a high negative predictive value and a high positive predictive value is not yet available, as described in a recent systematic review. The most popular scores and their application are listed in Table 2. The presence of (persistent) organ failure is the key determinant for morbidity and mortality in acute pancreatitis, in particular (early) multiorgan failure is associated with high mortality. The International Association of Pancreatology (IAP)/American Pancreatic Association (APA) guidelines recommend using persistent systemic inflammatory response syndrome (SIRS) (>48 hours) as a marker to predict severity of acute pancreatitis.Persistent organ failure is also one of the key determinants of the severity of acute pancreatitis in the Revised Atlanta classification (1) serum LDH detected upon presentation was independently associated with the incidence of POF in patients with AP. The predictive value was superior to scores of SIRS and Ranson's criteria. Although a majority of patients with AP have a mild course of the disease, severe forms of AP require more attention because of its high morbidity and mortality. POF, the most widely seen cause of mortality within the first 2 weeks of disease onset, develops in 10%–20% of AP(5)

MORPHOLOGIC SCORING SYSTEMS

Morphologic abnormalities assessed by CECT can be used in a scoring system such as the Balthazar grade, pancreatic size index, extrapancreatic inflammation on CT score, CT severity index, or the modified CT severity index. Although all these scoring systems have been shown to correlate with morbidity and mortality, it remains difficult, at the time of their admission or early in the course of their hospitalization, to accurately identify individual patients who will develop clinically severe disease. A study comparing all radiologic scoring systems in the early prediction of severity in acute pancreatitis did not show an advantage of an early CT on admission as an independent predictor as compared to the more easily obtainable clinical scoring systems in terms of accuracy in predicting clinically severe acute pancreatitis and mortality.[1]

Ranson's prognostic criteria in gallstone pancreatitis score >3 suggests severe AP	Ranson's prognostic criteria in non-gallstone pancreatitis	Glasgow–Imrie prognostic criteria	Acute Physiology and Chronic Health Evaluation (APACHE II) score >8 points predicts 11 to 18% mortality
On admission: • Age >70 years • TC >18,000/cu mm • Blood sugar >220 mg% • LDH >400 IU/L • AST >250 IU/100 mL	On admission: • Age >55 years • TC >16,000/cu mm • Blood sugar >200 mg% • LDH >350 IU/L • AST >250 IU/100 mL	On admission: • Age >55 years • TC >15,000/cu mm • PaO_2 <60 mmHg • Blood urea >16 mmol/L (No response to IV fluids) • Blood sugar >200 mg% (no H/O diabetes)	• Equation includes the following factors: age, rectal temperature, mean arterial pressure, heart rate, PaO_2, arterial pH, serum potassium, serum sodium, serum creatinine, haematocrit, white blood cell count, Glasgow coma scale score.
Within 48 hours: • Haematocrit drop >10% • BUN rise >2 mg% • Serum calcium <8 mg% • Base deficit >5 mEq/L • Fluid sequestration >4 L	Within 48 hours: • Haematocrit drop >10% • BUN rise >5 mg% • PaO_2 <60 mmHg • Serum calcium <8 mg% • Base deficit >4 mEq/L • Fluid sequestration >6 L	Within 48 hours: • Serum calcium <2 mmol/L • Serum albumin <3.2 g/dl • LDH >600 U/L • AST/ALT >200 U/L	APACHE II modified (1996) LFT is added in biliary pancreatitis APACHE – O (Toh 1996) Obesity is added

Note:

Table 4

Grade and appearance	Score	Grade and appearance	Score
CT Grade:		**CT Grade:**	
A – Normal pancreas	0	A – Normal pancreas	0
B – Oedematous pancreatitis	1 2 3	B – Intrinsic pancreatic abnormalities with or without inflammatory changes in peripancreatic fat	2 4
C–B+ mild extrapancreatic changes	4	C – Pancreatic/ peripancreatic fluid collection or peripancreatic fat necrosis	0 2 4 2
D–Severe extrapancreatic changes with one fluid collection	0 2		
E – Extensive/ multiple extrapancreatic collections or gas bubbles in or adjacent to pancreas		**Necrosis score** None <30% >30% One or more extrapancreatic complications	
Necrosis score None <1/3rd			
More than 1/3rd less than 1/2 More than half	4 6	3 mL	10 days
CT SI (CT severity index) – CT grade + necrosis score 0–3 = mild; 4–6 = moderate; 7–10 = severe			

Table 5

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Clinical features:

Sudden onset of abdominal pain mostly in the epigastric region which is referred to back.

Pain is severe and relieved or reduced by leaning forward.

Vomiting, fever, tachypnoea Tenderness, rebound tenderness, guarding, rigidity and abdominal distension Jaundice (due to cholangitis).

Jaundice may also be due to bile duct disease / obstruction or cholestasis.

Features of shock and dehydration such as Oliguria, hypoxia and acidosis.

Grey-Turner's sign, Cullen's sign, Fox sign.

Hematemesis and malena due to duodenal necrosis, gastric erosions,

- decreased coagulability or disseminated intravascular coagulation.
- Hiccough is occasionally present and it is mostly refractory.
- Ascites
- Paralytic ileus is common.[3]
- Pleural effusion (20%), pulmonary oedema, consolidation, features of ARDS can occur.
- Neurological abnormalities occurs due to toxaemia, fat embolism, hypoxia, respiratory distress. It may ranges from mild psychosis to coma.
- **Metabolic and biochemical changes**

- Hypovolemia due to capillary leak and vomiting. It causes raise in haematocrit, blood urea, serum creatinine levels.
- Hypoalbuminemia which becomes more relevant after fluid correction.
- Hypocalcaemia is either due to decreased level of albumin or loss of ionized calcium. Hypocalcaemia occurring due to reduced ionisedcalcium carries poor prognosis. Response of calcium reserve in bone to PTH is also reduced.
- Total count is raised with significant neutrophilia
- Thrombocytopenia, raised FDP, decreased fibrinogen, prolonged partial thromboplastin time and PT—are common. Later it can lead to the development of DIC.
- Hypochloraemic metabolic alkalosis is common due to repeated vomiting.
- Reduced insulin secretion, increased glucagon and catecholamine secretion lead to the development of hyperglycaemia. It is more pronounced in diabetics.
- Hyperbilirubinaemia due to biliary stone/ obstruction or cholangitis or non-obstructive cholestasis.
- Hypertriglyceridaemia is more common especially in patients with hyperlipidemia.
- Methemalbuminemia occurs rarely and when it occurs in acute pancreatitis , it indicates poor prognosis.

DIFFERENTIAL DIAGNOSIS OF PANCREATITIS

Fig4

PHASES OF ACUTE PANCREATITIS

- Traditionally, acute pancreatitis was described as running a biphasic course with two peaks of mortality: early and late.
- The early phase is characterized by a SIRS and lasts about 1 to 2 weeks.
- The late phase is characterized by a compensatory, antiinflammatory response syndrome (CARS), which can run a protracted course from weeks to months.
- More recent data suggest that the biphasic course is outdated and that there are not two peaks in the incidence of organ failure and mortality.
- Organ failure may already be present at the first presentation to the emergency department
- More severe cases, organ failure is usually diagnosed in the early SIRS phase at a median of 2 days after admission.
- Overall, approximately one-half of the patients who die in the early phase of acute pancreatitis have no infected necrosis but suffer from multiorgan failure.

- A recent systematic review of cohort studies demonstrated that the mortality of patients

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with organ failure in acute pancreatitis is 32%.Patients with both organ failure and infected necrosis had a mortality of 43%.[1]

Management of acute pancreatitis:

- The management of acute pancreatitis covers a wide range of spectrum of disease.
- It ranges from mild , moderate to severe acute pancreatitis.
- The duration of hospital stay and mortality varies between these categories.
- The mortality of mild pancreatitis is less than 1% and for moderate pancreatitis , the mortality is around 10%.
- The mortality of severe acute pancreatitis ranges from 20 to even greater than 50%.
- Early identification of these categories is thus important. Various scoring systems such as RANSON'S score, and various serum markers are used in assessing the severity of acute pancreatitis.
- The diagnosis of AP requires two of the following three features to be present according to international consensus:
- 1) abdominal pain consistent with AP (acute onset of a persistent, severe, epigastric pain often radiating to the back),
- 2) a threefold or higher elevation of serum amylase or lipase levels above the upper laboratory limit of normal, or
- 3) characteristics findings of pancreatitis by imaging.
- The serum half-life of amylase (10 hours) is shorter than that of lipase (6.9–13.7 hours) and therefore normalizes faster (3–5 vs. 8–14 days, respectively).
- In patients who do not present to the emergency department within the first 24 to 48 hours after the onset of symptoms, determination of lipase levels is a more sensitive indicator to establish the diagnosis.
- Lipase is also a more specific marker of AP because serum amylase levels can be elevated in a number of conditions, such as peptic ulcer disease, mesenteric ischemia, salpingitis, and macroamylasemia.
- Patients with AP are typically hyperglycemic; they can also have leukocytosis and abnormal elevation of liver enzyme levels.
- T he elevation of alanine aminotransferase levels in the serum in the context of AP confirmed by high pancreatic enzyme levels has a positive predictive value of 95% in the diagnosis of acute biliary pancreatitis.[2]
- Serum lactescence which is a metabolite of triglycerides. Most specific in hereditary hyperlipidaemia or in pancreatitis due to alcohol. Serum trypsin is most accurate for pancreatitis, but it is not commonly[3]
- Trypsinogen activation polypeptide (TAP) assay in serum and urine. It is useful in assessing the severity of the acute pancreatitis.
- CRP (>150 mg/L) is also useful(7). Phospholipase A2, LDH levels are also often assessed. These are useful in assessing the severity of acute pancreatitis.[3]

Imaging Studies

- Imaging studies are not required for diagnosis, but may be helpful in determining need for intervention in severe AP or elucidating an elusive etiology.
- Although simple abdominal radiographs are not useful for diagnosis of pancreatitis, they can help rule out other conditions, such as perforated ulcer disease. Nonspecific findings in patients with AP include air-fluid levels suggestive of ileus,[2]
- cutoff colon sign as a result of colonic spasm at the splenic flexure and widening of the duodenal C loop caused by severe pancreatic head edema.
- Ultrasound abdomen has got 95 percent sensitivity,in diagnosing gall stone
- Combined elevations of liver transaminase and pancreatic enzyme levels and the presence of gallstones on ultrasound have an even higher sensitivity (97%) and specificity (100%) for diagnosing acute biliary pancreatitis
- Contrast-enhanced computed tomography (CT) is currently the best modality for pancreas evaluation, especially when performed with a multidetector CT scanner.
- Diagnostic uncertainty, confirmation of severity based on clinical predictors, failure to respond to conservative treatment, or clinical deterioration are all indications for CT.
- The portal venous phase (65-70 seconds after injection of contrast material) is the most valuable contrast phase for evaluating the pancreatic parenchyma
- Because it allows evaluation of the viability of the pancreatic parenchyma, amount of peripancreatic inflammation, and presence of intraabdominal free air or fluid collections.

- Noncontrast CT scanning can help identify fluid collections or extraluminal air in the setting of renal failure.
- Abdominal magnetic resonance imaging (MRI) is also useful to evaluate the extent of necrosis, inflammation, and presence of free f luid.
- However, its cost and availability and the fact that patients requiring imaging are critically ill and need to be in intensive care units limit its applicability in the acute phase.
- Although MRCP is not indicated in the acute setting of AP
- It has an important role in the evaluation of patients with unexplained or recurrent pancreatitis because it allows noninvasive complete visualization of the biliary and pancreatic duct anatomy.
- Endoscopic ultrasound (EUS) may be useful in assessing persistent choledocholithiasis in the setting of gallstone pancreatitis.
- Several studies have shown that routine ERCP for suspected gallstone pancreatitis reveals no evidence of persistent obstruction in the majority of cases and may even worsen symptoms due to gland manipulation.
- EUS has been shown to be sensitive for detecting choledocholithiasis; it allows examination of the biliary tree and pancreas without risk of pancreatitis worsening.
- When persistent choledocholithiasis is confirmed by EUS, ERCP can be used as a therapeutic option.[2]

Prediction of severity:

- Most of the classifications are used to predict the past or present severity of the disease, it is important to predict the future outcome of the patient.
- This prediction is important in making decisions about fluid therapy, and the need for ERCP.
- The most widely used method is Ranson's or modified Glascow's score. Both use clinical and biochemical parameters for predicting severity. These parameters are scored over 48 hours of admission. If there are 3 or more positives, it is considered as severe disease.
- Another score used is APACHE II which is scored at 24 hours of admission. If there are 8 or more positives, it is considered severe.
- C- reactive protein is also used to predict the severity. CRP level > 150mg/dl has similar accuracy of predicting severity as Ranson's score.([6])
- The more recently proposed score is BISAP (Bedside Index for Severity of Acute Pancreatitis)is calculated from blood urea nitrogen (>25mg/dl), altered mental status (GCS<15), age >60years, presence of severe inflammatory response syndrome (SIRS) and pleural effusion.

Early and late organ failure:

- Acute pancreatitis runs a biphasic course. The first phase is severe inflammatory response syndrome and lasts for about 2 weeks.
- The second phase is counteractive anti-inflammatory response syndrome (CARS) which is characterised by stage of immunosupression. [(1)]
- Organ failure in SIRS phase is related to severe systemic inflammation and not to local infection whereas organ failure in CARS phase is related to secondary infections such as infected necrosis of the pancreas.
- Infection can also occur in SIRS phase but mostly of bacteremia and pneumonia.
- Organ failure occurs in all organs but most commonly involves pulmonary and cardiovascular system.
- The gastrointestinal system is also affected by acute pancreatitis and it is reflected by urine output. Organ failure
- SIRS occurs two days after admission. Half of those who die from acute pancreatitis die from organ failure rather than infected necrosis.
- The clinical course of necrotizing pancreatitis varies greatly, and there may be a transition between the SIRS and CARS phases.
- Distinguishing three scenarios may be useful in understanding the underlying pathophysiologic processes:
- Early onset organ failure - occurs in the first week. Intensive care and supportive measures are required. Clinical deterioration occurs between weeks 3 and 5, indicating infective necrosis.
- If the clinical condition is suddenly complicated by deterioration without organ failure, there is a high risk of infected necrosis.
- If there is no improvement after 2 to 3 weeks of intensive care treatment, In this patient's condition, a fine-needle aspiration of the collection should be performed to distinguish between persistent SIRS and infected necrosis.

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- However, if gas bubbles are detected on a CT scan, no further diagnostic procedures are required.[1]

TREATMENT:

Conservative management:

- SIRS phase: Adequate fluid resuscitation with pain management is the mainstay of treatment.
- Pain is the main complaint of pancreatitis and its management is a clinical priority.
- Intravenous analgesia is mandatory before starting oral intake. Those patients with a mild pain are managed with NSAIDs such as methimazole 2g/8h IV and those with severe pain are managed with opioids such as buprenorphine 0.3mg/ 4h IV.
- Other analgesics used in the management of acute pancreatitis are pentazocine, procaine hydrochloride, meperidine.
- Morphine should be avoided because it causes spasm of sphincter of oddi. [7]
- A diuresis-guided fluid regimen (1 mL/kg/hr urine production) is required in the initial phase.
- Close monitoring and intravenous fluid supplementation in the initial 24 hours are most important.
- Crystalloid resuscitation volumes as high as 20 L may be required.
- In this phase, there is no need for radiological interventions as resuscitation is most important in this phase.
- If the patient's condition does not improve or worsens, peripancreatic fluid collections or infected necrosis must be ruled out.
- The presence of gas bubbles on CECT is indicative of infected necrosis. According to some authors, FNAC of the collection can be used to diagnose peripancreatic collections or infected necrosis.
- **Infection prevention**: The most common organism causing pancreatitis is enteral bacteria.
- Bacteremia or ventilator-associated pneumonia occurs within 8 days of disease onset, whereas infectd necrosis occurs within 25 days of disease onset.
- Bacteraemia raises the risk of necrosis infection by 38 to 65%. Persistent organ failure and bacteremia are the most powerful predictors of death.
- Intravenous antibiotics, enteral nutrition, selective bowel decontamination, and probiotics have all been tried to reduce infection rates.

Enteral nutrition:

- Enteral nutrition is hypothesized to reduce bacterial overgrowth and also to improve intestinal mucosal barrier function and thereby reducing infections
- . In patients of mild disease, oral feeding can be started as early as possible.
- In patients of severe pancreatitis, enteral feeding through nasojejunal route can be started by 3 days if the patient is not expected to resume oral diet.
- It reduces both infections and mortality compared to total parenteral nutrition.

Systemic intravenous antibiotics:

- Many studies suggests the use of prophylactic systemic antibiotics in lowering the rate of infected necrosis.
- A recent metaanalysis showed that there is no beneficial effect in prophylactic use of antibiotics.
- Selective bowel decontamination (SBD): Since the small bowel is the main source of infection, selective bowel decontamination is needed in acute pancreatitis.
- There was one randomised control study in which compared the effect of norfloxacin, amphotericin, colistin in paients with acute pancreatitis which showed significant reduction in mortality by reducing the chances of infection with gram negative organisms.

Interventional treatment:

- USIRS phase: (first and second weeks) Interventions in this phase are only to treat the acute life threatening complications or prevention of further deterioration.
- Currently the only means to prevent deterioration is by ERCP with sphicterotomy, but its therapeutic significance is yet to be established.
- Surgical necrosectomy in this phase is associated with higher mortality and hence it is contraindicated within 72hours
- Since the main clinical picture in this phase is systemic inflammation rather than presence or absence of infected necrosis.[1]
- Only abdominal compartment syndrome, bowel ischemia, perforation, and severe bleeding unresponsive to angiographic coiling warrant intervention.

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- The 2007 international consensus meeting defined abdominal compartment syndrome as intraabdominal pressure greater than 20mmHg with signs of new organ failure.
- If drainage fluid is present, percutaneous drainage can be used as an initial treatment.
- If percutaneous drainage does not immediately lower the abdominal pressure or if there is more free fluid, laparatomy and decompression are recommended.
- It is not recommended to explore the pancreas because it is too early to safely remove the necrosis and there is a risk of infection entering the necrosis
- Percutaneous drainage of sterile collections is not recommended because there is a risk of introducing iatrogenic infection.
- The current concept of acute biliary pancreatitis is a gallstone released from the gallbladder into the common bile duct, causing temporary obstruction at the level of the ampulla of vater,
- Resulting in obstruction of the pancreatic duct with obstruction of pancreatic flow and secondary damage to exocrine pancreas cells due to autodigestion of exocrine pancreas.
- Theoretically, ERCP with sphicterotomy causes early relief by stopping the process of acute pancreatitis at an early stage and thus reducing the complications.
- But recent study showed that there is no benefit of routine ERCP in patients of severe biliary pancreatitis in absence of cholangitis.
- A recent study demonstrated that ERCP with endoscopic sphincterotomy reduces the complication rate in patients with predicted severe biliary pancreatitis and cholestasis (bilirubin >2.3 mg/dL [>40 μmol/L] and/or dilated common bile duct). [(1)]

Intervention in CARS Phase

- During the second phase, the patient experiences another episode of systemic infection or sepsis caused by secondary necrosis infection.
- The most acceptable indication for intervention is evidence of pancreatic or peripancreatic necrosis combined with signs of sepsis.
- Less common indications for intervention during this stage include gastric outlet obstruction, abdominal compartment syndrome, bleeding, bowel perforation, and common bile duct obstruction.
- Intervention can be performed via open laparatomy with necrosectomy, minimally invasive surgery, endoscopic or radiographic percutaneous methods, or a combination of the two.
- Intervention for infected necrosis: (third week and later) It is preferable to wait until intra or extrapancreatic collections have been encapsulated before intervening.
- This encapsulation process takes about 4 weeks, and the encapsulated collections are known as 'walled off necrosis'.
- Antibiotics may be required to allow for encapsulation while the patient's clinical condition is closely monitored, and a CECT scan performed at regular intervals is a viable option to postpone surgical intervention.
- Necrosectomy was performed 27 days after disease onset, with a 25% mortality rate. When performed within the first two weeks, the mortality rate was approximately 75%. According to current thinking, deferring surgical intervention until four weeks after disease onset is the best course of action.
- This interval time is primarily determined by the extent of encapsulation and the patient's clinical condition. This policy applies only to patients who survive the initial phase of SIRS and develop necrosis infection with signs of sepsis during the CARS phase.[(1)]

Types of intervention:

- Catheter drainage: It is the least invasive method for treating infected necrosis. The drain can be placed percutaneously through the left retroperitoneum or transabdominally or can be placed through the wall of stomach or duodenum transluminally.
- In around 55% of the patients with necrotising pancreatitis, percutaneous drainage is the only intervention needed for cure.
- The technical success rate for this method was 99% and the mortality rate was 17%a RCT study showed that the feasibility of this percutaneous drainage was 99%.
- If the patient donot improve after adequate drainage, necrosectomy should be done as the next step. The percutaneous drain can be used as a guidance for minimally invasive necrosectomy.
- This two stage approach – drainage followed by minimally invasive necrosectomy is called as stepup approach and now considered as

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standard of care in patients with infected necrosis.

- Minimally invasive necrosis: The most commonly used minimally invasive procedure is video assisted retroperitoneal debridement (VARD). The first step consists of placement of left sided percutaneous retroperitoneal drain through the left flank. The patient is then placed in supine position with the left side elevated.
- A 5-7cm incision is made and the necrotic collection is opened by the guidance of the drain. Initially the pus and-the necrotic material are removed blindly.
- Then a 0-degree laparascope is introduced to remove all the necrotic material under direct vision. The loosely adherent necrotic pieces are only removed in order to minimize bleeding.
- In contrast to percutaneous drainage, VARD allows removal of large pieces of necrotic material. The more degree of encapsulation, the more easier the necrosectomy.
- Following this near total necrosectomy, two suction drains are kept. Postoperatively, continuos lavage with increasing amounts of 0.9% saline (2, 4. 6L) per day in the first 3 days.
- In a dutch study , the results of minimally invasive step-up approach and open necrosectomy was compared which showed significant difference with respect to development of complications and costs was observed all in favour of VARD and there was no significant difference in mortality.
- A purely percutaneous minimally invasive retroperitoneal necrosectomy using operating nephroscope developed by Carter at al suggested a decrease in mortality by using this technique.[7]

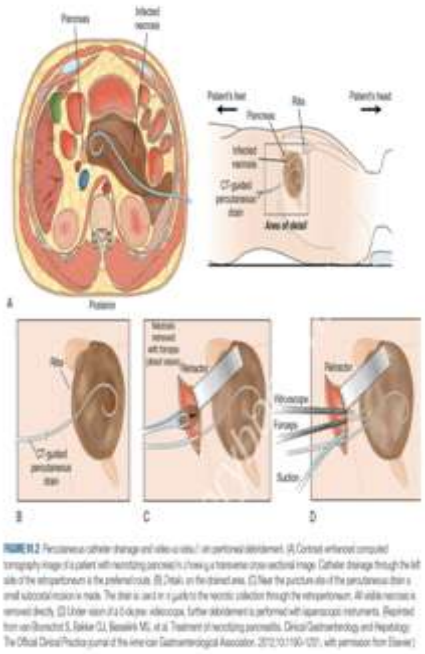

Fig 5

Endoscopic transluminal necrosectomy:

- If VARD technique is not feasible, due to difficulty in reaching the necrotic site, endoscopic transluminal or transgastric necrosectomy can be done.
- The success rates ranges from 80% to 93% with mortality of 0% to 6%. The advantages of this technique are that no abdominal incision(s) are required, and chances of external pancreatic fistula may not occur, because an internal fistula to the stomach is iatrogenically created.
- The chances of occurrence of incisional hernia, often difficult to treat after open necrosectomy, is also less. The major disadvantage of this technique is that the need for repeated, multiple procedures to remove sufficient amounts of necrosis.

Open necrosectomy:

- Till the results of the PANTER study were published, primary open necrosectomy was considered as the standard treatment in patients with infected necrotizing pancreatitis.

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- The most commonly used technique of open necrosectomy is laparotomy with a retroperitoneal lavage system placement after complete necrosectomy has been performed.(1)
- In this technique, drains are placed in the lesser sac after necrosectomy. Continuous lavage with increasing amounts (2, 4, then 6 L) of 0.9% saline are given per day for about 3 days.
- Lavage is useful for many purposes such as mechanical debridement, prevention of tube obstruction, and dilution of pancreatic juice.
- The mortality of this technique is approximately 25%. Another open technique is open necrosectomy with closed packing.
- A group from Boston used transmesenteric approach of open necrosectomy with 11% mortality rate.
- The necrosed part is approached through transverse mesocolon and debridement was done bluntly, with the aim of removing all necrotic tissue and debris.
- The cavity is packed with gauze-stuffed Penrose drains which are removed one by one after a week.
- Some-of the surgeons continue to use an open abdomen strategy with regular, relaparotomies as a routine, done every 3 to 5 days.
- The mortality of this procedure is around 70%, hence it is advised to use this technique only as a part of rescue strategy, when it is difficult to close the abdomen.

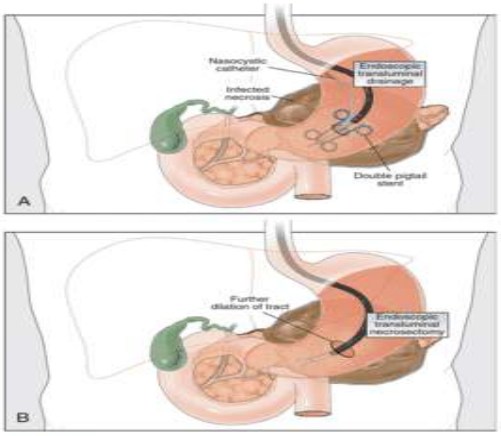

FIGURE 91.3 Endoscopic transluminal drainage and endoscopic transluminal necrosectomy. (A) Through the stomach wall the necrotic collection is punctured and a guidewire is placed in the collection, under guidance of endoscopic ultrasound if needed. Over the guidewire the tract is balloon dilated. Two pigtail drains and a nasocystic catheter are placed in the collection for continuous lavage. (B) Further dilation of the cystogastrostomy and the collection is performed via endoscope. A necrosectomy can be performed under direct vision. (Reprinted from van Brunschot S, Bakker OJ, Besselink MG, et al. Treatment of necrotizing pancreatitis. Clinical Gastroenterology and Hepatology: The Official Clinical Practice journal of the American Gastroenterological Association. 2012;10:1190–1201, with permission from Elsevier.)

Fig 6

Prevention of recurrent pancreatitis:

- If gallstones are the cause of pancreatitis, cholecystectomy with bile duct clearance is required to prevent recurrent attacks.
- Early cholecystectomy in patients with severe pancreatitis, such as necrotizing pancreatitis, may be harmful because these collections may become infected as a result of the procedure(1)

Complications

- . Pseudocyst formation,
- acute fluid collections,
- pancreatic abscess and infected necrosis,
- pancreatic ascites,
- pancreatic-pleural effusion, pancreaticoenteric and pancreaticocutaneous fistulas are all complications of acute pancreatitis.(1)

PANCREATIC PSEUDOCYST

- A pseudocyst is a localised collection of pancreatic secretions surrounded by a granulation tissue or fibrous tissue wall that develops as a result of acute or chronic pancreatic inflammation, pancreatic trauma, or obstruction of the pancreatic duct by a neoplasm. (1)
- Pseudocysts account for between 50% and 75% of pancreatic cystic lesions.
- They differ from other peripancreatic fluid collections such as cystic neoplasms, parasitic cysts, and congenital cysts in that they lack an epithelial lining and have a high concentration of pancreatic enzymes within the pseudocyst, and they form at least 4 weeks after an episode of acute pancreatitis or pancreatic trauma.
- Pseudocysts are formed as a result of the inflammatory response this occurs following extravasated pancreatic collections.
- The pseudocyst capsule may be thin fibrous tissue that eventually thickens as the pseudocyst matures.
- Following that, the pseudocyst's liquid contents are gradually reabsorbed by the body, after which the pseudocyst resolves.
- It means that the communication between the pseudocyst and the pancreatic duct has been cut off.
- Persistence of a pseudocyst indicates that the pseudocyst is communicating with the pancreatic duct.
- Acute fluid collections form early in the course of acute pancreatitis and lack a distinct wall of fibrous or granulation tissue.

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- They are common in patients suffering from severe pancreatitis. Acute fluid collections occur in approximately 30% to 50% of acute pancreatitis cases.
- The majority of these lesions regress on their own without any special treatment, such as drainage. The majority of acute fluid collections do not communicate with the pancreatic duct.
- They are simply a swollen or exudative response to inflammation and trauma. They are also known as pseudo pseudo cysts because they do not communicate with the pancreatic duct
- A pancreatic abscess is a circumscribed collection of purulent fluid containing little necrotic material that develops as a result of acute pancreatitis or pancreatic trauma
- An abscess of the pancreas-occurs late in the course of severe acute pancreatitis (i.e. 4 weeks after symptoms first appear).
- To distinguish from infected pancreatic necrosis, the presence of a purulent exudate, a positive culture, and the absence of necrotic pancreatic material are used.
- This distinction is necessary for management because the treatment for both conditions differs.
- Percutaneous drainage can be used to treat a pancreatic abscess, whereas operative debridement is required for infected pancreatic necrosis.

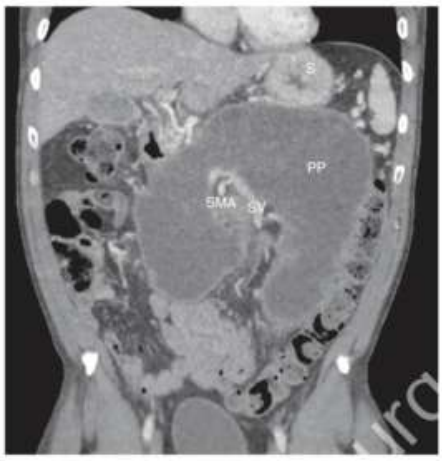

FIGURE 93.1 Computed tomography scan showing a large horseshoe-shaped pancreatic pseudocyst (*PP*) after acute gallstone pancreatitis. *S*, Stomach; *SMA*, superior mesenteric

Fig 7

- Pseudocysts are more common in men than in women, owing to their association with alcoholic pancreatitis.
- The majority of pseudocysts form in the pancreas's head, but they can also form in the pancreas's neck, body, and tail. In about 90% of patients, the most common symptom of pseudocyst is abdominal pain.
- A pseudocyst has also been linked to increased or refractory pain in chronic pancreatitis patients.
- Other symptoms include premature satiety, nausea and vomiting, jaundice, fever, and weight loss.
- Upper abdominal tenderness and a palpable abdominal mass are frequently found during a clinical examination. Early satiety symptoms, nausea, and vomiting are all due to gastroduodenal obstruction caused by the pseudocyst's mass effect.
- Patients with pseudocysts may not seek medical attention until complications arise. Sepsis secondary to infection, hypovolemic shock due to haemorrhage, jaundice due to CBD obstruction, and severe abdominal pain due to intraperitoneal rupture of a pseudocyst are all complications of pancreatic pseudocyst.
- A pancreas injury is unusual. It can happen after a sharp or penetrating injury. Penetrating or blunt trauma can cause pancreatic ductal disruption, which can contribute to pseudocyst formation.
- These injuries could go unnoticed during the initial radiologic evaluation or laparotomy.
- The diagnosis of pseudocyst is made many weeks after the injury in this case.[1]

DIAGNOSIS OF PSEUDOCYST

- Persistently elevated amylase after resolution of acute pancreatitis should suggests formation of pseudocyst.
- Some have mild leukocytosis, elevated liver enzymes indicating compression of the biliary tree.
- CT scan is used for diagnosis of a pancreatic pseudocyst. Ultrasound examination is also used to identify pseudocysts. It is least invasive and it is used in follow up of the size of the cysts.[1]

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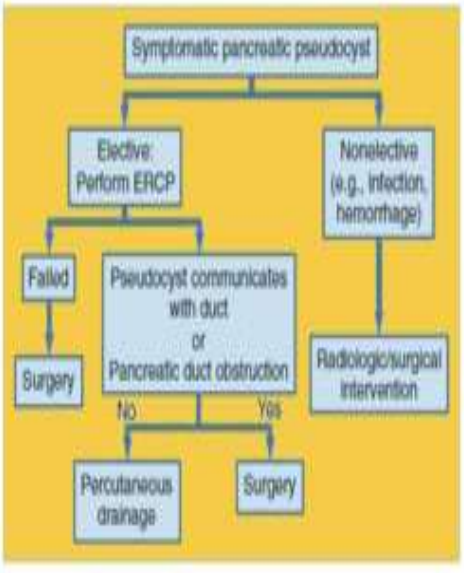
Fig 8

TABLE 93.3 Cyst Fluid Concentrations for Commonly Aspirated Cystic Pancreatic Lesions

Cyst Fluid Element	Pancreatic Pseudocyst	Serous Cystadenoma	Mucinous Cystic Neoplasm	Intraductal Papillary Mucinous Neoplasm
CEA	Low	Low	High	Low to high
Amylase	High	Low	Low	High
Mucin	Absent	Absent	Present usually	Present usually

CEA, Carcinoembryonic antigen.
Data from Brugge WR, Lauwers GY, Sahani D, Fernandez-del Castillo C, Warshaw AL. Cystic neoplasms of the pancreas. *N Engl J Med.* 2004;351(12):1218-1226.



ERCP ALGORITHM
TABLE 6

BOX 93.1 Management Options for Pancreatic Pseudocysts

- Observation
- Percutaneous catheter drainage
- Endoscopic internal drainage
  - Cystogastrostomy
  - Cystoduodenostomy
  - Transpapillary pancreatic duct stenting/pseudocyst drainage
- Surgical interventions (open or laparoscopic)
  - Cystogastrostomy
  - Roux-en-Y cystojejunostomy
  - Cystoduodenostomy
  - Longitudinal pancreaticojejunostomy
  - Partial pancreatic resection
  - External catheter drainage

Table 7

MANAGEMENT

- In patients who do not meet the criteria for conservative non-operative management, intervention in the management of pseudocyst is required.
- For ductal disruption, biliary obstruction, and chronic pancreatitis, simultaneous intervention is required
- . Current management options for a pseudocyst patient include percutaneous drainage, endoscopic drainage, operative internal or external drainage, and resection.
- Percutaneous aspiration and percutaneous drainage should be distinguished clearly. The goal of percutaneous aspiration is to remove all pseudocyst fluid in one operation without leaving a drainage catheter in place.
- This technique should be used on less than half of the patients who have pseudocysts. The remaining patients will require the aspiration technique to be repeated.
- According to a review of patients, the most successful percutaneous aspiration applicants were given pseudocystsIn the tail of the pancreas, less than 100 ml in total volume, with low intracystic amylase.

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Percutaneous catheter drainage:

- It involves placement of a catheter into a pseudocyst by the Seldinger technique usually done under USG or CT guidance.
- The pseudocyst tract is dilated to accept a catheter of size 7 to 14 French after it is entered through the flank or transgastric approach.
- To ensure patency, saline irrigation should be administered through the catheter two to three times per day
- The presence of necrosis or solid debris in the pseudocyst, haemorrhage within the pseudocyst, a lack of a safe access route, and complete obstruction of the main pancreatic duct are all contraindications to percutaneous drainage
- Infection of the drain tract, persistent or recurrent pseudocyst, and pancreaticcutaneous fistula are all complications of percutaneous drainage.[(1)]

Endoscopic procedures:

- Endoscopic methods in the treatment of pseudocysts have recently evolved. Currently the technique involve the use of flexible scope in localizing and draining the pseudocysts by creating a tract between the pseudocyst and the stomach or duodenum
- This fistulous tract communication is created with electrocautery and an endoprosthesis is placed to keep the fistula open.
- Endoscopic drainage technique requires the pseudocyst to be located in the head or body of the pancreas and the pseudocyst should be bulging into the wall of stomach or duodenum.
- Endoscopic ultrasound (EUS) is used to identify the pseudocyst and a suitable drainage site.
- Hemorrhage and perforation are two complications of this procedure. Endoscopic and percutaneous techniques are typically used in tandem to identify and drain pseudocysts that are adjacent to the stomach or duodenum wall but do not bulge into the lumen. [(1)]

Transmural endoscopic drainage:

- Approximately 50% of pseudocysts can be drained by transmural approach based on location of the pseudocyst and its relation to the stomach or duodenum.
- Successful drainage was achieved in 86% of patients of pseudocyst, whereas 11% of the patients had a recurrence following this approach.

TRANSAMPULLARY PANCREATIC STENT

- Transampullary pancreatic stents are used to treat chronic pancreatitis, pancreatic ductal disruption, pancreatic fistulas, and pseudocysts.
- This method of pseudocyst drainage has been tried in patients with pseudocysts communicating with the main pancreatic duct.
- The stents are inserted through the ampulla, along the pancreatic duct, and into the pseudocyst lumen.
- When it is difficult to insert the stent into the pseudocyst lumen, the stent tip is positioned as close to the site of communication between the pseudocyst and pancreatic duct as possible.
- Complications related to thisMild post-procedure pancreatitis, bleeding, and abscess formation are all risks of this approach.
- The abscess develops as a result of stent blockage.
- Endoscopic transpapillary nasopancreatic drainage has lately been employed to treat pseudocysts in unusual places such as the mediastinum, intrahepatic, intrasplenic, and pelvic regions.
- Transampullary drainage and pancreatic stenting may be used to empty a pseudocyst in some patients.
- Endoscopic and percutaneous drainage complications include sepsis, bleeding, shock, renal failure, and ventilator-dependent respiratory failure.
- Even after endoscopic and percutaneous draining, some individuals require surgical intervention.
- These percutaneous/endoscopic drainage failures were easily discovered by ERCP, which detects major pancreatic duct stricture and other pancreatic ductal abnormalities. Percutaneous drainage can be used to treat pancreatic duct type I and II anomalies.
- Types V through VII types of ducts are associated with severe duct strictures and stones, they are handled with surgical or endoscopic internal drainage or surgical resection.
- Types III and IV can be treated using either of these approaches. However, due of the presence of underlying duct strictures, percutaneous drainage alone may be associated

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with greater risks of recurrence in this condition.

- Some salvage procedures are followed after failed percutaneous and endoscopic drainage.
- These procedures include cyst debridement and external drainage, internal pseudocyst drainage, and pancreatic resection.
- Morbidity following these salvage procedures was about 56% with complications include haemorrhage, wound infection, and pulmonary complications.

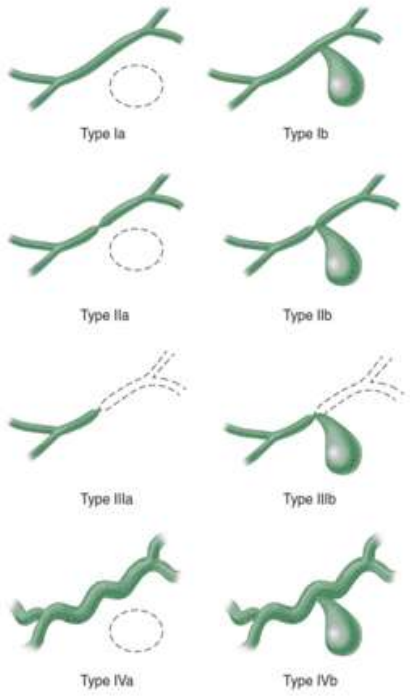

FIGURE 93.4 Nealon classification of pancreatic ductal disruption and pseudocyst formation. Type I is a normal main pancreatic duct. Type II is a pancreatic duct stricture. Type III is pancreatic duct occlusion (disconnected pancreatic duct syndrome). Type IV depicts chronic pancreatitis. Subtypes *a* represent no radiographically demonstrable communication between the pancreatic duct and the pseudocyst. Subtypes *b* represent communication between the pancreatic duct and the pseudocyst.

Fig 9

- The preferred approach for uncomplicated pseudocysts requiring surgical intervention is internal drainage.
- Internal drainage can be done by three methods viz cystojejunostomy to a Roux-en-Y jejunal limb, cystogastrostomy and cystoduodenostomy.
- Cystojejunostomy is the most commonly used method andis mostly done when the pseudocyst is located at the base of the transverse mesocolon and is not adherent to the gastric or duodenal wall.

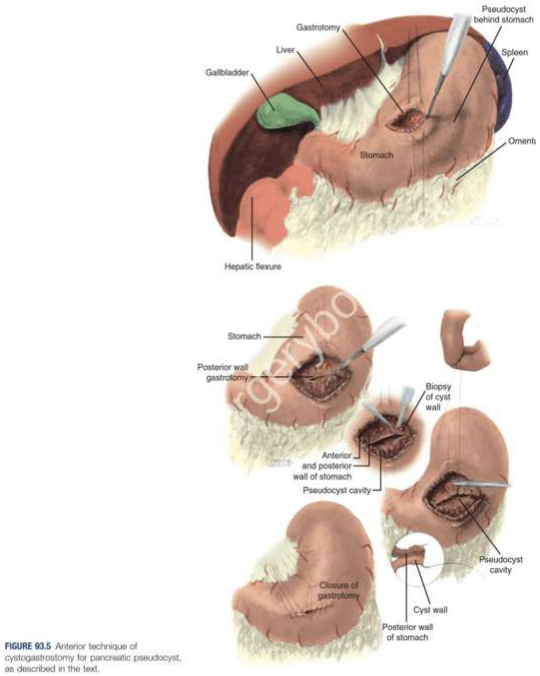

FIGURE 93.5 Anterior technique of cystogastrostomy for pancreatic pseudocyst, as described in the text.

Fig 10

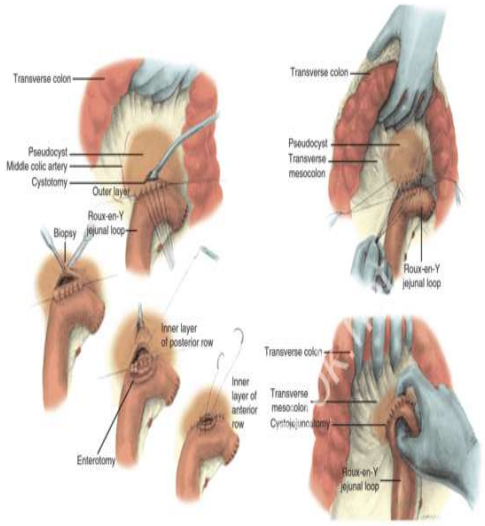

FIGURE 93.6 Roux-en-Y cystojejunostomy for pancreatic pseudocyst, focusing on the actual anastomosis between the pseudocyst and the Roux-en-Y limb.

Fig 11

- Cystogastrostomy is a quicker treatment utilised when the pseudocyst is adhered to the stomach or duodenal wall.

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- The least common operation is cystoduodenostomy, which is usually utilised for pseudocysts in the head or uncinate process that is within 1 cm of the duodenum's lumen.
- Cystoduodenostomy is conducted in the same manner as cystogastrostomy. Both of these treatments involve opening the lateral wall of the duodenum and establishing connection between the pseudocyst and the duodenum.
- This communication is made possible through medial duodenotomy. Because of the hazards associated with cystoduodenostomy, such as duodenal leak and fistula, it is rarely done nowadays.
- **Laparascopic drainage procedures**:
- Laparoscopic approaches are beneficial in cases of large retrogastric pseudocysts. It includes transgastric and extragastric approaches.
- It also allows biopsy of the cyst wall and cyst debridement.

Natural orifice transluminal endoscopic surgery (NOTES)

- **Cystogastrostomy**
- There are various surgical procedures available for different pancreatic diseases. Resections include pancreatic head resections (classical, pylorus-preserving and duodenum-preserving partial pancreatoduodenectomies), segmental resections, distal resections, total pancreatectomies, enucleations and others.
- In addition, palliative procedures such as biliodigestive anastomosis and gastric bypass procedures are frequently carried out, as well as special procedures such as necrosectomy or pancreatic pseudocyst drainage (cystogastrostomy or cysto-jejunostomy)[8]
- It produces comparable outcomes to the open and laparoscopic approaches. It combines EUS-guided pseudocyst drainage with transoral cystogastrostomy anastomosis through a flexible endoscopic shaft and a stapler.
- This method is also used to debride infected pancreatic necrosis. Some pseudocysts respond better to pancreatic excision.
- In the instance of a pseudocyst in the body or tail of the pancreas, a distal pancreatectomy is performed. Distal pancreatectomy is a difficult procedure due to the presence of peripancreatic and peripseudocyst inflammation.
- A Roux-en-Y pancreaticojejunostomy is performed after a distal pancreatectomy. This procedure is used to decompress a clogged or atypical pancreatic duct.[1]
- In patients with symptomatic pseudocysts in the head of the pancreas, pancreaticoduodenectomy is required.
- In this case, pylorus-preserving pancreaticoduodenectomy is the choice of procedure. Less commonly done procedures, such as duodenum-preserving resection of the head of the pancreas, may be performed in some patients.

- **External drainage**:
- External drainage of a pancreatic pseudocyst is indicated when gross infection is present at the time of surgery or presence of immature, thin-walled pseudocyst that will not allow for internal drainage.

COMPLICATIONS

- Infections, haemorrhage, obstruction or compression of adjacent structures and rupture are the most frequently reported complications.

INFECTION:

- Although some pseudocysts contain bacteria, the pseudocyst fluid is not purulent, and patients show no clinical signs of infection.
- In individuals with pseudocysts, however, genuine infections were manifested by fever, leukocytosis, and increased discomfort.
- Aspiration of purulent fluid from the pseudocyst indicates infection.
- The Atlanta International Symposium defines these patients as having pancreatic abscess, and the term "infected pseudocyst" should be avoided.
- The bacteriology of pancreatic abscesses varies greatly, although up to 60% contain aerobic and anaerobic gram-negative organisms.
- A pancreatic abscess is a medical ailment that can be treated with percutaneous drainage. Furthermore, percutaneous drainage has a lower fatality rate and avoids a large open surgical procedure.
- However, certain patients mayneed operative external drainage. Percutaneous catheters often do not provide for quick drainage or cannot fully address multi-loculated collections.

- Interventional radiologists strategies include upsizing drainage catheters, biweekly imaging, and aggressive manipulation to break up loculations.
- These are not always effective strategies,
- .Open operating drainage facilitates the complete evacuation of all infected material and outside drains can be placed under direct vision.
- Percutaneous endoscopic techniques have most recently allowed necrotic tissue debridement in direct endoscopic vision.
- Uteroscopes are advanced into the retroperitoneum via earlier percutaneous drainage channels, and devitalised tissues are removed while the cavity is constantly irrigated.
- It produces great effects while putting the patients under minimal stress.
- According to recent research, transampullary drainage is not only an effective way to resolve an abscess.
- Multivariable analysis revealed that patients who received pancreatic duct stenting improved. If the patient does not improve, surgical drainage should be performed.[(1)]

Haemorrhage:

- Arterial haemorrhage occur in a few patients with pancreatic pseudocysts.
- The splenic artery is the most common source of pseudocyst-associated bleeding.
- The gasproduodenal and pancreaticoduodenal arteries also cause significant hemorrhagic events.
- Bleeding also occurs from the portal, superiormesenteric, or splenic veins but that bleeding is less common.
- The aetiology of arterial bleeding begins with artery wall degradation, which leads to pseudo aneurysm development and rupture.
- Sentinel haemorrhage occurs after massive haemorrhage. As a result, bleeding linked with a pseudocyst should be checked as soon as possible.
- Angiography is required for diagnosis and provides a treatment option.
- The pseudo aneurysm is initially embolized, a process performed by trained radiologists. [(1)]
- Most haemorrhages are treatable with modern embolic treatments.
- When embolic treatment fails or the patient becomes hemodynamically unstable, emergent surgical exploration is required.
- The pseudocyst should be drained with large-bore catheters once the blood vessel has been ligated.
- If resection is required, the most usual surgeries are distal pancreatectomy, splenectomy, and splenic artery ligation. Rarely, an emergency pancreaticoduodenectomy is required.

Obstruction:

- Because of their mass effect on other structures, pancreatic pseudocysts may become symptomatic.
- While duodenal obstruction is secondary to pseudocyst formation as the most common type of mechanical obstruction, stomach obstruction, oesophagus, jejunum and colon may be identified.
- Hypertension, splenomegaly, and stomach varices can be caused by obstruction of the mesenteric vasculature and the portal venous system.
- Other retroperitoneal structures, such as the inferior vena cava and ureters, are also obstructed by pseudocysts.
- Pseudocysts with mediastinal and pleural extension have also been recognised as restricting cardiac output.
- Pseudocyst can also induce biliary blockage, which can lead to problems like jaundice, biliary cirrhosis, and cholangitis.
- Cholangiography is used to diagnose biliary blockage in a patient with a pancreatic pseudocyst.

RUPTURE:

- Spontaneous rupture, the least common complication of the development of pseudocysts, occurs in less than 3% of patients, but it may lead to dramatic clinical manifestations.
- Spontaneous rupture of a pseudocyst into the peritoneal cavity can result chemical peritonitis which causes severe acute abdominal pain.
- These patients are treated as a surgical emergency, especially those without a known pseudocyst.
- In these cases,history of pseudocyst, acute abdominal pain should raise the intraperitoneal

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rupture or rupture into an associated hollow viscus, most commonly a segment of the gastrointestinal tract.

- Rupture may be due to progressive growth, but it also signify an infected or hemorrhagic pseudocyst.
- Silent rupture of a pseudocyst also occur. Some pseudocysts are presumed to resolve by rupture or fistulization into an associated portion of the stomach.
- Pseudocysts that rupture silently anteriorly into the peritoneal cavity or posteriorly into the pleural cavity may lead to the development of pancreatic ascites or pancreatic pleural effusion.
- Endoscopic and minimally invasive therapy for complications of acute pancreatitis:

ENDOSCOPIC NECROSECTOMY:

- Endoscopic necrosectomy has developed as a therapeutic alternative to surgery for individuals with organised pancreatic necrosis that is near to the gastric or duodenal wall.
- This procedure comprises either direct puncture or endoscopic ultrasound-guided needle penetration and subsequent wire guide access to the necroma.
- Once the tract has been accessible, it is dilated with a graduated dilating catheter, needle knife sphincterotome, or cystotome.
- Following dilatation to 15 to 20 mm with a balloon dilator to facilitate passage of an upper endoscope into the necroma.
- Debridement is then performed using a mix of endoscopic accessories.
- Tract maintenance is accomplished by inserting two or more pigtail stents or a self-expanding metal stent through the gastric orduodenal wall into the cavity.It allows repeated access and debridement after initial necrosectomy.[1]
- In some circumstances, a nasocystic drain may be required for continuous lavage.
- Although the procedure is beneficial in preventing surgery and pancreaticocutaneous fistulas, it has significant drawbacks, including a high morbidity and mortality rate.
- In some circumstances, if the necroma is not adhering to the stomach wall, air supplied during endoscopic insufflation might dissect easily into the smaller sac, resulting in air emboli.
- Furthermore, large-diameter balloon dilatation may raise the risk of bleeding, particularly in patients with left-sided portal hypertension and stomach varices.
- Another disadvantage of endoscopic necrosis is that it requires numerous procedures that may not be possible on consecutive days with the same patient.

PERCUTANEOUS DRAINAGE AND COMBINED MODALITY THERAPY:

- Another method for Necrosis drainage is large-caliber percutaneous draining in necroma.
- Drains were gradually expanded to a maximum of 30 French (F) for the debridement of liquefied necrotic tissue.
- The biggest disadvantage of this approach is the high rate of chronic pancreaticocutaneous fistula development.
- This is especially common in patients with disconnected duct syndrome, in which drains were inserted in the disconnected portion of the pancreas.
- Before percutaneous drainage, when disconnected duct syndrome is suspected based on central necrosis with normal perfused distal pancreatic tissue, endoscopic or magnetic resonance pancreatography should be performed.[1]
- A combined-modality approach involves placement of large-caliber percutaneous drains into the necroma followed by endoscopic placement of double pigtail stents into the necrotic cavity.
- The pigtail stents are placed to create an internal fistula. Then the transenteric stents were allowed for redirection of pancreatic juice into the GI tract.
- In disconnected duct syndrome patients, it prevents the formation of chronic pancreaticocutaneous fistula.
- A recent comparison among standard percutaneous drainage and combined modality therapy has shown that the latter technique lowers hospital stay, external drainage duration and radiographic resource utilisation.
- In the combined drainage group there was no procedural mortality.
- Combined modality drainage is only used in patients whose necrosis is confined to the lesser sac where the cavity is within close proximity to the gastric or duodenal wall.[1]

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RELEVANCE:

- Acute pancreatitis is an inflammatory condition of the pancreas most commonly caused by bile stones or excessive use of alcohol.
- In most patients, the disease takes a mild course, where moderate fluid resuscitation, management of pain and nausea, and early oral feeding result in rapid clinical improvement.
- The severe form comprising about 20–30% of the patients is a life-threatening disease with hospital mortality rates of about 15% .
- The most commonly used classification system for acute pancreatitis is the 2012 revision of the Atlanta classification and definitions based on international consensus.
- Most patients (80–85%) will develop a mild disease course (self-limited, mortality < 1–3%), but around 20% will have a moderate or severe episode of AP, with a mortality rate from 13 to 35% .
- Thus, it is import ant to diagnose (or better predict) an episode of severe acute pancreatitis (SAP), and to identify the patients with high risk of developing complication[9]
- CRP is a kind of the non-specific acute-phase reac tive protein generated from the hepatocytes under the tumor necrotic factors released by the mononuclear macrophages in case of inflammation and injury of tissues, with the effects to facilitate the phagocytosis, activate the complement and regulate the immune func- tions.
- The level of CRP is always in the physiologically normal range, but CRP arises rapidly several hours after any acute infection or tissue injuries, and peak level is attained within 24 and 48 h.
- CRP serves as an indicator reflecting the inflammation and evaluating the severity of pancreatitis[10]
- LDH, as a kind of glycolytic enzyme, extensively distributes in the cytoplasm of tissues, mainly in the myocardium, skeletal muscle, kidney and liver,
- SAP patients are more susceptible to the dysfunctions in heart, lung or kidney, with an increase in the level of LDH.
- Thus, LDH, in spite of its poor organ specificity, can effectively reflect the pancreatitis-caused damage to other organs, and, accordingly, should be served as an indicator evaluating the progression and severity of Acute pancreatitis[10]
- Thus assessing these inflammatory markers within 48 hrs of onset of symptoms can easily predicts the severity of acute pancreatitis
- By this assessment we can plan early intervention rather than waiting for other cumbersome classification and also acquiring an imaging especially a contrast ct scan in our setup.

II. METHODOLOGY:

- **Study setting**

Department of General Surgery, Govt. Medical College, Thrissur.

Study design

Cross sectional study

- **Study population**

Patients admitted with acute pancreatitis in Govt. Medical College, Thrissur.

Inclusion criteria

Patients admitted with acute pancreatitis and those who have done CT evaluation in Department of Surgery, Govt. Medical College, Thrissur.

Exclusion criteria

- Patients who has diseases which will elevate CRP and LDH levels such as Rheumatoid arthritis,Lupus,inflammatory bowel diseases such as crohns disease and ulcerative colitis,those with diagnosed tumours,pericarditis,those with severe respiratory tract infection,UTI
- Those patients treated with antibiotics and anti inflammatory drugs prior to admission to study setting
- Those patients on immunosuppressive drugs
- Age more than 65 yr old

- **Sample size formula**

Sample size was calculated using the formula given below and it comes to sample population of 91.Variables have been obtained from study conducted by Dr Gayathri at Govt medical college coimbatore,standard deviation of CRP being 13.77 in severe group (higher standard deviation value kept to calculate sample size)and absolute error 3 formula applied as follows

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$$n = \left(\frac{(Z_{\alpha/2})(\sigma)}{E}\right)^2$$

Standard deviation =13.77, absolute error (E)= 3

- **Study Tools**
  1. Clinical evaluation, CT severity index
  2. Laboratory records

- **Study Period**

One year from the date of Ethical Committee Clearance.

Methodology

All patients diagnosed to have acute pancreatitis by clinical methods laboratory values and imaging assessed for severity by CT severity index and C Reactive protein quantitative ,LACTATE DEHYDROGENASE had been sent at the time of admission and compared with CT severity index and mean value to mild, moderate and severe form of acute pancreatitis obtained .All results hab been entered into an excel sheet and tabulated and analysed using SPSS 17

Statistical Analysis

Student t-test has been used to compare two arithmetic means. Chi- square test has been used to compare two proportions or percentages. 5%or <.05 taken as the level of statistical significance (p),which falls in above 95%confidence interval

EXPECTED OUTCOME OF THE STUDY

This study will help to evaluate relation of the elevation of CRP and LDH associated with differing severity levels of acute pancreatitiswhen compared with CTSI and helps in early intervention

ETHICAL ISSUES EXPECTED AND HOW THESE WILL BE ADDRESSED

There are no major ethical concerns with the study as tools required for study are now currently in practice and easily available at the centre.Informed written consent obtained prior from patients .

I. RESULTS:

91 patients who have been admitted with acute pancreatitis less than 65yr old without any other immune suppressive condition ,assessed with CRP and LDH within 48 hrs of onset of symptoms and the severity is compared with CTSI .

GENDER DISTRIBUTION

Sex	Frequency	Percent
Male	79	86.8
Female	12	13.2
Total	91	100

Table 8

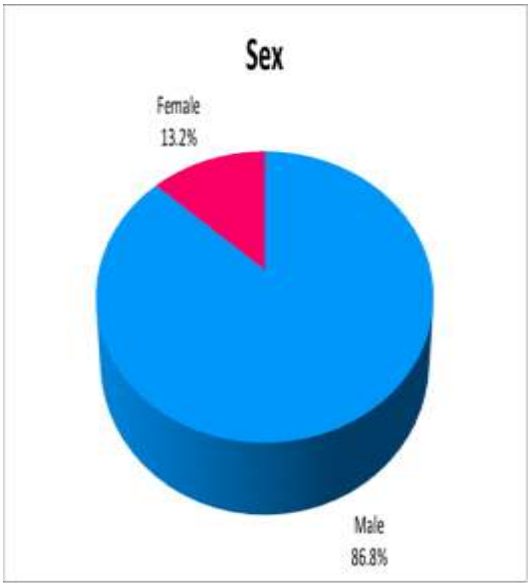

Fig 12

In this study 86.8% turnout to be male population and 13.2% females have been diagnosed with acute pancreatitis.

AGE DISTRIBUTION

Age	Frequency	Percent
<30	13	14.3
30-60	66	72.5
>60	12	13.2
Total	91	100

Table 9

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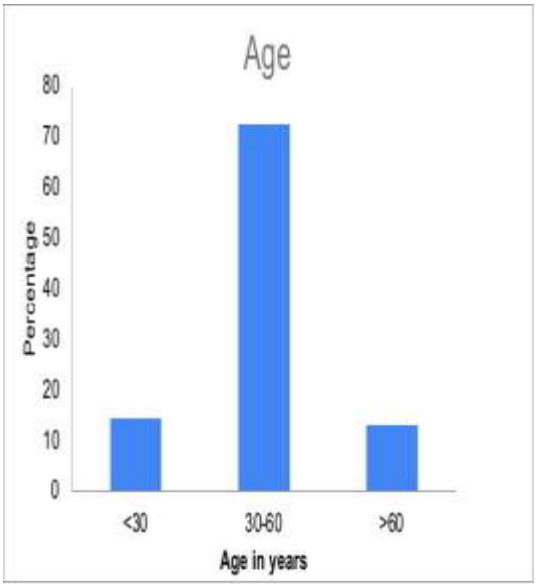

Fig 13

During this study most of patients falls in age group between 30 to 60 frequency comes around 72.5%, those who are more than 60 but less than 65yrs comes up to 13.2% only and those less than 30 yrs of age comes around 14%

CTSI DISTRIBUTION

CTSI	Frequency	Percent
Mild	60	65.9
Moderate	17	18.7
Severe	14	15.4
Total	91	100

Table 10

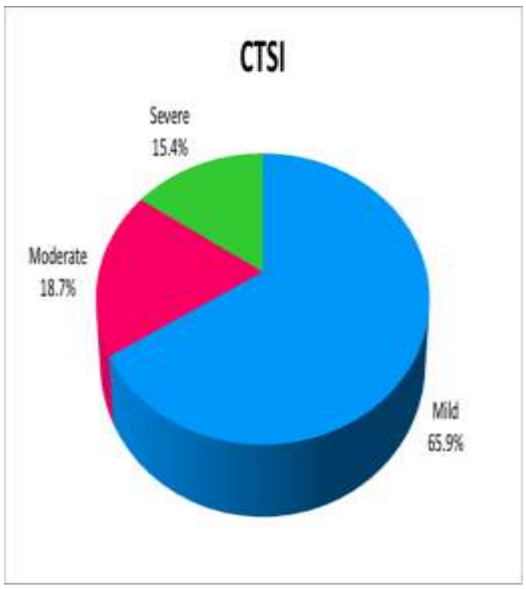

Fig 14

Here Those who have admitted with disease have been done with imaging study using CT and severity calculated via CTSI and 65.9% population fall in mild category and 15.4% had severe acute pancreatitis and around 18.7% comes in moderate category

CRP DISTRIBUTION

C REACTIVE PROTEIN(mg/dl)	Frequency	Percent
≤10	33	36.3
11-100	35	38.5
101-200	10	11
201-300	11	12.1
>300	2	2.2
Total	91	100

Table 11

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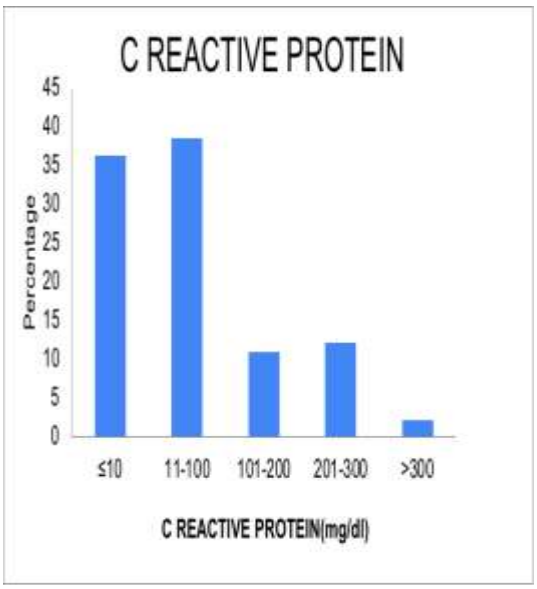

Fig 15

Here c reactive protein quantitative values had been obtained via blood test and around 64.8.% had value less than 100mg/dl,while, around 14.3%had value more than 200 mg/dl when combined and 11% falls in between this range

LDH DISTRIBUTION

LACTATE DEHYDROGENASE(IU/L)	Frequency	Percent
≤200	3	3.3
201-300	48	52.7
301-400	9	9.9
401-500	10	11
501-600	7	7.7
>600	14	15.4
Total	91	100

Table 12

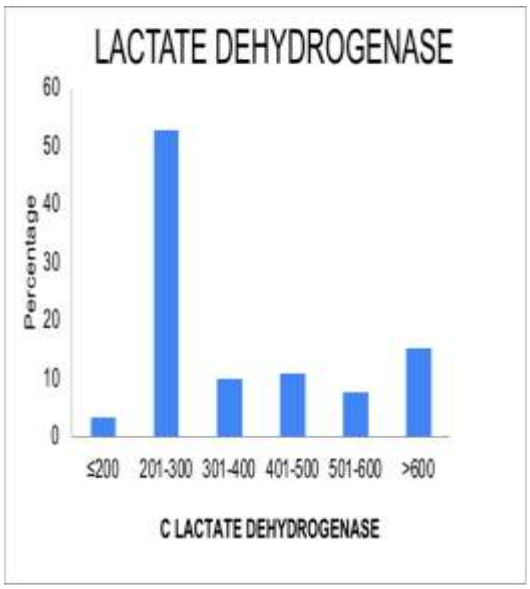

Fig 16

Here we took samples to ascertain LDH values in which around 65.9% value comes less than 400 IU/L which approximately falls in to normal Ldh range and around 15.4% had values above 600Iu/L and rest 18.7% falls category in between

Sex	Severity						Total	
	Mild		Moderate		Severe			
	N	%	N	%	N	%	N	%
Male	48	81.4	17	94.4	14	100	79	86.8
Female	11	18.6	1	5.6	0	0	12	13.2
Total	59	100	18	100	14	100	91	100

	χ^2	df	p
Chi-Square test	4.577	2	0.101

Table 13

Above depicted table we could see the gender predisposition to differing severity levels ,notable point is that out of 79 males in study 48 males had mild disease and out of 12 females 11 had mild disease and only 1 comes in moderate category,while in severe disease all 14 ,ie 100% were males

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Age in years	Severity						Total					
	Mild		Moderate		Severe							
	N	%	N	%	N	%	N	%				
<30	9	15.3	2	11.1	2	14.3	13	14.3				
30-60	42	71.2	14	77.8	10	71.4	66	72.5		χ^2	df	p
>60	8	13.6	2	11.1	2	14.3	12	13.2	Chi-Square test	.329	4	0.988
Total	59	100	18	100	14	100	91	100				

Table 14

Here age distribution according to severity can be analysed and most of the disease severity falls between 30 to 60 years

C REACTIVE PROTEIN(mg/dl)	Severity						Total	
	Mild		Moderate		Severe			
	N	%	N	%	N	%	N	%
≤10	33	55.9	0	0	0	0	33	36.3
11-100	26	44.1	9	50	0	0	35	38.5
101-200	0	0	8	44.4	2	14.3	10	11
201-300	0	0	1	5.6	10	71.4	11	12.1
>300	0	0	0	0	2	14.3	2	2.2
Total	59	100	18	100	14	100	91	100

	χ^2	df	p
Chi-Square test	108.894	8	<0.001

Table 15

The table above shows the comparison of severity score obtained by CTSI and c reactive protein value obtained during studyhere those with mild severity had c reactive protein values below 100 mg/dl and in which 55.9% had values less than or equal to 10 while moderate severity majority falls under 101 to 200 mg/dl

The severe category 71.4% had values above 200 but less than 300 ,while 14.3% had CRP more than 300 and p value <.001

LACTATE DEHYDROGENASE(/dl)	Severity						Total	
	Mild		Moderate		Severe			
	N	%	N	%	N	%	N	%
≤200	3	5.1	0	0	0	0	3	3.3
201-300	48	81.4	0	0	0	0	48	52.7
301-400	8	13.6	1	5.6	0	0	9	9.9
401-500	0	0	10	55.6	0	0	10	11
501-600	0	0	5	27.8	2	14.3	7	7.7
>600	0	0	2	11.1	12	85.7	14	15.4
Total	59	100	18	100	14	100	91	100

	χ^2	df	p
Chi-Square test	139.818	10	<0.001

Table 16

In the above depicted table we can see comparisons between CTSI and Lactate dehydrogenase values obtained

Majority of mild cases around 81.4% fall in value between 201 to 300 ,while severe cases had value more than 600 IU/L and it accounts around 85.7% among severe cases

55.6% cases falls in moderate category and their value lies between 400 and 500 and comparison p value <.001

II. DISCUSSION :

- From this crossectional study accumulating CTSI,CRP and LDH values, we could see the following results:
- Around 86.8% were males to which alcoholism was attributed as a factor for this,other important factor was gall stone
- And age distribution shows shows most between 30 to 60 years of age corresponding to our social scenario
- When analysing the CTSI we can see that 65.9% in the mild category and 18.7 % in moderate and 15.4% in severe acute pancreatitis category
- This comparison when over layer on CRP and LDH values we can see that around 74% patients had CRP value less than 100 ,and11% had values between 101 and 200 and around 14.3% more than 200 and p value <.001 which is statistically significant

- LDH values around 65.9% had values less than 400 and those with >600 are about 15.1%
- Around 18% comes in between these two ranges
- As the p value for CRP and LDH falls <.001,statistically it's very significant
- In the United States, more than 75% of cases of acute pancreatitis are attributable to either gallstones or alcohol.
- In general, a diagnosis of acute pancreatitis can be made with the presence of two of the following three features: (1) characteristic abdominal pain (acute onset of severe, persistent epigastric pain often radiating to the back);
- (2) serum lipase (or amylase) levels at least three times greater than the upper limit of normal; and
- (3) findings of acute pancreatitis on contrast-enhanced CT or MRI.
- Approximately 80% of cases of acute pancreatitis are mild, associated with minimal systemic derangements, and generally resolve within 5 to 7 days, even with minimal therapy.
- Severe acute pancreatitis accounts for about 20% of cases and is defined as acute pancreatitis associated with one or more of the following:
- pancreatic necrosis,
- distant organ failure, and
- the development of local complications such as haemorrhage, abscess, or pseudocyst.
- The mortality rate associated with severe acute pancreatitis ranges from 10% to 20%, with half of the deaths in the first 2 weeks as the result of SIRS-induced multi system organ failure and the remaining occurring later as the result of pancreatic necrosis/infection.[11]
- So To predict the course of disease easily we can assess the inflammatory markers like CRP and Lactate Dehydrogenase which is cheap and readily available and results will be obtained with in hours
- So we can see that both CRP and LDH values are statistically significant and comparable to CTSI
- In a prospective study done in Sri Ramachandra Medical College and Hospital, Chennai, India, from April 2012–September 2014.
- All patients with a diagnosis of acute pancreatitis were included in this study (110 consecutive patients).
- Limitation of study is it cannot find out the effect of disease in long progression
- Alsopatients with other inflammatory could not be assessed in this study,
- Strength of study is that it can assess the immediate outcome of patient.
- Patients with chronic pancreatitis and pancreatic malignancy were excluded from the study. Patients were classified into mild, moderate and severe acute pancreatitis based on Ranson's score, Glasgow scoring and CT severity index (CTSI).
- Among the 50 patients who required intensive care the minimum duration of stay was 5 days and the maximum was 21 days. Admission values in patients who died had a high CRP value (1.6 minimum value and 4.3 max value) and maximum LDH value of 980.[12]
- In another study , aim of this study was to investigate the associations of high serum lactate dehydrogenase (LDH) levels with AP severity and systemic complications. Methods. AP patients treated from July 2014 to December 2020 were retrospectively enrolled.
- AP severity, the Ranson, MODS, BISAP, APACHE II, and CTSI scores were significantly higher in the elevated LDH group than those in the normal group was the conclusion of study [13]
- In another study included 26 patients with severe acute pancreatitis identified among the 211 patients with acute pancreatitis admitted to our hospital between January 2014 and December 2018.
- Patients with and without WON (WON and non-WON groups, respectively) were compared to identify potential factors involved in the onset of this condition.
- The study could conclude that maximum CRP level was identified as a predictive factor for the onset of WON, and a high proportion of patients with WON exhibited elevated CRP levels within 3 days after diagnosis.[14]
- Another study conducted in Tamilnadu had 100 patients as study population which assessed ct severity with CRP and LDH ,This study concluded that there is significant relation for CRP and ldh to predict severity of acute pancreatitis [15]

III. CONCLUSION:

- From this study we could conclude that we can assess the severity of acute pancreatitis and CRP,LDH values when the CTSI for severity is available
- Thus this inflammatory markers can serve as simple and efficient tools to aid in treatment especially in early intervention and resuscitation of patients
- Those patients who will go into serious morbidities can be easily identified using these markers.
- From this study we could conclude that those patients with CRP <100mg/dl and LDH less than 300 IU/L fall to mild severity
- Those with CRP more than 200 and LDH more than 600 falls in to severe acute pancreatitis
- So even without other sophisticated investigations we can assess the severity of acute pancreatitis,using these inflammatory markers

REFERENCES:

[1]. Shackelfords surgery of Alimentary Tract,Eight edition
[2]. Sabiston Textbook surgery,21st edition
[3]. SRB's Manual of surgery,6th edition
[4]. Komolafe O, Pereira SP, Davidson BR, Gurusamy KS. Serum C- reactive protein, procalcitonin, and lactate dehydrogenase for the diagnosis of pancreatic necrosis. Cochrane Database of Systematic Reviews. 2017(4).
[5]. Cui J, Xiong J, Zhang Y, Peng T, Huang M, Lin Y, Guo Y, Wu H, Wang C. Serum lactate dehydrogenase is predictive of persistent organ failure in acute pancreatitis. Journal of critical care. 2017 Oct 1;41:161-5.
[6]. Frossard JL, Hadengue A, Pastor CM. New serum markers for the detection of severe acute pancreatitis in humans. American journal of respiratory and critical care medicine. 2001 Jul 1;164(1):162-70.
[7]. Schwartz principles of surgery,11th edition
[8]. Gillen S, Kleeff J, Kranzfelder M, Shrikhande SV, Friess H, Feussner H. Natural orifice transluminal endoscopic surgery in pancreatic diseases. World Journal of Gastroenterology: WJG. 2010 Aug 8;16(31):3859.
[9]. Leppäniemi A, Tolonen M, Tarasconi A, Segovia-Lohse H, Gamberini E, Kirkpatrick AW, Ball CG, Parry N, Sartelli M, Wolbrink D, van Goor H. 2019 WSES guidelines for the management of severe acute pancreatitis. World journal of emergency surgery. 2019 Dec;14(1):1-20.
[10]. Yin X, Xu J, Zhang Q, Yang L, Duan Y. Quantification analysis of lactate dehydrogenase and C-reactive protein in evaluation of the severity and prognosis of the acute pancreatitis. Cellular and Molecular Biology. 2020 Apr 20;66(1):122-5.
[11]. Greenfield's surgery,surgical principle and practice,sixth edition
[12]. Vengadakrishnan K, Koushik AK. A study of the clinical profile of acute pancreatitis and its correlation with severity indices. International journal of health sciences. 2015 Oct;9(4):410.
[13]. Huang DN, Zhong HJ, Cai YL, Xie WR, He XX. Serum Lactate Dehydrogenase Is a Sensitive Predictor of Systemic Complications of Acute Pancreatitis. Gastroenterology Research and Practice. 2022 Oct 25;2022.
[14]. Fujiwara J, Matsumoto S, Sekine M, Mashima H. C- reactive protein predicts the development of walled- off necrosis in patients with severe acute pancreatitis. JGH Open. 2021 Aug;5(8):907-14
[15]. Gayathri B. Study of Role of C-Reactive Protein and Lactate Dehydrogenase as Prognostic Factors of Severity in Acute Pancreatitis (Doctoral dissertation, Coimbatore Medical College, Coimbatore).

LIST OF ABBREVIATIONS:

CRP – c reactive protein
LDH. Lactate dehydrogenase
SIRS. Systemic inflammatory response syndrome
MODS Multiple organ Dysfunction syndrome
MRCP. Magnetic resonance cholangiopancreaticography
ERCP Endoscopic retrograde cholangiopancreaticography
EUS Endoscopic ultrasound
CECT. Contrast enhanced computed tomography

CARS. Compensatory anti inflammatory response syndrome
CBD Common Bile Duct
VARD Video Assisted Retroperitoneal Debridement