ROLE OF C- REACTIVE PROTEIN, SERUM AMYLASE AND APACHE II SCORING SYSTEM IN PREDICTING THE SEVERITY OF ACUTE PANCREATITIS

BY

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Dissertation submitted to the

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In partial fulfillment of the requirements for the degree of

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DEPARTMENT OF GENERAL SURGERY

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APRIL- 2017

DECLARATION BY THE CANDIDATE

I, **Dr. M.THAMARAI KANNAN** , solemnly declare that this Dissertation "**ROLE OF C- REACTIVE PROTEIN, SERUM AMYLASE AND APACHE II SCORING SYSTEM IN PREDICTING THE SEVERITY OF ACUTE PANCREATITIS "** was done by me in the Department of General Surgery, Thanjavur Medical College and Hospital , Thanjavur under the Guidance and Supervision of my U **Unit Chief Dr. V. KOPPERUNDEVI M.S**., Department of General Surgery, Thanjavur Medical College, Thanjavur during the period from October 2015 to September 2016.

This Dissertation is submitted to The Tamilnadu Dr. M.G.R Medical University , Chennai in partial fulfilment of University requirements for the award of M.S Degree (Branch –I) in GENERAL SURGERY.

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This is to certify that this dissertation titled " **ROLE OF C REACTIVE PROTEIN, SERUM AMYLASE AND APACHE II SCORING SYSTEM IN PREDICTING THE SEVERITY OF ACUTE PANCREATITIS "** is a bonafide research work done by **Dr.THAMARAI KANNAN.M ,** in partial fulfillment of the requirement for the degree of **M.S. GENERAL SURGERY – BRANCH I** to be held in April 2017.

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CERTIFICATE

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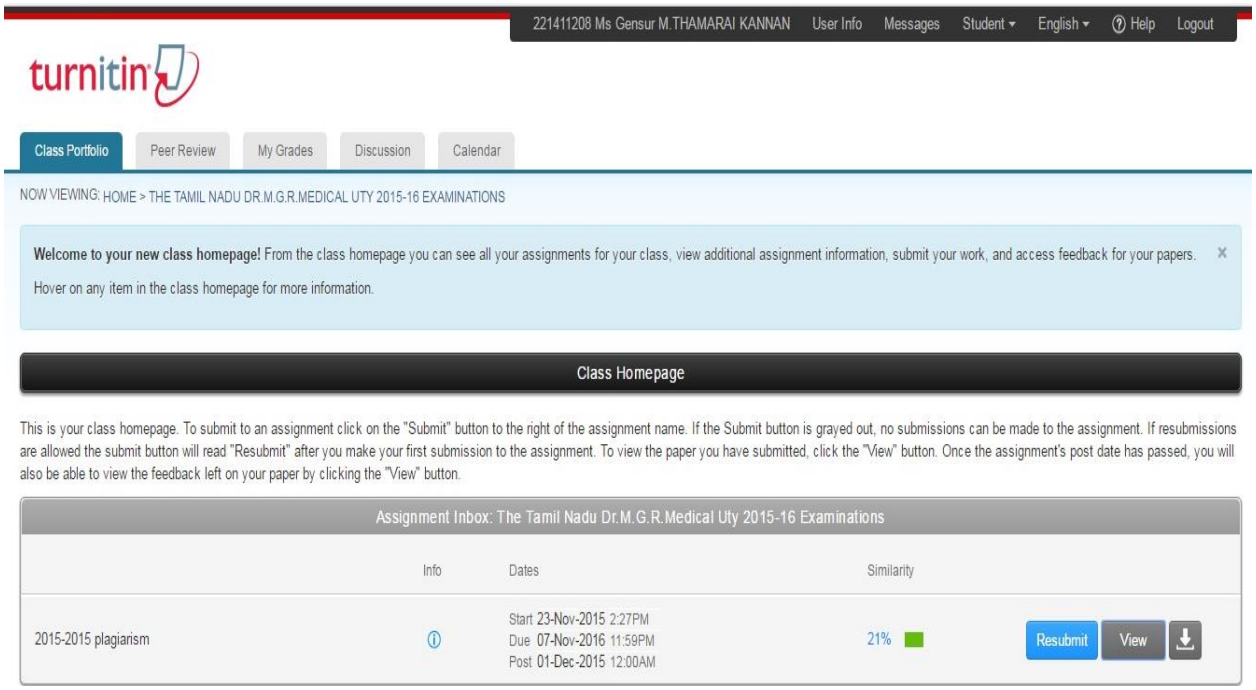
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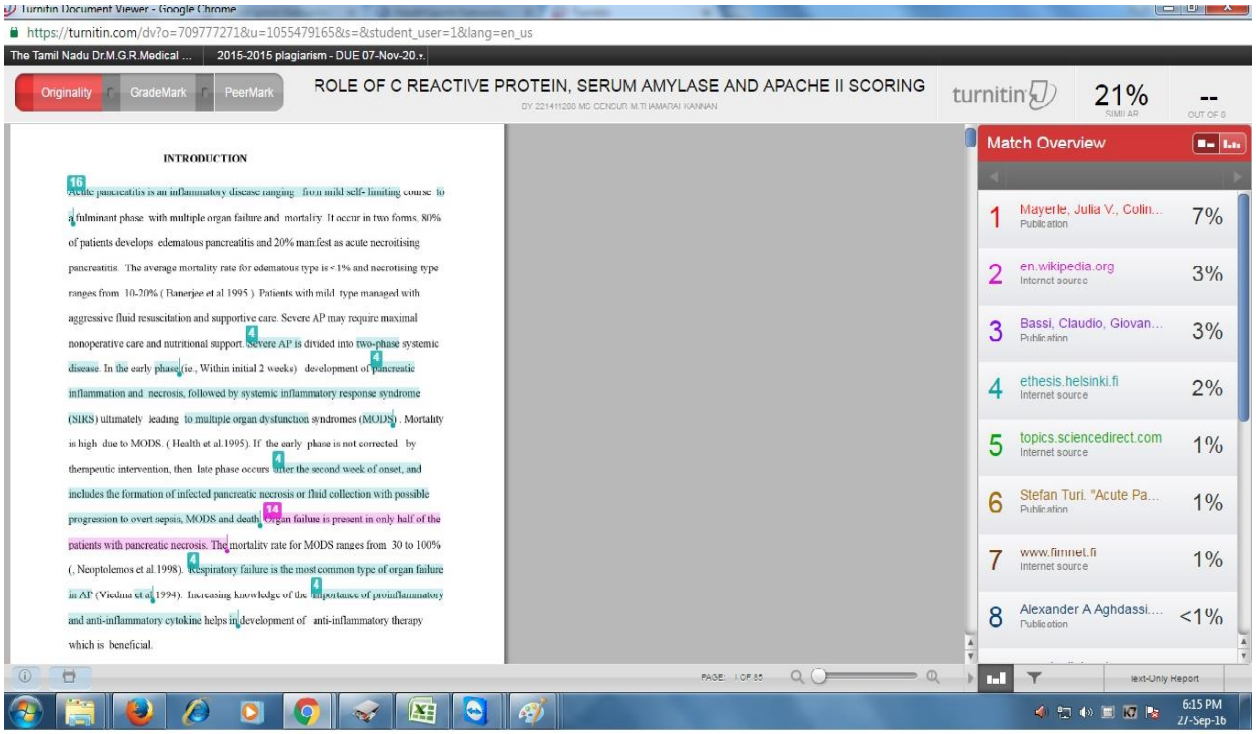
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INTRODUCTION
Acute pancreatitis is an inflammatory disease ranging from mild self- limiting course to a fulminant phase with multiple organ failure and mortality. It occur in two forms. 80% of patients develops edematous pancreatitis and 20% manifest as acute necroitising pancreatitis. The average mortality rate for edematous type is < 1% and necrotising type ranges from 10-70% (Banerjee et al 1995). Patients with mild type managed with aggressive fluid resuscitation and supportive care. Severe AP may require maximal nonoperative care and nutritional support. Severe AP is divided into two-phase systemic disease. In the early phase (ie., Within initial 2 weeks) development of pancreatic inflammation and necrosis, followed by systemic inflammatory response syndrome (SIRS) ultimately leading to multiple organ dysfunction syndromes (MODS). Mortality is high due to MODS. (Health et al.1995). If the early phase is not corrected by therapeutic intervention, then late phase occurs after the second week of onset, and includes the formation of infected pancreatic necrosis or fluid collection with possible progression to overt sepsis, MODS and death. Organ failure is present in only half of the patients with pancreatic necrosis. The mortality rate for MODS ranges from 30 to 100% (. Neoptolemos et al.1998). Respiratory failure is the most common type of organ failure in AP (Vinuela et al 1994). Increasing knowledge of the importance of proinflammatory and anti-inflammatory cytokine helps in development of anti-inflammatory therapy which is beneficial.
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INTRODUCTION

Acute pancreatitis is an inflammatory disease ranging from mild self- limiting course to a fulminant phase with multiple organ failure and mortality. It occur in two forms, 80% of patients develops edematous pancreatitis and 20% manifest as acute necrotising pancreatitis. The average mortality rate for edematous type is <1% and necrotising type ranges from 10-20% (Banerjee et al.1995). Patients with mild type managed with aggressive fluid resuscitation and supportive care. Severe AP may require maximal non operative care and nutritional support. Severe AP is divided into two-phase systemic disease. In the early phase (Within initial 2 weeks) development of pancreatic inflammation and necrosis, followed by systemic inflammatory response syndrome (SIRS) ultimately leading to multiple organ dysfunction syndromes (MODS) . Mortality is high due to MODS. (Health et al.1995). If the early phase is not corrected by therapeutic intervention, then late phase occurs after the second week of onset, and includes the formation of infected pancreatic necrosis or fluid collection with possible progression to overt sepsis, MODS and death. Organ failure is present in only half of the patients with pancreatic necrosis. The mortality rate for MODS ranges from 30 to 100% (Neoptolemos et al.1998). Respiratory failure is the most common type of organ failure in AP (Viedma et al.1994). Increasing knowledge of the importance of proinflammatory and anti-inflammatory cytokine helps in development of anti-inflammatory therapy which is beneficial.

REVIEW OF LITERATURE

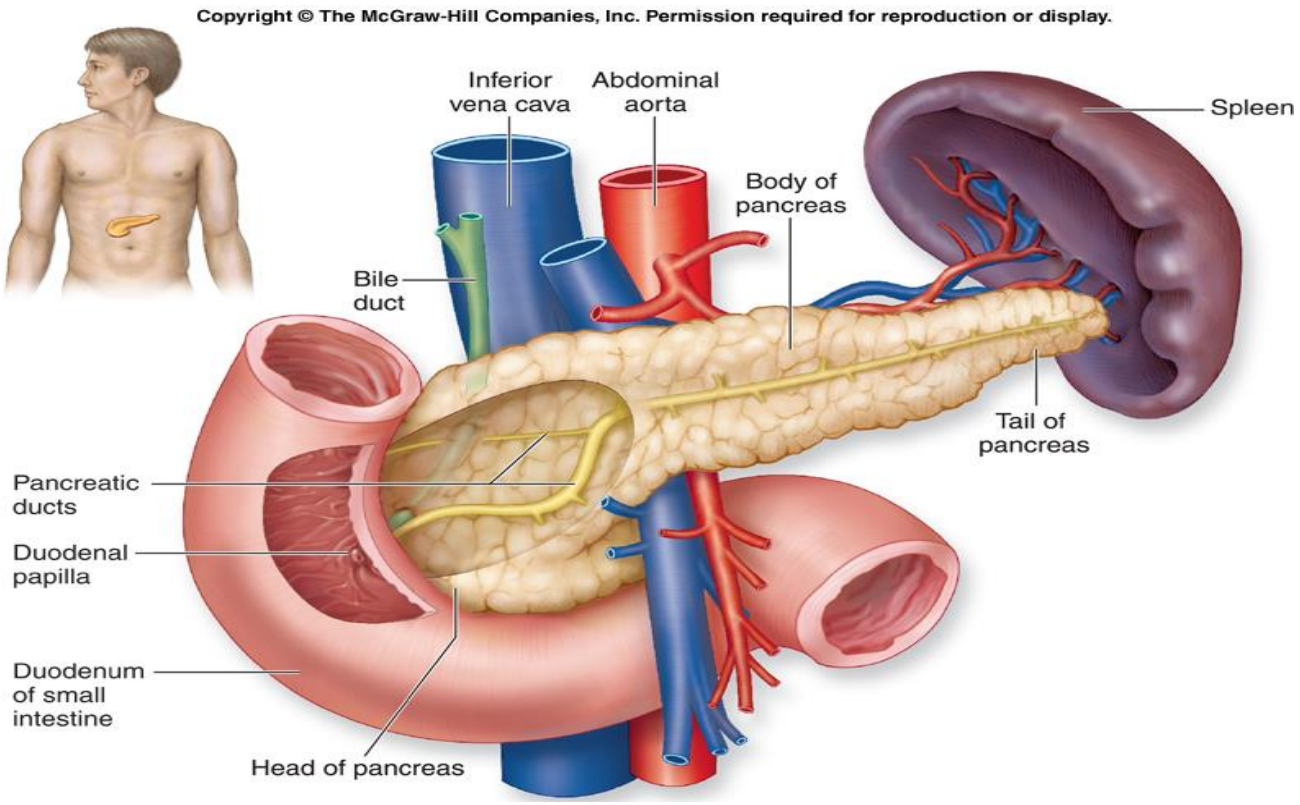
HISTORY & BACKGROUND:

The definition and classification of pancreatitis is characterized mainly by the distinction between acute and chronic pancreatitis as stated by Lagerlof in 1942 (Lagerlof,1942). These two clinical conditions seems to be different pathologic processes. But with the help of imaging studies, particularly magnetic resonance imaging (MRCP), the classic distinction between acute and chronic pancreatitis is clearly made.

The mechanism causing fibrosis in the glandular tissue begins with acute abdominal pain with increase levels of serum amylase and lipase. Acute pancreatitis developing into chronic pancreatitis due to obstruction secondary to edema or inflammatory response of the sphincter of Oddi.

The most typical natural history regarding the correlation between acute and chronic pancreatitis is so-called recurrent pancreatitis. It is not possible to define this condition per se, but it represents the best example of the new comprehension of the pathologic process. Nevertheless, today we must keep in mind a classification of acute and chronic pancreatitis to help in developing diagnostic and therapeutic algorithms.

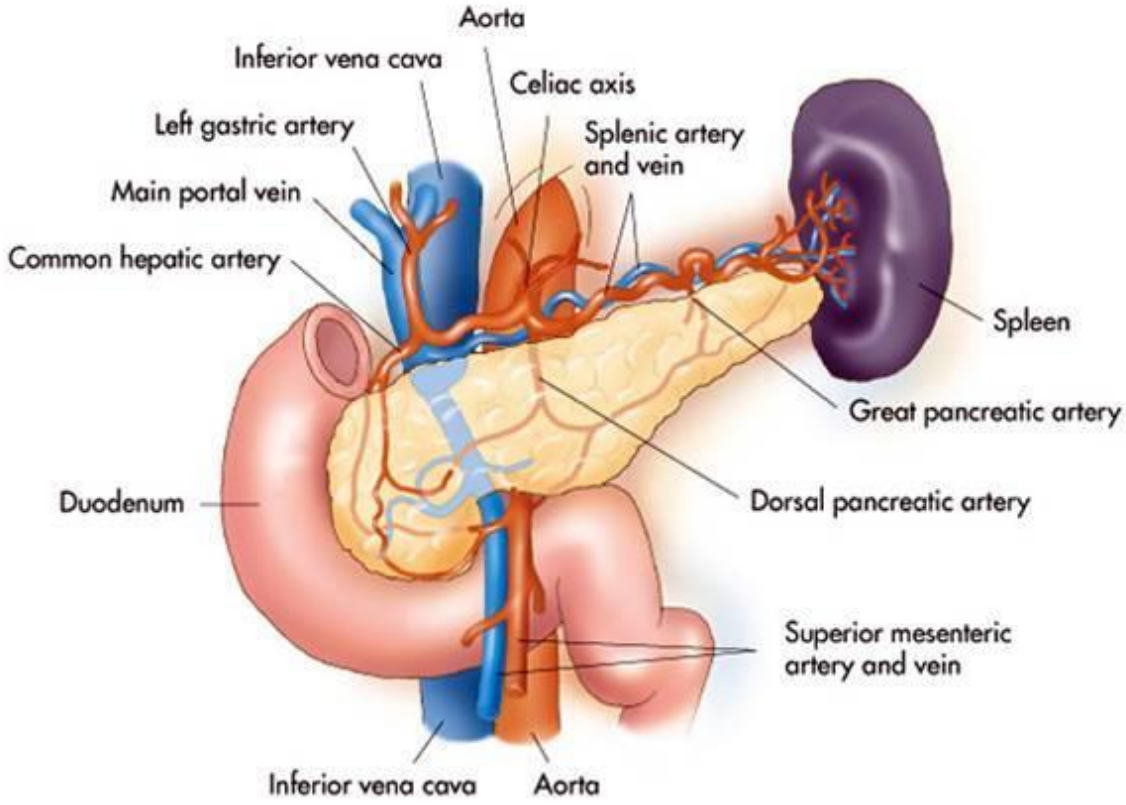
ANATOMY – PANCREAS



The pancreas, derived from GREEK (pan-all; kreas – flesh) lies in the upper left part of the abdomen. It is found behind the stomach. The pancreas is about 6 inches long. Anatomically the pancreas is divided into the head, neck, body and tail. The head is surrounded by duodenum in its concavity. Uncinate process arise from the back of head. The neck is 2.5 cms long and lies infront of the superior mesenteric artery and vein. The body is the largest part of the pancreas and lies behind the pylorus. The tail ends by abutting the spleen.

The pancreas is the secretory structure with an internal hormonal role (ENDOCRINE) and an external digestive role (EXOCRINE). It has two main ducts, MAIN PANCREATIC DUCT and ACCESSORY PANCREATIC DUCT. These drain enzymes through the ampulla of vater into the duodenum.

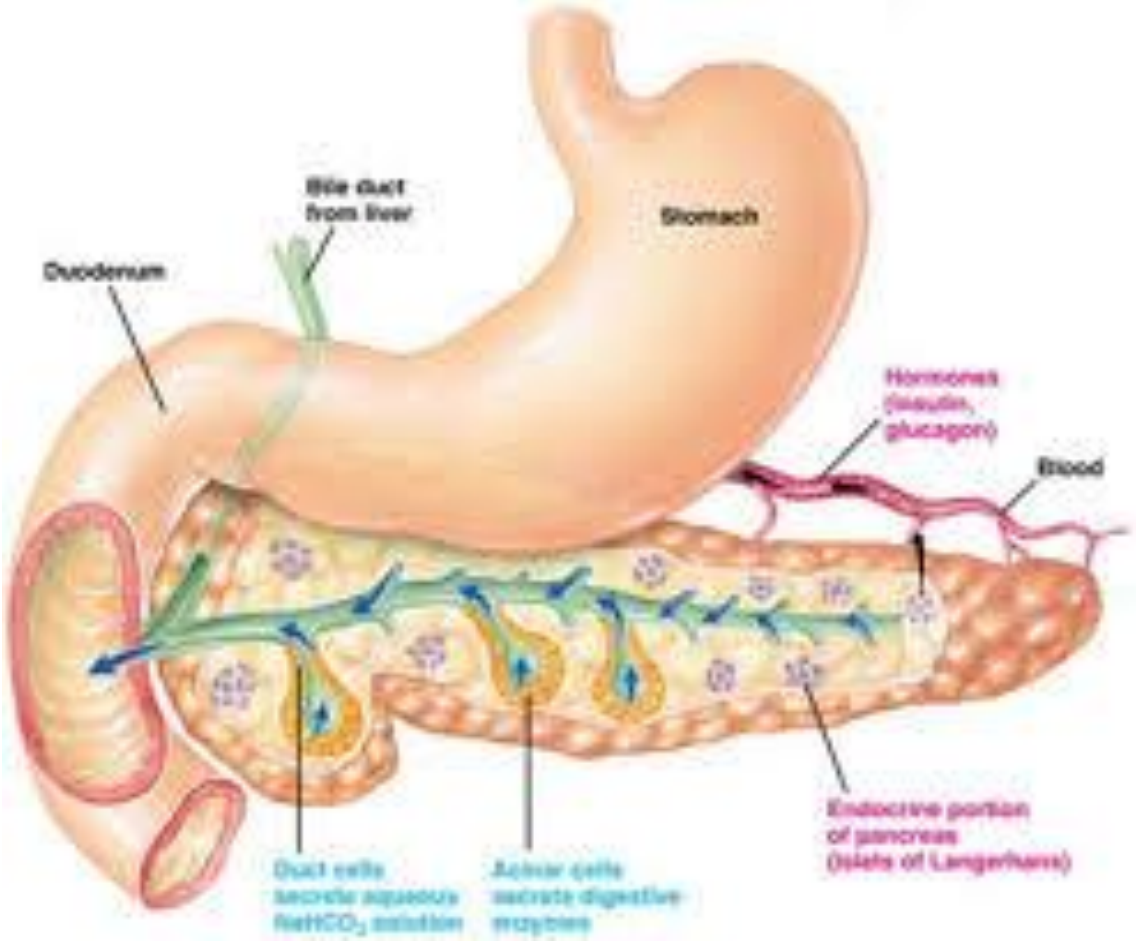
The pancreas receive blood supply from the celiac artery and superior mesenteric artery. The splenic artery supplies the neck, body and tail through its pancreatic branches. The head is supplied by superior & inferior pancreaticoduodenal arteries and drains into the superior mesenteric and portal veins. The body and neck drains into splenic vein.



BLOOD SUPPLY OF PANCREAS

HISTOLOGY OF THE PANCREAS

The pancreas contains tissue with an endocrine and exocrine role. The tissues with endocrine role be seen under staining as lightly stained clusters of cells, called PANCREATIC ISLETS OF LANGERHANS. Darker staining cells form clusters called ACINI. The secretory cells of each acinus surround a small INTERCALATED DUCT. The intercalated ducts drain into larger ducts within the lobule, and finally interlobular ducts. The ducts are lined by a single layer of columnar epithelium.



HISTOLOGY OF PANCREAS

CLASSIFICATION OF ACUTE PANCREATITIS:

The classifying systems for acute pancreatitis comes under following tables

ATLANTA CLASSIFICATION

Based on Atlanta classifying system, acute pancreatitis is divided into:

Table 1: Definition of severity of acute pancreatitis according to different classification systems

Atlanta Classification	Revised Atlanta Classification	Determinant based classification
Mild AP - *Minimal organ dysfunction and uneventful recovery* - *Absence of organ failure and/or local complications* **Severe AP** - *Organ failure and/or local complications*	**Mild AP** - *No organ failure* - *No local or systemic complications* **Moderately severe AP** - *Transient organ failure AND/OR local or systemic complication OR exacerbation of pre-existing co-morbidities.* **Severe AP** - *Persistent organ failure (single or multiple)*	**Mild AP** - *No organ failure* - *No (peri)pancreatic necrosis* **Moderate AP** - *Sterile (peri)pancreatic necrosis AND/OR transient organ failure* **Severe AP** - *Infected (peri)pancreatic necrosis OR persistent organ failure* **Critical AP** - *Infected (peri)pancreatic necrosis AND persistent organ failure*

INTERNATIONAL CLASSIFICATION:

i) Acute pancreatitis

   a) Mild

   b) Severe

ii) Acute interstitial pancreatitis

iii) Acute necrotizing pancreatitis

   a) Infected

   b) Sterile

iv) Pancreatic abscess

v) Pseudocyst pancreas

vi) According to clinico anatomical types

   a) Haemorrhagic

   b) Fatty

   c) Edematous

vii) According to necrotic distribution

   a) Focal

b) Diffuse

CAMBRIDGE CLASSIFIATION of PANCREATITIS:

- Acute
- Chronic

MANCHESTER CLASSIFICATION:

1. Mild
2. Moderate
3. End stage

MARSEILLE CLASSIFICATION:

i) Acute

ii) Chronic

iii) Chronic inflammatory

REVISED MARSEILLE CLASSIFICATION:

- Acute
- Chronic
- Obstructive chronic types.

ETIOPATHOGENESIS OF ACUTE PANCREATITIS

Pancreatitis, an inflammatory disorder caused by increased intakeof alcohol or the migration of gallstones. The tabular column shows the etiology of pancreatitis:

Aetiology	Incidence
Alcoholism	16 (35.6%)
Gallstones	10 (22.2%)
Post-traumatic	9 (20%)
Idiopathic	6 (13.3%)
Post-ERCP	4 (8.9%)

ERCP: endoscopic retrograde cholangiopancreatography

Table 2: Aetiology of mild pancreatitis (n=34)

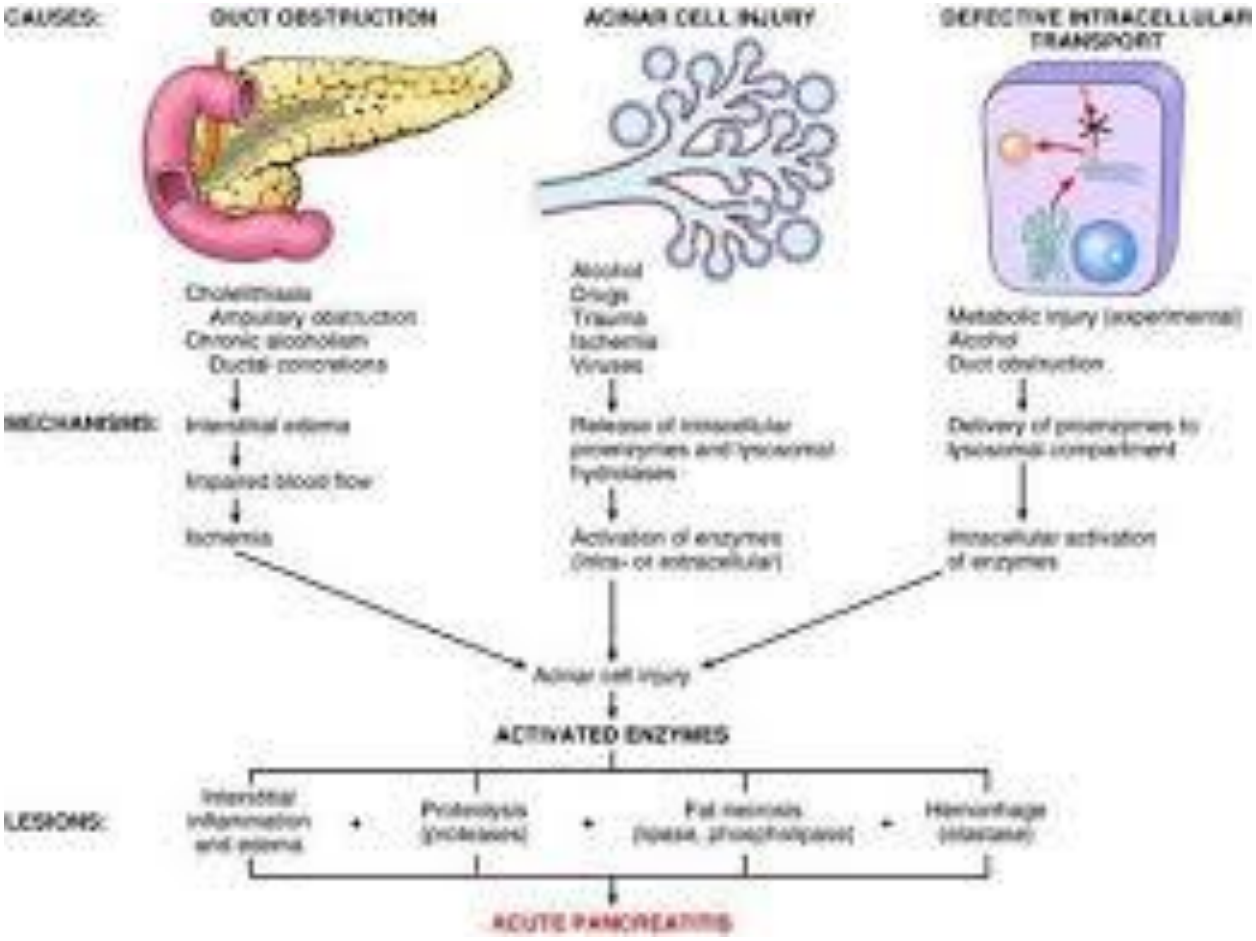
Aetiology	Incidence
Alcoholism	14 (41.2%)
Gallstones	8 (23.5%)
Post-traumatic	6 (17.6%)
Idiopathic	4 (11.8%)
Post-ERCP	2 (5.9%)

ERCP: endoscopic retrograde cholangiopancreatography

Table 3: Aetiology of severe acute pancreatitis (n=11)

Aetiology	Incidence
Post-traumatic	3 (27.3%)
Alcoholism	2 (18.2%)
Gallstones	2 (18.2%)
Idiopathic	2 (18.2%)
Post-ERCP	2 (18.2%)

PATHOGENESIS OF ACUTE PANCREATITIS



EtioPathogenesis of Biliary Pancreatitis:

The incidence of cholelithiasis induced pancreatitis is 30%. The prevalence of gallstones is 18.8% (Volzke et al, 2005). Male/Female ratio ranges from 1: 1.7 to 1: 1.4, suggesting that men are at a greater risk of developing biliary pancreatitis than women. Above 60 years of age, the age risk was reversed; more women than men had biliary pancreatitis (Imrie & Blumgart, 1975).

The socioeconomic impact of gallstone disease remains one of the highest in gastroenterology, in spite of the fact that the risk of developing pancreatitis remains < 1% for gallstone carriers.

Eugene Lindsay Opie, first to address biliary pancreatitis. In 1901, he reported two post-mortem reports and he concluded that two characteristic mechanisms exist for gallstone-induced pancreatitis (Opie, 1901a, 1901b). The first case showed that an impacted gallstone occludes the orifice of the Wirsung duct causing acute pancreatitis (Opie, 1901a). When Opie simulated this finding by pancreatic duct ligation in animals, he proposed the pancreatic outflow obstruction and named as “Impaired outflow hypothesis”.

In second case, the impacted stone at duodenal papilla created a communication between the common bile duct and the wirsung duct permiting the infected bile to enter the pancreatic duct. Opie proposed this triggering mechanism as “common channel hypothesis”.

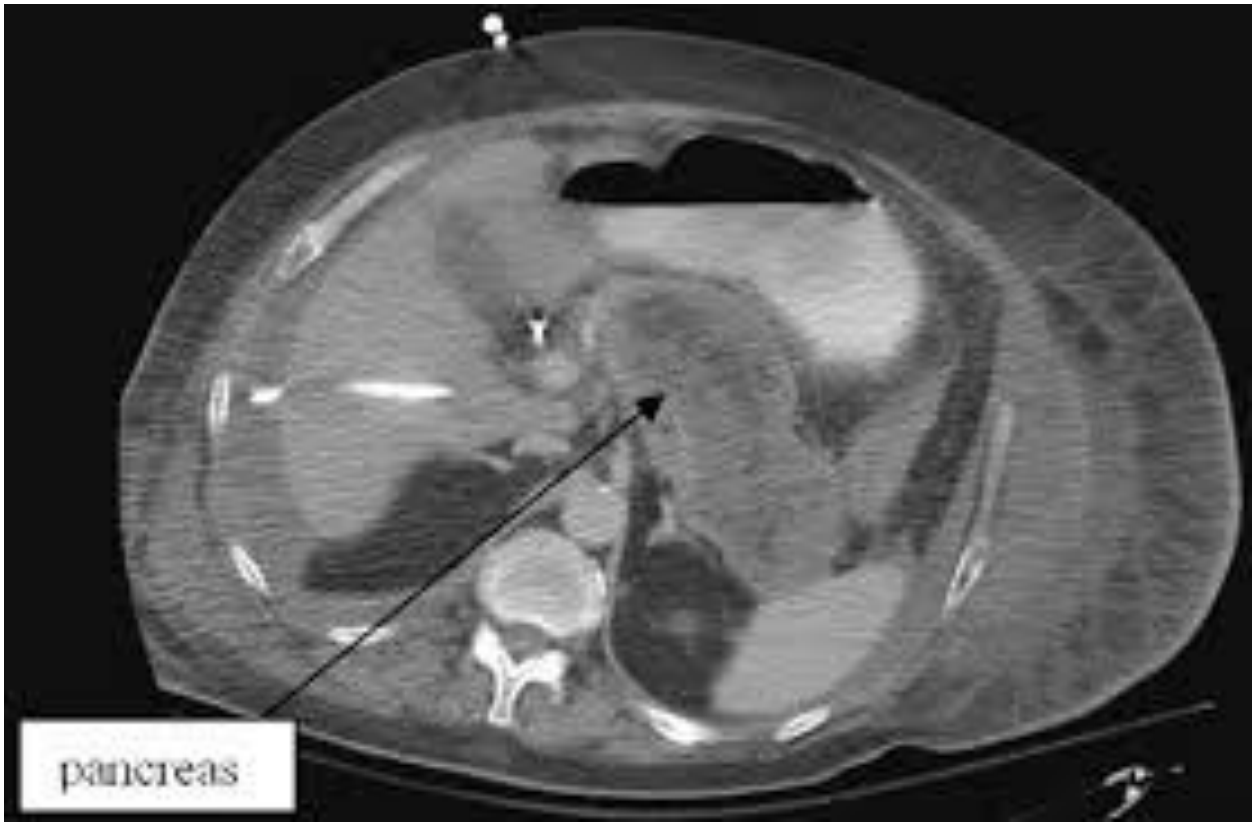


FIGURE 1. CT ABDOMEN IMAGE SHOWING INFLAMMED PANCREAS WITH PERIPANCREATIC FAT STRANDING

MECHAMISM OF ALCOHOLIC PANCREATITIS :

Alcohol abuse is the second most important cause of acute pancreatitis. Most patients with acute pancreatitis of this etiology drink at least 100 g of alcohol per day for long periods. Honest information from the patient can be difficult to obtain, and it is important to question relatives and close friends. Measurement of blood alcohol levels can be helpful. Alcohol is the most common etiology in prospective studies from New York and Helsinki (Ranson et al, 1974; Saino et al, 1995). In each study, approximately 75% to 80% of patients had alcohol abuse as the etiology with a young median age of around 40 years compared with the common median age for biliary etiology of 53 years. The exact mechanism by which alcohol induces acute pancreatitis is uncertain, but possible that cigarette smoking is also a factor because most of these patients smoke 20 or more cigarettes daily. Alcohol cause spasm of the sphincter of Oddi, and acute alcohol ingestion increases human pancreatic bicarbonate and protein secretion, whereas chronic intake decreases bicarbonate secretion and increases the viscosity of pancreatic juice so that it contains a higher concentration of proteins.

The relationship between acute and chronic pancreatitis in patients who regularly abuse alcohol is uncertain. The duration between the onset of alcohol intake and occurrence of symptoms ranges from 7 to 29 years.

EFFECTS OF ETHANOL IN PANCREAS:

1. Direct pancreatic acinar injury

2. Alcohol induced elevation of serum lipids, fatty acids and ethyl ester metabolites causing pancreatic injury.

3. Formation of pancreatic juice which contains high enzymatic activity cause enzyme activation, precipitation of proteins leading to intraductal plug formation and obstruction causing pancreatic injury.

4. Alcohol cause sphincter of Oddi dysfunction leading into pancreatic ductal obstruction and ends in pancreatitis.

5. Pancreatic injury caused by release of free O2 radicals.

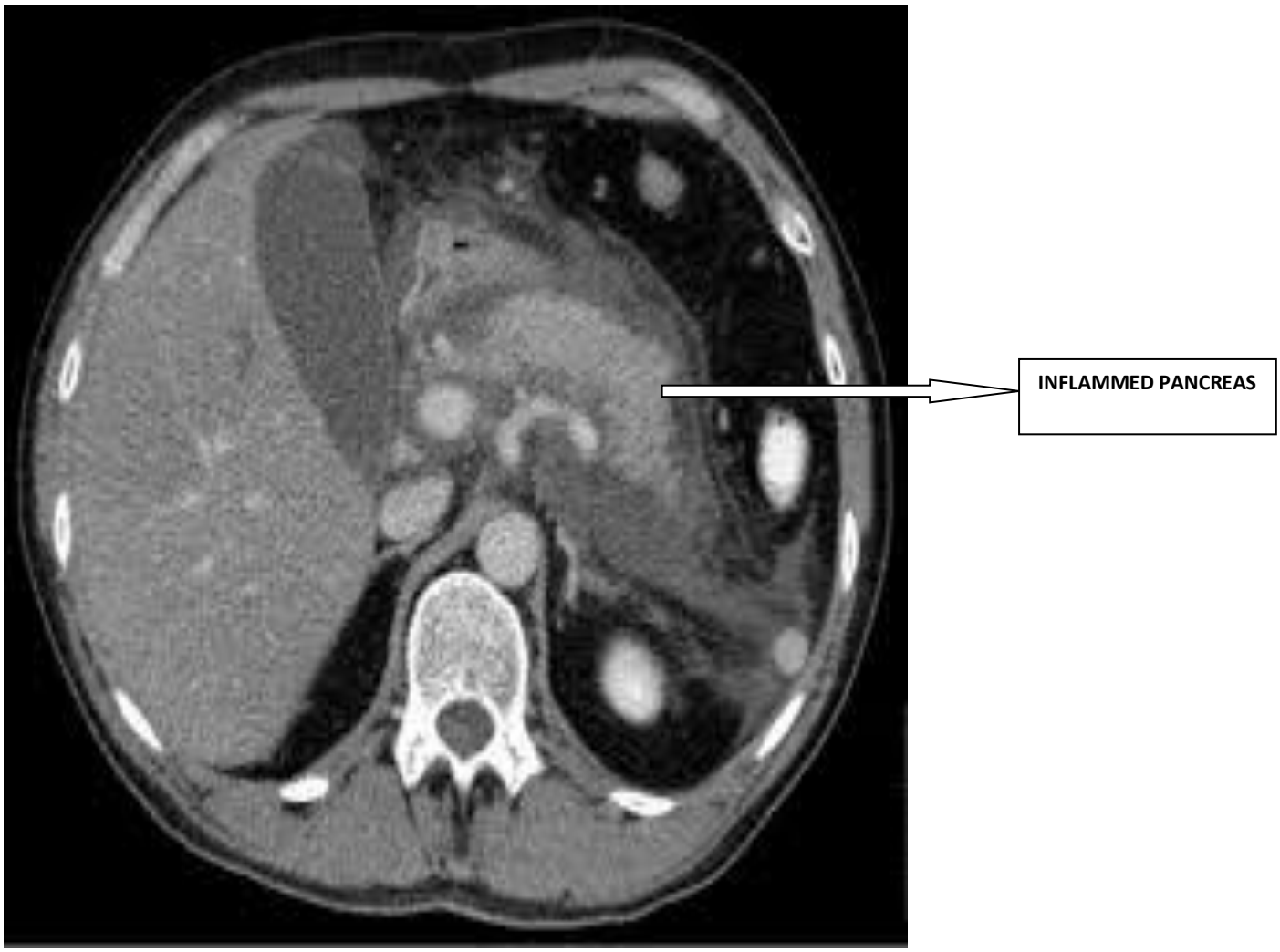


FIGURE2. CT ABDOMEN CONTRAST SHOWING EDEMATOUS PANCREATITIS.

EtioPathogenesis of Nonbiliary and Nonalcoholic Pancreatitis:

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Hyperlipidemia

Acute pancreatitis presents usually with varying degrees of severity such as infected necrosis and pseudocyst formation, which also occurs in hypertriglyceridemia patients. According to Fortson (1995), patients with hyperlipidemic patients fulfills the following criterias to develop pancreatitis, they are patients whose is hyperglycaemic and out of control, alcoholic patients with hyperlipidaemia, euglycaemic, lean and non-alcoholic patients with hypertriglyceridemia caused by medication or nutrition; or patients with familial hypertriglyceridemia.

The diagnosis of acute hyperlipidemic pancreatitis is based on serum and urinary lipase levels which remains within the above normal limits. (Lesser & Warshaw, 1975). The reason for this phenomenon reveals that there is an interference of the test assay with the plasma lipids or an unknown amylase inhibitor in plasma and urine that impairs the assay. This inhibitor has not been identified so far. In initial days the diagnostic parameter of hyperlipidemic pancreatitis was significant increase in ratio of urinary amylase and creatinine. (Warshaw et al, 1975). Triglycerides are above 1000 mg/dL, and therapy includes enteral nutrition regimens. Extracorporeal lipid reduction therapy should be considered in patients if triglyceride level does not reduce.

Drug-Induced Pancreatitis:

According to World Health Organization (WHO) list of 525 drugs published that cause acute pancreatitis. The overall incidence ranges from 0.1% and 2% of pancreatitis per cases. Drugs like mesalazine, azathioprine and simvastatine carries high adverse effects of developing acute pancreatitis. But the underlying pathophysiologic mechanisms remains sparse and the disease course is usually mild or even subclinical (Nitsche et al, 2010).

Infectious Causes:

VIRAL ILLNESS:

The incidence of acute pancreatitis in infectious patients was less than 2%.

The most frequent association of a viral infection with acute pancreatitis is mumps. The incidence of pancreatitis in mumps patient (parvovirus B19) is 0.3% to 14%. Acute pancreatitis mainly occurs 2 weeks after the initial onset of disease. Sometimes acute pancreatitis may occur 1 week prior to parotitis. The disease is usually mild, symptoms persist only 3 to 7 days. Serum lipase activity is elevated. Amylase measurements also elevated but results in an incorrect diagnosis due to increased activity from affected salivary gland. No specific treatment is available.

The second most common viral infection associated with pancreatitis is coxsackie virus B. The incidence of acute pancreatitis in coxsackie virus ranges from 0% to 11%. (Parenti et al, 1996). Other viruses such as Hepatitis virus A, B, or C also cause acute pancreatitis. In mild form of acute hepatitis, an increase in amylase activity can be detected in upto 30% of patients. In severe and ultimately lethal patients, post-mortem report shows 44% of patients with acute pancreatitis.

Post mortem studies also reported incidence of pancreatitis in 50% of patients who died from AIDS. Other virus infections like Epstein-Barr virus, rubella, adenovirus, rubeola, herpes simplex virus, rotavirus also cause pancreatitis.

BACTERIAL CAUSES:

Bacterial infections causing acute pancreatitis, such as Yersinia enterocolitica and Y. pseudotuberculosis, Salmonella enteritis and S.typhimurium, Campylobacter jejuni, and Mycoplasma pneumoniae. Acute pancreatitis induced by a Campylobacter is a rare. Amylase and lipase levels elevated three to six times above normal and the disease course is mild.

PARASITIC CAUSES:

Parasites causing acute pancreatitis includes ascariasis and clonorchis sinensis. The ascariasis is the most common type of helminthic infection in humans. An endemic manifestation is found mainly in tropical and subtropical countries. The mechanism causing pancreatitis includes worms migrates from intestines to either the biliary or pancreatic ducts, leading to obstruction causing pancreatitis. The disease can be diagnosed by ultrasonography or endoscopic retrograde cholangiopancreatography (ERCP). The appropriate treatment is a combination of antihelminthic therapy with standard treatment for acute pancreatitis.

The Chinese liver fluke Clonorchis sinensis also cause pancreatitis in which it lodges in the biliary tract. C. sinensis infection also causes periductal fibrosis, adenomatous proliferation and squamous metaplasia causing obstruction and results in acute pancreatitis.

ERCP related Pancreatitis:

Endoscopic retrograde cholangiopancreaticography is the imaging modality in various hepato biliary pancreatic disease. It is used for both diagnostic and therapeutic purposes in hepato biliary pancreatic tree. The most common complication is procedure-related acute pancreatitis, occurs in 2% to 9% of individuals. It ranges from mild variant with complete recovery to severe disease with prolonged hospitalisation, pancreatic necrosis,

multiorgan failure and even death. About 10% of patients have tendency to develop complications and up to 1% have fatal course (Freeman, 2001). The mechanism includes increase pressure in the pancreatic duct, chemical injury from the injection of contrast media which leads to initiation of inflammatory cytokine release and protease activation cascade that ultimately results in disease. Individuals with low pH of 6.9 in contrast media cause activation of the α-cation channel expressed on C and Aδ fibers of primary sensory neurons called transient receptor potential vanilloid 1 (TRPV1) neurons results in pancreatitis.

HEREDITARY CAUSES OF PANCREATITIS:

Patients with unknown cause for pancreatitis comes under idiopathic pancreatitis. Hereditary pancreatitis is a rare entity with tendency to occur before 20 years of age and progress to develop chronic pancreatitis for next 20 years and finally leads to pancreatic cancer at 70 years. Mutations associated are R122H, N291 and A16v of cationic trypsinogen gene. Other mutations include cystic fibrosis transmembrane conductance regulator and intracellular trypsin inhibitor (KAZAL, type I). No treatment is available yet.

DIAGNOSIS OF ACUTE PANCREATITIS

CLINICAL PRESENTATION:

Patient presents with sudden-onset of upper abdominal pain and vomiting.. The location of the pain may be in the epigastrium or right or left upper quadrant of the abdomen radiating towards to the back. An encircling upper abdominal pain is less common. Severe pain is often present for the initial 48 hours, which then settles over the next 96 hours. The accompanying vomiting exacerbates the tendency to hypovolemia and hypotension. A mild degree of obstructive jaundice may be present, and occasionally cholangitis co-exists with acute pancreatitis. Bowel sounds may be present at the time of initial presentation, but frequently a degree of paralytic ileus occurs. Despite this paralytic ileus, early enteral feeding either by the nasojejunal or the nasogastric route can be routinely practiced (Eatock et al, 2005). Mild acute pancreatitis usually resolves fairly quickly with simple treatment measures. Other clinical findings, such as ecchymosis of the flank (Grey Turner sign) or periumbilical area (Cullen sign) are noted.

BIOCHEMICAL ANALYSIS:

Serum Amylase & Lipase

Diagnosis of acute pancreatitis is confirmed by measuring the serum amylase and lipase values. An elevated serum amylase value 3 times than its normal value and lipase level 2 times more than its normal value confirms the diagnosis. But comparing these two parameters, Serum lipase high sensitivity and specificity than serum amylase since amylase level increases in other acute inflammatory conditions. The normal value of serum amylase and lipase is 40-140U/L and 0-160 U/L.

C-Reactive Protein

C-reactive protein (CRP) is a non specific biomarker for inflammation. It is synthesised by release of inflammatory cytokines. Serum concentration of >150 mg/L predicts the severity of the disease with high sensitivity.

Trypsinogen Activation Peptide

Trypsinogen activation peptide (TAP) level elevation in urine and plasma helps in detecting the severity of acute pancreatitis. Due to unavailability of the TAP test kit it is withdrawn. An automated assay technique is needed for clinical practise.

Hematocrit

Packed cell volume or Hematocrit, a prognostic tool for assessing the severity of acute pancreatitis. A hematocrit of > 44% indicates pancreatic necrosis and multiorgan failure.(Brown et al, 2000). This is mainly due to the third space fluid loss.

Procalcitonin

Procalcitonin(PCT), which is the precursor of calcitonin, new serum marker used in detecting acute pancreatitis. The synthesis and release of PCT mediated by proinflammatory cytokines and lipopolysaccharides. To differentiate mild and severe type of acute pancreatitis, PCT rapid semiquantitative assay is used.

Imaging Assessment of Acute Pancreatitis

The radiological investigation of choice is dynamic contrast enhanced computed tomography scan. It has high sensitivity and accuracy rate in detecting the severity of acute pancreatitis.

The following table shows the BALTHAZAR CT scoring system for predicting the severity:

SEVERITY INDEX IN ACUTE PANCREATITIS

Grade of Acute pancreatitis		Points
A	NORMAL PANCREAS	0
B	PANCREATIC ENLARGMENT ALONE FOCAL OR DIFFUSE WITH CONTOUR IRREGULARITIES AND INHOMOGENOUS ATTENUATION	1
C	B+PERIPANCREATIC INFLAMMATION	2
D	C+ONE PERIPANCREATIC FLUID COLLECTION	3
E	D+TWO OR MORE PERIPANCREATIC OR RETROPERITONEAL FLUID COLLECTION OR GAS COLLECTION	4
DEGREE OF PANCREATIC NECROSIS		
1	NO – NECROSIS	0
2	NECROSIS OF <33% PANCREASE	2
3	NECROSIS OF 33%-50% OF PANCREASE	4
4	NECROSIS OF > 50% OF PANCREASE	6
CT SEVERITY INDEX (CT SI)BALTHAZAR SCORE+NECROSIS SCORE		
CT GRADE + NECROSIS GRADE (0 - 4) + (0 – 6) → (0 – 10)		

TABLE SHOWING CT FINDINGS IN COMPLICATED TYPES OF ACUTE PANCREATITIS:

Acute Peripancreatic Collection	Acute Necrotic Collection
- < 4 weeks - In interstitial pancreatitis - Homogeneous - fluid density - *No fully definable wall* - Adjacent to pancreas - Confined by normal fascial planes	- < 4 weeks - In necrotizing pancreatitis - Heterogeneous collection - *No fully definable wall* - Intra- or extrapancreatic
Pseudocyst	**Walled-off Necrosis**
- > 4 weeks - In interstitial pancreatitis - Homogeneous - fluid density - *Well defined wall* - Adjacent to pancreas - No non-liquid component	- > 4 weeks - In necrotizing pancreatitis - Heterogeneous collection - *Well-defined wall* - Intra- or extrapancreatic

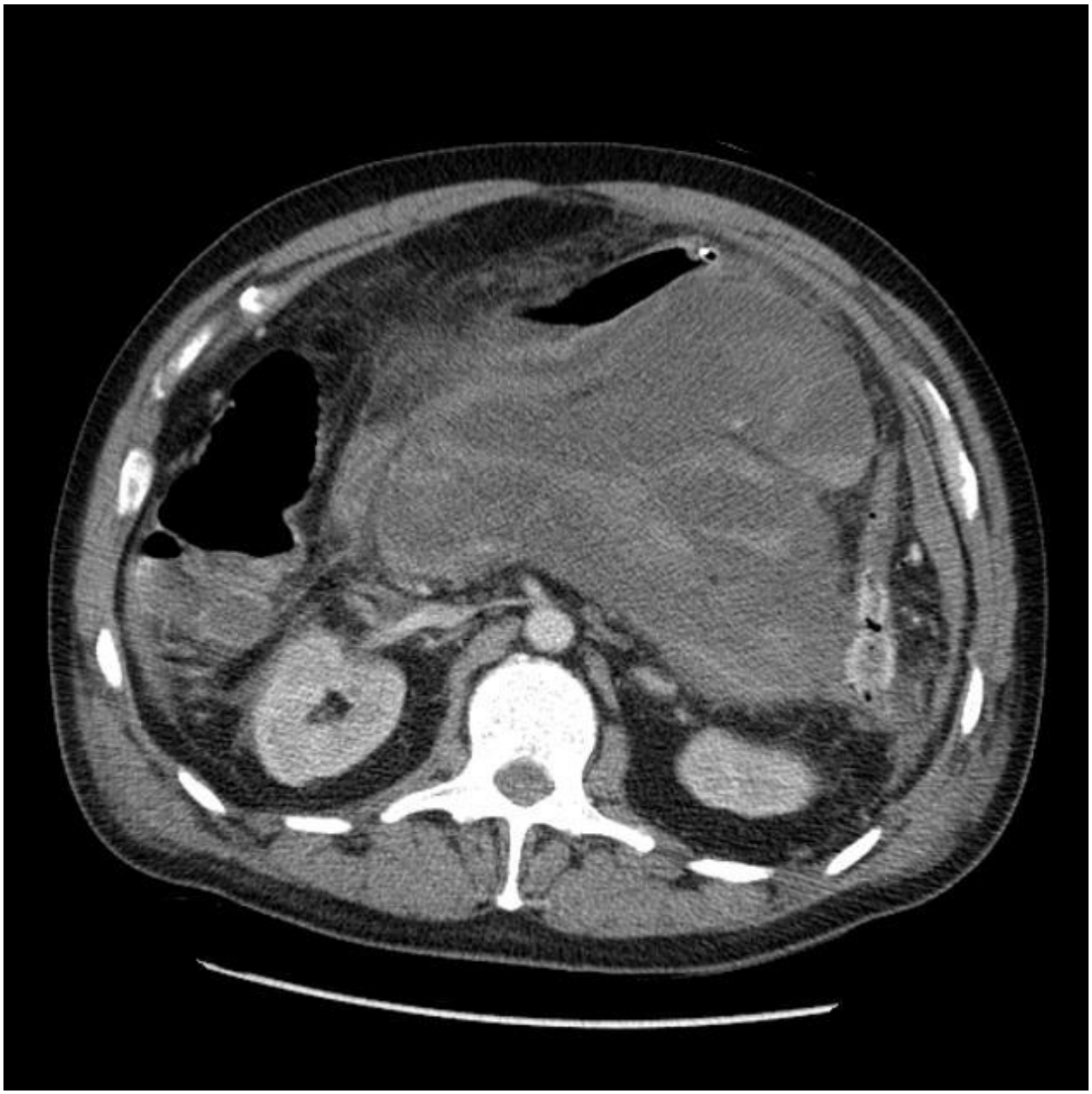


FIGURE SHOWING ACUTE NECROTISING PANCREATITIS WITH PERIPANCRATIC COLLECTION

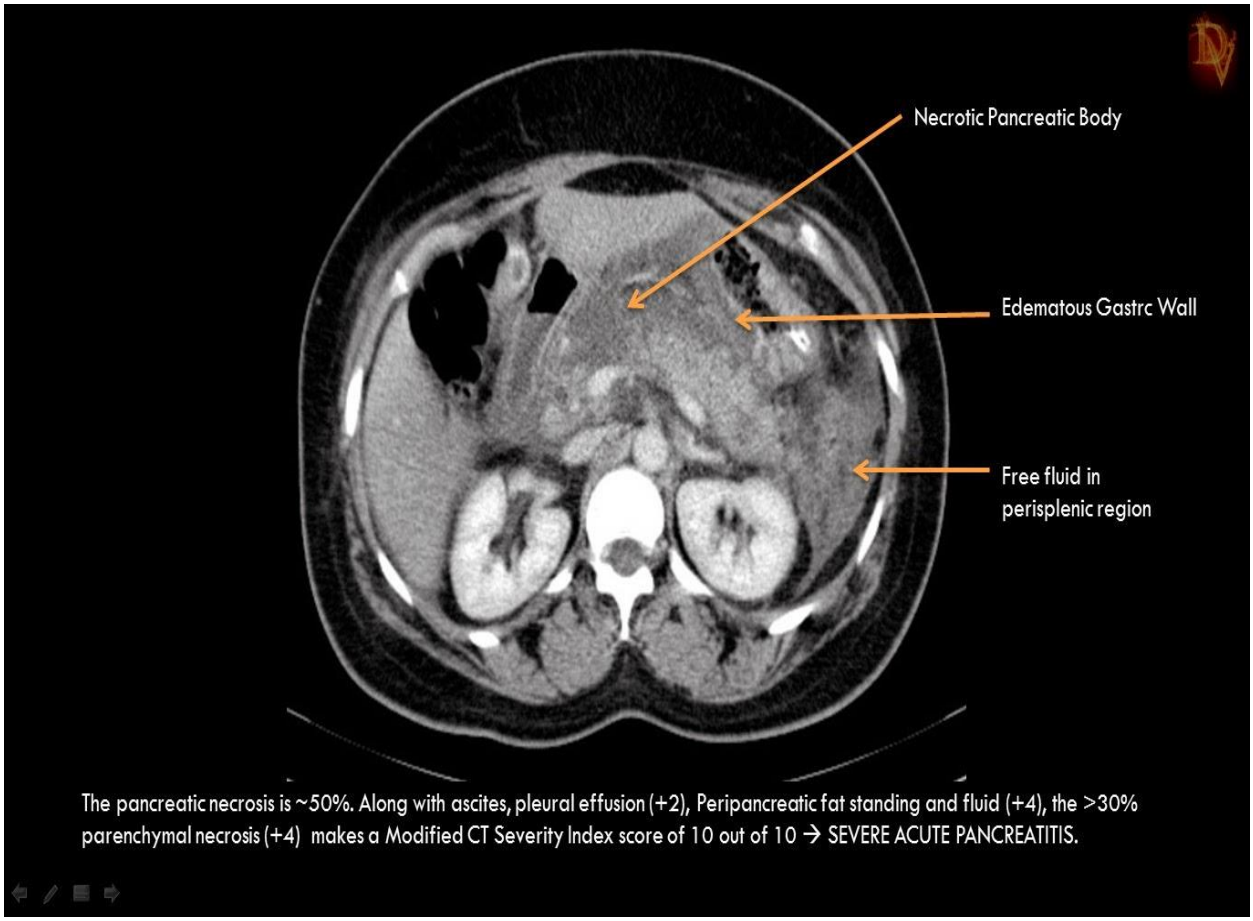


FIGURE SHOWING ACUTE NECROTISING PANCREATITIS

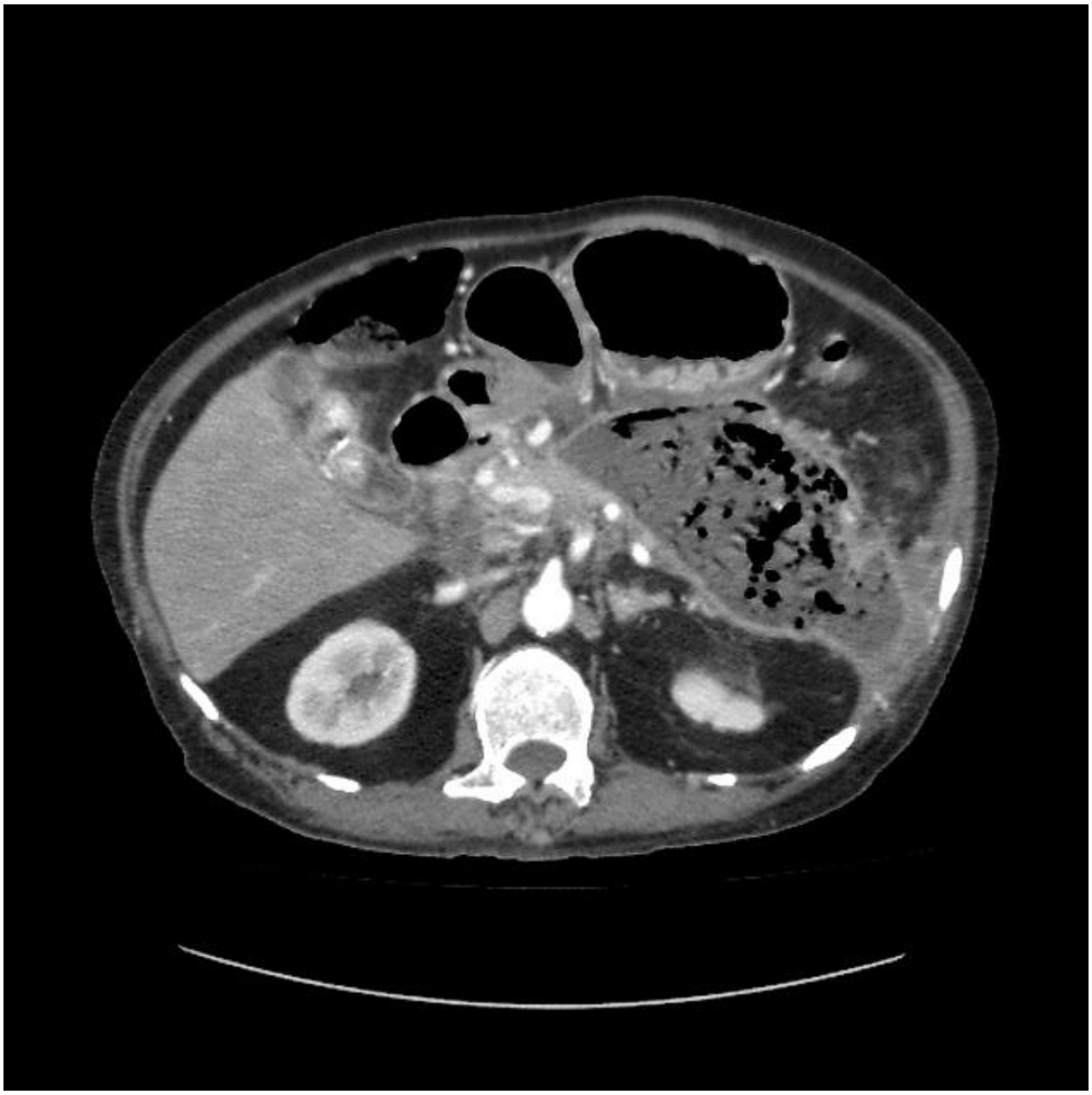


FIGURE SHOWING WALLED OFF PANCREATITIS

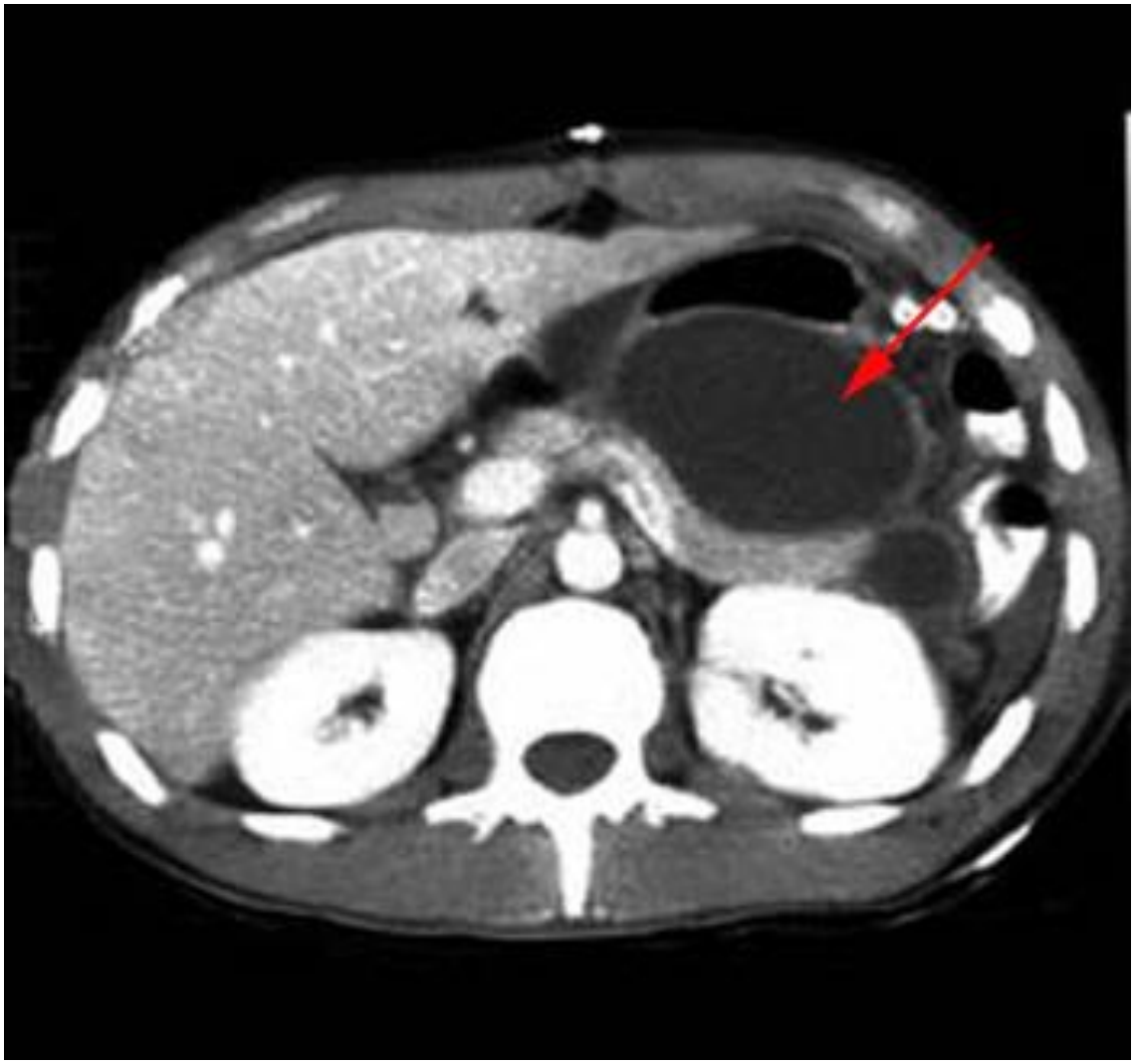


FIGURE SHOWING PSEUDOCYST FORMATION IN ACUTE PANCREATITIS

Though the imaging modality of choice is CECT scan, MRI may also helps in delineating the vein thrombosis and pseudoaneurysm in severe pancreatitis. MRI also plays a role in delineating the anatomical orientation of pancreas.

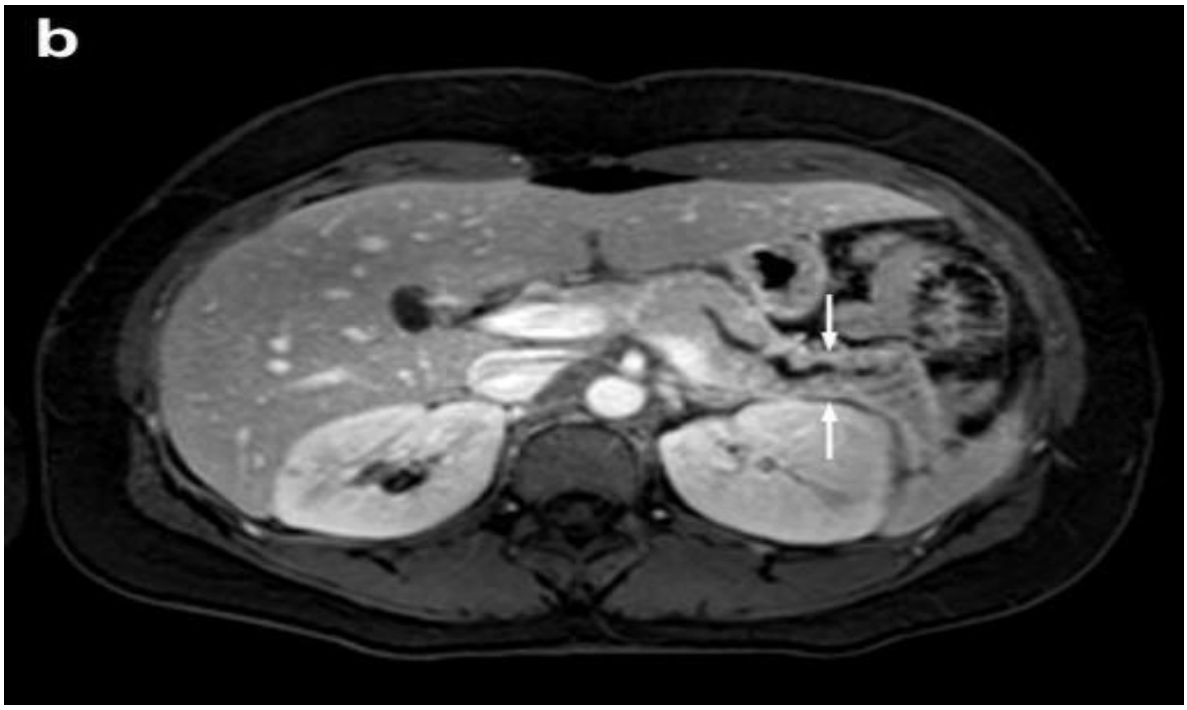


FIGURE SHOWING MRI IMAGE OF ACUTE PANCREATITIS

DISEASE OF SEVERIY ASSESSMENT

To identify the cause and severity in a particular episode of pancreatitis it is important to identify the group with maximum risk of death and main complications. To identify this group, numerous grading systems have been devised. An initial system (Ranson et al,1974) was more relevant to patients with alcohol-associated disease. Ranson subsequently introduced a modified grading system for patients with biliary disease (Ranson, 1979). This modification has complicated the system because a dilemma exists as to which to use when the patient is initially admitted. There has not been a problem with the Glasgow grading system, which has been increasingly used in the United Kingdom and elsewhere (Imrie et al, 1978; Osborne et al, 1981; Wilson et al, 1988). The system comprises eight factors; the presence of any three or more within 48 hours of admission defines the patient as having severe disease .

The single biomarker for detecting the clinical severity, C- Reactive protein level. The normal value is 10mg/L and the value >150mg/L indicates severe disease. Other biochemical markers includes IL-8,IL-6, TNF soluble receptors, polymorphonuclear elastase, serum procalcitonin, soluble IL-2 receptors and soluble E- selectin. Trypsinogen activation peptide is an additional marker useful in determining the prognosis.

CLINICAL SCORING SYSTEMS

Clinical scoring systems such as Ranson which contains 11 biochemical criteria, Glasgow and Acute physiology and chronic health evaluation 2 score contains 12 biochemical variables are used to predict outcomes in acute pancreatitis.

SEVERITY SCORING SYSTEMS

ACUTE PANCREATITIS **SPECIFIC** SCORING SYSTEMS

- Ranson score
- Glagsow score
- **B**edside **I**ndex for **S**everity in **A**cute **P**ancreatitis(BISAP) score
- **H**armless **A**cute **P**ancreatitis **S**core(HAPS)
- Hong Kong Criteria

ACUTE PANCREATITIS **NON-SPECIFIC** SCORING SYSTEMS (ICU SCORING SYSTEMS)

- **A**cute **P**hysiology **A**nd **C**hronic **H**ealth **E**valuation(APACHE) II score
- **S**equential **O**rgan **F**ailure **A**ssessment(SOFA) score

APACHE II SCORING SYSTEM:

Table 2

APACHE II Score[9]

APACHE II score = (acute physiology score) + (age points) + (chronic health points)

Physiologic Variable	+4	+3	+2	+1	0	+1	+2	+3	+4
Rectal temperature (C)	≥ 41	39-40.9		38-38.9	36-38.4	34-35.9	32-33.9	30-31.9	≤ 29.9
Mean arterial pressure (mm Hg)	≥ 160	130-159	110-129		70-109		50-69		≤ 49
Heart rate (bpm)	≥ 180	140-179	110-139		70-109		55-69	40-54	≤ 39
Respiratory rate (bpm)	≥ 50	35-49		25-34	12-24	10-11	6-9		≤ 5
Oxygen delivery (mL/min) OR PaO_2 (mm Hg)	≥ 500	350-499	200-349		< 200 > 70	61-70		55-60	< 55
Arterial pH	≥ 7.7	7.6-7.69		7.5-7.59	7.33-7.49		7.25-7.32	7.15-7.24	< 7.15
Serum sodium (mmol/L)	≥ 180	160-179	155-159	150-154	130-149		120-129	111-119	≤ 110
Serum potassium (mmol/L)	≥ 7	6-6.9		5.5-5.9	3.5-5.4	3-3.4	2.5-2.9		< 2.5
Serum creatinine (mg/dL)	≥ 3.5	2-3.4	1.5-1.9		0.6-1.4		< 0.6		
Hematocrit (%)	≥ 60		50-59.9	46-49.9	30-45.9		20-29.9		< 20
White cell count (10^3/mL)	≥ 40		20-39.9	15-19.9	3-14.9		1-2.9		< 1

Age Points

Age	Points
≤ 44	0
45-54	2
55-64	3
65-74	5
≥75	6

Chronic Health Points

History of Severe Organ Insufficiency	Points
Nonoperative patients	5
Emergency postoperative patients	5
Elective postoperative patients	2

RANSON SCORING SYSTEM

Ranson's Criteria on Admission :

- age greater than 55 years
- a white blood cell count of > 16,000/µL
- blood glucose > 11 mmol/L (>200 mg/dL)
- serum LDH > 350 IU/L
- serum AST >250 IU/L

Ranson's Criteria after 48 hours of admission :

- fall in hematocrit by more than 10 percent
- fluid sequestration of > 6 L
- hypocalcemia (serum calcium < 2.0 mmol/L (<8.0 mg/dL))
- hypoxemia (P_{O_2} < 60 mmHg)
- increase in BUN to >1.98 mmol/L (>5 mg/dL) after IV fluid hydration
- base deficit of >4 mmol/L

The prognostic implications of Ranson's criteria are as follows :

- Score 0 to 2 : 2% mortality
- Score 3 to 4 : 15% mortality
- Score 5 to 6 : 40% mortality
- Score 7 to 8 : 100% mortality

Modified Marshall Scoring System for organ failure

Organ System	0	1	2	3	4
Respiratory PO_2/FiO_2 (mmHg)	>300	226–300	151–225	76–150	≤ 75
Renal serum creatinine (μmol/liter)	≤ 100	101-200	201–350	351–500	>500
Hepatic serum bilirubin (μmol/l)	≤ 20	21–60	61–120	121–240	>240
Cardiovascular PAR[1)]	≤ 10,0	10,1–15,0	15,1–20,0	20,1–30,0	>30,0
Hematologic platelets/nl	>120	81–120	51–80	21-50	≤ 20
Neurologic Glasgow Coma Score	15	13–14	10–12	7–9	≤ 6

Table 3: Bedside Index for Severity of Acute Pancreatitis (BISAP)

- BUN > 25
- Impaired mental status
- SIRS (> 2 criteria)
- Age > 60 yrs
- Pleural effusion on CT scan
- 1 point for the presence of each finding.

BUN, blood urea nitrogen; SIRS, systemic inflammatory response syndrome

BISAP Score	Observed Mortlity
0	0.1%
1	0.4%
2	1.6%
3	3.6%
4	7.4%
5	9.5%

Adapted from: Wu BU, Johannes RS, Sun X, et al. The early prediction of mortality in acute pancreatitis: A large population-based study. *Gut* 2008;57:1698-1703.

Modified Glasgow/PANCREAS score

- **P**a**O2** < 8kPa (60mmhg)
- **A**ge > 55 years
- **N**eutrophils: **WBC** >15 x109/l
- **C**alcium < 2mmol/l
- **R**enal function: (**Urea** > 16mmol/l)
- **E**nzymes: (AST/ALT > 200 iu/L or **LDH** > 600 iu/L)
- **A**lbumin < 32g/l
- **S**ugar: (Glucose >10mmol/L)

***Applicable for both** gallstone and alcohol induced pancreatitis within 48 hours of admission
***Omission of age/serum transaminase** increases the predictive valve of scoring system as serum transaminase did not differ significantly between mild and severe pancreatitis
*Bold 4 factors are **independently significant** in predicting the severity

COMPLICATION OF ACUTE PANCREATITIS

i. Shock

ii. Septicaemia

iii. Elevated serum calcium

iv. Acute kidney injury

v. Disseminated intravascular coagulation

vi. Pleural effusion

vii. Pseudoaneurysm in pancreas

viii. Pancreatic abscess

ix. Chronic pancreatitis

MANAGEMENT:

I. CONSERVATIVE MANAGEMENT:

SIRS PHASE:

Aggressive fluid resuscitation in order to replenish extravascular or third space fluid loss. To maintain the intravascular volume, fluid flow rate of >200mL/h is recommended. Close monitoring , intravenous fluid supplementation and pain relief are most important. Crystalloid resuscitation volumes as high as 20 L may e required.

COUNTERACTIVE ANTIINFLAMMATORY RESPONSE SYNDROME PHASE:

During this phase, fatal complications have to be ruled out if the patient deterioates from the initial phase.

PREVENTION OF INFECTION:

Enterogenous bacteria is the most common cause for infections. Systemic intravenous antibiotics, enteral nutrition and probiotics are used to prevent infection.

II. INTERVENTIONAL TREATMENT:

SYSTEMIC INFLAMMATORY RESPONSE SYNDROME PHASE (FIRST & SECOND WEEKS)

Intervention in this phase aims to prevent fatal complications or further deterioration. Currently the intervention is ENDOSCOPIC RETROGRADE CHOLANGIOPANCREATOGRAPHY with SPHINCTEROTOMY.

INDICATIONS FOR ACUTE INTERVENTIONS:

1. Abdominal compartment syndrome
2. Bowel ischaemia
3. Perforation
4. Severe bleeding unresponsive to angiographic coiling.

INTERVENTION IN THE SECOND COUNTERACTIVE ANTIINFLAMMATORY RESPONSE SYNDROME PHASE:

INDICATIONS ARE:

1. Gastric outlet obstruction
2. Bowel perforation
3. Bleeding
4. Abdominal compartment syndrome

The surgical management of choice is OPEN LAPAROTOMY WITH NECROSECTOMY and minimally invasive procedures (PERCUTANEOUS, ENDOSCOPIC and LAPAROSCOPIC TECHNIQUES)

AIMS & OBJECTIVES

AIMS OF THE STUDY:

1. To correlate and analyse the various clinical presentations of acute pancreatitis.
2. To predict the severity of acute pancreatitis with available clinico biochemical parameters.
3. To assess the severity in relation to serum C REACTIVE PROTEIN.
4. To prognosticate and assess the severity of the disease.
5. To predict the outcome of acute pancreatitis with regard to serum CRP.

MATERIALS & METHODS:

54 patients are included in this study who presented with acute pancreatitis in the department of surgical emergency, Thanjavur medical college during the period of October 2015- August 2016.

STUDY DESIGN:

Prospective study from October 2015- August 2016.

INCLUSION CRITERIA:

Patients diagnosed as acute pancreatitis irrespective of sex and age over 25 years.

EXCLUSION CRITERIA:

1. Patient age <25 years.
2. Patient diagnosed as chronic pancreatitis.
3. Withdrawl / refusal to consent

DIAGNOSTIC CRITERIA:

Acute pancreatitis is diagnosed clinically and biochemically with elevated serum amylase value above the upper reference limit. The diagnosis is further confirmed by PLAIN X-RAY ABDOMEN, USG and CONTRAST ENHANCED CT SCAN.

MATERIALS USED ARE:

I. Serum CRP and Serum Amylase

II. Serum electrolytes

III. Arterial blood gas analysis

IV. CBC and urine investigations

V. Serum creatinine and urea.

OBSERVATION & DISCUSSION

ANALYSIS AND OBSERVATIONS:

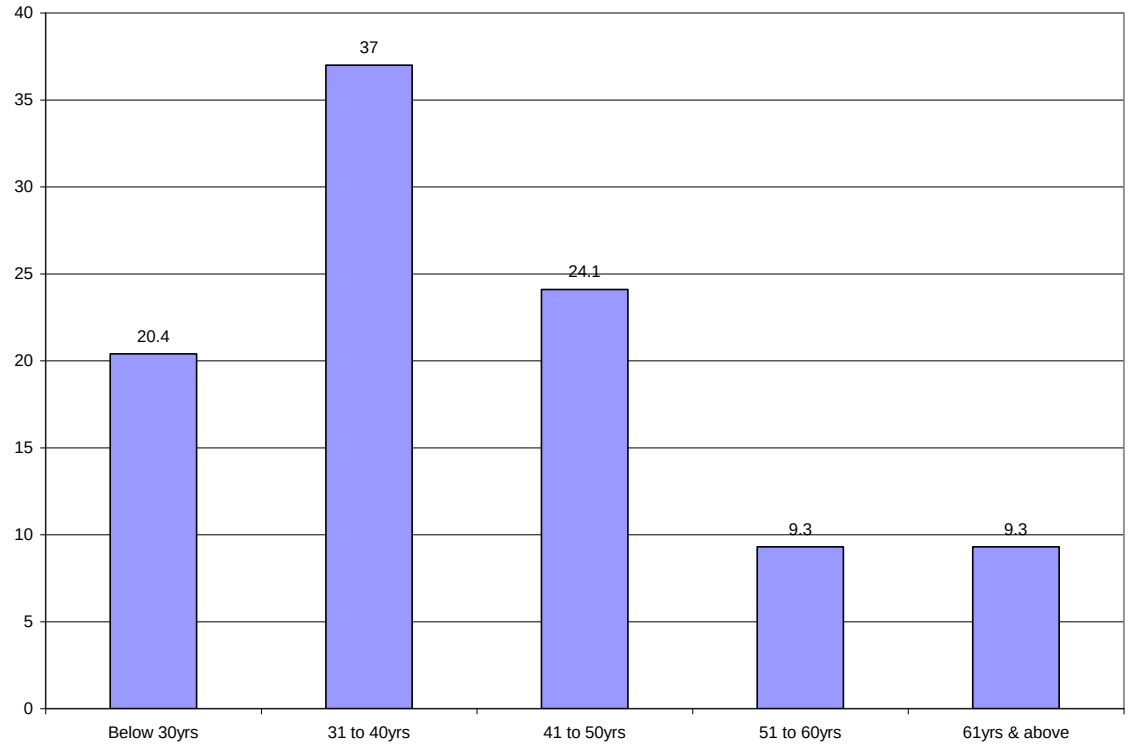
1. RELATIONSHIP of AGE AND SEX IN ASSESSING THE DISEASE:

A total of 54 patients incorporated in this study. Among them 47 are males and 7 are females. The average age of occurrence of the disease is 31 to 40 years which is shown in the following tables and charts.

I.TABLE SHOWING AGE PREPONDERANCE (Frequency Table)

Particulars	**No.of respondents (n=54)**	**Percentage (100%)**
Below 30yrs	11	20.4
31 to 40yrs	20	37.0
41 to 50yrs	13	24.1
51 to 60yrs	5	9.3
61yrs & above	5	9.3

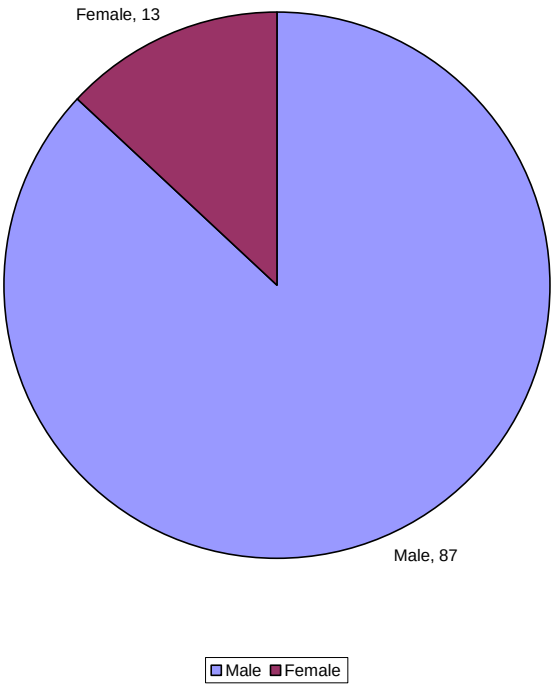
A. AGE CHART


40
35
30
25
20
15
10
5
0
37
24.1
20.4
9.3
9.3
Below 30yrs
31 to 40yrs
41 to 50yrs
51 to 60yrs
61yrs & above

II. TABLE SHOWING SEX PREPONDERANCE

Particulars	**No.of respondents (n=54)**	**Percentage (100%)**
Male	47	87.0
Female	7	13.0

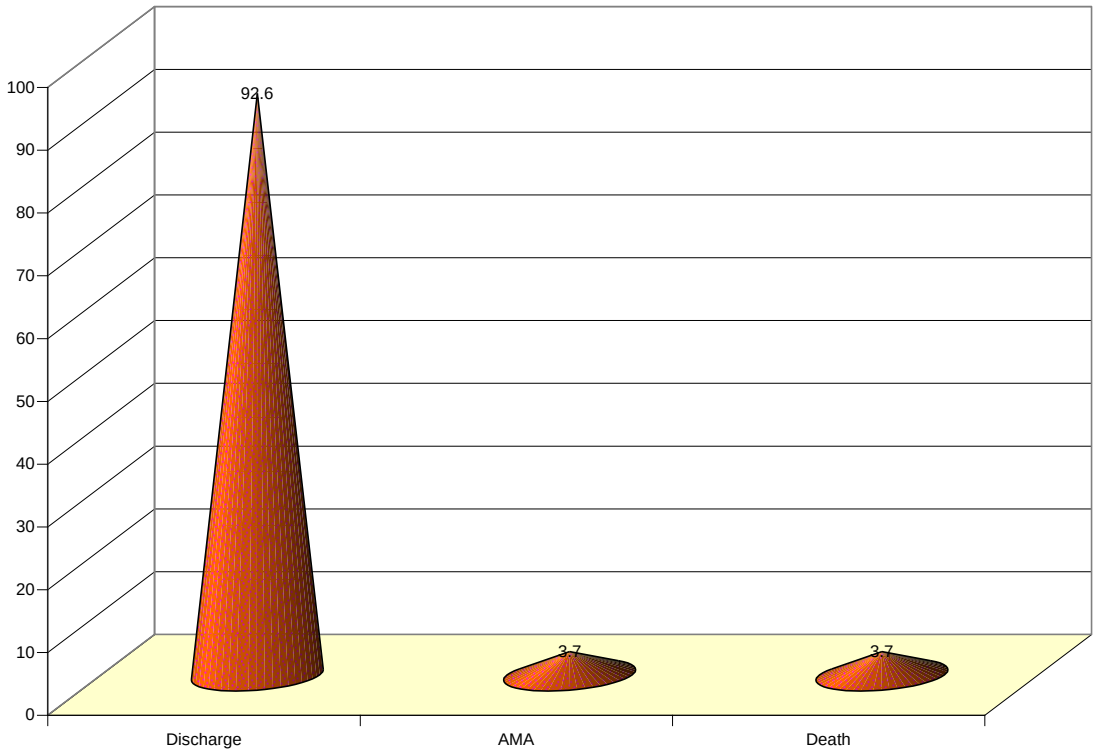
B. DIAGRAM SHOWING GENDER FREQUENCY



III. TABLE SHOWING DISCHARGE/ DEATH/AMA

Particulars	**No.of respondents (n=54)**	**Percentage (100%)**
Discharges	50	92.6
AMA	2	3.7
Death	2	3.7

C.GRAPH SHOWING % OF DISCHARGE/DEATH/AGAINST MEDICAL ADVICE(AMA)



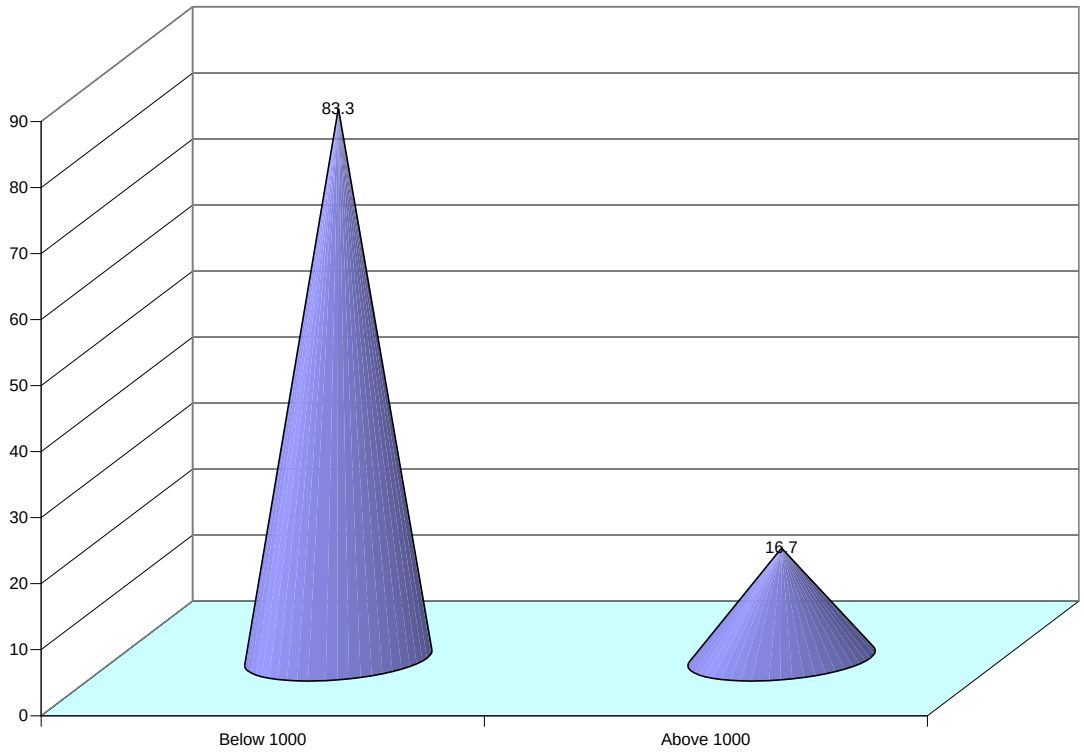
2. RELATIONSHIP OF SERUM AMYLASE, SERUM CRP AND APACHE II SCORE:

A total of 54 patients studied in this study. The following table (4,5,6) shows the serum biomarkers and scoring system values in both mild and severe pancreatitis patients.

IV. TABLE SHOWING SERUM AMYLASE LEVEL IN MILD & SEVERE DISEASE

Particulars	**No.of respondents (n=54)**	**Percentage (100%)**
Below 1000	45	83.3
Above 1000	9	16.7

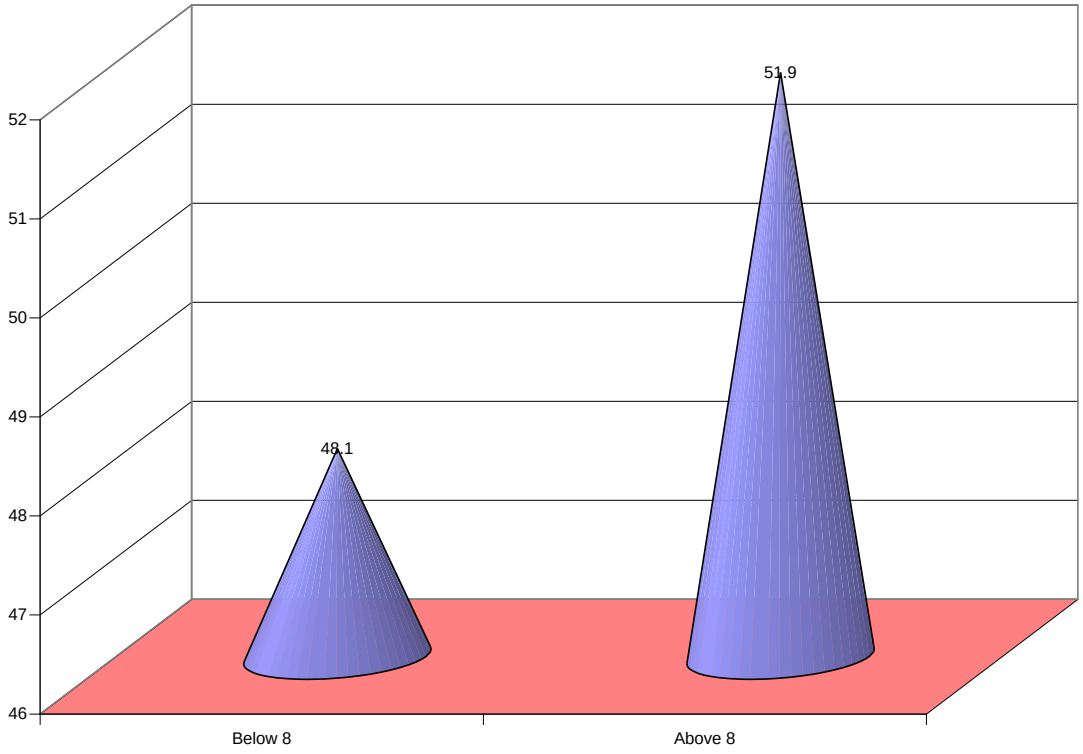
D.GRAPH SHOWING % OF SERUM AMYLASE IN MILD AND SEVERE DISEASE



V. TABLE SHOWING FREQUENCY OF APACHE II SCORE

Particulars	**No.of respondents (n=54)**	**Percentage (100%)**
Below 8	26	48.1
Above 8	28	51.9

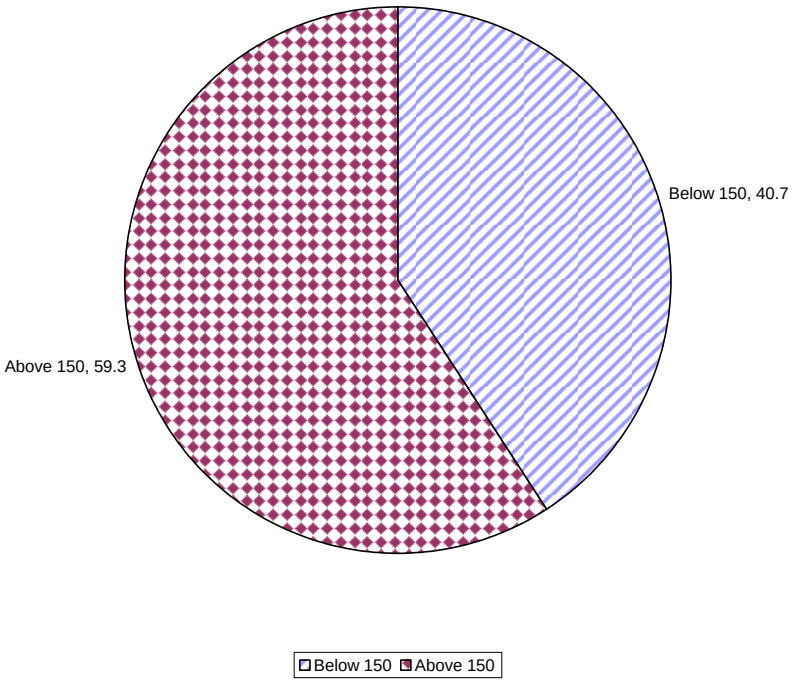
E.GRAPH SHOWING APACHE II SCORE IN MILD AND SEVERE DISEASE



VI. TABLE SHOWING % OF SERUM CRP

Particulars	**No.of respondents (n=54)**	**Percentage (100%)**
Below 150	22	40.7
Above 150	32	59.3

F.**PIE CHART SHOWING SERUM CRP IN MILD AND SEVERE DISEASE**



3. RELATIONSHIP BETWEEN CLINICAL SCORING AND BIOCHEMICAL MARKERS:

It is found in this study that there is a linear progression between the biochemical marker and scoring system by comparing the statistical inference as shown in the following tables and charts.

VII. TABLE SHOWING DESCRIPTIVE STATISTICS

	N	**Min.**	**Max.**	**Mean**	**S.D**
AGE	54	26	71	41.33	11.237
SR. AMYLASE IU/L	54	154	1520	603.26	359.472
APACHEII score	54	3	28	9.54	4.875
SR. CRP mg/L	54	68	198	139.39	26.992

VIII. TABLE SHOWING T-Test AND ITS SIGNIFICANCE

PARAMETER	**n**	**Mean**	**S.D**	**t**	**df**	**Statistical inference**
SR. AMYLASE IU/L						
Below 1000	45	134.78	27.178	-3.014	52	.004<0.05
Above 1000	9	162.44	6.821			Significant
APACHEII score						
Below 8	26	122.81	26.883	-5.374	52	.000<0.05
Above 8	28	154.79	15.822			Significant

IX. **TABLE COMPARING Chi-SQUARE TEST AND ITS SIGNIFICANCE OF BOTH SERUM AMYLASE AND APACHE II SCORE WITH SERUM CRP**

	SR. CRP mg/L						**Statistical inference**
	Below 150		**Above 150**		**Total**		
	n	**%**	**n**	**%**	**n**	**%**	
SR. AMYLASE IU/L							
Below 1000	22	100.0%	23	71.9%	45	83.3%	X^2=7.425 Df=1 .006<0.05 Significant
Above 1000	0	.0%	9	28.1%	9	16.7%	
APACHEII score							
Below 8	19	86.4%	7	21.9%	26	48.1%	X^2=21.717 Df=1 .000<0.05 Significant
Above 8	3	13.6%	25	78.1%	28	51.9%	
Total	**22**	**100.0%**	**32**	**100.0%**	**54**	**100.0%**	

X**. TABLE COMPARING Chi-square test OF SERUM AMYLASE AND SERUM CRP WITH APACHE II SCORE**

	APACHEII score						**Statistical inference**
	Below 8		**Above 8**		**Total**		
	n	**%**	**n**	**%**	**n**	**%**	
SR. AMYLASE IU/L							
Below 1000	24	92.3%	21	75.0%	45	83.3%	X^2=2.908 Df=1 .088>0.05 Not Significant
Above 1000	2	7.7%	7	25.0%	9	16.7%	
SR. CRP mg/L							
Below 150	19	73.1%	3	10.7%	22	40.7%	X^2=21.717 Df=1 .000<0.05 Significant
Above 150	7	26.9%	25	89.3%	32	59.3%	
Total	**26**	**100.0%**	**28**	**100.0%**	**54**	**100.0%**	

XI. **TABLE COMPARING Chi-square test OF SERUM CRP AND APACHE II SCORE WITH SERUM AMYLASE**

	AMYLASE IU/L						Statistical inference
	Below 1000		Above 1000		Total		
	n	%	N	%	N	%	
SR. CRP mg/L							
Below 150	22	48.9%	0	.0%	22	40.7%	X^2=7.425 Df=1 .006<0.05 Significant
Below 150	23	51.1%	9	100.0%	32	59.3%	
APACHEII score							
Below 8	24	53.3%	2	22.2%	26	48.1%	X^2=2.908 Df=1 .088>0.05 Not Significant
Above 8	21	46.7%	7	77.8%	28	51.9%	
Total	45	100.0%	9	100.0%	54	100.0%	

STATISTICS METHOD:

Results were expressed as mean±SE. Statistical analyses were made using Student *t* test, Chi square test. P value < 0.05 were accepted as statistically significant.

RESULTS:

Among 54 patients, 32 had severe disease and 22 had mild disease based on serum CRP ($P<0.05$). Serum amylase and Apache ii scoring system were analysed on the first day of admission. Serum CRP taken at 48 hours of admission. The average age of occurrence 31 to 40 years. Males are more commonly affected than females. Alcohol was the leading cause of death in both mild and severe disease. In this study upper limit for serum amylase were 1000U/L, Apache II score >8 and serum CRP >150mg/L. The percentile of patients for mild and severe pancreatitis for serum amylase, Apache ii score and serum CRP includes 83.8%, 48%, 40.7% and 16.7%, 51.9%, 59.3%. The standard deviation of serum amylase, Apache ii score and serum CRP includes 359.472, 4.875, 26.992. The statistical inference of all the three parameters comparing one value with other parameters shows serum CRP has significant value of $P<0.05$. Among the 32 patients with severe disease, two patients died after developing multiple organ failure. Others had pancreatic necrosis, renal and respiratory failure.

FOLLOW UP:

Out of 54 patients, 50 patients are discharged, 2 patients died and 2 patients went on against medical advice. Among the 50 patients, 20 patients did not turn up for follow up. It was found that 16 of 20 patients who had alcoholic pancreatitis had recurring episodes and had repeated hospital admissions. About 4 out of 20 patients who had biliary pathology had recurred and these were due to retained calculi in biliary tract.

CONCLUSION:

1. Serum CRP is the important single prognostic marker of predicting the severe pancreatitis with the cut off value of 150mg/L.
2. CRP levels increase significantly in early stages of pancreatic necrosis.
3. CRP plays a critical role in initial process of diagnosis, as an early predictive indicator of severity of the disease and helps in detecting the mortality in this study.
4. Serum CRP plays a major role in stratifying the patients for early aggressive intervention of acute pancreatitis to reduce morbidity and mortality.

ANNEXURE

CASE PROFORMA

Name: Age/SEX: IP.NO: UNIT:

Date of admission: Date of Discharge:

1. COMPLAINTS:

a. Pain Abdomen

i) Duration ii) Mode of onset

iii) Site of pain IV) Nature

v) Radiation VI) Shifting of pain

vii) Aggravating / Relieving factors

b. Vomiting

i) Duration ii) Frequency

iii) Vomitus: Bilious / Blood / Faecal / otherwise.

c. Fever

i) Duration ii) Degree

iii) Chills / Rigors

d. Distension of abdomen

i) Present / absent:

ii) Duration (if present):

e. Bowel symptoms.

i) Diarrhoea ii) Constipation

iii)Hematemesis/Melaena

f. H/o reduced / nil urine output.

g. Other complaints (if any):

2. PAST HISTORY:

a) H/o Previous operation

b) H/o Trauma

c) Any comorbid illness:

SHT/DM/TB/COPD/CAD.

3. PERSONAL HISTORY:

a) Diet : Mixed / Vegetation

b) Appetite: Good / Impaired

c) Habits: Smoker / Alcoholic

4. FAMILY HISTORY:

Any other member of the family suffered from similar disease

5. GENERAL PHYSICAL EXAMINATION:

a) Vital signs:

Pulse: Blood Pressure:

Temperature: Respiratory rate:

b) Built and Nutrition:

c) State of hydration

d) Anaemia / Jaundice / Pedal edema / Lymphadenopathy

6. EXAMINATION OF THE ABDOMEN

a) Inspection

1. Shape 2.Umbilicus

3. Skin 4. Movement with respiration

5. Visible veins 6. Visible pulsation

7. Hernial orifices 8. External genitalia

9. Distension

b) Palpation:

1. Site of tenderness 2. Guarding / Rigidity

3. Whether liver / spleen palpable

c) Percussion

Liver dullness obliterated

d) Auscultation

Bowel sounds: Present / Absent

e) Per rectal examination

7. EXAMINATION OF OTHER SYSTEMS

a. Cardiovascular system

b. Respiratory system

c. Nervous system

8. INVESTIGATION

Blood investigations:

1.Hb% TC Blood Group

2. Urine Sug Alb Micro

3. RBS, Urea, Creatinine, Serum electrolytes, Serum amylase, Serum lipase, Serum CRP, Arterial blood gas analysis

4.Special: X-ray abdomen (Erect / lateral decubitus)

X-ray Chest PA view

5. USG abdomen & pelvis

6. CT abdomen & pelvis

9. CLINICAL DIAGNOSIS

10. MANAGEMENT

Medical Management

Pre operative treatment

Operative details

Post operative management

11. FINAL DIAGNOSIS

12. COMPLICATIONS

13. OUTCOME

MASTER CHART

S.NO	NAME	AGE/SEX	IP NO	DOA	DOD/AMA/DEATH	SR. AMYLASE IU/L	APACHEII SCORE	SR. CRP mg/L
1	PANCHAVARNAM	65/F	50621	03.10.15	12.10.15/DIS	625	17	152
2	SELVAKUMAR	35/M	51236	06.10.15	27.10.15/DIS	860	10	165
3	TAMILSELVAN	45/M	52218	08.10.15	19.10.15/DIS	608	13	198
4	BALACHANDRAN	51/M	53640	26.10.15	06.11.15/DIS	436	13	151
5	RAJARAJAN	30/M	54263	02.11.15	14.11.15/DIS	520	7	144
6	BALAN	35/M	58719	06.11.15	21.11.15/DIS	632	9	153
7	NATARAJAN	71/M	58958	06.11.15	13.11.15/DEATH	726	24	161
8	BOOPATHY	43/M	59102	09.11.15	26.11.15/DIS	1032	10	153
9	PITCHAIKANNU	44/M	59626	09.11.15	24.11.15/DIS	638	9	155
10	SUNDARESAN	70/M	59784	09.11.15	17.11.15/DEATH	1250	28	157
11	RAJAPANDI	26/M	59906	09.11.15	16.11.15/DIS	216	9	119
12	VENKATESH	34/M	61252	12.11.15	14.11.15/AMA	724	8	151
13	BALACHANDAR	50/M	61528	13.11.15	23.11.15/DIS	325	9	119
14	KUMAR	55/M	64206	15.11.15	02.12.15/DIS	1429	16	176
15	ALAGESAN	29/M	65122	16.11.15	30.11.15/DIS	376	6	124
16	KALIYAMOORTHY	35/M	65848	18.11.15	29.11.15/DIS	173	7	68
17	SHANMUGAVEL	28/M	68456	18.11.15	28.11.15/DIS	455	7	113
18	RAMESH	37/M	68975	18.11.15	26.11.15/DIS	255	8	108
19	SURESH	30/M	69802	21.11.15	29.11.15/DIS	450	9	151
20	PERIYASAMY	47/M	69865	21.11.15	02.12.15/DIS	652	12	159
21	RAJA	42/M	69902	23.11.15	05.12.15/DIS	1520	18	162
22	SELVARAJ	33/M	75210	30.11.15	12.12.15/DIS	198	7	84
23	KASINATHAN	35/M	79620	05.12.15	19.12.15/DIS	320	7	129
24	MURUGESAN	50/M	80546	09.12.15	23.12.15/DIS	480	9	151
25	JAYAKUMAR	35/M	84230	14.12.15	28.12.15/DIS	240	6	130
26	SARAVANAN	29/M	89745	19.12.15	26.12.15/DIS	180	3	95
27	RAJESH	28/M	90123	21.12.15	30.12.15/DIS	362	4	116
28	ANTHONIRAJ	38/M	95102	23.12.15	01.01.16/DIS	154	5	78
29	CHANDRAMOHAN	37/M	98745	25.12.15	05.01.16/DIS	168	6	86
30	ULAGANATHAN	40/M	99156	25.12.15	09.01.16/DIS	980	9	154
31	MAHALINGAM	45/M	10241	27.12.15	14.01.16/DIS	236	8	112
32	SUBBAIYAN	41/M	15201	29.12.15	10.01.16/DIS	330	5	106
33	SUBRAMANI	30/M	1025	05.01.16	16.01.16/DIS	675	10	152
34	PURUSOTHRAJ	38/M	1536	11.01.16	29.01.16/DIS	1275	10	160
35	NOORJAHAN	65/F	1856	15.01.16	29.01.16/DIS	395	14	120
36	THIRUGNAGAM	26/M	1965	16.01.16	02.02.16/DIS	534	8	152
37	PANDIDURAI	55/M	1994	18.01.16	31.01.16/DIS	859	14	153
38	SATHISH	34/M	2013	24.01.16	14.02.16/DIS	1086	4	162
39	ARUNRAJ	39/M	2145	28.01.16	19.02.16/DIS	1032	5	158

40	BOOPATHI	42/M	2235	01.02.16	03.03.16/DIS	1280	11	168
41	SARAVANAN	43/M	2531	15.02.16	25.02.16/DIS	436	8	151
42	HARIHARAN	40/M	2687	19.02.16	05.03.16/DIS	424	8	152
43	VASUKI	40/F	2745	23.02.16	12.03.16/DIS	386	4	136
44	SHANMUGAVEL	28/M	4920	25.03.16	13.03.16/DIS	410	5	142
45	SELVI	32/F	5201	06.04.16	26.04.16/DIS	234	4	116
46	GUNASEKARAN	37/M	6987	26.04.16	09.05.16/DIS	1216	9	166
47	VADIVEL	40/M	7832	05.05.16	21.05.16/DIS	287	5	120
48	PALANISAMY	29/M	8984	30.05.16	12.06.16/DIS	687	5	156
49	KALA	40/F	9520	16.06.16	29.06.16/DIS	256	6	104
50	MURUGAN	48/M	10654	29.06.16	10.07.16/DIS	940	13	161
51	KUMARESAN	45/M	15874	19.07.16	03.08.16/DIS	510	15	157
52	VELAMMAL	52/F	19380	05.08.16	23.08.16/DIS	435	12	153
53	GANDHI	53/M	25016	16.08.16	05.09.16/DIS	840	14	152
54	PATTAMMAL	63/F	39523	31.08.16	14.09.16/AMA	829	13	156

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