Original Research Paper General Surgery

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

Dr.V.Kopperundevi	AssociateProfessor, Department of General Surgery, Thanjavur Medical college, Thanjavur, TN MGR University, Tamilnadu, India
Dr.K.Sathyabama	Professor, Department of General Surgery, Thanjavur Medical college, Thanjavur
Dr.M.Thamarai kannan	(MS) Post graduate, Department of General Surgery, Thanjavur Medical college, Thanjavur

ABSTRACT

Acute pancreatitis is a disease with wide clinical variation, which makes its diagnosis complex. Serum/urinary amylase measurement is a standard diagnostic method, although it was shown to be unable to recognize one fifth of Acute pancreatitis patients. Most of the cases are mild and conservative treatment results in a rapid recovery .. However, severe Acute pancreatitis constitutes 15-20% of all cases and mortality rate approaches 2-10%. The main value of C Reactive protein is a guide to the severity of pancreatic inflammation. An accurate prediction of severity and prognostic monitoring are necessary to anticipate the early and late complications so as to consider aggressive treatment.

Aim : The aim of the study is to analyse the role of C-Reactive Protein, Serum Amylase and APACHE II Scoring System in predicting the severity of Acute Pancreatitis. This prospective study was conducted in the department of General Surgery, Thanjavur Medical College. About 54 patients were included in this study during the period of October 2015 to September 2016.

KEYWORDS Acute Pancreatitis, CRP, Serum amylase

I. INTRODUCTION

Acute Pancreatitis is an acute inflammation of the prior normal gland parenchyma which is usually reversible with raised pancreatic enzymes level in blood and urine. Acute attack can also occur in pre existing chronic pancreatitis. Although the disease process may be limited to pancreatic tissue, it also can involve peri pancreatic tissues or more distant organ sites. .Mild acute pancreatitis has a very low mortality rate (less than 1 percent), whereas the death rate for severe acute pancreatitis can be 10 to 30 percent depending on the presence of sterile versus infected necrosis. The revised Atlanta classification system divides acute pancreatitis into two broad categories.

- Interstitial edematous acute pancreatitis, which is characterized by acute inflammation of the pancreatic parenchyma and peripancreatic tissues, but without recognizable tissue necrosis
- Necrotizing acute pancreatitis, which is characterized by inflammation associated with pancreatic parenchymal necrosis and/or peripancreatic necrosis

According to the severity, acute pancreatitis is divided into the following:

- Mild acute pancreatitis which is characterized by the absence of organ failure and local or systemic complications
- Moderately severe acute pancreatitis which is characterized by transient organ failure (resolves within 48 hours) and/or local or systemic complications without persistent organ failure (>48 hours)
- Severe acute pancreatitis which is characterized by persistent organ failure that may involve one or multiple organs

Local complications of acute pancreatitis include acute peripancreatic fluid collection, pancreatic pseudocyst, acute necrotic collection, and walled-off necrosis.

Organ failure is defined as a score of two or more for any one of three organ systems (respiratory, cardiovascular, or renal) using the modified Marshall scoring system.

Modified Marshall scoring system for organ dysfunction

Organ system	Score				
	0	**1**	**2**	**3**	**4**
Respiratory (PaO_2/FiO_2)	>400	301-400	201-300	101-200	≤101
Renal*					
(serum creatinine, micromol/L)	≤134	134-169	170-310	311-439	>439
(serum creatinine, mg/dL)	<1.4	1.4-1.8	1.9-3.6	3.6-4.9	>4.9
Cardiovascular (systolic blood pressure,mmHg)¶	>90	<90, fluid responsive	<90, not fluid responsive	<90, pH <7.3	<90, pH <7.2
For nonventilated patients, the FiO_2 can be estimated from below:					
Supplemental oxygen (L/min)	FiO_2 (percent)				
Room air	21				
2	25				
4	30				
6-8	40				
9-10	50				

A score of 2 or more in any system defines the presence of organ failure.

* A score for patients with pre-existing chronic renal failure depends on the extent of further deterioration of baseline renal function. No formal correction exists for a baseline serum creatinine ≥134 micromol/L or ≥1.4 mg/dL. Off inotropic support.

Etiology

There are many causes of acute pancreatitis, which can be easily identified in 75%-85% of patients. In developed countries, obstruction of the common bile duct by stones (38%) and alcohol abuse (36%) are the most frequent causes of acute pancreatitis. The other causes are listed below

Mechanical	Gallstones, biliary sludge, ascariasis, periampullary diverticulum, pancreatic or periampullary cancer, ampullary stenosis, duodenal stricture or obstruction
Toxic	Ethanol, methanol, scorpion venom, organophosphate poisoning
Metabolic	Hyperlipidemia (types I, IV, V), hypercalcemia
Drugs	Didanosine, pentamidine, metronidazole, stibogluconate, tetracycline furosemide, thiazides, sulphasalazine, 5-ASA, L-asparaginase, azathioprine, valproic acid, sulindac, salicylates, calcium, estrogen
Infection	Viruses-mumps, coxsackie, hepatitis B, CMV, varicella-zoster, HSV, HIV
	Bacteria-mycoplasma, Legionella, Leptospira, salmonella
	Fungi-aspergillus
	Parasites-toxoplasma, cryptosporidium, Ascaris
Trauma	Blunt or penetrating abdominal injury, iatrogenic injury during surgery or ERCP (sphincterotomy)
Congenital	Cholodochocele type V pancreas divisum
Vascular	Ischemia, atheroembolism, vasculitis (polyarteritisnodosa, SLE)
Miscellaneous	Post ERCP, pregnancy, renal transplantation, alpha-1-antitrypsin deficiency
Genetic	CFTR and other genetic mutations (cystic fibrosis transmembrane conductance regulator)

Two factors have been suggested as the possible initiating event in gallstone pancreatitis: reflux of bile into the pancreatic duct due to transient obstruction of the ampulla during passage of gallstones or obstruction at the ampulla secondary to stone(s) or edema resulting from the passage of a stone.

It is unclear why alcohol-induced pancreatitis occurs only after many years of alcohol abuse and not after a single binge in individuals not habituated to alcohol use. However, several mechanisms have been proposed.

- Sensitization of acinar cells to cholecystokinin (CCK)-induced premature activation of zymogens
- Potentiation of the effect of CCK on the activation of transcription factors, nuclear factor kB, and activating protein-1
- Generation of toxic metabolites such as acetaldehyde and fatty acid ethyl esters
- Sensitization of the pancreas to the toxic effects of coxsackie virus B3
- Activation of pancreatic stellate cells by acetaldehyde and oxidative stress and subsequent increased production of collagen and other matrix proteins

In hyperlipidemia, free fatty acids are released from serum triglycerides in toxic concentrations by the action of pancreatic lipase within pancreatic capillaries

Premature activation of pancreatic zymogens within the pancreas has also been proposed as the pathogenetic mechanism for the acute attacks of pancreatitis seen in patients with hereditary pancreatitis.

Mutations in the **s**erine **p**rotease **in**hibitor **K**azal type **1 gene** (SPINK1, also called pancreatic secretory trypsin inhibitor gene) also may present in an autosomal recessive pattern. CFTR-associated disorders include chronic pancreatitis with minimal lung disease, and this trait may occur in multiple family members. Autosomal dominant hereditary pancreatitis — This is most often associated with mutations in the serine protease 1 gene (PRSS1) on chromosome 7q35, which encodes trypsin-1 (cationic trypsinogen)

The exocrine pancreas synthesizes and secretes a variety of digestive enzymes that normally become activated after reaching the duodenum. Small amounts of trypsinogen are spontaneously activated, but the pancreas has mechanisms to quickly remove activated trypsin:

- The first line of defense is the pancreatic secretory trypsin inhibitor (PSTI or SPINK1), which can bind and inactivate about 20 percent of the trypsin activity.
- The second line of defense is autolysis of prematurely activated trypsin. Absence of this mechanism is postulated to cause hereditary pancreatitis.
- Another defense mechanism involves mesotrypsin and enzyme Y, which lyses and inactivates trypsin.
- Nonspecific antiproteases such as alpha-1 antitrypsin and alpha-2-macroglobulin are present in the pancreatic interstitium.

The underlying mechanism in the acute pancreatitis is the intraacinar activation of these pancreatic enzymes, which ultimately leads to an autodigestive injury to the gland. The normal defense mechanisms of the pancreas are overwhelmed by the large amounts of trypsin released. In addition, the intrapancreatic release of trypsin leads to activation of more trypsin, and other pancreatic enzymes such as phospholipase, chymotrypsin, and elastase. Trypsin also activates other enzyme cascades including complement, kallikrein-kinin, coagulation, and fibrinolysis. As the inflammatory meadiators releases in to the circulation , systemic complications can arise such as haemodynamic instability, bacterimia (due to translocation of gut flora) acute respiratory distress syndrome, pleural effusion renal failure,and disseminated intra vascular coagulation

The majority of patients with acute pancreatitis have acute onset of severe upper abdominal pain. Patients may have associated nausea and vomiting. On physical examination, patients have abdominal tenderness on palpation.Patients may have abdominal distention and hypoactive bowel sounds due to an ileus secondary to inflammation.

Patients may have scleral icterus due to obstructive jaundice due to choledocholithiasis In 3 percent of patients with acute pancreatitis, ecchymotic discoloration may be observed in the periumbilical region (Cullen's sign) or along the flank (Grey Turner sign . These findings, although nonspecific, suggest the presence of retroperitoneal bleeding in the setting of pancreatic necrosis. Patients with severe acute pancreatitis may have fever, tachypnea, tachycardia, hypoxemia, and hypotension.

1.1 Investigations:

Serum amylase rises within 6 to 12 hours of the onset of acute pancreatitis. Amylase has a short half-life of approximately 10 hours and in uncomplicated attacks returns to normal within three to five days. Serum amylase elevation of greater than three times the upper limit of normal has a sensitivity for the diagnosis of acute pancreatitis of 67 to 83 percent and a specificity of 85 to 98 percent. Given the short half-life of amylase, the diagnosis of acute pancreatitis may be missed in patients who present >24 hours after the onset of pancreatitis. In addition, elevations in serum amylase are not specific for acute pancreatitis and may be seen in other conditions like acute cholecystitis parotitis

Serum lipase — Serum lipase has a sensitivity and specificity for acute pancreatitis ranging from 82 to 100 percent . Serum lipase rises within four to eight hours of the onset of symptoms, peaks at 24 hours, and returns to normal within 8 to 14 days.Lipase elevations occur earlier and last longer as compared with elevations in amylase and are therefore especially useful in patients who present >24 hours after the onset of pain Serum lipase is also more sensitive as compared with amylase in patients with pancreatitis secondary to alcohol.

Other enzymes and products — Trypsinogen activation peptide (TAP), a five amino-acid peptide that is cleaved from trypsinogen to produce active trypsin, is elevated in acute pancreatitis. Since

activation of trypsin is likely an early event in the pathogenesis of acute pancreatitis, TAP may be useful in detection of early acute pancreatitis and as a predictor of the severity of acute pancreatitis . Urinary and serum trypsinogen-2 levels are elevated in early acute pancreatitis. However, additional studies are needed to determine their role in the diagnosis of acute pancreatitis . Other pancreatic digestive enzymes that leak into the systemic circulation and are elevated in serum include trypsin, phospholipase, carboxypeptidase, carboxylester lipase, colipase, and pancreatic isoamylase.

Markers of immune activation — Activation of granulocytes and macrophages in acute pancreatitis results in release of a number of cytokines and inflammatory mediators. Acute pancreatitis is associated with elevations in C-reactive protein (CRP), interleukin (IL)-6, IL-8, IL-10, tumornecrosis factor (TNF), and PMN elastase

A CRP level above 150 mg/dL at 48 hours is associated with severe pancreatitis.— C-reactive protein (CRP) is one of the acute phase reactants made by the liver in response to interleukin-1 and interleukin-6. Levels of CRP above 150 mg/L at 48 hours discriminate severe from mild disease. At 48 hours, CRP above 150 mg/L has a sensitivity, specificity, positive predictive value, and negative predictive value of 80, 76, 67, and 86 percent, respectively, for severe acute pancreatitis.

Patients with pancreatitis may have leukocytosis and an elevated hematocrit from hemoconcentration due to extravasation of intravascular fluid into third spaces. Metabolic abnormalities including elevated blood urea nitrogen (BUN), hypocalcemia, hyperglycemia, and hypoglycemia may also occur.

Chest radiographs — A pleural effusion and/or pulmonary infiltrates during the first 24 hours may be associated with necrosis and organ failure.

CT scan — CT scan is probably the most frequently used radiologic investigation when severe AP is suspected. It is used to look for pancreatic necrosis and extrapancreatic inflammation. Intravenous contrast-enhanced CT distinguishes between edematous and necrotizing pancreatitis, since areas of necrosis and exudates do not enhance. CT is more accurate than ultrasonography for the diagnosis of severe pancreatic necrosis (90 versus 73 percent in one report).CT scan is not required on the first day unless there are other diagnoses are being considered.

MRI is as effective as CT in demonstrating the presence and extent of pancreatic necrosis and fluid collections, and is probably superior for indicating the suitability of such collections for nonsurgical drainage.MRI can characterize the "pancreatic necrosis" seen on CT as necrotic pancreatic parenchyma, peripancreatic necrotic fluid collections, or hemorrhagic foci. One study found that MRI was reliable for staging the severity of AP and predicting prognosis with fewer contraindications than CT. It can also detect pancreatic duct disruption, which can occur early in the course of AP.

1.2 SCORING SYSTEMS — Many scoring systems have been reported to assess the severity of acute pancreatitis.Ranson's criteria — A score based upon Ranson's criteria is one of the earliest scoring systems for severity in AP. Ranson's criteria consist of 11 parameters. Five of the factors are assessed at admission and six are assessed during the next 48 hours. A later modification for biliary pancreatitis included only 10 points. Mortality increases with an increasing score.

The APACHE II score — The Acute Physiology and Chronic Health Examination (APACHE) II score was originally developed for critically ill patients in intensive care units (ICUs). It has 12 physiologic measures and extra points based upon age and presence of chronic disease .It is probably the most widely studied severity scoring system in AP. It has good negative predictive value and modest positive predictive value for predicting severe AP and can be performed daily. Decreasing values during the first 48 hours suggest a mild attack, while increasing values suggest a severe attack. Studies suggest that mortality is less than 4 percent with a score <8 and is 11 to 18 percent with a score >8.

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

Some limitations of the APACHE II score are that is complex and cumbersome to use, it does not differentiate between interstitial and necrotizing pancreatitis, and it does not differentiate between sterile and infected necrosis. Finally, it has a poor predictive value at 24 hours.

Several additional variables were added to APACHE II to improve its accuracy leading to the development of APACHE III. Both APACHE II and III scores use physiology, age, and chronic health to calculate prognosis; they differ in total score, the number of physiologic variables (12 for APACHE II versus 17 for APACHE III), and the assessment of chronic health status. However, the APACHE III system does not appear to be as useful as APACHE II for distinguishing mild from severe attack.

Systemic inflammatory response syndrome score includes Two or more of the following conditions:

Temperature >38.3°C or <36.0°C
Heart rate of >90 beats/minute
Respiratory rate of >20 breaths/minute or $PaCO_2$ of <32 mmHg
WBC count of >12,000 cells/mL, <4000 cells/mL, or >10 percent immature (band) forms

It can reliably predict the severity of pancreatitis and has the added advantage that it can be applied easily at the bedside every day.A study found that the severity of AP was greater among patients with AP and SIRS on day one, particularly in those with three or four SIRS criteria, compared with those without SIRS on day one. Thus, it appears that the SIRS score is inexpensive, readily available, and compares favourably with other more complicated scores.

CT severity index — A CT severity score (the Balthazar score) has been developed based upon the degree of necrosis, inflammation, and the presence of fluid collections

CT findings and grading of acute pancreatitis (CT severity index [CTSI])

Grading based upon findings on unenhanced CT		
Grade	Findings	Score
A	Normal pancreas - normal size, sharply defined, smooth contour, homogeneous enhancement, retroperitoneal peripancreatic fat without enhancement	0
B	Focal or diffuse enlargement of the pancreas, contour may show irregularity, enhancement may be inhomogeneous but there is no peripancreatic inflammation	1
C	Peripancreatic inflammation with intrinsic pancreatic abnormalities	2
D	Intrapancreatic or extrapancreatic fluid collections	3
E	Two or more large collections of gas in the pancreas or retroperitoneum	4

Necrosis score based upon contrast enhanced CT	
Necrosis, percent	Score
0	0
<33	2
33-50	4
≥50	6

CT severity index equals unenhanced CT score plus necrosis score: maximum = 10, ≥6 = severe disease.

American College of Gastroenterology (ACG) - ACGguidelines recommend the following clinical findings associated with a severe course for initial risk assessment:

Patient characteristics
-Age >55 years
Obesity (body mass index >30 kg/m^2)
Altered mental status
Comorbid disease

The systemic inflammatory response syndrome
-Presence of >2 of the following criteria:
-Pulse >90 beats/min
-Respirations >20/min or $PaCO_2$>32 mm Hg
-Temperature >38 or <36°C
-White blood cell count >12,000 or <4,000 cells/mm^3 or >10 percent immature neutrophils (bands)

Laboratory findings
-Blood urea nitrogen (BUN) >20 mg/dl
-Rising BUN
-Hematocrit (HCT) >44 percent
-Elevated creatinine
-Rising HCT

* Radiology findings
-Pleural effusions
-Pulmonary infiltrates
- Multiple or extensive extrapancreatic collections

The presence of organ failure and/or pancreatic necrosis defines severe acute pancreatitis.

1.3 MANAGEMENT
Acute pancreatitis is treated with supportive care including pain control, intravenous fluids especially during the first 24 hours, and correction of electrolyte and metabolic abnormalities. The majority of patients with mild pancreatitis require no further therapy, and recover within three to seven days. Patients with moderately severe or severe acute pancreatitis, signs of sepsis, or clinical deterioration 72 hours after initial presentation, should undergo a contrast-enhanced CT scan to assess the presence of pancreatic or extra pancreatic necrosis and local complications. Local complications of acute pancreatitis include acute peri pancreatic fluid collection, pancreatic pseudocyst, acute necrotic collection (ANC), and walled-off necrosis .Patients with severe pancreatitis are at increased risk for intra-abdominal hypertension and abdominal compartment syndrome due to tissue edema from aggressive fluid resuscitation, peripancreatic inflammation, ascites, and ileus. Patients in the intensive care unit should be monitored for potential abdominal compartment syndrome with serial measures of urinary bladder pressures. Management initially consists of careful observation and supportive care. In some cases abdominal compartment decompression is required

Infected necrosis should be suspected in patients with pancreatic or extrapancreatic necrosis who deteriorate (clinical instability or sepsis , increasing white blood cell count, fevers) or fail to improve after 7 to 10 days of hospitalization.

These patients should either undergo initial CT-guided fine needle aspiration (FNA) for Gram stain and culture to guide use of appropriate antibiotics or empiric antibiotics may be started without CT-guided FNA.Necrosectomy should be accomplished initially by a minimally invasive approach (endoscopic or percutaneous radiologic). Open surgical necrosectomy should be used if minimally invasive methods are not possible or fail In patients with gallstone pancreatitis, we recommend urgent (<24 hours) endoscopic retrograde cholangiopancreatography (ERCP) and sphincterotomy for patients with cholangitis. Cholecystectomy should be performed after recovery from acute pancreatitis in all operable patients with gallstone pancreatitis or biliary sludge.

II.RESULTS
A total of 54 patients included 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.

Relationship of age and sex in assessing the disease:

TABLE.1 SHOWING AGE PREPONDERANCE

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

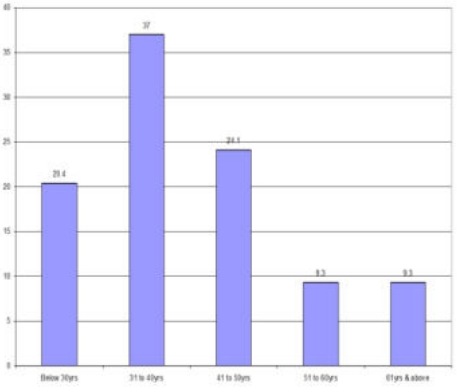
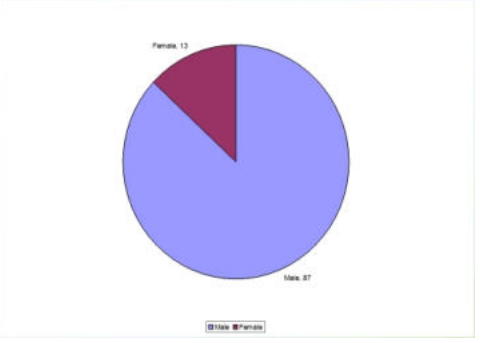


TABLE.2 SHOWING SEX PREPONDERANCE

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



Relationship of Serum Amylase, Serum CRP and apache II score:

TABLE.3 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

GRAPH SHOWING % OF SERUM AMYLASE IN MILD AND SEVERE DISEASE

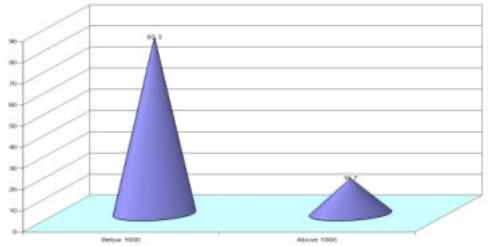


TABLE.4 SHOWING FREQUENCY OF APACHE II SCORE

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

GRAPH SHOWING APACHE II SCORE IN MILD AND SEVERE DISEASE

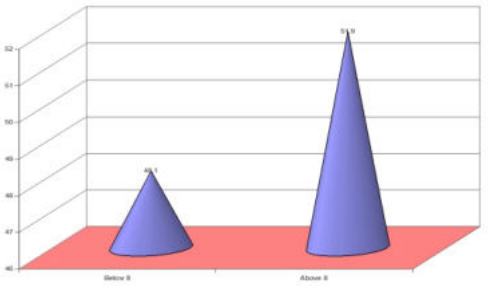


TABLE.3 SHOWING DISCHARGE/ DEATH/AMA

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

GRAPH SHOWING %OF DISCHARGE/DEATH/AGAINST MEDICAL ADVICE

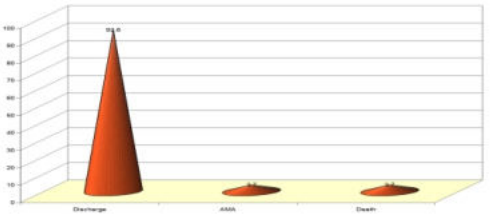
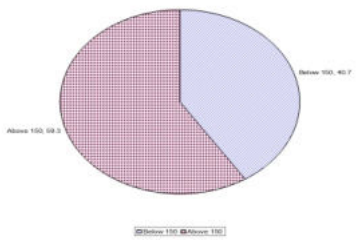


TABLE .4 SHOWING % OF SERUM CRP

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

PIE CHART SHOWING SERUM CRP IN MILD AND SEVERE DISEASE



1.1 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

TABLE 5.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

TABLE 6.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

TABLE.7 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%**	

TABLE 8. 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%**	

TABLE 9. 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 %	

2.2 STATISTICS:
Results were expressed as mean±SE. Statistical analyses were made using Student t test, Chi square test. P value less than 0.05 were accepted as statistically significant.

Among the 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. Male are more commonly affected than females. Alcohol were 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. Hospital stay was significantly longer in the group with severe disease compared to mild disease (P<o.05).

2.3 FOLLOW UP:
Out of 54 patients, 50 patients are discharged, 2 patients died and 2 patients went on against medical advice. . 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.

III.CONCLUSION
Serum CRP is the important single prognostic marker of predicting the severe pancreatitis with the cut off value of 150mg/ml.CRP levels increase significantly in early stages of pancreatic necrosis. 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. Serum CRP plays a major role in stratifying the patients for early aggressive intervention of acute pancreatitis to reduce morbidity and mortality.

IV.REFERENCES

1. Acosta and Ledesma, 1974. Acosta JM, Ledesma CL: Gallstone migration as a cause of acute pancreatitis. N Engl J Med 1974; 290:484-487.
2. Ammori, 2003. Ammori BJ, et al: The biochemical detection of biliary etiology of acute pancreatitis on admission: a revisit in the modern era of biliary imaging. Pancreas 2003; 26:32-35.
3. Armstrong, 1985. Armstrong CP, et al: The biliary tract in patients with acute gallstone pancreatitis. Br J Surg 1985; 72:551-555.
4. Aube, 2005. Aube C, et al: MR cholangiopancreatography versus endoscopic sonography in suspected common bile duct lithiasis: a prospective comparative study. AJR Am J Roentgenol 2005; 184:55-62.
5. Balthazar, 1990. Balthazar EJ, et al: Acute pancreatitis: value of CT in establishing prognosis. Radiology 1990; 174:331-336.
6. Beger, 1986. Beger HG, et al: Hemodynamic data patterns in patients with acute pancreatitis. Gastroenterology 1986; 90:74-79.
7. Blamey, 1983. Blamey SL, et al: The early identification of patients with gallstone associated pancreatitis using clinical and biochemical factors only. Ann Surg 1983; 198:574-578.
8. Bradley, 1993. Bradley EL: A clinically based classification system for acute pancreatitis. Arch Surg 1993; 128:586-590.
9. Bradley and Allen, 1991. Bradley III EL, Allen KA: Management of necrotizing pancreatitis: results of prospective longitudinal trial. Am J Surg 1991; 161:19-25.
10. Bradley, 1983. Bradley EL, et al: Haemodynamic consequence of severe acute pancreatitis. Ann Surg 1983; 198:130-133.
11. British Society of Gastroenterology, 1998. British Society of Gastroenterology : United Kingdom guidelines for the management of acute pancreatitis. Gut 1998; 42(suppl 2):S1-S3.
12. Buchler, 1986. Buchler M, et al: Correlation of imaging procedure, biochemical parameters and clinical stage in acute pancreatitis. In: Malfertheiner P, Ditschuneit H, ed. Diagnostic Procedures in Pancreatic Disease, Berlin: Springer-Verlag; 1986:122-129.
13. Buter, 2002. Buter A, et al: Dynamic nature of early organ dysfunction determines outcome in acute pancreatitis. Br J Surg 2002; 87:298-302.
14. Corfield, 1985. Corfield AP, et al: Prediction of severity in acute pancreatitis: prospective compareson of three prognostic indices. Lancet 1985; 2:403-407. 1. Russo MW, Wei JT, Thiny MT, Gangarosa LM, Brown A, Ringel Y, et al. Digestive and liver diseases statistics, 2004. Gastroenterology. 2004;126:1448–53.
15. Triester SL, Kowdley KV. Prognostic factors in acute pancreatitis. J Clin Gastroenterol. 2002;34:167–76.
16 Dervenis C, Johnson CD, Bassi C, Bradley E, Imrie CW, McMahon MJ, et al. Diagnosis, objective assessment of severity, and management of acute pancreatitis. Santorini consensus conference. Int J Pancreatol. 1999;25:195–210.
17. Otte M. Chronic pancreatis and pancreatic carcinoma in the elderly. Schweiz Rundsch Med Prax. 2005;94:943–948. [PubMed]
18. Mechanisms of alcoholic pancreatitis. Proceedings of a conference. Chicago, Illinois, USA, November 2002. Pancreas 2003; 27:281.
19. Opie EL. The etiology of acute hemorrhagic pancreatitis. Bull Johns Hopkins Hosp 1901; 12:182.
20. Lerch MM, Saluja AK, Rünzi M, et al. Pancreatic duct obstruction triggers acute necrotizing pancreatitis in the opossum. Gastroenterology 1993; 104:853.
21. Toskes PP. Hyperlipidemic pancreatitis. Gastroenterol Clin North Am 1990; 19:783.
22. Block S, Maier W, Bittner R, et al. Identification of pancreas necrosis in severe acute pancreatitis: imaging procedures versus clinical staging. Gut 1986; 27:1035.
23. Morgan DE, Baron TH, Smith JK, et al. Pancreatic fluid collections prior to intervention: evaluation with MR imaging compared with CT and US. Radiology 1997; 203:773.