“PROSPECTIVE STUDY OF CLINICAL OUTCOME IN ALCOHOLIC ACUTE PANCREATITIS”

DISSERTATION SUBMITTED TO

THE TAMIL NADU DR.MGR MEDICAL UNIVERSITY, TAMILNADU

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE

DEGREE OF

MASTER OF SURGERY

IN

GENERAL SURGERY

DEPARTMENT OF GENERAL SURGERY

GOVERNMENT MOHAN KUMARAMANGALAM MEDICAL

COLLEGE HOSPITAL, SALEM

Year : 2017-2020

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ACKNOWLEDGEMENT

I am extremely thankful to **Prof.Dr.K.THIRUMALBABU, MD., DM**., Dean, Govt. Mohan Kumaramangalam Medical College and Hospital, Salem for allowing me to utilize the hospital facilities for doing this work.

I am also thankful to **Prof.Dr.P.V.DHANAPAL,M.S.,** Medical Superintendent,Govt.Mohan Kumaramangalam Medical College Hospital, Salem for his whole hearted support and encouragement for the completion of this dissertation.

I express my deep sense of gratitude and indebtedness to **Prof.Dr.C.RAJASEKARAN,M.S**., Head of the Department of General Surgery and **Prof.Dr.P.SUMATHI,M.S., DGO.,** Unit Chief, Guide for giving me inspiration, valuable guidance and his unstinting help in completing the

I thank all surgical unit chiefs **Prof.Dr.K.VIJAYAKUMAR,M.S., Prof.Dr.K.KESAVALINGAM,M.S., Prof.Dr.G.RAJASHOK,M.S., Prof.Dr.M.RAJASEKAR,.M.S.,** for their advice and kind help.

I also thank my registrar **Dr.M.ARULKUMARAN,M.S.,DA.,** who guided me to success this study.

It is my privileged duty to profusely thank my assistant professors **Dr.S.Sreedevi MS., Dr.P.Mohan MS., Dr.Ganganesamy MS,Dr.S.Prasad MS.,** who helped and guided me in many aspects of this study.

I take this opportunity to thank my senior PG's **Dr.P.DHAKSHINAMOORTHY M.S., Dr. B.SIVASUBRAMANIAN,M.S.,** who despite of my shortcoming were eager to teach me. I thank my colleague **Dr.N.NANTHAKUMAR.**

I thank my junior PG's **Dr.VIDHYABHARATHI, Dr. BHUKYA VIJAYAKUMAR Dr.GOPINATH, Dr.SUBASHCHANDRU** my other post graduate colleagues and my house surgeons who shared majority of my duties so that I could complete this study with ease.

I would like to Thank my husband **Dr. V.ANAND, MD.,DM.,** for helping me to complete my study.

I thank my daughter A.RAGHAVARSHINI for her cooperation throughout my dissertation work.

I cordially thank my parents, my family and friends who have always been there with me whenever I needed their help and cooperation.

I am deeply obliged to my patients, without whose help the present study would not have been possible.

DR. R.DEVIPRIYA

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Communication of Decision of the Institutional Ethics Committee(IEC)

Ref. No. GMKMC&H/4341/IEC/01/2017-10 **Date: .01.2018**

Protocol title	**"PROSPECTIVE STUDY OF CLINICAL OUTCOME IN ACUTE ALCOHOLIC PANCREATITIS"**
Guide/Principal Investigator	DR. P. SUMATHI, MS., Associate Professor of General Surgery, GMKMC, Salem-30.
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Name & Address of Institution	Govt. Mohan Kumaramangalam Medical College & Hospital, Salem, Tamil Nadu.
Type of Review	☑ New review ☐ Revised review ☐ Expedited review
Date of review (D/M/Y)	17.11.2017
Date of previous review, if revised application:	Nil
Decision of the IEC	☑ Recommended ☐ Recommended with suggestions ☐ Revision ☐ Rejected
Suggestions/ Reasons/ Remarks:	Nil
Recommended for a period of :	3 Years

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ABBREVIATIONS

AP	-	Acute Pancreatitis
ALT	-	Alanine Aminotransferase
AST	-	Aspartate Aminotransferase
APACHE Score	-	Acute Physiology and Chronic Health Evaluation
BISAP	-	Bedside Index for Severity in Acute Pancreatitis
CECT	-	Contrast Enhanced Computed Tomography
CRP	-	C - reactive protein
HAPS	-	Harmless Acute Pancreatitis Score
LDH	-	Lactate Dehydrogenase
LFT	-	Liver Function Tests
MAP	-	Mild Acute Pancreatitis
SAP	-	Severe Acute Pancreatitis
SIRS	-	Systemic Inflammatory Response Syndrome

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ABSTRACT

STUDY ON ACUTE ALCOHOLIC PANCREATITIS

Background

Acute Alcoholic pancreatitis is a common disease with wide clinical variation. It may vary in severity, from mild self-limiting to pancreatic necrosis with life-threatening sequelae. Severity of acute Alcoholic pancreatitis is linked to the presence of systemic organ dysfunctions and/or necrotizing pancreatitis.

Aim and objectives

The present study was aimed to assess the clinical profile and to assess the efficacy of various severity indices in predicting the outcome of patients.

Methodology:

This was a prospective study done in Salem Medical College and Hospital . All patients with a diagnosis of acute Alcoholic pancreatitis were included in this study. Along with routine lab parameters, serum amylase, lipase, lipid profile, calcium, CRP, LDH, CT abdomen, CXR and 2D Echo were done.

Results

Out of 142 patients alcohol induced pancreatitis was higher (51%) than gall Stone induced pancreatitis. This can be explained by the greater incidence of alcohol abuse in Tamilnadu. Incidence of alcoholic pancreatitis is mostly seen in young males, particularly of middle age group. All the patients had significant alcohol history. Out of them 85.7% were associated with smoking

history. In this study alcohol which is mostly abused by men than women and younger age group than old prevalence is more in young males. Most of the patients had no comorbidities (73.8%) , ICU stay seen in 46.4 % **, < 2week .**

(77.4%) patients of the study population had mild pancreatitis, while> 2 weeks(22.6%) patients had Severe acute pancreatitis. .Duration of discharge is directly proportional to the severity of pancreatitis. (70.2.%) patients of the study population had mild pancreatitis, while (29.7%) patients had SAP as determined by CT, which is taken as standard to predict the severity of pancreatitis for the most common symptom of abdominal pain or it could be that of a referral bias.

Three pancreatic scores were taken in the study HAPS, BISAP ,GLASCOW and SIRS, all of which have easily obtainable variables and can be calculated at the time of admission.

Conclusion:

Initial assessment of , LDH , HAPS Score ,SIRS and Glascow score could be reliable indicators of outcome in acute pancreatitis

Keywords: Acute pancreatitis, C-Reactive Protein, LDH, Severity index,

Prolonged hazardous drinking can result in progressive and irreversible damage to the pancreas gland. This occurs on the background of pancreatic inflammation, acinar atrophy and, ultimately, fibrosis and can result in significant exocrine and endocrine insufficiency. Some individuals may develop this condition with alcohol intakes as low as 20 g/day; others may need

to drink in excess of 200 g/day before evidence of the disease develops; others may never develop this condition no matter how much they drink or for how long. In susceptible individuals the longer the duration of drinking the greater the risk of developing significant pathology.

Acute alcohol-related pancreatitis may present as an acute episode of abdominal pain, nausea and vomiting and in severe cases can be accompanied by profound metabolic abnormalities and circulatory collapse. These acute episodes may recur, often precipitated by an increase in alcohol intake. Complications such as narrowing of the common bile duct, localized leakage of pancreatic fluid and pancreatic exocrine and endocrine insufficiency may develop resulting in jaundice, pseudocyst formation, malabsorption and diabetes. In some individuals, however, the clinical course is insidious with progression to pancreatic insufficiency without acute inflammatory episodes.

INTRODUCTION

Acute pancreatitis (AP) is a major disastrous condition of GIT, with increased incidence at present.[1-2]

It also shows unpredictable outcome. Patient may improve with supportive care as in two thirds or may show serious local and systemic complications due to an intense inflammatory response, such as multi organ failure or necrosis .[3-7]

These patients should be triaged as severe pancreatitis group requiring intensive resuscitation (SAP). Initial intensive fluid resuscitation within first 24-48 hours management may alter the course of SAP. If there is a > 24 hour delay in treating with fluids mortality rate doubles[8]. Outcome in AP depends on pancreatic necrosis. Pancreatic necrosis patients show morbidity of 80% and a mortality ranging from 6 to 40%.

Scores and Variables

Atlanta Classification is most widely used, which is based on clinical manifestations. Ranson and APACHE II scores, as well as the presence/ absence of organ failure and intrapancreatic pathology[9] may also be useful.

Its drawbacks were corrected and SIRS was added by The Acute Pancreatitis Classification Working Group in 2012.[10-12]

To differentiate MAP and SAP Organ Failure Assessment (SOFA), Logistic Organ Dysfunction (LOD) or the Multiple Organ Dysfunction (MODS) scores or pancreatitis specific severity scores such as the Ranson criteria[13] were used.

Acute alcohol-related pancreatitis is characterized by acute episode of abdominal pain, nausea and vomiting. In SAP there is metabolic abnormalities and circulatory collapse.

Aims of the study

1. To assess the clinical profile of acute Alcoholic pancreatitis
2. To assess the efficacy of various severity indices in predicting the outcome of patients.
3. To determine the role of Bedside Index for Severity in Acute Pancreatitis (BISAP), Harmless Acute Pancreatitis Score (HAPS) and Systemic Inflammatory Response Syndrome (SIRS) scores in predicting acute Alcoholic pancreatitis.
4. To determine if a correlation exists between CTSI and BISAP, HAPS and SIRS scores in predicting acute Alcoholic pancreatitis.

Review of Literature

History

Discovery of the Pancreas- The Anatomical Perspective

Pancreas is being defined through centuries. A Greek anatomist Herophilus gave first report of pancreas (335–280 BC). **After** hundred years Ruphos, another Greek anatomist, named "pancreas"(1st or 2nd Century AD) meant "all flesh"[14]

A roman physician Claudius Galenus(138-201 AD), described that the pancreas was a cushion to protect the large blood vessels. After 15 centuries in 1543 Vesalius predicted anatomical elucidation of the pancreas. This disproved Galen dogma

The Duct of Wirsüng was described in 1642 by Johann Georg Wirsüng. He predicted that it was a excretory duct of the pancreas and it drains into the duodenum .In 1720 Vater described papilla duodeni major. Second excretory pancreatic duct was described by Santorini in 1724, which was regarded as a normal finding.[15]

Discovery of the Pancreas-Pancreatic Secretion

Sylvius proposed the role of the pancreatic juice in1659. Willy Kuhne (1837-1900) discovered trypsin. Lipase and pepsin was discovered by Alexander Marcet and Theodor Schwann respectively in 1815.

The effect of pancreatic juice on digestion was given by Claude Bernard (1813-1878). Paul Langerhans described in 1869 the islet of Langerhans[16]

Acute Pancreatits(AP)

Initially acute necrosis of the pancreas were described by Aubert (1578-1579) followed by acute pancreatitis by Nicholas Tulp in 1652 . H e identified patients with fatty stools and predicted it to be of a pancreatic etiology .Following an autopsy the first pancreatic pseudocyst was described by Morgagni (1761) [17]

Historical Events in Pancreatitis[18-19]

Year	Event
1842	Anatomy and clinical features of AP- Classen
1856	Pathogenesis- altered pancreatic drainage- AnceletEdouard
1861	Necrosis of the pancreas in vivo- Oppolzer
1865	Etiology -Hemorrhagic and suppurative - Rokitansky
1878	Role Of alcohol– Friedreich
1882	Association with gall stones- Prince

1882	Adipose tissue necrosis in AP caused by pancreatic lipase- Porlich,
1889	Description of hemorrhagic and suppurative pancreatitis and fat necrosis - Reginald H. Fitz
1896	Necrotizing pancreatitis – pathogenesis as autodigestion – Chiari
1901	Obstruction at the ampulla of vater can cause acute pancreatitis - Opie
1927	Role of Serum amylase in acute pancreatitis – Elman

Epidemiology

The incidence of acute pancreatitis is on the raising trend. It may be due to better diagnostic modalities.

In 1961-1967 Trapnell and Duncan [20] from Bristol reported incidence of 5.4 / 10^5population /year. Jakkola from Finland reported incidence of 73.4 / 10^5population /year in 1989.[21]

The incidence may vary from 5 to 80 per 100,000 population worldwide. Highest incidence are reported from the United states and Finland[22]. Japan reported incidence of acute pancreatitis to be 12.1/100 000 [23].

India have not reported pancreatic epidemiological data .According to data from All India Institute of Medical Sciences (AIIMS), New Delhi, 276 patients with AP were hospitalized from January 1997 to June 2002, i.e. about 55 patients per year[24]. This data is similar to that of England. US Census Bureau reported incidence of AP to be 313,256 considering the population of the country as 1, 065, 070, 607.[25]

25% of patients may present with severe acute pancreatitis (SAP).This severity depends on the presence of systemic organ dysfunction and/or pancreatic necrosis [26-27]. Incidence of necrotising pancreatitis is 10–15% with acute edematous pancreatitis showing mortality of 27% to 86% [28].

Age-related demographics[29]

The average age of onset of acute alcoholic pancreatititis is 39 years. Biliary disease is 69 years. Pancreatitis following trauma is seen in sixth decade. Drug-induced pancreatitis mostly occur in fourth decade. In third decade it may be due to ERCP, vasculitis and AIDS . As age increases hopitalisation due to pancreatitis increases.

Sex-related demographics

Males are more commonly affected than females. This may be due to the effect of steroid hormones. The most common etiology in males

being alcohol and females being biliary disease . Idiopathic pancreatitis may occur in both sexes.[30]

Definition & Diagnosis of acute pancreatitis[31-35]

Two among the three criteria is required for the diagnosis of AP:

(1) onset of severe epigastric pain radiating to back.

(2) More than 3 times the rise of Serum amylase or lipase .

(3) Radiological confirmation by contrast-enhanced computed tomography (CECT) / magnetic resonance imaging (MRI) or abdominal ultrasonography .

Physiology

The main biological function of exocrine pancreas is synthesis and secretion of digestive enzymes. Enterokinase causes proteolytic activation in small intestine. Trypsin gets activated from trypsinogen, which in turn activates all other enzymes. Usually they are in inactive state even after secretion in the pancreatic duct. The enzymes are in intracellular area which prevents their activation. Trypsin is activated in the acinar cell.

Pathophysiology

Pancreatitis occur by premature activation of zymogenases predicted by Chiari[37]. Intrapancreatic activation can also result in acute pancreatitis.[37]

The pathophysiology can be divided into Acinar Cell Events, Pancreatic and Peripancreatic Events, Cell Death and Systemic Events

Acinar Cell Events

Zymogen Activation And Inhibition Of Secretion

Acute pancreatitis occurs due to activation of trypsin from trypsinogen. Subsequently it leads to activation of vascular endothelium, interstitium, and acinar cells [38-40]. Acinar cell insult causes cytosolic calcium elevation.

The probable causes of Trypsin activation

1. Localization of the enzymes and hydrolases
2. Bile reflux causes reflux of duodenal contents into pancreatic ducts,exposing ductal contents into pancreatic parenchyma.[41]

3.Inactivation of pancreatic secretory trypsin inhibitor[37]

Mutations in cationic trypsinogen causes enhancement of its activation or prolonged activation of trypsin which in turn causes hereditary pancreatitis[42].

Cytokine And Chemokine Generation

Activation of c5a causes recruitment of polymorphonuclear leukocytes and macrophages which then releases proinflammatory cytokines [43](IL-1, IL-6, IL-8, TNF and platelet-activating factor). It is counteracted by anti inflammatory cytokines IL-2, IL-10, IL-11.[44, 45]

Pancreatic and Peripancreatic Events

Edema

Expression of endothelial adhesion molecules occurs following injury to acini which triggers inflammatory response causing microcirculatory changes . It increases vascular permeability causing edema of the gland

Changes in Paracellular Cell Permeability

When loss of tight junctions occur in the acinar and duct cells, pancreatic duct leak into the interstitial space .It causes increase in serum levels of pancreatic enzymes and decrease in pancreatic secretion [46]

Vascular Changes

Pancreatic blood flow decreases following theses events which is then aggravated by decreasing intravascular volume .compression of the vascular structures leads to local microcirculatory failure. In acute pancreatitis further vascular damage occurs causing thrombosis and hemorrhage which in turn leads to pancreatic necrosis. [47]

Pseudocyst occurs when ischemia of the pancreas causes disruption of the pancreatic excretory ducts.[48, 49]

Pancreatic infection

Bacterial translocation from the colon or hematogenous spread causes infection of the cyst. The immunologic and morphologic factors if defective in healthy individuals causes infection[50]. Hypovolemia and pancreatitis-induced arteriovenous shunting occurs further aggravating bacterial translocation.[51]

Systemic Events

SIRS occurs due to release of cytokines and activated pancreatic enzymes as seen in SAP into the portal circulation.[52] These induces hepatic secretion of cytokines into the systemic circulation by the kupfer cells. These in turn releases IL-1, IL-6, IL-8, TNF,CRP. All these events leads to SIRS and in turn to MODS.[53]

SIRS is characterized by fever, pleural effusion (s), acute respiratory distress syndrome (ARDS), myocardial depression, acute kidney injury, shock and metabolic complications.

Pathogenesis of ARDS- active phospholipase A digests lecithin. It causes loss of surfactant causing ARDS. Myocardial depressant factor, vasoactive peptides and hypovolemia causes myocardial depression. Hypovolemia and hypotension predisposes to acute kidney injury.

Hypo or hyperglycemia, hyperlipidemia or a decrease in the serum calcium are seen in pancreatitis. Hypocalcemia occurs due to calcium-soap formation theory in older concept. According to newer concepts free fatty acid–albumin complexes bind with calcium. This leads to translocation of calcium to the intracellular compartment leading to hypocalcemia.[54]

Phases of acute pancreatitis[55]

The initial phase occurs for 5 to 7 days

Early phase

Local pancreatic injury causing systemic changes occurs up to 7 days. This is due to the release of proinflammatory cytokines. It in turn leads to SIRS. Persistent SIRS leads to MODS. Organ failure is defined by the Modified Marshall scoring system for organ dysfunction (Table1)

Table 1: Modified Marshall scoring system for organ dysfunction

	Score				
Organ system	**0**	**1**	**2**	**3**	**4**
Respiratory (PaO_2/FiO_2)	>400	301–400	201–300	101–200	≤101
Renal*					
(serum creatinine, μmol/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, mm Hg)†	>90	<90, fluid responsive	<90, not fluid responsive	<90, pH<7.3	<90, pH<7.2
For non-ventilated patients, the FiO_2 can be estimated from below:					
Supplemental oxygen (l/min)	**FiO_2 (%)**				
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 μmol/l or ≥1.4 mg/dl.
†Off inotropic support.

Transient organ failure resolves within 48 hours. If the duration exceeding more than 48 hours it is called as persistent organ failure.[31]

Late phase

Systemic signs of inflammation, organ failure and local complications occurs in late phase of pancreatitis.

Complications

Local

Local complications may be pancreatic or extra pancreatic complications. Pancreatic complications are acute peripancreatic fluid collection, pancreatic pseudocyst, acute necrotic collection and walled-off necrosis. Extrapancreatic complications include gastric outlet dysfunction, splenic and portal vein thrombosis, and colonic necrosis.

Recurrent pain, increases in serum pancreatic enzyme activity, organ dysfunction, with clinical signs of sepsis characterizes local complication.

Systemic complications

Worsening of a pre-existing co-morbidity by the inflammation leads to systemic complication.

ETIOLOGY AND CLASSIFICATION

Based on pathology, etiology, severity of disease, or the presence of necrosis AP can be classified.

Commonest - gallstones (40 –70 %), alcohol (25– 35%). Idiopathic 10–20% of patients.

Specific Etiologies

Gallstone pancreatitis and Microlithiasis

Incidence of AP is 0.17% / year. Biliary pancreatitis occurs in 2%. Long course of cystic duct and CBD, Small gallstones < 5 mm predisposes. AP due to gallstone presents with a transient elevation of liver enzymes especially alanine aminotransferase >150 IU/ml.

Alcohol :

An important public health problem is alcohol intake. Highest average volume of drinking is seen in Western Europe, eastern part of Europe and in North America. Lowest in the eastern Mediterranean region and parts of southeast Asia, including India [56]. A recent study predicts that if these practices are not intervened still people's health will be deteriorating.[57].

In Tamilnadu especially in males also the alcohol intake is more[58].

Calculating units

1 unit = 10ml or 8g alcohol.

standard measure is alcohol by volume (ABV).

ABV is a measure of the amount of pure alcohol as a percentage of the total volume of liquid in a drink.

- strength (ABV) x volume (ml) ÷ 1,000 = units

Drinks and units

A 750ml bottle of red, white or rosé wine (ABV 13.5%) contains 10 units.

Type of drink	Number of alcohol units
spirits * (25ml, ABV 40%)	1 unit
Alcopop (275ml, ABV 5.5%)	1.5 units
white/red/ rosé wine (125ml, ABV 12%)	1.5 units
Beer (330ml, ABV 5%)	1.7 units
Can of lager/beer/cider (440ml, ABV 5.5%)	2 units
Pint of lager/beer/cider (ABV 3.6%)	2 units
Standard glass of red/white/rosé wine (175ml, ABV 12%)	2.1 units
Pint of higher-strength lager/beer/cider (ABV 5.2%)	3 units
Large glass of red/white/rosé wine (250ml, ABV 12%)	3 units

*Gin, rum, vodka, whisky, tequila, sambuca. Large (35ml) single measures of spirits are 1.4 units.

ALCOHOL METABOLISM

An oxidative and a non-oxidative pathway occurs in the liver in alcohol metabolism. Haber et al[59] published mechanism of oxidative metabolism. This study correlated with Gukovskaya et al[60], which was done with isolated pancreatic acini. Ethanol is converted to acetaldehyde by alcohol dehydrogenase. Cytochrome P-450 has a role in metabolism of 20% of ethanol [61,62].The presence of cytochrome P-450 CYP2E1 has been demonstrated in rat pancreas[63] as well as human pancreas[64]. The expression of CYP2E1 in rat pancreas[63]occurs in chronic intake of alcohol [65].

Synthesis of FAEE (fatty acid ethyl esters) using FAEE synthase is the non-oxidative pathway[66] of metabolism. Gukovskaya et al[60] predicted FAEE synthase activity in pancreas. The correlation of oxidative and non oxidative pathways of ethanol is given by Werner et al[67,68]. Following inhibition of oxidative metabolism shift to non oxidative metabolism occurs resulting in an increase of FAEE. Carboxyl ester lipase (CEL) catalyze FAEE synthesis from fatty acids and ethanol. Alcohol induced pancreatitits[69] is associated with CEL gene polymorphism. But this fact requires further discussion.

ROLE OF ETHANOL METABOLISM IN PANCREATIC INJURY

According to ethnical workup alcohol causes Sphincter of Oddi spasm. Ductal-Plug hypothesis by Sarles and his colleagues[70] was also considered. Pluggig of proteins in small ductules occurs due to alcohol causing pancreatic injury. The mechanism of alcohol induced effect in animal model was given by Saluja and Bhagat[71. Transient increase of pancreatic amylase output and plasma cholecystokinin (CCK) levels occurs due to ethanol, mediated by CCK releasing factors. Inhibition of apoptosis and the downstream apoptosis executor caspase-3 occurs in animals when compared with the controls[72]. Endotoxin causes pancreatic necrosis .The results from this study showed that the pancreas exposed to alcohol is more sensitive to necrotic cell death.

When there is appropriate trigerring factor acinar cells metabolises alcohol and causes gland injury. Role of stellate cells with involvement of acinar cells in causing pancreatic fibrosis [73] also documented. Acetaldehyde interfere with the binding of secretagogue to their receptors[74]. This in turn stimulates secretion from isolated pancreatic acini[74]. All these events leads to microtubule dysfunction. All these in turn affects exocytosis from acinar cells[75].

Hydrogen ions and reducing equivalents are released in alcohol induced damage[76];imbalance between free radicals and antioxidant

defense mechanism occurs due to release of NADH. Loss of mitochondrial glutathione and inactivation of GPX occurs, with inactivation of respiratory complexes[77]. Upregulation of CYP2E1and catalase[78] occurs in chronic conditions. They compete the mitochondrial electron transport system causing localized and transient hypoxia in tissues. All these events eventually forms ROS .

Products of non – oxidative ethanol metabolism FAEEs causes pancreatic injury *in vivo*[79] and *in vitro*[80] . Uncoupling of mitochondrial and oxidative phosphorylation[81] occurs due to hydrolysis of FFA. Direct binding to the intracellular membrane occurs causing permeability of cell membrane[82].Increase in lysosomal fragility releasing hydrolase's causes production of cholestryl esters. They act on the zymogen granule membrane releasing trypsin[83]

Impairment of blood flow to pancreatic acinarcells ,alters hemodynamic parameters .McCord[84] explained reoxygenation induced injury following hypoxia.

EFFECT OF ALCOHOL ON CELL SIGNALING PATHWAY

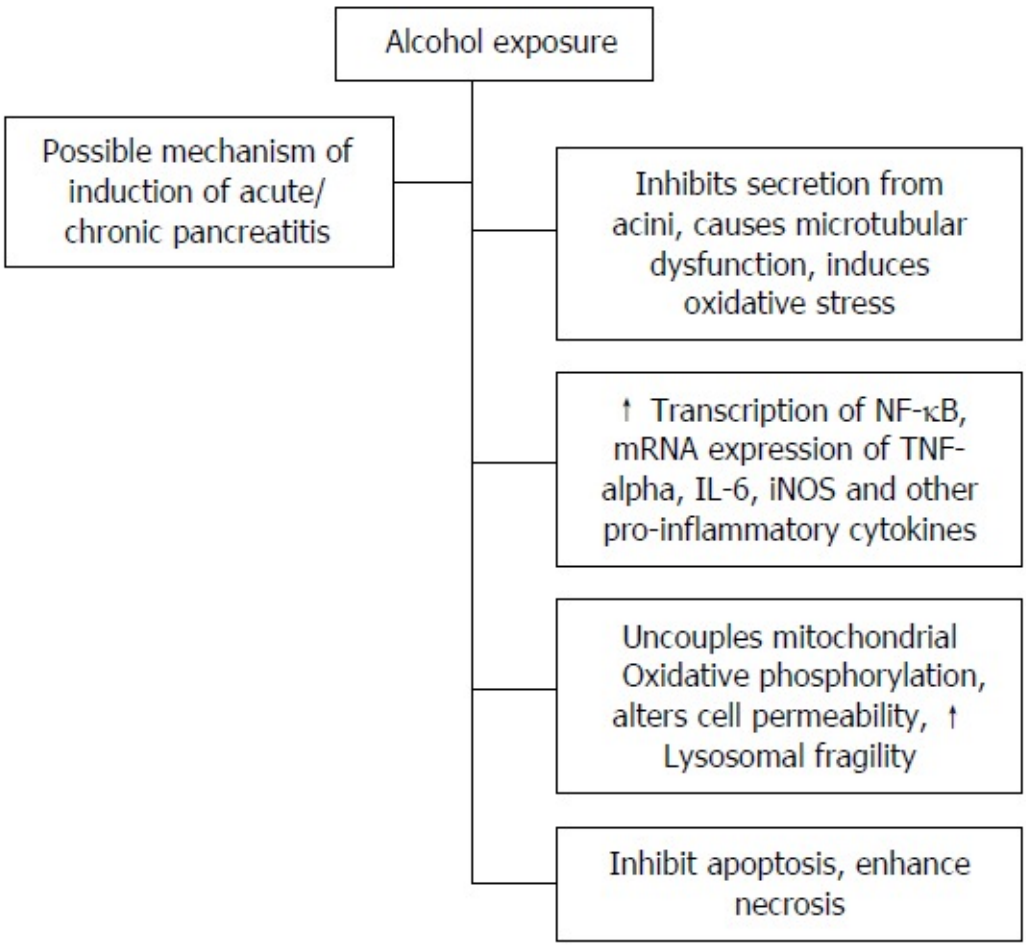
In a study as animals fed on alcohol do not develop pancreatitis explains that there are factors other than alcohol to produce pancreatitis. Alcohol is found to sensitize pancreas, thereby injuring pancreas[85].Ethanol diet was given to animals intragastrically and CCK -

8 infusion according to Pandolet al[86]. It releases NF-κB, AP-1 and other cytokine and inflammatory molecules. It results in increased trypsin release.

CIGARETTE SMOKING AND PANCREATITIS:

Alcoholic chronic pancreatitis is predisposed by Cigarette smoking. About 80%-95% of alcoholics smoke, 25%-30% of smokers donot drink alcohol[87]. The occurrence of acute pancreatitis is more common in smokers than nonsmokers (about 10%). Intermittent nicotine administration in rats enhanced ethanol uptake according to Blomqvist et al[88]. Mesolimbic dopamine neurons responsiveness to both nicotine and alcohol in increased with subchronic nicotine doses.

Events to alcohol exposure lead to cause alcoholic pancreatitis



Recurrent pancreatitis,[89,90] and alcoholic pancreatitis is caused by alcohol with an incidence of 5%. Three theories proposed are

1.Toxic theory

2.Stone theory

3. Necrosis fibrosis theory.

Hyperlipidemia

When the triglyceride exceeds 1000 mg/dl, acute pancreatitis develops. Toxic free fatty acids are produced from triglycerides causing damage to the small pancreatic blood vessels. This in turn cause injury to the endothelial cells. The inflammatory cells are recruited causing thrombosis, and ischemia .

Hereditary pancreatitis

Mutations in the cationic trypsinogen gene (PRSS1), pancreatic secretory trypsin inhibitor gene (serineprotease inhibitor Kazal type 1 or SPINK-1) and cystic fibrosis transmembrane conductance regulator gene are involved in the pathogenesis of pancreatits.

Post-ERCP pancreatitis

Its incidence is about 5%. younger age group, normal pancreatic duct, specialist, multiple injections of the pancreatic duct with acinarization, pancreatic sphincterotomy, SOD, and biliary or pancreatic manometry may predispose to post ERCP pancreatitis.

Other causes of AP

Hypercalcemia and hyperparathyroidism, drugs such as 6-mercaptopurine, azathioprine, and 2', 3'-dideoxyinosine are predisposing factors but data is not conclusive. CMV, Ascariasis and some infections

may cause AP. Recent abdominal trauma and autoimmune pancreatitis are other causes.

Structural Causes

Failure of fusion of the ventral and dorsal and pancreatic ducts causes pancreatic divisum. The incidence of pancreatic divisum is 5–10% .clear evidence in causing pancreatitis is not available. In case of mass obstructing the pancreatic duct idiopathic AP may occur (5-14%).

Idiopathic pancreatitis

No etiology is available. May be due to microlithiasis, congenital abnormalities, pancreatic and genetic causes.

Definitions of severity in Acute Pancreatitis

Mild acute pancreatitis[91]

No organ failure or complications (local or systemic). These patients are discharged within few days .

Moderately severe acute pancreatitis[92]

It presents with transient organ failure or local and systemic complications. Prognosis varies. Some may require extended hospital stay with or without interventional procedures (like those with sterile necrosis), while others resolve spontaneously.

Severe acute pancreatitis[93,94]

Organ failure occurs in severe acute pancreatitis. These patients have a mortality ranging from 36-50 %.With infected necrosis mortality increases.

Definition of Types Of Acute Pancreatitis[55]

Interstitial oedematous pancreatitis

It is characterized by inflammation of the pancreatic parenchyma and the peripancreatic tissues. They donot cause tissue necrosis.

CECT criteria: These patients donot show peripancreatic necrosis. They show only enhancement of parenchyma.

Necrotising pancreatitis

Patients present with pancreatic parenchymal necrosis and/or peripancreatic necrosis along with inflammation.

CECT criteria: These patients have non enhancement of the pancreatic parenchyma after an intravenous contrast agent

Acute peripancreatic fluid collection

Peripancreatic fluid collection within the first month of interstitial oedematous pancreatitis is called acute peripancreatic fluid collection. They donot show features of a pseudocyst.

CECT criteria :These patients will have a homogeneous collection with fluid density in peripancreatic fascial planes. There is no wall encapsulating the collection. There is no intrapancreatic extension.

Pancreatic pseudocyst

They present with interstitial oedematous pancreatitis after 4 weeks. They have encapsulated fluid collection. They also present with well-defined inflammatory wall with or without necrosis.

CECT criteria: These patients present with well circumscribed lesion showing homogeneous fluid density. They have well-defined wall. Onset of pseudocyst is after 4 weeks

Acute necrotic collection

Apart from collections necrosis occurs in pancreatic parenchyma and/or peripancreatic tissue.

CECT criteria: These patients have features of acute necrotizing pancreatitis. No definable wall encapsulating the collection. Location could be intrapancreatic and/or extrapancreatic.

Walled-off necrosis

After 4 weeks of onset of necrotizing pancreatitis. It is characterized by encapsulated collection of necrotic tissue, with a well-defined inflammatory wall.

CECT criteria: These patients present with heterogeneous collection of liquid and non-liquid density material .Loculations with well-defined wall occur.

Infected pancreatic necrosis

Characterized by the presence of extraluminal gas. On CECT or in fine-needle aspiration patients have bacteria and/or fungi .

CLINICAL PRESENTATION[47]

Pancreatic type of pain, signs are of importance in diagnosing pancreatitis.

History

Patient tells typical epigasric and right hypochondrial pain radiating to back. It may be rapid onset or reaches a peak in 10 to 20 minutes. When exudates track to left colon pain occurs in the lower abdomen. Mostly associated with nausea and vomiting. This occurs due

to inflammation of the posterior gastric wall. In SAP Oliguria, breathlessness, GI bleed, fever occur.

Physical Examination

Patients with MAP may or may not show clinical features. This may vary from mild abdominal tenderness to guarding, abdominal distension. Bowel sounds are reduced or absent. In hemorrhagic pancreatitis ecchymosis around the periumbilical area (Cullen's sign) or flanks (Grey Turner's sign) occur.

Due to the release of inflammatory mediators from the inflamed pancreas third-space fluid losses occur causing hypotension and fever. This occurs after 3 days.

Extrapancreic manifestations like dyspnea, tachypnea, pleural effusion, atelectasis, ARDS, or congestive heart failure occur in SAP. Due to electrolyte imbalance, hypoxemia, fever, hypotension, or due to the toxins CNS manifestations of hallucinations, disorientation or coma may occur. Icterus in AP could indicate bile duct obstruction in course of edema of head of the pancreas or common bile duct stones with coexistent liver disease. Subcutaneous fat necrosis, thrombophlebitis, and polyarthritis are rare manifestations of the disease.

LABORATORY DIAGNOSIS

Pancreatic Enzymes[47,95,9,97]

Elevation of Amylase, lipase, Elastase, Phospholipase A2 and CarboxypeptidaseB .

Serum Amylase

There are many causes for elevation of amylase levels. Pancreatic pathology contributes to 40- 45 %. These values begin to rise in 6-12 hours. They remain in circulation for a duration of about 5 days.

Sensitivity of amylase in predicting pancreatitis is 85 %.serum amylase is also raised in hypertriglyceridemia. Hyperamylasemia is seen in salivary gland or fallopian tube, ruptured viscus. It is also seen in Renal failure. If the pathogenesis involves biliary system, marked elevations >2000 IU/L occur. This indicates that amylase is a supporting investigation.

Lipase

Sensitivity of 85% - 100% is seen with serum lipase. Lipasestarts rising from day one of illness. It remains raised during the entire pathology. The ratio of lipase to amylase is more than in biliary diseases.

Standard Blood Tests

Raised white blood cell count, Hyperglycemia levels, raised mean corpuscular volume (MCV) are characteristic.

Gallstone etiology presents with elevated Alanine aminotransferase. It is the most sensitive liver enzyme to diagnose acute biliary obstruction in AP. Hypertriglyceridemia and hypocalcemia are also noted in some patients.

Diagnostic Imaging

Abdominal Radiography[47]

Patients may present with normal x ray or localized ileus of a segment of small intestine. It is called sentinel loop which is seen in MAP. In severe disease *colon cut-off sign* is seen. Retroperitoneal gas is seen in pancreatic abscess.

Chest Radiography[47]

X ray show pleural effusion, atelectasis which are usually basal or an elevation of a hemidiaphragm. Left sided pleural effusions rather than bilateral presentations are seen.

***Sonography*[47, 96]**

The visualization of pancreas is usually obscured by the presence intestinal gas or adipose tissue. Gland may be enlarged or may be hypoechoic. Rather than collections it reveals presence of gall stones.

***Endosonography*[47, 96]**

Endoscopic ultrasound (EUS) is useful in gall stones and CBD stones. It can diagnose presence pseudocysts after 4 weeks and congenital abnormalities of the gland or for therapeutic intervention.

Computerized Tomography

Apart from diagnosis it also diagnose stages of acute pancreatitis [97].Contrast show 90 % sensitivity and specificity. But CECT is not used routinely as most patients have a mild pancreatitis. It is done after 48 – 72 hours of treatment. It is used to determine the onset of complications.[99] It shows 85 % sensitivity in detecting bile-duct stones .

CECT can detect

1.Enlarged pancreas with lobular effacement

2.Inhomogenous pancreatic parenchyma

3.Peripancreatic fat stranding

4. Fluid collection[96].

Pancreas would have a uniform perfusion; pancreatic necrosis show perfusion defects usually after 48 to 72 hours [47].

Based on the severity it can be of into five grades (A to E) with fluid collection, and when pancreatic necrosis is added to it , it is called the CT severity index(CTSI) as suggested by Balthazar[100].

When CTSI is more than 7 it shows higher mortality[101].

***Magnetic Resonance Imaging* (MRI)**

MRI is comparable to CECT in the diagnosis[98]. Diffuse or focal enlargement of the pancreatic gland with blurred pancreatic margins is seen in T1 weighted images .In necrosis there is no contrast enhancement.

MRCP can detect

1. Duct disruption
2. Duct anomalies
3. Choledocholithiasis.
4. Secretin MRCP - idiopathic pancreatitis and recurrent pancreatitis

Non-contrast MRI

1. Pancreatic necrosis
2. Solid debris in a pancreatic fluid collection

PROGNOSTICATORS OF SEVERITY

General or Pancreatic specific scores, imaging scores, or individual markers of severity are predictors.

General Severity Scores

1.Acute Physiology and Chronic Health Evaluation Score: APACHE II[13, 47]

APACHE II consists of 12 parameters.

Score of 8 and above is considered as SAP. It can be used on admission to determine severity . Reassessment of severity and disease progression is done again. It is best predictor of mild disease. But it has complexity, low sensitivity and not a better predictor after 48 hours

2. Organ Failure Based Scoring Systems[13]

The sequential Organ Failure Assessment (SOFA), Logistic Organ Dysfunction (LOD) and Multiple Organ Dysfunction (MODS) scores are scores that evaluate organ dysfunction and correspond it to mortality. They take into account the number of systems involved and the degree of severity with 6 parameters.

MODS uses 5 parameters. MODS have equal predictability to APACHE II.

They show equal efficacy in predicting mortality in patients with SAP.

3. Organ Failure [47]

Mortality of around 36% is seen in organ failure. The Modified Marshall Scoring System for organ failure is used to define organ failure.

Pancreatitis Specific Scores

1. Ranson Criteria[102]

Ranson criteria :

11 variables

5 on admission

6 >48 hrs.

Score of ≥ 3 is SAP. Sensitivity is 67-84%, specificity is 76-90%. Disadvantages are that assessment can only be done after 48 hrs. some investigations like lactate dehydrogenase, base excess, and fluid sequestration are not easily available

Its advantage is that it excludes severe pancreatitis.[103]

Its variant is modified glascow score with same disadvantages.

2. The Pancreatic Outcome Prediction Score[104]

Used in ICU setting .Has 8 variables in the first 24 hrs of admission (0-40 score range).

3.*Bedside Index for Severity in Acute Pancreatitis (BISAP)*[105]

Disadvantages of Ranson's and APACHE are overcome by the BISAP score .It was developed by Singh et al. A series of 17,922 cases of AP from 2000 to 2001 were studied. It was further validated in 18,256 cases from 2004 to 2005.

BISAP uses five variables to determine mortality:

1. Blood urea nitrogen > 25mg/dL,

2. Impaired mental status

3. Presence of SIRS

4. Age > 60 years

5. Pleural effusion.

Score of each is 1, value of ≥ 3 - organ failure and mortality. In predicting mortality in the first 24 hours APACHE II and BISAP are the same.

Score of 0 – mortality <1 %

Score of 5 – 22 %

score greater than 3- 7-12 fold increase in developing organ failure.

Advantage :

1.Accurate

2.Easier to use

4. Harmless Acute Pancreatitis Score[106]

More recently, HAPS is being used for a mild self-limiting type of disease. HAPS is one of the simplest scores to predict severity of AP.

It includes:

1.Absence of rebound tenderness or guarding

2. Normal hematocrit

3. Normal serum creatinine score.

3 present –Harmless course of disease(98% accuracy)

Advantage : Easy to determine.

5.Systemic Inflammatory Response Syndrome (SIRS)

It depends on vital signs and leukocyte count.

The presence of any of the following two is defined as SIRS

- Heart rate >90 beats/min
- Core temperature <36°C or >38°C

▸ WBC count <4000 or >12000/mm^3▸ Respiration >20/min or PCO2 <32 mm Hg.

SIRS in the first 24 hours of admission reveals organ failure (85%) and death (100%). It lacks specificity for predicting severe disease (41%). Persistent inflammation of longer than 48 hours is linked to organ dysfunction and death.

In early phase of pancreatitis a clinical response to the pro-inflammatory mediators occurs resulting in SIRS. Pathogenesis involves decreased vascular tone, a decrease in systemic vascular resistance and increased capillary permeability . It results in third space volume loss leading on to hypotension and a hyperdynamic circulation. If uninterrupted leads to disasterous effects. According to Mofidi et al mortality of 0,8 and 25 percent with no, not persistent and persistent SIRS is seen.

Advantages

1.Inexpensive

2.Readily available

3.compares favorably with other more complicated scores.

IMAGING SCORES

1. Computed Tomography Severity Index[100](CTSI)

Detects development of complications and mortality.CTSI is graded on a 10 point scale . 2 radiologic criteria are pancreatic inflammation and of fluid collections .

4 points graded A-E

Pancreatic necrosis with 6 points

Balthazar Grades

Grade A: Normal pancreas consistent with mild pancreatitis

Grade B: Focal or diffuse enlargement of the gland, including contour irregularities and inhomogeneous attenuation but without peripancreatic inflammation

Grade C: Grade B plus peripancreatic inflammation

Grade D: Grade C plus associated single fluid collection

Grade E: Grade C plus two or more peripancreatic fluid collections or gas in the pancreas or retroperitoneum

Balthazar grade score: A = 0, B = 1, C = 2, D = 3, E = 4

Balthazar Necrosis score

Absence = 0, up to 33% = 2, from 33% to 50% = 4, Necrosis of >50% = 6

CTSI = Balthazar Grading plus Necrosis Score: Highest attainable score is 10.

A score of 7-10 - 92% morbidity and 17% mortality

CTSI :

48 hrs, 72 hrs and 1week after hospital admission.

Modified CTSI score -CTSI with the addition of extrapancreatic complications.

Combination of Ranson score and CTSI is very useful for the diagnosis of severe AP [49]

2. The Extrapancreatic Inflammation on Computed Tomography Score[107](EPIC)

It may be associated with pleural effusions, ascites, retroperitoneal inflammation, and mesenteric ischemia.

In the first 24 hrs of admission, pancreatic necrosis could not be diagnosed hence may be associated with all these factors. It is efficacious in predicting mortality when the score ≥ 4.

Single Markers of Severity

Methaemalbuminaemia is useful to predict haemorrhagic pancreatitis, hypoxemia, fibrinogen, complement products. They cannot predict severe AP with >90% accuracy.

1. Hematocrit

Decrease in plasma volume occurs with increased hematocrit. 44% increase in hematocrit occurs and reverts back within 24 hours .It is an early predictor of pancreatic necrosis [108]. According to Whitcomb et al risk of necrosis is less if hematocrit is <40%[109].

2. Blood Urea Nitrogen **(BUN)**

It shows changes in intravascular volume status similar to hematocrit. It evaluates mortality risk.

Increase by 5 mg/dl- mortality increases by odds ratio of 2.2 within the first 24 hrs of admission [110]. Risk of death was also higher when BUN was ≥ 20 mg/dl at admission. It is also a component of Ranson and APACHE prognostic scores.

3. Serum Creatinine

In pancreatic necrosis, increase in serum creatinine in the first 48 hours of admission is seen[111] with minimal literature support.

MARKERS OF PANCREATIC INJURY

1. *Trypsinogen Activation Peptide*(TAP)

It is produced during cleavage of trypsinogen to trypsin.

TAP > 30 nmol/L in urine –severe pancreatitis.

80% PPV and NPV close to 100% has been reported when urine analysis was done in the first 12 hours. Similar to APACHE II at 24 hrs after admission and even more after 48 hrs it is useful[112]. Not useful in monitoring as early secretion of TAP decline after 72 hours .

2. Carboxypeptidase B activation peptide(CBAP)

CBAP is more stable than TAP. Easier to measure. Severity assessment using urinary CBAP at 48 hrs is as good as APACHE II.

An early rise in CBAP levels follows a rapid decline. Hence not useful for monitoring purpose.[113]

3. Trypsinogen-2[13]

Trypsinogen has two major isoenzymes, trypsinogen-1 (cationic) and trypsinogen-2 (anionic). They are excreted in the urine. In dipstick test TRY-2 in the urine is sensitive and specific marker.

MARKERS OF INFLAMMATION

1. C-Reactive Protein

An acute phase protein. Its levels increase in nearly all acute and chronic inflammatory diseases. It is useful in measuring activity of inflammatory bowel disease and pancreatitis. It is a good biochemical marker for predicting the severity of AP [115]; not specific for pancreas.

Useful after 48 hours from the onset of symptoms rather than early phase of AP. May vary from 120 to 210 mg/L [114].

Cutoff level-150 mg/L in the first 48 hours of symptom.

Sensitivity and specificity - 80-86% and 61-84%,

2. Interleukins[13,115]

They are proinflammatory cytokines (IL-6 and IL-8). They peak at 72 hrs after the clinical onset of disease. The 2009 Atlanta Classification group suggested IL-6 to be more superior to IL-8, also to CRP and APACHE-II on Day 1. They also predict organ failure and necrotizing pancreatitis. TNF, MIP, CD 40, IL-18 are the other cytokines used to assess the severity of AP.

3. Procalcitonin(PCT)

It is a propeptide of calcitonin. It is released by hepatocytes, monocytes and G-cells of the thyroid gland.

At an early stage, an increased PCT is seen.

PCT > 0.5 ng/mL is an indicator of severe AP (specificity 73 %-87%)[13] .

PCT > 3.8 ng/mL within 48-96 hrs of symptom - organ failure and pancreatic infection [116]

4. Polymorphonuclear Leukocyte Elastase(PLE)

PLE is an enzyme released and activated by granulocytes.

values > 110 μg/L within 24-72 hrs-severe AP [117].

PLE rises early in pancreatitis than other parameters.

Other prognostic markers

Coagulation Parameters:

In severe AP Coagulation profile is deranged. Presence of DIC, levels of Tissue factor, Protein C, D-dimer levels are useful.

Obesity

It is a poor prognostic indicator , usually a BMI >30 kg/m2

Other Novel Markers

Small studies are available for the evaluation of Nitric Oxide and other free radicles, activated protein C-protein C inhibitor complex in plasma, E-Cadherin in predicting SAP.

Length of hospital stay :

Severity depends on the natural progression and associated morbidity[118]

According to Atlanta classification:

Mild, Moderately severe, Severe AP.

Depends on

1. Organ failure (OF)

2. local and systemic complications.[119]

Mostly it results in mild AP with a brief and uncomplicated hospital course.[120]

Moderately severe and severe AP has been associated with increased morbidity and mortality.

MATERIALS AND METHODS

I. Type of study : Observational study

II. Setting : Salem Medical College and Hospital

IV. Duration of Study : April 2017–September 2019

V.Ethical Clearance **:** Ethical clearance was obtained
Copy of the letter is enclosed in Annexure I

VI. Consent :Informed consent was obtained before taking up each case for the study

VII. Inclusion criteria:

1.Age> 18 years .

2.All patients with history of alcohol intake > 21units /week

3.The Atlanta classification was used for diagnosis of AP.

4.They were followed prospectively for 6 months after discharge from the hospital or till death, whichever was earlier

VIII. Exclusion criteria:Patients who had any of the following were excluded from the study

1.Pancreatitis of other etiologiy.

2.Chronic Pancreatitis

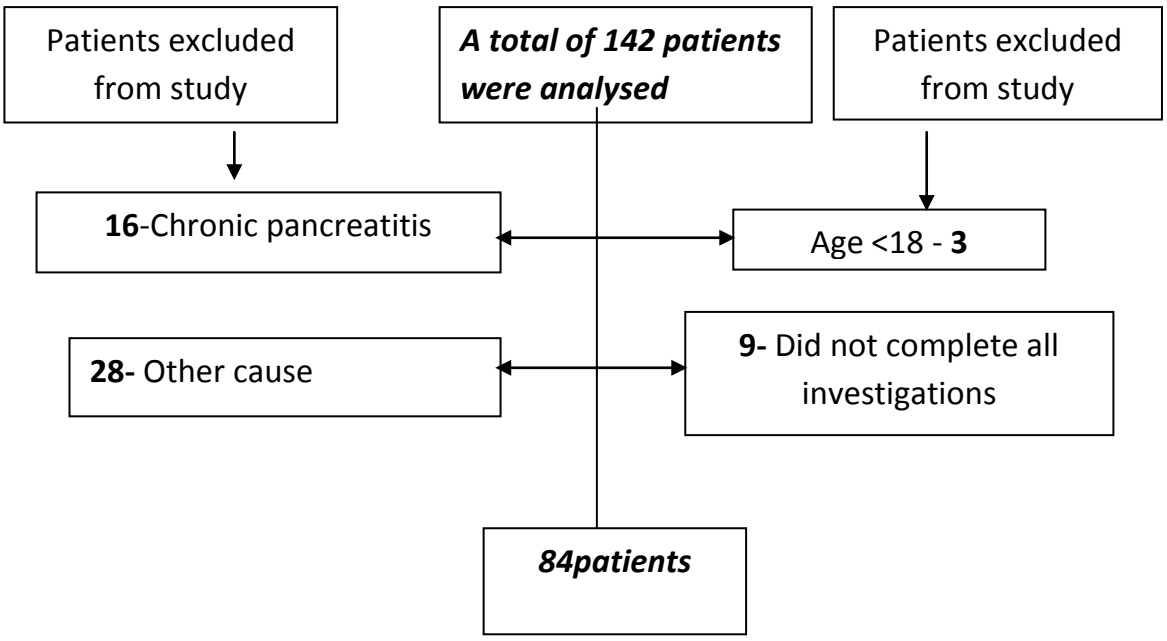
3.Patient had known comorbid disorders of respiratory, cardiovascular or renal systems.

4.Patient refused participation

IX. Materials

A total of 142 patients of acute pancreatitis were enrolled in the study based on the inclusion criteria and the set of exclusion. The exclusion of other patients is given in Figure below

Figure 1: Patient flow chart



Methods

Selected socio-demographic, clinical and laboratory data were elicited from these patients and recorded in a proforma. (Annexure II)

1. Socio-demographic data
   - Age
   - Sex
2. Clinical data
   - Clinical history was elicited in detail with special emphasis on abdominal pain, abdominal distention, decreased urine output, vomiting, blood vomitus, blackish stool, breathlessness, chest discomfort, swelling of legs, fever, yellowish discoloration of eyes or urine and substance abuse (alcohol and smoking)
   - Blood pressure, Pulse rate, Temperature, Respiratory rate, Oxygen saturation in peripheral blood (SpO2) was measured using standard procedures.
3. Clinical examination was done with special attention for abdominal guarding, rebound tenderness, impaired mental status, respiratory system for breath sounds
4. Laboratory data
   - Hematocrit: Estimated by 5 part cell counter (Pentra ES 60, Japan)

- Serum Amylase: Estimation was done by kinetic colorimetric method (Spin React ,Spain)
- Serum Lipase: Estimation was done by kinetic colorimetric method (Spin React ,Spain)
- Liver Function Tests, Blood urea, Serum creatinine, Blood Glucose, Serum Triglycerides, Serum Calcium : Estimation was done using COBAS autoanalyzer

5. Computerised Tomography: was done using Toshiba Aquilion 64 (Japan)

XI. Conflict of Interest : Nil

XII. Financial support : This study did not receive any financial support from any organization.

Data were entered in a predetermined proforma and later entered into a Microsoft excel spread sheet and analysed using SPSS Package 19.0

XIII. Limitations of the study

1. Investigations to rule out other causes of hyperamylasemia were not carried out

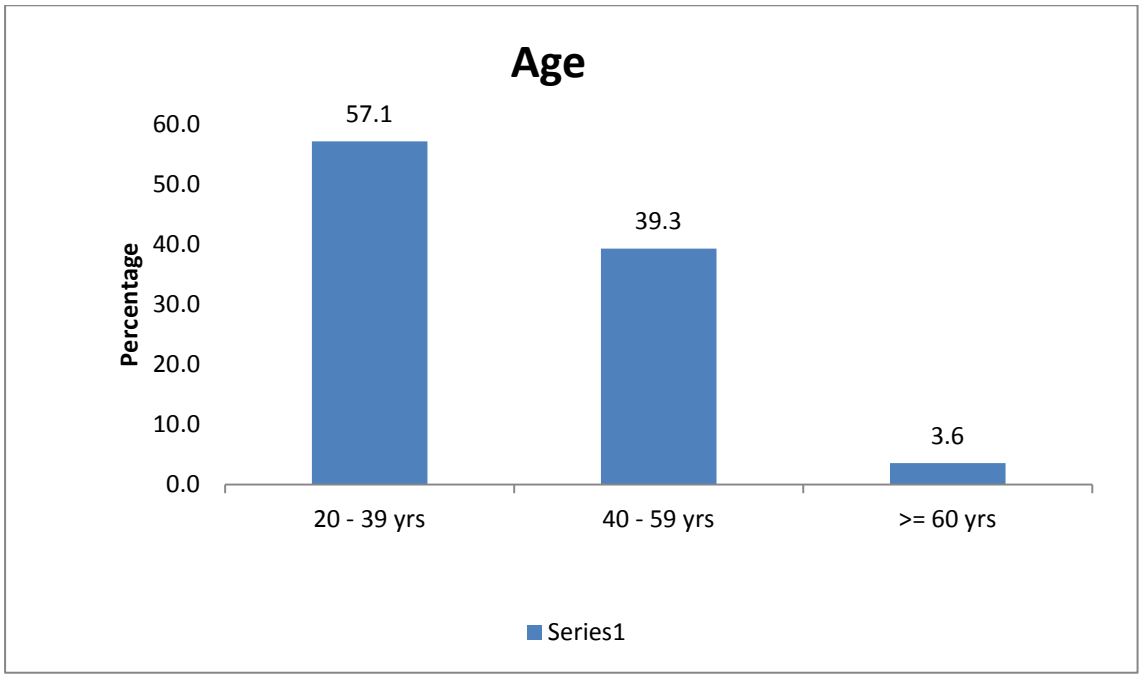
2.In the BISAP score Blood urea was taken into consideration not Blood urea nitrogen

The strength of the study is that it included an adequate number of patients with necessary investigations. It was done in a resource limited setting with no external funding. We could do the minimum required investigations for assessment of acute pancreatitis but could not do other specific markers as mentioned earlier. We could not repeat initial lab values for all patients but we definitely monitored renal function, amylase and lipase for all patients. In view of the above reasons we could not calculate the scores at different times of hospital stay. Though the detailed scoring systems offer significant advantage of risk assessment we could infer that initial lab makers especially CRP, LDH and lipase could be useful for initial triaging and predicting morbidity and mortality.

RESULTS

Age distribution:

CHART 1 : **Age distribution**



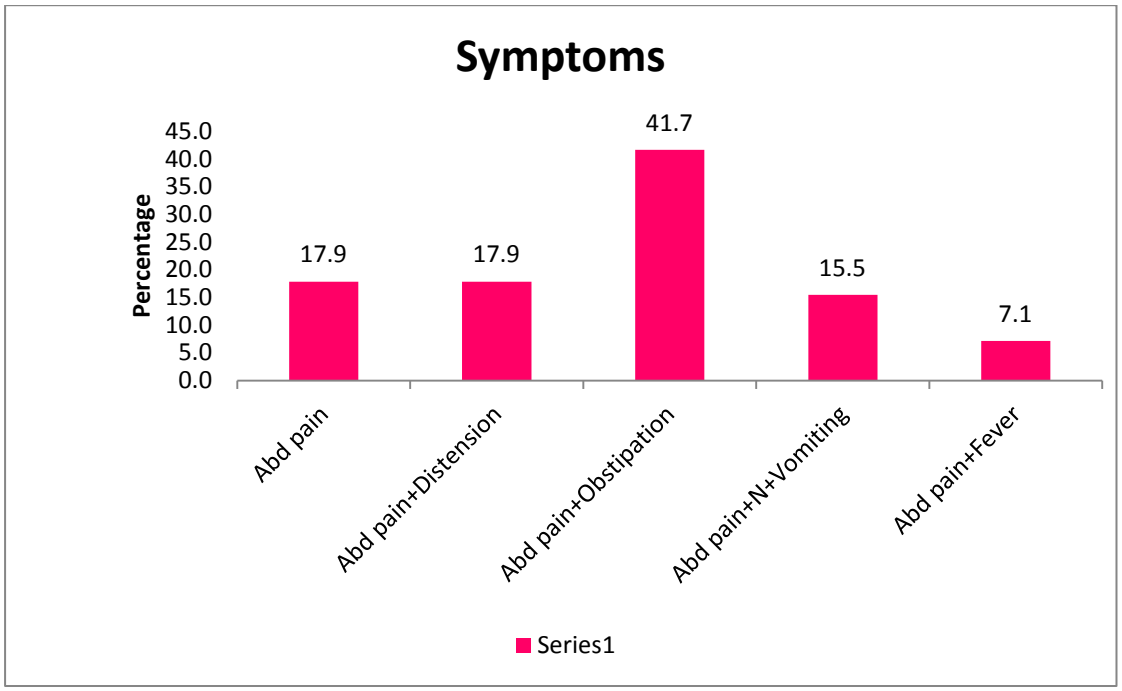
The prevalence of acute pancreatitis in the age group of 20-39 years is 57.1%, 40-59 years is 39.3 %,>60 years is 3.6 %.

TABLE 1 Age distribution:

	Frequency	Percent
20 - 39 yrs	48	57.1
40 - 59 yrs	33	39.3
>= 60 yrs	3	3.6
Total	84	100.0

Symptoms :

CHART 2: symptoms



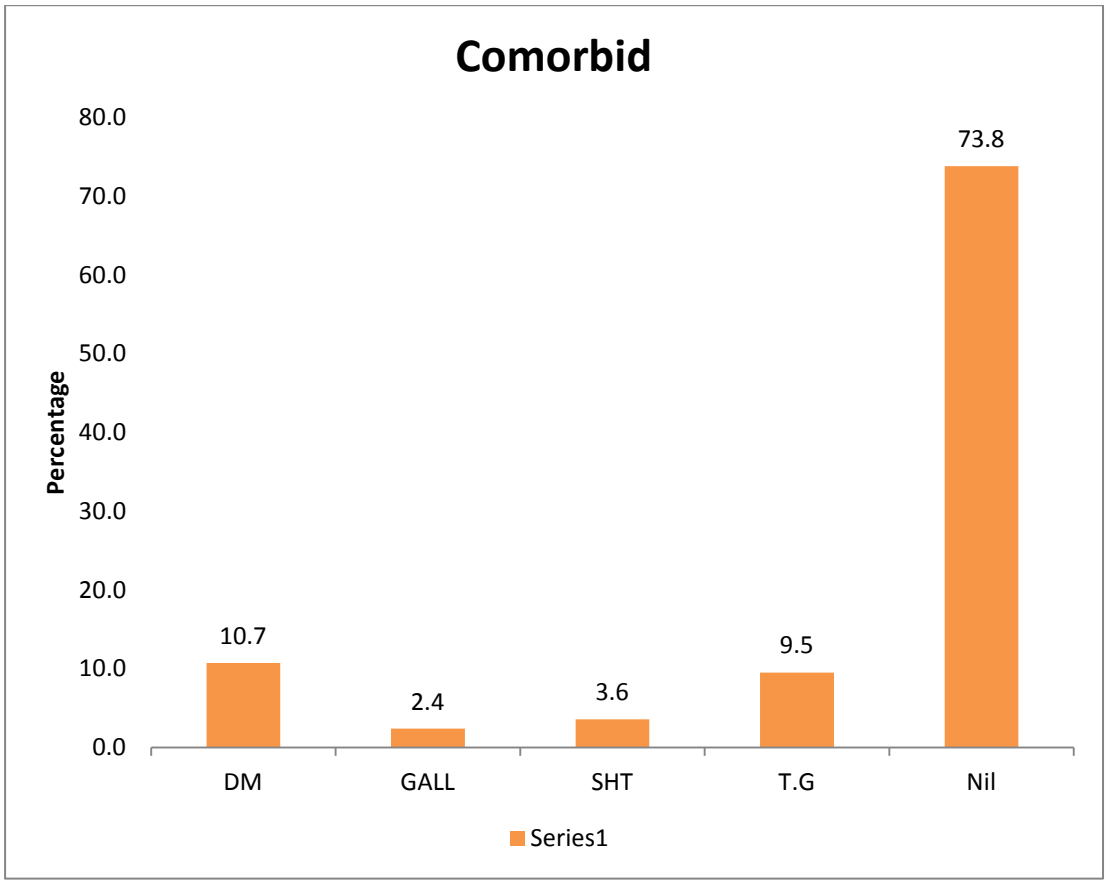
Abdominal pain with obstipation is the predominant presenting complaint in 41.7%(35 Patients). Presentation with abdominal pain or abdominal pain with distension is more or less equal (17.9%)(15 patients). 15.5 % (13 patients) presented with abdominal pain ,nausea and vomiting.7.1 % (6 patients) presented with abdominal pain and fever

TABLE 2- symptoms

	Frequency	Percent
Abd pain	15	17.9
Abd pain+Distension	15	17.9
Abd pain+Obstipation	35	41.7
Abd pain+N+Vomiting	13	15.5
Abd pain+Fever	6	7.1
Total	84	100.0

Comorbidities :

CHART 3 : Comorbidities :



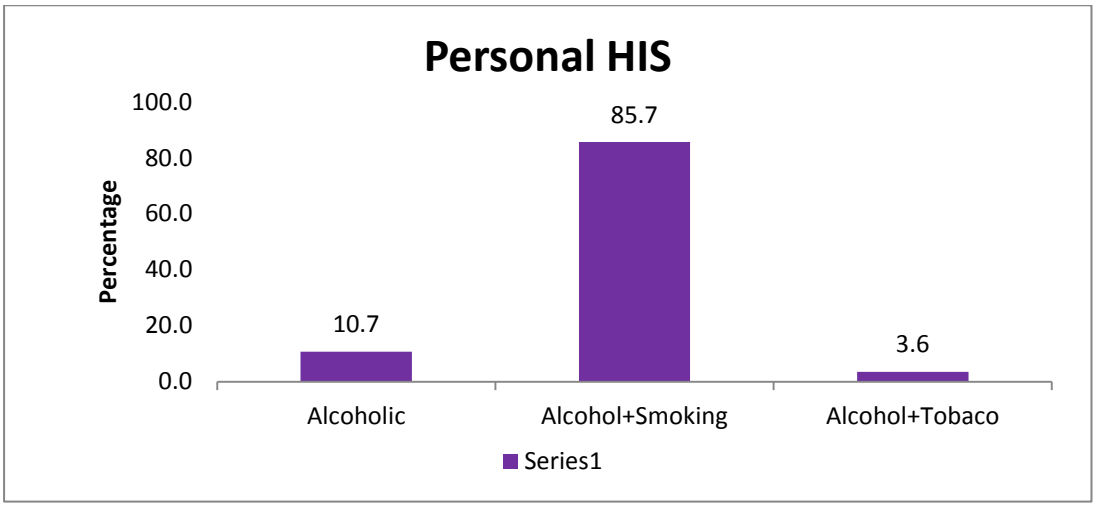
As most of the patients were young, 73.8% (62 patients) had no other comorbidities.10.7%(9 patients) had associated Diabetes mellitus. 9.5 % (8 patients) had hypertriglyceridemia, Hypertension is seen in 3.6%(3 patients). 2.4% (2 patients) had associated gall stones.

	Frequency	Percent
DM	9	10.7
GALL	2	2.4
SHT	3	3.6
T.G	8	9.5
Nil	62	73.8
Total	84	100.0

Table 3- Comorbidities :

Personal history:

CHART 4 : Personal history



85.7 % (72 patients) were both smoker and alcoholics.10.7 %(9 patients) were only alcoholics. 3.6 %(3patients) were alcoholics and tobacco users.

TABLE 4- Personal history:

	Frequency	Percent
Alcoholic	9	10.7
Alcohol+Smoking	72	85.7
Alcohol+Tobaco	3	3.6
Total	84	100.0

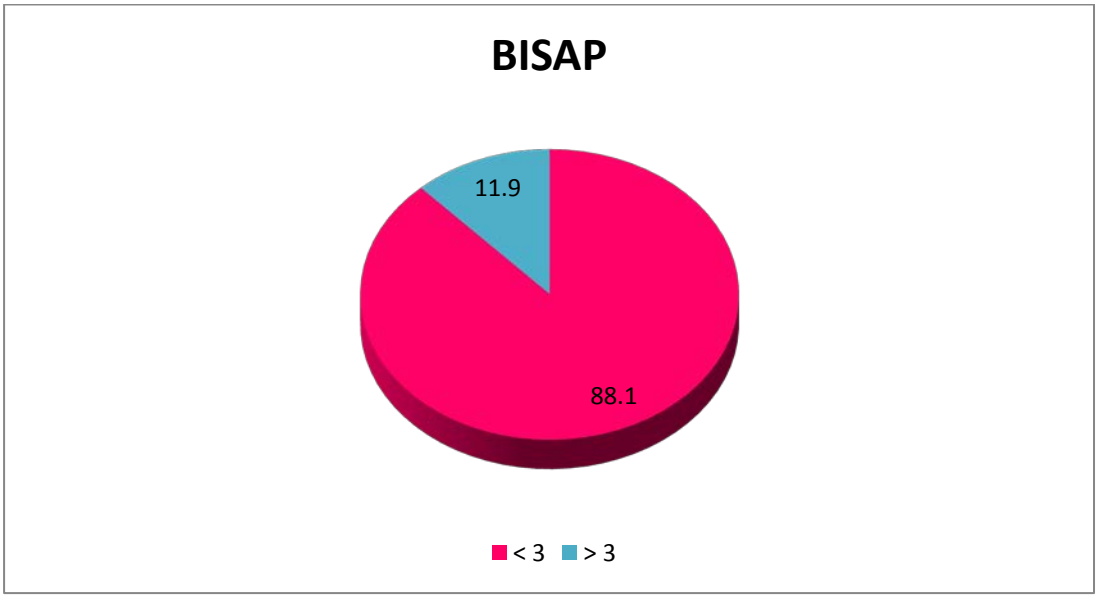
Alcohol units:

All patients had taken > 24 units /week

Scoring systems:

BISAP score:

CHART 5 : **BISAP score**



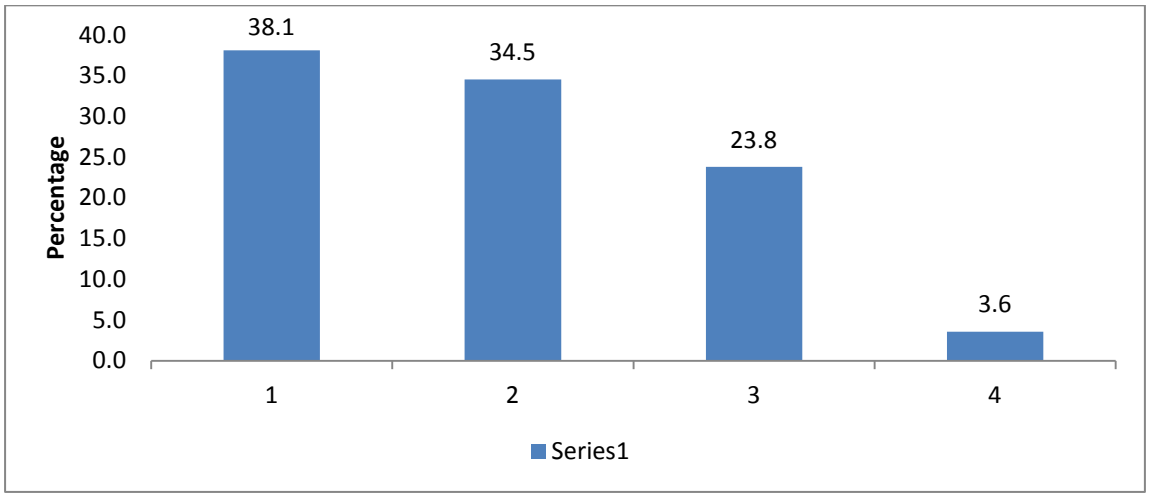
88.1 % (74 Patients) were grouped as with BISAP score <3. 11.9 % (10 patients) were grouped as with BISAP score > 3.

TABLE 5- BISAP score

	Frequency	Percent
< 3	74	88.1
> 3	10	11.9
Total	84	100.0

HAPS score:

CHART 6: HAPS score



38.1% (32 patients) had score of 1.

34.5 % (29 patients)had score of 2.

23.8%(20 patients) had score of 3.

3.6 %(3 patients) had score of 4.

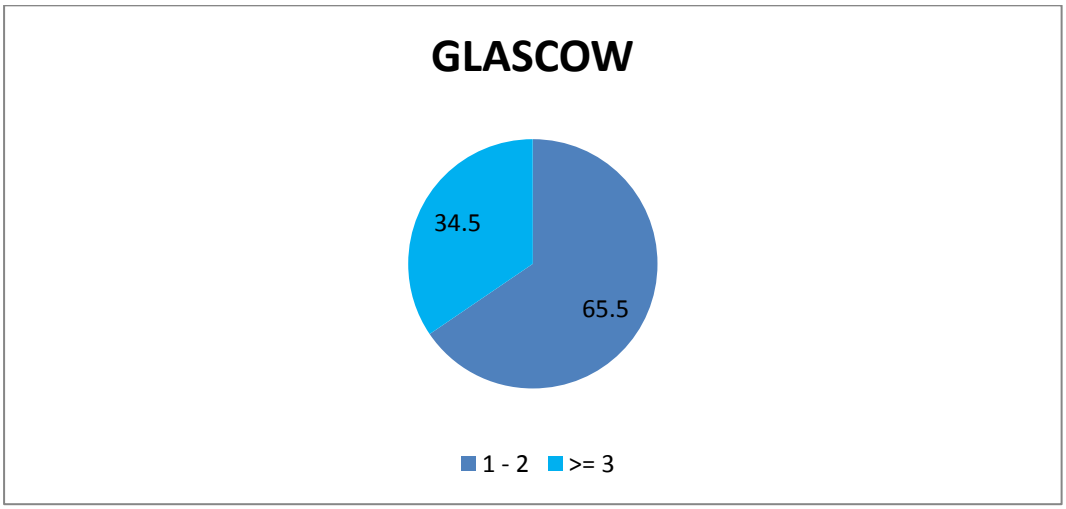
Hence majority (38.1% of patients) were grouped under the score of 1

TABLE 6- HAPS score

	Frequency	Percent
1	32	38.1
2	29	34.5
3	20	23.8
4	3	3.6
Total	84	100.0

Glascow score:

CHART 7: **Glascow score**



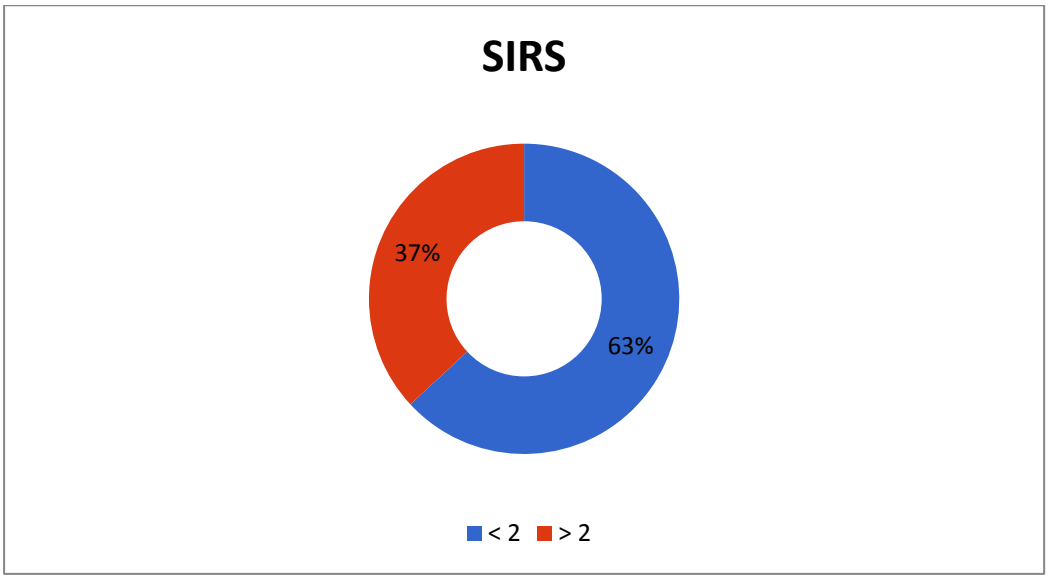
Score of 1-2 is seen in 65.5 % (55 patients). 34.5 % (29 patients) presented with score of >= 3

TABLE 7-Glascow score:

	Frequency	Percent
1 - 2	55	65.5
>= 3	29	34.5
Total	84	100.0

SIRS:

CHART 8: SIRS:



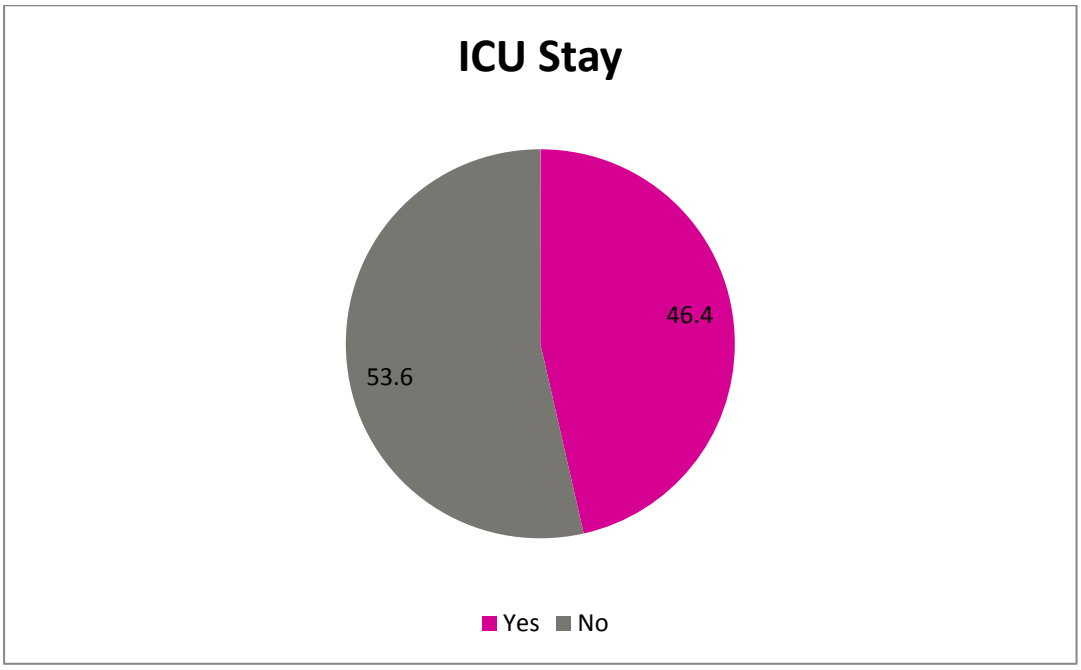
63 .1% (53 patients) had score of <2. 36.9 % (31 patients) had score >2.

TABLE 8-SIRS:

	Frequency	Percent
< 2	53	63.1
> 2	31	36.9
Total	84	100.0

ICU stay:

CHART 9: ICU stay



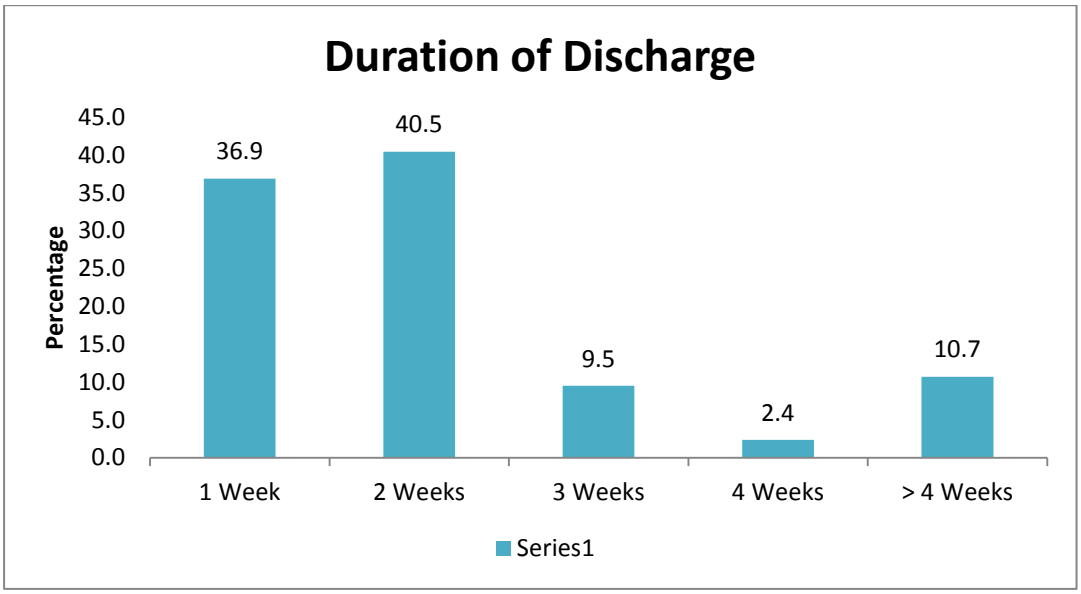
46.4 % (39 patients) had severe pancreatitis and have been admitted in ICU. Remaining 45 patients (53.6%) had no necessity for ICU stay.

TABLE 9- ICU STAY

	Frequency	Percent
Yes	39	46.4
No	45	53.6
Total	84	100.0

Duration of ICU stay:

CHART 10: Duration of ICU stay



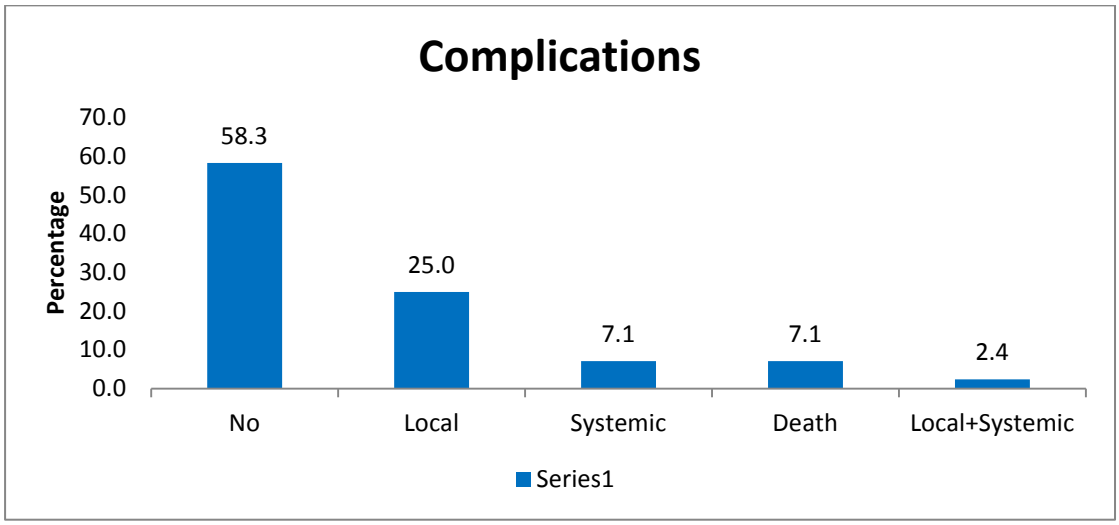
Longer length of hospital stay is seen in 10.7 % (9 patients) i.e. greater than 4 weeks.2.4 % (2 patients) had hospital stay of 4 weeks.9.5 % (8 patients) had stay of 3 weeks. 40.5 % (34 patients) had stay of 2 weeks. 36.9 % (31 patients) had hospital stay of 1 week.

TABLE 10-Duration of ICU stay

	Frequency	Percent
1 Week	31	36.9
2 Weeks	34	40.5
3 Weeks	8	9.5
4 Weeks	2	2.4
> 4 Weeks	9	10.7
Total	84	100.0

Complications :

CHART 11: Complications



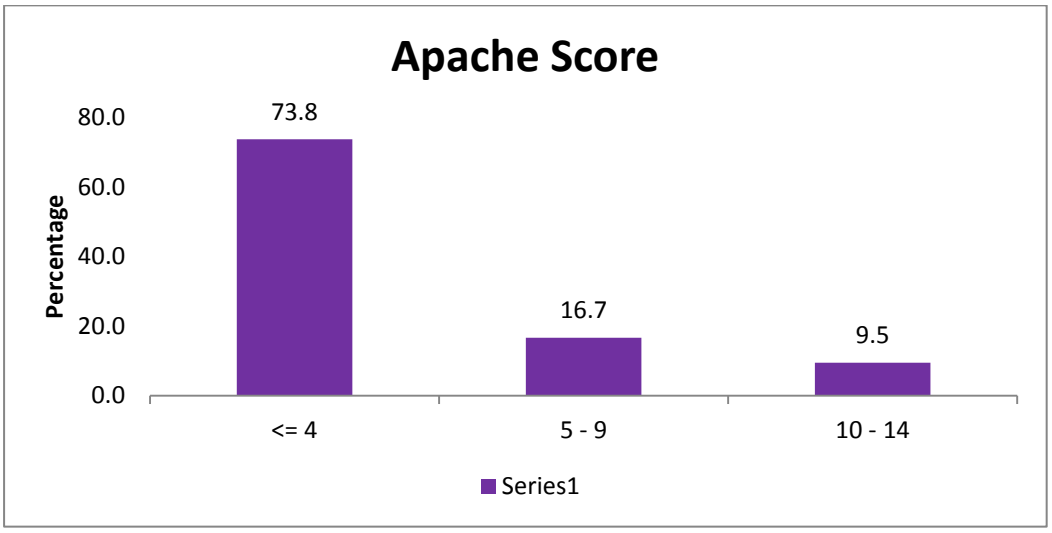
58.3 % (49) patients had no complications due to effective initial resuscitation.25% (21 patients)had local complications.7.1 %(6 patients) had systemic complications.2.4% (2 patients)had both systemic and local complications.7.1 % (6 patients)died.

TABLE 11- Complications;

No	49	58.3
Local	21	25.0
Systemic	6	7.1
Death	6	7.1
Local+Systemic	2	2.4
Total	84	100.0

APACHE score:

CHART 12: APACHE score



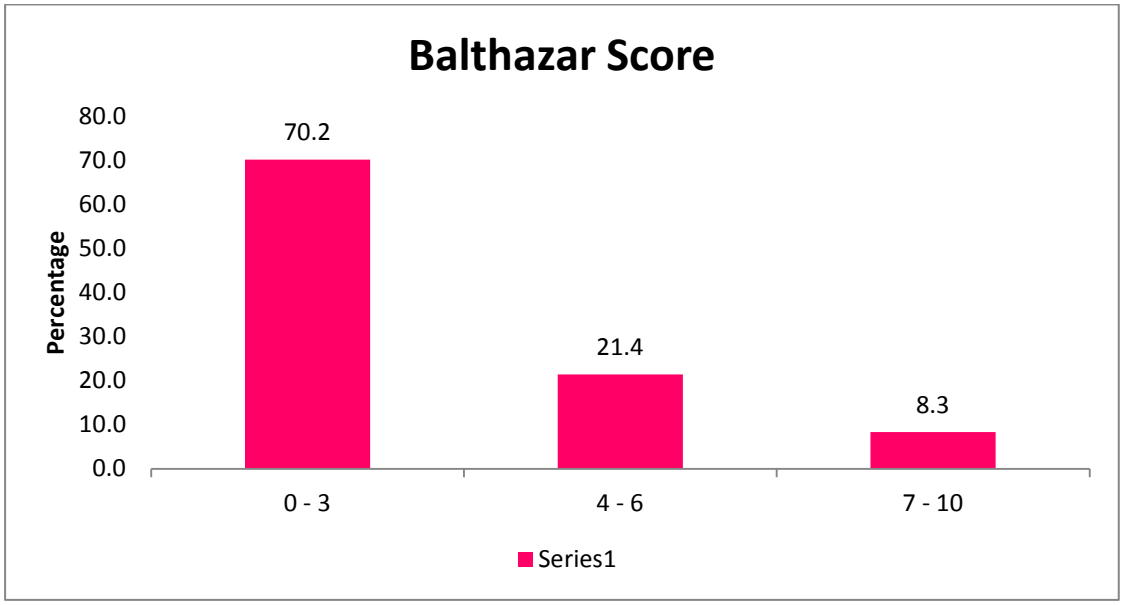
73.8% (62 patients)had score of <4.16.7 %(14 patients) had score of 5-9. 9.5 %(8 patients) had score of 10-14.

TABLE 12 -APACHE score

	Frequency	Percent
<= 4	62	73.8
5 - 9	14	16.7
10 - 14	8	9.5
Total	84	100.0

Modified BALTHAZAR score:

CHART 13: Modified BALTHAZAR score



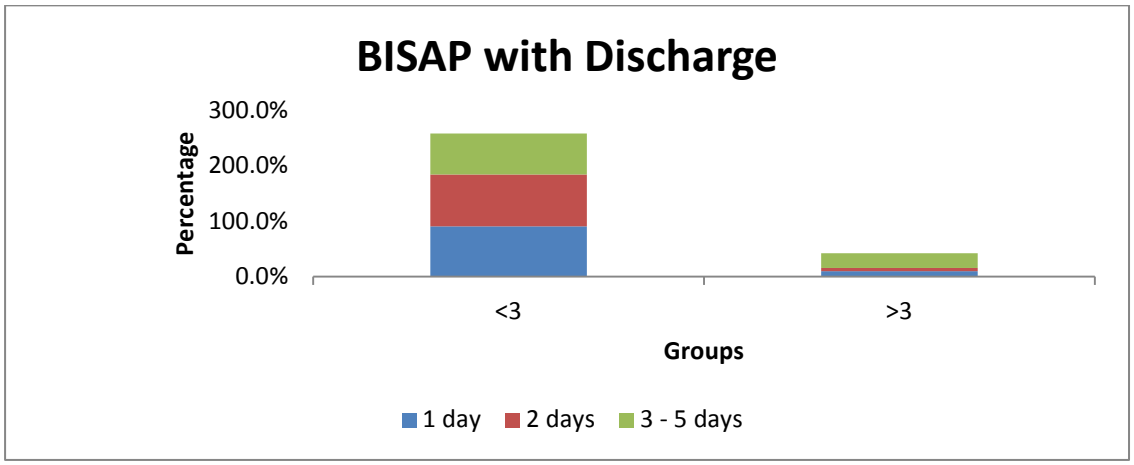
Majority of patients i.e. 70.2 % (59) had score of 0-3. 21.4%(18 patients) had score 4-6. 8.3% (7 patients) had score 7-10.

TABLE 13- Modified BALTHAZAR score

	Frequency	Percent
0 - 3	59	70.2
4 - 6	18	21.4
7 - 10	7	8.3
Total	84	100.0

Comparison between BISAP and Discharge:

CHART 14: Comparison between BISAP and Discharge



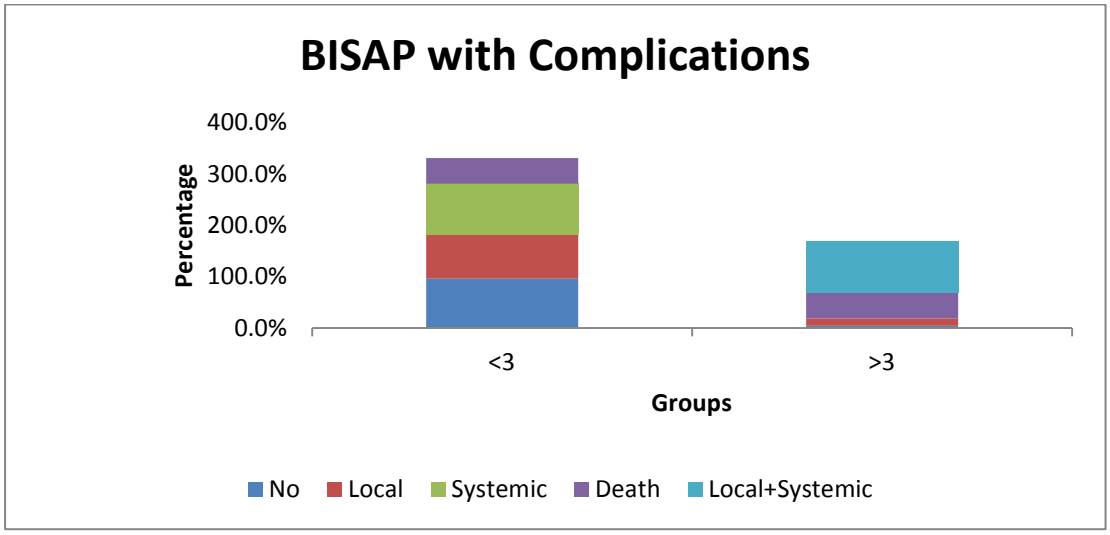
BISAP score does not have significant p value in predicting discharge of patients.

TABLE 14: Comparison between BISAP and Discharge

Comparison between BISAP with Discharge								
			Discharge			Total	⍰ 2 - value	P-value
			1 day	2 days	3 - 5 days			
BISAP	< 3	Count	28	32	14	74	5.085	0.079 #
		%	90.3%	94.1%	73.7%	88.1%		
	> 3	Count	3	2	5	10		
		%	9.7%	5.9%	26.3%	11.9%		
Total		Count	31	34	19	84		
		%	100.0%	100.0%	100.0%	100.0%		
# No Significant at P < 0.05 level								

Comparison of BISAP with complication:

CHART 15 : Comparison of BISAP with complication

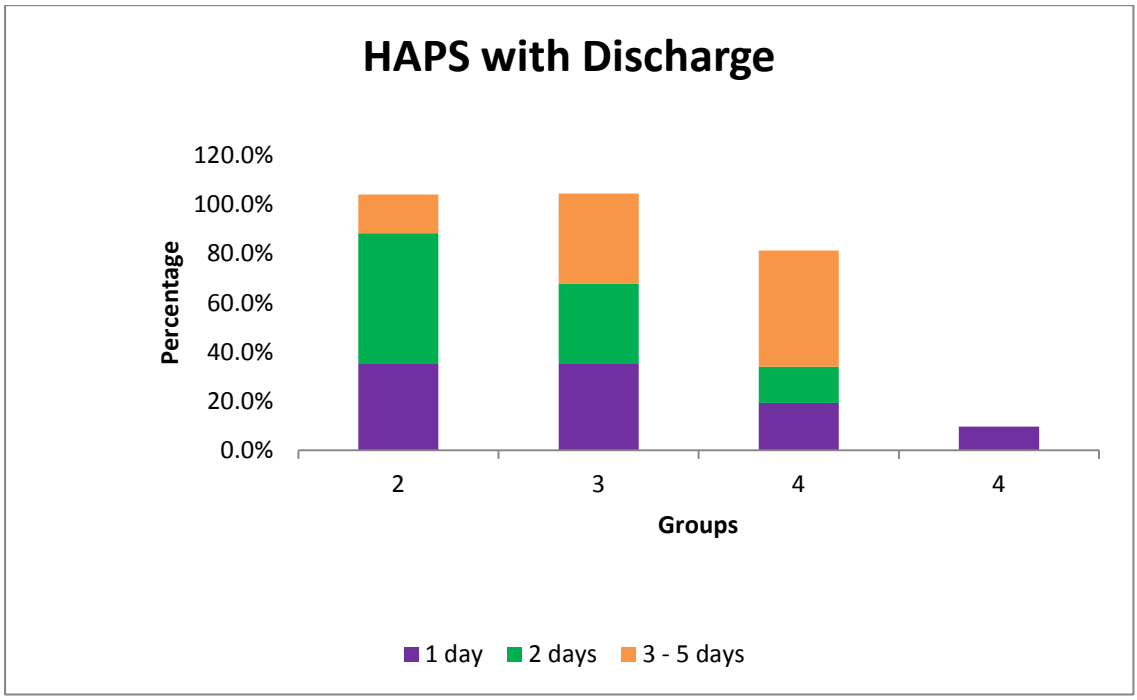


BISAP showed highly significant p value in predicting complications.

TABLE 15 : Comparison of BISAP with complication

			COMPLICATIONS					
			No	Local	Systemic	Death	Local+Systemic	Total
BISAP	< 3	Count	47	18	6	3	0	74
		% within COMPLICATIONS	95.9%	85.7%	100.0%	50.0%	0.0%	88.1%
	> 3	Count	2	3	0	3	2	10
		% within COMPLICATIONS	4.1%	14.3%	0.0%	50.0%	100.0%	11.9%
Total		Count	49	21	6	6	2	84
		% within COMPLICATIONS	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%

CHART 16: Comparison between HAPS and Discharge

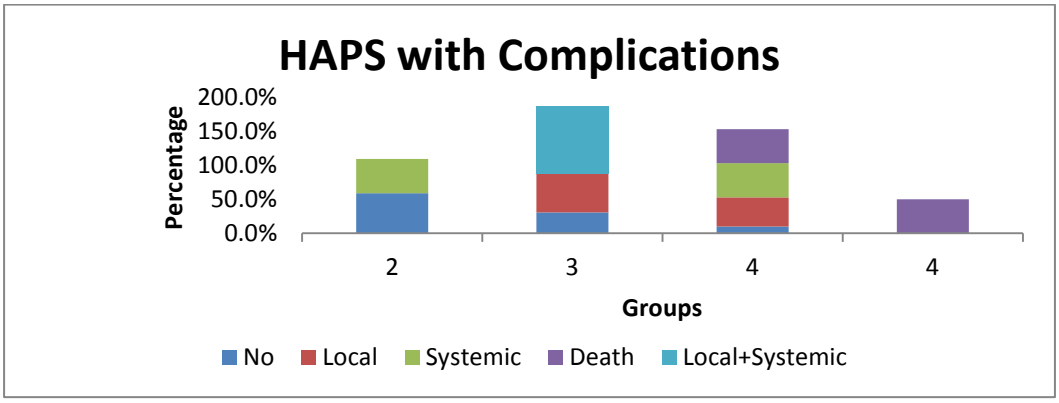


HAPS score does not have significant p value in predicting discharge of patients.

TABLE 16: Comparison between HAPS and Discharge

Comparison between HAPS with Discharge								
			Discharge			Total	⍰ 2 – value	P-value
			1 day	2 days	3 - 5 days			
HAPS	1	Count	11	18	3	32	15.588	0.016 #
		%	35.5%	52.9%	15.8%	38.1%		
	2	Count	11	11	7	29		
		%	35.5%	32.4%	36.8%	34.5%		
	3	Count	6	5	9	20		
		%	19.4%	14.7%	47.4%	23.8%		
	4	Count	3	0	0	3		
		%	9.7%	0.0%	0.0%	3.6%		
Total		Count	31	34	19	84		
		%	100.0%	100.0%	100.0%	100.0%		
# No Significant at P < 0.05 level								

Comparison of HAPS with complication:CHART 17 : Comparison of HAPS with complication

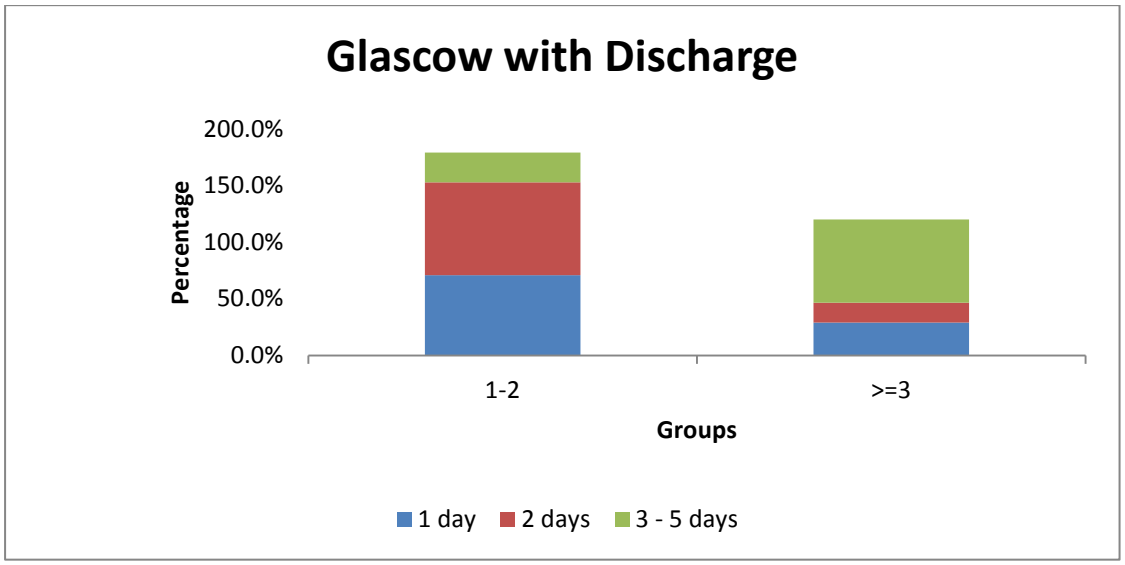


HAPS showed highly significant p value (<0.01)in predicting complications.

TABLE 17 : Comparison of HAPS with complication

Comparison between HAPS with Complications										
			COMPLICATIONS					Total	2 - value	P-value
			No	Local	Systemic	Death	Local+ Systemic			
HAPS	1	Count	29	0	3	0	0	32	76.890	0.0005 **
		%	59.2%	0.0%	50.0%	0.0%	0.0%	38.1%		
	2	Count	15	12	0	0	2	29		
		%	30.6%	57.1%	0.0%	0.0%	100.0%	34.5%		
	3	Count	5	9	3	3	0	20		
		%	10.2%	42.9%	50.0%	50.0%	0.0%	23.8%		
	4	Count	0	0	0	3	0	3		
		%	0.0%	0.0%	0.0%	50.0%	0.0%	3.6%		
Total		Count	49	21	6	6	2	84		
		%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%		
** Highly Significant at P < 0.01 level										

CHART 18: Comparison of Glascow score and Discharge

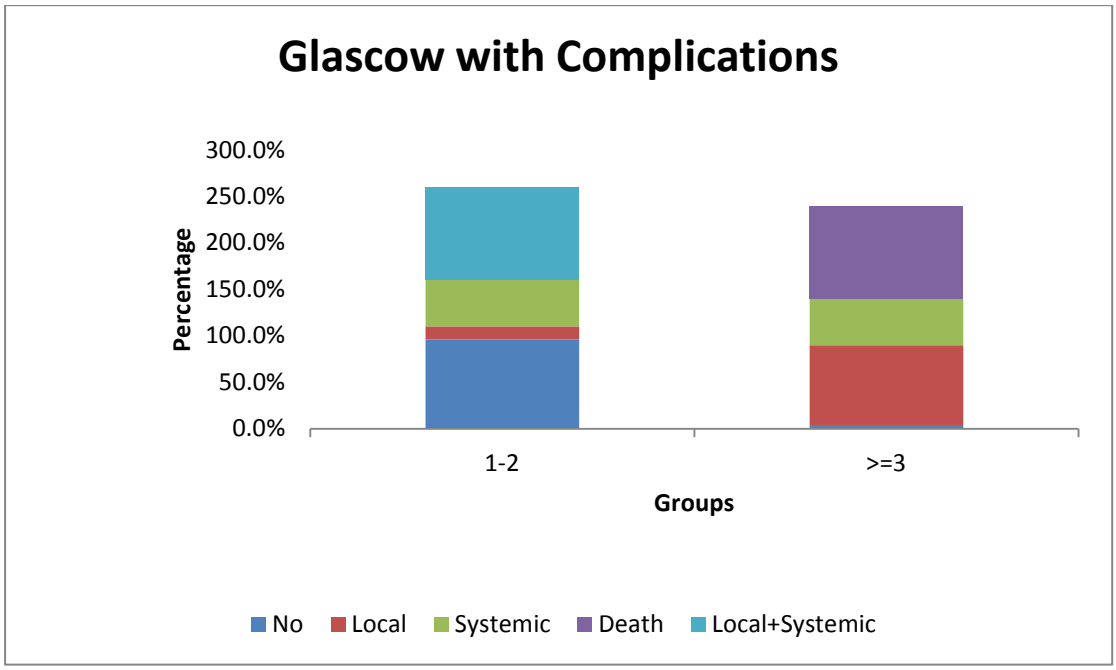


Glascow score highly significant p value (<0.01) in predicting discharge.

TABLE 18: Comparison of Glascow score and Discharge

Comparison between GLASCOW with Discharge								
			Discharge			Total	⍰ 2 - value	P-value
			1 day	2 days	3 - 5 days			
GLASCOW	1 - 2	Count	22	28	5	55	17.587	0.0005 **
		%	71.0%	82.4%	26.3%	65.5%		
	>= 3	Count	9	6	14	29		
		%	29.0%	17.6%	73.7%	34.5%		
Total		Count	31	34	19	84		
		%	100.0 %	100.0 %	100.0 %	100.0 %		
** Highly Significant at P < 0.01 level								

CHART 19: Comparison of Glascow score with complication



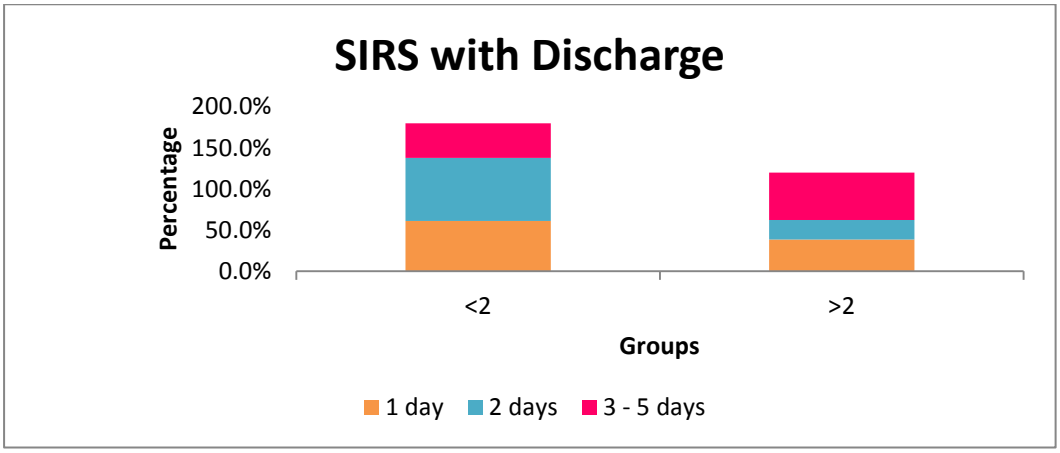
Highly significant p value (<0.01)in predicting complications

Comparison between GLASCOW with Complications										
			COMPLICATIONS					Total	⍰ 2 - value	P-value
			No	Local	Systemic	Death	Local+Systemic			
GLASCOW	1 - 2	Count	47	3	3	0	2	55	57.502	0.0005 **
		%	95.9%	14.3%	50.0%	0.0%	100.0%	65.5%		
	>= 3	Count	2	18	3	6	0	29		
		%	4.1%	85.7%	50.0%	100.0%	0.0%	34.5%		
Total		Count	49	21	6	6	2	84		
		%	100.0%	100.0%	100.0%	100.0%	100.0%	100.0%		
** Highly Significant at P < 0.01 level										

TABLE 19: Comparison of Glascow score with complication

SIRS and discharge:

CHART 20: SIRS and discharge

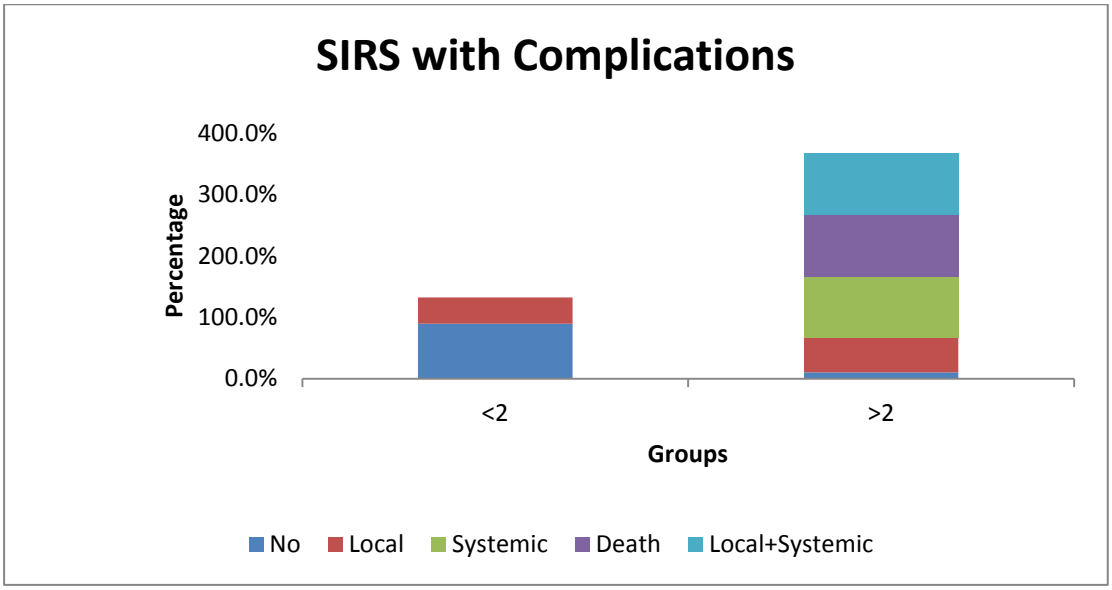


SIRS score does not have significant p value in predicting discharge of patients.

TABLE 20: SIRS and discharge

Comparison between SIRS with Discharge								
			Discharge			Total	⍰ 2 - value	P-value
			1 day	2 days	3 - 5 days			
SIRS	< 2	Count	19	26	8	53	6.251	0.044 #
		%	61.3%	76.5%	42.1%	63.1%		
	> 2	Count	12	8	11	31		
		%	38.7%	23.5%	57.9%	36.9%		
Total		Count	31	34	19	84		
		%	100.0%	100.0%	100.0%	100.0%		
# No Significant at P < 0.05 level								

CHART 21: Comparison of SIRS with complication

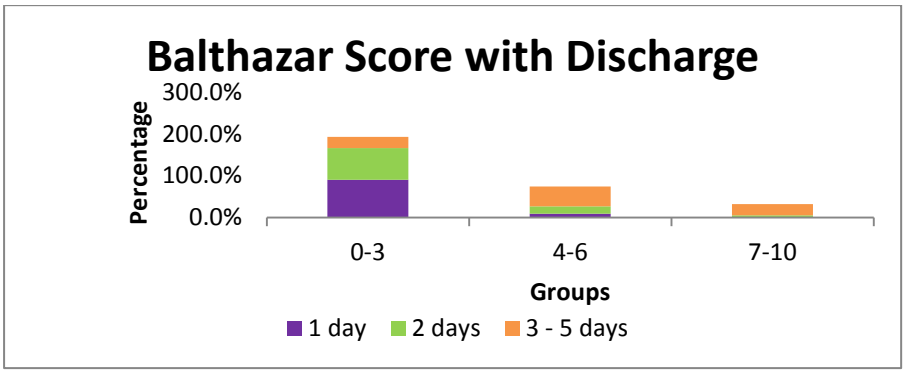


SIRS showed highly significant p value in predicting complications.

TABLE 21-Comparison of SIRS with complication:

Comparison between SIRS with Complications										
			COMPLICATIONS					Total	⍰ 2 – value	P-value
			No	Local	Systemic	Death	Local+Systemic			
SIRS	< 2	Count	44	9	0	0	0	53	42.632	0.0005 **
		%	89.8 %	42.9 %	0.0%	0.0%	0.0%	63.1 %		
	> 2	Count	5	12	6	6	2	31		
		%	10.2 %	57.1 %	100.0 %	100.0 %	100.0%	36.9 %		
Total		Count	49	21	6	6	2	84		
		%	100.0 %	100.0 %	100.0 %	100.0 %	100.0%	100.0 %		
** Highly Significant at P < 0.01 level										

CHART 22: Modified BALTHAZAR score with discharge

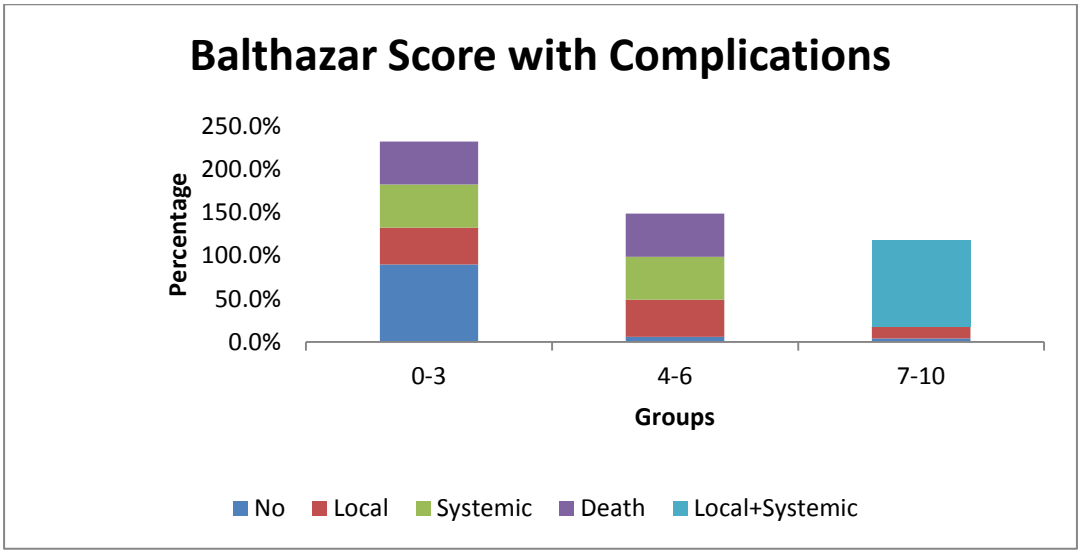


Modified BALTHAZAR score highly significant p value in predicting discharge of patients

TABLE 22: Modified BALTHAZAR score with discharge

Comparison between Modified BALTHAZAR score with Discharge								
			Discharge			Total	⍰ 2 - value	P-value
			1 day	2 days	3 - 5 days			
Modified BALTHAZAR score	0 - 3	Count	28	26	5	59	25.579	0.0005 **
		%	90.3%	76.5%	26.3%	70.2%		
	4 - 6	Count	3	6	9	18		
		%	9.7%	17.6%	47.4%	21.4%		
	7 - 10	Count	0	2	5	7		
		%	0.0%	5.9%	26.3%	8.3%		
Total		Count	31	34	19	84		
		%	100.0 %	100.0 %	100.0 %	100.0 %		
** Highly Significant at P < 0.01 level								

CHART 23: Modified BALTHAZAR score with complications



Modified BALTHAZAR score showed highly significant p value (0.01) in predicting complications.

CHART 23: Modified BALTHAZAR score with complications

Comparison between Modified BALTHAZAR score with Complications										
			COMPLICATIONS					Total	χ^2 - value	P-value
			No	Local	Systemic	Death	Local+Systemic			
Modified BALTHAZAR score	0-3	Count	44	9	3	3	0	59	44.994	0.0005 **
		%	89.8%	42.9%	50.0%	50.0%	0.0%	70.2%		
	4-6	Count	3	9	3	3	0	18		
		%	6.1%	42.9%	50.0%	50.0%	0.0%	21.4%		
	7-10	Count	2	3	0	0	2	7		
		%	4.1%	14.3%	0.0%	0.0%	100.0%	8.3%		
Total		Count	49	21	6	6	2	84		
		%	100.0 %	100.0 %	100.0%	100.0 %	100.0%	100.0 %		
** Highly Significant at P < 0.01 level										

All scores had a significant association in predicting complications, but none were superior to the other in predicting with pancreatitis with respect to duration of discharge.

Discussion

Pancreatitis show varying morbidity and mortality especially the severe necrotizing type. This mortality will be even more if there is bacterial contamination. Severe form of the disease may have a lesser mortality if diagnosed and treated early. Eventhough scoring systems like (Ranson's criteria, APACHE II score, Glasgow scoring system) and radiological scoring systems (CTSI /Balthazar scoring system) are available, management of both mild and severe forms remains cumbersome.

In our study pancreatitis predicting scores SIRS, BISAP, APACHE, GLASCOW, HAPS and CTSI were analyzed to predict the severity of pancreatitis. simpler and easily available parameters containing scores and markers were taken for the study. With limited resources , the simplest and the most economical of the scores or markers would be of great help in management of cases.

Epidemiology

Out of 142 patients alcohol induced pancreatitis was higher (51%) than gall Stone induced pancreatitis. This can be explained by the greater incidence of alcohol abuse in Tamilnadu. Incidence of alcoholic pancreatitis is mostly seen in young males, particularly of middle age group. All the patients had significant alcohol history. Out of them 85.7% were associated with smoking history. In this study alcohol which is mostly abused by men than women and younger age group than old prevalence is more in young males. Most of the patients had no comorbidities (73.8%), because Prevalence of alcoholic pancreatitis was high in the young healthy males who were addicted to the alcohol. According to Venkata Krishnan et al. from Chennai epidemiology of study were similar.

Symptoms

Abdominal pain (100%) & followed by obstipation were the predominant complaints seen in the study population. This is similar to the study by Milheiroet al[121]who stated the predominant symptom in AP as abdominal pain in 100% followed by vomiting in 69.2%. Extrapancreatic manifestations have greater correlation to length of the hospital stay, which showed that the presence of Extra pancreatic manifestations in acute pancreatitis had a high probability to be

associated with Severe acute pancreatitis . In this study there was a significant association with fever, dyspnea and oliguria which is similar to the study by Abbasi[122]and Jacobs et al[123] .Thus this study further emphasizes the well-known fact of the need of aggressive fluid management in AP thus preventing volume depletion, which may lead on to the development and progression of Severe acute pancreatitis.

In this study alcohol was the predominant cause of AP seen in 68.8% while a biliary cause of pancreatitis was seen only in 10.9% of the study population, in contrary to Roberts who reported from the UK that 36.9% patients had gallstone as the predominant etiology of AP followed by alcohol (22.0%). Sekimoto[23]showed, in the Japanese population alcohol contributed to 37% of AP and the biliary system contributed to 20%, while Abbasi et al showed in African Americans, alcohol was the predominant cause in 53 % of patients, these studies have shown parallel results to the present study. This study goes with the literature, which states that alcohol intake is more common followed by gall stones in causing pancreatitis. In south India due to dietary pattern incidence of gallstone is low than the North India.

Other Causes of AP were seen in 28 patients in the study group. They were excluded from the study, 3 patients had carcinoma pancreas, one was tropical pancreatitis and 2 patients had pancreatic divisum. In 8

patients etiology was not known and an evaluation was not attempted due to the first episode of acute pancreatitis

Duration of discharge:

53.9% had no ICU stay, ICU stay seen in 46.4 % **, < 2week .**

77.4% patients of the study population had mild pancreatitis, while> 2 weeks 22.6%patients had Severe acute pancreatitis. Duration of discharge is directly proportional to the severity of pancreatitis. 70.2.% patients of the study population had mild pancreatitis, while 29.7% patients had SAP as determined by CT, which is taken as standard to predict the severity of pancreatitis. Incidence of SAP is similar to study by Svetlana[95]but higher than that of studies by Davor[124], Banks[31]and as suggested by the Atlanta study group[55]. The reason could be that in our population there is a delay in presentation to the hospital as the patients seek over the counter medications or complementary and alternative forms of medicine for the most common symptom of abdominal pain or it could be that of a referral bias.

Analysis of Pancreatic Scores with Duration of discharge, computerized Tomography and complication.

Three pancreatic scores were taken in the study HAPS, BISAP, GLASCOW and SIRS, all of which have easily obtainable variables and can be calculated at the time of admission. This study evaluated the

efficacy of these scores in comparison with hospital stay, complications and CT severity in predicting Severe acute pancreatitis. This study suggests all the pancreatic predictive scores have an excellent predictive value in predicting severe acute pancreatitis. None of the scores were superior to the other in predicting SAP. This is in similarity with studies by Papachristou, Park, and Khanna [125-127]

BISAP score in this study had a <3 -<1 week duration for discharge 37.8 %, 1- 2 weeks duration of discharge is in 43%. BISAP score does not have significant p value in predicting discharge of patients. But in score <3- 63.5% had no complication.> 3 of BISAP had 80% of complication including death in all patients. BISAP showed highly significant p value in predicting complications. The study correlates with the study by Gompertzet al 's study in Spain who reported a BISAP sensitivity, specificity, positive and negative predictive value of 71.4, 99.1, 83.3 and 98.3% respectively.[128]

HAPS score in this study had a similar value to BISAP score, HAPS score 0 - early discharge (90.60). High score had death in all patients. HAPS showed highly significant p value (<0.01)in predicting complications. This is concordance with the study by Lankisch et al who stated that HAPS had a 98% efficacy in predicting SAP.[106]

Singh et al showed that SIRS when present on admission had a sensitivity 85%-100% in predicting SAP and a NPV of 98%-100% in the absence of SIRS on the day of admission to develop SIRS; but in this study even though SIRS predicted <2 score had 35.8 % early discharge <1 week.>2 score also had 38.7 % early discharge. SIRS score does not have significant p value in predicting discharge of patients.< 2 score 83 % had no complication &> 2 had only 16 % no complication.

Glascow score-<2, 50 patients had early discharge (< weeks)&>3 hospital stay in 73.7 %. Glascow score< 2 – 85.4 % patients had no complication &> 2 – 93.1 % had complications. This is similar to Deepa et al Glasgow criteria had high sensitivity (85.1%), NPV (79.4) in predicting ICU-admission.

Mortality

There were 6 (7.1%) deaths in the study population; all the patients had high pancreatic predicting scores. The mortality is higher than as reported by Mann and the national survey of Japan[23]

CONCLUSION

- Incidence of Alcoholic pancreatitis is more common in middle age males (20-39years)
- When alcohol intake is combined with smoking, risk of developing pancreatitis is more.
- With regard to prognosis 46.4% had severe pancreatitis and were managed in Icu. 53.6%had mild pancreatitis. All patients included in study had significant alcohol intake
- Correlation with length of hospital stay, mortality and complication rates was determined in comparison with different scoring systems.
- In predicting complications every scoring system is more or less equal.
- To predict duration of discharge Glascow score and modified Balthazaar score or valuable.
- HAPS is a simple bedside score with equal efficacy to other scoring systems. It helps in the disposal of these patients to an appropriate management setting. Moreover, its parameters are easy to remember; can be determined quickly and its laboratory components are available in most health facilities.

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ACUTE PANCREATITIS

PATIENT PROFORMA

Study no: _____

Name: _____ O.P/ I.P no: _____

Age ☐☐ (completed years) Sex : Male ☐1 Female ☐2

History

Symptoms	Yes	No	Symptoms	Yes	No
severe upper abdominal pain					
Radiating To Back			Nausea		
Diarrhea			Vomiting		
Fever and chills			Loss of Appetite		
Rapid heartbeat			Abdomen Distension		
Hiccups			Not passed flatus & Motion		
Difficulty In Breathing					

Co- Morbidities

Condition	Yes	No	Condition	Yes	No
Diabetes Mellitus			SHT		
Ischemic Heart Disease			Tuberculosis		
Gall Stones			High T.G		

Personal History

Habits	Yes	No	Habits	Yes	No
Smoking			Alcohol Intake		
Tobacco Chewing			If Yes,>24 U/week		
Skipping of Meals			Emotional Stress		
Sleep Pattern Disturbance			NSAID,Any Drug Intake		

Family History

Please Specify: _____

GENERAL EXAMINATION

Signs	Yes	No	Signs	Yes	No
Conscious/oriented:			Pallor		
Icterus			Cyanosis		
Clubbing			Edema		
Lymphadenopathy			Febrile		

Other signs:

VITALS:	HT:	WT:	BMI:
PULSE:	BP:	TEMP:	URINE OUTPUT:

Systemic Examination:

Oral Cavity: _____

P/A: _____

RS: _____

CVS: _____

CNS: _____

INVESTIGATIONS:

Hb		ESR	
TC		DC	P L E M
BT		CT	
Pl COUNT		RBS	
UREA		CREATININE	
ECG			
Chest X Ray			
USG Abdomen			
Albumin		AST	
Sr.Calcium		LDH	
PaO2		T.G	

SCORE

BISAP score		**HAPS Score**	
Ranson score		**Glasgow criteria**	
APACHE II		**Balthazar score**	
CRP		**SIRS**	

Localregional Complications			Systemic Complications		
Antibiotics	YES	NO	ICU Admission	YES	NO
Duration Of Hospital stay			Oral stats on	DAY -	

MASTER CHART

Acute Pancreatitis															
S.NO	SEX	AGE	SYMPTOMS	COMORBID	PERSONAL HIS	ALCOHOL	BISAP	HAPS	GLASCOW	SIRS	ICU STAY	>1 WEEK	DURATION OF DISCHARGE	COMPLICATIONS	APACHE SCORE	BALTHAZAR SCORE
1	1	1	3	7	2	2	1	3	2	1	1	2	5	2	1	3
2	1	1	3	5	2	2	1	3	2	1	1	2	5	2	2	2
3	1	1	1	7	2	2	1	1	1	1	2	3	2	1	1	1
4	1	1	3	4	2	2	1	4	2	2	1	1	1	4	3	2
5	1	2	3	7	2	2	1	1	1	1	2	3	1	1	1	1
6	1	2	2	7	2	2	1	1	1	1	2	3	2	1	1	1
7	1	2	5	7	2	2	1	1	1	1	2	3	2	1	1	2
8	1	2	1	7	1	2	1	1	1	1	2	3	1	1	1	1
9	1	1	3	7	3	2	1	1	1	1	2	3	2	1	1	1
10	1	2	2	7	2	2	1	3	2	2	1	1	2	3	2	1
11	1	1	3	5	2	2	2	3	2	2	1	2	5	2	2	2
12	1	2	4	7	2	2	1	2	2	2	2	3	1	2	1	1
13	1	2	3	1	2	2	2	3	2	2	1	1	1	4	3	1
14	1	2	1	7	2	2	1	2	2	1	2	3	2	2	1	1
15	1	2	4	7	1	2	1	2	2	2	1	1	3	2	1	1
16	1	1	3	7	2	2	1	1	1	1	2	3	1	1	1	1
17	1	2	4	7	2	2	1	2	1	1	2	3	1	1	1	1
18	1	1	2	1	2	2	1	2	1	1	2	3	2	1	1	1
19	1	1	2	7	1	2	1	1	1	1	2	3	2	1	1	1
20	1	1	2	7	2	2	1	1	1	2	1	1	3	3	1	2
21	1	1	1	7	2	2	1	2	1	2	1	1	1	1	1	1
22	1	1	3	7	2	2	1	2	1	2	1	1	2	2	1	2
23	1	2	5	7	2	2	1	3	1	1	2	3	1	1	1	1
24	1	3	1	1	2	2	1	1	1	1	1	1	2	1	2	1
25	1	1	3	3	2	2	1	2	2	1	1	1	3	1	1	1

26	1	1	3	7	2	2	1	1	1	1	2	3	1	1	1	1
27	1	1	4	7	2	2	1	2	1	1	2	3	1	1	1	1
28	1	1	4	7	2	2	1	2	1	1	2	3	2	1	1	1
29	1	1	3	7	2	2	2	2	1	2	1	2	4	5	2	3
30	1	1	3	5	2	2	2	3	1	2	1	1	2	1	3	3
31	1	1	3	4	2	2	1	4	2	2	1	1	1	4	3	2
32	1	2	3	7	2	2	1	1	1	1	2	3	1	1	1	1
33	1	2	2	7	2	2	1	1	1	1	2	3	2	1	1	1
34	1	2	5	7	2	2	1	1	1	1	2	3	2	1	1	2
35	1	2	1	7	1	2	1	1	1	1	2	3	1	1	1	1
36	1	1	2	7	2	2	1	1	1	2	1	1	3	3	1	2
37	1	1	1	7	2	2	1	2	1	2	1	1	1	1	1	1
38	1	1	3	7	2	2	1	2	1	2	1	1	2	2	1	2
39	1	2	5	7	2	2	1	3	1	1	2	3	1	1	1	1
40	1	3	1	1	2	2	1	1	1	1	1	1	2	1	2	1
41	1	1	3	3	2	2	1	2	2	1	1	1	3	1	1	1
42	1	1	3	7	3	2	1	1	1	1	2	3	2	1	1	1
43	1	2	2	7	2	2	1	3	2	2	1	1	2	3	2	1
44	1	1	3	5	2	2	2	3	2	2	1	2	5	2	2	2
45	1	2	4	7	2	2	1	2	2	2	2	3	1	2	1	1
46	1	2	3	1	2	2	2	3	2	2	1	1	1	4	3	1
47	1	2	1	7	2	2	1	2	2	1	2	3	2	2	1	1
48	1	1	3	7	2	2	1	3	2	1	1	2	5	2	1	3
49	1	1	3	5	2	2	1	3	2	1	1	2	5	2	2	2
50	1	1	1	7	2	2	1	1	1	1	2	3	2	1	1	1
51	1	2	4	7	1	2	1	2	2	2	1	1	3	2	1	1
52	1	1	3	7	2	2	1	1	1	1	2	3	1	1	1	1
53	1	2	4	7	2	2	1	2	1	1	2	3	1	1	1	1
5	1	1	2	1	2	2	1	2	1	1	2	3	2	1	1	1
55	1	1	2	7	1	2	1	1	1	1	2	3	2	1	1	1
56	1	1	3	7	2	2	1	1	1	1	2	3	1	1	1	1
57	1	1	4	7	2	2	1	2	1	1	2	3	1	1	1	1
58	1	1	4	7	2	2	1	2	1	1	2	3	2	1	1	1
59	1	1	3	7	2	2	2	2	1	2	1	2	4	5	2	3
60	1	1	3	5	2	2	2	3	1	2	1	1	2	1	3	3
61	1	2	3	7	2	2	1	1	1	1	2	3	1	1	1	1
62	1	2	2	7	2	2	1	1	1	1	2	3	2	1	1	1
63	1	2	5	7	2	2	1	1	1	1	2	3	2	1	1	2

64	1	2	1	7	1	2	1	1	1	1	2	3	1	1	1	1
65	1	1	3	7	3	2	1	1	1	1	2	3	2	1	1	1
66	1	2	2	7	2	2	1	3	2	2	1	1	2	3	2	1
67	1	1	3	5	2	2	2	3	2	2	1	2	5	2	2	2
68	1	2	4	7	2	2	1	2	2	2	2	3	1	2	1	1
69	1	2	4	7	2	2	1	2	1	1	2	3	1	1	1	1
70	1	1	2	1	2	2	1	2	1	1	2	3	2	1	1	1
71	1	1	2	7	1	2	1	1	1	1	2	3	2	1	1	1
72	1	1	2	7	2	2	1	1	1	2	1	1	3	3	1	2
73	1	1	1	7	2	2	1	2	1	2	1	1	1	1	1	1
74	1	1	3	7	2	2	1	2	1	2	1	1	2	2	1	2
75	1	2	5	7	2	2	1	3	1	1	2	3	1	1	1	1
76	1	3	1	1	2	2	1	1	1	1	1	1	2	1	2	1
77	1	2	3	1	2	2	2	3	2	2	1	1	1	4	3	1
78	1	2	1	7	2	2	1	2	2	1	2	3	2	2	1	1
79	1	2	4	7	1	2	1	2	2	2	1	1	3	2	1	1
80	1	1	3	7	2	2	1	1	1	1	2	3	1	1	1	1
81	1	1	3	7	2	2	1	3	2	1	1	2	5	2	1	3
82	1	1	3	5	2	2	1	3	2	1	1	2	5	2	2	2
83	1	1	1	7	2	2	1	1	1	1	2	3	2	1	1	1
84	1	1	3	4	2	2	1	4	2	2	1	1	1	4	3	2

KEY TO MASTER CHART

M- 1 :F-2

20-39 YRS-1; 40-59- 2;>60 YRS -3

Abd pain -1: A.P + DISTENSION -2 ; A.P + OBSTIPATION-3 :A.P +N+VOMOITING -4 :A.P + FEVER -5: A.P+obs+ DYSPONEA – 6

D-M-1 ;IHD-2;GALL-3;SHT- 4; T.G-5;T.B-6 nil -7

ALCOH -1 : A LCHOLOL +SMOKING-2: ALCOHOL +TOBACO-3

<24 U/WEEK-1: >24U/WEEK-2

<3- 1: >3-2

0-1:1-2: 2-3: 3-4

1-2 -1 ;;>3 -2

<2- 1; >2-2

YES-1; NO-2

<1WEEK -1 ;>1 WEEK -2;NIL- -3

1WEEK- 1;2 WEEK-2 3WEEK-3.4WEEKS-4>4 WEEKS-5

NO-1; LOCAL-2; SYSTEMIC-3;DEATH-4;LOCAL +SYSTEMIC-5;

<4POINTS-1;5-9- 2 ;10-14-3;15-19- 4

0-3 -1 ; 4-6- 2 ; 7-10-3

ANNEXURES

PATIENT CONSENT FORM

STUDY TITLE:

"PROSPECTIVE STUDY OF CLINICAL OUTCOME IN ALCOHOLIC ACUTE PANCREATITIS"IN GMKMCH, SALEM.

Department of General surgery, GMKMCH

PARTICIPANT NAME : AGE : SEX:

I.P. NO :

I confirm that I have understood the purpose of surgical/invasive procedure for the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.
I have been explained about the possible complications that may occur during and after medical procedure. I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.
I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.
I hereby consent to participate in this study.
Time :

Date : Signature / Thumb Impression Of Patient

Place :

Patient's name:

Signature of the investigator: _____

Name of the investigator : _____