A Thesis in General Surgery

A PROSPECTIVE STUDY ON NEUTROPHIL LYMPHOCYTE RATIO AND PLATELET LYMPHOCYTE RATIO AS EARLY PREDICTOR OF NECROSIS IN ACUTE PANCREATITIS

Submitted in partial fulfillment of the requirements for the

DEGREE OF M.S GENERAL SURGERY (BRANCH I)

KILPAUK MEDICAL COLLEGE & HOSPITAL,

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

MAY 2020

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation titled "**A PROSPECTIVE STUDY ON NEUTROPHIL LYMPHOCYTE RATIO AND PLATELET LYMPHOCYTE RATIO AS EARLY PREDICTOR OF NECROSIS IN ACUTE PANCREATITIS"** is a bonafide and genuine research work carried out by me under the guidance of **Prof. Captain. Dr. S. Nedunchezhian, M.S., D.Ortho., MCA.,** Professor, Department of General Surgery, Kilpauk Medical College,Chennai.This dissertation is submitted to The Tamilnadu Dr.M.G.R. MEDICAL UNIVERSITY, CHENNAI in partial fulfillment of the requirements for the degree of M.S. General Surgery examination to be held in May 2020.

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This is to certify that this dissertation work titled **"A PROSPECTIVE STUDY ON NEUTROPHIL LYMPHOCYTE RATIO AND PLATELET LYMPHOCYTE RATIO AS EARLY PREDICTOR OF NECROSIS IN ACUTE PANCREATITIS**" of the candidate **Dr. M. Mohamed Ibrahim** with **Registration Number 221711156** for the award of Masters in Surgery in the branch of Branch I- General Surgery. I personally verified the www.urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **17%** of plagiarism in the dissertation.

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Finally, I would like to thank God, my parents, sister and my niece for their unconditional love and support in my journey towards becoming a surgeon.

TABLE OF CONTENTS

INTRODUCTION

Acute pancreatitis (AP) is an inflammatory disease of the pancreas with an rapidincrease over the past 30years. At present, AP results more than 270,000 hospital admissions per year in the United Kingdom, which is more than any other GI-related cause of hospitalization. This leads to a high economic burden, exceeding 2.5billion dollars annually in the United States.

Acute pancreatitis is adynamic inflammatory process that starts with local acinar cell injury with unpredictable involvement of other nearby tissues or remote organ systems. Though the large amount of acute pancreatitis cases are mild and self-limiting, severe cases can be associated with complications such as necrosis or organ failure in approximately 15- 20% of patients. In such severe acute pancreatitis (SAP), high mortality rates of up to 70% have been recorded..

The severity of acute pancreatitis depends on systemic organ failure secondary to the systemic inflammatory response of the patient, and a poor prognosis of SAP is thought to be the result of uncontrolled systemic inflammatory response syndrome or multi-organ dysfunction syndrome. White blood cell (WBC) counts and C-reactive protein (CRP) levels are recent markers associated with systemic inflammation that can be measured using routine haematological tests. In addition, the WBC count is correlated with

worse prognosis as part of Ranson's criteria, Glasgow score, Acute Physiology and Chronic Health Evaluation-II (APACHE II), and Bedside Index of Severity in Acute Pancreatitis (BISAP). However, the total WBC count can be changed based on various physiological and pathological conditions including hydration status, stress, and pregnancy. Changes in peripheral blood components are been used to predict the prognosis of many diseases, such as coronary heart disease, esophageal cancer, colorectal cancer, and hepatocellular carcinoma. Neutrophil-lymphocyte ratio (NLR) and platelet Lymphocyte ratio (PLR) are new markers used to this, on which there are several studies available in the literature.

These markers are especially thought to show inflammation response of the patient. Now, it has been shown that PLR-NLR combination could be used to predict disease prognosis as well. Although we have found past studies showing NLR and PLR usage to predict prognosis of acute pancreatitis, there is not a single study that compares these markers and necrosis prediction in acute pancreatitis.

So, in this study, we aimed to investigate the prognostic importance of PLR-NLR combination for patients diagnosed with acute pancreatitis and its relationship with necrosis.PLR and NLR reflects the immune response better than that of total WBC count. Past studies have shown the correlation ship between peripheral lymphocytopenia and the severity of acute pancreatitis. In addition, one study established the superiority of the PLR over NLR.

AIM AND OBJECTIVES

The aim of my study is to calculate the Neutrophil-Lymphocyte Ratio (NLR) and Platelet- Lymphocyte Ratio (PLR) among acute pancreatitis patients and to investigate if this ratio is helpful as early predictor of necrosis in acute pancreatitis.

REVIEW OF LITERATURE

HISTORICAL BACKGROUND:

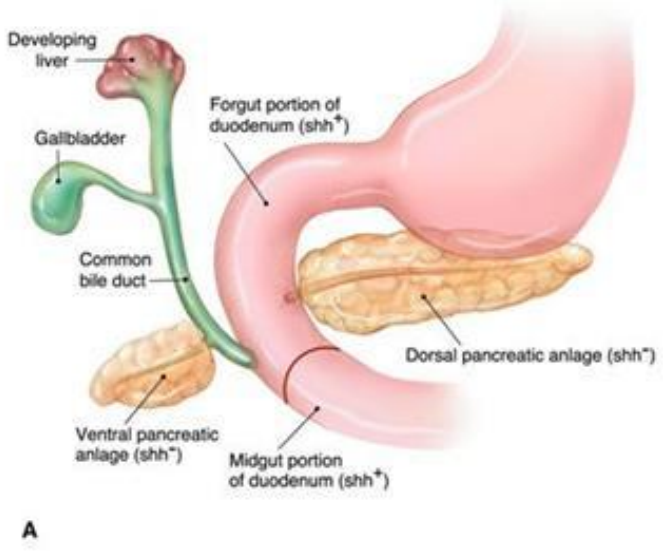
The pancreas is one of the last organs in the abdomen to be studied by anatomists, physiologists, physicians, and surgeons. History dates back to the Rabbinic Judaism (Babylonian Talmud) which describes pancreas as the "finger of liver". The word pancreas is derived from a Greek concept of pan kreas(meaning "all flesh") based on the hypothesis by Hippocrates that all glandular structures were composed of flesh.[1] Vesalius was the first initiated the formal structural elucidation of pancreas. The formal structure of pancreas was first quoted by Vesalius. The physiologic function of pancreas was defined by R. de Graaf. The association of diabetes mellitus with pancreas was identified by O. Minkowski. With regard to the digestive property of pancreas, fat digestion was described by J. Purkinje and role of trypsin in proteolysis by W. Kuhne. NicholaesTulp from Amsterdam was the first to describe acute pancreatitis in 1652. However, Guy Patin from Paris made a similar observation, but published a decade later.[2]

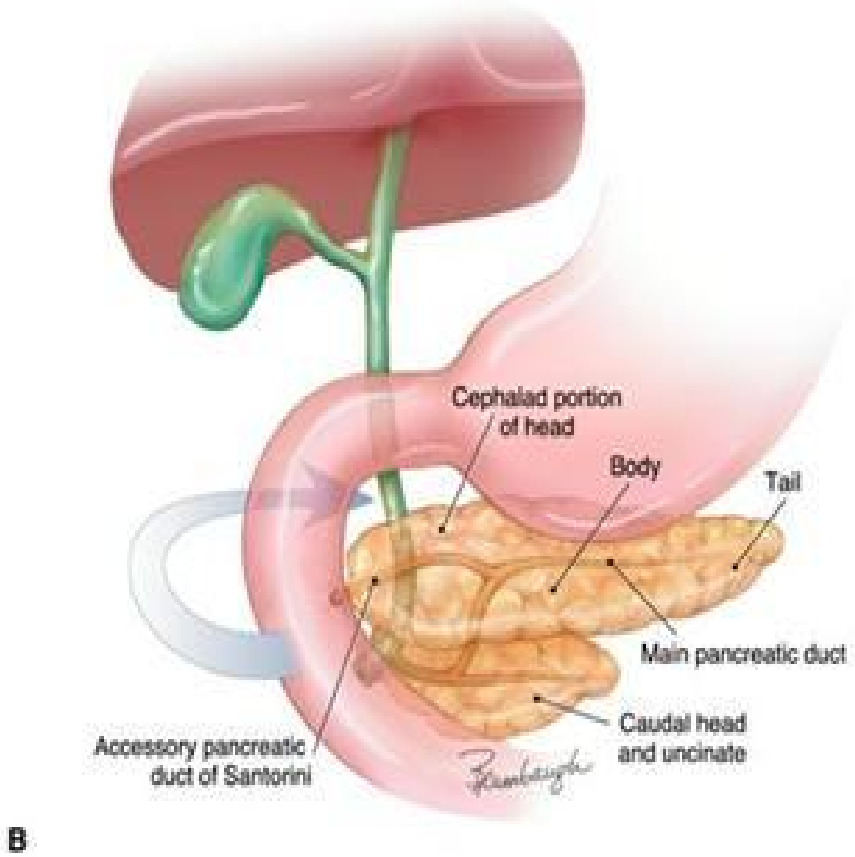
[1]Busnardo et al., 'History of the Pancreas'.

PANCREAS

EMBRYOLOGY

The pancreas develops from two outgrowths of the foregut distal to the stomach. The ventral diverticulum gives rise to the common bile duct, gallbladder, liver and the ventral pancreatic anlage that becomes a part of the head of the pancreas and the uncinate process and its ductal system. The dorsal pancreatic anlage gives rise to a part of the head, the body, and tail of the pancreas including a major duct that is continuous through the three regions. Fusion of the duct systems results in the formation of the main pancreatic duct from the ducts of dorsal and ventral anlagen. The caudal part of the head of the pancreas (uncinate) and the major papilla (ampulla of Vater) are derived from the ventral bud. The minor papilla that drains the duct of Santorini is derived from the dorsal bud.

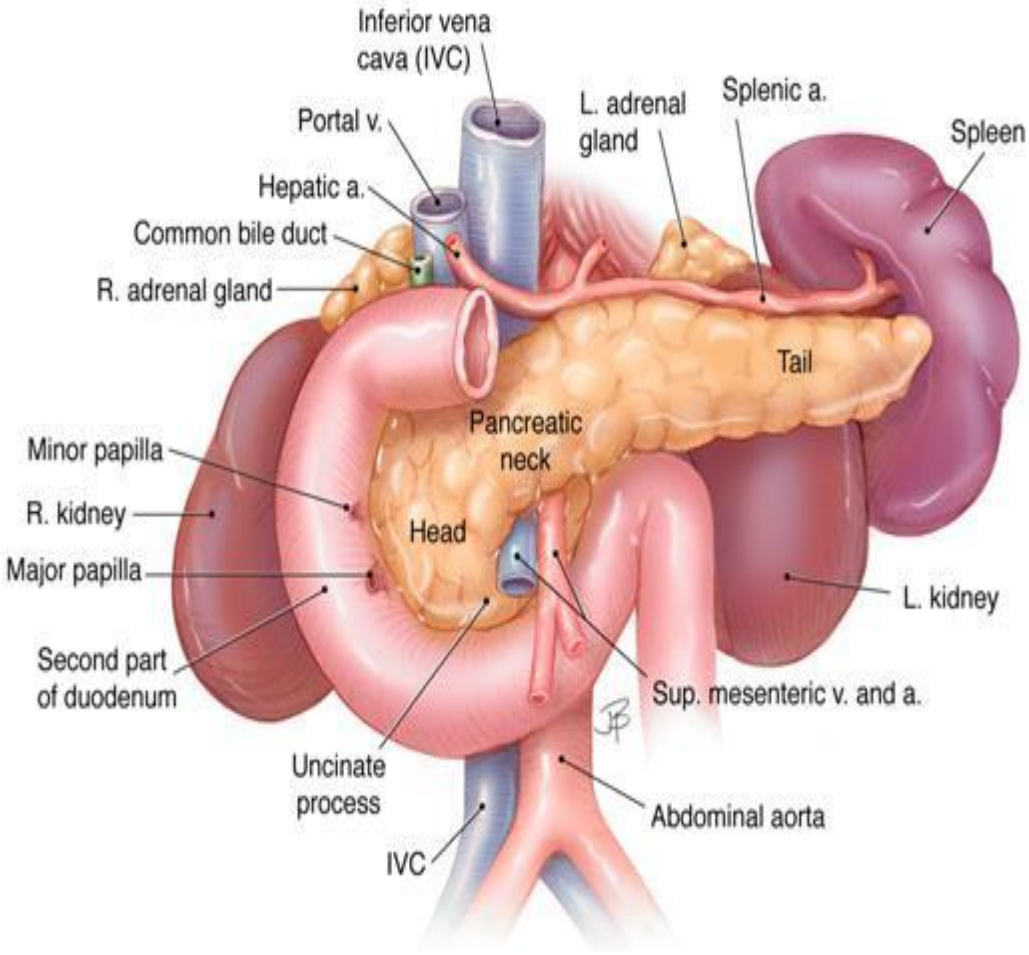




Anatomic relationships of the pancreas with surrounding organs and structures.

- The head of the pancreas lies within in the loop of the duodenum
- The tail of the pancreas lies near the hilum of the spleen.
- The body of the pancreas lies posterior relation to the distal portion of the stomach between the tail and the neck.

• The portion of the pancreas that lies anterior to the aorta is somewhat thinner than the adjacent portions of the head and body of the pancreas. This region is sometimes called as the neck of the pancreas and marks the junction between head and body.



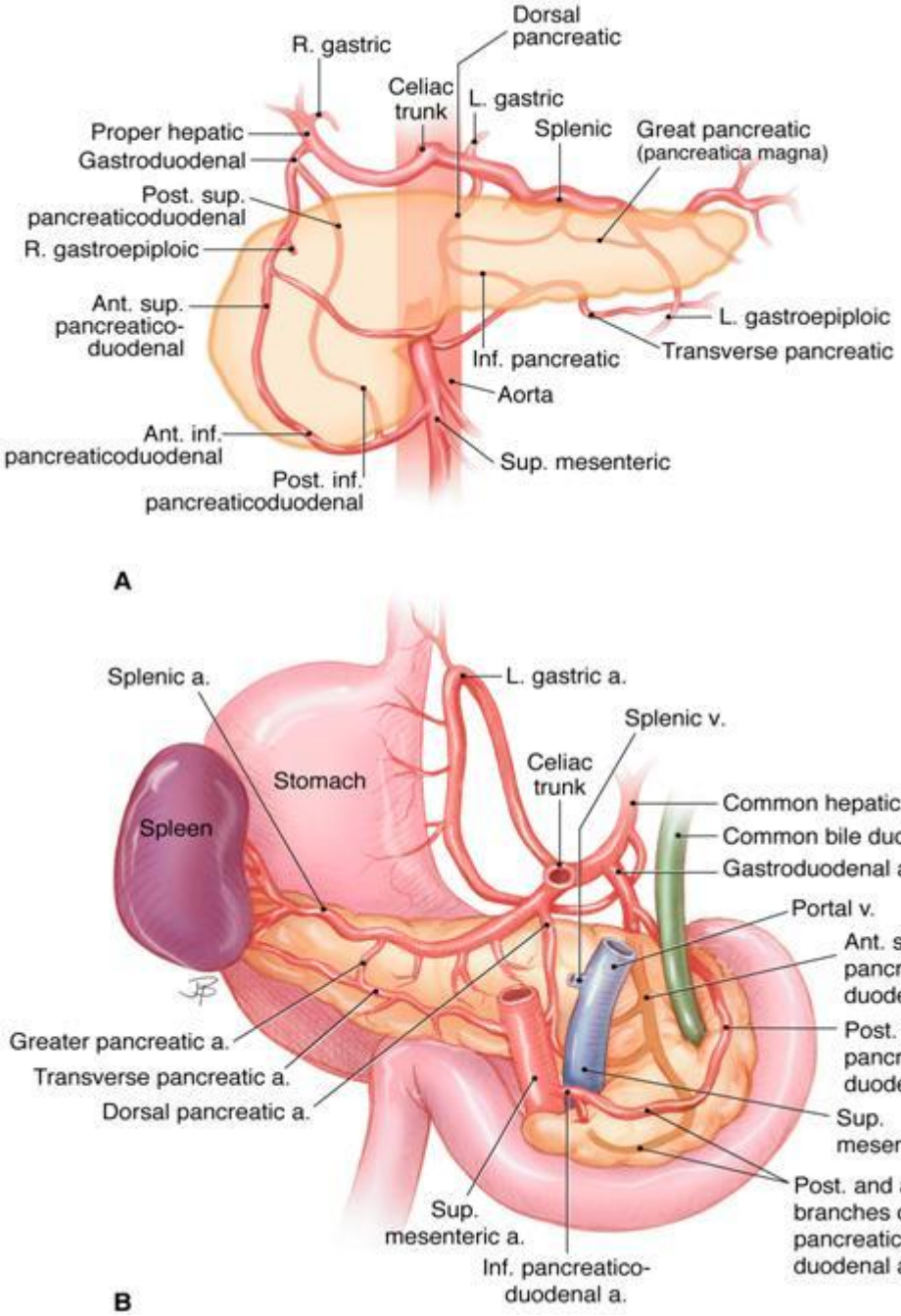
- The neck of the pancreas lies in a close proximity to major blood vessels posteriorly including the superior mesenteric artery, superior mesenteric vein, portal vein, inferior vena cava, and aorta.

- The common bile duct passes through the head of the pancreas to join the main duct of the pancreas near the duodenum.

•The minor papilla where the accessory pancreatic duct drains into the duodenum and the major papilla (ampulla of Vater) where the main pancreatic duct enters the duodenum.[2]

[2]Longnecker, 'Anatomy and Histology of the Pancreas'.

BLOOD SUPPLY



The celiac trunk and the superior mesenteric artery both arise from the abdominal aorta. Both have multiple branches that supply several organs including the pancreas. The anastomosis of their branches around the pancreas provides collateral circulation that generally assures a secure arterial supply to the pancreas[3]. Most of the arteries are accompanied by veins that drain into the portal and splenic veins as they pass behind the pancreas. The superior mesenteric vein becomes the portal vein when it joins the splenic vein.

Function

The exocrine tissues secrete a clear, watery, alkaline juice that contains several enzymes. These break down food into small molecules that can be absorbed by the intestines.

THE ENZYMES INCLUDE:

- Trypsin and chymotrypsin to digest proteins
- Amylase to break down carbohydrates
- Lipase, to break down fats into fatty acids and cholesterol

[3]Covantev, Mazuruc, and Belic, 'The Arterial Supply of the Distal Part of the Pancreas'.

The endocrine portion, or islets of Langerhans, secrete insulin and other hormones.

Pancreatic beta cells release insulin when blood sugar levels rise.

INSULIN:

- moves glucose from the blood into muscles and other tissues, for use as energy

- helps the liver absorb glucose, storing it as glycogen in case the body needs energy during stress or exercise

When blood sugar falls, pancreatic alpha cells release the hormone glucagon.

Glucagon causes glycogen to be broken down into glucose in the liver.

The glucose then enters the bloodstream, restoring blood sugar levels to normal.[4]

[4] 'Pancreas'.

ACUTE PANCREATITIS

Acute pancreatitis is a most common clinical condition seen in surgical practice. Acute pancreatitis is a common cause of the "acute abdomen". Aetiology, however, varies from country to country. Sex incidence is approximately equal, but gallstones are more common in females and alcoholism being aetiology is more common in males. Currently, AP results in 270,000 hospital admissions per year in the United States[5], which is more than any other GI-related cause of hospitalization. This leads to a high economic burden exceeding 2.5 billion dollars annually in the United States alone[6].Though the volume of cases is hugh, acute pancreatitis has a great challenge to the treating physician.

Acute pancreatitis is an inflammation of the pancreatic tissue secondary to acinar cell necrosis. It occurs due to auto digestion by pancreatic enzymes[7].Most patients develop a mild and a self-limited course, however 10%-20% of patients have a rapidly progressive course with prolonged length of hospital stay and significant morbidity and mortality. Mild pancreatitis is associated with a mortality rate of less than 1% but, it increases up to 10%-30% in severe pancreatitis.[8]Inspite of treatments, acute pancreatitis leads to high morbidity, mortality and complications.

[5]Fagenholz et al., 'Increasing United States Hospital Admissions for Acute Pancreatitis, 1988-2003'.
[6]Fagenholz et al.
[7]'The Role of Ca2+ in the Pathophysiology of Pancreatitis'.

Most deaths occur in one of two settings

1: During the initial period of hypovolemic shock

2: After > 2 weeks of septic illness that leads to multiorgan failure in those who have infected pancreatic necrosis.[8]

Hence, determination of its prognosis is of vital importance. Several scoring systems such as Ransonscore , Atlanta classification 9, acute physiology and chronic health evaluation (APACHE)-2, the bedside index for severity in acute pancreatitis (BISAP) (6), and laboratory parameters such as C-reactive protein (CRP) are used for this purpose. Inspite of all these scoring systems and laboratory parameters, it may still be difficult to determine its prognosis.

Aetiology and pathology

Gall stones and Alcohol consumption are the leading causes among a multitude of reported factors. In several western countries including UK, gall stones account for one-half to two – thirds of attacks of acute pancreatitis[10]. Acute biliary pancreatitis is more common in those individuals with small gallstones and a long common pancreaticobiliary channel. Lodgement of stone

[8]Garg et al., 'Association of Extent and Infection of Pancreatic Necrosis with Organ Failure and Death in Acute Necrotizing Pancreatitis'.
[9]Banks et al., 'Classification of Acute Pancreatitis--2012'.
[10]Yadav and Lowenfels, 'The Epidemiology of Pancreatitis and Pancreatic Cancer'.

at the papilla, so allowing reflux of bile along the pancreatic duct, is the likely pathogenesis

- Alcohol consumption can be precipitated by an alcohol binge, nearly most of them have history chronic alcohol abuse. Pathogenesis may be due to pancreatic hypersecretion, direct cellular toxicity, disturbed microcirculation in pancreas.[11]

- Post-operative pancreatitis- following procedures like ERCP and surgical procedure in vicinity of papilla- cause papillary oedema or ductal overdistension. Amylase level is increased after ERCP, but clinical pancreatitis accounts for 2-4% only.[12]

- Acute fulminating pancreatitis following low cardiac output state

[11]Chowdhury and Gupta, 'Pathophysiology of Alcoholic Pancreatitis'.
[12]Thaker, Mosko, and Berzin, 'Post-Endoscopic Retrograde Cholangiopancreatography Pancreatitis'.

METABOLIC CAUSES

- Hypertriglyceridemia -Patients with types I and V hyperlipoproteinemia can experience episodes of abdominal pain, and these often occur in association with marked hypertriglyceridemia. TGL level >1000 is often associated with episodes of acute pancreatitis.

- Hypercalcemia

- Hypothermia

- Hyperparathyroidism

Drugs

Certain drugs are known to be capable of causing acute pancreatitis. These include the thiazide diuretics, furosemide, oestrogens, azathioprine,l-asparaginase,6-mercaptopurine,methyldopa, sulphonamides, tetracycline, pentamidine, procainamide, nitrofurantoin, dideoxyinosine, valproic acid, and acetylcholinesterase inhibitors. In addition, lipid-based intravenous drugs and solutions, such as propofol, can also cause acute pancreatitis. A history of verified or suspected drug-induced pancreatitis should serve as a contraindication to prescribing that medication again.[13]

[13]Bellocchi, Campagnola, and Frulloni, 'Drug-Induced Acute Pancreatitis'.

Infection

- Viral- Mumps, Coxsackie A, HIV, CMV
- Bacterial - Mycobacterium tuberculosis, salmonella enteritis
- Fungal - Mycoplasma
- Round worm in pancreaticobiliary tree.

Uncommon Causes

• Vascular causes and vasculitis (Ischemic-hypoperfusion states after cardiac surgery)

• Connective tissue disorders and thrombotic thrombocytopenic purpura (TTP)

• Cancer of the pancreas

• Periampullary diverticulum

- Duodenal diverticula,
- Annular pancreas,
- Choledochocele

Hereditary Pancreatitis

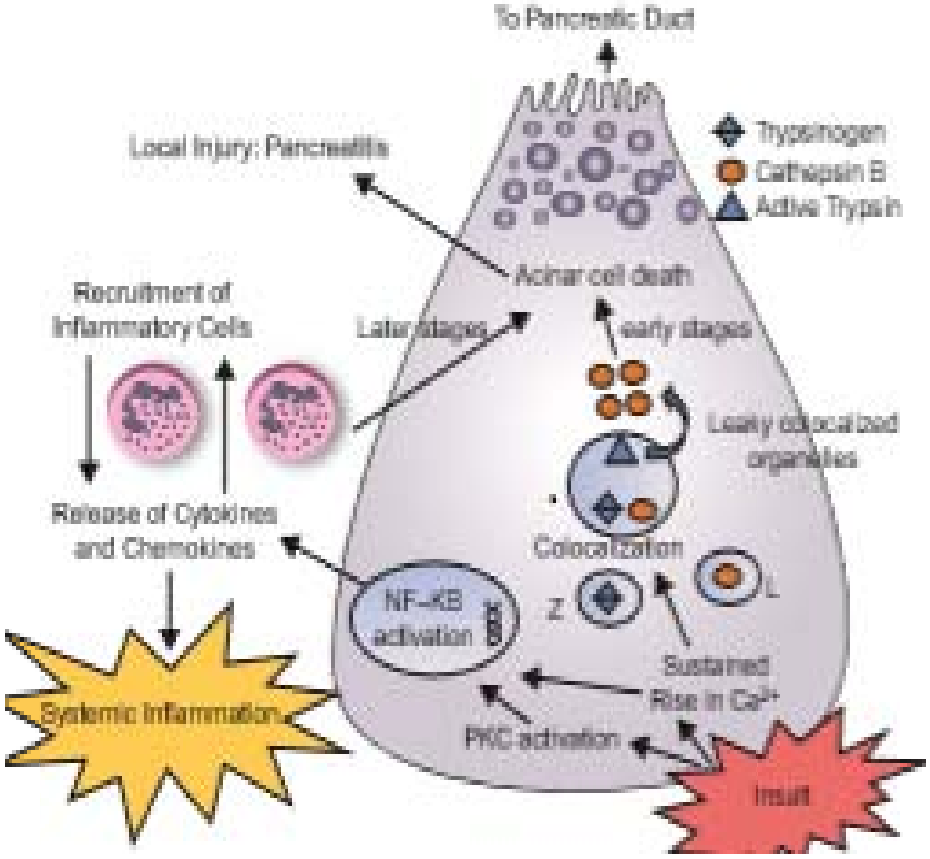
Hereditary pancreatitis is an autosomal dominant disorder associated with mutations related to cationic trypsinogen gene (PRSS1). PRSS1 mutations cause premature activation of trypsinogen to trypsin and cause abnormal of ductal secretion, which promotes acute pancreatitis. Mutations in the SPINK1 protein, which blocks the active binding site of trypsin, is also likely to have a role in predisposing to acute pancreatitis. Variations in penetration and phenotype are common and there are many other mutations that may become implicated. Mutant enzymes activated within acinar cells can overwhelm the first line of defence (pancreatic secretory trypsin inhibitor) and resist backup defences (e.g., proteolytic degradation, enzyme Y, and trypsin itself) allowing activated mutant cationic trypsin to trigger the entire zymogen activation cascade.

PATHOBIOLOGY OF THE ACINAR CELL IN ACUTE PANCREATITIS

INFLAMMATORY SIGNALLING OF PANCREATITIS

The prime event initiating the disease process is the excessive release of Ca^{2+} from intracellular stores, followed by excessive entry of Ca^{2+} from the interstitial fluid. However, Ca^{2+} release and subsequent entry are also the processes that control the physiological secretion of digestive enzymes in response to stimulation via the vagal nerve or the hormone cholecystokinin[14]. Inflammation is the hallmark of AP and the inflammatory response begins in the acinar cell. Most of the cases, the acute inflammatory response is limited to the pancreas, but in severe cases there can be progression to a systemic inflammatory response syndrome (SIRS) causing organ failure which can lead to mortality. SIRS is mediated by pancreas-generated increased[15] levels of circulating cytokines that affect several organs especially the lungs leading to ARDS.

[14]'The Role of Ca2+ in the Pathophysiology of Pancreatitis'.
[15]Han and Logsdon, 'CCK Stimulates Mob-1 Expression and NF-KappaB Activation via Protein Kinase C and Intracellular Ca(2+)'.



When acinar cells are pathologically stimulated, their lysosomal (L) and zymogen (Z) contents colocalize, then trypsinogen is activated to trypsin by cathepsin B. There is an increase in cytosolic calcium. Once trypsin has permeabilized the contents of the cytosol, cathepsin B [16] and other contents of these colocalized organelles are released. Once in the cytosol, cathepsin B activates apoptosis by causing cytochrome c to be released from the mitochondria. Activation of PKC results in a sudden activation of nuclear factor kappa beta (NFκβ) which in turn triggers the release of cytokines that

[16]Lerch and Halangk, 'Human Pancreatitis and the Role of Cathepsin B'.

attract inflammatory response cells which mediate local and systemic inflammation cascades[17]

The studies that show that the acinar cell is the initial site of inflammatory signalling come from experiments that show that this cell produces a variety of inflammatory mediators with stressors that cause pancreatitis. These mediators are then involved in the recruitment of neutrophils followed by macrophages, monocytes, and lymphocytes into the pancreas. Importantly, infiltrating inflammatory cells (both neutrophils and macrophages) mediate the pathologic, intra-acinar activation of trypsinogen which is involved in the promotion of the acinar cell injury and is a key feature of pancreatitis.

Furthermore, the inflammatory cell infiltrate exacerbates pancreatic necrosis. Although all the mechanisms for promotion of necrosis are not elucidated, another feature of inflammation is that it shifts apoptosis–necrosis balance of acinar cell death towards necrosis of the parenchymal tissue which is associated with a greater severity of disease. The severity of pancreatitis in experimental models improves with various strategies that inhibit inflammatory cells recruitment including neutralizing antibodies. Genetic deletion of specific integrins or inhibition of complement.

[17]Han and Logsdon, 'CCK Stimulates Mob-1 Expression and NF-KappaB Activation via Protein Kinase C and Intracellular Ca(2+)'.

Although the exact mechanisms involved in initiating inflammatory signalling in the acinar cell are not completely understood, there are key transcription factors that are involved which are generally known to regulate inflammatory mediators. These include nuclear factor kappa-B, activator protein-1(AP-1), and nuclear factor of activated T-cells(NFAT).

These transcription factors are, in turn, regulated by upstream intracellular signalling systems that include [Ca^{2+}], calcineurin, novel isoforms of protein kinase them, the studies cited show that in animal models and invitrostudies using acinar cells, the inhibition of the pathways leads to attenuation of the severity of pancreatitis(and cellular injury) pointing to the central role played by the acinar cell and its inflammatory signalling in pancreatitis.

The pancreatic microcirculation

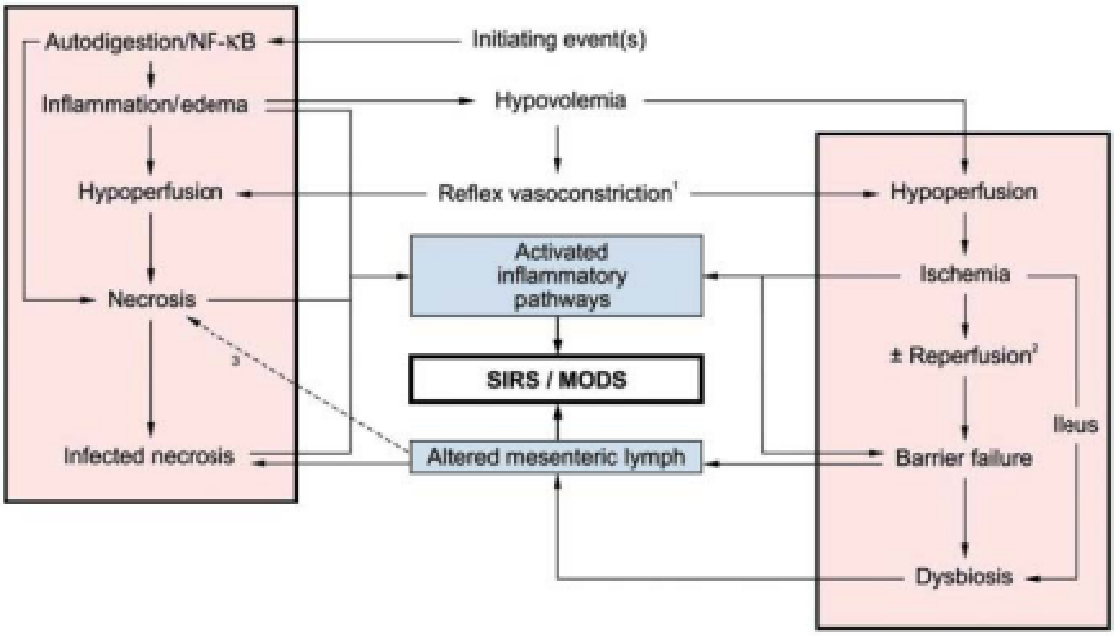
Evidence suggests that disrupted perfusion of the pancreatic microcirculation is an important factor in the transition from mild interstitial oedematous pancreatitis to severe necrotizing pancreatitis[18]. Several causes are implicated in disrupting the pancreatic microcirculation in AP including hypovolemia, increased capillary permeability, hypercoagulability with microthrombi, and

[18]Lewis, Reber, and Ashley, 'Pancreatic Blood Flow and Its Role in the Pathophysiology of Pancreatitis'.

endothelial damage from oxidative free radicals[19]. Regardless of the underlying pathophysiologic aetiology, these disruptions increase the degree of pancreatic ischemia, the release of cytokines and inflammatory mediators, and local vasodilatation and vascular permeability. This can lead to the systemic inflammatory response syndrome (SIRS) and multiorgan failure and increase the risk for severe AP with pancreatic necrosis

[19]Plusczyk et al., 'ET A and ET B Receptor Function in Pancreatitis-Associated Microcirculatory Failure, Inflammation, and Parenchymal Injury'.

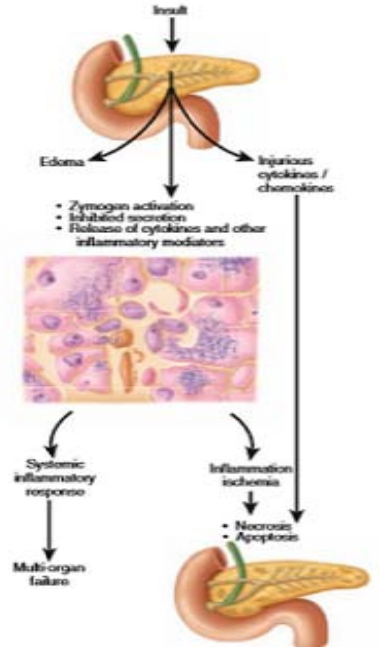
Complex interactions between the pancreas and the intestine in the pathogenesis of severe acute pancreatitis



The pathophysiological events in these organs and the complex interplay between them are important in driving the disease course. These events include activation of the inflammatory pathways,microcirculatory failure, alterations in the microbiome, and breakdown of the intestinal barrier.[20]

[20]Kinnala et al., 'Splanchnic and Pancreatic Tissue Perfusion in Experimental Acute Pancreatitis'.

Events of acute pancreatitis



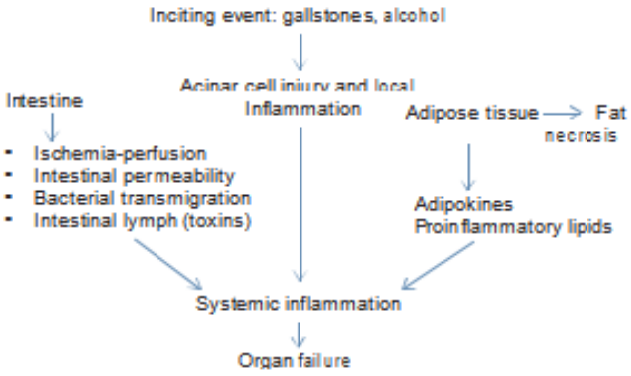


Figure 2C.3 Drivers of systemic inflammation in acute pancreatitis: apart from the pancreas itself, intestine and adipose tissue play major roles in the development of MODS.

Diagnosis and Classification of acute pancreatitis.[21]

The diagnosis of Acute Pancreatitis is established when two out of the three following criteria are present:

(i) pancreatic-type abdominal pain, upper abdominal

(ii) elevated serum amylase[22]and/or lipase more than three times the upper limit of normal, and/or

(iii)imaging findings consistent with Acute pancreatitis.[23]

The diagnosis of AP should be considered when patients present with acute onset, severe, upper abdominal pain that often radiates to the back, and is associated with nausea and vomiting. Physical examination reveals epigastric tenderness but usually without peritoneal signs.

In patients with symptoms typical for Acute pancreatitis, the measurement of elevated serum pancreatic enzymes (amylase and/or lipase) three times the upper limit of normal can confirm the diagnosis of Acute pancreatitis[24]. Studies have shown that the three fold elevation criteria are associated with a moderate sensitivity (55–100%) and a high specificity (93–99%) and that this is more accurate than lowercut- off values.

[21]Banks et al., 'Classification of Acute Pancreatitis--2012'.
[22]Winslet et al., 'Relation of Diagnostic Serum Amylase Levels to Aetiology and Severity of Acute Pancreatitis'.
[23]Chatila, Bilal, and Guturu, 'Evaluation and Management of Acute Pancreatitis'.
[24]Winslet et al., 'Relation of Diagnostic Serum Amylase Levels to Aetiology and Severity of Acute Pancreatitis'.

Revised Atlanta classification

The revised Atlanta classification defines three grades of severity.

- **Severe AP** is defined by the presence of persistent organ failure,
- **moderate severity** by transient organ failure (less than48 hours), local complications, including infected pancreatic necrosis, and/or exacerbation of existing comorbidities, and
- **mild severity** when the aforementioned features are absent.[25]

Table 7.1 Collections as defined in the revised Atlanta classification.

Definition	Description
Acute fluid collection (less than 4 weeks after onset)	• Homogenous fluid density • Confined by normal peripancreatic fascial planes • No definable wall encapsulating the collection • Adjacent to pancreas (not intrapancreatic)
Pseudocyst (usually more than 4 weeks after onset)	• Well circumscribed, usually round/oval • Homogenous fluid density • Well-defined wall and completely encapsulated • Adjacent to pancreas (not intrapancreatic)
Acute necrotic collection (less than 4 weeks after onset)	• Heterogeneous and nonliquid density • No definable wall encapsulating the collection • Location: intrapancreatic and/or extrapancreatic
Walled-off necrosis (usually more than 4 weeks after onset)	• Heterogeneous and nonliquid density • Well-defined wall and completely encapsulated • Location: intrapancreatic and/or extrapancreatic

[25]Foster et al., 'Revised Atlanta Classification for Acute Pancreatitis'.

Determinant-based classification of acute pancreatitis.[26]

The determinant-based classification defines four severity categories based on local and systemic complications.

- **Critical** is defined by the presence of both infected pancreatic necrosis and persistent organ failure,
- **Severe** by infected pancreatic necrosis or persistent organ failure,
- **Moderate** by sterile pancreatic necrosis and / or transient organ failure, and
- **Mild** – rest of the cases.[27]

[26]Dellinger et al., 'Determinant-Based Classification of Acute Pancreatitis Severity'.
[27]Acevedo-Piedra et al., 'Validation of the Determinant-Based Classification and Revision of the Atlanta Classification Systems for Acute Pancreatitis'.

Atlanta1992	MildAP No	SevereAP Yes		
Local[a]complications				
		And/or		
Organfailure[b]	No	Yes		
		And/or		
APACHE-II≥8orRanson's≥3	No	Yes		
RAC	MildAP No	ModeratelysevereAP Yes	SevereAP	
Local[c]orcomorbidities[d]				
		And/or		
Organfailure[e]	No	Transient	Persistent	
DBC	MildAP	ModerateAP	SevereAP	CriticalAP
(Peri)pancreaticnecrosis	No	Sterile	Infected	Infected
	And	And/or	Or	And
Organfailure[f]	No	Transient	Persistent	Persistent

Scores	Year	Cutoff	Variablesassessedatadmissionand48hours
Ranson's	1974	3	Admission:age(>55y),WBC(>16,000/mL),glucose(>200mg/dL),LDH (>350IU/mL),AST(>250IU/mL)48hours:hematocrit (decrease>10%), BUN(increase>5mg/dL),calcium(<8mg/dL),PaO_2 (<60mmHg),base deficit(>4mEq/L),fluidsequestration(>6L)
Glasgow	1984	2	Age(>55y),WBC(>15,000/mL),glucose(>180mg/dL),BUN(>45mg/dL), PaO_2 (<60mmHg),calcium(<8g/dL),albumin(<3.2g/dL),LDH(>600IU/L)
APACHE-II	1989	8	Age,temperature,MAP,heartrate,respiratoryrate,A-aPaO_2orPaO_2, arterialpHorHCO_3,sodium,potassium, creatinine, hematocrit,WBC,
SIRS	2006	2	Temperature(<36°Cor>38°C),heartrate(>90/min),respiratoryrate (>20/minor$PaCO_2$ <32mmHg),WBC(<4000/mm^3,>12,000/mm^3 or >10% bands)
Panc3	2007	1	Hematocrit(>44%),BMI(>30kg/m^2), pleuraleffusion
POP	2007	9	Age,MAP,PaO_2:FiO_2,arterialpH,BUN,calcium[a]
BISAP	2008	2	BUN(>25mg/dL),impairedmentalstatus(GlasgowComaScore<15),SIRS (≥2),age(>60y),pleuraleffusion
JSS	2009	2	Baseexcess(≤3mEq/L),PaO_2 (≤60mmHgorrespiratoryfailure),BUN (≥40mg/dL)orCr(≥2mg/dL),LDH(≥2×upperlimitofnormal),platelet (≤100,000/mm^3), calcium(≤7.5mg/dL),CRP(≥15mg/dL),SIRS(≥3),age (≥70y)
HAPS	2009	1	Abdominaltenderness,hematocrit (>43%formenor>39.6%forwomen), creatinine(>2mg/dL)

Ranson'swas the first to use clinical criteria to predict Acute pancreatitis severity, and they have been widely used in clinical practice and research for four decades. The Ranson's criteria comprise11 variables that are scored at 2 time points on admission and within48hours.A score of 3 or more is required for predicted severe Acute pancreatitis and is usually associated with a worse outcome. Since the development of Ranson's score, several additional clinical scores for predicting severity have been developed[28]. They incorporate clinical, laboratory, and occasionally radiographic findings and include in chronological order

- the Glasgow criteria (also known as Imrie score),
- the acute physiology and chronic health examination (APACHE) II score,
- the systemic inflammatory response syndrome (SIRS) score,
- Panc 3 score,
- the pancreatitis outcome prediction (POP) score,
- the bedside index for severity in acute pancreatitis (BISAP)score,
- the revised Japanese severity score (JSS), and
- the harmless acute pancreatitis score (HAPS).[29]

A recent large study that head-to-head compared all available clinical scores in a large cohort of prospectively enrolled Acute pancreatitis patients and

[28]Mounzer et al., 'Comparison of Existing Clinical Scoring Systems to Predict Persistent Organ Failure in Patients with Acute Pancreatitis'.
[29]'Comparison of Ranson, Glasgow, MOSS, SIRS, BISAP, APACHE-II, CTSI Scores, IL-6, CRP, and Procalcitonin in Predicting Severity, Organ Failure, Pancreatic Necrosis, and Mortality in Acute Pancreatitis'.

subsequently validated the results in an independent cohort showed that all perform with moderate accuracy (around 80%) and are comparable in predicting severe disease[27] .One major limitations of the available scoring systems is that they mainly convert continuous into binary values of equal weight and thus fail to capture synergistic effects based on the interactions of interdependent systems . It appears that the current clinical predictive scores have reachedtheir maximum efficacy, and novel approaches for severity prediction are needed. Pancreatic societies and expert recommendations have proposed **SIRS**[30]as an easy-to- remember and easy-to-apply clinical predictive score, which is based on vital sign measurements and simple laboratory values

.

It involves four criteria and is positive when two or more of them are present:

- Heart rate >90beats/min,
- Core temperature <36 or >38°C,
- White blood count <4000 or >12,000/mm^3, and
- Respirations >20/min or PCO_2 <32mmHg:

[30]Mofidi et al., 'Association between Early Systemic Inflammatory Response, Severity of Multiorgan Dysfunction and Death in Acute Pancreatitis'.

Ranson's criteria (Non-Gallstone Pancreatitis)[31]

At admission

- Age in years > 55years
- Leucocyte count > 16000cells/mm^3
- Blood glucose > 10 mmol/L (> 200mg/dL)
- Serum AST > 250IU/L
- Serum LDH > 350IU/L

At 48 hours

- Calcium (serum calcium < 8.0mg/dL)
- Haematocrit fall>10mmol/l
- Oxygen (hypoxemia PO2 < 60mmHg)
- BUN increased by 5 or more mg/dL after IV fluid hydration
- Base deficit (negative base excess) > 4mEq/L
- Sequestration of fluids > 6L

[31]Basit, Ruan, and Mukherjee, 'Ranson Criteria'.

At admission

- Age in years > 70years
- Leucocyte count > 18000cells/mm^3
- Blood glucose > 220mg/dL)
- Serum AST > 250U/100mL
- Serum LDH > 400 IU/L At **48hours**
- Calcium (serum calcium < 8.0mg/dL)
- Haematocrit fall>10mmol/l
- BUN increased by 2 or more mg/dL after IV fluid hydration
- Base deficit (negative base excess) > 5mEq/L
- Sequestration of fluids > 4L

Interpretation:

- Score < 3 - Mild Pancreatitis
- Score 4 to 6 - Moderate Pancreatitis
- Score > 7 - Severe Pancreatitis

CTSI (CT **severity index)**

Balthazar Grades

Grade A: Normal pancreas consistent with mild pancreatitis (0 points)

Grade B: Focal or diffuse enlargement of the gland without peripancreatic inflammation (1 point)

Grade C: Peripancreatic inflammation (2 points)

Grade D: Peripancreatic inflammation with single fluid collection (3 points)
Grade E: Peripancreatic inflammation with two or more peripancreatic fluid collections or gas in the pancreas or retro peritoneum (4 points)

Necrosis score:

Absence of necrosis (0 point) Up to 33% necrosis (2 points)

33% to 50% necrosis (4 points)

>50% necrosis (6 points)

CTSI = Balthazar Grade Score + Necrosis Score Interpretation:

0-3 points: Mild pancreatitis

4-6 points: Moderate pancreatitis

7-10 points: Severe pancreatitis

APACHE II Score

I. Physiological variable

- Rectal temperature(ºC)
- Mean arterial pressure (MAP) in mmHg
- Heart rate inbeats/min
- Respiratory rate inbreaths/min
- PaO2 in mmHg
- Arterial pH
- Serum sodium in mEq/l
- Serum potassium in mEq/l
- Serum creatinine in mg/dl

- Haematocrit in percentage

ØWBC count/ mm^3

- Glasgow Coma Score

The total acute physiology score = sum of above points

II. AgePoints

55-64 years (3 points) 65-74 years (5 points) ≥75 years (6 points)
III. Chronic Health Points – points are assigned as below if the patient gives a history of severe organ insufficiency or is immunocompromised:For nonoperative or emergency postoperative patients (5Points) For electivepostoperative patients (2 points)
The APACHE II score = I+II+III Interpretation: A score of > 8 is considered as severe pancreatitis

Less than 44 years (0 point)

45-54 years (2 points)

Modified Glascow index (Imrie score)

- Age >55 yearsold
- PaO2 <8kPa
- Neutrophilia – Leucocyte count>15x10(9)/L
- Calcium <2mmol/L
- Urea >16mmol/L

•AST >200 IU/L; LDH > 600 IU/L

- Serum Albumin <3.2g/dl
- Blood glucose >180mg/dl

Interpretation:

Scores 3 or more it indicates severe pancreatitis

BISAP score

- Blood urea nitrogen more than 25mg/dL,
- Mental status impairment,
- SIRS (Systemic inflammatory response syndrome), defined as two or more of the following

- Temperature < 36 or > 38°C
- Respiratory rate > 20 breaths/min or $PaCO_2$< 32mmHg
- Pulse rate > 90beats/min
- WBC counts < 4000 or > 12,000 cells/ mm^3 or > 10%immature

➢ bands

➢ More than 60 years of age,and/or

➢ Pleuraleffusion

➢ Interpretation:

➢ A Score of more than 3 is associated with 7 to 12 fold increase in risk of organ failure.

Other biochemical markers include as follows:

C - reactive protein (CRP):

CRP is an acute-phase reactant produced by the liver and is used extensively as a marker of severe pancreatitis. However, it is nonspecific as its levels rise in most inflammatory conditions. Themajor limitations with CRP is that it can be measured only after 48hrs as it lacks sensitivity before 48hrs.

Polymorphonuclear Leukocyte Elastase:

Polymorphonuclear leukocyte elastase rises very early, even before CRP, in acute pancreatitis. High levels have been reported to differentiate severe from mild disease.

Phospholipase A2 (PLA2):

PLA2 plays a vital role in degrading surfactant in the lung. It plays a important role in the pulmonary dysfunction associated with acute pancreatitis. Levels of type II PLA2 are used to differentiate between mild and severe disease within 24 hours of admission.

Urinary TAP may serve as an early predictor of severity in patients with acute pancreatitis. Urinary TAP more than >30 nmol/L is associated with severe disease. The test must be done within 12 hours of hospital admission. Elevated TAP test prediction is about 80% and the negative predictive value approaches 100%.

Procalcitonin:

This procalcitonin is one another acute-phase reactant that has been used to differentiate mild from severe acute pancreatitis within the first 24 hours of symptoms onset. This test that has a sensitivity of 86% and a specificity of

95% in detecting organ failure. It has a drawback that it is not available at all centres and is expensive.

Interleukin-6 (IL-6):

IL-6 is acytokine thatinduces hepatic synthesis of CRP. Numerous studies has beenreportedas a reasonable marker to differentiate mild from severe disease, but the test is not readily available in all centres and is very expensive.

Serum AmyloidA:

Serum amyloid A is another early acute-phase reactant that is synthesized in the liver and is associated with the extent of tissue inflammation. Studies have demonstrated that the level of this serum protein can differentiate mild from severe disease. However, it is expensive and not available in peripheral centres.It can be noticed that the limitation with other parameters is that they are very costly and not easily available.On realizing the importance of acute pancreatitis, extensive studies were conducted by numerous medical practitioners regarding evaluation of the severity of acute pancreatitis and designed various scoring systems. They also even compared these scoring systems with one another to find out a single best possible way to predict the severity of acute pancreatitis. The following are few examples of such studies.

Thomas L Bollen et al compared the radiological and clinical scoring systems

in acute pancreatitis in 2002 and that routine studied that CT abdomen, at time of admission is not recommended routinely in a case of acute pancreatitis for assessing its severity.

RawadMounzer et al, compared all clinical scoring systems to predict organ failure, in cases of acute pancreatitis. He finally concluded that all scoring systems have reasonable accuracy in predicting persistent organ failure, but the Glascow score was found to be the best.[32]

In 2012 , Fabre et al studied several scoring systems in paediatric age group presenting with acute pancreatitis. He studied the sensitivity and specificity of each score and found that the best parameter to assess the severity of acute pancreatitis in paediatric population is CT severity score.[33]

Zhang WW et al, compared the clinical scoring and CT severity scoring in 2011, he found that CT has superior role than clinical scoring and he also found that CT severity index has good correlation with APACHE II and Ranson's scores.

[32]Mounzer et al., 'Comparison of Existing Clinical Scoring Systems to Predict Persistent Organ Failure in Patients with Acute Pancreatitis'.
[33]'Acute Pancreatitis in Children'.

In 2011, Wu et al. published the first RCT trial on early fluid resuscitation in AP and compared the outcomes of fluid resuscitation with two crystalloid fluids, lactated Ringer's solution versus normal saline, during the first 24 hours of admission in 40 consecutive patients with AP. They found a significant reduction in systemic inflammation with lactated Ringer's solution compared to normal saline as measured by SIRS and CRP[34]

In 2007, Ekrem et al studied definite relation between the elevation of CRP, BUN, LDH, CT severity index, APACHE score and mortality and morbidity in patients presenting with acute pancreatitis.[35]

In 2006, Yuk Pang et al, studied the comparison between Ransons score with APACHE scores in acute pancreatitis and concluded that APACHE II score is better and more accurate than that of Ranson‟s score in predicting the severity of acute pancreatitis.[36]

[34]Wu et al., 'Lactated Ringer's Solution Reduces Systemic Inflammation Compared with Saline in Patients with Acute Pancreatitis'.
[35]Vengadakrishnan and Koushik, 'A Study of the Clinical Profile of Acute Pancreatitis and Its Correlation with Severity Indices'.
[36]Yeung, Lam, and Yip, 'APACHE System Is Better than Ranson System in the Prediction of Severity of Acute Pancreatitis'.

In 2016 Seung Kook Cho studied Neutrophil to lymphocyte ratio and platelet to lymphocyte ratio can predict the severity of gallstone pancreatitis and concluded that NLR and PLR were significant independent predictive factors of POF in gallstone AP, than CRP, a traditionally used inflammatory marker and independent prognostic factor.[37]

In 2013, Suppiah A et al studied the divergence of these two components of the WBC counts - neutrophilia and lymphopeniathat raised the proposal of assessing the NLR as a single and more accurate predictive factor than either component alone. [38]

Hotchkiss et al, Ayala et al have observed apoptosis of lymphocytes which resulted in lymphocytopenia. Menges et al supported this with his flow cytometric assays which showed a decrease in T4- helper lymphocytes following multiple trauma and hence responsible for SIRS and MODS. It has been stated that lymphocytopenia not only indicates the severity of the stressful condition, but also reflects the efficacy and adaptability of the immune system.[39]

[37]Cho et al., 'Neutrophil to Lymphocyte Ratio and Platelet to Lymphocyte Ratio Can Predict the Severity of Gallstone Pancreatitis'.
[38]Suppiah et al., 'The Prognostic Value of the Neutrophil-Lymphocyte Ratio (NLR) in Acute Pancreatitis'.
[39]Menges et al., 'Changes in Blood Lymphocyte Populations after Multiple Trauma'.

MANAGEMENT OF ACUTE PANCREATITIS

Treatment of AP involves correction of these underlying aetiologies and control of the inflammatory process to prevent severe complications such as multiorgan failure and infected pancreatic necrosis

INTRAVENOUS FLUIDS

Long underappreciated intravenous fluid resuscitation is now recognized as the cornerstone of medical treatment for AP. The goal of fluid resuscitation is to adequately perfuse the pancreatic microcirculation to prevent pancreatic ischemia and hopefully limit progression to pancreatic necrosis, SIRS, and multiorgan failure[40]. Two studies have demonstrated that an elevated haematocrit admission or a failure to decrease haematocrit 24 hours after admission is a risk factor for the development of pancreatic necrosis

Another study found that the development of pancreatic necrosis was strongly associated with an increase in serum creatinine within48 hours of admission. Finally, in a metanalysis published in 2011 analysing 1043 cases of AP, a BUN level of 20mg/dL or greater at admission and BUN rise within24 hours of hospitalization were associated with an odds ratio of 4.6 and 4.3,

[40]Warndorf et al., 'Early Fluid Resuscitation Reduces Morbidity among Patients with Acute Pancreatitis'.

respectively, for increased mortality and death[41]. These simple laboratory markers illustrate the importance f intravascular volume in the progression of AP. Inadequate fluid resuscitation has been associated with the development of acute necrotizing pancreatitis.[4243]

Early versus late fluid resuscitation[44]

Early fluid resuscitation was defined as receiving greater than one-third of the total first 72hour fluid volume administered with in the first24 hours, and late resuscitation as receiving less than one-third[45]. The investigators found that patients in the early resuscitation group experienced less mortality than those in the late resuscitation group. Although they advocate early fluid resuscitation, they did not suggest a specific fluid volume to be infused

A retrospective analysis of 436 patients with AP similarly examining early versus late fluid resuscitation found that early resuscitation was associated with decreased SIRS, decreased organ failure at 72 hours, a lower rate of admission to the intensive care unit, and a decreased length of hospital stay

[41]Wu et al., 'Blood Urea Nitrogen in the Early Assessment of Acute Pancreatitis'.
[42]Gardner et al., 'Faster Rate of Initial Fluid Resuscitation in Severe Acute Pancreatitis Diminishes In-Hospital Mortality'.
[43]Brown, Orav, and Banks, 'Hemoconcentration Is an Early Marker for Organ Failure and Necrotizing Pancreatitis'.
[44]Warndorf et al., 'Early Fluid Resuscitation Reduces Morbidity among Patients with Acute Pancreatitis'.
[45]Fisher and Gardner, 'The "Golden Hours" of Management in Acute Pancreatitis'.

Regard less of the lack of specific guidelines, most experts recommend starting in AP with a rate between 250 and 300mL/h or enough to produce a urine output of at least 0.5 mL/kg. This infusion follows a 1–2Lfluid bolus given to the patient in the emergency department. A total fluid infusion of 2.5–4L in the first 24 hours will generally suffice to reach resuscitation goals.

As discussed previously, laboratory markers including haematocrit, BUN, and creatinine are in direct measures of intravascular fluid volume and perfusion of the pancreatic microcirculation and should be measured at admission and at 12 hours interval to guide fluid management. Symptoms and signs of pulmonary oedema should also be monitored.

The investigators concluded that the more pH-balanced lactated Ringer's solution may provide improved pH and electrolyte homeostasis when compared to normal saline, leading to less pancreatic and systemic inflammation [46]. Further randomized controlled trials are needed to evaluate fluid management in AP, but lactated Ringer's solution in initial fluid resuscitation may be preferable to normal saline.[47]

[46]Wu et al., 'Lactated Ringer's Solution Reduces Systemic Inflammation Compared with Saline in Patients with Acute Pancreatitis'.
[47]Wu et al.

Targeted pharmacologic therapy

Despite thousands of animal studies and numerous human trials published on the treatment of AP, there are still no proven pharmacological therapies. Several drugs have been evaluated that specifically target the pathophysiologic process of AP with no benefit in important outcomes in randomized controlled trials (RCT). These agents include those directed at reducing pancreatic secretions–specifically atropine, glucagon, cimetidine, somatostatin, and its long-acting analogue octreotide. A randomized controlled trial in 1994 of 302 patients with Ap treated with octreotide showed no significant difference in mortality or development of complications when compared with controls.

ANTIBIOTICS

Inpatients who survive the early phase of AP, the most common cause of death is infection of pancreatic necrosis by enteric bacteria. Patients with pancreatic necrosis have an especially high risk of infection which occurs in 50–70% of cases. Although only 5% of patients with AP develop infected pancreatic necrosis, this complication may account for upto70% of all deaths.

Therefore, there has been much interest in the use of prophylactic antibiotics to prevent these infections inpatients and reduce morbidity, mortality, and health-care costs. Antibiotic treatment in AP is subject of considerable debate with conflicting studies and no clear guidelines. The use of prophylactic antibiotics in severe AP to prevent pancreatic infection is currently not recommended If infection or sepsis is suspected, treatment with antibiotics is appropriate while conducting a thorough evaluation for infection including blood cultures and cultures of a fine-needle aspirate from the site of pancreatic necrosis. If the infectious work-up is negative, antibiotics should be stopped.

Enteral feeding

In severe AP or predicted severe pancreatitis, enteral feeding via tube feedings should be started within72 hours of hospitalization. Multiple studies have shown that enteral feeding is superior to parenteral feeding in severe AP as it maintains the gut barrier. Severe AP randomized to total parenteral nutrition versus total enteral nutrition, total enteral nutrition was superior regarding mortality, infectious complications, organ failure, and lower surgicalintervention rate.

If it is clear that the patient is not meeting nutritional goals within the first week of hospitalization with enteral feeding, parenteral nutrition should be initiated. However, enteral feeding should be continued even at low rates to maintain gut barrier function and prevent bacterial translocation.

In summary, despite high morbidity, mortality, and health-care costs, the medical treatment of AP remains largely supportive with no pharmacologic therapies verified to improve important clinical outcomes. Intravenous fluid resuscitation, especially within the first24 hours of presentation, is the cornerstone of treatment and critical to maintaining the microcirculation of the pancreas to prevent progression from mild to severe. AP and complications such as SIRS, multiorgan failure, and pancreatic necrosis. Further randomized controlled trials are needed to create specific guidelines on the optimal type, volume, and rate of intravenous fluid resuscitation. Antibiotics are not recommended in the prevention of infected pancreatic necrosis as they have shown no benefit in overall mortality in multiple meta analyses.

- For giving rest to the pancreas, patient is made NPO.

- To control abdominal pain, intravenous narcotic analgesics is given &

supplemental O2 (2 L) via nasal cannula.

- Every 6-8 hrs. Serial bedside monitoring of vital signs, oxygen saturation & change in physical examination is mandatory.

Special Considerations based on Etiology

In Gallstone Pancreatitis, Patients are increased risk of recurrence. If patients with evidence of ascending cholangitis within 24-48 h of admission they must undergo ERCP andso performing a cholecystectomy or endoscopic biliary sphincterotomy during the same admission or within 4-6 weeks of discharge is advisable.

In **HYPERTRIGLYCERIDEMIA** (Serum triglycerides > 1 000 mg/ dL) initial therapy may include insulin, heparin, or plasmapheresis, lipid lowering agents, weight loss, avoidance of drugs that elevate lipid levels.

- **Autoimmune pancreatitis** responds to glucocorticoid administration)

- **Post-ERCP pancreatitis** - Pancreatic duct stenting and rectal indomethacin administration are effective

MATERIALS AND METHODS

This study was carried out in General Surgery department, Government Royapettah Hospital from april 2019 to september 2019 after obtaining permission from the Institutions Research and Ethical committee.

SOURCE OF DATA:

The study was conducted on 130 patients diagnosed with acute pancreatitis in Government Royapettah Hospital.

METHOD OF COLLECTION OF DATA:

⦿ Patients with acute pancreatitis were diagnosed as per Atlanta symposium which is any two of the three findings:

- Abdominal pain consistent with acute pancreatitis, i.e., severe and persistent epigastric pain, acute in onset, radiating to the back
- Serum amylase or lipase: three or more times the normal limit.
- CECT (Contrast Enhanced Computerized Tomography) findings characteristic with acute pancreatitis and less commonly with MRI or Ultrasonography of abdomen

⦿ Informed consent was obtained from patients for including them in my

study

⦿ Blood samples were taken at the time of admission and sent for Serum Amylase, Sr. urea, Sr. creatinine and liver function test analysis

⦿ Similarly, Samples were sent for total WBC count and differential count

- At the time of admission
- At24hrs
- At48hrs

⦿ Neutrophil lymphocyte ratio (NLR) and platelet lymphocyte ratio was calculated which is the ratio of the Absolute Neutrophil count (in %) and Absolute Lymphocyte count (in%), ratio of platelet and absolute Lymphocyte count

⦿ Appropriate tests were conducted like Sr. Creatinine, Blood Pressure monitoring and Spo2 as and when needed to look for features of organ failure.

⦿ NLR and PLR values were correlated with the CECT Abdomen of pancreatitis patient.

INCLUSION CRITERIA:

- All cases of acute pancreatitis admitted in our hospital from my study period

EXCLUSION CRITERIA:

- Patients with chronic pancreatitis
- Recurrent pancreatitis
- Patients with known haematological disorder
- Patient diagnosed with malignancy

METHOD OF STATISTICAL ANALYSIS:

The NLR and PLR for day 0, day1 and day 2 for mild pancreatitis and severe pancreatitis were analysed using independent sample t test. A „p" value of <0.05 is indicated as statistically significant.

OBSERVATION AND RESULTS:

SEX DISTRIBUTION:

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	MALE	121	93.1	93.1	93.1
	FEMALE	9	6.9	6.9	100.0
	Total	130	100.0	100.0	

In my study, out of 130 patients 121 patients were male and 9 were female, which showed that there is higher preponderance for pancreatitis in male patients

AGE DISTRIBUTION:

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	20 - 30 YEARS	15	11.5	11.5	11.5
	31 - 40 YEARS	52	40.0	40.0	51.5
	41 - 50 YEARS	50	38.5	38.5	90.0
	51 - 60 YEARS	13	10.0	10.0	100.0
	Total	130	100.0	100.0	

	N	Minimum	Maximum	Mean	Std. Deviation
AGE	130	26	60	40.65	7.502
Valid N (listwise)	130				

It is observed that the most common age group affected in pancreatitis was 31-40 years, accounting for 40%

NLR at hr,24 hr and 48 hrs

NLR		%		%		%
POSITIVE	48	39.9	42	32.3	35	26.9
NEGATIVE	82	63.1	88	67.7	95	73

In this study it is observed that there is progressive decline in NLR over the course of time in hospital

NLR AND CT

AT ADMISSION

			CT FINDINGS		
			Positive	Negative	Total
NLR- At time of admission	Positive	Count	27	21	48
		% within NLR- At time of admission	56.3%	43.8%	100.0 %
	Negative	Count	12	70	82
		% within NLR- At time of admission	14.6%	85.4%	100.0 %
Total		Count	39	91	130
		% within NLR- At time of admission	30.0%	70.0%	100.0 %

AT 24 HOURS:

			CT FINDINGS		
			Positive	Negative	Total
NLR- At 24 Hours	Positive	Count	29	13	42
		% within NLR- At 24 Hours	69.0%	31.0%	100.0%
	Negative	Count	10	78	88
		% within NLR- At 24 Hours	11.4%	88.6%	100.0%
Total		Count	39	91	130
		% within NLR- At 24 Hours	30.0%	70.0%	100.0%

AT 48 HOURS:

			CT FINDINGS		
			Positive	Negative	Total
NLR - At 48 Hours	Positive	Count	27	8	35
		% within NLR - At 48 Hours	77.1%	22.9%	100.0%
	Negative	Count	12	83	95
		% within NLR - At 48 Hours	12.6%	87.4%	100.0%
Total		Count	39	91	130
		% within NLR - At 48 Hours	30.0%	70.0%	100.0%

PLR AT ADMISSON,24 HRS AND 48 HOURS

PLR		%		%		%
POSITIVE	56	43.1	46	35.4	41	31.5
NEGATIVE	74	56.9	84	64.4	89	68.5

PLR VERSUS CT:

AT ADMISSION:

			CT FINDINGS		Total
			Positive	Negative	
PLR- At time of admission	Positive	Count	25	31	56
		% within PLR- At time of admission	44.6%	55.4%	100.0%
	Negative	Count	14	60	74
		% within PLR- At time of admission	18.9%	81.1%	100.0%
Total		Count	39	91	130
		% within PLR- At time of admission	30.0%	70.0%	100.0%

AT 24 HOURS:

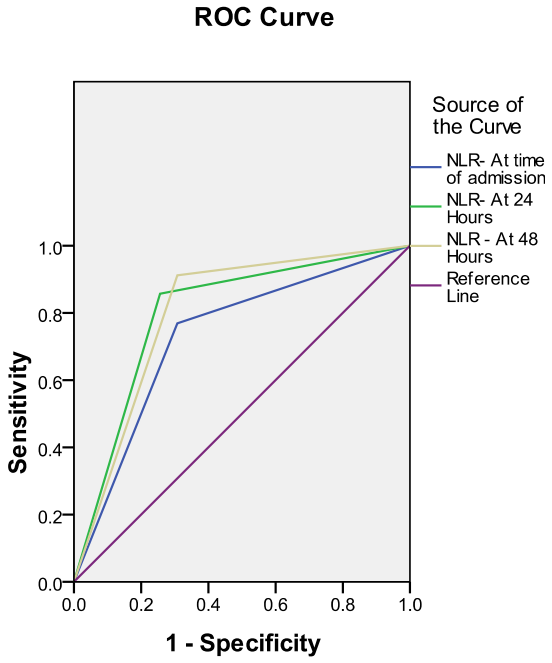
			CT FINDINGS		
			Positive	Negative	Total
PLR - At 24 Hours	Positive	Count	21	25	46
		% within PLR - At 24 Hours	45.7%	54.3%	100.0%
	Negative	Count	18	66	84
		% within PLR - At 24 Hours	21.4%	78.6%	100.0%
Total		Count	39	91	130
		% within PLR - At 24 Hours	30.0%	70.0%	100.0%

AT 48 HOURS:

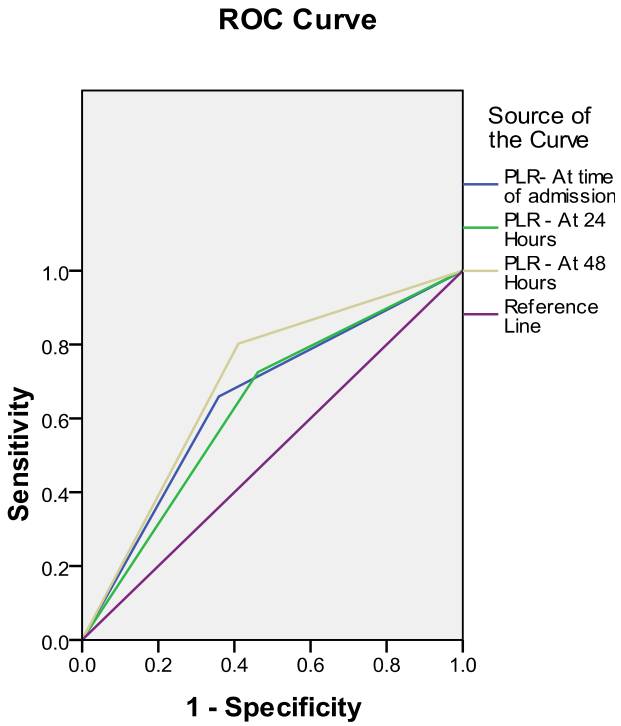
			CT FINDINGS		
			Positive	Negative	Total
PLR - At 48 Hours	Positive	Count	23	18	41
		% within PLR - At 48 Hours	56.1%	43.9%	100.0%
	Negative	Count	16	73	89
		% within PLR - At 48 Hours	18.0%	82.0%	100.0%
Total		Count	39	91	130
		% within PLR - At 48 Hours	30.0%	70.0%	100.0%

SENSITVITY AND SPECIFICITY OF NLR AND PLR

	O HR		24 HR		48 HR	
	NLR	PLR	NLR	PLR	NLR	PLR
SENSITIVITY	69.2	64.1	74.4	53.8	62.2	59
SPECIFICITY	76.9	65.9	85.7	72.5	91.2	80.2



Diagonal segments are produced by ties.



Diagonal segments are produced by ties.

Area Under the Curve

Test Result Variable(s)	Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
PLR- At time of admission	.650	.053	.007	.546	.754
PLR - At 24 Hours	.632	.055	.017	.525	.739
PLR - At 48 Hours	.696	.053	.000	.592	.800

The test result variable(s): PLR- At time of admission, PLR - At 24 Hours, PLR - At 48 Hours has at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased.

a. Under the nonparametric assumption

b. Null hypothesis: true area = 0.5

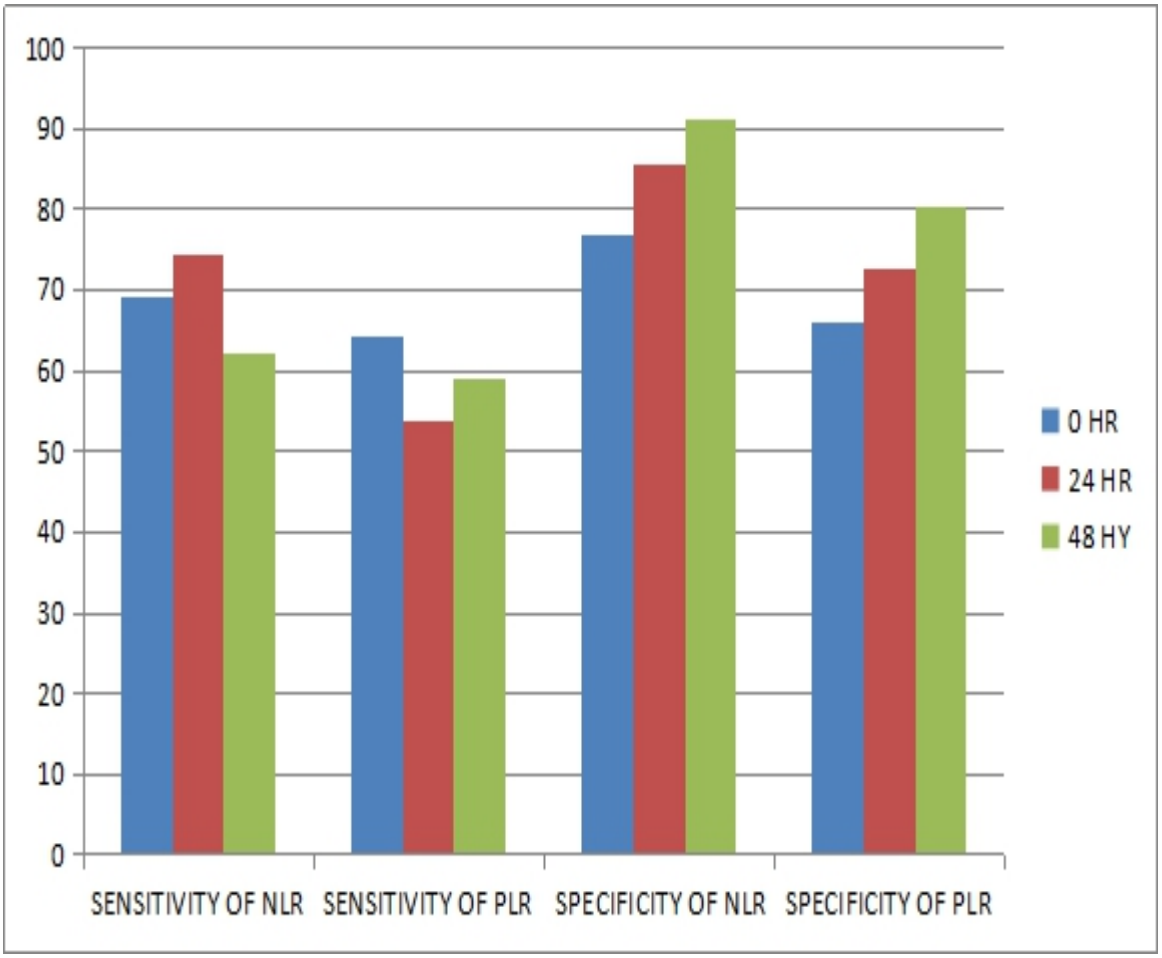
Area Under the Curve

Test Result Variable(s)	Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
NLR- At time of admission	.731	.050	.000	.633	.829
NLR- At 24 Hours	.800	.046	.000	.710	.891
NLR - At 48 Hours	.802	.048	.000	.709	.896

The test result variable(s): NLR- At time of admission, NLR- At 24 Hours, NLR - At 48 Hours has at least one tie between the positive actual state group and the negative actual state group. Statistics may be biased.

a. Under the nonparametric assumption

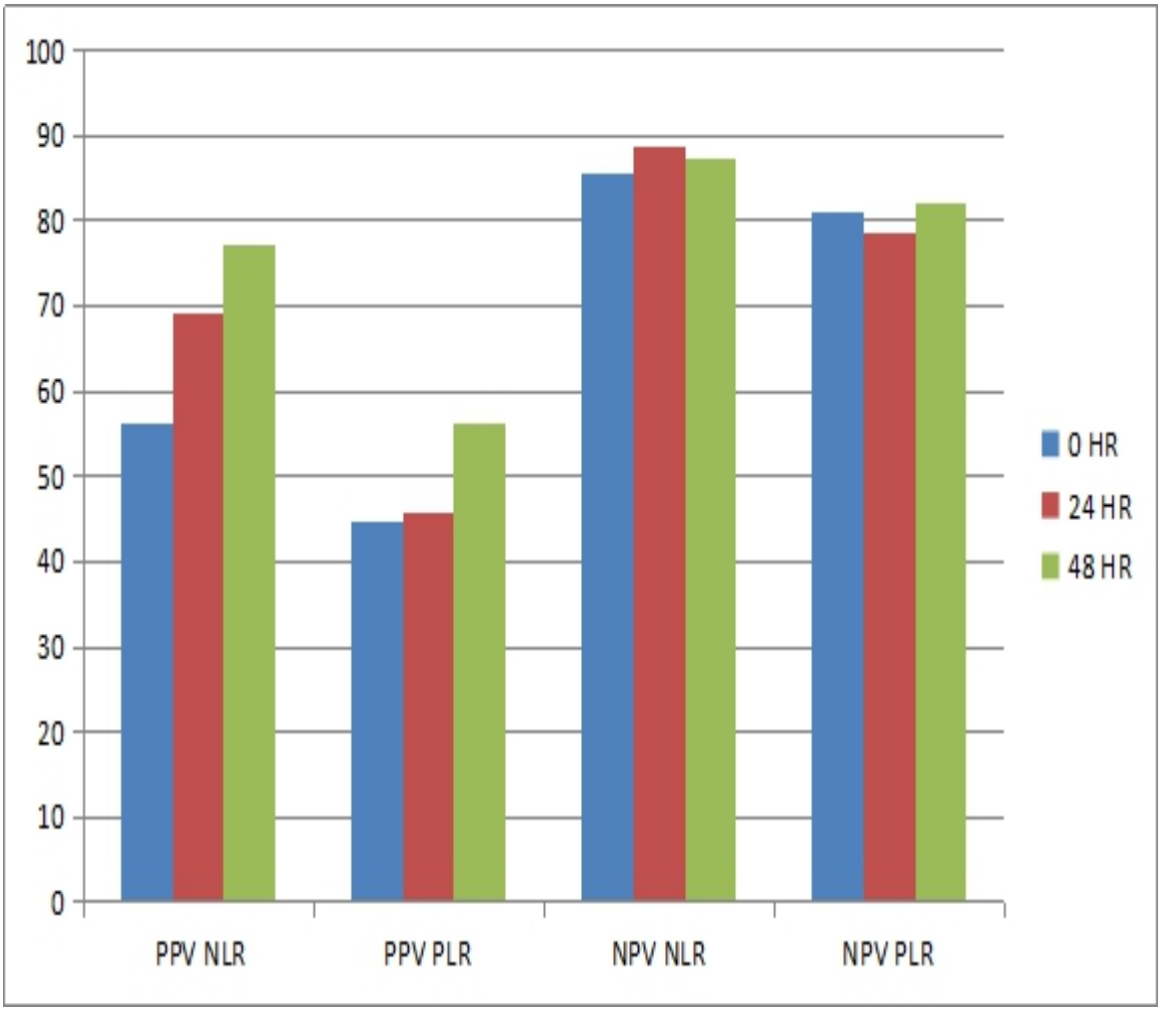
b. Null hypothesis: true area = 0.5


100
90
80
70
60
50
40
30
20
10
0
SENSITIVITY OF NLR
SENSITIVITY OF PLR
SPECIFICITY OF NLR
SPECIFICITY OF PLR
0 HR
24 HR
48 HY

PPVAND NPV OF NLR AND PLR:

	0 HR		24 HR		48 HR	
	NLR	PLR	NLR	PLR	NLR	PLR
PPV	56.3	44.6	69.3	45.7	77	56.1
NPV	85.4	81.1	88.6	78.6	87.4	82

PPV AND NPV OF NLR AND PLR



The present study concluded that, out of 130 patients 121 were male, which showed that male patients were most commonly affected by pancreatitis than female

Most common age group affected were between 31-40 years, comprising of 40 %

Mean age affected was 40.65 with a standard deviation of 7.502

30 % of patients were diagnosed as having pancreatitis by CT imaging

According to ATLANTA classification,77.7% were diagnosed as having mild pancreatitis,22.3% were categorized under severe pancreatitis

The sensitivity of NLR at admission,24 hours and 48 hours were 69.2%,74.4% and 62.2& respectively

The sensitivity of PLR at admission ,24 hours and 48 hours were 64.1%,53.8%&59% respectively

The specificity of NLR at admission,24 hours and 48 hours were 76.95,85.7% &,91.2% respectively

The specificity of PLR at admission ,24 hours and 48 hours were 65.9%,72.5%,80.2% respectively

The PPV of NLR and PLR

at admission were56.3% and 44.6%,

at 24 hours 69.3%& 45.7%,

at 48 hours 77% &56.1%

The NPV of NLR& PLR

At admissionà85.4% &81.1%

At 24 hours 88.6%&78.6%

At 48 hours 87.4%&82%

The sensitivity and NPV of NLR was higher than PLR in diagnosing acute necrotizing pancreatitis

NLR was also superior in terms of predicting intensive care admission and shorter hospital stay.

When NLR and PLR were combined, both good statistical correlation as an early predictor of necrotizing pancreatitis.

From the study it is concluded that the combination of NLR and PLR was found to have the highest AUC in terms of predicting necrosis earlier than other scoring systems

DISCUSSION

In my study on 130 patients of acute pancreatitis, Males are (93.1%) than females are(6.9%). This observation may be due to the fact that alcohol consumption is more common among males in our epidemiology

Majority of the patients in mystudy are between age group of 31- 40yr (40% of my study population). Next, 38.5 % of my study population were among age group of 40-50 yrs. The mean age was around 40.3%.

In my study, the most of the acute pancreatitis patients were alcoholic which was around 96% comparingthe study done by Savio G Barreto et al, where alcohol was found as the causative agent in 92.6% and gallstones in 19%.

Of the 130 patients, 77.7% of them had mild pancreatitis and 22.3% had severe pancreatitis. My study looks comparable to the rate of incidence of mild and severe pancreatitis as per Atlanta symposium, in which the rate of mild pancreatitis is 70-80 % and 20-30 % in severe pancreatitis patients.

In my study, mortality rate 7.69% and all the patients were suffering from acute necrotising pancreatitis due to organ failure.

In my study, I also noticed that, serum amylase was elevated (≥ 3times the normal) in only 18% of patients while it was < 3 times the normal in 82% of

patients. Studies showed lower levels of amylase in patients with acute pancreatitis were due to severe destruction of the pancreas. they reported that it was especially true in pancreatitis caused by alcohol, where the amylase level was lower at the time of admission. As alcohol induced pancreatitis was the major cause in my study group the lower amylase values may be attributed to it.

The primary finding in my study is that the Neutrophil Lymphocyte Ratio (NLR) and Platelet lymphocyte ratio(PLR) were elevated significantly in patients with acute necrotising pancreatitis comparing acute pancreatitis patients .

In the present study, we investigated the value of NLR and PLR as predictive markers of necrosis in AP patient. We found that NLR and PLR were well correlated with CT finding in patients with acute necrotising pancreatitis patients. NLR was first introduced as an easy reproducible and measurable parameter assessing systemic inflammation in critically ill patients in ICU. Then, PLR was also found to be an inflammatory marker, and the role of platelets as a critical factor between inflammation and microvascular dysfunction leading to SIRS and ORGAN FAILURE. The prognostic and predictive value of these two parameters has been confirmed in a variety of clinical conditions, and PLR was shown to be superior to NLR in certain cancers and inflammatory conditions.

AP is an inflammatory condition characterized by activation of both innate and acquired immune responses. Activation and controlled influence of neutrophils and platelets play a crucial role in establishing host defences in settings of systemic inflammation, however in some scenario, excessive and extensive inflammatory response causes massive cell migration to the pancreas and subsequent release of aggressive defence molecules, resulting in destruction of the pancreas and subsequent necrosis.

However, despite the demonstrated superiority of PLR over NLR in predicting the outcome of inflammation in several clinical conditions, there were no studies investigated the predictive value of PLR at the time of admission Necrosis in Acute pancreatitis patients.

Therefore, we investigated the value of PLR in predicting the necrosis in Acute pancreatitis and compared differences between NLR and PLR patterns in prediction of necrosis.

CONCLUSION

In my study, NLR AND PLR has proved to be a indicator in predicting the necrosis in acutepancreatitis. NLR and PLR can be easily calculated from basic investigations done in all patients. Beinga basic investigation, it adds no additional cost to the patient.

NLR and PLR correlates well with predicting necrosis in acute pancreatitis. Regular monitoring on each day will provide a dynamic reflection immune response of the host to pancreatitis and hence predict the necrosis and the prognosis of the patient earlier. In my study statically, NLR seems to superior to PLR in prediction of necrosis in acute pancreatitis patient.

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ANNEXURES

Urkund Analysis Result

Analysed Document: ur 1.docx (D57791757)
Submitted: 28/10/2019 09:34:00
Submitted By: ibu.van@gmail.com
Significance: 16 %

Sources included in the report:

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சுயஒப்புதல்படிவம்

ஆய்வுசெய்யப்படும்தலைப்பு **'A PROSPECTIVE STUDY ON NEUTROPHIL LYMPHOCYTE RATIO AND PLATELET LYMPHOCYTE RATIO AS EARLY PREDICTOR OF NECROSIS IN ACUTE PANCREATITIS, Department of General Surgery, GRH.**

பங்குபெறுபவரின்பெயர்:

பங்குபெறுபவரின்வயது: பங்குபெறுபவரின்எண் :

மேலேகுறிப்பிட்டுள்ளமருத்துவஆய்வின்விவரங்கள்எனக்கு விளக்கப்பட்டது.
நான்இவ்வாய்வில்தன்னிச்சையாகபங்கேற்கிறேன்.
எந்தகாரணத்தினாலோஎந்தசட்டசிக்கலுக்கும்உட்படாமல் நான்இவ்வாய்வில்இருந்துவிலகிக்கொள்ளல்லாம்என்றும் அறிந்துகொண்டேன்.

இந்தஆய்வுசம்பந்தமாகவோ,
இதைசார்ந்துமேலும்ஆய்வுமேற்கொள்ளும்போதும்இந்தஆய்வில்பங்குபெறும்மருத்துவர்என்னுடையமருத்துவஅறிக்கைகளைபார்ப்பதற்குஎன்அனுமதிதேவையில்லைஎனஅறிந்துகொள்கிறேன்.
இந்தஆய்வின்மூலம்கிடைக்கும்தகவலையோ,
முடிவையோபயன்படுத்திக்கொள்ளமறுக்கமாட்டேன்.
இந்தஆய்வில்பங்குகொள்ளஒப்புக்கொள்கிறேன்.
இந்தஆய்வைமேற்கொள்ளும்மருத்துவஅணிக்குஉண்மையுடன்இருப்பேன்என்றும்உறுதியளிக்கிறேன்.

பங்கேற்பவரின்கையொப்பம்

இடம்:

தேதி:

ஆய்வாளரின்கையொப்பம்

ஆய்வாளரின்கையொப்பம்

S.NO.	NAME	AGE	SEX	IP. NO.							AMYL	USG/CT FINDINGS	ATLANTA
					NLR	PLR	NLR	PLR	NLR	PLR			CLASSIFICATION
1	RAMALINGAM	48	M	28543	14	348	13.3	342	12.4	340	413	E EDEMATOUS PANCREA	MILD
2	MANOHAR	42	M	23479	13.33	340	14.18		12.4	342	568	ACUTE NECROTISING PAI	SEVERE
3	SURYA	58	M	29938	12.59	347	12.59	345	11.7	343	339	ACUTE NECROTISING PAI	MILD
4	SUBASH	36	M	29945	11.62	341	12.03	340	12.5	342	224	ACUTE PANCREATITIS	MILD
5	NATRAJ	51	M	29903	19.56	376	20	370	22.6	368	631	ACUTE NECROTISING PAI	SEVERE
6	PONNUSAMY	40	M	29001	16.31	350	15.86	338	14.2	340	245	ACUTE PANCREATITIS WI	SEVERE
7	ROSEMARY	40	F	29932	12.5	343	15.13	330	14.4	333	475	ACUTE NECROTISING PAI	MILD
8	ANBARASAN	48	M	28345	12.62	338	16.83	335	15.3	335	487	ACUTE NECROTISING PAI	SEVERE
9	SEKAR	32	M	29370	13.57	345	12.46	340	13.2	339	247	ACUTE PANCREATITIS	MILD
10	KANNAN	39	M	28955	14.93	347	14.75	345	14.6	343	416	ACUTE EDEMATOUS PAN	MILD
11	THARANIVELU	60	M	29000	14.44	339	13.62	340	13.3	340	371	ACUTE PANCREATITIS WI	MILD
12	BABU	28	M	28824	19.5	380	21	378	21.7	370	719	ACUTE NECROTISING PAI	SEVERE
13	SATHYA	50	M	28159	14.44	340	13.6	338	3.33	332	371	ACUTE PANCREATITIS	MILD
14	DEVA	38	M	28687	12.3	341	12	340	11.9	340	301	ACUTE PANCREATITIS	MILD
15	SUGUMAR	40	M	29940	12.8	342	12.5	340	12.5	338	316	ACUTE NECROTISING PAI	MILD
16	SANTHOSH	30	M	29981	13.4	348	13.1	347	12.9	341	398	ACUTE PANCREATITIS	MILD
17	PERUMAL	48	M	28967	14.4	350	14.2	348	14.1	348	415	ACUTE PANCREATITIS	MILD
18	RAVI	53	M	29453	16.7	360	16.6	359	16	350	467	ACUTE PANCREATITIS	MILD
19	SIVA	55	M	29567	19.6	376	19.4	374	18.8	370	667	ACUTE NECROTISING PAI	SEVERE
20	PRABHAKARAN	43	M	28970	12.3	345	12	343	11.9	340	345	ACUTE PANCREATITIS	MILD
21	SELVARARJ	32	M	28742	14.5	342	13.6	343	13.5	342	387	ACUTE PANCREATITIS WI	MILD
22	BASKAR	40	M	29956	12.7	343	12	343	12	340	370	ACUTE PANCREATITIS	MILD
23	SEKAR	56	M	28796	14.3	351	14	348	13.4	345	401	ACUTE NECROTISING PAI	SEVERE
24	SHANKAR	35	M	29964	13.8	346	13	344	13.1	342	415	ACUTE PANCREATITIS	MILD
25	RAMAN	45	M	29754	12.7	348	12.5	345	12.4	344	383	ACUTE PANCREATITIS	MILD
26	LINGA	48	M	29564	13.8	356	13.4	350	13	349	408	ACUTE PANCREATITIS	MILD
27	RAMESH	41	M	29346	20.1	389	19.9	380	19.5	375	764	ACUTE NECROTISING PAI	SEVERE
28	PRADEEP	39	M	29567	13.7	340	13	338	13	340	389	ACUTE PANCREATITIS	MILD
29	THANGAVEL	45	M	28857	12	344	12.1	340	11.7	337	315	ACUTE PANCREATITIS	MILD
30	SUNDAR	29	M	27091	13.3	348	13	344	12.8	341	356	ACUTE EDEMATOUS PAN	MILD
31	RAJAVEL	34	M	29076	15.1	352	15.1	350	14.8	346	409	ACUTE NECROTISING PAI	MILD
32	MURALI	29	M	27459	13.2	342	13	340	12.8	337	378	ACUTE PANCREATITIS	MILD
33	ASHOK	51	M	28967	14.1	348	13.3	342	13	340	350	ACUTE PANCREATITIS	MILD
34	RAVI	43	M	29075	12	340	12.3	341	12.2	340	301	ACUTE PANCREATITIS	MILD
35	RAGHU	40	M	30562	18.8	371	18	367	16.9	356	799	ACUTE NECROTISING PAI	SEVERE
36	PADMANATHAN	32	M	29867	11.9	338	12.3	341	12	338	287	ACUTE PANCREATITIS	MILD
37	RAVANNAN	47	M	29632	13.7		13.4		13		356	ACUTE PANCREATITIS	MILD

						345		342		340				
38	VEERAMANI	40	M	30321	14.3		14		13.8		402	ACUTE PANCREATITIS	MILD	
						346		340		338				
39	VELU	41	M	29871	14		13.6		13.4		345	ACUTE PANCREATITIS	MILD	
						341		338		335				
40	MAARI	52	M	29994	20.4		19.7		19		1002	ACUTE NECROTISING PA	SEVERE	
						383		376		370				
41	SELVAM	43	M	29832	13.3		13		12.9		278	ACUTE PANCREATITIS	MILD	
						340		338		337				
42	RAVI	39	M	28714	12.7		12.9		12.5		301	ACUTE EDEMATOUS PAN	MILD	
						337		337		335				
43	SHAIK AHMED	43	M	25347	14.9		14,5		14		418	ACUTE PANCREATITIS	MILD	
						343		340		339				
44	JEEVAN	40	M	28996	13.5		13		13.1		408	ACUTE PANCREATITIS	MILD	
						348		345		342				
45	RAGHU	37	M	29641	12.5		12.3		12		279	ACUTE PANCREATITIS	MILD	
						341		12.1		12				
46	JOHN	38	M	30532	13		12.7		12.5		309	ACUTE PANCREATITIS	MILD	
						338		335		337				
47	REKHADEVI	34	F	29898	14.3		14		13.7		348	ACUTE PANCREATITIS WI	MILD	
						345		341		338				
48	RAGHUL	49	M	30101	13.8		13.4		13		407	ACUTE PANCREATITIS	MILD	
						348		345		340				
49	FRANCIS	32	M	29953	13		12.7		12		289	ACUTE PANCREATITIS WI	MILD	
						340		338		337				
50	RAMALINGAM	49	M	30104	17.8		17.3		17		661	E NECROTISING PANCREA	SEVERE	
						366		362		358				
51	PANDI	39	M	29947	12.9		12,4		12		305	ACUTE PANCREATITIS	MILD	
						342		340		338				
52	MUTHU	45	M	28510	12		12.3		12		315	ACUTE PANCREATITIS WI	MILD	
						340		337		335				
53	GOPALAN	42	M	29931	12		12		13		267	ACUTE EDEMATOUS PAN	MILD	
						345		333		345				
54	DHAYALAN	40	M	30421	11.9		11.2		14		280	ACUTE EDEMATOUS PAN	MILD	
						336		326		335				
55	SATHISH	45	M	30452	14		16		17		400	ACUTE NECROTISING PA	SEVERE	
						380		336		360				
56	SIVA	55	M	30635	17		18		18		366	ACUTE NECROTISING PA	SEVERE	
						365		376		377				
57	THANGAVEL	44	M	29081	12.6		12.7		12.5		280	ACUTE PANCREATITIS	MILD	
						336		338		338				
58	DHANANJAYAN	39	M	29635	11.9		11		11.8		300	ACUTE PANCREATITIS	SEVERE	
						340		345		342				
59	REKHA DEVI	42	F	24585	13		12,4		12.9		310	ACUTE EDEMATOUS PAN	MILD	
						338		342		336				
60	SWAPANA	45	F	29991	12.8		11.8		12.7		311	ACUTE PANCREATITIS	MILD	
						346		342		342				
61	PRIYA	40	F	26685	16.5		14.5		16.6		500	ACUTE NECROTISING PA	SEVERE	
						381		396		380				
62	RAGU	38	M	30551	11.9		12.4		11.8		328	ACUTE PANCREATITIS	MILD	
						338		335		336				
63	RAGHAV	39	M	30110	12		13		12.1		310	ACUTE NECROTISING PA	MILD	
						342		345		340				
64	KISHORE	54	M	30155	13.1		12.1		13.2		324	ACUTE PANCREATITIS WI	MILD	
						343		342		343				
65	RAMU	45	M	28552	12.1		14.4		12		280	ACUTE PANCREATITIS	MILD	
						337		338		335				
66	SHIVAJI	50	M	29668	18		17		17.8		490	ACUTE NECROTISING PA	SEVERE	
						396		386		391				
67	BASHA	51	M	30405	12.2		11.2		12.4		245	ACUTE PANCREATITIS	MILD	
						351		352		350				
68	PRANAV	36	M	30410	16		17		161		480	ACUTE NECROTISING PA	SEVERE	
						396		386		390				
69	PRAVEEN	37	M	30452	14		13		13.9		268	ACUTE PANCREATITIS	MILD	
						344		345		342				
70	SHANKAR	41	M	29658	13.5		12.5		13.4		276	ACUTE PANCREATITIS	MILD	
						321		322		330				
71	RAGHUL	41	M	28111	12.9		12		12.7		276	ACUTE EDEMATOUS PAN	MILD	
						324		335		323				
72	LAKSHMI	39	F	29992	12.4		11.8		12.1		224	ACUTE PANCREATITIS	MILD	
						330		345		345				
73	SATHISH	48	M	28991	17		18		18.1		380	ACUTE NECROTISING PA	SEVERE	
						365		376		363				
74	JEEVAN	44	M	29668	11.2		12.2		11		252	ACUTE PANCREATITIS	MILD	
						321		332		330				
75	SUBASH	46	M	30101	18.8		17.8		18		396	ACUTE NECROTISING PA	SEVERE	
						386		399		384				

76	SURIYA	42	M	30112	13.5		12.5		13		250	ACUTE PANCREATITIS	MILD
						335		336		332			
77	SARAN	44	M	30521	12.7		12,4		12.1		270	ACUTE PANCREATITIS	MILD
						348		348		346			
78	DHANANJAYAN	49	M	30401	18		18		17.8		444	ACUTE NECROTISING PAI	SEVERE
						376		382		370			
79	KRISHNA	37	M	29685	19.1		18		19		496	ACUTE NECROTISING PAI	SEVERE
						394		396		396			
80	NEHA	36	F	28883	11.2		11.9		11.4		301	ACUTE PANCREATITIS	MILD
						335		332		333			
81	NANDHINI	41	F	28891	11.9		12.2		12		311	ACUTE EDEMATOUS PAN	MILD
						331		332		334			
82	PRAKASH	46	M	29012	12.1		13.1		12.5		290	ACUTE PANCREATITIS	MILD
						334		340		332			
83	RISHI	47	M	29658	13.1		12.1		12.9		283	ACUTE NECROTISING PAI	MILD
						342		340		340			
84	ROSHINI	38	F	30120	12.5		12,4		12.6		280	ACUTE PANCREATITIS	MILD
						348		342		342			
85	HARI	35	M	30225	19		18.3		18		480	ACUTE NECROTISING PAI	SEVERE
						380		382		386			
86	GOVINDAN	42	M	30213	14		13.8		13.5		238	ACUTE PANCREATITIS	MILD
						272		270		265			
87	PERUMAL	27	M	30322	13.6		13.2		12.5		248	ACUTE PANCREATITIS WI	MILD
						270		268		266			
88	AMUDHAN	30	M	30312	14.2		13.9		13.6		302	ACUTE PANCREATITIS	MILD
						284		283		262			
89	VARADHAN	42	M	30342	15.1		14.3		14.4		336	ACUTE PANCREATITIS	MILD
						282		277		279			
90	KRISHNASAMY	45	M	30311	18.2		18		17.8		489	ACUTE NECROTISING PAI	SEVERE
						398		396		378			
91	GAJENDRAN	55	M	30085	11		11.4		12.2		492	ACUTE PANCREATITIS	MILD
						226		248		252			
92	RANJITH	26	M	30332	13.8		12		13.1		237	ACUTE NECROTISING PAI	MILD
						277		234		212			
93	SEKAR	41	M	30312	12.4		13		12.6		254	ACUTE PANCREATITIS WI	MILD
						213		213		218			
94	VEERAMANI	50	M	30356	12.5		13.4		12.7		265	ACUTE PANCREATITIS	MILD
						235		265		237			
95	MADHAN	38	M	30321	12		13		13		245	ACUTE PANCREATITIS	MILD
						265		214		249			
96	SANTHOSH	45	M	30387	14		16		15		263	ACUTE NECROTISING PAI	SEVERE
						245		256		238			
97	BASHA	39	M	30398	13.5		13		13.1		253	ACUTE PANCREATITIS	MILD
						213		224		213			
98	ARASU	43	M	30218	13.2		12.4		13.6		247	ACUTE NECROTISING PAI	MILD
						234		227		234			
99	RAJA	34	M	30222	11.7		13.4		11.4		297	ACUTE PANCREATITIS	MILD
						215		212		245			
100	JAYA KUMAR	29	M	30123	14		11.3		13.9		217	ACUTE PANCREATITIS	MILD
						289		235		213			
101	PALANI	48	M	29989	13.2		12.7		12.5		267	ACUTE NECROTISING PAI	MILD
						222		237		253			
102	KARTHCK	34	M	30101	12		13.5		12.7		275	ACUTE PANCREATITIS	MILD
						287		285		248			
103	IFZATH ALI	28	M	29987	13.8		11.7		11.3		214	ACUTE PANCREATITIS	MILD
						246		263		274			
104	RAJALINGAM	33	M	30003	12.8		12.5		12.6		257	ACUTE PANCREATITIS	MILD
						271		292		219			
105	PERIASAMY	45	M	29873	11.2		12.6		13.4		247	ACUTE PANCREATITIS	MILD
						216		214		298			
106	KANNAN	37	M	30289	16.9		17.1		16.8		414	ACUTE NECROTISING PAI	SEVERE
						255		333		356			
107	RAMU	39	M	29976	11.6		13.6		12.6		238	ACUTE PANCREATITIS	MILD
						283		246		216			
108	SURESH	49	M	30009	13.5		12.7		13.2		218	ACUTE PANCREATITIS	MILD
						279		264		268			
109	RAVINDRAN	26	M	30199	11.5		12.6		12		289	ACUTE NECROTISING PAI	MILD
						267		214		218			
110	RAJU	44	M	29987	12.3		13.7		12.4		216	ACUTE PANCREATITIS	MILD
						213		269		239			
111	RAVI	49	M	30219	14		15		12		232	ACUTE PANCREATITIS	SEVERE
						287		267		264			
112	YUVARAJ	41	M	30321	13		12.4		13		213	ACUTE PANCREATITIS	MILD
						215		231		213			
113	KATHIRESAN	39	M	30319	12		12.6		12		241	ACUTE PANCREATITIS	MILD
						245		253		241			
114	PALANI	45	M	30312	12.5		13		13		251	ACUTE PANCREATITIS	MILD

						224		223		231			
115	KUMAR	51	M	29991	13.4		14		12.4		236	ACUTE PANCREATITIS	MILD
						231		241		245			
116	SELVAN	34	M	29841	12.7		12.5		12.1		309	ACUTE NECROTISING PAI	MILD
						239		234		230			
117	RAGAVENDRA	30	M	30341	12.4		12.6		12		298	ACUTE PANCREATITIS	MILD
						244		243		240			
118	AHMED	38	M	299510	14.5		14,.0		13.8		381	ACUTE PANCREATITIS	MILD
						240		241		240			
119	SHANMUGAM	29	M	30295	15.6		15		14.9		367	ACUTE NECROTISING PAI	SEVERE
						255		250		246			
120	RAVI	34	M	28973	13.3		13		12.1		275	ACUTE PANCREATITIS	MILD
						240		238		238			
121	SHANKAR	30	M	29941	16.3		16		15.4		415	ACUTE PANCREATITIS	SEVERE
						258		255		246		WITH NECROSIS	
122	REVANTH	32	M	30178	15,2		14.3		14		278	ACUTE PANCREATITIS	MILD
						250		246		243			
123	ABDUL	34	M	288310	12		11.9		11.5		267	ACUTE PANCREATITIS	MILD
						244		240		239			
124	PRAKASH	28	M	29961	11.8		11.5		11		251	ACUTE PANCREATITIS	MILD
						236		234		230			
125	PRABHU	32	M	28578	16.9		16.7		16		463	ACUTE NECROTISING PAI	SEVERE
						266		260		250			
126	CHOZHAN	37	M	27761	13.3		13		12.7		267	ACUTE PANCREATITIS WI	MILD
						245		243		240			
127	CHANDRAN	31	M	26719	14		13.9		13.5		301	ACUTE NECROTISING PAI	MILD
						250		245		240			
128	FRANCIS	38	M	30156	13.5		13		13	240	270	ACUTE PANCREATITIS	MILD
						245		242					
129	RAGHU	33	M	27719	13		12.7		12.5	233	259	ACUTE PANCREATITIS WI	MILD
						243		240					
130	SANTHOSH	28	M	30321	15.1		14.9		14		299	ACUTE NECROTISING PAI	MILD
						251		246		240			