
**“ROLE OF PANC3 SCORE AND HSCR P IN
PREDICTING SEVERITY OF ACUTE
PANCREATITIS IN PATIENTS ADMITTED AT
TERTIARY CARE CENTRE, BELAGAVI-A ONE
YEAR CROSS SECTIONAL STUDY”**

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LIST OF ABBREVIATIONS

AP	Acute Pancreatitis
SAP	Severe Acute Pancreatitis
MAP	Mild Acute Pancreatitis:
MSAP	Moderately Severe Acute Pancreatitis
CAP	Complex Acute Pancreatitis
CRP	C-Reactive Protein
HsCRP	High-sensitivity C-Reactive Protein
BMI	Body Mass Index
HCT	Haematocrit
POF	Persistent Organ Failure
SOFA	Sequential Organ Failure Assessment
APACHE	Acute Physiology and Chronic Health Evaluation

ABSTRACT

Introduction: Acute pancreatitis (AP) is a severe inflammatory condition of the pancreas, often caused by gallstones or alcohol consumption. The updated Atlanta classification system categorizes it into Mild Acute Pancreatitis (MAP), Moderately Severe Acute Pancreatitis (MSAP), and Severe Acute Pancreatitis (SAP). Early prediction of severity is essential for appropriate management.

Methodology: This study evaluated the effectiveness of the PANC 3 scoring system and High-Sensitivity C-Reactive Protein (HsCRP) levels in predicting AP severity. A cohort of patients diagnosed with AP was assessed using PANC 3 parameters (Body Mass Index, Haematocrit, and Pleural Effusion) and HsCRP levels. Clinical outcomes were compared to determine the accuracy of these markers.

Results: The findings indicated that the PANC 3 score and HsCRP levels were effective in predicting severe AP. A significant correlation was observed between higher scores and adverse clinical outcomes, demonstrating the utility of these tools in early severity assessment.

Conclusion: The PANC 3 scoring system and HsCRP levels are reliable and practical markers for predicting the severity of acute pancreatitis. Their implementation in clinical practice can aid in early intervention and improve patient management.

Keywords: Acute Pancreatitis, PANC 3 Score, HsCRP, Severity Prediction, APACHE, SOFA, Ranson Score.

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INTRODUCTION

Acute pancreatitis (AP) is most severe among the abdominal visceral illnesses. AP can result from a variety of causes.^[1,2] It is defined as acute pancreatic inflammation induced by the digestive enzymes activation within gland, which affects pancreas, surrounding tissues, other organs.^[3,4] With occurrence of 30-50 out of 100,000 people,^[5] this AP is typically caused by bile stones or increased alcohol usage.

Although the majority of instances are minor and confined, the symptom spectrum has been wide-ranging. Nearly 20% have severe variant, which causes systemic problems and increased deaths ^[3]. Common symptoms typically include sudden and severe upper abdominal pain that radiates to the back, accompanied by nausea, vomiting, and localized inflammation of the abdominal lining (peritonitis) in the epigastric region. In some cases, the condition can also affect the circulatory system, and may be characterized by increased pancreatic enzymes in blood or urine.

Updated Atlanta categorisation system classifies AP into 3 categories: mild AP (MAP), moderately severe AP (MSAP), and severe AP (SAP). This classification has been based on whether or not are any local or systemic complications. "Mild Acute Pancreatitis (MAP) refers to pancreatitis without any complications. Moderately Severe Acute Pancreatitis (MSAP) is characterized by the presence of local complications such as fluid collections, necrotic collections, pseudocysts, or walled-off necrosis, and/or transient organ failure lasting less than 48 hours"^[6].

Patients who have had chronic organ failure for > 48 hours are classified into SAP^[6]. Primary worry in the therapy is progression of MAP to more severe variants, which are associated with higher morbidity and death^[7,8]. MSAP and SAP are frequently grouped jointly into complex acute pancreatitis (CAP)^[9,10].

The severity of AP determines the management^[6], and those with severe disease may require urgent care from the time symptoms appear. As a result, predicting the disease's progression as early as possible is critical^[3]. Following the initial diagnosis, treatment is determined by early evaluation of disease severity. Early detection of SAP reduces mortality from 50% to 8%, emphasising the need of early illness identification^[11].

Since Ranson's study in 1974,^[12] other studies have proposed markers and scores for identifying people at risk of developing SAP.^[13] However, tests cause challenges in practical practice.^[14] There are currently no consensuses regarding the best strategy, and an objective way to predict the severity of AP is still required.^[15] Different markers have been assigned prognostic value around the world, with varying sensitivity and specificity, but the exploration for the ideal indicator continues.

Clinicians are always attempting in integrating such indicators in order to frame scoring system instead of systems designed to achieve highest potential prediction value. Despite the fact that several clinical grading systems have been devised, they have only little accuracy to predict POF in AP.^[13-16] The majority of these indications are time-consuming, need extensive laboratory setup, and take > 48 hours to assess severity categorization. "The 2012 Atlanta classification system, a widely used framework, categorizes acute pancreatitis severity based on early

prognostic indicators, organ failure, and local complications. Other commonly employed scoring systems to assess disease severity include the Ranson score, Glasgow score, Balthazar score, APACHE II, and SOFA score."

Brown et al devised the PANC 3 scoring system, which they claimed was an approachable, assessable scoring system that used only three parameters: HCT, BMI, pleural effusion. It is quick and simple for use, requires minimal hospital equipment, performs as others to predict outcome of AP attack. ^[17] The PANC 3 score has the advantage of using generally available, rapid, and easy-to-measure tests, as well as having excellent accuracy in predicting severe acute pancreatitis. ^[18]

Although Ranson, Glasgow, and APACHE II were once widely employed, they have limitations for a variety of reasons. ^[13] These limitations prompted development of single markers to predict CAP. ^[16] "C-Reactive Protein (CRP) is a significant predictor, serving as an acute-phase reactant that is produced by the liver in response to inflammatory conditions, thereby indicating the presence of inflammation." ^[17]

Mayer et al. presented the first report on its use as a CAP predictor in 1984 [19]. From then, there were conflicting findings on its use to predict CAP [13, 20]. Furthermore, there is debate concerning appropriate timing for assessing CRP and the optimal cut-offs. ^[13, 21]

To complicate matters, a CRP interval change of > 90 is offered to be superior predictor. ^[22] "There is currently no established consensus on the optimal timing and threshold values for C-Reactive Protein (CRP) in predicting disease severity, therefore, different timings and values must be evaluated and compared to other commonly used predictive markers."

AIMS AND OBJECTIVES

AIM:

"The aim is to assess the effectiveness of the PANC 3 score and high-sensitivity C-Reactive Protein (HsCRP) in predicting the severity of acute pancreatitis and their correlation with the clinical outcomes of the disease."

OBJECTIVES:

1. To estimate PANC 3 score and HsCRP in patients of AP.
2. To assess clinical outcomes in patients of AP.
3. "This study aims to investigate the predictive value of the PANC 3 score and high-sensitivity C-Reactive Protein (HsCRP) for determining the severity of acute pancreatitis and exploring their relationship with patient outcomes."

REVIEW OF LITERATURE

ACUTE PANCREATITIS

Acute pancreatitis (AP), frequent condition, is major reason for hospitalisation for GI diseases. Severity of AP can range from mild cases requiring minimal management upto severe and complex cases with significant morbidity and mortality. While diagnosing acute presentation is relatively straightforward, the challenge lies in predicting the disease's progression and outcome.

The Atlanta classification divides AP into 2 types:

- Interstitial edematous pancreatitis: Inflammation of pancreatic tissue and surrounding areas, typically results in swelling of the pancreas, and
- Necrotizing pancreatitis, a more severe form, leads to cell death (necrosis) of pancreatic tissue and surrounding tissues, resulting in significant damage and potential complications.

AP is classified into 3 kinds based on its severity: mild, moderate, and severe.

- MAP does not cause organ failure or other problems.
- Moderately Severe Acute Pancreatitis (MSAP) is characterized by presence of local complications, with or without transient organ failure that lasts < 48 hours.
- SAP involves POF for > 48 hours, affecting 1/ more organs.

Anatomy:

Pancreas is digestive gland located in the posterior abdominal wall, positioned in front of the L1 and L2 vertebrae. Pancreas primary purpose is to secrete both exocrine and endocrine hormones. Exocrine secretions (pancreatic juice from acinar cells) reach duodenum via main and accessory pancreatic ducts, while endocrine secretions (glucagon and insulin from pancreatic islets of Langerhans) enter bloodstream.^[24]

Etiology

Most common causes are gallstones, alcohol usage, and hypertriglyceridemia

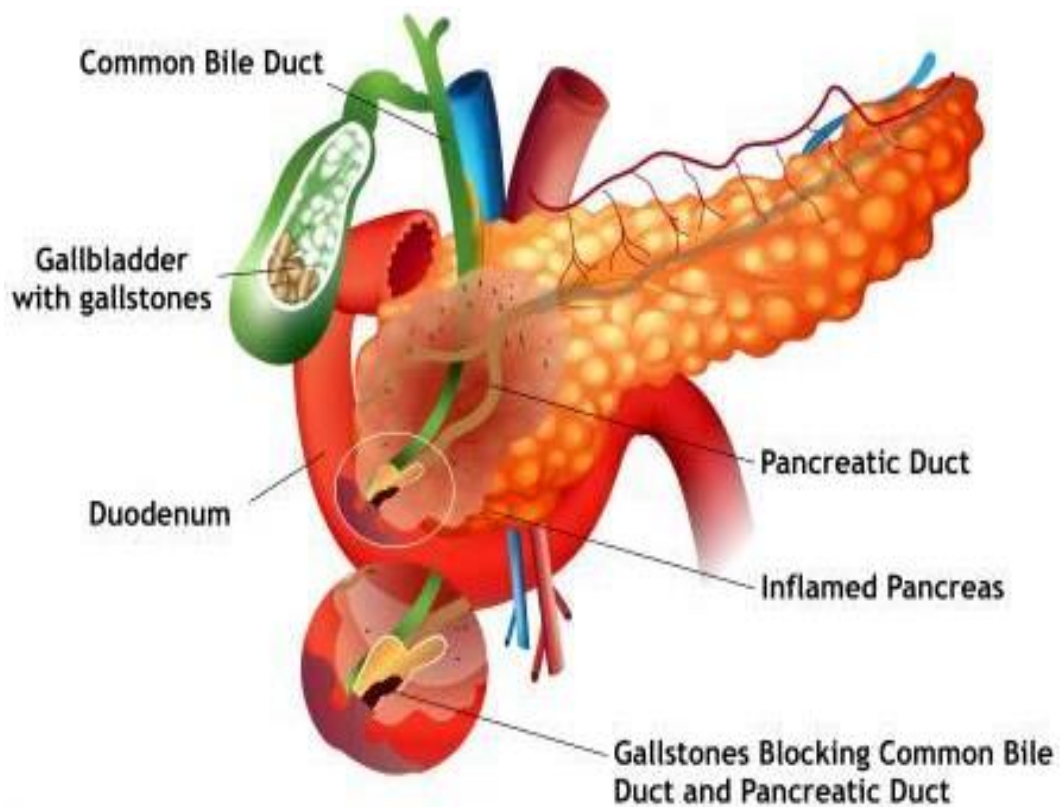


Figure 1: Acute Pancreatitis, Gallstones

Causes of AP.^[25]

Mechanical	Toxic and metabolic	Others
Gall stones	Alcohol	Ischemia
Pancreatic duct obstruction	Hyperlipidemias	Iatrogenic injury
Dysfunction of sphincter of Oddi	Drugs	infection
Trauma	Scorpion venom	Hereditary
Ampullary obstruction	Organophosphate poisoning	Autoimmune
Congenital malformations	Hypocalcaemia	Cystic fibrosis

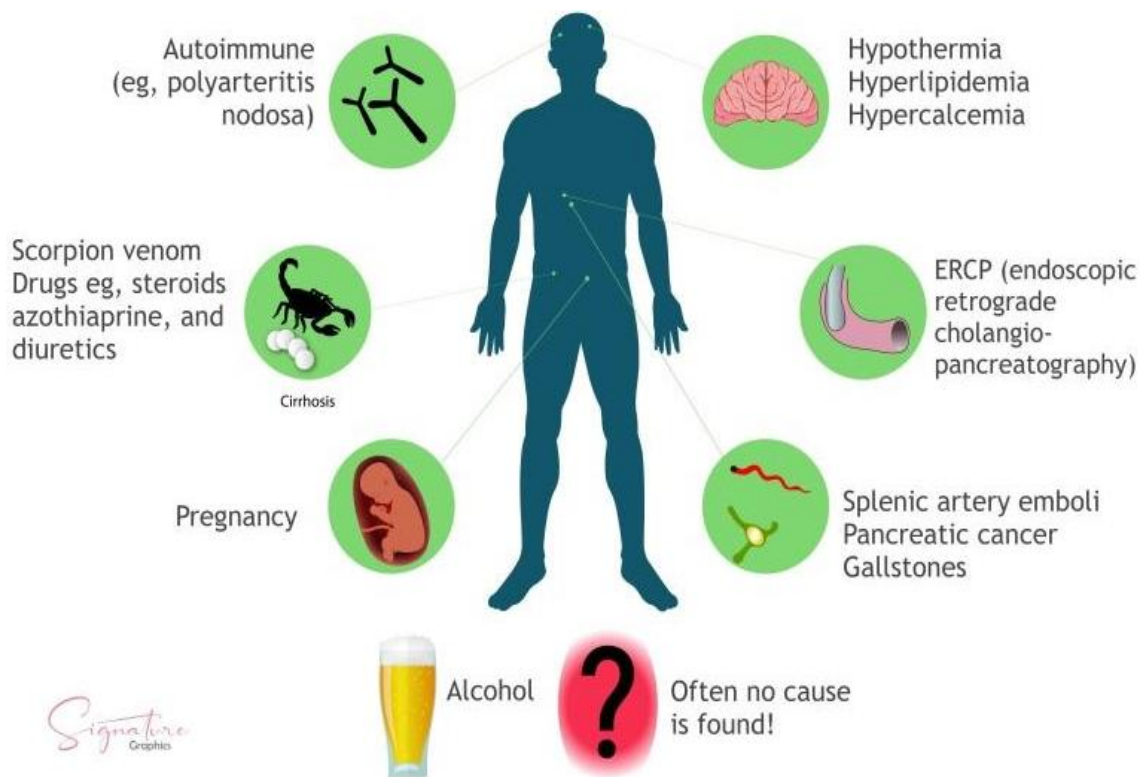


Figure 2: Causes of Pancreatitis

Pathophysiology

Pathophysiology of pancreatitis involves localized pancreatic damage accompanied by a systemic inflammatory response. Primary triggering event is premature activation of trypsinogen to trypsin within acinar cells, rather than in the ductal lumen. This is often caused by increased ductal pressure due to obstruction, and disruptions in calcium homeostasis and pH balance. Various toxins that induce pancreatitis can deplete ATP, leading to elevated intra-acinar calcium levels, which in turn can trigger the early activation of trypsinogen to trypsin, activating other enzymes like elastase and phospholipases.

Premature activation of these zymogens triggers significant tissue damage and release of Damage-Associated Molecular Patterns (DAMPs). This recruits neutrophils and activates inflammatory cascade, driving systemic progression of AP. As inflammatory response intensifies, it increases capillary permeability and causes endothelial damage, leading to microvascular thrombosis. These events culminate in multi-organ dysfunction syndrome (MODS), the primary cause of morbidity and mortality in AP.

Some people have a hereditary susceptibility to pancreatitis, which leads to repeated AP and eventually chronic pancreatitis. Unsurprisingly, linked genes play a role in converting trypsinogen into trypsin. Several genes have been implicated in recurrent pancreatitis, including the cystic fibrosis transmembrane conductance regulator (CFTR) gene, gain-of-function mutations in the cationic trypsinogen gene (PRSS1), mutations in the chymotrypsin C gene (CTRC), and the pancreatic secretory trypsin inhibitor gene (SPINK1). These genetic factors contribute to a spectrum of

pancreatic disorders, ranging from acute pancreatitis (AP) to chronic pancreatitis, and are being increasingly recognized as underlying causes of these conditions.

Classification of Acute Pancreatitis:

Classification of AP based on the 2012 Atlanta Classification of AP

Severity of AP	Characteristics
Mild AP	The most frequent form No organ failure No local or systemic complications Usually resolves in the first week
Moderately severe AP	Transient organ failure resolving within 48 h Local or systemic complications without persistent organ failure Exacerbation of co-morbid disease
Severe AP	Persistent organ failure > 48 h
Criterion of AP	Characteristics
OF and systemic complications of AP	Respiratory System: $P_{aO_2}/F_{iO_2} \leq 300$ Cardiovascular System: SBP < 90 mm Hg Urinary System: serum creatinine $\geq 170 \mu\text{mol/L}$
Local complications of AP	Acute peripancreatic fluid collections Pancreatic and peripancreatic necrosis (sterile or infected) Pancreatic pseudocyst Walled-off pancreatic necrosis (sterile or infected)

Clinical

Patients with AP often present with moderate to severe abdominal pain in the epigastric region, accompanied by nausea and loss of appetite. Pain characteristics

differ depending on underlying cause, with biliary obstruction typically described as sharp, radiating to the back, and having a sudden onset. But, metabolic and toxicologic causes, such as alcohol consumption, frequently begin with dull, generalized discomfort in the epigastric region that develops gradually. A thorough history of alcohol use, medication, and smoking should be obtained, noting that heavy alcohol consumption for over 5 years is typically required to develop alcohol-related pancreatitis, and that smoking is an additional risk factor of AP.

Investigating family history is crucial, especially when common causes of pancreatitis are absent, as rare hereditary cases of familial pancreatitis may be responsible. Physical examination frequently reveals fever, tachycardia, and, in severe cases, hypotension. Abdominal assessment typically shows epigastric tenderness, muscle guarding, rigidity, and reduced bowel sounds. In cases of retroperitoneal hemorrhage, Grey-Turner's sign—bruising on the flanks—may be present, while Cullen's sign, characterized by periumbilical bruising, suggests peritoneal hemorrhage.

Evaluation

The Revised Atlanta Classification defines AP as meeting at least 2 of 3 criteria:

1. Lipase or amylase levels are 3 times higher than normal.
2. Abdominal pain suggests pancreatitis.
3. Abdominal imaging indicates acute pancreatitis.

To identify the underlying cause of AP, a comprehensive initial assessment is conducted, complemented by laboratory testing. A detailed history is essential, focusing on symptoms of gallstone disease, unexplained weight loss, new-onset diabetes, alcohol use, medication history, prior surgery or trauma,

hypertriglyceridemia, hypercalcemia, autoimmune disorders, and a family history of recurring AP. These factors provide critical clues to identifying the underlying cause of the condition.

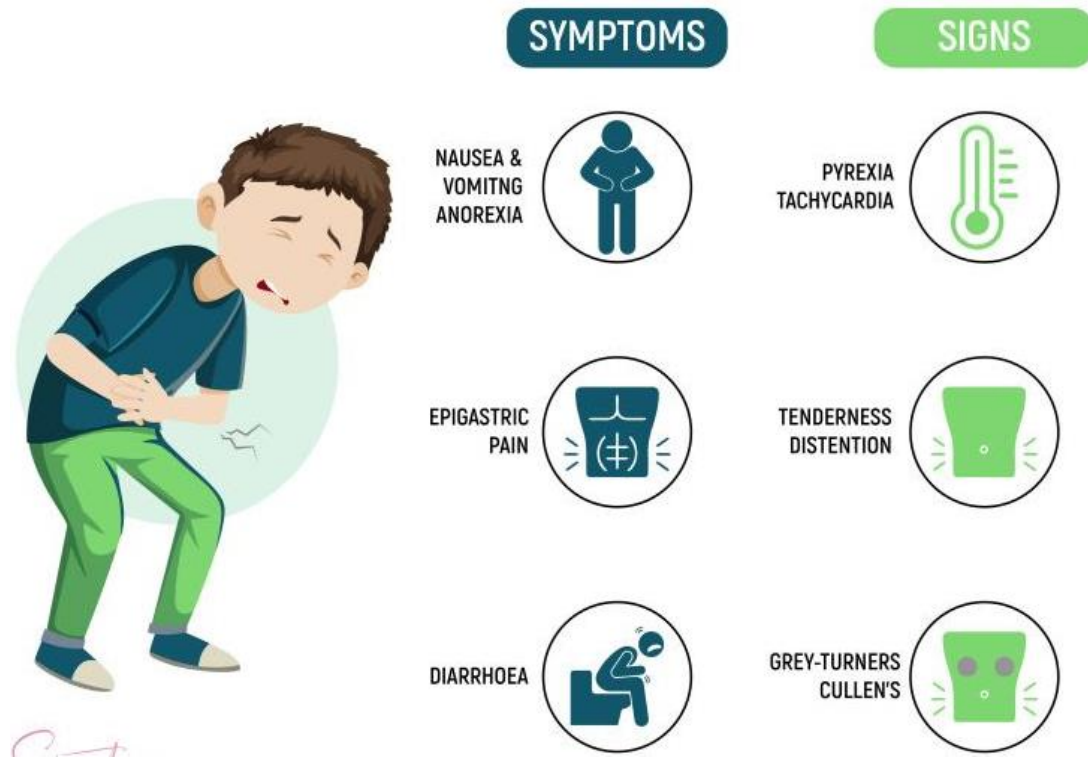


Figure 3: AP, Signs and Symptoms

A thorough diagnostic evaluation for acute pancreatitis (AP) involves a comprehensive initial assessment, supplemented by laboratory tests, to determine the underlying cause. A meticulous medical history is essential, encompassing questions about gallstone disease symptoms, unexplained weight loss, recent-onset diabetes, alcohol consumption, medication regimen, past surgical or traumatic events, hypertriglyceridemia, hypercalcemia, autoimmune disorders, and familial recurrence of AP, as these elements can offer critical insights into the condition's etiology.

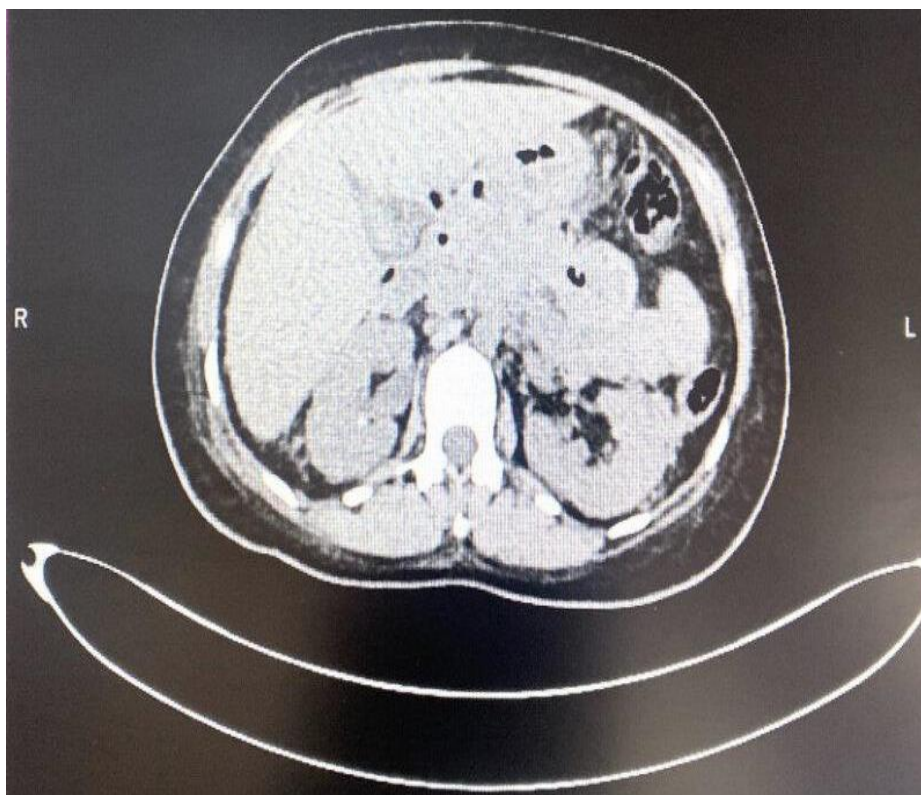


Figure 4: Peripancreatic Necrotic Fluid (CT)

Detailed evaluation of patient with suspected AP

History	Gallstone disease Alcohol abuse Medication use History of weight loss New onset diabetes Previous surgery or trauma Hypertriglyceridemia or hypocalcaemia Autoimmune disease Genetic causes
Examination	Fever Tachycardia Epigastric tenderness or palpable mass in the epigastric area Jaundice Cullen's sign Turner's sign
Laboratory evaluation	Elevated serum amylase and lipase levels- 3-5 times normal Elevated white blood count cell Elevated serum triglycerides Hypocalcaemia Elevated serum bilirubin, AST, ALT, LDH, and alkaline phosphatase
Imaging	Chest and abdominal X-rays Abdominal ultrasound CT abdomen
Serological Test	IgG4
Genetic Tests	PRSS1 SPINK1 CFTR CTRCCASRClaudin-2

When the initial evaluation fails to determine the cause of pancreatitis, session to gastrointestinal specialist is frequently needed for additional testing with advanced imaging modalities. MRCP and EUS are commonly used. While MRCP is non-invasive and does not require contrast, its sensitivity is limited for detecting small biliary stones (<3mm) and early chronic pancreatitis, making EUS the preferred option. If MRCP and EUS are occupied, a pancreatic-protocol CT scan is an alternative

Treatment

The cornerstone of acute pancreatitis (AP) treatment is fluid resuscitation, with LR's solution being preferred fluid. Preliminary treatment involves administering a 15-20mL/kg bolus, followed by 3mL/kg/hr (approximately 250-500mL/hr) over initial 24 hours, assuming no contraindications. To ensure effective fluid

resuscitation, monitoring includes regular laboratory tests, such as BUN, HCT, and urine output, each 4-6 hours during initial 24-hour period. This allows for adjustments to the fluid administration rate as needed. Failure to respond to early fluid resuscitation indicates a high risk of developing multiple organ dysfunction syndrome (MODS) and necessitates escalation of care to a higher level of treatment.^[27]

Nutrition plays a crucial role in the management of acute pancreatitis (AP). Traditionally, patients are kept nil per os (NPO) until symptoms such as abdominal pain, vomiting, nausea, loss of appetite, and ileus subside. In SAP or poor oral intake, nasojejunal feeding is preferred over parenteral nutrition, as it helps maintain the intestinal barrier and reduce bacterial translocation. Prophylactic antibiotics are often unnecessary, but empiric antibiotics may be used when an infection is suspected, pending culture results. Antibiotic treatment is only initiated in confirmed pancreatic necrosis with infection.

The treatment of pain is top goal when treating AP. Almost all patients with AP report stomach pain at some point during condition. Some practice guidelines ignore recommendations for pain treatment. Others, on other hand, make strong recommendations for pain evaluation and appropriate pharmacological therapy but do not go into enough depth about analgesia type, dose, route, and frequency.^[28] While managing acute pancreatitis (AP) may seem straightforward in clinical practice, effective pain management is crucial. Initially, WHO analgesic ladder is in use, which involves a stepwise approach using nonsteroidal anti-inflammatory drugs (NSAIDs) and strong opioid analgesics, as well as interventional procedures. Various analgesic options are available, including opioids like fentanyl and meperidine, and NSAIDs, offering a range of choices for optimal pain control.

WHO analgesic ladder

Step 1	NSAID
Step 2	low potent opioid ± NSAID ± adjuvant drugs
Step 3	High powerful opioid ± NSAID ± adjuvant drugs
Step 4	interventional treatment ± high potent opioid ± NSAID ± adjuvant drugs [23]

Previously, opioids were believed to induce spasms of the Oddi sphincter, but recent evidence challenges this notion. A Cochrane review of five randomized controlled trials (RCTs) involving 227 participants found no significant difference in complication rates or adverse events between opioids and other analgesic treatments. Furthermore, a meta-analysis by Stigliano et al. concluded that there is no compelling evidence to contraindicate the use of morphine for pain management in acute pancreatitis (AP).^[29]

Acute pancreatitis (AP) can lead to local complications, including the accumulation of fluid around the pancreas, pancreatic or peripancreatic necrosis, and the formation of a pancreatic pseudocyst (typically after 4 weeks). In the majority of cases, acute peripancreatic fluid collections resolve on their own, with fewer than 10% progressing to form a pancreatic pseudocyst. Most pseudocysts are managed conservatively with observation and periodic monitoring using CT or MRI scans.

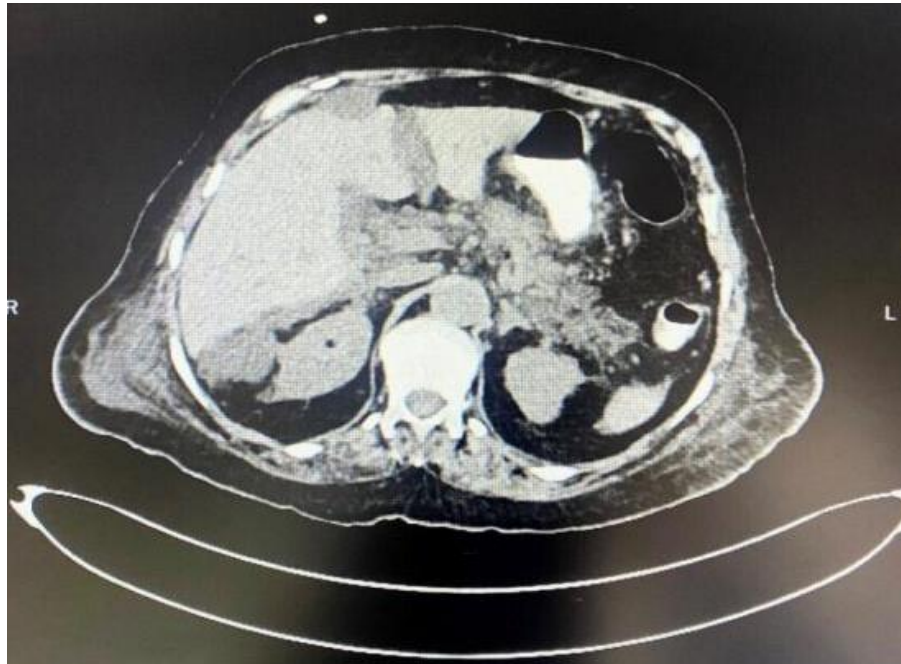


Figure 5: Necrotising Pancreatitis With Extensive Peripancreatic Nodularity

Drainage of these pseudocysts is only suggested in infection and fast growth. Endoscopic (transmural or transpapillary) and percutaneous drainage options are available, with endoscopic route being the preferable method.

Managing necrotic collections in acute pancreatitis is challenging. Intervention is required when sterile collections cause symptoms like nausea, vomiting, chronic abdominal pain, gastric outlet or intestinal obstruction, or a ruptured pancreatic duct. However, approximately 1/3rd of necrotic collections become infected, leading to clinical decline, prolonged recovery, and increased death. Imaging studies and CT-guided percutaneous aspiration cultures revealing gas bubbles confirm infected necrosis. Patients who clinically deteriorate despite antimicrobial therapy undergo surgical necrosectomy. Antibiotics are administered for 4-6 weeks before necrosectomy, allowing collection wall to mature.

PREDICTORS OF SEVERITY OF ACUTE PANCREATITIS

AP is pancreatic inflammation that is not caused by microorganisms. Updated Atlanta classification.^[6] categorises its severity as mild, moderately severe, or severe based on development of local complications and/or organ failures. MSAP and SAP account for around 15%-25% of all cases. Death rate varies with severity; mild AP has extremely low mortality, but severe AP has mortality rates as high as 36-50%.^[6]

Early identification of acute pancreatitis (AP) severity is vital for effective treatment. Previous studies have identified several predictors of AP severity, including clinical characteristics, laboratory tests, radiologic findings, and scoring systems. Commonly used laboratory tests to assess AP severity include hematocrit (HCT), blood urea nitrogen (BUN), C-reactive protein (CRP), and serum creatinine. Additional biomarkers that have been investigated include interleukins, procalcitonin, trypsinogen activation peptide, and various pancreatic enzymes. Several scoring systems are also employed to predict AP severity, such as the Ranson score, Glasgow score, APACHE II score, and Balthazar score, as well as radiologic scoring systems like the computed tomography severity index (CTSI) score. Furthermore, specialized scores like the bedside index of severity in AP and the harmless AP score are also used to assess AP severity.^[30]

PANC-3 Scoring System to predict SAP

PANC-3 scoring system is a simple clinical tool designed for predicting likelihood of SAP early in course of disease. It is based on three easily measurable clinical parameters, making it practical for bedside evaluation.

Understanding Acute Pancreatitis & the Need for Severity Prediction

Acute pancreatitis (AP) is a pancreatic inflammatory condition that can manifest as a mild, self-limiting illness or progress to a severe, life-threatening disease characterized by organ failure, necrosis, sepsis, and potentially catastrophic complications such as multi-organ dysfunction syndrome (MODS).

Early assessment of patients at risk for severe pancreatitis is crucial for initiating aggressive fluid resuscitation, intensive monitoring, and appropriate interventions.

PANC-3 Criteria

A patient is assessed for the following three parameters:

1. Hematocrit (Hct) > 44%

- An elevated hematocrit suggests hemoconcentration due to significant fluid loss.
- This indicates early hypovolemia and increased risk of pancreatic necrosis.
- If hematocrit rises further at 24 hours, it strongly predicts severe pancreatitis.

2. BMI > 30 kg/m²

- Obesity is related with increased inflammation, cytokine release, and metabolic dysfunction.
- Higher BMI correlates with more severe pancreatitis, increased complications, and worse outcomes.

3. Pleural Effusion on Imaging

- Presence of pleural effusion suggests systemic inflammation and third-spacing of fluids into the pleural cavity.
- Associated with severe disease, pancreatic necrosis, and higher mortality.

Interpretation of PANC-3 Score

- All 3 criteria positive → High probability of severe pancreatitis.
- Less than 3 positive → Lower probability of severe disease.
- The specificity of PANC-3 is high, meaning that if a patient meets all three criteria, they are very likely to develop SAP.

PANC-3 Score	Risk of Severe Pancreatitis
3/3	High risk (strong predictor)
<3/3	Lower risk (less predictive)

Clinical Relevance of PANC-3

- Best used for early risk assessment in the emergency department (ED) or wards.
- Helps guide decisions regarding ICU admission, aggressive fluid resuscitation, and close monitoring.
- May be combined with other scores (e.g., BISAP, SIRS criteria) for better prediction.
- More useful at resource-limited settings where advanced scoring systems might not be feasible.

The PANC-3 scoring system is a fast, easy-to-use bedside tool to predict SAP. Its high specificity makes it valuable to early identify high-risk patients, allowing to timely intervene. However, it is best used alongside other clinical and laboratory findings for a more complete risk assessment.

CRP to Predict SAP

CRP is acute-phase inflammatory protein consisting of 5 identical subunits. Tillet and Francis.^[31] discovered this highly conserved plasma protein in 1930 while studying blood samples from people with acute Pneumococcus infection. It was called "C-reactive" because it interacts with Pneumococcus bacterium's capsular (C)-polysaccharide.^[31] CRP is first created as separate monomers, which are subsequently combined into pentameric form within endoplasmic reticulum of the generating cells. This pentameric CRP is stored in endoplasmic reticulum of hepatocytes via contacts with 2 carboxylesterases, gp60a and gp50b.^[32]

When there is no inflammation, CRP slowly exits endoplasmic reticulum. Rising levels of inflammatory cytokines, such as IL-6, IL-1, and TNF- α , reduce CRP binding to carboxylesterases, leading to fast release.^[33] CRP production is activated by pro-inflammatory cytokines, including IL-6, IL-1, and TNF- α .^[34]

C-Reactive Protein (CRP) levels surge in response to injury, infection, and inflammation, serving as a sensitive biomarker. Recent research has expanded our understanding of CRP's role in the inflammatory process, revealing its significance as a key mediator of the acute-phase response. Following an inflammatory event, CRP production is largely driven by IL-6-dependent hepatic biosynthesis, highlighting its critical position downstream of the inflammatory cascade.^[35, 36]

C-Reactive Protein (CRP) plays a pivotal role in the inflammatory response by activating the C1q molecule within the complement system, leading to the opsonization of pathogens. Notably, CRP can trigger both fluid-phase and cell-mediated mechanisms of host defense through complement pathway activation and binding to the Fc region of immunoglobulin G (IgG) receptors.^[35] This binding causes

the release of pro-inflammatory cytokines. Additionally, CRP possesses the ability to distinguish between self and foreign molecules through pattern recognition, setting it apart from other complement activators like IgG. This distinction arises from CRP's capacity to recognize a broader range of molecular patterns, whereas IgG is limited to identifying specific antigenic epitopes.^[37] CRP is recognised as an important indication of inflammation since it rises in cases of tissue injury or necrosis. It is commonly used in clinical settings to assess, diagnose, and track the progression of inflammatory diseases.^[38]

CRP is an acute phase reactant produced by liver in response to systemic inflammation. In context of AP, CRP is an important biomarker for determining disease severity and predicting complications.

CRP as a Prognostic Indicator:

- **Timing of Measurement:** CRP begin to rise within first 24 hours of AP onset, peaking around 48 to 72 hours. Measuring CRP at 48 hours post-admission provides a reliable indicator of disease severity.
- **Optimal Cutoff Values:** Research indicates that CRP exceeding 190 mg/L at 48 hours is associated with SAP, pancreatic necrosis, and increased in-hospital mortality.

EVIDENCE FROM PREVIOUS LITERATURE

- 1. Sunil Kumar Meena,et al.,** did cross-sectional study over 50 patients admitted with acute pancreatitis. After making the clinical diagnosis, PANC -3 score, modified ATLANTA score, APACHE II were done. CRP and CTSI (computed tomography sensitivity index) were calculated and correlated. Mean age was 44.74 years and most common cause was biliary tract pathology. Mortality observed in 5 patients, 11 patients had severe disease. Sensitivity of PANC- 3 was 81.82%, specificity -92.31% with 75% PPV and 94.7% NPV. PANC-3 can be used to predict the severity of pancreatitis as efficiently as Modified ATLANTA classification/APACHE II. It uses only three criteria which are easily done, and available in the basic health care setup. ^[39]
- 2. Murilo Gamba BEDUSCHI,et al.,** did study for evaluating efficacy of PANC 3 to predict acute pancreatitis severity and its relation to clinical outcome. Acute pancreatitis patients were assessed as to sex, age, body mass index (BMI), etiology of pancreatitis, intensive care need, length of stay, length of stay in intensive care unit and mortality. The PANC 3 score was determined within the first 24 hours after diagnosis and compared to acute pancreatitis grade of the Revised Atlanta classification. Out of 64 patients diagnosed with acute pancreatitis, 58 met the inclusion criteria. The PANC 3 score was positive in five cases (8.6%), pancreatitis progressed to a severe form in 10 cases (17.2%) and five patients (8.6%) died. Patients with a positive score and severe pancreatitis required intensive care more often, and stayed for a longer period in intensive care units. The PANC 3 score showed sensitivity of 50%, specificity of 100%, accuracy of 91.4%, positive predictive value of 100% and negative predictive

value of 90.6% in prediction of severe acute pancreatitis. The PANC 3 score is useful to assess acute pancreatitis because it is easy and quick to use, has high specificity, high accuracy and high predictive value in prediction of severe acute pancreatitis. ^[40]

- 3. Anandaravi BN, et al.,** conducted observational study (2020-2021) in Mysore to use PANC-3 criteria for early prediction of severity of AP. PANC3 Scoring system is simple, easy to access readily available and economic, as it involves these criteria (HCT, BMI, and pleural effusion). It is a prospective study in which 100 patients were evaluated with PANC3 Scoring, who were diagnosed with AP and admitted to the department of general surgery, Mysore Medical College and Research Institute. The results showed that PANC3 score had a 96.25% specificity, 70% sensitivity, 82.35% PPV, and 92.77% NPV. ^[41]
- 4. Chandra Mohan S Monica, et al.,** did Observational study at KSHEMA, Mangalore from patients admitted during the time period from December 2020 to November 2021 to evaluate the efficacy of the PANC 3 score in predicting severity of acute pancreatitis and its relation with the clinical outcome of the disease. The PANC 3 score was determined by measuring three variables obtained within the first 24 h after diagnosis of acute pancreatitis: 1) Haematocrit; 2) body mass index (BMI); and 3) pleural effusion on the chest X-ray. It is a prospective study in which 60 patients were evaluated with PANC3 Scoring, who were diagnosed with acute pancreatitis and admitted to DEPT OF GENERAL SURGERY, K.S. Hegde Medical Academy, Mangalore. The results showed that PANC3 score had a sensitivity of 80%, and the specificity was 95.5% The PPV was 85.7%, and the NPV was 93.5%, the diagnostic accuracy is 91% in predicting severe acute pancreatitis. “ PANC3 SCORE is a useful tool to approach and

predict the severity of acute pancreatitis, due to its high efficacy, easy application and rapid results, which enables classification of cases and early treatment".^[42]

5. Rami Ahmad, et al., conducted analysis of AP in 225 patients (2014-2018). Out of 225 patients, 122 were female while 103 patients were male. CAP developed in 47 patients (20.9%) while 178 (79.1%) patients had mild AP. Overall, in-hospital mortality rate was 1.8% (n=4). ROC analysis demonstrated that CRP at admission had low discriminatory value (AUC= 0.54, p-value=0.74). CRP at 48 hours had AUC of 0.70 (p-value=0.007). At a cut-off of 150, the positive predictive value (PPV) of 150 was 30 %. The PPV of CRP at 48 hours at a cut-off of 190 was 28%. Interval change in CRP at 48 hours greater than 90 had a PPV of 26 %. Further comparison of CRP with other scoring systems like Glasgow scoring system (AUC= 0.65), NL ratio (AUC=0.54), and MCTSI was performed. Among the single predictors, although, NL ratio showed good sensitivity at a cut-off value of 4.7 (87.23%), however, its discriminatory power was negligible (AUC=0.542, p-value=0.513). The overall best performance was achieved by the MCTSI scoring system at a cut-off of 3 (AUC=0.90, sensitivity=83.33 %, specificity=100%, diagnostic accuracy=94.49%). CRP measured at admission or at 48 hours has a very limited role in the prediction of CAP. Along with other scoring systems, its negative predictive value should be used to predict cases with mild AP which can help in clinical decision making for early discharge or management of such patients on ambulatory care basis.^[43]

6. Hongsheng Wu, et al., did meta-analysis to explore its ability to predict the severity of acute pancreatitis. 41 studies with 6156 cases of acute pancreatitis, retrieved from PubMed, Cochrane Library Springer, and Embase databases, were incorporated. SROC analysis yielded an AUC of 0.85 with a sensitivity of 0.76

and specificity of 0.79. The combined NLR, PLR and DOR were 0.30, 3.66 and 12.19 respectively. Sensitivity analysis demonstrated the stability of our results after omitting any study. Finally, meta-regression analysis indicated that the description of the reference test, prospective design, blinding method and spectrum of the disease could account for heterogeneity in this meta-analysis. CRP has significant value as a biomarker for assessing AP severity.^[44]

7. Aaron D. Stirling et al. did a retrospective study of all first-time AP cases. ROC curve analysis revealed that the second day was the most useful day for repeat CRP testing. A CRP interval change of >90 mg/dL at 48 hours (+LR 2.15, -LR 0.26) corresponded to an absolute value of >150 mg/dL within 48 hours (+LR 2.32, -LR 0.25). The optimal cut-off for absolute CRP based on the revised, more stringent definition of severity was >190 mg/dL (+LR 2.72, -LR 0.24). Interval change in CRP is comparable to absolute CRP. According to this study, a rise of more than 90 mg/dL from admission, or an absolute value of more than 190 mg/dL at 48 hours, accurately predicts severe illness.^[45]

8. Shanjidah Hoque et al. conducted a prospective observational study (2016-2017) on 35 hospitalised patients diagnosed with AP at BIRDEM General Hospital in Dhaka, Bangladesh, to predict the severity of AP using biochemical markers. Among patient parameters, the P value for CRP was 0.047, and the P value for PCT was 0.032. PCT is a better diagnostic for predicting the degree and progression of organ failure in AP than CRP. The total white blood cell count was high in both the moderate and severe acute groups of pancreatitis patients. Haemoglobin levels and other baseline measures did not differ significantly between the two groups of individuals. Serum amylase and serum lipase levels were elevated in both groups. The size of the increase in amylase and lipase does

not indicate disease severity. CRP and PCT distinguish between MAP and SAP. PCT had a sensitivity of 91% and a specificity of 81%, which was higher than previous tests.^[46]

- 9. Avreen Singh Shah,et al.,** did observational study on 100 cases to see the prospects of PANC3 scoring in predicting the severity of acute pancreatitis as decided by modified Marshals score. The results showed that PANC3 score had a 96.43% specificity, 75% sensitivity, 80% positive predictive value, and 95.29% negative predictive value. Hence, the PANC3 score is a cost-effective, promising score that helps in predicting the severity of acute pancreatitis leading to prompt treatment and early referral to higher center.^[47]

MATERIALS AND METHODS

STUDY DESIGN:

Hospital based single centered, cross sectional study.

STUDY SETTING:

Medical Intensive Care Unit in the Tertiary Care Centre, Belagavi, India.

STUDY PERIOD:

Duration of 12 months from August 2023 to July 2024 after obtaining approval from IRB (Ethical and Scientific Clearance).

STUDY POPULATION:

Patients admitted to Tertiary Care Centre, Belagavi, India with clinical symptoms of AP and elevated serum amylase and lipase levels to 3 times upper limit of normal.

Inclusion criteria:

- Age > 18 years
- All patients diagnosed with acute pancreatitis (AP) typically present with a characteristic clinical history, including severe upper abdominal pain that radiates to the back and persists for more than 24 hours.
- All patients with elevated serum amylase and lipase levels to 3 times upper limit of normal.

Exclusion criteria:

- Chronic pancreatitis (including acute on chronic pancreatitis).
- Pregnant women.
- History of pancreatic carcinoma.
- Patients with:
 - Hematological diseases.
 - Connective tissue disorders.
 - Collagen vascular diseases.

SAMPLE SIZE:

Formula used is,

$$n = \frac{(1 - Sp) Z_{\alpha/2}^2}{(1 - Prev) * d^2}$$

Formula used for is, $n = Sp (1 - 5p) Z_{\alpha/2}^2 (1 - Prev) / d^2$ where n is sample size required, Sp is pre-determined values of specificity, d is the maximum marginal error required, $Z_{\alpha/2}$ is value corresponding to level of confidence required and prevalence is prevalence of disease. Specificity of PANC-3 is 92.31% in predicting severity of acute pancreatitis.

Assuming 50% prevalence, at 95% CL and 10% maximum error, sample size is given by, $n = 0.9231 \times (1 - 0.9231) \times 1.962^2 / (0.5 \times 0.1)^2 = 54.5402655$ Hence, minimum sample size required was 55.

SAMPLING TECHNIQUE:

Convenient sampling.

METHODOLOGY:

- This hospital-based, cross-sectional study included patients admitted to the Tertiary Care Centre in Belagavi, India.
- Obtained informed written consent following ethical approval.
- Patients were informed about the study's implications and outcomes in their language. They were told that they may choose whether or not to participate in the study, and that doing so would have no effect on the therapy procedure.
- This study included patients with acute pancreatitis who experienced significant upper abdomen pain extending to the back for more than 24 hours and had serum amylase and serum lipase levels three times higher than normal.

Assessment of parameters:

- Detailed history was taken from the patients or attendants including demographic details and past medical history.
- A complete physical examination with monitoring of vitals (temperature, PR, RR, and BP) was done.
- PANC 3 score is calculated by assessing 3 variables in 24 hours of diagnosis of AP: HCT, BMI, and pleural effusion on CXR.

If HCT was > 44 mg/dl, BMI was > 30 kg/m², and CXR revealed pleural effusion, case was classified as positive.

- Patients underwent comprehensive evaluation using a combination of scoring systems, including the PANC 3 score (which assesses body mass index, hematocrit, and pleural effusion), the modified Atlanta criteria, and the APACHE-II scoring system.
- C-Reactive Protein (CRP) levels were measured at 48 hours, with a threshold value of 120 mg/L or higher indicating severe acute pancreatitis.

Investigations:

- PANC 3 score was estimated on day of admission by the respective reports obtained CXR, BMI, hematocrit. HsCRP sample were sent within 24 hrs of admission.
- Other blood parameters such as serum PCT, RFT and LFT, ABG analysis along with contrast abdomen scan were considered to determine severity of AP based on Revised Atlanta Classification. Also organ failure in Revised Atlanta Classification will be determined using Modified Marshall Scoring system.

Methodology:

- Patients provided informed consent.
- Data was collected using a pre-structured and tested proforma.
- Baseline data, including socio-demographics and medical history, were gathered.
- Clinical examination and appropriate investigations were conducted.

- Cause of AP was diagnosed using history and imaging testing (ultrasound, computed tomography, and magnetic resonance).
- The study assessed hospital stay, critical care unit stay, and fatality rates.

STATISTICAL ANALYSIS

- Data was imported to MS Excel and analysed with Statistical Package for Social Sciences software version 28.
- Descriptive statistics were used to summarise quantitative factors in clinical data. The standard deviation was determined as a measure of variability.
- Qualitative factors were reported as percentages with 95% CI.
To test for statistical significance, mean values were compared between groups using the student's t test or Mann Whitney test for non-normal distributions.
- Chi-square and Fisher's exact tests were used to compare proportional differences.
- The Pearson correlation coefficient was used to investigate the type and magnitude of correlation.
- The one-way ANOVA/Kruskal Wallis test was performed to evaluate mean/distribution of variables across many groups.
- Logistic regression and Receiver Operating Characteristic (ROC) curves were used to assess the predictability of acute pancreatitis severity using PANC-3 and HsCRP levels.
- Cut-off values were determined by maximising both sensitivity and specificity.
- The level of significance (P-Value) was fixed at $P < 0.05$.

RESULTS

Table 1: Age-wise distribution of subjects

Age	Frequency	Percentage
≤ 20 years	2	3.6%
21 - 30 years	6	10.9%
31 - 40 years	20	36.4%
41 - 50 years	17	30.9%
51 - 60 years	7	12.7%
> 60 years	3	5.5%
Total	55	100%
Mean age: 40.9±11.98 years		

Graph 1: Pie chart showing Age-wise distribution of subjects

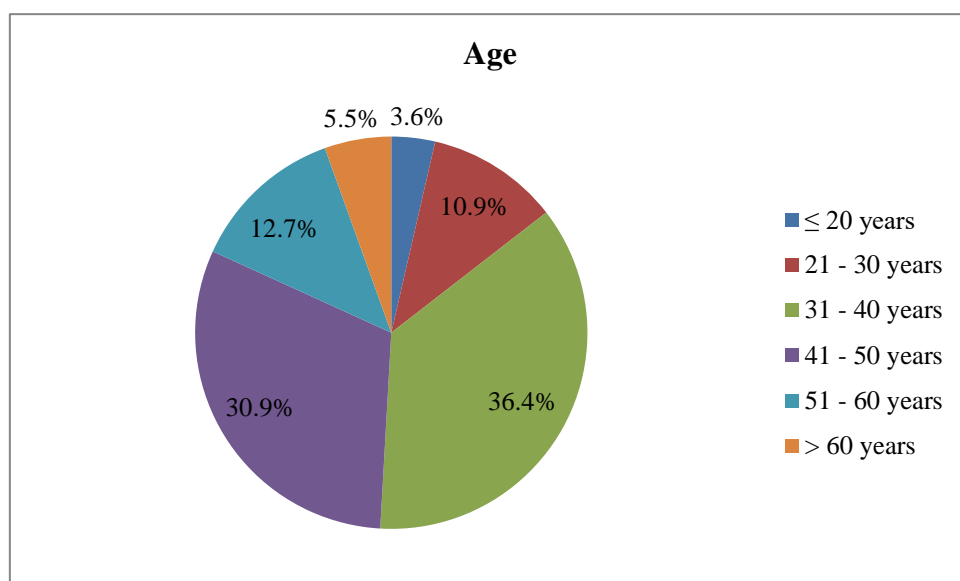


Table 1 displays the Age-wise distribution of subjects. The youngest of the 55 patients was 18 years old, while the oldest was 70. In the study population, 2 (3.6%) patients were under the age of 20; 6 (10.9%) patients were between the ages of 21 and 30; 20 (36.4%) patients were between the ages of 31 and 40; 17 (30.9%) patients were between the ages of 41 and 50; 7 (12.7%) patients were between the ages of 51 and 60; and 3 (5.5%) patients were over the age of 60. The average age was 40.9 ± 11.98 years.

Table 2: Distribution of subjects according to gender

Gender	Frequency	Percent
Males	44	80%
Females	11	20%
Total	55	100%

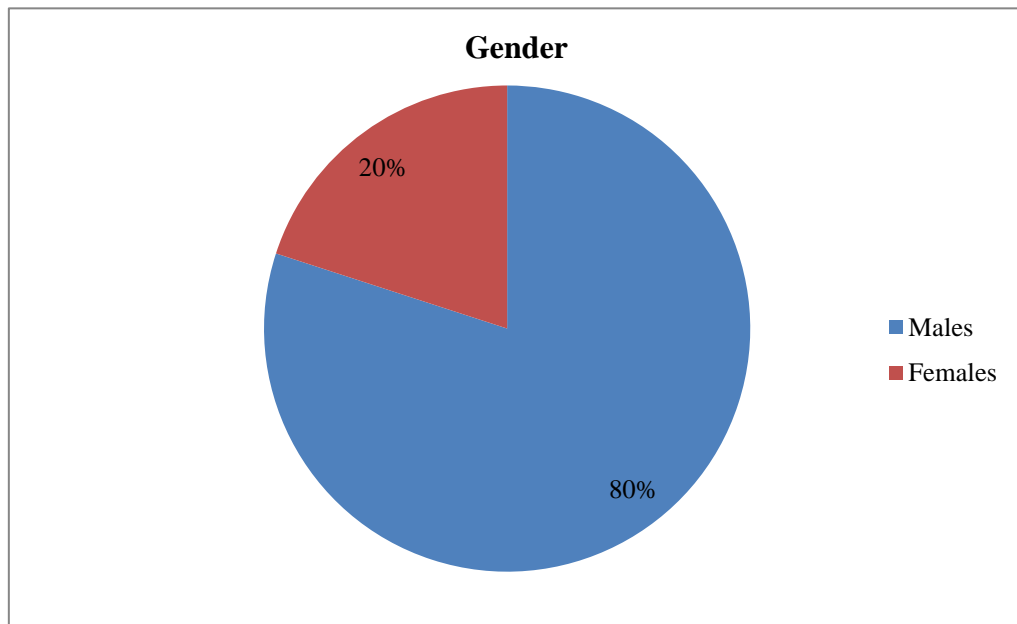
Graph 2: Pie chart showing gender distribution among subjects

Table 2 displays gender distribution of individuals. The majority of the 55 patients were male, accounting for 44 instances (80%). 11 patients were female, accounting for 20% of all patients. The male to female ratio was 4:1.

Table 3: Distribution of subjects according to age and gender

Age vs Gender	Males	Females	Total
≤ 20 years	2 (4.5%)	0 (0%)	2 (3.6%)
21 - 30 years	4 (9.1%)	2 (18.2%)	6 (10.9%)
31 - 40 years	16 (36.4%)	4 (36.4%)	20(36.4%)
> 40 years	22 (50%)	5 (45.5%)	27 (49.1%)
Total	44 (100%)	11 (100%)	55 (100%)
Mean (years)	40.4 ± 11.39	43.0 ± 14.54	40.9 ± 11.98

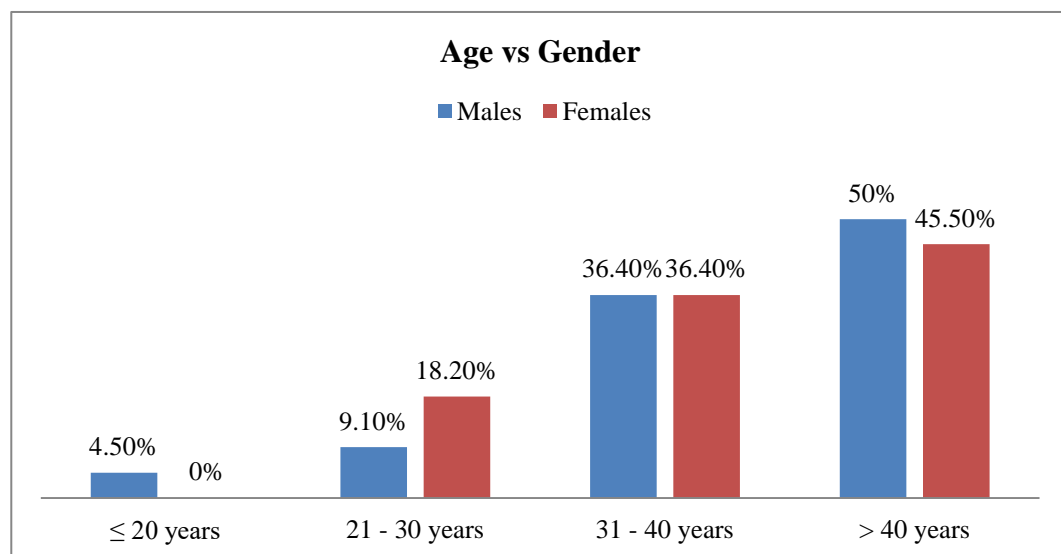
Graph 3: Column diagram showing distribution of age and gender

Table 3 illustrates the distribution of subjects by age and gender. In our study of 55 cases, the age & gender distribution the majority of patients was >40 years of which Male were 50% and Female were 45.5%, followed by 31-40 years of which Male and Female were 36.4%, followed by 21-30 years of which Male were 9.1% and Female were 18.2%, ≤ 20 years of which Male were 2.5% and none were Female.

Table 4: Distribution of subjects according to habits

Habits	Frequency	Percent
Smoking	4	7.3%
Alcohol	14	25.5%
Both	9	16.4%
None	28	50.9%
Total	55	100%

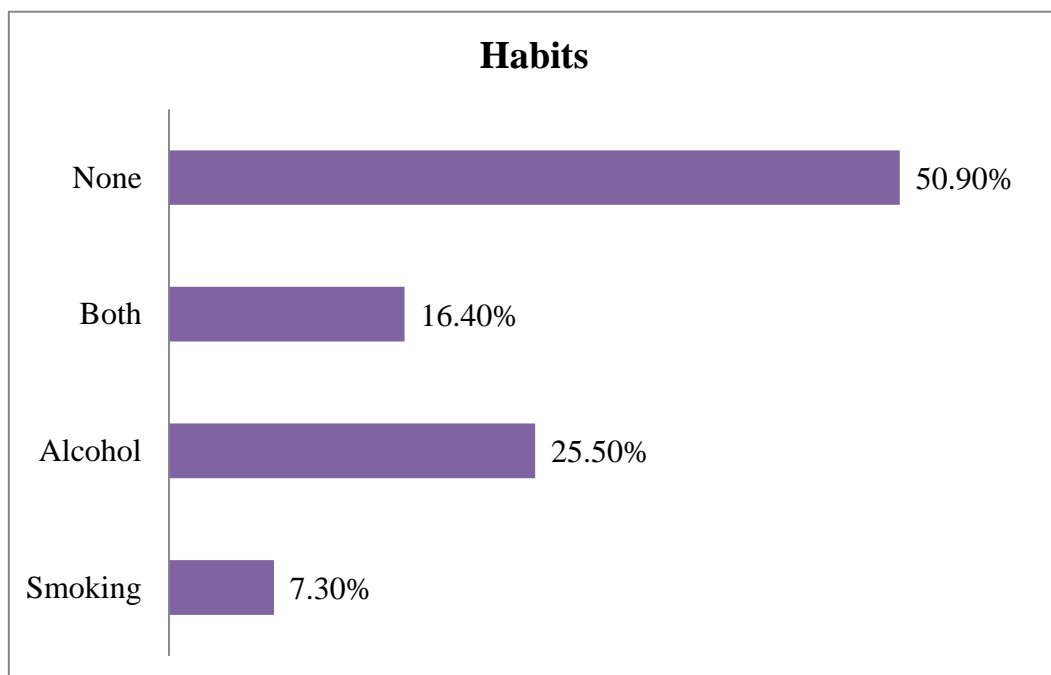
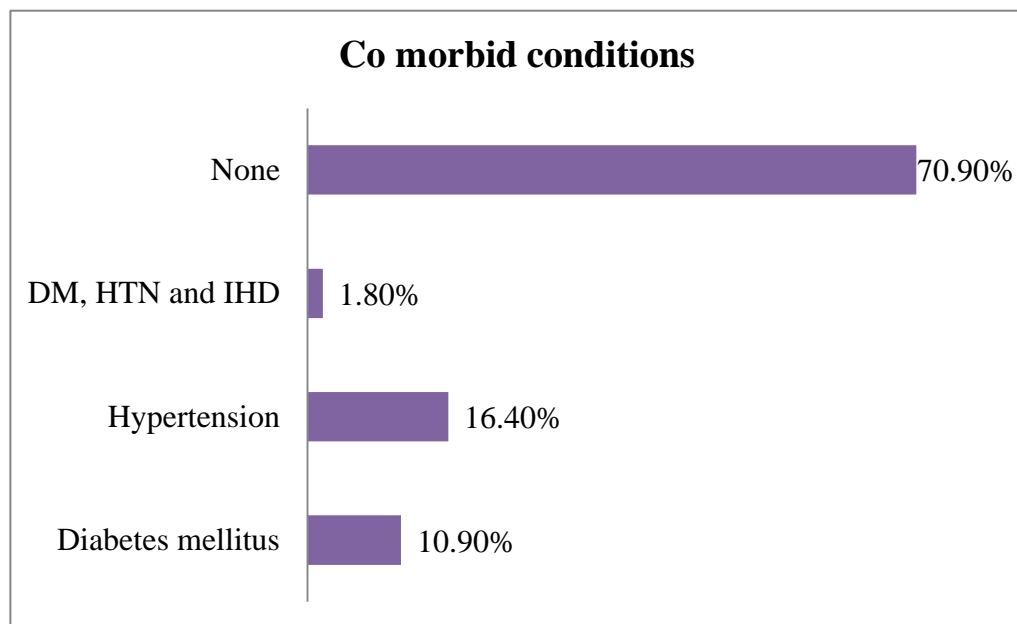
Graph 4: Bar diagram showing distribution of subjects according to habits

Table 4 illustrates distribution of subjects according by habits. Commonest was alcohol consumption which was seen in 14 (25.5%) cases, smoking was present in 4 (7.3%) cases, 9 (16.4%) patients were both smokers and consumed alcohol.

Table 5: Distribution of subjects according to co morbid conditions

Co morbid conditions	Frequency	Percent
Diabetes mellitus	6	10.9%
Hypertension	9	16.4%
DM, HTN and IHD	1	1.8%
None	39	70.9%
Total	55	100%

Graph 5: Bar diagram showing distribution of subjects according to co morbid conditions

Illustrates distribution of subjects according byco morbid conditions. Commonest co morbid condition was Hypertension which was seen in 9 (16.4%) cases, 6 (10.9%) patient hadDiabetes mellitus, 1 (1.8%) patient hadDM, HTN and IHD.

Table 6: Distribution of subjects according to BMI

Body Mass Index (kg/m ²)	Frequency	Percent
< 18.5 (Underweight)	0	0%
18.5 - 24.9 (Normal)	13	23.6%
25 - 29.9 (Overweight)	22	40%
≥ 30 (Obese)	20	36.4%
Total	55	100%
Mean BMI = 28.1 ± 3.31 kg/m²		

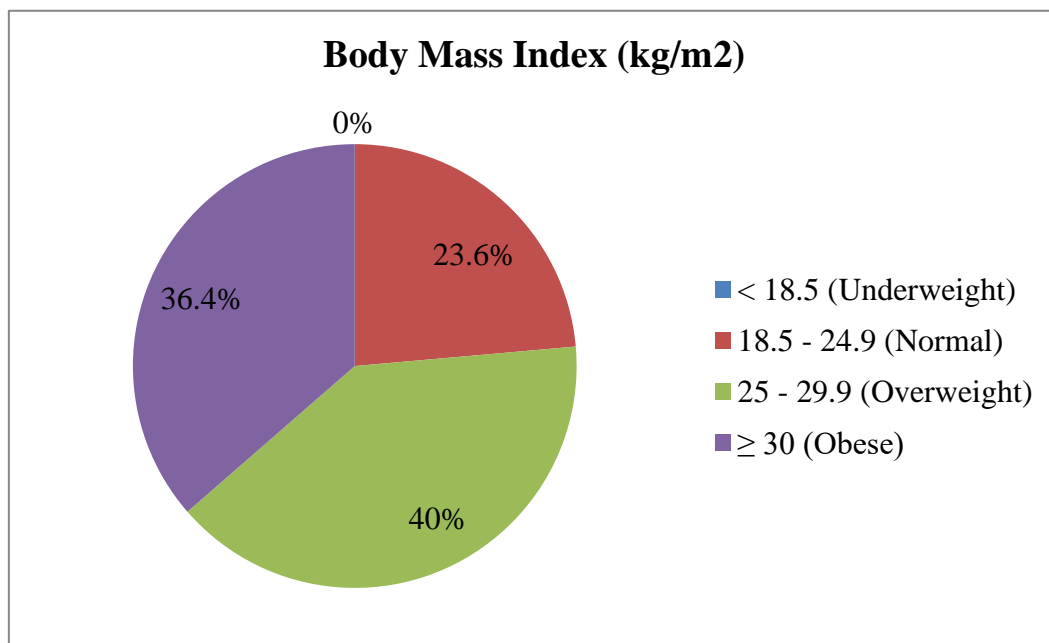
Graph 6: Pie chart showing BMI distribution among subjects

Table 6 illustrates distribution of subjects according by BMI. 13 (23.6%) patients had normal BMI, 22 (40%) patients were overweight and 20 (36.4%) patients were obese.

Graph 7: Distribution of subjects according to Lab parameters

Lab parameters	Mean	SD
Amylase (U/L)	663.5	474.82
Lipase (U/L)	1056.2	1073.57
Serum Procalcitonin (ng/mL)	2.6	11.74
Total Bilurubin (mg/dL)	2.0	1.99
Direct Bilurubin (mg/dL)	1.2	1.53
Indirect Bilurubin (mg/dL)	0.8	0.74
Total Protein (g/dL)	6.7	0.79
Albumin (g/dL)	3.8	0.77
Globulin (g/dL)	2.9	0.68
A:G Ratio	1.4	0.41
ALT (SGPT) (U/L)	52.2	69.85
SGOT (AST) (U/L)	84.2	132.77
Alkaline Phosphatase (IU/L)	107.9	51.21
RBS (mg/dL)	167.5	106.63
Blood Urea (mg/dL)	39.2	39.69
BUN (mg/dL)	18.4	18.51
Serum Creatinine (mg/dL)	1.6	1.95

Sodium (mmol/L)	133.9	5.44
Potassium (mmol/L)	4.4	1.01
Chloride (mmol/L)	98.5	5.74
Bicarbonate (mmol/L)	18.7	4.65
Haemoglobin (g/dL)	13.5	3.10
Hematocrit (L/L)	42.4	9.11
RBC Count (million cells/mcL)	4.6	1.03
WBC Count(cells/mcL)	12.4	6.16
Platelet Count (thousand cells/mcL)	253.3	157.56
Neutrophils(%)	78.2	9.07
Lymphocytes(%)	12.9	7.22
Monocytes(%)	1.2	1.92
Eosinophils(%)	6.5	2.32
Basophils(%)	0	0

Table 7 illustrates distribution of subjects by Lab parameters.

Table 8: Distribution of subjects according to severity of AP based on Revised Atlanta Classification

Severity of AP based on Revised Atlanta Classification	Frequency	Percent
A: MAP	37	67.3%
B: MSAP	5	9.1%
C: SAP	13	23.6%
Total	55	100%

Graph 7: Pie chart showing severity of AP based on Revised Atlanta Classification

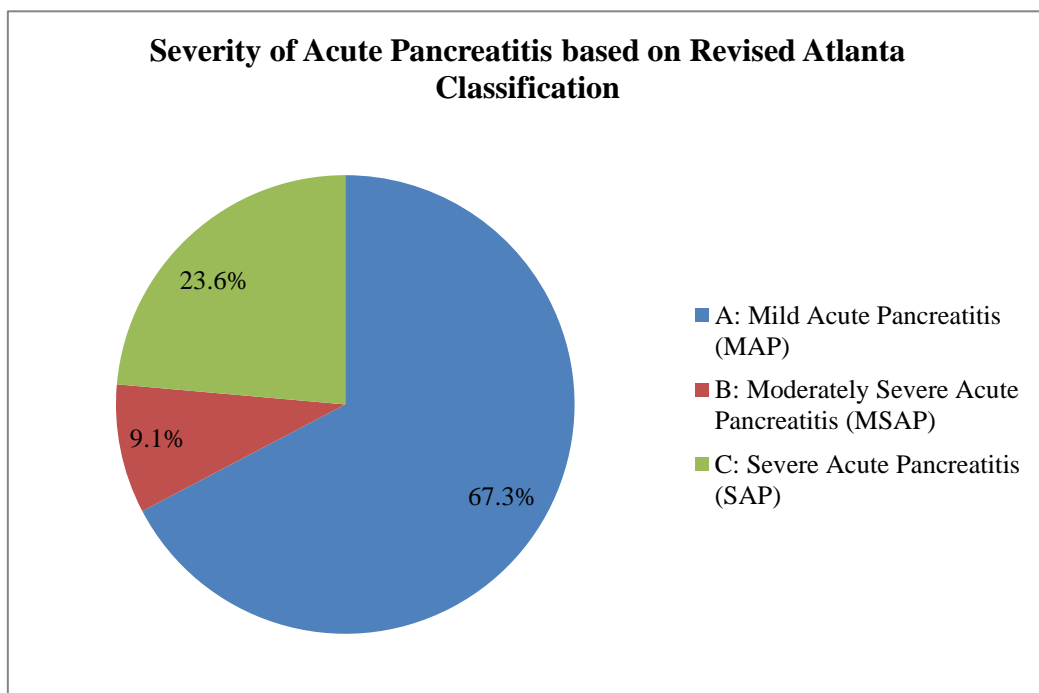


Table 8 shows the severity of AP based on Revised Atlanta Classification. 37 (67.3%) patients had MAP, 5 (9.1%) patients had MSAP and 13 (23.5%) patients had SAP.

Table 9: Distribution of subjects according to PANC-3 Score

PANC-3 Score	Frequency/ Mean	Percent/ SD
Positive	7	12.7%
BMI (kg/m²)	31.6	1.41
Hematocrit (mg/dL)	55.2	12.16
Pleural Effusion	7	100%
Negative	48	87.3%
BMI (kg/m²)	27.6	3.19
Hematocrit (mg/dL)	40.5	6.98
Pleural Effusion	0	0%

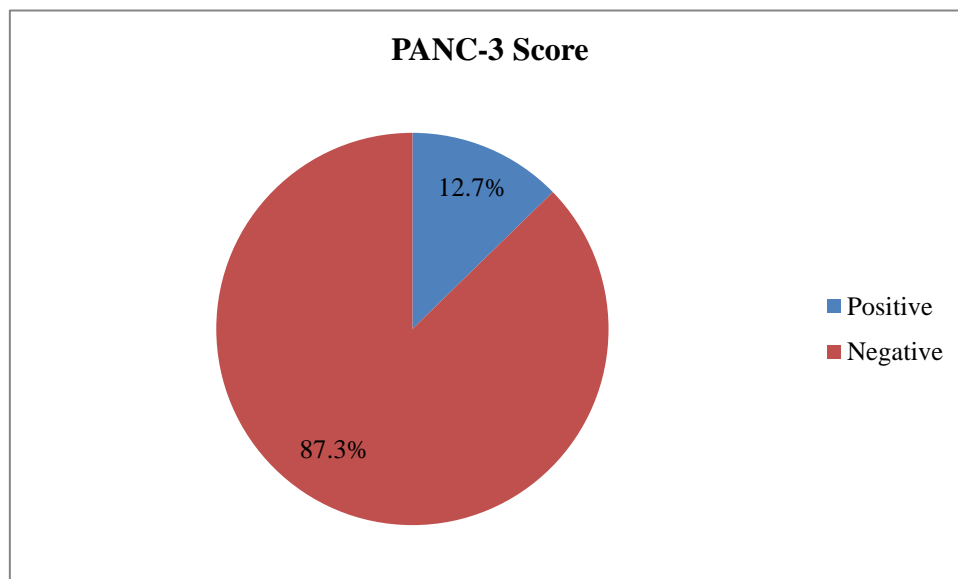
Graph 8: Pie chart showing distribution of PANC-3 Score

Table 9 shows the distribution of subjects according to PANC-3 Score. In the PANC-3 Score, the case was considered positive if HCT was >44 mg/dl, BMI >30 kg/m², and pleural effusion was detected on CXR. Among 55 patients in this study 7 (12.7%) patients had positive score.

Table 10: Comparison of PANC-3 Score with Revised Atlanta Classification among subjects

PANC-3 Score	Revised Atlanta Classification n (%)		Total n (%)
	SAP	MAP or MSAP	
Positive	9 (60%)	6 (40%)	15 (100%)
Negative	4 (10%)	36 (90%)	40 (100%)
Total	13 (23.6%)	42 (76.4%)	55 (100%)
p- value	0.026	χ^2 value	4.989

Table 10 shows the Comparison of PANC-3 Score with Revised Atlanta Classification.

Among 15 patients with +PANC-3 Score, 9 (60%) patients had SAP according to Revised Atlanta Classification while 6 (40%) patients had MAP or MSAP. Among 40 patients with Negative PANC-3 Score, 4 (10%) patients had SAP while 36 (90%) patients had MAP or MSAP.

On comparison of PANC-3 Score with Revised Atlanta Classification, p-value (p=0.026) was significant.

Table 11: Distribution of subjects according to severity of AP based on Hs-CRP

Severity of AP based on Hs-CRP	Frequency	Percent
> 100mg/L (SAP)	20	36.4%
≤ 100 mg/L	35	63.6%
Total	55	100%
Mean Hs-CRP = 90.8 ± 83.37 mg/L		

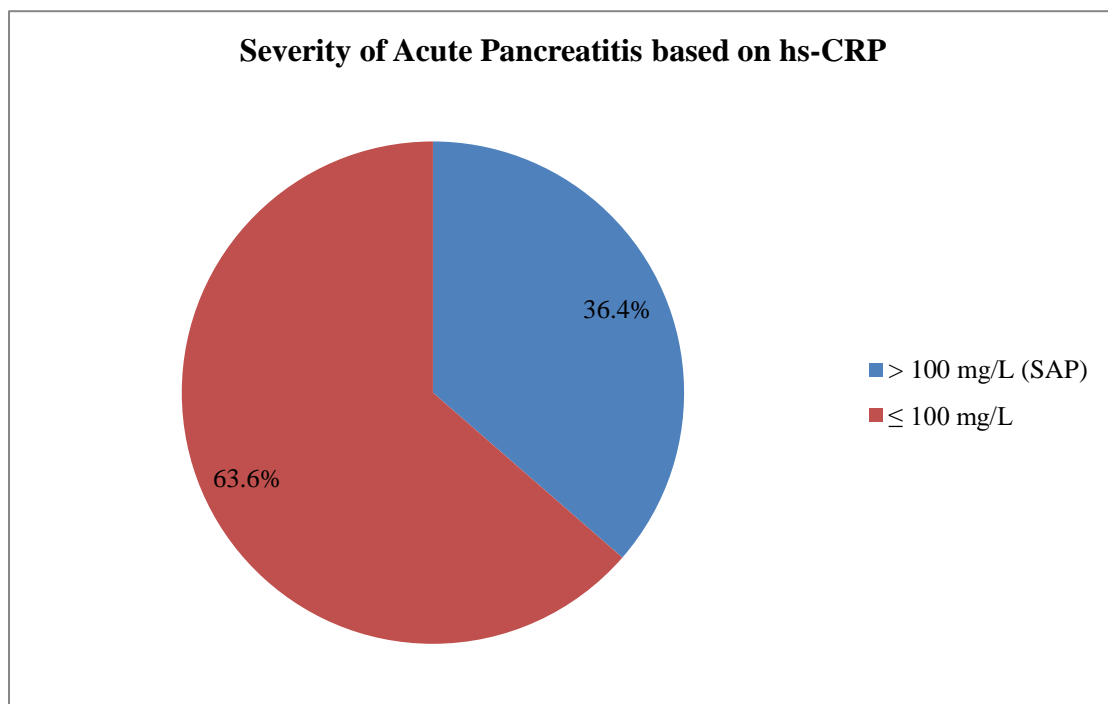
Graph 9: Pie chart showing distribution of Hs-CRP

Table 11 illustrates distribution of subjects by Hs-CRP. Among 55 patients in this study 20 (36.4%) patients had Hs-CRP levels > 100 mg/L. Mean Hs-CRP level was 90.8 ± 83.37 mg/L.

Table 12: Comparison of Hs-CRP levels with Revised Atlanta Classification among subjects

Hs-CRP	Revised Atlanta Classification n (%)		Total n (%)
	SAP	MAP or MSAP	
> 100mg/L (SAP)	11 (55%)	9 (45%)	20 (100%)
≤ 100 mg/L	2 (5.7%)	33 (94.3%)	35 (100%)
Total	13 (23.6%)	42 (76.4%)	55 (100%)
Mean (mg/L)	134.7±77.34	84.3±83.02	90.8 ± 83.37
p- value	< 0.001		

Table 12 shows the Comparison of Hs-CRP levels with Revised Atlanta Classification.

Among 20 patients with Hs-CRP levels > 100 mg/L, 11 (55%) patients had SAP according to Revised Atlanta Classification while 9 (45%) patients had MAP or MSAP. Among 35 patients with Hs-CRP levels ≤ 100 mg/L, 2 (5.7%) patients had SAP while 33 (94.3%) patients had MAP or MSAP.

On comparison of Hs-CRP levels with Revised Atlanta Classification, p-value (p <0.001) was significant.

Table 13: Analysis of PANC-3 Score and Hs-CRP

Test variable	PANC-3 Score	Hs-CRP
Sensitivity	69.2%	84.6%
Specificity	85.7%	78.6%
PPV	60%	55%
NPV	90%	94.3%
Diagnostic Accuracy	81.8%	80%

Table 13 shows the sensitivity, specificity, PPV, NPV and diagnostic accuracy of PANC-3 Score and Hs-CRP to predict the severity of AP.

For PANC-3 Score, the sensitivity was 69.2% and specificity was 85.7% PPV and NPV were 60% and 90% respectively in predicting severity of AP. Diagnostic accuracy was 81.8%.

For Hs-CRP, the sensitivity and specificity were 84.6% and 78.6% respectively, PPV and NPV were 55% and 94.3% respectively in predicting severity of AP. Diagnostic accuracy was 80%.

Graph 10: Receiver Operator Characteristic Curveshowing the diagnostic ability ofHs-CRP levelsto predict severity of AP

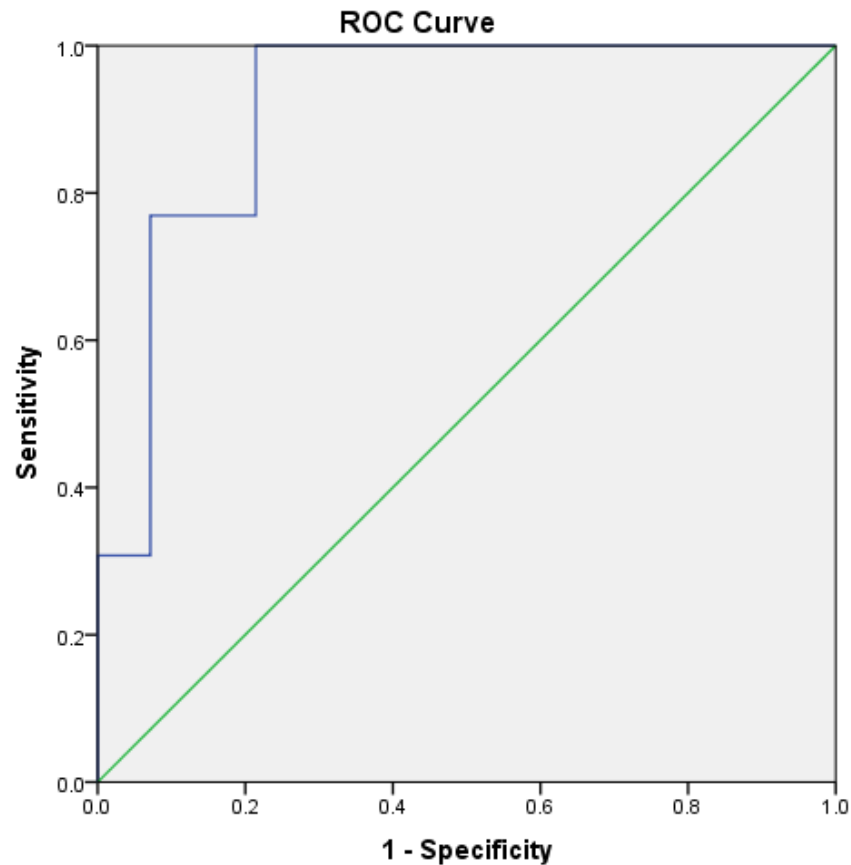


Figure 10 illustrates the Receiver Operator Characteristic Curve showing the diagnostic ability of Hs-CRP levels to predict severity of AP.

There exists a significant association among the Hs-CRP levels and severity of Acute Pancreatitis. The AUC-ROC is 0.918 (p-value < 0.001). By using Receiver Operator Characteristic curve, Hs-CRP levels > 100 mg/L (optimum cut off criterion) predicts SAP with a sensitivity of 84.6% and specificity of 78.6%.

DISCUSSION

AP is gastrointestinal emergency with highly variable clinical course, varying from mild self-limiting inflammation to severe necrotizing pancreatitis with systemic complications. Early detection of patients at risk for severe disease is critical for appropriate treatment and better prognosis. We assessed role of PANC 3 score and HsCRP to predict severity of AP in patients at tertiary hospital over one year.

Our findings suggest that the PANC 3 score is consistent tool for early risk stratification in AP. Patients with a higher PANC 3 score were significantly likely to develop SAP, as defined by the Revised Atlanta Classification. This aligns with previous research indicating that the PANC 3 score, which incorporates HCT, BMI and pleural effusion, effectively predicts disease severity. Elevated hematocrit suggests hemoconcentration and potential pancreatic necrosis, while obesity (high BMI) is recognized risk factor to poor outcomes in AP. The presence of pleural effusion further indicates systemic inflammatory response and disease progression.

Similarly, HsCRP levels measured within 48 hours of admission were strongly related with severity of AP. CRP, an acute-phase reactant, is a well-established inflammatory marker that correlates with pancreatic necrosis and organ failure in SAP. Our results support existing literature that HsCRP values above a specific threshold serve as a valuable biomarker in predicting complications such as POF and necrotizing pancreatitis.

When comparing predictive performance of PANC 3 score and HsCRP, both demonstrated significant associations with disease severity. However, combining

these markers may provide a more comprehensive risk assessment, allowing for better triaging and early management decisions.

Baseline characteristics of the study participants:

The subjects' age distribution was as follows: the majority (36.4%) were between 31 and 40 years old, 3.6% were under 20 years old, 10.9% were between 21 and 30 years old, 30.9% were between 41 and 50 years old, 12.7% were between 51 and 60 years old, and 5.5% were over 60 years old. The average age in this study was 40.9 ± 11.98 years. The majority of them were guys, accounting for 80%. 11 patients were female, accounting for 20% of all patients. The male to female ratio was 4:1.

Sunil Kumar Meena et al.^[39] conducted a cross-sectional study to assess PANC-3 score in predicting SAP. The study population was predominantly comprised of individuals between 31-40 years old (26%), followed by those aged 51 to 60 years (20%). The mean age of participants is 44.74 yrs, with a standard deviation of 15.69 years. Notably, the age demographics of this study align with those of previous investigations.

Murilo Gamba BEDUSCHI, et al.^[40] conducted a cross-sectional study to investigate the usefulness of the PANC 3 score in predicting acute pancreatitis severity and its relationship to clinical outcomes. The final study population consisted of 58 patients, the majority of whom were women (63.8%), with an average age of 48.5 years (range 20-86). The demographic profiles of the participants in this study were comparable to those in the current investigation.

Anandaravi BN et al.,^[41] conducted a prospective analytical observational study to apply PANC-3 criteria to predict severity of AP. The majority of patients

were between the ages of 35 and 55, with a mean age of 48 when they presented with severe AP. The bulk of the patients (84) were men (84%). The baseline characteristics of the participants in this study were similar to those of the current study participants.

Chandra Mohan S Monica et al.^[42] conducted a prospective observational study to assess PANC 3 score to predict severity of AP and relationship to disease's clinical prognosis. The majority of patients were between ages of 35 and 55, with severe acute pancreatitis presenting at an average age of 48 years. The bulk of our study's patients (54) were men (90%). The baseline characteristics of the participants in this study were similar to those of the current study participants.

Rami Ahmad et al.^[43] conducted a retrospective analysis of prospectively collected data on acute pancreatitis episodes was conducted to evaluate the diagnostic accuracy and predictive value of C-Reactive Protein (CRP) at various time points and cut-off values, in comparison to other predictors such as neutrophil-to-lymphocyte ratio (NLR), Glasgow scoring system, and modified CT severity index (MCSTI). The analysis was based on 225 cases, consisting of 122 females and 103 males, with a median age slightly above 65. Notably, the demographic characteristics of this study's participants differed from those of current study.

Study by Hongsheng Wu et al.,^[44] which was a systematic review and meta-analysis, sought to investigate CRP's potential to predict the severity of acute pancreatitis. The participants in this study had similar baseline characteristics in terms of age to those in the current investigation.

Aaron D. Stirling et al.^[45] conducted a retrospective analysis to evaluate interval changes in CRP with absolute CRP measurement, in order to determine

whether this gives equivalent, or better, severity categorisation for AP. The cohort's median age was 42 years (range 12-91). The participants in this study had similar baseline characteristics in terms of age to those in the current investigation.

The study by Shanjidah Hoque et al.,^[46] which was a prospective observational study, sought to predict the severity of acute pancreatitis using biochemical markers. C-reactive protein (CRP) and procalcitonin (PCT) can distinguish moderate from severe acute pancreatitis. 54% were male, with the remaining 46% female. As a result, men participants outnumbered females, with a ratio of 1.2:1. The age distribution of all patients ranged from 28 to 76 years, with the youngest patient being 28 and the oldest being 76. Ten (29%) of the total patients were between the ages of 46 and 55, while eight (23%) were between the ages of 56 and 65. The participants in this study had similar baseline characteristics in terms of age to those in the current investigation.

The study by Avreen Singh Shah et al.,^[47] observational study, sought to determine PANC3 scoring to predict severity of AP as determined according to modified Marshals score. Majority of patients were between the ages of 30 and 45, with a mean age of 47.3 years when they presented with severe acute pancreatitis. It was discovered that all kinds of pancreatitis were more prevalent in men than in women. The demographic profiles of the participants in this study were consistent with those of the current study's population.

Morbidity profile of the study participants:

In this current investigation, the most common habit was alcohol drinking, which was observed in 25.5% of the cases; smoking was found in 7.3% of the cases; and 16.4% of the patients were both smokers and drinkers.

In terms of co-morbid disorders, the most prevalent was hypertension, which was seen in 16.4% of cases; 10.9% of patients had diabetes mellitus; and 1.8% had DM, HTN, and IHD.

Sunil Kumar Meena et al.^[39] conducted a cross-sectional investigation and discovered that biliary tract pathology was the most common etiological component in our study (70%), alcohol (30%). The features of the participants were similar to those of the current study participants.

Murilo Gamba BEDUSCHI et al.,^[40] in a cross-sectional investigation, found that the aetiology of AP includes biliary tract illness (84.5%), hypertriglyceridemia (5.2%), alcohol usage (3.4%), neoplasms (3.4%), and idiopathic cases. Features of the participants in this study were similar to those of the current study participants.

Study by Anandaravi BN, et al.,^[41] which was a prospective analytical observational study, found that the most common aetiology was alcohol consumption (70% of cases), with gallstones coming in second.

Study by Chandra Mohan S Monica et al.,^[42] which was a prospective observational analysis, found that common etiology is alcohol consumption (85%), with gallstones coming in second. The features of the participants in this study were similar to those of the current study participants.

Rami Ahmad et al.^[43] found that the most common cause of index episodes of acute pancreatitis was biliary pancreatitis (57.8%). The features of the participants in this study were similar to those of the current study participants.

Aaron D. Stirling et al. ^[45] conducted a retrospective research and discovered that the leading cause was alcohol (n = 157, 47%), followed by biliary causes (n = 120, 36%). Idiopathic AP was discovered in 39 individuals (12%).

Study by Avreen Singh Shah et al., ^[47] which was a prospective observational study, found that common etiology was alcohol consumption (50%), with gallstones coming in second place. The features of the participants in this study were related to current study participants.

Clinical characteristics:

23.6% of the patients had a normal BMI, 40% were overweight, and 36.4% were obese. Sunil Kumar Meena et al. ^[39] conducted a cross-sectional study and found that mean BMI was 23.65 kg/m² among moderate type and 25.27 kg/m² among severe type (p-value = 0.001). Features of participants in this study were similar to those of the current study participants. Murilo Gamba BEDUSCHI et al. ^[40] conducted a cross-sectional investigation and found that the mean BMI was 29.9 kg/m². The features of the participants in this study were similar to those of the current study participants.

Anandaravi BN et al., ^[41] in a prospective analytical observational analysis, found that patients' mean BMI was 25.08 kg/m² in the mild, 26.09 kg/m² in moderate type, and 30.87 kg/m² in the severe AP. The features of the participants in this study were similar to those of the current study participants.

According to Chandra Mohan S Monica et al., ^[42] mean BMI was 25.076 kg/m² in mild type, 26.09 kg/m² in moderate, and 30.87 kg/m² in severe AP. This

was a prospective observational study. The features of the participants in this study were similar to those of the current study participants.

Avreen Singh Shah et al. ^[47] conducted a prospective observational study and found that mean BMI was 25.04 kg/m² in mild type, 26.08 kg/m² in moderate type, and 30.99 kg/m² in severe type. The features of the participants in this study were similar to those of the current study participants.

Severity of AP based on Revised Atlanta Classification

In the present study, 67.3% of patients had MAP, 9.1% had MSAP, and 23.5% had SAP.

Sunil Kumar Meena et al. ^[39] conducted a cross-sectional investigation and found that 22% (11 patients) had severe illness according to the modified ATLANTA categorisation. The participants in this study had similar baseline characteristics in terms of age to those in the current investigation. Murilo Gamba BEDUSCHI et al. ^[40] conducted a cross-sectional investigation and found MAP in 75.9%, MSAP in 6.9%, SAP in 17.2%. Features of the participants in this study were comparable to those of the current study participants. Anandaravi BN et al., ^[41] in a prospective analytical observational analysis, found that 55% of cases were light AP, 25% were moderate AP, and 20% were severe kind. The features of the participants in this study were similar to those of the current study participants. Study by Chandra Mohan S Monica et al., ^[42] which was a prospective observational study, found that in 60 patients, 55% had MAP, 25% had MSAP, and 20% had SAP. Features of the participants in this study were similar to those of the current study participants.

According to Rami Ahmad et al.,^[43] an investigation of prospectively maintained data for index bouts of acute pancreatitis revealed that CAP developed in 47 patients (20.9%) using the updated Atlanta classification. Mild AP was observed in 178 cases (79.1%). The features of the participants in this study were similar to those of the current study participants. Avreen Singh Shah et al.,^[47] in a prospective observational analysis, found that 59% of cases were MAP, 25% were moderate AP, and 16% were SAP. Features of the participants in this study were similar to those of the current study participants.

PANC-3 Score:

In the current investigation, a positive PANC-3 Score was defined as serum haematocrit >44 mg/dl, BMI >30 kg/m², and pleural effusion on chest X-ray. Among the 55 patients in this study, 12.7% had a favourable score. Among the 15 patients with a positive PANC-3 score, 9 (60%) had SAP according to the Revised Atlanta Classification, while 40% had MAP or MSAP. Out of 40 individuals with a negative PANC-3 score, 10% had SAP according to the Revised Atlanta Classification, while 90% had MAP or MSAP. A significantly significant p-value ($p = 0.026$) was obtained when the PANC-3 Score was compared to the Revised Atlanta Classification.

Sunil Kumar Meena et al.^[39] conducted a cross-sectional study and discovered that of 38 patients with one + or - PANC score, 25 had MAP, 11 had moderate AP, and 2 had SAP. Among 9 individuals with two + criteria, one had MAP, two had moderate pancreatitis, and six had SAP, whereas three had severe acute pancreatitis. The participants in this study had similar baseline characteristics in terms of age to those in the current investigation.

Murilo Gamba BEDUSCHI et al.^[40] conducted a cross-sectional investigation and found that PANC 3 score was + in 8.6%. All patients with a positive PANC-3 score had severe acute pancreatitis. However, half of the patients had severe illness, while the rest got a negative score. The features of the participants in this study were similar to those of the current study participants.

Anandaravi BN et al.^[41] conducted a prospective analytical observational study and found that mean BMI was 25.07 kg/m² in MAP, 26.09 kg/m² in moderate, and 30.87 kg/m² in SAP. Pleural effusion was detected among 25 cases, with SAP accounting for 17 of them (85%). HCT was calculated, and average HCT in mild, moderate, and SAP patients was 34.5%, 42.6% and 46.8%, respectively. Among 20 SAP cases, 14 (70%) had all three parameters positive, four (20%) had two parameters positive, and two (10%) had only one parameter positive. The features of the participants were similar to current study participants.

The study by **Chandra Mohan S Monica, et al.**,^[42] which was a Prospective Observational study showed that mean BMI were 25.07 kg/m² in MAP, 26.09 kg/m² in moderate AP, and 30.87 kg/m² in SAP. Pleural effusion in 18 patients, 13 patients of SAP had pleural effusion (73%). Mean HCT in patients of MAP, moderate, and SAP was 34.5%, 42.6% and 46.8%. Among 15 cases of SAP, 12(80%) had all 3 parameters +, 2(13%) had 2 parameters +, and 1(7%) had 1 parameter +. These characteristics were similar to that of the current study participants.

Avreen Singh Shah et al.^[47] conducted a prospective observational study and found that the mean BMI was 25.03 kg/m² in MAP, 26.08 kg/m² in moderate and 30.99 kg/m² in SAP. There was pleural effusion in 18 individuals. Majority of SAP patients (87.5%) had a pleural effusion. HCT in mild, moderate, and SAP patients

was 35.5%, 41% and 45.91%. Among 16 SAP cases, 12 (75%) had all three parameters positive, two (12.5%) had two parameters positive, and two (12.5%) only had one parameter positive. All patients had at least one parameter positive, resulting in an abnormal PANC3 score. These characteristics of the participants of were similar to current study participants.

Hs-CRP:

In the current investigation, 36.4% of patients had Hs-CRP values greater than 100 mg/L. The average hsCRP level was 90.8 ± 83.37 mg/L.

Among 20 patients with Hs-CRP levels greater than 100 mg/L, 55% had SAP according to the Revised Atlanta Classification, while 45% had MAP or MSAP. Among 35 patients with Hs-CRP levels ≤ 100 mg/L, 5.7% had SAP and 94.3% had MAP or MSAP, according to the Revised Atlanta Classification.

A significantly significant difference ($p < 0.001$) was seen between Hs-CRP levels and the Revised Atlanta Classification.

Sunil Kumar Meena et al.^[39] conducted a cross-sectional investigation and found that CRP at 48 hours were 4.7 mg/dl in MAP, 8.9 mg/dl in moderate and 12.7 mg/dl in SAP. The features of the participants in this study were similar to those of the current study participants.

Aaron D. Stirling et al.^[45] conducted a retrospective analysis and discovered that interval variations in CRP (DCRP) correlated well with Atlanta graded disease severity. The mean DCRP was considerably higher in the severe AP group compared to the mild group at 24, 48, and 72 hours. The biggest difference in mean DCRP between mild and severe disease occurred at 48 hours (29.5 mg/dL versus 318.9

mg/dL), representing a 10.8-fold increase. This corresponds to the lowest p value according to 2-way ANOVA ($p = 0.00022$) The features of the participants in this study were comparable to current study.

Study by Shanjidah Hoque et al.,^[46] which was a prospective observational analysis, found that CRP levels were elevated in 16 patients out of 21 with MAP and 12 of 14 with SAP. About 80% of patients had elevated CRP. CRP levels were higher in patients with SAP than in those with MAP. Circulating C-reactive protein levels with MAP (range: 3.7-101) and SAP (range: 6.0-113.6). CRP has been shown to be an effective severity predictor, with a cut-off level of 100 mg/l used to discriminate between mild and severe disease. The features of the participants in this study were similar to those of the current study participants.

Analysis of PANC-3 Score and Hs-CRP:

In this study, the PANC-3 Score had a sensitivity of 69.2% and a specificity of 85.7%. The positive and negative predictive values were 60% and 90%, respectively, in predicting the severity of Acute Pancreatitis. The diagnosis accuracy was 81.8 percent.

The sensitivity and specificity of Hs-CRP were 84.6% and 78.6%, respectively, with positive and negative predictive values of 55% and 94.3% to predict severity of AP. The diagnosis accuracy was 80 percent.

Sunil Kumar Meena et al.^[39] conducted a cross-sectional investigation that found PANC 3 to be 81.82% sensitive and 92.31% specific. The PPV was 75% and NPV was 94.7% to predict SAP (p -value=0.005), indicating considerable and fair

level of agreement. The features of the participants in this study were similar to those of the current study participants.

Murilo Gamba BEDUSCHI et al.^[40] conducted a cross-sectional investigation and found that PANC 3 score had a sensitivity of 50%, specificity of 100%, accuracy of 91.4%, PPV of 100%, and NPV of 90.6% in predicting SAP. The features of the participants in this study were similar to those of the current study participants.

Anandaravi BN et al.^[41] in a prospective analytical observational analysis, found that sensitivity of the PANC3 score was 70%, specificity was 96.25%, PPV was 82.35%, and NPV was 92.77% to predict SAP. Diagnosis accuracy is 91 percent. The features of the participants in this study were similar to those of the current study participants.

According to Chandra Mohan S Monica et al.^[42] a prospective observational study, the PANC3 score had a sensitivity of 80%, specificity of 95.5%, PPV of 85.7%, and NPV of 93.5% in predicting SAP. Diagnosis accuracy is 91 percent. The features of the participants in this study were similar to those of the current study participants.

According to Hongsheng Wu et al.'s^[44] systematic review and meta-analysis, the NLR and PLR of CRP for predicting AP severity were 0.30 (0.23-0.40) and 3.66 (2.94-4.55), respectively. The specificity subgroup analysis revealed that when the cut-off value was ≥ 200 mg/L, the specificity of CRP for predicting AP severity reached 0.88 (0.82-0.94), considerably higher than the other two subgroups with the random-effect model ($\chi^2 = 12.88$, $df=2$, $p<0.01$). The features of the participants in this study were similar to those of the current study participants.

Aaron D. Stirling et al.,^[45] in a retrospective research, found that interval change in CRP is similar to absolute CRP in predicting severe illness at 24, 48, and 72 hours. Notably, absolute CRN values exhibited higher Area Under the Curve (AUC) values, although this difference was not statistically significant. The optimal time interval for calculating CRP changes to predict severe disease accurately was found to be 48 hours, with a maximum AUC of 0.816 (95% CI: 0.756-0.867). Interestingly, the AUC remained above 0.75 across all time intervals, demonstrating sufficient discriminatory power. For moderate disease, the 48-hour time point also yielded the highest predictive accuracy (AUC: 0.760, 95% CI: 0.693-0.817). Furthermore, the demographic characteristics of this study's participants closely aligned with current study.

Study by Shanjidah Hoque et al.,^[46] which was a prospective observational study, reported that the sensitivity of CRP was 78% and the specificity was 69% with a cut-off value of 100 mg/L. The NPV and PPV for CRP were 89% and 47%, respectively. The features of the participants in this study were similar to those of the current study participants.

Avreen Singh Shah et al.,^[47] in a prospective observational research, found that sensitivity of PANC3 was 75% and specificity was 96.43%. Predicting SAP had a PPV of 80% and an NPV of 95.29%. The diagnosis accuracy is 93 percent. The features of the participants in this study were similar to those of the current study participants.

STRENGTHS:

1. Current study highlights utility of the PANC 3 score and HsCRP as early, cost-effective, and easily accessible biomarkers to predict severity of AP. Their use in can aid in prompt and timely risk stratification and management.
2. Both PANC 3 score and HsCRP provide objective, quantifiable parameters that minimize subjectivity in assessing disease severity, enhancing the reliability of severity prediction.
3. Conducted in a tertiary care center, the study reflects real-world patient demographics, disease burden, and resource availability, increasing its clinical relevance and applicability.
4. The study includes a one-year duration, allowing for the assessment of seasonal variations and diverse patient presentations, strengthening the robustness of the findings.
5. By evaluating both the PANC 3 score and HsCRP, the study provides insight into their individual and combined predictive capabilities, potentially guiding future research and clinical protocols.
6. The findings support use of them in timely decision-making, which could lead to timely interventions, reduced complications, and improved patient outcomes.

LIMITATIONS:

1. Since study was conducted at single tertiary hospital, the findings might not be generalizable to other healthcare settings, including primary or secondary care hospitals with different patient demographics and resource availability.
2. The study design limits ability to ascertain causality. A prospective longitudinal study could provide good perceptiveness of predictive value of PANC 3 score and HsCRP over time.
3. The study was conducted over one year, which may have limited the number of patients included. A larger sample size could provide stronger statistical power and more conclusive results.
4. Patients admitted to a tertiary care center may represent more severe cases of AP, which could influence severity distribution and predictive performance of the studied markers.
5. Although HsCRP is known to be a reliable inflammatory marker, its levels can fluctuate based on the timing of measurement. Variations in the exact time of sample collection after symptom onset may have affected the consistency of results.
6. While the study evaluates PANC 3 and HsCRP, it does not compare their predictive accuracy against other established severity scores like BISAP, APACHE II, or Ranson's criteria, which could have provided additional insights.

7. The study does not consider confounding factors including pre-existing inflammatory disorders, chronic diseases, or drugs that could impact HsCRP levels and severity of AP.
8. The study primarily relies on biomarker-based severity prediction, but additional imaging findings (such as CT severity index) and long-term clinical outcomes could provide a more comprehensive assessment of disease progression.
9. Since the study focuses on initial severity prediction, long-term outcomes, complications, and mortality rates were not evaluated, which limits the assessment of the prognostic value of these markers.

CONCLUSION

This study highlights the significance of the PANC 3 score and HsCRP as valuable tools to predict severity of AP in patients admitted to tertiary hospital. The findings demonstrate that a higher PANC 3 score and elevated HsCRP levels are significantly associated with severe AP, making them useful markers for early risk stratification.

The PANC 3 score, which incorporates HCT, BMI, and pleural effusion, effectively identifies patients at higher risk of severe disease. Similarly, HsCRP, a widely available inflammatory biomarker, correlates well with disease severity and systemic complications. The combination of these markers enhances the accuracy of severity prediction, potentially guiding timely interventions and improving clinical outcomes.

Despite certain limitations, including the single-center design and the lack of direct comparison with other severity scoring systems, this study reinforces clinical utility in routine practice.

In conclusion, integrating the PANC 3 score and HsCRP into standard assessment protocols can facilitate timely classification of high-risk patients, enabling prompt management strategies to reduce morbidity and mortality connected with SAP.

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ANNEXURE – I - INFORMED CONSENT FORM

“ROLE OF PANC3 SCORE AND HSCR P IN PREDICTING SEVERITY OF ACUTE PANCREATITIS IN PATIENTS ADMITTED AT TERTIARY CARE CENTRE,BELAGAVI-A ONE YEAR CROSS SECTIONAL STUDY”

Name of Student/Principal Investigator:

Name of Guide/Co Investigators:

- **Introduction:** Acute pancreatitis is the most terrible of all the calamities that occur in connection with the abdominal viscera. It may be defined as an acute pancreatic inflammation due to activation of digestive enzymes present in the interior of the gland, which affect the pancreas, adjacent tissues and other organs^(1,2).
- The typical findings observed are acute onset of upper abdominal pain with radiation to the back, nausea and vomiting, local peritonitis located in the epigastrium and sometimes an effect on the circulatory system, in combination with elevated pancreatic enzymes in blood or urine.
- With the Incidence between 30/10,000 to 50/100,000 population,⁽³⁾ and miscellaneous etiological factors^(4,5) this inflammatory condition is most commonly caused by bile stones or excessive use of alcohol.
- After the initial diagnosis, the treatment depends upon the assessment of disease severity as early as possible. 50% mortality associated with severe acute pancreatitis can be reduced to 8% by its early recognition implicating the importance of early diagnosis of the disease⁽⁶⁾. In such a situation we need an indicator which can predict the outcome of an attack, as severe or mild, as early as possible and it should be sensitive and specific enough to trust upon.
- Most of the indicators used for prognosis are cumbersome, requires advanced laboratory set up, take greater than 48 hours to enable complete severity stratification.
- PANC 3 scoring system developed by Brown et al of Harvard Medical School claimed to be an approachable, assessable scoring which uses only 3 parameters such as hematocrit, body mass index (BMI) and pleural effusion. It is rapid, easy to apply, needs facilities of a basic hospital and is as good as other scoring systems in predicting the outcome of an attack of an acute pancreatitis.⁽⁷⁾
- Biochemical markers like highly sensitive C-reactive protein (HsCRP), pro-calcitonin and the like are under evaluation as prognostic indicators. HsCRP estimation is cheap and easily available. In this study an attempt will be made to see if a early

estimation of HsCRP levels is an effective predictor of morbidity and mortality in acute pancreatitis.

- Tools like HsCRP and PANC 3 score are useful tools in acute pancreatitis approach, due to their high efficacy, easy application and rapid results, which enables classification of cases and early treatment. Thus an attempt will be made to evaluate PANC3 and HsCRP levels as a prognostic tool in this study.
- By the knowledge from such studies, better programs can be designed to help the patients and improve their quality of life.

Explanation of procedure: PATIENTS PRESENTING WITH CLINICAL SYMPTOMS OF ACUTE PANCREATITIS AND THOSE WITH ELEVATED SERUM AMYLASE AND LIPASE LEVELS (THREE TIMES THE UPPER LIMIT OF NORMAL) WILL BE SELECTED IN THIS STUDY. ON THE DAY OF ADMISSION PANC3 SCORE WILL BE ASSESSED BY CALCULATING BODY MASS INDEX, HEMATOCRIT AND DOING A CHEST XRAY. HSCRP TEST WILL BE DONE AT 24-48 HRS OF ADMISSION. RESULTS WILL BE ASSESSED AND INTERPRETED TO PREDICT THE SEVERITY OF DISEASE ACCORDINGLY.

Withdrawal from participation in the study: Participation in this study is voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will get benefits by participating in this study. The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Cost of investigations done during the course of study will be paid by the **principal investigator / Participant.**(Strike out which is not applicable)

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case o

' If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights

CONSENT STATEMENT

I am making a voluntary decision to participate in the study "**ROLE OF PANC3 SCORE AND HSCRp IN PREDICTING SEVERITY OF ACUTE PANCREATITIS IN PATIENTS ADMITTED AT TERTIARY CARE CENTRE,BELAGAVI-A ONE YEAR CROSS SECTIONAL STUDY**"

". My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

ANNEXURE – II -

PROFORMA

CASE NO	
NAME	
IP NO	
AGE	YEARS
SEX	
ADDRESS	
OCCUPATION	
Complaints at presentation	
Past history	
Personal history	

Vitals:

Temperature	
Pulse	
Respiratory rate	
Blood pressure	
Height	
Weight	
BMI	

PHYSICAL EXAMINATION:

	Yes	NO
Pallor		
Icterus		
Lymphadenopathy		
Cyanosis		
Clubbing		
Edema		

SYSTEMIC EXAMINATION:

Cardio vascular system	
Respiratory System	
Central Nervous System	
Per Abdomen	

INVESTIGATIONS:

HB	
HCT	
TLC	
PLATELETS	
SGOT	
SGPT	
TOTAL BILIRUBIN	
DIRECT BILIRUBIN	
INDIRECT BILIRUBIN	
ALP	
CREATININE	
UREA	
HsCRP	
PROCALCITONIN	
AMYLASE	
LIPASE	
ABG	
PH	
PO2	
PCO2	
SO2	
HCO3-	
LACTATE	

IMAGING:**Chest xray -Pleural effusion****CECT(A+P) findings(additional):****REVISED ATLANTA CRITERIA**

A.Mild acute pancreatitis:	
1)organ failure	Yes/no
2)local or systemic complications	Yes/no
B. Moderate acute pancreatitis:	
1)organ failure <48 hours –	Yes/no
2)local or systemic complications without organ failure- yes /no:	Yes/no
C. Severe acute Pancreatitis :	
1)organ failure >48 hours –	Yes/no
2)local or systemic complications-yes/no:	Yes/no

ANNEXURE III – MASTER CHART

SL NO		AGE	SEX	OCCUPATION	HABITS	PAST HISTORY	BMI	AMYLASE	LIPASE	HSCRIP	SERUM PROCALCITONIN	TOTAL BILIRUBIN	DIRECT BILIRUBIN	INDIRECT BILIRUBIN	TOTAL PROTEIN	ALBUMIN	GLOBULIN	A/G RATIO	ALT (SGPT)	AST (SGOT)	ALK. PHOSPHATASE	GLUCOSE (R)	UREA	BUN	CREATININE	SODIUM	POTASSIUM	CHLORIDE	BICARBONATE	HAEMOGLOBIN	HAEMATOCRIT/PCV	RBC COUNT	WBC COUNT	PLATELET COUNT	NEUTROPHILS	L	M	E	BASOPHILS	CHEST X RAY (PLEURAL EFFUSION)	REVISED ATLANTA CLASS	CECT (A/P)
1	10032754	37	F	HOME MAKER	NIL	NIL	29	733	297	135	0.68	0.68	0.25	0.43	5.9	3.1	2.8	1.1	17	23	102	106	16.9	7.9	0.76	136	4.30	104	20.1	9.5	30.7	3.39	9.7	410	77	13	4	6	0	NO	A	
2	10041459	45	M	BUSINESS	ALCOHOL	HTN	31	372	844	13	0.04	1.39	0.88	0.51	6.9	4.2	2.7	1.6	52	84	102	208	16.9	7.9	0.57	131	3.23	95	17.0	14.5	44.8	4.40	10.3	80	82	13	0	5	0	YES	A	
3	10044109	37	M	EX ARMY	ALCOHOL	NIL	32	384	719	270	1.47	8.13	6.21	1.92	4.9	3.3	1.6	2.1	50	129	34	70	30.3	14.2	1.05	133	5.42	97	18.7	13.4	43.7	4.96	8.2	117	74	14	2	10	0	YES	C	
4	10045723	18	M	STUDENT	NIL	NIL	24	313	1033	3	0.33	0.91	0.54	0.37	6.8	4.3	2.5	1.7	77	46	55	199	6.9	6.2	0.60	139	3.64	104	18.0	15.0	45.3	5.03	12.3	216	88	6	0	6	0	NO	A	
5	10039772	41	M	BUSINESS	ALCOHOL	DM	28	313	199	27	0.29	3.01	2.58	0.43	7.3	2.6	4.7	0.6	41	78	184	94	17.8	8.3	0.78	131	5.26	100	19.6	9.1	30.8	3.30	14.0	243	75	16	1	8	0	NO	A	
6	10047724	35	M	FARMER	TOBACCO	NIL	32	404	562	270	0.64	6.85	5.46	1.39	4.8	3.0	1.8	1.7	12	39	72	266	35.0	16.4	1.00	127	5.30	94	21.5	12.0	33.8	2.55	20.5	274	84	7	0	9	0	YES	C	
7	10047897	56	M	BUSINESS	SMOKING, ALCOHOL	HTN	29	574	2888	100	0.09	0.59	0.21	0.38	7.0	3.7	3.3	1.1	10	26	162	121	14.4	6.7	1.24	123	3.36	84	21.1	13.0	40.3	5.06	8.4	241	86	8	0	6	0	NO	C	
8	10052115	25	M	STUDENT	NIL	NIL	24	456	522	76	0.21	3.57	1.14	2.43	6.2	3.8	2.4	1.6	29	73	66	360	47.8	22.3	1.06	141	4.10	109	14.2	14.1	43.3	4.52	12.3	196	75	6	7	6	0	NO	A	
9	10055879	48	M	BANK EMPLOYEE	SMOKING, ALCOHOL	NIL	23	369	219	213	0.23	1.62	0.85	0.77	7.5	3.6	3.9	0.9	22	28	82	115	18.3	8.6	0.74	133	4.21	98	21.5	12.8	41.4	4.30	11.2	337	72	19	0	9	0	NO	A	
10	10058246	70	M	DEPENDENT	NIL	NIL	21	305	180	56	0.61	1.22	0.94	0.28	6.3	3.6	2.7	1.3	80	76	119	96	31.5	14.7	1.27	136	3.36	94	26.7	14.7	44.0	5.83	28.9	175	91	5	0	4	0	YES	A	
11	10059852	41	M	BANK EMPLOYEE	SMOKING, ALCOHOL	NIL	31	1450	3458	152	2.59	2.59	0.39	2.30	7.0	3.8	3.2	1.2	51	307	63	150	57.8	27.0	2.46	135	7.86	101	13.8	22.5	72.6	7.44	16.5	193	80	6	0	4	0	YES	C (DEATH)	
12	10060571	55	F	FARMER	TOBACCO	HTN	28	571	365	43	0.32	0.32	0.21	0.11	6.8	3.9	2.9	1.3	11	20	126	87	16.8	7.9	0.64	138	3.29	105	18.9	8.9	30.0	3.97	10.3	503	63	26	1	10	0	NO	A	
13	10066763	34	M	BUSINESS	NIL	NIL	25	369	644	18	0.05	1.05	0.22	0.83	7.7	4.9	2.8	1.8	15	26	99	227	31.7	14.8	0.70	136	5.15	93	14.9	16.7	51.9	5.47	8.7	233	60	31	2	7	0	NO	A	
14	10067196	31	M	TEACHER	SMOKING,ALCOHOL	NIL	29	532	893	11	0.11	1.44	0.47	0.97	7.4	4.8	2.6	1.8	146	192	141	136	23.9	11.2	1.05	135	4.18	94	28.1	16.9	49.7	4.99	6.8	156	78	16	1	5	0	YES	B	
15	10070671	35	M	SOFTWARE	NIL	NIL	26	400	702	1	0.05	0.53	0.23	0.30	7.7	4.6	3.1	1.5	10	18	93	209	23.0	10.8	0.69	138	3.62	99	19.2	14.7	43.5	4.40	9.3	201	63	23	4	10	0	NO	A	
16	10077170	24	F	HOME MAKER	NIL	NIL	31	1138	1673	190	5.89	0.37	0.26	0.11	6.2	2.9	3.3	0.9	12	18	160	58	150.0	70.1	9.95	131	4.34	94	10.0	8.4	28.7	3.86	25.0	341	83	7	1	9	0	YES	C (DEATH)	
17	10031353	45	M	FARMER	ALCOHOL	NIL	29	1695	3778	38	0.30	1.51	0.86	0.65	7.0	4.2	2.8	1.5	92	143	116	137	41.1	19.2	0.70	136	4.79	102	20.1	13.4	41.9	4.38	6.4	83	88	6	0	6	0	NO	A	
18	10029357	48	M	FARMER	SMOKING,ALCOHOL	DM	31	610	1724	139	0.32	0.78	0.33	0.45	6.6	3.5	3.1	1.1	55	47	89	501	19.3	9.0	1.14	126	4.33	93	7.9	17.2	51.6	5.30	12.5	133	83	9	0	8	0	YES	B	
19	10029352	33	M	SOFTWARE	ALCOHOL	NIL	30	490	287	210	4.19	1.16	0.61	0.55	5.4	2.9	2.5	1.2	31	62	30	76	205.0	95.7	8.31	146	5.59	114	9.7	12.4	44.0	4.80	4.7	94	71	18	3	8	0	YES	C (DEATH)	
20	10026027	45	M	BUSINESS	ALCOHOL	HTN	32	2550	2388	208	2.74	2.81	1.40	1.41	5.9	3.5	2.4	1.5	48	197	21	403	66.8	31.2	4.18	132	5.20	99	13.4	12.8	43.5	3.53	10.8	151	84	10	0	6	0	YES	C (DEATH)	
21	10025618	34	F	HOME MAKER	ALCOHOL	NIL	27	1685	4213	78	0.17	1.15	0.42	0.73	6.7	4.1	2.6	1.6	125	77	148	141	21.0	9.8	0.64	135	5.13	100	16.2	13.0	42.2	4.53	3.0	110	78	19	0	3	0	YES	A	
22	10015968	39	M	BUSINESS	ALCOHOL	NIL	28	567	1044	68	0.16	8.89	5.66	3.23	6.0	2.8	3.2	0.9	35	190	119	114	10.0	4.7	0.38	127	4.23	92	23.5	8.4	30.1	2.01	6.3	110	77	15	0	8	0	NO	A	
23	10016932	66	M	FARMER	TOBACCO	DM,HTN, IHD	28	1192	1822	9	2.60	1.33	0.64	0.69	7.0	4.3	2.7	1.6	22	41	32	149	19.4	9.1	0.80	137	4.08	100	20.3	14.5	43.5	4.50	11.1	217	86	8	0	6	0	NO	A	
24	10010170	42	M	TEACHER	NIL	NIL	32	486	205	102	12.90	6.89	5.52	1.37	5.4	3.3	2.1	2.6	151	914	146	160	70.8	33.1	4.75	138	3.95	100	12.7	17.2	51.0	5.94	5.5	30	70	22	2	6	0	YES	C	
25	10013306	39	F	TEACHER	NIL	NIL	31	1092	895	47	0.09	0.75	0.34	0.41	7.1	4.2	2.9	1.4	176	91	130	70	19.9	9.3	0.81	132	4.18	96	20.0	12.0	38.0	4.93	9.9	296	79	15	0	6	0	NO	A	
26	10006717	52	F	HOME MAKER	NIL	DM	31	1622	3729	204	86.80	1.38	0.74	0.64	5.2	3.3	1.9	1.7	28	138	47	112	70.0	32.7	1.58	143	4.59	110	21.0	12.0	44.0	4.40	18.5	202	75	7	1	3	0	YES	C	
27	10005335	58	M	FARMER	ALCOHOL	NIL	22	1250	1678	67	0.18	1.30	0.58	0.72	7.8	5.0	2.8	1.8	26	30	108	152	32.9	15.4	0.82	141	4.85	98	25.3	16.1	48.0	5.13	17.0	221	92	4	0	4	0	NO	A	
28	10065900	45	M	BUSINESS	ALCOHOL	HTN	31	305	751	13	0.04	1.35	0.64	0.71	7.0	4.3	2.7	1.6	25	38	84	207	18.5	8.6	0.61	132	3.16	93	19.7	16.8	51.9	5.54	18.7	127	83	11	0	6	0	NO	A	
29	10063857	45	M	BUSINESS	NIL	NIL	26	337	204	135	0.49	2.09	0.81	1.28	6.7	3.9	2.8	1.4	23	42	67	136	36.2	16.9	1.02	137	4.33	98	22.2	18.0	54.6	5.20	17.9	215	94	4	0	2	0	NO	A	
30	10063359	37	M	BUSINESS	NIL	NIL	25	291	717	45	0.04	0.82	0.25	0.57	8.0	5.0	3.0	1.7	40	24	122	189	17.6	8.2	0.93	137	3.56	99	18.7	16.7	52.9	5.72	20.7	339	85	9	0	5	0	NO	A	
31	10062791	32	M	GOVT EMPLOYEE	NIL	NIL	28	795	423	19	0.03	0.35	0.14	0.21	6.4	3.0	3.4	0.9	25	20	116	86	13.1	6.1	0.78	137	3.57	102	23.5	11.9	37.3	4.41	7.1	410	58	31	4	7	0	NO	A	
32	10060911	58	M	BUSINESS	NIL	NIL	28	376	348	121	0.14	2.34	1.30	1.04	6.8	4.1	2.7	1.5	122	40	141	84	33.0	15.4	0.98	135	4.11	98	20.8	14.0	42.0	4.60	7.8	185	75	17	0	8	0	NO	A	

33	10037622	58	M	FARMER	ALCOHOL	DM	29	596	476	46	0.25	0.63	0.14	0.49	6.6	3.2	3.4	0.9	54	83	281	153	95.0	44.4	1.66	115	5.08	84	17.2	14.5	42.5	4.41	10.2	562	78	13	1	8	0	YES	B	
34	10041334	26	M	ACCOUNTANT	NIL	NIL	30	349	482	227	0.24	1.13	0.80	0.33	7.4	2.9	4.5	0.6	24	23	149	97	39.5	18.5	0.86	133	4.46	96	21.4	11.6	35.0	4.01	23.4	415	81	7	0	6	0	NO	A	
35	10044566	32	M	ELECTRICAL ENGINEER	NIL	NIL	25	442	414	4	0.90	0.81	0.47	0.34	7.0	3.6	3.4	1.1	12	23	70	91	21.6	10.1	0.69	133	4.43	96	24.5	11.0	34.0	5.00	7.0	229	77	13	0	10	0	YES	A	
36	10048595	49	F	HOME MAKER	NIL	HTN	32	565	1255	41	0.53	0.63	0.40	0.23	7.5	3.5	4.0	0.9	8	6	231	452	148.0	69.2	6.36	126	6.33	99	10.0	8.0	28.1	3.22	14.3	859	88	7	0	5	0	NO	B	
37	10049520	26	M	STUDENT	NIL	NIL	24	795	423	19	0.03	0.71	0.37	0.34	7.7	5.0	2.7	1.9	27	28	73	319	26.3	12.3	0.86	134	4.47	98	21.1	16.5	50.3	5.80	9.6	232	86	11	0	3	0	NO	A	
38	10052942	48	M	BUSINESS	ALCOHOL	DM	35	289	275	272	1.98	1.83	1.14	0.69	5.8	3.3	2.5	1.3	70	98	77	204	48.9	22.9	1.95	137	3.20	106	13.2	14.7	45.8	3.69	5.7	105	75	10	1	2	0	YES	C	
39	10067196	31	M	BUSINESS	NIL	NIL	28	376	348	121	0.14	2.34	1.30	1.04	6.8	4.1	2.7	1.5	122	40	141	84	33.0	15.4	0.98	135	4.11	98	20.8	14.0	42.0	4.60	7.8	185	75	17	0	8	0	NO	A	
40	10067287	31	M	SOFTWARE	NIL	NIL	30	930	1561	15	0.26	3.22	0.70	2.52	7.1	7.2	2.9	1.4	11	25	79	121	18.3	8.6	0.80	137	4.02	102	21.0	15.0	43.9	4.30	6.3	212	74	18	2	6	0	NO	A	
41	10068410	43	M	FARMER	NIL	HTN	29	986	805	9	0.34	0.54	0.27	0.27	7.2	4.1	3.1	1.3	12	23	108	84	16.4	7.6	1.04	134	3.77	98	18.5	9.4	30.0	5.51	8.3	322	65	20	5	10	0	YES	A	
42	10073730	39	M	BUSINESS	NIL	NIL	28	689	332	5	0.10	0.55	0.28	0.27	6.3	4.0	2.3	1.7	24	27	106	166	15.5	7.2	0.79	136	4.00	102	22.2	12.0	37.3	4.30	17.0	312	72	20	3	5	0	NO	A	
43	10063661	19	M	STUDENT	NIL	NIL	22	236	1035	4	1.60	3.22	1.06	2.16	6.7	4.2	2.5	1.7	9	13	99	74	23.7	11.1	0.97	134	4.58	98	20.3	13.8	42.0	4.65	9.1	180	79	10	5	6	0	YES	A	
44	10069845	48	M	GOVT EMPLOYEE	SMOKING,ALCOHOL	NIL	23	386	258	36	0.02	0.56	0.19	0.37	7.5	3.8	3.7	1.0	7	24	80	114	14.1	6.6	0.69	138	4.00	100	18.4	12.5	38.8	4.61	9.4	235	57	28	6	9	0	NO	A	
45	10070454	39	M	MECH ENGINEER	NIL	HTN	25	151	183	9	0.80	1.05	0.52	0.53	6.4	4.4	2.0	2.2	105	102	95	98	13.8	6.5	0.68	138	3.32	98	23.5	15.2	45.8	4.64	6.4	159	62	26	2	10	0	YES	A	
46	10078705	39	M	BUSINESS	ALCOHOL	NIL	32	251	208	113	4.63	0.75	0.34	0.41	7.4	4.3	3.1	1.4	11	14	99	202	23.9	11.2	0.72	129	4.78	99	11.6	13.8	47.8	5.60	7.9	254	77	12	2	9	0	YES	A	
47	10077170	24	F	HOME MAKER	NIL	NIL	32	565	1255	41	0.53	0.63	0.40	0.23	7.5	3.5	4.0	0.9	8	6	231	452	148.0	69.2	6.36	126	6.33	99	10.0	8.0	28.1	3.22	14.3	859	88	7	0	5	0	NO	B	
48	10066190	33	F	HOME MAKER	NIL	NIL	28	990	1348	56	0.34	1.12	0.88	0.24	5.9	3.7	2.2	1.7	462	190	90	78	12.0	5.6	0.51	138	3.75	104	15.9	13.9	42.8	4.53	24.1	312	97	2	0	1	0	YES	A	
49	10053980	41	M	BUSINESS	ALCOHOL	DM	28	313	199	27	0.29	3.01	2.58	0.43	7.3	2.6	4.7	0.6	41	78	184	94	17.8	8.3	0.78	131	5.26	100	19.6	9.1	30.8	3.30	14.0	243	75	16	1	8	0	NO	A	
50	10049276	35	M	FARMER	TOBACCO	NIL	32	404	562	270	0.64	6.85	5.46	1.39	4.8	3.0	1.8	1.7	12	39	72	266	35.0	16.4	1.00	127	5.30	94	21.5	12.0	33.8	2.55	20.5	274	84	7	0	9	0	YES	C	
51	10052583	56	F	BUSINESS	SMOKING,ALCOHOL	HTN	29	574	2888	100	0.09	0.59	0.21	0.38	7.0	3.7	3.3	1.1	10	26	162	121	14.4	6.7	1.24	123	3.36	84	21.1	13.0	40.3	5.06	8.4	241	86	8	0	6	0	NO	C	
52	10042391	25	M	STUDENT	NIL	NIL	24	456	522	76	0.21	3.57	1.14	2.43	6.2	3.8	2.4	1.6	29	73	66	360	47.8	22.3	1.06	141	4.10	109	14.2	14.1	43.3	4.52	12.3	196	75	6	7	6	0	NO	A	
53	10046627	48	M	BANK EMPLOYEE	SMOKING,ALCOHOL	NIL	23	369	219	213	0.23	1.62	0.85	0.77	7.5	3.6	3.9	0.9	22	28	82	115	18.3	8.6	0.74	133	4.21	98	21.5	12.8	41.4	4.30	11.2	337	72	19	0	9	0	NO	A	
54	10070781	70	F	DEPENDENT	NIL	NIL	21	305	180	56	0.61	1.22	0.94	0.28	6.3	3.6	2.7	1.3	80	76	119	96	31.5	14.7	1.27	136	3.36	94	26.7	14.7	44.0	5.83	28.9	175	91	5	0	4	0	YES	A	
55	10072786	41	M	BANK EMPLOYEE	SMOKING,ALCOHOL	NIL	31	1450	3458	152	2.59	2.59	0.39	2.30	7.0	3.8	3.2	1.2	51	307	63	150	57.8	27.0	2.46	135	7.86	101	13.8	22.5	72.6	7.44	16.5	193	80	6	0	4	0	YES	C (DEATH)	