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THE ROLE OF RISK FACTORS AND BIOMARKERS IN CORRELATION WITH SEVERITY IN ACUTE PANCREATITIS

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ABSTRACT

Acute pancreatitis is a sudden inflammatory condition of the pancreas with a wide clinical spectrum ranging from mild self-limiting illness to severe disease associated with organ failure and high mortality. Early prediction of severity is crucial for timely management and prevention of complications. This prospective observational study was conducted to evaluate the correlation between demographic characteristics, etiological risk factors, comorbidities, biochemical biomarkers, and disease severity in patients diagnosed with acute pancreatitis. A total of 100 patients admitted to a tertiary care teaching hospital were included in the study over a period of six months. Data regarding age, gender, etiological factors, comorbid conditions, laboratory parameters, and radiological findings were collected. Disease severity was assessed using the Revised Atlanta Classification, BISAP score, Ranson criteria, and Modified CT Severity Index. The study population predominantly consisted of males (68%), with the majority of cases observed in the 31–50 year age group. Gallstones were identified as the most common etiological factor followed by alcohol consumption. Mild acute pancreatitis was observed in 68% of patients, while 21% had moderately severe disease and 11% had severe acute pancreatitis. Statistically significant associations were found between disease severity and age, gender, comorbidities, and gallstone etiology. The study concludes that

integrating clinical risk factors with biomarkers and scoring systems aids in early severity assessment and improves clinical decision-making in acute pancreatitis.

KEYWORDS: Acute pancreatitis, biomarkers, risk factors, disease severity.

INTRODUCTION OF PANCREAS

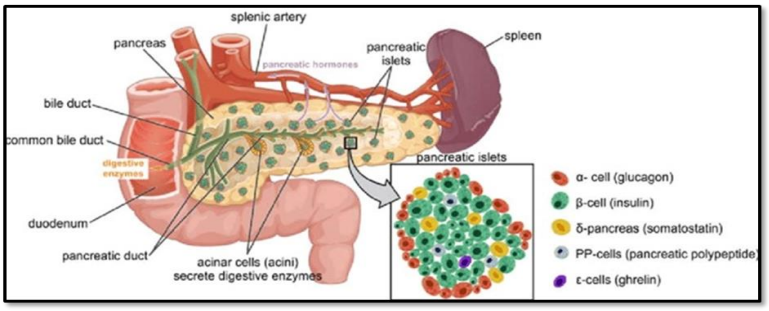
The pancreas is a vital retroperitoneal organ located posterior to the stomach in the upper abdomen. It performs both exocrine and endocrine functions, making it essential for digestion and glucose homeostasis. Anatomically, it is divided into the head, neck, body, and tail and lies in close proximity to major vascular structures, including the superior mesenteric and splenic vessels. The pancreatic duct joins the common bile duct to form the ampulla of Vater, which opens into the duodenum.

The exocrine pancreas secretes digestive enzymes such as amylase, lipase, and proteases, which facilitate the digestion of carbohydrates, fats, and proteins. The endocrine pancreas, composed of the islets of Langerhans, secretes hormones including insulin and glucagon that regulate blood glucose levels. Due to its deep anatomical location and nonspecific symptoms of disease, pancreatic disorders often require imaging and biochemical investigations for diagnosis. Inflammatory conditions such as pancreatitis are among the most common pancreatic disorders and represent a major cause of hospital admissions worldwide.

Anatomy of the Pancreas

Functionally, the pancreas consists of an exocrine component responsible for enzyme secretion and an endocrine component involved in hormonal regulation. The exocrine portion constitutes approximately 85–90% of the organ and drains into the duodenum via the main pancreatic duct. The endocrine portion comprises the islets of Langerhans, predominantly located in the tail region, and contains specialized cells responsible for hormone secretion.

The pancreas receives arterial blood supply from branches of the celiac trunk and superior mesenteric artery, with venous drainage into the portal circulation. Lymphatic drainage follows the arterial pathways, while autonomic innervation regulates both enzyme and hormone secretion. This complex vascular and neural network plays a crucial role in maintaining pancreatic function.



Functions of the Pancreas

The pancreas performs two primary functions.

Endocrine Function

The endocrine pancreas secretes hormones such as insulin, glucagon, somatostatin, and pancreatic polypeptide, which regulate blood glucose levels and metabolic processes. Insulin lowers blood glucose by promoting cellular uptake, while glucagon increases glucose levels through glycogenolysis and gluconeogenesis.

Exocrine Function

The exocrine pancreas produces alkaline pancreatic juice containing digestive enzymes. These enzymes include proteases for protein digestion, lipases for fat digestion, amylase for carbohydrate digestion, and nucleases for nucleic acid breakdown. Pancreatic secretion is regulated by neurohormonal mechanisms involving secretin and cholecystokinin.

Pancreatitis

Pancreatitis is an inflammatory disorder of the pancreas caused by premature activation of pancreatic enzymes within the gland, leading to autodigestion and systemic inflammatory response. It is broadly classified into acute and chronic pancreatitis.

Acute pancreatitis presents as a sudden inflammatory episode that may range from mild, self-limiting disease to severe necrotizing pancreatitis with organ failure. Chronic pancreatitis is characterized by persistent inflammation, fibrosis, and irreversible loss of pancreatic function. Less common forms include autoimmune and hereditary pancreatitis.

Acute Pancreatitis

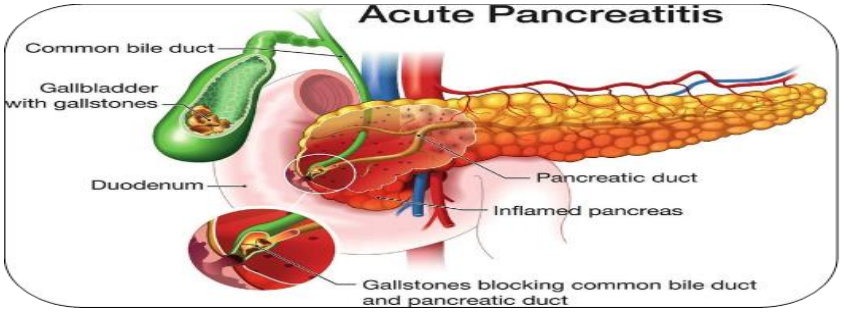
Acute pancreatitis is one of the most common gastrointestinal emergencies worldwide. It typically presents with severe upper abdominal pain and elevated serum amylase and lipase

levels. Histologically, it is characterized by edema, inflammatory cell infiltration, necrosis, and hemorrhage in severe cases.

Based on the revised Atlanta classification, acute pancreatitis is categorized into interstitial edematous pancreatitis and necrotizing pancreatitis. Interstitial edematous pancreatitis is the most common and usually resolves with supportive care. Necrotizing pancreatitis is associated with tissue necrosis, infected collections, organ failure, and increased mortality.

Complications of Acute Pancreatitis

Local complications include acute peripancreatic fluid collections, pseudocysts, acute necrotic collections, and walled-off necrosis. Systemic complications include systemic inflammatory response syndrome, organ failure, sepsis, and metabolic disturbances. The severity of complications determines prognosis and clinical outcomes.



Causes and Risk Factors of Acute Pancreatitis

Gallstone-Induced Acute Pancreatitis

Gallstones are the most common cause of acute pancreatitis worldwide, accounting for approximately 35–60% of cases depending on the studied population. Gallstone pancreatitis occurs when gallstones migrate from the gallbladder into the common bile duct and become lodged at the ampulla of Vater. This obstruction leads to impaired drainage of pancreatic secretions, increased intraductal pressure, and reflux of bile into the pancreatic duct. The resultant disruption triggers premature activation of trypsinogen to trypsin within pancreatic acinar cells, initiating autodigestion and inflammatory cascades. Smaller gallstones (<5 mm) and biliary sludge are particularly implicated due to their higher likelihood of transient ductal obstruction.

Alcohol-Related Acute Pancreatitis

Alcohol consumption represents the second most common cause of acute pancreatitis, contributing to nearly 30% of cases globally. The risk increases with chronic heavy alcohol intake, typically defined as consumption of 50–150 grams of alcohol per day for at least five years. Alcohol exerts a direct toxic effect on pancreatic acinar cells and promotes ductal obstruction through protein plug formation. Additionally, alcohol alters intracellular calcium signaling, increases zymogen secretion, and sensitizes pancreatic tissue to injury. Despite prolonged heavy drinking, only 5–10% of individuals develop acute pancreatitis, suggesting the influence of genetic susceptibility and environmental modifiers.

Hypertriglyceridemia

Hypertriglyceridemia accounts for approximately 1–4% of acute pancreatitis cases and is typically observed when serum triglyceride levels exceed 1000 mg/dL. In this condition, pancreatic lipase hydrolyzes triglycerides into free fatty acids, which exert cytotoxic effects on pancreatic tissue and vascular endothelium. These free fatty acids promote ischemia, inflammation, and acinar cell damage. Hypertriglyceridemia-induced pancreatitis is frequently associated with uncontrolled diabetes mellitus, obesity, metabolic syndrome, and inherited lipid disorders.

Post-Endoscopic Retrograde Cholangiopancreatography (Post-ERCP) Pancreatitis

Acute pancreatitis is a recognized complication following diagnostic or therapeutic endoscopic retrograde cholangiopancreatography (ERCP), with an incidence ranging from 3–10%. The risk is higher in young individuals, females, and patients undergoing difficult cannulation or pancreatic duct instrumentation. Mechanical trauma, hydrostatic injury from contrast injection, and chemical or thermal injury contribute to pancreatic inflammation in post-ERCP pancreatitis.

Drug-Induced Acute Pancreatitis

Drug-induced pancreatitis is relatively rare, accounting for less than 2% of cases. Various medications can cause pancreatic inflammation through hypersensitivity reactions, direct toxicity, or metabolic disturbances. Commonly implicated drugs include azathioprine, mercaptopurine, valproic acid, tetracyclines, didanosine, thiazide diuretics, loop diuretics, and estrogen-containing medications, particularly due to their triglyceride-elevating effects.

Hypercalcemia

Hypercalcemia, often associated with primary hyperparathyroidism or malignancy, is a rare but established cause of acute pancreatitis. Elevated intracellular calcium levels in pancreatic acinar cells lead to inappropriate enzyme activation, mitochondrial dysfunction, and cellular injury, thereby triggering inflammatory processes.

Genetic Causes

Inherited genetic mutations contribute to recurrent or early-onset acute pancreatitis, particularly in younger individuals without obvious external risk factors. Mutations in the PRSS1 gene increase trypsin activity, SPINK1 mutations impair trypsin inhibition, and CFTR mutations result in thick pancreatic secretions and ductal obstruction, collectively predisposing individuals to pancreatic inflammation.

Trauma

Blunt abdominal trauma can directly injure pancreatic tissue or the pancreatic duct, leading to acute pancreatitis. This is more commonly observed in children and young adults following motor vehicle accidents or sports-related injuries.

Infectious Causes

Certain viral infections, particularly in tropical regions, have been linked to acute pancreatitis. Viruses such as mumps, coxsackievirus, hepatitis viruses, HIV, and cytomegalovirus may cause direct pancreatic injury or immune-mediated inflammation.

Autoimmune Pancreatitis

Autoimmune pancreatitis is a distinct inflammatory condition characterized by elevated IgG4 levels and systemic autoimmune features. Prompt recognition is essential as it responds well to corticosteroid therapy, unlike other etiologies.

Idiopathic Acute Pancreatitis

Despite thorough evaluation, 10–30% of acute pancreatitis cases remain idiopathic. Advanced imaging modalities and genetic testing have significantly reduced this proportion, often revealing microlithiasis or occult biliary disease during detailed assessments.

Signs and Symptoms

Acute pancreatitis presents with characteristic clinical features that vary in severity depending on the extent of pancreatic inflammation and systemic involvement. The hallmark

symptom is sudden-onset, severe epigastric pain radiating to the back, often described as deep and persistent. Pain typically worsens after food intake and is partially relieved by leaning forward. Associated gastrointestinal symptoms include nausea, vomiting, abdominal bloating, and reduced oral intake.

On physical examination, patients exhibit abdominal tenderness with guarding in severe cases. Bowel sounds may be diminished due to paralytic ileus. Rare but severe manifestations include Cullen's sign and Grey Turner's sign, which indicate hemorrhagic pancreatitis (Figure 3). Systemic signs such as fever, tachycardia, hypotension, and hypoxia reflect systemic inflammatory response syndrome or sepsis.

Complications

Acute pancreatitis can result in both local and systemic complications. Local complications include pancreatic necrosis, pseudocysts, acute peripancreatic fluid collections, and walled-off necrosis (Figure 1). Systemic complications include organ dysfunction affecting the lungs, kidneys, cardiovascular system, and central nervous system.

Pathogenesis

The pathogenesis of acute pancreatitis begins with premature intracellular activation of digestive enzymes, particularly trypsinogen to trypsin, within pancreatic acinar cells. This abnormal activation results from lysosomal fusion with zymogen granules, calcium overload, and mitochondrial dysfunction. Activated enzymes initiate autodigestion, leading to acinar cell necrosis rather than apoptosis.

The subsequent release of damage-associated molecular patterns triggers a robust inflammatory response mediated by cytokines such as TNF-α, IL-1β, IL-6, and IL-8. Neutrophil infiltration, oxidative stress, microvascular dysfunction, ischemia-reperfusion injury, and endothelial damage exacerbate pancreatic injury. When inflammatory mediators enter systemic circulation, systemic inflammatory response syndrome develops, potentially progressing to multiorgan dysfunction syndrome.

DIAGNOSIS

Diagnosis of acute pancreatitis requires fulfillment of at least two of the three revised Atlanta criteria: characteristic abdominal pain, serum amylase or lipase levels exceeding three times the upper limit of normal, and imaging findings consistent with pancreatitis. Clinical

evaluation remains the cornerstone of early diagnosis, supported by biochemical and radiological investigations.

Biochemical Markers

Serum amylase and lipase are key diagnostic enzymes, with lipase preferred due to higher sensitivity and specificity. C-reactive protein levels above 150 mg/L at 48 hours indicate severe disease. Procalcitonin is useful in predicting infected necrosis. Liver function tests assist in identifying biliary etiology, while serum calcium and LDH levels contribute to severity assessment.

Radiological Biomarkers

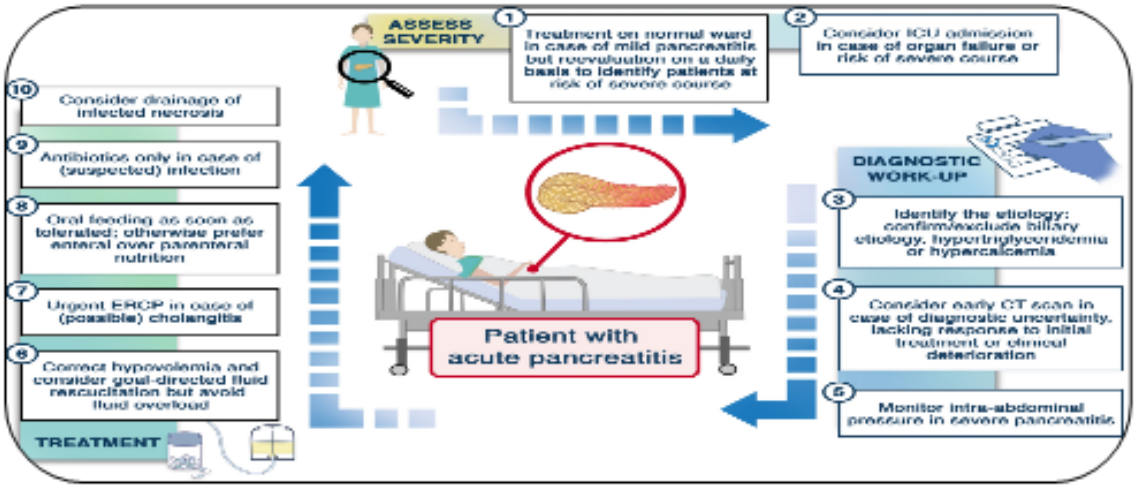
Radiological investigations such as ultrasonography, contrast-enhanced CT, MRI, and endoscopic ultrasound play a vital role in diagnosing etiology, assessing severity, and identifying complications. CECT remains the gold standard for evaluating pancreatic necrosis and severity scoring using CTSI.

Severity Scoring Systems

Severity assessment using BISAP, Ranson's criteria, and CT Severity Index aids in early risk stratification, prognosis, and clinical decision-making. Higher scores correlate with increased risk of organ failure and mortality.

Treatment

Management of acute pancreatitis is primarily supportive. Pain control using opioids, aggressive fluid resuscitation with lactated Ringer's solution, and early enteral nutrition are cornerstone therapies. Antibiotics are reserved for infected necrosis.



MATERIALS AND METHODS

Need of the Study

Acute pancreatitis exhibits a wide clinical spectrum ranging from mild self-limiting disease to severe forms associated with organ failure and high mortality. Early identification of patients at risk for severe disease remains a major clinical challenge. Risk factors such as gallstones, alcohol consumption, and metabolic abnormalities significantly influence disease onset and progression. Additionally, biochemical markers and radiological findings play a crucial role in assessing disease severity. This study was undertaken to evaluate the correlation between risk factors, biochemical and radiological biomarkers, and severity of acute pancreatitis, with the aim of improving early prediction, patient stratification, and clinical outcomes.

AIM

To evaluate the role of risk factors and biomarkers in correlation with disease severity in patients with acute pancreatitis through a prospective observational study.

OBJECTIVES

The objectives of the study were to identify and analyze major risk factors contributing to acute pancreatitis, including gallstones, alcohol consumption, and metabolic abnormalities; to assess the role of radiological and biochemical biomarkers in predicting disease severity; to evaluate the effectiveness of combined severity scoring systems in predicting complications; to explore the relationship between risk factors, biomarkers, and clinical outcomes; and to determine the prognostic value of early biomarkers in disease progression.

MATERIALS

The study utilized a structured patient profile form to collect demographic details, clinical history, and risk factors. An informed consent form was used to obtain written consent from all participants prior to enrollment. Relevant laboratory reports, imaging records, and clinical data were reviewed and documented using a standardized data collection format.

METHODOLOGY

This prospective observational study was conducted in the Department of General Medicine at Malla Reddy Hospital, Suraram, Hyderabad, Telangana. A total of 100 patients diagnosed with acute pancreatitis were enrolled over a period of six months.

Study Criteria

Patients aged above 18 years diagnosed with acute pancreatitis based on clinical presentation, biochemical markers, and radiological findings were included. Only patients willing to provide informed consent and those with identifiable risk factors such as gallstones, alcohol consumption, or metabolic disorders were enrolled.

Pregnant or lactating women, patients with incomplete clinical or investigative data, individuals with other abdominal pathologies mimicking pancreatitis, and patients unwilling to provide consent or lost to follow-up were excluded.

Study Procedure

Eligible patients admitted with acute pancreatitis were prospectively evaluated. Data were collected from medical records using a predesigned data collection sheet. Information included demographic details, clinical history, comorbidities, and risk factors. Biochemical parameters such as serum amylase, lipase, liver function tests, serum calcium, triglycerides, blood glucose, blood urea nitrogen, C-reactive protein, and procalcitonin were recorded. Radiological data from ultrasonography, contrast-enhanced computed tomography, or magnetic resonance imaging were analyzed for pancreatic inflammation, necrosis, and peripancreatic collections.

Disease severity was assessed using the Revised Atlanta Classification, Modified CT Severity Index, Ranson's criteria, and the Bedside Index of Severity in Acute Pancreatitis (BISAP). Patients were followed until discharge to monitor disease progression, complications, and clinical outcomes.

Statistical Analysis

Collected data were entered into Microsoft Excel and analyzed using the Statistical Package for Social Sciences (SPSS) for Windows. Descriptive and inferential statistical methods were applied to evaluate associations between risk factors, biomarkers, and disease severity.

RESULTS AND DISCUSSION

RESULTS

A total of 100 patients diagnosed with acute pancreatitis were included in the present study. The analysis primarily focused on identifying **risk factors** and evaluating the role of **biochemical and radiological biomarkers** in relation to disease severity.

Risk Factors Associated with Acute Pancreatitis

Gallstones emerged as the most common etiological risk factor, observed in **56%** of patients, followed by **alcohol consumption (27%)** and **fatty liver disease (17%)**. This finding reinforces the established role of biliary pathology as the leading cause of acute pancreatitis. Statistical analysis revealed a **significant association between gallstone etiology and increased disease severity (p = 0.049)**, indicating that patients with gallstone-induced pancreatitis were more likely to develop moderate to severe disease compared to other etiologies.

Alcohol-related pancreatitis constituted over one-fourth of the study population and was associated with a higher proportion of moderate and severe cases, suggesting the contributory role of chronic alcohol exposure in worsening pancreatic injury. Fatty liver disease, though less frequent, was also associated with increased severity, possibly reflecting underlying metabolic dysfunction.

Impact of Comorbidities on Severity

The presence of comorbid conditions significantly influenced disease severity. Patients with comorbidities such as **diabetes mellitus, hypertension, and dyslipidemia** showed a higher incidence of moderate to severe acute pancreatitis. Statistical analysis demonstrated a **significant association between comorbidities and disease severity (p = 0.032)**. These findings suggest that metabolic and cardiovascular comorbidities may exacerbate systemic inflammation and impair recovery, thereby contributing to worse clinical outcomes.

Radiological Biomarkers and Severity Assessment

Radiological severity assessment using the **Modified CT Severity Index (MCTSI)** revealed that **22% of patients** had moderate to severe radiological disease. Patients with higher MCTSI scores demonstrated increased pancreatic necrosis, peripancreatic fluid collections, and local complications, correlating well with clinical severity. The findings support the role of contrast-enhanced CT as a reliable radiological biomarker for predicting disease progression and complications.

Biochemical Biomarkers and Prognostic Significance

Biochemical markers played a crucial role in severity stratification. Elevated levels of **serum amylase and lipase** were observed in all patients, confirming their diagnostic utility.

However, **C-reactive protein (CRP)** showed stronger correlation with disease severity, particularly in patients with moderate to severe pancreatitis, highlighting its prognostic value. Patients with elevated **blood urea nitrogen (BUN)**, **serum triglycerides**, and **low serum calcium levels** were more likely to develop severe disease, consistent with their inclusion in established severity scoring systems. Elevated **procalcitonin levels** were noted in severe cases, indicating an increased risk of infected necrosis and systemic complications.

Correlation of Severity with Scoring Systems

Severity stratification using **Revised Atlanta Classification**, **Ranson's criteria**, and **BISAP score** demonstrated consistent findings. Patients classified as severe by these scoring systems showed strong associations with adverse biomarkers and high-risk etiologies. A higher BISAP score was significantly associated with increased severity, reinforcing its value as an early bedside predictor.

The present study demonstrates that **gallstones remain the most significant risk factor** for acute pancreatitis and are strongly associated with increased disease severity. Alcohol consumption and metabolic disorders further contribute to disease progression. Biochemical markers such as **CRP, BUN, serum calcium, and procalcitonin**, along with radiological indices like **MCTSI**, serve as reliable predictors of severity and clinical outcomes.

The combined evaluation of **risk factors, biochemical biomarkers, and radiological findings** allows for early identification of high-risk patients and facilitates timely intervention. These findings support the integration of multiple assessment tools for improved prognostication and management of acute pancreatitis.

DISCUSSION

The present prospective observational study involving 100 patients with acute pancreatitis provides valuable insight into the demographic distribution, etiological risk factors, comorbid conditions, and the effectiveness of various severity assessment tools and biomarkers in predicting disease progression. The findings emphasize the multifactorial nature of acute pancreatitis and highlight the importance of integrating clinical, biochemical, and radiological parameters for optimal patient management.

A clear **male predominance (68%)** was observed in this study, which is consistent with several previously published studies reporting a higher incidence of acute pancreatitis among

males. This trend may be attributed to increased exposure to modifiable risk factors such as alcohol consumption, smoking, and metabolic abnormalities. The **31–50 years age group**, accounting for **48% of cases**, was the most affected, suggesting that individuals in this economically productive age range are particularly vulnerable. This susceptibility could be linked to lifestyle-related factors, including dietary habits, alcohol use, and stress-related metabolic disorders, which tend to peak during this period of life.

Among the identified etiological factors, **gallstones were the most common risk factor (56%)**, reaffirming biliary pathology as the leading cause of acute pancreatitis in the studied population. Gallstone-induced pancreatitis occurs due to transient obstruction of the pancreatic duct, leading to premature enzyme activation and pancreatic inflammation. **Alcohol consumption (27%)** emerged as the second most prevalent risk factor and was associated with a greater tendency toward moderate and severe disease, likely due to its direct toxic effects on pancreatic acinar cells and its role in promoting chronic inflammation. **Fatty liver disease (17%)**, though less frequent, reflects the growing burden of metabolic disorders and their indirect contribution to pancreatic injury.

The study further demonstrated a significant association between **metabolic comorbidities** and disease severity. **Diabetes mellitus (35%)** was the most common comorbidity, followed by **hypertension (28%)** and **dyslipidemia (23%)**. These findings support the growing body of evidence linking metabolic syndrome to increased susceptibility and worse outcomes in acute pancreatitis. Chronic hyperglycemia, insulin resistance, and lipid abnormalities may exacerbate systemic inflammation, impair pancreatic microcirculation, and delay tissue recovery, thereby increasing the likelihood of complications. Early identification and management of these comorbid conditions may therefore play a critical role in reducing disease severity and improving outcomes.

Severity stratification using established scoring systems revealed that the majority of patients presented with **mild acute pancreatitis**. According to the **Revised Atlanta Classification**, **68%** of patients were classified as mild, while **21%** and **11%** had moderate and severe disease, respectively. Similar trends were observed with the **Modified CT Severity Index**, **Ranson criteria**, and **BISAP score**, all of which categorized most cases as mild or low risk. The consistency across multiple scoring systems highlights their reliability and usefulness in routine clinical practice.

Radiological assessment using **contrast-enhanced CT and MRI** proved essential in identifying local complications such as pancreatic necrosis, peripancreatic fluid collections, and inflammatory changes. Patients with higher CT severity scores showed a stronger correlation with clinically severe disease, emphasizing the prognostic value of imaging studies in acute pancreatitis. Radiology thus serves not only as a diagnostic tool but also as a critical component of severity assessment and treatment planning.

Biochemical markers played a pivotal role in correlating disease severity and progression. While **serum amylase and lipase** were effective for diagnosis, markers such as **C-reactive protein (CRP)** and **procalcitonin** showed stronger associations with disease severity and systemic inflammatory response. Elevated CRP levels were particularly useful in identifying patients at risk of developing severe pancreatitis and complications. The integration of biochemical biomarkers with clinical scoring systems enhances early risk stratification and enables timely therapeutic interventions.

Overall, the findings of this study underscore the importance of a **multidimensional assessment approach**, combining risk factor evaluation, metabolic profiling, biochemical markers, radiological findings, and validated scoring systems. Such an approach allows for early identification of high-risk patients and facilitates personalized management strategies.

CONCLUSION

This study highlights the critical role of **early recognition of etiological risk factors and metabolic comorbidities** in the effective management of acute pancreatitis. Gallstones, alcohol consumption, and metabolic disorders were identified as key contributors to disease development and progression. The presence of comorbid conditions such as diabetes mellitus, hypertension, and dyslipidemia significantly influenced disease severity, emphasizing the need for comprehensive metabolic risk management.

The findings demonstrate that **integrated use of clinical scoring systems, biochemical biomarkers, and radiological assessments** provides a reliable and practical framework for predicting disease severity and guiding clinical decision-making. Early severity stratification enables timely interventions, reduces complications, and may improve overall patient outcomes.

In conclusion, adopting a **personalized, risk-based approach** that incorporates early biomarker assessment and standardized severity scoring may help optimize treatment strategies, minimize disease burden, and enhance quality of care for patients with acute pancreatitis. These results may contribute to the development of improved clinical protocols and evidence-based guidelines for the management of acute pancreatitis.

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