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Comparative Study to Assess the Bisap Score vs Ranson Score in Predicting Mortality among Acute Pancreatitis

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Abstract

The upper abdomen contains the pancreas. It performs endocrine and exocrine tasks. The exocrine system secretes chemicals into the environment; this includes hormones secreted from excretory ducts and pancreatic enzymes secreted into the gastrointestinal (GI) tract via the pancreatic duct. The hormonal processes internal secretion; and pancreatic duct hormone secretion, which involves the direct bloodstream release of somatostatin, glucagon, and insulin. Clinical, pathologic, biochemical, and bacteriologic data relate to four entities in acute pancreatitis: necrotizing pancreatitis, interstitial edematous pancreatitis, pancreatic pseudocyst, pancreatic abscess, pancreatic parenchymal necrosis, extrapancreatic retroperitoneal fatty tissue necrosis, biologically active substances in pancreatic ascites, and infection of necrosis. Severe complications account for over 80% of fatalities. The purpose of the study is to compare the predictive power of the BISAP score and the Ranson score for acute pancreatitis mortality. Objects are as follows to evaluate patients with acute pancreatitis based on their BISAP and Ranson scores. To contrast the BISAP rating and the RANSON score in individuals with acute pancreatitis. To correlate certain demographic characteristics with the BISAP and RANSON scores of patients with acute pancreatitis. For this study, a quantitative research methodology was applied. The present study employs an evaluative research approach. In this study, a descriptive research design was employed. The study was carried out at Saveetha Medical College and Hospital. Based on the inclusion criteria, a convenience sample methodology was used to recruit a total of 30 study participants. A self-structured questionnaire method was used to collect the demographic data, according to the study's findings, the RANSON score has a higher mortality prediction than the BISAP score.

Keywords: *Acute Pancreatitis, Assess, BISAP Score, Hormone, RANSON Score.*

Introduction

Acute pancreatitis is one of the most frequent gastrointestinal reasons for hospital admission in the United States and is on the rise globally. Over the last ten years, significant progress has been achieved in comprehending the pathophysiological mechanisms behind acute pancreatitis. Research has clarified the mechanisms underlying calcium-induced acinar cell damage and death, as well as the significance of calcium entry channels that are controlled by stores and mitochondrial permeability transition pores. The cytoprotective function of autophagy and the unfolded protein response in averting prolonged endoplasmic reticulum stress, apoptosis, and necrosis, together with the pivotal role of unsaturated fatty acids in inducing pancreatic organ failure, have also been documented. The

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identification of possible molecular targets for upcoming treatment trials has resulted from the characterization of these pathways. Several seminal clinical trials have informed management strategies regarding nutritional support and interventions for infected pancreatic necrosis, leading to significant changes to acute pancreatitis management paradigms. Two classification systems have been developed at the patient level to categorize the severity of acute pancreatitis into prognostically meaningful groups. A review of current developments in acute pancreatitis is presented in this review, with a focus on the pathophysiological causes and clinical management of the condition [1].

One frequent clinical issue is acute pancreatitis. This condition has varying degrees of severity; some people have moderate, self-limiting attacks, while others have severe, extremely morbid, and often fatal attacks. It's still unknown exactly how several etiological elements cause an attack. Acinar cells are thought to be the site of the initial events in acute pancreatitis. Early in acute pancreatitis, acinar cell damage triggers a local inflammatory response. A systemic inflammatory response syndrome develops if this inflammatory reaction is severe (SIRS). Multiple organ dysfunction syndrome (MODS) and distant organ damage are caused by an overactive SIRS. The main factor contributing to morbidity and death in cases of acute pancreatitis is MODS. Inflammatory mediators have been linked to the pathophysiology of acute pancreatitis and the subsequent MODS, according to recent research. However, as a factor in determining the severity of pancreatitis, new studies have shown the significance of acinar cell death in the form of necrosis and apoptosis. We will talk about what we now know about the pathophysiology of acute pancreatitis in this review [2].

Clinical, pathologic, biochemical, and bacteriologic data all relate to four entities in acute pancreatitis. The most common clinical signs are necrotizing pancreatitis and interstitial edematous pancreatitis; pancreatic pseudo cysts and pancreatic abscess appear as late sequelae, arising three to five weeks following necrotizing pancreatitis. Pancreatic parenchymal necrosis, extrapancreatic retroperitoneal fatty tissue necrosis, biologically active substances in pancreatic ascites, and infection of necrosis are factors that influence the natural course of acute pancreatitis. Multiple organ failure occurs early in the course of acute pancreatitis as a result of several inflammatory mediators generated from the inflammatory process and activated leukocytes drawn to the pancreatic damage. The second week of the late course is when local and systemic septic problems predominate. Severe complications from acute pancreatitis account for over 80% of fatalities. The pancreaticrotizing pancreatitis infection. The majority of the bacteria found in intraoperative smears and aspirates are gram-negative and originate from the colon; Escherichia coli is the most common kind of these germs. It has been established that a sizable portion of individuals experience chronic exocrine and endocrine insufficiency following necrotizing pancreatitis [3].

Although there has been a significant impact of this classification on pancreatitis research, several issues still exist. Physicians can distinguish between acute pancreatitis with a first onset and advanced chronic pancreatitis; nevertheless, there are definitional challenges between these two extremes. Since pathological specimens are rarely accessible, the distinction between relapsing acute and chronic relapsing pancreatitis is typically made based on function; nevertheless, the majority of patients with pancreatic disease are handled without function tests. There have been some misunderstandings and inaccurate quotes as a result of the official Marseille publication being difficult to obtain. One interpretation that has been made is that excessive alcohol

use only results in chronic pancreatitis; however, it is now understood that alcohol-induced pancreatitis can also manifest promptly and that the illness is not always progressive [4].

The pancreas's autodigestive process causes acute pancreatitis. In human disease, auto-digestion is a rare phenomenon. The issue is that the 100 g pancreas produces a large amount of active lipolysis, amylolysis, and proteolytic enzymes, none of which function until they are in the small intestine. Therefore, to protect the pancreas against its enzymes, a series of defence mechanisms is required. Inactive precursors provide this defence inside cells, the mucous layer covering the duct epithelium in the tissue provides this defence, and the free and prompt release of pancreatic juice seals the deal. If pancreatic juice gets into the bloodstream, it comes into contact with a strong system of enzyme inhibitors. The primary defence mechanism is intrinsic cell metabolism, which creates a one-way permeability to stop released materials from returning to the glandular epithelial cells [5].

The upper abdomen contains the pancreas. Its functions are both endocrine and exocrine. The exocrine processes include hormone secretion from excretory ducts, pancreatic enzyme secretion into the gastrointestinal (GI) tract via the pancreatic duct, and other exterior secretions. The secretion of hormones by the pancreatic duct, which includes the direct release of somatostatin, glucagon, and insulin into the bloodstream, is one of the endocrine functions. The inflammation of the pancreas known as acute pancreatitis can cause the organ to digest itself using its enzymes. Acute pancreatitis is a reasonably common, but potentially fatal, inflammatory disease that can cause bleeding, edema, and varying degrees of auto-digestion and fat necrosis [6].

The pancreas undergoes auto-digestion by its enzymes as a result of acute pancreatitis, a dangerous and occasionally fatal inflammatory condition. The degree of the pathogenic changes varies. The degree of tissue loss and inflammation determines the severity of pancreatitis, which can range from modest involvement indicated by oedema and inflammation to necrotizing hemorrhagic pancreatitis. Diffuse bleeding of pancreatic tissue accompanied by fibrosis and tissue death is a characteristic of the severe form of pancreatitis [7]. With an annual cost of more than 2.5 billion dollars, acute pancreatitis is the most common gastrointestinal reason for hospitalization in the United States [8].

According to the most recent revised Atlanta classification, the severity of acute pancreatitis can be defined as mild, moderate, or severe, affecting the prognosis. Only 15-20% of individuals experience severe acute pancreatitis (severe abdominal pain); the majority of patients have mild or moderate acute pancreatitis [9]. Notably, the mortality of mild or moderate Acute Pancreatitis is far less than that of Severe Abdominal Pain. The mortality is approximately 1% among all Acute Pancreatitis patients, but Reaching as high as 20% to 30% among those with severe course [10]. Because of its high death rate, severe acute pancreatitis must be detected as soon as possible to be managed and risk-categorized. Pancreatitis has varying incidence rates among nations, depending on factors such as alcohol consumption, gallstones, metabolic disorders, drug use, etc [11].

Surgeon John H. C. Ranson, MD, oversaw the General Surgery division at NYU and was also its director. He created a widely utilized method for forecasting the prognosis of pancreatic illness and contributed to the improvement of pancreatitis treatment [12]. Acute Pancreatitis was co-authored by Dr. Ranson. A clinical prediction rule for estimating acute pancreatitis mortality is called a ransom criteria [13]. Age, white cell count, blood glucose, serum lactate dehydrogenase (LDH), aspartate transaminase (SGOT), hematocrit level, blood urea nitrogen (BUN), PaO2, base deficit, serum calcium, and fluid

sequestration are the factors that determine the severity of acute pancreatitis [14].

The Bedside Index for Mortality in Acute Pancreatitis (BISAP) score is a tool used to identify patients who are at high risk of mortality or severe disease early in the course of acute pancreatitis, as well as to estimate the risk of in-hospital mortality in patients with acute pancreatitis [15]. Its objective is to measure how well the BISAP score predicts mortality and severe acute pancreatitis. The BISAP takes into account five factors: age greater than 60 years old, presence of pleural effusion, development of systemic inflammatory response syndrome, blood urea nitrogen level >25 mg/dl, and poor mental capacity [16].

Methods and Materials

Study design: To forecast the mortality of individuals with acute pancreatitis, a descriptive study design was used. **Study Setting:** This study was carried out in the Medical Intensive Care Unit, Saveetha Medical College of Hospital for a month. **Study Participants:** Thirty patients who met the inclusion criteria and had acute pancreatitis in the chosen location (n = 30) were enrolled in the study. Patients with acute pancreatitis met the inclusion criteria for study participants**. Informed consent:** Patients with acute pancreatitis were given a thorough explanation of the study's goal, which included predicting their death, and their informed agreement was obtained. The formal permission was obtained from the Principal, of Saveetha Medical College & Hospital, Chennai and the Medical Superintendent, of Saveetha Medical College & Hospital, Chennai. Informed consent was obtained from the acute pancreatitis patients. Confidentiality of information collected was assured to the samples. The demographic characteristics like age, gender, occupation, religion, residence, the habit of smoking and alcoholism and clinical characteristics of Blood Urea Nitrogen, mental status, Systematic inflammatory response syndrome, pleura effusion, Blood investigations like WBC count, serum AST, LDH, Calcium levels, hematocrit fall, sequestration of fluid were collected by using the tool developed for the study [17]. The severity of the acute pancreatitis among the patients was assessed by using the BISAP and RANSON methods. The data collected were then coded and entered into Excel for further data analysis and interpretation. **Results**: According to the study's findings, the RANSON score has a higher mortality prediction than the BISAP score.

Results

The analysis revealed that in most of the patients with acute pancreatitis, the Majority of 12(40%) were aged between 46 – 65 years, 15(50%) were male and female respectively, 14(46.7%) were moderate workers, 25(83.3%) were Hindus, 9(30%) were residing in urban area, 22(73.3%) had no habit of smoking and 23(76.7%) had no habit of alcoholism. The analysis revealed in shows that the demographic variable residence (χ^2=18.938, p=0.026) had shown statistically significant association with RANSON score of predicting mortality among patients with acute pancreatitis at $p<0.05$ level and the other demographic variables had not shown statistically significant association with RANSON score of predicting mortality among patients with acute pancreatitis.

The BISAP scoring system includes Blood urea of more than 25 mg/dl, presence of impaired mental status, presence of systemic response syndrome, Age more than 60 years and presence of pleural effusion given score as one absence of all these parameters as given as zero. A score of 0-2 points indicates lower mortality and 3-5 indicates a higher mortality rate in acute pancreatitis. The analysis revealed in shows that by BISAP score, 20(66.7%) had higher mortality (>15%) and

10(33.3%) had lower mortality (<2%).

RANSON scoring system is assessed on admission and 48 hours after admission. The following parameters were assessed age, Total WBC count, LDH, AST and blood glucose level. After 48hrs of assessment serum calcium level less than 2mmol/litre, drop in hematocrit of 10% and fall in partial pressure of oxygen less than 60%, elevation of blood urea nitrogen of more than 5mg/dl, base negative of more than 4 mEq/l, Sequestration of fluids more than 6 litres. Score interpretation includes 0-2 indicates 2% mortality, score 3-4 indicates 15% mortality, score 5-6 indicates 40% mortality, score 7-8 indicates 100% mortality. The results shows that by Ranson score, 20(66.7%) had 100% mortality, 5(16.7%) had 40% mortality, 4(13.3%) had 15% mortality and only 1(3.3%) had 2%mortality. According to the study's findings, the RANSON score has a higher mortality prediction than the BISAP score.

Table 1. Frequency and Percentage Distribution of Demographic Variables of Patients with Acute Pancreatitis**.**

N = 30

Demographic Variables	**Frequency**	**Percentage**
Age in years		
18 – 30	5	16.7
31 – 45	8	26.6
46 – 65	12	40.0
>65	5	16.7
Gender		
Male	15	50.0
Female	15	50.0
Others	-	-
Occupation		
Sedentary worker	7	23.3
Moderate worker	14	46.7
Heavy worker	9	30.0
Religion		
Hindu	25	83.3
Muslim	2	6.7
Christian	3	10.0
Others	-	-
Residence		
Urban	9	30.0
Semi-urban	8	26.7
Rural	5	16.6
Slum	8	26.7
Habit of smoking		
Yes	8	26.7
No	22	73.3
Habit of alcoholism		
Yes	7	23.3
No	23	76.7

The data shows that most of the 12(40%) were aged between 46 – 65 years, 15(50%) were male and female respectively, 14(46.7%) were moderate workers, 25(83.3%) were

Hindus, 9(30%) were residing in an urban area, 22(73.3%) had no habit of smoking and 23(76.7%) had no habit of alcoholism tabulated in Table I and Figure I.

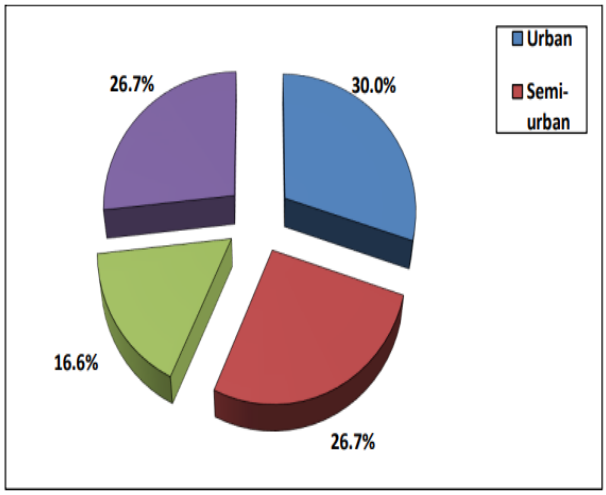

Figure 1. Percentage Distribution of Residence of Patients with Acute Pancreatitis

Table 2. Frequency and Percentage Distribution of RANSON Score in Predicting Mortality Among Patients with Acute Pancreatitis.

N = 30

RANSON Score	F	%
2% mortality	1	3.3
15% mortality	4	13.3
40% mortality	5	16.7
100% mortality	20	66.7

The above data shows that by Ranson score, 20(66.7%) had 100% mortality, 5(16.7%) had 40% mortality, 4(13.3%) had 15% mortality and only 1(3.3%) had 25 mortality tabulated in Table 2 and Figure 2.

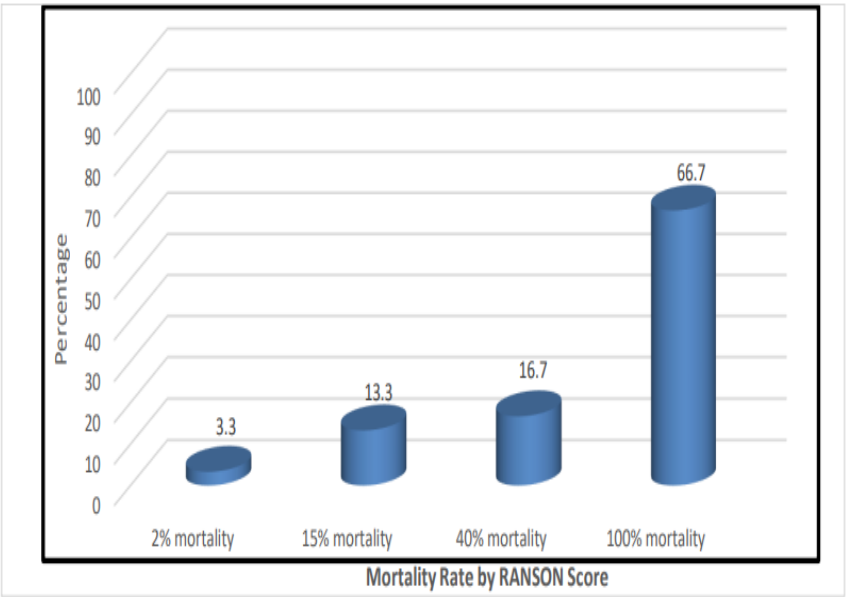

Figure 2. Percentage Distribution of Mortality Rate by RANSON Score

Table 3. Frequency and Percentage Distribution of BISAP Score in Predicting Mortality Among Patients with Acute Pancreatitis

N = 30

BISAP Score	F	%
Lower mortality (<2%)	10	33.3
Higher mortality (>15%)	20	66.7

The above data shows that by BISAP score, 20(66.7%) had higher mortality (>15%) and 10(33.3%) had lower mortality (<2%) tabulated in Table 3 and Figure 3.

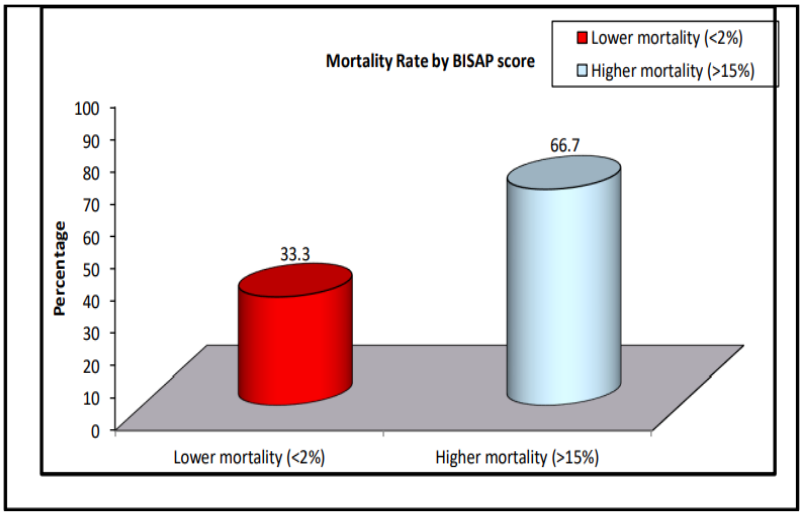

Figure 3. Percentage Distribution of Mortality Rate by BISAP Score

Table 4: Comparing the Mortality Prediction of RANSON and BISAP Score Frequency and Percentage

N = 30

	Frequency	Percentage	Chi-square & P value
BISAP score			
Lower mortality (<2%)	10	33.3	χ^2=0.600 d.f=1 p=0.439 N.S
Higher mortality (>15%)	20	66.7	
RANSON			
2% mortality	1	3.3	χ^2=18.938 d.f=9 p=0.026 S*
15% mortality	4	13.3	
40% mortality	5	16.7	
100% mortality	20	66.7	

*P<0.05, S – Significant, N.S – Not Significant

The above Table 4 shows that by Ranson score, 20(66.7%) had 100% mortality, 5(16.7%) had 40% mortality, 4(13.3%) had 15% mortality and only 1(3.3%) had 25 mortality. The above table shows that by BISAP score, 20(66.7%) had higher mortality (>15%) and 10(33.3%) had lower mortality (<2%).

Discussion

Acute pancreatitis is a common gastrointestinal condition that causes hospital admissions in the United States and globally.

Over the past decade, significant progress has been made in understanding the pathophysiological mechanisms behind acute pancreatitis. Research has identified the mechanisms underlying calcium-induced acinar cell damage and death, as well as the significance of calcium entry channels controlled by stores and mitochondrial permeability transition pores. The cytoprotective function of autophagy and the unfolded protein response in preventing prolonged endoplasmic reticulum stress, apoptosis, and necrosis, along with the pivotal role of unsaturated fatty acids in inducing pancreatic organ failure, have also been documented.

The diagnosis of acute pancreatitis relies on clinical, biochemical and/or radiological criteria and can be made if two of the following three features are present acute onset of severe abdominal pain often radiating to the back; serum lipase and/or amylase activity at least three times the upper limit of normal [17]. It is important to identify patients with acute pancreatitis who are at risk for developing persistent organ failure early in the course of the disease. Several scoring systems have been developed to predict which patients are most likely to develop persistent organ failure [18]. According to the study, individuals with acute pancreatitis can effectively forecast their mortality using the BISAP score and the Ranson score as prognostic tools. However, according to the study findings, the RANSON score might perform better at the bedside in terms of accuracy, usability, and simplicity than the BISAP score. In particular, we discovered that when it came to identifying patients who were at risk of dying, the RANSON score outperformed the BISAP score in terms of sensitivity, specificity, and positive predictive value.

The BISAP scoring system includes Blood urea of more than 25 mg/dl, presence of impaired mental status, presence of systemic response syndrome, Age more than 60 years and presence of pleural effusion given score as one absence of all these parameters as given as zero. A score of 0-2 points indicates lower mortality and 3-5 indicates a higher mortality rate in acute pancreatitis. The analysis revealed in shows that by BISAP score, 20(66.7%) had higher mortality (>15%) and 10(33.3%) had lower mortality (<2%). RANSON scoring system is assessed on admission and 48 hours after admission. The following parameters were assessed age, Total WBC count, LDH, AST and blood glucose level. After 48hrs of assessment serum calcium level less than 2mmol/litre, drop in hematocrit of 10% and fall in partial pressure of oxygen less than 60%, elevation of blood urea nitrogen of more than 5mg/dl, base negative of more than 4 mEq/l, Sequestration of fluids more than 6 litres.

According to the study's findings, the RANSON score has a higher mortality prediction than the BISAP score. The results were supported by The Ranson score shows a high sensitivity in predicting severe acute pancreatitis and related consequences, according to results corroborated. Better risk categorization is made possible by the Ranson score's comprehensiveness, especially in complex circumstances.

The scores of the BISAP, Ranson's, APACHE-II, and CTSI, which further corroborated the findings [19]. They discovered that although BISAP is effective, Ranson's score which has numerous parameters offers a more sophisticated picture of the patient's state, which is essential for comprehensive treatment and long-term prognosis. Ranson’s score of more than 3 and the BISAP score of less than or equal to 3 had the best accuracy in predicting the severity of acute pancreatitis. Both Ranson’s score and BISAP score showed higher sensitivity in the prediction of systemic complications than that

of local complications.

This study is also supported by Balasubramanium V (2021) Many scoring systems help predict mortality in acute pancreatitis patients some of the scoring systems are also helpful as the Early Warning Score (EWS) is a widely used scoring system for monitoring patient progress, which we have previously shown to predict the outcome from acute pancreatitis [20]. According to the study's findings, the RANSON score has a higher mortality prediction than the BISAP score.

Conclusion

According to the study's findings, the RANSON score has a higher mortality prediction than the BISAP score. The BISAP score is frequently chosen in contemporary clinical practice because of its simplicity, quickness, and convenience of use, even though the Ranson score offers a more thorough assessment. Depending on the clinical context, the severity of the issue, and the available resources, one should choose between the two. In many instances, combining the two scores can offer a well-rounded strategy that makes the most of both to improve patient care.

Conflict of Interest

None.

Acknowledgement

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