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Acute pancreatitis: predictors of mortality, pancreatic necrosis and intervention

Ramlal Prajapati[1], Priyadarshini Manay[2], Kavin Sugumar[1], Vinay Rahandale[3], Rajeev Satoskar[3]

[1] Department of Surgery, University Hospitals, Seidman Cancer Center, Cleveland, United States
[2] Department of Transplant Surgery, University of Iowa Hospitals & Clinics, Iowa City, United States
[3] Department of Surgery, Seth G.S Medical College and King Edward Memorial Hospital, Mumbai, India

ABSTRACT

Objective: Several predictive scoring systems are used in the prognostication of acute pancreatitis (AP). However, the quantity of evidence of these prognostic systems in the Indian population remains sparse. The aim of our study was to evaluate the usefulness of such prognostic scores to predict mortality, incidence of pancreatic necrosis and intervention in AP.

Material and Methods: This was an observational study of patients diagnosed with AP between June 2012 and November 2013 in a tertiary referral center in India. Vital signs, biochemical tests and CT-findings were recorded to identify SIRS, Ranson's score and CT-severity index at diagnosis. Chi square test was used to compare incidence of mortality, pancreatic necrosis, and intervention between mild versus severe acute pancreatitis groups.

Results: A total of 100 patients with AP were treated during out study period. Ranson's score more than 7 and presence of pancreatic necrosis were significantly associated with increased mortality ($p< 0.05$). SIRS, CTSI score more than 7, inotropic support, and complications were more frequently associated with patients with necrosis. Prophylactic antibiotics did not decrease mortality, but decreased intervention rate ($p< 0.05$). Presence of systemic inflammatory response syndrome (SIRS), Ranson's score > 7, necrosis, inotropic support and presence of complications were associated with a greater rate of interventions including surgery and percutaneous procedures ($p< 0.05$).

Conclusion: We validate SIRS, Ranson's, and CTSI score as prognostic markers for AP in the Indian population. These predictors, when used in combination, can direct early monitoring and aggressive management in order to decrease mortality associated with severe AP.

Keywords: Acute pancreatitis, prognostic score, Ranson's score, CT-severity index, necrosis, SIRS

INTRODUCTION

Acute pancreatitis (AP) is one of the most common causes of inpatient admission worldwide, with an annual incidence of 15-36 among 100,000 persons (1). With advances in our understanding of the pathophysiology of AP, outcomes have improved over the last few decades. However, severe forms of AP are still associated with a high morbidity of 25-70% (2) and a mortality rate of 13.5% (3). Early prognostication of AP is crucial to reduce morbidity and mortality in such patients. Several prognostic biochemical, imaging and clinical scores have been created for treating AP in the past. These include but are not limited to Ranson's score (4), CT-severity index (CTSI) (5), BISAP (6), SOFA (7), Glasgow (8), APACHE-II (9), to name a few. Although many studies comparing the effectiveness of these scoring systems have been conducted worldwide, there is sparse evidence in the Indian subcontinent.

Our aim was to demonstrate the predictive effect of clinical signs and scoring systems in identifying AP patients at the highest risk of mortality, pancreatic necrosis, and need for intervention.

MATERIAL and METHODS

Patient Selection

This study was conducted at a tertiary referral center in Mumbai, India from June 2012 to November 2013. We included all patients between 18 and 70 years of age who presented to our outpatient department or emergency department for the first time with clinical presentation suggestive of AP. Patients were excluded if they

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Corresponding Author
Kavin Sugumar
E-mail: dr.kavinsugumar@gmail.com

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were diagnosed as acute or chronic pancreatitis or if they presented following any surgical or percutaneous intervention at an outside facility or after five days of initial symptom onset.

Covariates and Outcomes

Demographic information including age, gender, history of chronic alcohol intake and gallstone disease was obtained. On admission, vital signs including temperature, pulse, blood pressure, respiratory rate, and lab investigations such as white blood cell count (WBC), random blood sugar (mg/dl), and C-reactive protein (CRP, mg/L) were recorded. Ranson's score at admission was calculated using biochemical and clinical parameters. We did not calculate the Ranson's score at 48 hours, as we preferred the ability to predict who would have severe AP without having to wait two days for an elevated Ranson's score. In conjunction with Ranson's score at admission, presence of systemic inflammatory response syndrome (SIRS) was identified on the basis of the following parameters: temperature, pulse, respiratory rate, and white blood cell counts (10). Contrast enhanced CT of the abdomen was obtained on day fifth of symptom onset. Based on clinical evaluation by the attending surgeon, patients were started on prophylactic antibiotics prior to any form of imaging. Both local and systemic complications were recorded.

Ranson's score was utilized to demonstrate biochemical severity and CTSI was used to grade radiological severity. Hence, a comprehensive triumvirate assessment was made combining clinical and biochemical (SIRS and Ranson's score) and radiological findings (CTSI score). Ranson's score more than 3 was considered as severe (4), and CTSI more than 7 was considered severe (5).

The main outcomes of this study included mortality, rate of intervention and presence of parenchymal necrosis. Intervention included any form of surgical or percutaneous intervention that the patient underwent during the in-patient course. The proportion of pancreatic necrosis was broken down into involvement of less than and more than 30% of the parenchyma on imaging.

Statistical Analysis

Continuous variables were represented as median with range, and categorical variables were represented as frequency with percentages. Categorical variables were analyzed using Pearson Chi square test or Fishers Exact test. All statistical analysis was performed by SPSS (SPSS Inc. Released 2007. SPSS for Windows, Version 16.0. Chicago, SPSS Inc) and significance was defined as p-value less than 0.05.

RESULTS

Patient Demographics

A total of 100 consecutive patients diagnosed with AP were included in the study. Baseline patient characteristics are shown in Table 1.

Table 1. Patient characteristics

Parameter	n (%) or median (with range)
Age (years)	36.5 (18-75)
Sex	
Male	74 (74%)
Female	26 (26%)
Etiology	
Alcoholic	64 (64%)
Gallstone	28 (28%)
Idiopathic	8 (8%)
Pancreatic necrosis	
No necrosis	58 (61%)
<30% parenchymal necrosis	10 (10%)
>30% parenchymal necrosis	27 (28%)
SIRS*	77 (77%)
Intervention	
Surgery	13 (13%)
ERCP*	2 (2%)
Image guided drainage	3 (3%)
Non-image guided drainage	2 (2%)
Status at discharge	
Alive	95 (95%)
Dead	5 (5%)

*SIRS: Systemic inflammatory response syndrome, ERCP: Endoscopic retrograde cholangiopancreaticography.

Factors Associated With Mortality

Ninety-eight patients fell into mild Ranson's score category, of which 3 died (3%) (Table 2). Only two patients had a severe Ranson's score and both died (100%). There was a significant association between Ranson's score and mortality (p= 0.004). Presence of pancreatic necrosis resulted in mortality in five patients (5/42, 12%). There was a significant association between necrosis and mortality (p= 0.022).

There was no association between mortality and the following parameters: SIRS (p= 0.52), CTSI (p= 0.563), inotropic support (p= 0.215), ventilatory support (p= 1), CRP (p= 0.6), random blood sugar (alcoholic pancreatitis, p= 0.31; gall stone pancreatitis, p= 0.14), antibiotic use (p= 0.353), and complications (p= 0.578).

Factors Associated With Pancreatic Necrosis

Among those who developed SIRS, 49% had pancreatic necrosis, whereas among those that did not have SIRS, only 18% had necrosis (Table 3). SIRS was more commonly associated with necrosis (p= 0.006). All patients who had a severe CTSI score > 7 had necrosis (21/21, 100%, p< 0.0001). 83% of patients (n= 10/12) who were on inotropic support had pancreatic necrosis. There was a significant relationship between the need for inotropic support and necrosis (p= 0.002).

Table 2. Factors associated with mortality

Parameter	Alive	Dead	p
SIRS*			
Absent	23 (100%)	0 (0%)	0.52
Present	72 (93.5%)	5 (6.5%)	
Ranson's score			
Mild	95 (97%)	3 (3%)	**0.004**
Severe	0 (0%)	2 (100%)	
CT-severity index			
Mild	76 (96%)	3 (4%)	0.563
Severe	19 (90%)	2 (10%)	
Pancreatic necrosis			
Absent	58 (100%)	0 (0%)	**0.022**
Present	37 (88%)	5 (12%)	
Inotropic support			
Yes	10 (83%)	2 (17%)	0.215
No	85 (96%)	3 (4%)	
Ventilator support			
Yes	1 (100%)	0 (0%)	1
No	85 (96%)	3 (4%)	
C-reactive protein			
<150 mg/L	23 (88%)	3 (12%)	0.6
>150 mg/L	4 (80%)	1 (20%)	
Random blood sugar			
Alcoholic pancreatitis			0.31
<200 mg/dl	63 (95%)	3 (5%)	
>200 mg/dl	1 (66%)	1 (34%)	
Gall stone pancreatitis			0.14
<220 mg/dl	26 (100%)	0 (0%)	
>220 mg/dl	1 (50%)	1 (50%)	
Antibiotic use			
Prophylactic antibiotics	40 (97%)	1 (3%)	0.353
Therapeutic antibiotics	41 (93%)	3 (7%)	
Complications			
Absent	68 (95%)	3 (5%)	0.578
Present	27 (93%)	2 (7%)	

*SIRS: Systemic inflammatory response syndrome.

Eighty-five patients received either prophylactic or therapeutic antibiotics. Prophylactic antibiotics was less frequently associated with presence of necrosis (3/41, 7%, p< 0.0001). Five patients out of 58 (18%) without pancreatic necrosis developed local or systemic complications, whereas 24 of 37 patients (65%) with necrosis developed complications. There was a significant relationship between pancreatic necrosis and complication rate (p< 0.0001).

Out of 56 patients with no complications, 3 patients (5%) had pancreatic necrosis <30% and 53 patients (95%) did not have necrosis. Out of 12 patients with complications, 7 patients (58%) had pancreatic necrosis <30%; complications included respiratory complications (2/7, 28%), infected necrosis (1/7, 14%), ascites (1/7, 14%), and pseudocyst (3/7, 42%). The remaining 5 patients (42%) did not have necrosis and developed complications including respiratory complications (5/5, 100%) and pseudocyst (1/5, 20%). There was a significant association between necrosis (none vs <30%) and complication rate (p= 0.00015).

Among 11 patients with no complications, 3 patients (27%) had pancreatic necrosis <30% and 8 patients (73%) had necrosis >

Table 3. Factors associated with pancreatic necrosis

Parameter	Necrosis	No necrosis	p
SIRS*			
Absent	4 (18%)	19 (82%)	**0.006**
Present	38 (49%)	39 (51%)	
Ranson's score			
Mild	40 (41%)	58 (59%)	0.34
Severe	2 (100%)	0 (0%)	
CT-severity index			
Mild	21 (26%)	58 (74%)	**<0.0001**
Severe	21 (100%)	0 (0%)	
Inotropic support			
Yes	10 (83%)	2 (17%)	**0.002**
No	32 (36%)	56 (64%)	
Ventilator support			0.238
Yes	1 (100%)	0 (0%)	
No	41 (41%)	58 (59%)	
C-reactive protein			
<150 mg/L	16 (61%)	10 (39%)	0.67
>150 mg/L	2 (40%)	3 (60%)	
Random blood sugar			
Alcoholic pancreatitis			
<200 mg/dl	30 (49%)	31 (51%)	0.554
>200 mg/dl	2 (67%)	1 (34%)	
Gall stone pancreatitis			
<220 mg/dl	5 (18%)	22 (82%)	0.051
>220 mg/dl	1 (100%)	0 (0%)	
Antibiotic use			
Prophylactic antibiotics	3 (7%)	38 (93%)	**<0.0001**
Therapeutic antibiotics	24 (54%)	20 (46%)	
Complications			
Absent	8 (13%)	53 (87%)	**<0.0001**
Present	24 (83%)	5 (17%)	
Complications	No necrosis	<30% necrosis	
Absent	53 (95%)	3 (5%)	**0.0001**
Present	5 (42%)	7 (58%)	
Complications	<30% necrosis	>30% necrosis	
Absent	3 (27%)	8 (73%)	1
Present	7 (22%)	24 (78%)	

*SIRS: Systemic inflammatory response syndrome.

30%. Out of 31 patients with complications, 7 patients (22%) had pancreatic necrosis <30%. Twenty-four patients (78%) that had necrosis > 30% had complications including respiratory complications (15/24, 62%), infected necrosis (7/24, 30%), ascites (5/24, 21%), and pseudocyst (5/24, 21%). There was no association between extent of necrosis (<30% vs >30%) and complication rate (p= 1).

There was no significant association between pancreatic necrosis and Ranson's score (p= 0.34), ventilator support (p= 0.238), CRP (p= 0.67), and random blood sugar (alcoholic pancreatitis, p= 0.554; gall stone pancreatitis, p= 0.051).

Factors Associated With the Rate of Intervention

Eighteen percent of the patients with SIRS required intervention, compared to 9% of patients without SIRS who required the

Table 4. Factors associated with the rate of intervention

Parameter	No intervention	Intervention	p
SIRS*			
Absent	63 (82%)	14 (18%)	**0.0345**
Present	21 (91%)	2 (9%)	
Ranson's score			
Mild	84 (86%)	14 (14%)	**0.0484**
Severe	0 (0%)	2 (100%)	
CT-severity index			
Mild	68 (86%)	11 (14%)	0.2735
Severe	16 (76%)	5 (24%)	
Pancreatic necrosis			
Absent	55 (95%)	3 (5%)	**0.001**
Present	29 (69%)	13 (31%)	
Inotropic support			
Yes	78 (89%)	10 (11%)	**0.001**
No	6 (50%)	6 (50%)	
Ventilator support			
Yes	83 (84%)	16 (16%)	0.661
No	1 (100%)	0 (0%)	
C-reactive protein			
<150 mg/L	18 (69%)	8 (31%)	1
>150 mg/L	3 (34%)	2 (66%)	
Random blood sugar			
Alcoholic pancreatitis			0.507
<200 mg/dl	50 (82%)	11 (18%)	
>200 mg/dl	2 (67%)	1 (36%)	
Gall stone pancreatitis			
<220 mg/dl	24 (89%)	3 (11%)	0.724
>220 mg/dl	1 (100%)	0 (0-%)	
Antibiotic use			
Prophylactic antibiotics	41 (100%)	0 (0%)	**<0.0001**
Therapeutic antibiotics	31 (70%)	13 (30%)	
Complications			
Absent	22 (61%)	14 (39%)	**0.000028**
Present	62 (97%)	2 (3%)	

*SIRS: Systemic inflammatory response syndrome.

same (Table 4). There was a significant relationship between SIRS and rate of intervention (p= 0.0345). Out of 98 patients with mild Ranson's score, only 14 patients (14%) underwent intervention while both patients (100%) with severe Ranson's score needed intervention. There was a significant association between Ranson's score and the need for intervention (p= 0.0484).

Thirty-one percent of the patients with necrosis required intervention compared to 5% who did not have necrosis. There was a significant relationship between necrosis and rate of intervention (p= 0.001). Fifty percent of the patients who were on inotropic support required intervention, compared to 11% of those who did not require it. There was a significant relationship between inotropic support and rate of intervention (p= 0.001). None of the patients who were administered antibiotics required any form of intervention (p< 0.0001).

Out of 64 patients with no complications, 2 patients (3%) underwent intervention, and out of 36 patients with complications, 14 patients (18%) underwent intervention. There was a significant association between complication and intervention rate (p= 0.000028).

There was no significant relationship between the rate of intervention and CTSI (p= 0.2735), ventilator support (p= 0.661), CRP (p= 1), and random blood sugar (alcoholic pancreatitis, p= 0.507; gall stone pancreatitis, p= 0.724).

DISCUSSION

Severe acute pancreatitis (SAP) develops in 15-25% of the patients diagnosed with AP (11). Such patients have a protracted hospital course with a higher rate of morbidity and mortality (12). Hence, the early identification of severity is one of the most essential steps in the management of AP. Several prognostic scoring indices like Ranson's score, CTSI, Glascow scoring system, APACHE=II, and BISAP score (13) have proven to be useful to ascertain the severity of disease in the past. They have varying sensitivity ranging from 55-90% in predicting severe AP, but the accuracy depends on the cut-off value and time of scoring8. Ranson's and APACHE score are limited by complexity in a number of parameters included but at the same time have maximum likelihood of predicting mortality (14). CTSI score has a similar sensitivity of predicting severity, yet it has limited use in earlier stages of AP as CT findings within 72 hours of symptoms are usually normal, and local complications like hemorrhage and abscess formation occur much later in the course of AP (14). BISAP is a much simpler bed side prognostic score compared to other scoring systems with equivalent predictive value (13).

This study showed that SIRS was not associated with a higher mortality rate. A study by Buter et al. investigating the effect of SIRS and MODS on mortality has concluded that MODS but not SIRS is associated with mortality on multivariable analysis16. The reason for this finding can be explained by the fact that even though transient mild SIRS is common in early stages of AP, it does not translate to mortality but persistent worsening of SIRS score during the inpatient course which indicates progression to sepsis and organ dysfunction would have a higher probability of death.

We found that SIRS was associated with pancreatic necrosis and an increase in intervention rate which could be considered as a surrogate marker of morbidity. A study by Singh et al. evaluating the role of SIRS on assessing AP severity has found that SIRS predicts AP severity and complications including necrosis with a high sensitivity of 85-100%. The higher the SIRS score on day 1 of admission, the greater the risk of severe AP (17). A study by Gregoric et al. investigating the role of SIRS and IL-6 in AP has found that it correlated with in-hospital morbidity (18). In our study, we studied the effect of SIRS as a prognostic factor as it represents the body's initial inflammatory response to an insult. The insult can be compounded by necrosis which could result to rapid deterioration of the patient's status during hospital stay. Similar sequential insults can cause a maladaptive response leading to multi-organ dysfunction syndrome (MODS). When sepsis supervenes, it can result in worse prognosis.

In this study, higher Ranson's score was associated with higher mortality but not with the presence of pancreatic necrosis. Khanna et al. have conducted a retrospective cohort study comparing various prognostic systems and concluded that Ranson's score was more reliable in determining mortality but not as accurate in predicting pancreatic necrosis compared to CTSI score, CRP and IL-6 on ROC analysis (8). Kumar et al. have similarly studied the predictive value of various prognostic systems and found that pancreatic necrosis was most accurately determined by CTSI followed by APACHE and then Ranson's score by ROC analysis (15). Ranson's score is a composite marker consisting of clinical and biochemical parameters which reflect the systemic status of the patient, hence a good marker of mortality. It is calculated at and within 48 hours of diagnosis, during which parenchymal necrosis development is not complete; which could explain why it is not a better predictor of necrosis compared to CTSI score that quantifies severity based on local complications. In addition, our study showed that Ranson's score was associated with higher intervention rate comprising percutaneous procedures and laparotomy. In our literature review, we did not find similar studies comparing Ranson's score to intervention rate, which can be considered as a surrogate marker of degree of morbidity.

In our study, modified CTSI score was used for assessing severity and was associated with pancreatic necrosis. CTSI score consists of presence of fluid collection in the vicinity of the pancreas and also quantifies the collection or necrosis (19). However, CTSI score was not associated with mortality. A metanalysis conducted by Miko et al. has evaluated the predictive value of mortality between CTSI and other prognostic systems and concluded that CTSI score has a sensitivity of 79% in predicting mortality which was lower compared to APACHE, Ranson, and BISAP score on ROC analysis (19). Similarly, Georgios et al. have compared CTSI, BISAP, Ranson's, and APACHE score in predicting mortality and concluded that CTSI had lower predictive ability compared to APACHE, Ranson's and BISAP score (20). Hence, CTSI may be more important as a radiological marker of severity in terms of local complications like necrosis and hemorrhage in comparison to clinical status of the patient.

Our study showed that pancreatic necrosis was associated with higher morbidity rate in the form of local and systemic complications. Balthazar et al. have studied the prognostic value of CT in predicting severity of disease and concluded that pancreatic necrosis on CT is associated with morbidity in up to 80% of patients (21). Even though our study showed the relationship between necrosis and overall morbidity, it did not depend on the percentage of necrosis. This finding can be due to many reasons. Even though increasing parenchymal necrosis would increase the likelihood of infection or local complications, it does not necessarily lead to systemic complications like pleural

effusion, acute kidney injury (AKI) which may vary by case-to-case basis. Another reason could be that in our cohort, most systemic complications were mild, thus not correlated with percentage of necrosis. Also, we showed that necrosis increases the intervention rate. Pancreatic necrosis requires surgical intervention in 10% of cases, due to infection, hemorrhage, abscess, or bowel perforation (22).

Our study showed that pancreatic necrosis was associated with increased mortality. Overall mortality of AP is around 1-2% in the US population (23) but in severe cases with parenchymal necrosis, mortality is increased to nearly 40% (24). Pancreatic necrosis can lead to a multitude of local complications including infection, hemorrhage, bowel perforation, and fistula formation which considerably increases the mortality rate (25).

Our study put forth that those who had pancreatic necrosis and required intervention needed inotropic support. Alteration in pancreatic microcirculation due to circulating interleukins and TNF-alpha lead to overall fluid sequestration including the pancreatic parenchyma. This can result in hypovolemia coupled with hypoxic damage to the pancreas leading to pancreatic necrosis (26). The resulting hypoperfusion may result in the need for inotropic support. The resulting SIRS, when coupled with gut dysmotility and barrier dysfunction can lead to superinfection of necrosis. In addition, hypovolemia can result in pre-renal azotemia. Other systemic complications include acute respiratory distress syndrome, pleural effusion, anorexia, and electrolyte abnormalities to name a few. All of these complications may increase the overall rate of intervention.

Superinfection of pancreatic necrosis is believed to be due to bacterial translocation from the gut via the enteric blood vessels or lymphatic pathways to the pancreatic parenchyma. Even though the exact mechanism of bacterial translocation is still not clear, various possible mechanisms have been illustrated that include alteration in intestinal flora, impaired gut barrier, or maladaptive immune reponsse (28). Hence, there is a push for administering prophylactic antibiotics in patients with parenchymal necrosis. Our study showed that prophylactic antibiotic use was associated with lesser incidence of necrosis. Pancreatic necrotic patients still continued to have a higher rate of local and systemic complications. A recent meta-analysis has shown that prophylactic antibiotics decreases superinfection of pancreatic necrosis, but does not decrease mortality or rate of intervention (29). Even though prophylactic antibiotics prevent superinfection, they may not decrease the rate of necrosis or other systemic complications. Hence, the use of prophylactic antibiotics in patients with parenchymal necrosis is still controversial.

Our study revealed that the natural progression of AP leading to various systemic and local complications increased the need for intervention. Complications like infected necrosis and peripancreatic necrosis are known to increase the rate of interventions which are mainly necrosectomy (27).

The findings of our study must be elucidated in light of certain limitations. From a statistical standpoint, though this study included a large sample size of 100 patients, power analysis was not performed to determine adequate sample size. This might explain some of the non-significant results. We chose Ranson's score calculated at admission and SIRS as the prognostic markers for this study. Even though the use of Ranson's at 48 hours and other systems like APACHE would be preferred, it was not feasible due to their increased complexity.

To conclude, SIRS, Ranson's score and CTSI prove to be valuable indicators of AP severity in the Indian population. Patients having Ranson's score more than 3, SIRS, and pancreatic necrosis must be carefully monitored in an intensive care unit to achieve better outcomes.

Ethics Committee Approval: The approval for this study was obtained from Seth G.S Medical College and King Edward Memorial Hospital Ethics Committee (Decision no: IEC(II)/OUT/986/14 Date: 03.11.2014).

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REFERENCES

1. *Roberts SE, Akbari A, Thorne K, Atkinson M, Evans PA. The incidence of acute pancreatitis: Impact of social deprivation, alcohol consumption, seasonal and demographic factors. Aliment Pharmacol Ther 2013; 38(5): 539-48.* ***[CrossRef]***
2. *Zerem E. Treatment of severe acute pancreatitis and its complications. World J Gastroenterol 2014; 20(38): 13879-92.* ***[CrossRef]***
3. *Mann DV, Hershman MJ, Hittinger R, Glazer G. Multicentre audit of death from acute pancreatitis. Br J Surg 1994; 81(6): 890-3.* ***[CrossRef]***
4. *Ruan GJ, Bhimji SS. Ranson Criteria; 2018.* ***[CrossRef]***
5. *Sahu B, Abbey P, Anand R, Kumar A, Tomer S, Malik E. Severity assessment of acute pancreatitis using CT severity index and modified CT severity index: Correlation with clinical outcomes and severity grading as per the Revised Atlanta Classification. Indian Indian J Radiol Imaging 2017; 27(2): 152-60.* ***[CrossRef]***
6. *Sharma V, Rana SS, Sharma RK, Kang M, Gupta R, Bhasin DK. A study of radiological scoring system evaluating extrapancreatic inflammation with conventional radiological and clinical scores in predicting outcomes in acute pancreatitis. Ann Gastroenterol 2015; 28(3): 399-404.* ***[CrossRef]***

7. *Tee YS, Fang HY, Kuo IM, Lin YS, Huang SF, Yu MC. Serial evaluation of the SOFA score is reliable for predicting mortality in acute severe pancreatitis. Medicine (United States) 2018; 97(7): e9654.* **[CrossRef]**
8. *Khanna AK, Meher S, Prakash S, Tiwary SK, Singh U, Srivastava A, et al. Comparison of Ranson, Glasgow, MOSS, SIRS, BISAP, APACHE-II, CTSI Scores, IL-6, CRP, and procalcitonin in predicting severity, organ failure, pancreatic necrosis, and mortality in acute pancreatitis. HPB Surg 2013; 2013: 367581.* **[CrossRef]**
9. *Wu BU, Johannes RS, Sun X, Tabak Y, Conwell DL, Banks PA. The early prediction of mortality in acute pancreatitis: A large population-based study. Gut 2008; 57(12): 1698-703.* **[CrossRef]**
10. *Toliver-Kinsky T, Kobayashi M, Suzuki F, Sherwood ER. The systemic inflammatory response syndrome. In: Total Burn Care: Fifth Edition; 2018.* **[CrossRef]**
11. *Tenner S, Baillie J, Dewitt J, Vege SS. American college of gastroenterology guideline: Management of acute pancreatitis. Am J Gastroenterol 2013; 108(9): 1400-15.* **[CrossRef]**
12. *Chatila AT, Bilal M, Guturu P. Evaluation and management of acute pancreatitis. World J Clin Cases 2019; 7(9): 1006-20.* **[CrossRef]**
13. *Yadav J, Yadav SK, Kumar S, Baxla RG, Kumar Sinha D, Bodra P, et al. Predicting morbidity and mortality in acute pancreatitis in an Indian population: A comparative study of the BISAP score, Ranson's score and CT severity index. Gastroenterol Rep 2016; 4(3): 216-20.* **[CrossRef]**
14. *Cho JH, Kim TN, Chung HH, Kim KH. Comparison of scoring systems in predicting the severity of acute pancreatitis. World J Gastroenterol 2015; 21(8): 2387-94.* **[CrossRef]**
15. *Kumar AH, Griwan MS. A comparison of APACHE II, BISAP, Ranson's score and modified CTSI in predicting the severity of acute pancreatitis based on the 2012 revised Atlanta Classification. Gastroenterol Rep 2018; 6(2): 127-31.* **[CrossRef]**
16. *Buter A, Imrie CW, Carter CR, Evans S, McKay CJ. Dynamic nature of early organ dysfunction determines outcome in acute pancreatitis. Br J Surg 2002; 89(3): 298-302.* **[CrossRef]**
17. *Singh VK, Wu BU, Bollen TL, Repas K, Maurer R, Mortele KJ, et al. Early systemic inflammatory response syndrome is associated with severe acute pancreatitis. Clin Gastroenterol Hepatol 2009; 7(11): 1247-51.* **[CrossRef]**
18. *Gregoric P, Sijacki A, Stankovic S, Radenkovic D, Ivancevic N, Karamarkovicet A, al. SIRS score on admission and initial concentration of IL-6 as severe acute pancreatitis outcome predictors. Hepatogastroenterology 2010; 57(98): 349-53.* **[CrossRef]**
19. *Mikó A, Vigh É, Mátrai P, Soós A, Garami A, Balaskó M, et al. Computed tomography severity index vs. Other indices in the prediction of severity and mortality in acute pancreatitis: A predictive accuracy meta-analysis. Front Physiol 2019; 10: 1002.* **[CrossRef]**
20. *Papachristou GI, Muddana V, Yadav D, O'Connell M, Sanders MK, Slivka A, et al. Comparison of BISAP, Ranson's, APACHE-II, and CTSI scores in predicting organ failure, complications, and mortality in acute pancreatitis. Am J Gastroenterol 2010; 105(2): 435-41; quiz 442.* **[CrossRef]**
21. *Balthazar EJ, Robinson DL, Megibow AJ, Ranson JHC. Acute pancreatitis: Value of CT in establishing prognosis. Radiology 1990; 174(2): 331-6.* **[CrossRef]**
22. *Büchler MW, Gloor B, Müller CA, Friess H, Seiler CA, Uhl W. Acute necrotizing pancreatitis: Treatment strategy according to th status of infection. Ann Surg 2000; 232(5): 619-26.* **[CrossRef]**
23. *Banks PA, Freeman ML, Practice Parameters Committee of the American College of Gastroenterology. Practice guidelines in acute pancreatitis. Am J Gastroenterol 2006; 101(10): 2379-400.* **[CrossRef]**
24. *Bugiantella W, Rondelli F, Boni M, Stella P, Polistena A, Sanguinetti A, et al. Necrotizing pancreatitis: A review of the interventions. Int J Surg 2016; 28 Suppl 1: S163-71.* **[CrossRef]**
25. *Rashid MU, Hussain I, Jehanzeb S, Ullah W, Ali S, Jain AG, et al. Pancreatic necrosis: Complications and changing trend of treatment. World J Gastrointest Surg 2019; 11(4): 198-217.* **[CrossRef]**
26. *Lankisch PG, Apte M, Banks PA. Acute pancreatitis. In: The Lancet; 2015.* **[CrossRef]**
27. *Raraty MGT, Halloran CM, Dodd S, Ghaneh P, Connor S, Evans J, et al. Minimal access retroperitoneal pancreatic necrosectomy: Improvement in morbidity and mortality with a less invasive approach. Ann Surg 2010; 251(5): 787-93.* **[CrossRef]**
28. *Fritz S, Hackert T, Hartwig W, Rossmanith F, Strobel O, Schnieder L, et al. Bacterial translocation and infected pancreatic necrosis in acute necrotizing pancreatitis derives from small bowel rather than from colon. Am J Surg 2010; 200(1): 111-7.* **[CrossRef]**
29. *Lim CLL, Lee W, Liew YX, Tang SSL, Chlebicki MP, Kwa ALH. Role of antibiotic prophylaxis in necrotizing pancreatitis: a meta-analysis. J Gastrointest Surg 2015; 19(3): 480-91.* **[CrossRef]**

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Akut pankreatit: mortalite, pankreas nekrozu ve girişimlerin öngördürücüleri

Ramlal Prajapati[1], Priyadarshini Manay[2], Kavin Sugumar[1], Vinay Rahandale[3], Rajeev Satoskar[3]

[1] Üniversite Hastaneleri, Seidman Kanser Merkezi, Cerrahi Anabilim Dalı, Cleveland, Amerika Birleşik Devletleri
[2] Iowa Üniversitesi Hastane ve Kliniği, Organ Nakil Cerrahisi Anabilim Dalı, Iowa, Amerika Birleşik Devletleri
[3] Seth G.S Tıp Fakültesi ve King Edward Memorial Hastanesi, Cerrahi Anabilim Dalı, Mumbai, Hindistan

ÖZET

Giriş ve Amaç: Akut pankreatit (AP) prognozunu tahmin etmede birkaç skorloma sistemi kullanılmaktadır. Ancak, bu skorlama sistemlerinin Hint toplumundaki kanıt düzeyi kuşkuludur. Bu çalışmanın amacı, bu prognostik skorlama sistemlerinin AP'de mortalite, pankreatik nekroz insidansı ve girişimi öngörmedeki kullanışlılığını değerlendirmekti.

Gereç ve Yöntem: Bu çalışma, Hindistan'da üçüncü basamak bir merkezde Haziran 2012 ve Kasım 2013 tarihleri arasında AP tanısı alan hastaların gözlemsel bir çalışmaydı. Vital bulgular, biyokimyasal testler ve BT bulguları, SIRS, Ranson skoru ve BT-şiddet endeksini belirlemek amacıyla kaydedildi. Hafif ve şiddetli akut pankreatit grupları arasında mortalite, pankreatik nekroz ve girişimsel yaklaşım insidansını karşılaştırmak amacıyla Ki-kare testi kullanıldı.

Bulgular: Çalışma süresince toplamda 100 AP hastası tedavi edildi. 7'den yüksek Ranson skoru ve pankreatit nekroz varlığı yüksek mortalite ile anlamlı düzeyde ilişkiliydi (p< 0,05). SIRS, 7'den yüksek BT-şiddet endeksi skoru, inotrop desteği ve komplikasyonlar nekrozu olan hastalarda daha sık görülmüştü. Profilaktik antibiyoktikler mortaliteyi düşürmese de girişimsel yaklaşım oranını azalttı (p< 0,05). Sistemik enflamatuvar yanıt sendromu (SIRS), Ranson's skoru > 7, nekroz, inotropik destek ve komplikasyon varlığı cerrahi ve perkütan işlemler gibi girişimsel yaklaşımlarla daha yüksek oranda ilişkiliydi (p< 0,05).

Sonuç: Hint toplumunda SIRS, Ranson's skoru ve BT-şiddet endeksini AP prognostik belirteçleri olarak doğruladık. Bu öngördürücüler ek olarak kullanıldığı takdirde, şiddetli AP ile ilişkili mortaliteyi düşürmek için, erken izlem ve agresif tedaviye yönlendirmeyi sağlayabilir.

Anahtar Kelimeler: Akut pankreatit, prognostik skor, Ranson's skor, CTSI, SIRS

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