ORIGINAL ARTICLE

Utility of Ranson score, computed tomography severity index, and CRP criteria in risk stratification on the day of hospital admission in patients with acute pancreatitis: A cross-sectional analysis

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ABSTRACT

BACKGROUND: The early identification of severe acute pancreatitis (AP) remains a great challenge in clinical practice and novel predictors are needed to complement available scoring systems. This study aimed to investigate utility of Ranson score, and computed tomography severity index (CTSI) and C-reactive protein (CRP) criteria in determination of risk prognostic status in AP.

METHODS: A total of 104 patients with AP (median age: 71.5 (range, 21–102) years, (59.6% were males) were included in this cross-sectional study. Patients were divided into two groups according to risk prognostic status including good prognosis (n=67) and poor prognosis (n=37) groups, based on presence of at least one of the poor prognostic criteria including Ranson score ≥3, presence of pseudocyst and necrotizing fluid collection on ultrasonography or computed tomography imaging and CRP levels >15 mg/L. Data on patient demographics, etiology of AP, smoking, blood biochemistry and hemogram findings and inflammatory markers including CRP (mg/L), mean platelet volume (fL), neutrophil-lymphocyte ratio, and platelet-lymphocyte ratio were recorded.

RESULTS: Overall, 37 (35.6) patients with at least one these criteria comprised the poor prognosis group. Most of patients were considered to be in the poor prognosis group based on CTSI only (35.1%), CTSI + CRP (18.9%), and CTSI + Ranson (16.2%). Overall, 6 (5.8%) patients died, and all of them were in the poor prognosis group (p=0.002). Patients with poor versus good prognosis had significantly higher median (min-max) values for creatinine (1 [0.57–10.0] vs. 0.76 [0.5–8.4] mg/dL, p=0.004) and urea (48.0 [9.0–247.0] vs. 27.0 [10.0–111.0] mg/dL, p<0.001), and lower albumin values (3.5 [2.4–4.3] vs. 3.6 [2.7–4.6] g/L, p=0.021). Kappa values indicated presence of a moderate agreement between CTSI and CRP (kappa: 0.408), a fair agreement between CTSI and Ranson (kappa: 0.312), and a none to slight agreement between Ranson and CRP (kappa: 0.175). CTSI was able to discriminate all 6 patients (100.0%) with mortality, whereas Ranson and CRP each discriminated only 2 (33.3%) of 6 patients with mortality.

CONCLUSION: Our findings suggest a stronger individual prognostic value of CTSI alone, rather than CRP or Ranson score alone, in risk stratification of AP patients for severity of disease and related mortality risk on the day of admission, whereas emphasize the likelihood of using CRP or Ranson score complementary to CTSI to enable further identification of poor prognostic status.

Keywords: Acute pancreatitis; computed tomography severity index; C-reactive protein; inflammatory markers; peripheral blood; prognosis; ranson score; risk stratification.

INTRODUCTION

Acute pancreatitis (AP) is an inflammatory disease of highly variable clinical presentation and severity along with potential multi-organ involvement.[1–3] While AP is self-limited in 65–85% of cases not requiring specific treatment or resulting in sequelae, the remaining may suffer from severe attacks progressing to systemic inflammatory response syndrome (SIRS) with a high morbidity and mortality.[3–6] Hence, the early assessment of severity of disease through risk stratification,

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particularly on day of admission, is considered crucial to prevent pancreatic necrosis and organ failure in those assigned to higher risk categories through the interventions in this period of a window of opportunity.[3,6,7]

Several multi-factorial scoring system and imaging tools such as Ranson, acute physiology and chronic health evaluation (APACHE-II), SIRS, bedside index for severity in AP (BISAP), modified Marshall score, sequential organ failure assessment (SOFA) score, and computed tomography severity index (CTSI) as well as several biochemical markers such C-reactive protein (CRP) and procalcitonin have been defined in the early identification of severe AP.[6–11]

However, none of these current clinical scoring systems or biochemical markers are considered definitive tools with widespread applicability or consistent accuracy and all are associated with several limitations.[3,6,8–10] In this regard, the early identification of severe AP remains a great challenge in clinical practice and novel predictors are needed to complement available scoring systems.[7,12]

Apart from severity scores, there has been considerable interest in utility of certain laboratory parameters and rapid biomarkers in reliable prognosis prediction for AP.[7] Several direct or combined markers of systemic inflammation as readily available laboratory tests including white blood cell (WBC) and platelet counts, mean platelet volume (MPV), neutrophil-lymphocyte ratio (NLR), platelet-lymphocyte ratio (PLR), and CRP have also been proposed to show the inflammatory state and severity of disease in patients with AP.[7,13–16]

Nonetheless, the correlation of these laboratory parameters as well as the pancreatic enzymes, liver and kidney function tests with multi-factorial scoring systems and radiological severity has not been extensively explored in AP patients.[10,13,17,18]

Therefore, this study aimed to investigate utility of Ranson score, CTSI, and CRP levels in determination of risk prognostic status in patients with AP.

MATERIALS AND METHODS

Study Population

A total of 104 patients with AP (median age: 71.5 [range, 21–102] years, 59.6% were males) were included in this cross-sectional study. Although initially 156 patients were enrolled, due to exclusion of 52 patients with missing data on Ranson parameters and/or CRP, 104 patients were subjected to the final analysis. Patients were divided into two groups according to risk prognostic status including good prognosis (n=67) and poor prognosis (n=37) groups, based on presence of at least one of the poor prognostic criteria including Ranson score ≥3, presence of pseudocyst and necrotizing fluid collection on ultrasonography or CT imaging and CRP levels >15 mg/L (Table 1).

Table 1. Risk stratification

Risk stratification	n (%)
CTSI	
Balthazar scores 0–2 (A-C) plus	74 (71.2)
Pancreatic necrosis score 0-2	
Balthazar scores 3–4 (D-E) plus	30 (28.8)
Pancreatic necrosis score 4-6	
CRP	
≤15 mg/L	91 (87.5)
>15 mg/L	13 (12.5)
Ranson	
<3	89 (85.6)
≥3	15 (14.4)
POOR PROGNOSIS: Patients with at least one poor prognostic criteria (n=37)	
CTSI only	13 (35.1)
CRP only	2 (5.4)
Ranson only	5 (13.5)
CTSI+Ranson	6 (16.2)
CTSI+CRP	7 (18.9)
CTSI+CRP+Ranson	4 (10.8)
Total	37 (100.0)

CTSI: Computed tomography severity index; CRP: C-reactive protein.

Written informed consent was obtained from each subject following a detailed explanation of the objectives and protocol of the study which was conducted in accordance with the ethical principles stated in the "Declaration of Helsinki" and approved by the institutional ethics committee.

Assessments

Data on patient demographics (age and gender), etiology of AP, smoking status, blood biochemistry findings including glucose (g/dL), creatinine (mg/dL), urea (mg/dL), bilirubin (mg/dL), Aspartate aminotransferase (AST, IU/L), Alanine aminotransferase (ALT, IU/L), lactate dehydrogenase (LDH, U/L), amylase (U/mL), albumin (g/L), and calcium (Ca, mg/dL), hemogram findings including hemoglobin (g/dL), platelet (cells/mm^3), neutrophil (cells/mm^3), lymphocyte (cells/μL) counts, and inflammatory markers including CRP (mg/L), MPV (fL), NLR, and PLR were recorded at the time of hospital admission in each patient.

The diagnosis of AP is based on presence of 2 of the following three criteria: upper abdominal pain consistent with AP; increased serum lipase and/or amylase activity at least 3 times greater than the upper limit of normal; characteristic findings of AP on abdominal imaging.[19]

Ranson Criteria

Ranson criteria use 11 parameters to assess the severity of AP. The five parameters on admission are age (>55 years), WBC count (>16,000 cells/cmm, blood glucose ≥200 mg/dL (11 mmol/L), serum AST ≥250 IU/L, and serum LDH >350 IU/L. At 48 h, the remaining six parameters are: serum calcium <8.0 mg/dL (<2.0 mmol/L), hematocrit fall ≥10%, PaO_2 ≤60 mmHg, BUN increased by 5 mg/dL or more (1.8 mmol/L or more) despite intravenous (IV) fluid hydration, base deficit ≥4 mEq/L, and sequestration of fluids ≥6 L. Scores of "0–2", "3–4", "5–6" and "7–11" indicates mortality risk of 0–3%, 15%, 40%, and nearly 100%, respectively.[20] In this study, Ranson criteria assessment was based on hematocrit, BUN, Ca, arterial pO_2, and base deficit measurements on the day of hospital admission and scores ≥3 indicated poor prognosis.

CT Severity Index (CTSI)

The CTSI sums the Balthazar score (grading of pancreatitis: A-E) and grading the extent of pancreatic necrosis. Grading of pancreatitis (Balthazar score) is scored with 0 (A: normal pancreas), 1 (B: enlargement of pancreas), 2 (C: inflammatory changes in pancreas and peripancreatic fat), 3 (D: ill-defined single peripancreatic fluid collection), and 4 (E: two or more poorly defined peripancreatic fluid collections), while pancreatic necrosis is categorized as none (score 0), ≤30% (score 2), >30–50% (score 4) and >50% (score 6).[21,22]

Statistical Analysis

Statistical analysis was made using MedCalc® Statistical Software version 19.7.2 (MedCalc Software Ltd, Ostend, Belgium; https://www.medcalc.org; 2021). Chi-square test was used for analysis of categorical data. Mann–Whitney U test were used for analysis of the parametric variables. Agreement between Ranson, CRP and CTSI criteria were analyzed using kappa values with consideration of kappa values ≤0 as indicating no agreement, 0.01–0.20 as none to slight, 0.21–0.40 as fair, 0.41–0.60 as moderate, 0.61–0.80 as substantial, and 0.81–1.00 as almost perfect agreement. Data were expressed as "mean±standard deviation, median (min-max) and percent (%) where appropriate. P<0.05 was considered statistically significant.

RESULTS

Risk Prognostic Status

Ranson scores were ≥3 in 15(14.4%) patients and CTSI-radiological imaging revealed Balthazar scores of 3–4 (D-E) plus pancreatic necrosis scores of 4–6 in 30 (28.8%) patients, while CRP levels were >15 mg/L in 13 (12.5%) patients (Table 1).

Overall, 37 (35.6) patients with at least one these criteria comprised the poor prognosis group. Most of patients were considered to be in the poor prognosis group based on CTSI only (35.1%), CTSI + CRP (18.9%), CTSI + Ranson (16.2%), and Ranson only (13.5%) (Table 1).

Patient Demographics and Mortality According to Prognostic Groups

Patients in the poor prognosis group were significantly older than those in the good prognosis group (median age 74 vs. 64

Table 2. Demographic and clinical characteristics according to risk prognostic status

	Patients with acute pancreatitis			p-value
	Total (n=104)	Good prognosis (n=67)	Poor prognosis (n=37)	
Age (year), median (min-max)	71.5 (21–102)	64 (21–94)	74 (36–102)	0.033[1]
Gender, n (%)				
Male	62 (59.6)	37 (59.7)	25 (40.3)	0.308[2]
Female	42 (40.4)	30 (71.4)	12 (28.6)	
Etiology, n (%)				
Idiopathic	61 (58.7)	42 (68.9)	19 (31.1)	0.053[2]
Biliary	39 (37.5)	21 (53.8)	18 (46.2)	
Malignancy	4 (3.8)	4 (100.0)	0 (0.0)	
Active smoking, n (%)				
No	88 (84.6)	55 (62.5)	33 (37.5)	0.337[2]
Yes	16 (15.4)	12 (75.0)	4 (25.0)	
Survivorship status, n(%)				
Survived	98 (94.2)	67 (68.4)	31 (31.6)	0.002[3]
Died	6 (5.8)	0 (0.0)	6 (100.0)	

[1]Mann-Whitney U test, [2]χ^2 test, [3]Fisher Exact test.

Table 3. Blood parameters according to prognostic status

	Patients with pancreatitis						p-value
	Total (n=104)		Good prognosis (n=67)		Poor prognosis (n=37)		
	n	Median (min-max)	n	Median (min-max)	n	Median (min-max)	
Blood biochemistry							
Creatinine (mg/dL)	104	0.8 (0.5–10.0)	67	0.76 (0.5–8.40)	37	1.0 (0.57–10.0)	0.004
Urea (mg/dL)	104	34.0 (9.0–247.0)	67	27.0 (10.0–111.0)	37	48.0 (9.0–247.0)	<0.001
Bilirubin (mg/dL)	104	1.6 (0.1–14.9)	67	1.47 (0.1–14.9)	37	1.7 (0.4–8.9)	0.525
ALT (IU/L)	104	77.0 (6.0–1310)	67	72.0 (0.7–1310)	37	115.0 (6.0–851)	0.571
Amylase (U/mL)	104	729.0 (24.0–3292.0)	67	482 (24.0–3190)	37	749.0 (38.0–3292.0)	0.114
Albumin (g/L)	103	3.6 (2.4–4.6)	66	3.6 (2.7–4.6)	37	3.5 (2.4–4.3)	0.021
Hemogram							
Hemoglobin (g/dL)	104	12.7 (7.8–15.7)	67	12.8 (8.6–15.7)	37	11.9 (7.8–15.5)	0.093
Platelet (cells/mm^3)	104	204.0 (47.0–414.0)	67	206.0 (94.0–351.0)	37	202.0 (47.0–414.0)	0.669
Neutrophil (cells/mm^3)	103	2.70 (1.4–4.3)	66	2.75 (1.4–4.2)	37	2.70 (1.4–4.3)	0.836
Lymphocyte (cells/µL)	102	1.8 (1.0–2.9)	65	1.8 (1.0–2.9)	37	1.7 (1.0–2.8)	0.836
Inflammatory markers							
MPV (fL)	104	10.45 (5.67–13.4)	67	10.3 (6.86–12.6)	37	10.7 (5.67–13.4)	0.152
NLR	102	1.57 (0.88–3.15)	65	1.66 (0.90–3.15)	37	1.53 (0.88–3.07)	0.898
PLR	102	112.93 (27.65–351.0)	65	117.64 (54.83–351.0)	37	110.66 (27.65–276.0)	1.000

AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; LDH: Lactate dehydrogenase; CRP: C-reactive protein; MPV: Mean platelet volume; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; LOS: Length of hospital stay. Mann-Whitney U test.

years, p=0.033). No significant difference was noted between patients with good prognosis and those with poor prognosis in terms of gender, etiology of AP and the smoking status. Overall, 98 (94.2%) patients discharged from the hospital, while 6 (5.8%) patients died, and all of them were in the poor prognosis group (p=0.002) (Table 2).

Blood Parameters in Prognostic Groups

Patients with poor versus good prognosis had significantly higher median (min-max) values for creatinine (1 (0.57–10.0) vs. 0.76 (0.5–8.4) mg/dL, p=0.004) and urea.

(48.0 (9.0–247.0) vs. 27.0 (10.0–111.0) mg/dL, p<0.001), and lower albumin values (3.5 (2.4–4.3) vs. 3.6 (2.7–4.6) g/L, p=0.021) (Table 3).

ALT, amylase, bilirubin and hemoglobin levels, platelet, neutrophil and lymphocyte counts and MPV, NLR, and PLR values did not significantly differ between good prognosis and poor prognosis groups (Table 3).

Agreement between CTSI, Ranson, and CRP Criteria

Ranson and CRP were able to identify only 10 (33.3%) and 11 (36.7%) of 30 patients with poor CTSI criteria, respectively (p=0.001 and p<0.001). CTSI and CRP were able to identify 10 (66.7%) and 4 (26.7%) of 15 patients with poor Ranson criteria, respectively, (p=0.001 and p=0.073). Kappa values indicated presence of a moderate agreement between CTSI and CRP (kappa:0.408), a fair agreement between CTSI and Ranson (kappa: 0.312), and a none to slight agreement between Ranson and CRP (kappa: 0.175) (Table 4).

CTSI was able to discriminate all 6 patients (100.0%) with mortality, whereas Ranson and CRP each discriminated only 2 (33.3%) of 6 patients with mortality (Table 4).

DISCUSSION

Our findings revealed presence of at least one of the poor prognostic criteria (Ranson scores ≥3, Balthazar scores of 3–4 (D-E) plus pancreatic necrosis scores of 4–6 on CTSI-radiological imaging or CRP levels >15 mg/L) in 35.6% of patients presented with AP and mortality in 5.8% of them. CTSI (28.8%) compared to Ranson (14.4%) or CRP (12.5%) was able to discriminate more patients to be considered in the poor prognosis category and to more accurately identify the mortality risk.

The stronger association of CTSI in predicting SAP and mortality in our study support the consideration of CTSI to have a strong positive correlation with the development of complications and mortality in patients with AP.[18,22,23] In a study with 163 AP patients, pancreatic necrotic volume (PNV, cutoff

Table 4. Agreement between CTSI, Ranson score and CRP criteria

	Ranson			CRP		
	Good prognosis	**Poor prognosis**	**Total**	**Good prognosis**	**Poor prognosis**	**Total**
CTSI						
Good prognosis	69 (93.2)	5 (6.8)	74 (71.2)	72 (97.3)	2 (2.7)	74 (71.2)
Poor prognosis	20 (66.7)	10 (33.3)	30 (28.8)	19 (63.3)	11 (36.7)	30 (28.8)
Total	89 (85.6)	15 (14.4)	104 (100.0)	91 (87.5)	13 (12.5)	104 (100.0)
Kappa Value	0.312			0.408		
	CRP					
	Good prognosis	**Poor prognosis**	**Total**			
Ranson						
Good prognosis	80 (89.9)	9 (10.1)	89 (85.6)			
Poor prognosis	11 (73.3)	4 (26.7)	15 (14.4)			
Total	91 (87.5)	13 (12.5)	104 (100.0)			
Kappa Value	0.175					
	CTSI		**Ranson**		**CRP**	
	Good prognosis (n=74)	**Poor prognosis (n=30)**	**Good prognosis (n=89)**	**Poor prognosis (n=15)**	**Good prognosis (n=91)**	**Poor prognosis (n=13)**
Outcome						
Discharge (n=98)	74 (75.5)	24 (24.5)	85 (86.7)	13 (13.3)	87 (88.8)	11 (11.2)
Mortality (n=6)	0 (0.0)	6 (100.0)	4 (66.7)	2 (33.3)	4 (66.7)	2 (33.3)
p-value	<0.001		0.174		0.112	

CTSI: Computed tomography severity index; CRP: C-reactive protein. Chi square test.

value: 75 cc) in the CT was reported to show a linear correlation with hospital stay and a significant association with acute complications (i.e., infection, organ failure, need of treatment, or hospitalization at intensive care unit).[23] The authors also noted that PNV and thus necrosis volume was the best radiological biomarker correlated with AP complications.[23] In fact, identification of peripancreatic edema in endoscopic ultrasonography, a method allowing for the detailed visualization of the whole pancreas due to its high-resolution images,[6,24,25] is considered likely to be a novel imaging marker with favorable sensitivity (65.8%), specificity (75.5%), and accuracy (72.2%) for the early prediction of the severity of AP.[24]

Indeed, while CTSI alone was able to discriminate 35.1% of 37 patients with poor prognosis, the use of CRP (18.9%), Ranson (16.2%) or both (10.8%) complementary to CTSI enabled further identification of poor prognostic status in AP patients at the time of hospital admission. Hence, our findings seem to emphasize the significant prognostic value of CTSI alone in risk stratification of AP patients on initial admission, whereas further identification of poor prognosis with complementary use of Ranson or CRP which shows only fair-to-moderate agreement with CTSI.

In a study with 72 AP patients (31 patients had severe AP, 17 had pancreatic necrosis, and 9 [12.5%] died) area under curves (AUC) for Ranson, CTSI, and CRP were reported to be 0.85, 0.80, and 0.91, respectively, in predicting severe AP, to be 0.70, 0.75, and 0.90, respectively, for pancreatic necrosis, and to be 0.84, 0.57, and 0.75, respectively, for mortality.[9] The authors concluded the stronger role of CRP in early detection of severity and pancreatic necrosis whereas Ranson score in predicting AP related mortality.[9] Furthermore, in a study with 80 AP patients (19 had severe AP, and 9 (11.3%) died), AUC for Ranson score, CTSI, and CRP were reported to be 0.690, 0.619, and 0728, respectively, in predicting severe AP and to be 0.669, 0.615, and 0.799, respectively, for mortality.[26]

The moderate agreement between CTSI and CRP findings in our study seems to be in line with consideration of CRP as a good marker for prediction of complications and mortality in AP, particularly for predicting the pancreatic necrosis.[9,26–28]

Nonetheless, our findings support the use of CRP or Ranson as complementary methods to CTSI rather than alone in predicting severity of AP, supporting the previous studies

indicated that Ranson scores had lower sensitivity for complications, mortality, and the length of stay for AP than the Balthazar score.[29,30] This seems notable given that despite being a useful and readily available tool in clinical practice, CRP, when used alone, is considered a marker with a low specificity as a prognostic tool in AP.[31] Notably, in a retrospective chart review study with 119 AP patients, the CRP level and follow-up CRP titer were reported to be significantly correlate with the radiological grade, suggesting the utility of CRP with the radiological severity in estimation of the severe disease course.[18]

In the current study, in patients with poor prognostic status, serum creatinine and urea levels were also concomitantly higher and serum albumin levels were lower. The concomitant increase in serum levels of creatinine and urea in our poor prognosis group supports the consideration of acute kidney injury (AKI) as a serious and common complication of severe AP which increases the mortality risk,[17,32–34] while higher fluid sequestration in AP has been associated with more severe disease course and thus increased likelihood of organ failure.[17,35] Moreover, hypoalbuminemia is also considered a good clinical prediction index of consistent organ failure of AP.[36] and albumin levels ≤28.9 mg/L as a relatively accurate index to evaluate severe AP death risk with a similar accuracy rate to CRP.[37]

In addition, BISAP, which uses pleural effusion detected on imaging as one of the parameters in the prognostic scoring[38] was reported to be a valuable predictor for SAP[7] and to be better than Ranson in prediction of organ failure.[8] Moreover, Ranson score was reported to be positively correlated with CRP in AP patients,[13] while the disrupted control of microvascular pressure tone due to systemic inflammation is also considered likely to be an important factor leading to kidney injury.[17,39]

Among radiologic scoring systems including the Balthazar score, MTSI, EPIC, and renal rim grade, an EPIC score ≥6 was considered the most valuable for predicting both severity and mortality among all four of these radiologic scoring systems,[26,40] while NLR, as another inexpensive and widely available parameter, was suggested to have almost the same value as CRP for predicting severe AP and even markedly higher value to predict mortality than CRP value.[26] Our findings revealed no significant difference in blood cell counts, NLR and PLR as well as in amylase levels with respect to prognostic status. Likewise, past studies also reported no significant differences between patients with AP and healthy groups in terms of platelet numbers at the time of admission. [13,16,41] while the increase in MPV showing platelet activation has been described as an independent risk factor for AP.[41,42] suggesting that the severity of systemic inflammation is related to platelet volume.[13]

Certain limitations to this study should be considered. First, potential lack of generalizability is an important limitation due to single-center study design with relatively small sample size. Second, the cross-sectional design made it impossible to establish any cause-and-effect relationships.

Conclusion

Our findings suggest a stronger individual prognostic value of CTSI alone, rather than CRP or Ranson score alone, in risk stratification of AP patients for severity of disease and related mortality risk on the day of admission, whereas emphasize the likelihood of using CRP or Ranson score complementary to CTSI to enable further identification of poor prognostic status. Large population-based multicenter studies are needed to identify the utility of different multifactorial scoring systems in combination with biochemical markers for early assessment of the severity of AP in routine clinical practice.

Ethics Committee Approval: This study was approved by the Recep Tayyip Erdoğan University Non-interventional Clinical Research Ethics Committee (Date: 19.08.2021, Decision No: 2021/145).

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Conflict of Interest: None declared.

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ORİJİNAL ÇALIŞMA - *ÖZ*

Akut pankreatit hastalarında Ranson, bilgisayarlı tomografi şiddet indeksi ve CRP kriterlerinin yatış günü risk derecelendirmesinde kullanımı: Kesitsel bir çalışma

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AMAÇ: Şiddetli pankreatit, erken tanısı klinik pratikte zorluk teşkil etmeye devam eden bir hastalık olup, mevcut skorlama sistemlerini tamamlayıcı yeni öngördürücülere ihtiyaç vardır. Bu çalışmada, Ranson skoru, bilgisayarlı tomografi şiddet indeksi (BTŞİ) ve C-reaktif protein (CRP) kriterlerinin akut pankreatitte (AP) risk prognostik durum belirlenmesinde kullanımının incelenmesi amaçlandı.

GEREÇ VE YÖNTEM: Bu kesitsel çalışmaya AP tanılı 104 hasta (ortanca yaş: 71.5 (21–102) yıl, %59.6'sı erkek) dahil edildi. Hastalar Ranson skoru ≥3, ultrason veya CT görüntülemede psödokist ve nekrotize sıvı birikimi ve CRP düzeyleri >15 mg/L kriterlerinden en az birini karşılayanlar temelinde, risk prognostik durumlarına göre, iyi prognoz (n=67) ve kötü prognoz (n=37) olmak üzere iki gruba ayrıldı. Hastaların demografik özellikleri, sigara içme durumu, AP etiyolojisi, kan biyokimya ve hemogram bulguları ve CRP (mg/L), MPV (fL), nötrofil-lenfosit oranı (NLR), platelet-lenfosit oranı (PLR) olmak üzere enflamatuvar belirteçleri kaydedildi.

BULGULAR: Toplamda, 37 (%35.6) hasta kötü prognoz lehine en az bir kriter mevcuttu. Kötü prognoz grubundaki hastalar, çoğunlukla sadece BTŞİ (%35.1), BTŞİ+CRP (%18.9), BTŞİ+Ranson (%16.2) kriterleri bazında saptandı. Tümü kötü prognoz grubunda sınıflanmış 6 (%5.8) hastada ölüm gerçekleşti (p=0.002). Kötü prognoz gurunda iyi prognoz grubuna göre ortanca (min-mak) kreatinine (1 (0.57–10.0) vs. 0.76 (0.5–8.4) mg/dL, p=0.004) ve üre (48.0 (9.0–247.0) vs. 27.0 (10.0–111.0) mg/dL, p<0.001) değerleri anlamlı olarak daha yüksel, albümin değerleri (3.5 (2.4–4.3) vs. 3.6 (2.7–4.6) g/L, p=0.021) ise daha düşüktü. Kappa değerlerine göre, BTŞİ ile CRP arasında orta dereceli (kappa: 0.408), BTŞİ ile Ranson arasında ise ekseriyetle (kappa: 0.312) uyuşma olduğu saptandı. BTŞİ, ölümle sonuçlanan altı olgunun tümünü, Ranson ve CRP ise sadece ikişer olguyu öngörebildi.

TARTIŞMA: Bulgularımız, AP hastalarında risk derecelendirmesinde, BTŞİ'nin tek başına kullanımının, CRP ve Ranson skorunun tek başlarına kullanımına göre, hastalık şiddeti ve mortalite riski açısından daha güçlü bir prognostik değeri olduğunu düşündürmekte ve CRP veya Ranson skorunun BTŞİ'ye komplementer olarak kullanımının AP hastalarında kötü prognostik durumun daha iyi belirlenmesinde katkısı olacağına işaret etmektedir.

Anahtar sözcükler: Akut pankreatit; bilgisayarlı tomografi şiddet indeksi; CRP; enflamatuvar belirteçler; periferik kan; prognoz; Ranson skoru; risk derecelendirme.

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