ORIGINAL ARTICLE

Diagnostic utility of hematological indices in predicting adverse outcomes and severity of acute pancreatitis based on BISAP and modified Glasgow score

Gökhan Akdur, M.D.,[1] Okan Bardakcı, M.D.,[1] Murat Das, M.D.,[1] Okhan Akdur, M.D.,[1] Yavuz Beyazit, M.D.[2]

[1]Department of Emergency Medicine, Çanakkale Onsekiz Mart University Faculty of Medicine, Çanakkale-*Turkey*
[2]Department of Internal Medicine, Çanakkale Onsekiz Mart University Faculty of Medicine, Çanakkale-*Turkey*

ABSTRACT

BACKGROUND: The neutrophil-lymphocyte ratio (NLR), platelet-lymphocyte-ratio (PLR), and red blood cell distribution width (RDW) are simple indicators of inflammatory status previously established as a severity indicator in distinct disease states. This study aimed to determine the impact of these simple hematologic indices with conventional inflammation markers such as C-reactive protein (CRP) and white blood cells in acute pancreatitis (AP) patients and their relationship with AP risk stratification scores including Bedside Index for Severity of Acute Pancreatitis (BISAP) and modified Glaskow Prognostic score (mGPS) scores.

METHODS: This retrospective study was performed in the emergency department of Canakkale Onsekiz Mart University. A total of 171 patients (male/female: 68 [39.8%]/103 [60.3%]) with AP and 59 age and gender matched healthy subjects (male/female: 23 [39%]/36[61%]) as controls were enrolled in the present study. The patients were grouped according to severity and adverse outcomes according to BISAP and mGPS and a comparative analysis was performed to compare the NLR, PLR, and RDW between groups.

RESULTS: The mean NLR values of AP patients and control group were 9.62±6.34 and 2.04±1.08, respectively ($p<0.001$), while the mean PLR values of AP patients and control group were 221.83±122.43 and 83.30±38.89, respectively ($p<0.001$). Except from RDW, all the other hematologic indices were found to be elevated ($p<0.05$ for WBC; NLR, PLR, and CRP) on both mild and severe disease at disease onset. NLR and PLR showed significant predictive ability for estimating serious complications associated with AP.

CONCLUSION: The present study showed that NLR and PLR is increased in AP. Moreover, peripheral blood NLR and PLR values can predict disease severity and adverse outcomes associated with AP and can be used as an adjunctive marker for estimating disease severity.

Keywords: Acute pancreatitis; Bedside Index for Severity of Acute Pancreatitis; Modified Glaskow; mean platelet volume; neutrophil-lymphocyte ratio; platelet-lymphocyte-ratio; red blood cell distribution width.

INTRODUCTION

Acute pancreatitis (AP) is a serious and reversible inflammatory process of the pancreatic tissue characterized by abdominal/epigastric pain and elevated levels of pancreatic enzymes.[1] It is a common cause for admission to the emergency department (ED) and one of the most common emergent conditions resulting in admission to inpatient ward or intensive care units (ICU).[2] Although, the majority of the cases are mild with a favorable outcome, approximately one-fifth of the patients develops a severe form usually associated with complications such as, pancreatic abscess, necrosis, organ failure, and potentially death.[3] It is therefore crucial to perform early interventions to reduce adverse outcomes, shorten the length of hospitalization and to prevent the progression to death. In this context, prompt diagnosis and accurate staging

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Address for correspondence: Okan Bardakcı, M.D.
Çanakkale Onsekiz Mart Üniversitesi Tıp Fakültesi, Acil Tıp Anabilim Dalı, Çanakkale, Turkey
Tel: +90 286 - 263 59 50 / 1147 E-mail: drokanbardakci@gmail.com

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of AP severity is important due to the risk of rapid deterioration of the patients' clinical condition.

Due to the varied clinical presentations of AP, a series of risk stratification systems are currently accepted to identify the severity of AP including revised Atlanta classification (RAC), Ranson, Acute Physiologic Assessment and Chronic Health Evaluation II (APACHE II) score, Bedside Index for Severity in Acute Pancreatitis (BISAP), SOFA and modified Glaskow Prognostic score (mGPS) severity criteria.[4–6] Unfortunately, these systems are sometimes criticized for being unnecessarily complex, overly restrictive, and difficult to perform to patients outside of ICU because using too many parameters.[7,8] Moreover, also some of these parameters are not suitable for the evaluation of AP patients at the time of ED admission or shortly thereafter. Although several serum biochemical paramaters including erthyrocyte sedimentation rate (ESR), C-reactive protein (CRP), interleukin 6, procalcitonin, D-dimer, soluble fms-like tyrosinekinase-1, and tumor necrosis factor alpha (TNF-α) have been demonstrated to predict the severity of AP, it must be noted that some of these parameters are expensive, have low sensitivity and specificity, and cannot sufficiently predict the prognosis of AP in clinical settings.[9–12] Therefore, apart from these biochemical markers, new and simple severity predictors to complement the present scoring systems are strictly required to improve clinical practice and health outcomes.

During recent years, several easy to calculate hematologic indices such as neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), red cell distribution width (RDW) and mean platelet volume proposed to have valuable correlations with distinct inflammatory, cardiovascular and neoplastic conditions.[13–16] Therefore, it is not surprising to find out that there are considerable interest in the development of rapid hematologic biomarkers to predict the prognosis and severity of AP. In this context, NLR, PLR, and RDW have been shown to represent simple, inexpensive and reliable tests with a promising value to predict disease severity in AP.[17] The mechanism responsible from the alterations observed in these complete blood cell (CBC) count differentials is the damage and destruction of pancreatic cells during acute period of pancreatitis. Destruction of the pancreatic tissue leads to vascular endothelium dysfunction and increased vascular permeability with leukocyte migration to tissues, and activation of coagulation systems.[18]

Although, there are some literature suggesting that NLR and PLR could be used to predict adverse outcomes related to AP, there is only scarce data that compares the combination of these hematologic parameters with CRP and scoring systems such as BISAP and mGPS that is commonly used to predict prognosis and severity of AP. Hence, in this study, we aimed to explore the impact of NLR, PLR, and RDW with conventional inflammation markers such as CRP and white blood cells in AP patients and their relationship with AP risk stratification scores.

MATERIALS AND METHODS

Patient Selection

Patients with AP that were admitted to Canakkale Onsekiz Mart University (COMU) Hospital were retrospectively enrolled in the present study after approval from the Institutional Review Board for Human Research of COMU. A total of 171 patients presenting with AP to the Department of Emergency Medicine of COMU over a 2 years period (between December 2017 and December 2019) were included. AP diagnosis was made according to the revised 2012 Atlanta Classification.[19] The following data were extracted from COMU Hospital Information and Management System including age, gender, past medical conditions, radiologic imaging, laboratory data at onset and remission. Exclusion from the study included patients with cardiac failure, chronic disease conditions including hematologic, kidney and liver disease, diabetes mellitus, use of immunosupressive drugs, tumoral conditions, incomplete records or those with a doubtful diagnosis. Remission was considered at both clinical and biochemical levels briefly after symptom relief, starting with oral nutrition and pancreatic enzymes returned to normal.

About 59 healthy subjects (male/female: 23 [39%]/36 [61%]) without any history of acute/chronic inflammatory disorders with no drug use history were enrolled to the present study as controls.

Data Collection

Blood samples for hematological and biochemical data were obtained on onset and remission. CBC analysis was conducted using a Beckman Coulter (High Wycombe, UK) Gen-S automated analyzer. The WBC differentials were recorded for each patient. The NLR was defined as the absolute neutrophil count to absolute lymphocyte count and the PLR was defined as absolute platelet count to absolute lymphocyte count. Routine biochemistry parameters including blood urea nitrogen (BUN), creatinine, glucose, aspartate amino transferase, and alanine amino transferase were also obtained for each patient. Hospital length of stay was defined as day of hospital admission through day of discharge or in-hospital death.

Severity degree of patients was defined according to the BISAP and mGPS which can easily be applied in clinical settings. BISAP score was calculated based on the data (age >60 years, BUN >25 mg/dl, systemic inflammatory response syndrome, impaired mental status, and pleural effusion) obtained within 24 h of presentation. According to RAC the patients with BISAP ≥3 and at least one persistent organ failure were classified as severe AP. mGPS measured within 48 h after admission. Eight variables of mGPS were analyzed and these patients were graded mild AP (if the score <3) or severe AP (if the score ≥3).[5]

Statistical Analysis

Results were analyzed using SPSS (Statistical Package for the Social Sciences) 18.0 for Windows (IBM Corporation, Armonk, NY, USA). For the continuous data, test of the normality was tested by Kolmogorov—Smirnov test. For normally distributed data, mean±standard deviations are given, while in the case of non-normally distributed data median and range are given. All normally-distributed data were analyzed using Student's unpaired t-test. A $p<0.050$ is considered significant. Receiver operating characteristic (ROC) curve analysis was used to identify optimal cut-off values (mGPS ≥3 and BISAP ≥3) for PLR and NLR with other markers of inflammation to recognize maximum sensitivity and specificity for AP severity.

RESULTS

In this study, a total of 171 subjects and 59 healthy controls were selected according to the inclusion and exclusion criteria. The mean age of the AP patients and healthy controls was 68.02±15.99 years and 65.32±13.02, respectively. Of the 59 healthy subjects, 23 (39.0%) were men and 36 (61.0%) were women, while 171 AP patients 68 (39.8%) were men and 103 (60.3%) were women. There was no difference among patients in respect to age ($p=0.594$) or gender ($p=0.916$). Computerized tomography (CT) findings revealed a pancreatic necrosis rate of 5.8 %, pancreatic fluid collection rate of 4.7%, and abscess rate of 2.9%. The demographic, clinical and laboratory measurements of our patient population and the control group are summarized in Table 1.

The mean NLR values of AP patients and control group were 9.62±6.34 and 2.04±1.08, respectively ($p<0.001$), while the mean PLR values of AP patients and control group were 221.83±122.43 and 83.30±38.89, respectively ($p<0.001$). No significant differences were observed in respect to RDW levels between patients and controls ($p>0.05$). Inflammatory markers, such as WBC and CRP were found to be significantly elevated in AP compared to control patients.

After measuring the severity of the disease based on mGPS, 39 patients (22.8%) were classified as severe AP and 132 patients (77.2%) were classified as mild. According to the sever-

Table 1. Demographic and baseline characteristics of patients and healthy controls

	Study Group	Control Group	p
Age (year)	68.02±15.99	65.32±13.02	0.594
Male/female, n (%)	68 (39.8)/103 (60.3)	23 (39.0)/36 (61.0)	0.916
Initial laboratory*			
Hemoglobin (g/dl)	12.69±1.97	12.16±1.71	0.067
White blood cells (/mm^{3}x10^3)	12.40±5.09	7.42±1.65	<0.001
Platelet (/mm^{3}x10^3)	244.20±93.07	172.27±46.60	<0.001
Neutrophil-to-lymphocyte ratio	9.62±6.34	2.04±1.08	<0.001
Platelet-to-lymphocyte ratio	221.83±122.43	83.30±38.89	<0.001
Red cell distribution width (%)	14.78±1.68	14.03±1.24	0.055
C-reactive protein	7.62±7.85	0.64±0.28	<0.001
Glucose	147.99±60.25	114.74±37.38	<0.001
Urea	37.46±19.35	38.08±18.60	0.830
Creatinine	0.96±0.70	0.95±0.39	0.916
Alanine amino transferase	144.80±162.27	13.40±6.02	<0.001
Aspartate amino transferase	176.92±196.09	17.25±5.91	<0.001
Computerized tomography findings, n (%)			
Necrosis	10 (5.8)	–	
Pleural effusion	16 (9.4)	–	
Pancreatic fluid collection	8 (4.7)	–	
Pancreatic abscess	5 (2.9)	–	
≥2 complications	4 (2.3)	–	
Median Length of stay (days)	5 (4–8)		
Bedside Index for Severity in Acute Pancreatitis ≥3, n (%)	20 (11.7)		
Modified Glaskow Prognostic score ≥3, n (%)	39 (22.8)		

Table 2. Comparison of NLR and PLR with other inflammation markers at onset and remission of the disease according to disease severity based on mGPS

	Mild pancreatitis (n=132)			Severe pancreatitis (n=39)		
	Onset	Remission	p	Onset	Remission	p
White blood cells	11.10±4.12	6.98±2.41	<0.001	16.80±5.64	8.57±4.15	<0.001
Neutrophil-to-lymphocyte ratio	10.47±11.07	3.42±2.82	<0.001	17.43±12.95	4.95±4.21	<0.001
Platelet-to-lymphocyte ratio	247.3±202.1	152.0±74.8	<0.001	291.4±142.2	207.2±102.6	0.004
Red cell distribution width	14.66±1.37	14.59±1.50	0.670	15.17±2.45	15.36±2.11	0.724
C-reactive protein	6.29±6.86	4.72±4.70	0.032	12.14±9.22	7.15±7.01	0.009

NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; mGPS: modified Glaskow Prognostic score.

ity of the disease that is based on mGPS, comparison of NLR, PLR, and RDW with other markers of inflammation at onset and remission of the disease is shown in Table 2. Figure 1 shows NLR and PLR levels of AP patients and controls according to severity of the disease based on mGPS. Except from RDW, all the other hematologic indices were found to be elevated (p<0.05 for WBC; NLR, PLR and CRP) on both mild and severe disease at onset.

The ROC analyses of NLR and PLR were depicted in Table 3. According to these analyses, the AUC of NLR in the prediction of severe AP patients based on BISAP was found to be 0.574, and it was 0.571 for PLR. Based on the ROC curves, the best cut-off value for NLR was 7.61 with a sensitivity of 70.0% and a specificity of 50.1%, and for PLR it was 168.05, with a sensitivity of 75.0% and a specificity of 42.4% (Fig. 2a). The AUC, sensitivity and specificity of NLR and PLR in the prediction of severe AP patients based on mGPS was also demonstrated in Table 3. Same ROC curve analysis for NLR, PLR and for other inflammation markers was also shown on Figure 2b.

NLR and PLR showed significant predictive ability for estimating serious complications associated with AP (p<0.001 for NLR, p=0.040 for PLR). Comparison of NLR, PLR with other markes of inflammation according to complications are presented in Table 4.

DISCUSSION

In this study, we compared the predictive value of NLR, PLR, RDW and conventional inflammation based predictive markers with the two well known risk stratification systems namely BISAP and mGPS score in both onset and remission of AP. As a result, elevated levels of peripheral blood NLR and PLR was found to give high sensitivity, specificity and predictive values in predicting severe AP and were well correlated with BISAP and mGPS score. The AUC values of NLR, PLR and RDW to predict severe pancreatitis according to BISAP were 0.574 (95% CI 0.451–0.696), 0.521 (95% CI 0.400–0.642) and 0.722 (95% CI 0.610–0.834), respectively. According to mGPS, the AUC values of NLR, PLR and RDW to predict severe pancreatitis were 0.749 (95% CI 0.671–0.827), 0.667 (95% CI 0.580–0.754) and 0.540 (95% CI 0.431–0.649), respectively.

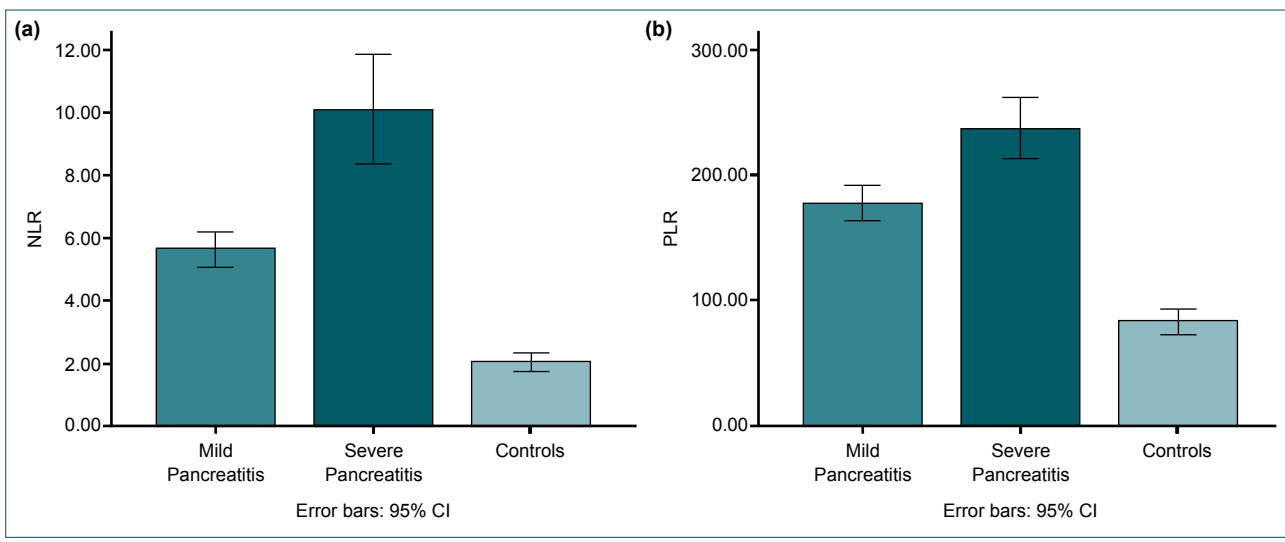

Figure 1. Bar plots and corresponding error bars demonstrating **(a)** NLR and **(b)** PLR levels of mild and severe AP patients in comparison to healthy controls.

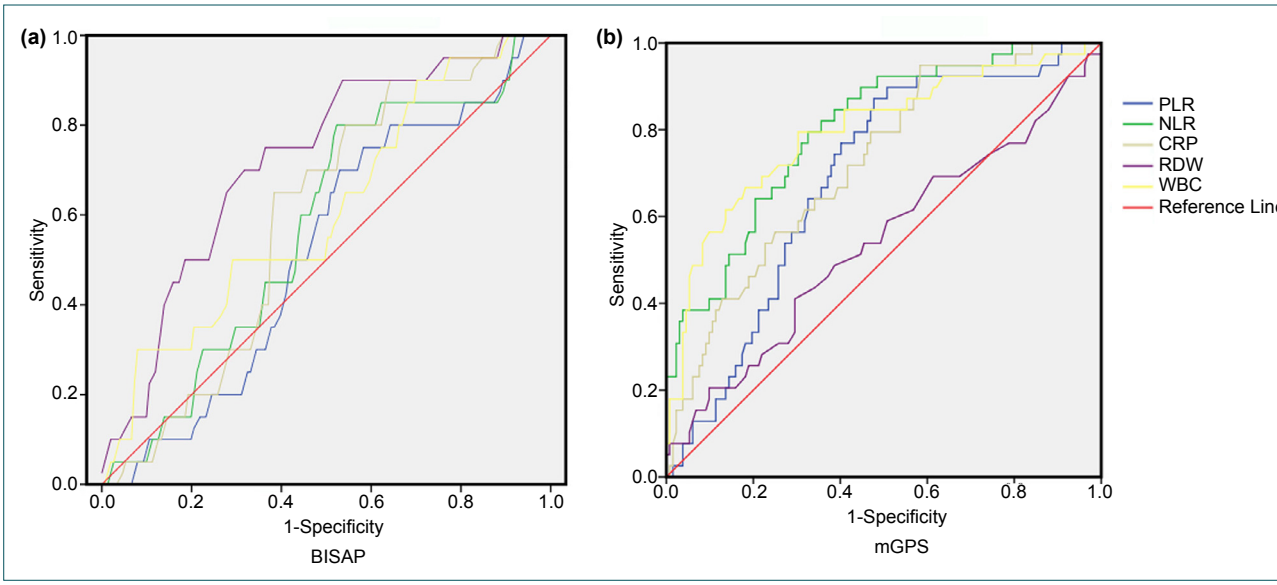

Figure 2. Receiver operating characteristic curve analysis for neutrophil-lymphocyte ratio and platelet-lymphocyte-ratio with other markers of inflammation to predict severity of the disease according to **(a)** bedside ındex for severity of acute pancreatitis and **(b)** modified Glaskow Prognostic score.

Moreover, NLR and PLR showed significant predictive ability for estimating serious complications associated with AP.

AP is a heterogeneous clinical condition characterized by inappropriate activation of pancreatic enzymes that leads to inflammatory cell infiltration of neutrophils and macrophages, and in some cases necrosis of the pancreatic tissue.[18] Despite improvements in diagnosis and management, AP is still the largest contributor to aggregate health related costs and the fifth leading reason of in-hospital mortality.[20] At present, early prediction of disease severity and outcome of acute severe pancreatitis achieved by combined use of clinical data, radiologic imaging, and biochemical analysis.[21] However, there are still no accurate and objective methods for diagnosing the disease in early stages and determine severe cases. Therefore, there is a need for an effective prognostic index that can facilitate therapeutic decision making and evaluation of AP in clinical settings.

The Ranson criteria, APACHE II score, BISAP score, and mGPS scores, are the most widely used risk assessment tools for AP in clinical settings.[22] Although, Ranson score and

Table 3. ROC analyses of NLR, PLR and other inflammation markers to predict severe acute pancreatitis based on BISAP and IMRIE scores

	Cut-off	AUC (95% CI)	Sensitivity (%)	Specificity (%)	NPV (%)	PPV (%)	Accuracy (%)
mGPS ≥3							
WBC	11.50	0.796 (0.710–0.882)	84.62	59.09	92.86	37.93	64.91
NLR	8.02	0.749 (0.671–0.827)	84.67	58.33	92.77	37.5	66.33
PLR	163.40	0.667 (0.580–0.754)	35.23	90.36	56.82	79.49	61.99
RDW	14.45	0.540 (0.431–0.649)	45.45	46.15	20.00	74.07	45.61
CRP	8.30	0.722 (0.637–0.808)	56.41	71.21	84.68	36.67	67.84
BISAP ≥3							
WBC	14.25	0.906 (0.479–0.739)	50.00	70.86	91.45	18.52	68.42
NLR	7.61	0.574 (0.451–0.696)	70.00	50.99	92.77	15.91	53.22
PLR	168.05	0.521 (0.400–0.642)	75.00	42.38	92.75	14.71	46.20
RDW	14.25	0.722 (0.610–0.834)	80.00	50.99	95.06	17.78	54.39
CRP	4.6	0.595 (0.486–0.705)	70.00	54.97	93.26	17.07	56.73

WBC: White blood cells; Plt: Platelet; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; RDW: Red cell distribution width; CRP: C-reactive protein; ALT: Alanine amino transferase; AST: Aspartate amino transferase; CT: Computerized tomography; BISAP: Bedside Index for Severity in Acute Pancreatitis; mGPS: modified Glaskow Prognostic score; CI: Confidence interval; ROC: Receiver operating characteristic.

Table 4. Comparison of study variables according to pancreatitis complications

	Complication (-)	Complication (+)	p
WBC	11.52±4.12	15.81±6.67	0.001
NLR	10.31±9.92	18.84±15.89	<0.001
PLR	244.3±192.4	307.68±177.42	0.040
RDW	14.60±1.35	15.48±2.50	0.006
CRP	6.26±6.90	12.93±9.09	<0.001

WBC: White blood cells; Plt: Platelet; NLR: Neutrophil-to-lymphocyte ratio; PLR: Platelet-to-lymphocyte ratio; RDW: Red cell distribution width; CRP: C-reactive protein.

APACHE II scores typically necessitates 48 hours or longer for an accurate calculation, mGPS score and BISAP can be measured in a short amount of time. In addition, previous studies reported that BISAP and mGPS scores were as good as APACHE II and Ranson scores in predicting severity and death of AP and can be measured more easily in emergency room.[23,24] For this reason, we used BISAP and mGPS scores as a measure of disease severity in AP patients.

This study demonstrated that PLR values are elevated in severe AP and can be used effectively to predict adverse outcomes. Another finding of this study is the determination of elevated PLR levels in the active phases of AP compared to clinical remission. PLR is a novel inflammatory marker that has been proposed to be a predictor of distinct disease states including inflammatory and thrombotic conditions.[25] The association between PLR and the disease severity in AP patients is a novel topic of interest and successfully explored in a recent article in which BISAP was one of the scoring systems used to define the severity of AP.[26] Authors demonstrated that NLR, PLR and RDW levels of the severe AP group were significantly increased compared to the mild AP group on admission. Similarly Kaplan et al.[17] demonstrated that PLR- had the highest AUC value in terms of predicting AP prognosis and had a similar diagnostic discrimination with other scoring systems including Ranson, RAC and BISAP. Contrary to these reports İlhan et al.[27] reported no significant association between PLR and AP severity in a patient cohort who developed AP in ongoing pregnancy.

Various studies in the last 10 years have confirmed that simple hematologic indices including NLR, PLR, and RDW might be usable for prognostic purposes in many diseases including malignant conditions, appendicitis, acute coronary syndrome, ulcerative colitis, and major vascular surgeries.[13–16,28] In this context, NLR is a simple and inexpensive index of systemic inflammatory burden and have been shown to have prognostic impact in estimating the severity of AP.[17] The basic explanation for the mechanism behind the association between NLR and negative outcomes is primarily based on neutrophil–epithelial cell interactions during ongoing inflammation in AP. As the inflammation proceeds activated neutrophils discharges several specific granule proteins, which are responsible for endothelial cell activation and changes in vascular permeability resulting in pancreatic tissue injury.[29,30] Based on the contributions of increased neutrophils to the pathology of severe AP with a relative decrease in lymphocytes forms the basis of the conclusions in various studies which reveals a positive association between NLR and adverse events.[31] Similarly, the results of the present study showed a significant association between NLR and disease severity as reflected by BISAP and mGPS score. Furthermore, based on our results, we suggest that a standardized cut-off point for NLR in predicting severity is of great importance. Due to the need to ensure an ideal treatment strategy as quickly as possible, increased NLR levels in conjunction with PLR can provide valuable insight to the clinician for estimating disease severity.

RDW is a quantitative parameter that measures variation in red blood cell size or red blood cell volume reflecting greater heterogeneity in cell sizes.[32] The increased levels may reflect an underlying inflammatory state such as cardiovascular disease, appendicitis, inflammatory bowel disease, obstructive jaundice and pneumonia.[33–36] Although, the exact cause of RDW alterations in inflammatory conditions is still a matter of debate, direct effects of inflammatory cytokines and alterations in iron metabolism in conjunction with inflammatory activity may diminish nitric oxide production in endothelial cells and may be responsible in the change of RDW.[37] In this study, we didn't observed any correlation between AP severity and RDW. Thus, we did not observe any association between RDW and adverse outcomes in AP patient. Contrary to our finding, Yao et al.[38] investigated whether RDW has a causal effect in leading to severe disease or mortality in patients with AP. RDW values was found to be significantly elevated in severe AP patients and with a cut-off level of 14.2 sensitivity and specificity of RDW to predict mortality was found to be 75.0% and 89.8%, respectively.

This study has several limitations that need to be addressed. First, it is a retrospective single center study with a relatively limited number of patients which makes a definitive comparison amongst the scoring systems difficult. Second, in spite of RAC, the definition of severe AP was based on BISAP and Imrie score. However, it must be noted that RAC does also have shortcomings such as underestimation of the effect of infected necrosis and extrapancreatic infections on the outcome of AP. Third, this study was undertaken in a tertiary referral center that might be resulted in disproportional inclusion of AP patients in more sever condition and tendency to progress adverse outcomes. Such selection bias might have overestimated the predictive value of elevated NLR or PLR. Moreover, finally, it might be noteworthy if we compare NLR and PLR with other inflammatory markers such as ESR, IL-6, IL-8, procalcitonin and TNF-α.

Conclusion

We explored severity stratification and adverse outcomes in AP patients and measured the predictive values of NLR, PLR, RDW and other conventional inflammatory markers in patients with AP admitted to ED at onset of the disease. Our results demonstrated that NLR and PLR can be useful tools in predicting which patients are more likely to develop severe disease at onset of their illness. Furthermore, evaluation NLR and PLR combination in AP patients has obvious advantages over other risk stratification scores such as being simple, easy-to-apply, and being highly sensitive for providing information related to severity and adverse outcomes without necessitating 48 h of assessment time.

Ethics Committee Approval: This study was approved by the Canakkale Onsekiz Mart University Ethics Committee (Date: 06.07.2020, Decision No: 2020-09).

Peer-review: Internally peer-reviewed.

Authorship Contributions: Concept: G.A.; Design: G.A.; Supervision: G.A., O.B., M.D., Y.B.; Resource: G.A., O.B., M.D., Y.B.; Data: G.A., O.B., M.D., Y.B.; Analysis: O.B., M.D.; Literature search: O.B., M.D.; Writing: Y.B.; Critical revision: O.A., Y.B.

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ORİJİNAL ÇALIŞMA - *ÖZ*

BISAP ve Modifiye Glaskow Skoru'na göre akut pankreatitde hastalık ciddiyetini ve komplikasyonlarını öngörmede hematolojik parametrelerin rolü

Dr. Gökhan Akdur,[1] Dr. Okan Bardakcı,[1] Dr. Murat Das,[1] Dr. Okhan Akdur,[1] Dr. Yavuz Beyazit[2]

[1]Çanakkale Onsekiz Mart Üniversitesi Tıp Fakültesi, Acil Tıp Anabilim Dalı, Çanakkale
[2]Çanakkale Onsekiz Mart Üniversitesi Tıp Fakültesi, Dahiliye Anabilim Dalı, Çanakkale

AMAÇ: Nötrofil-lenfosit oranı (NLR), trombosit lenfosit oranı (PLR) ve kırmızı kan hücresi dağılım genişliği (RDW), daha önce farklı hastalık durumlarında bir şiddet göstergesi olarak tanımlanmış iltihap durumunun basit göstergeleridir. Bu çalışma, bu basit hematolojik indekslerin akut pankreatit (AP) hastalarında CRP ve beyaz kan hücreleri gibi geleneksel enflamasyon belirteçleri ve bunların, Akut Pankreatit Şiddeti İçin İndeksi (BISAP) ve Modifiye Glaskow Prognostik (mGPS) skorları ile ilişkisini değerlendirmektedir.

GEREÇ VE YÖNTEM: Bu geriye dönük çalışma Çanakkale Onsekiz Mart Üniversitesi Acil Servisi'nde yapıldı. Toplam 171 hasta (erkek/kadın: 68 [%39.8) / 103 [%60.3)] AP'li ve 59 yaş ve cinsiyet uyumlu sağlıklı (erkek/kadın: 23 [%39] / 36 [%61]) kontroller bu çalışmaya dahil edildi. Hastalar BISAP ve mGPS'ye göre ciddiyet ve istenmeyen sonuçlara göre gruplandırıldı ve gruplar arasında NLR, PLR ve RDW değerlerini karşılaştırmak için analiz edildi.

BULGULAR: Akut pankreatit hastalarının ve kontrol grubunun ortalama NLR değerleri sırasıyla 9.62±6.34 ve 2.04±1.08 ($p<0.001$) iken, AP hastalarının ve kontrol grubunun ortalama PLR değerleri sırasıyla 221.83±122.43 ve 83.30±38.89 idi ($p<0.001$). RDW dışında, diğer tüm hematolojik indekslerin hastalık başlangıcında, hem hafif hem de şiddetli hastalıkta yükseldiği (WBC için $p<0.05$; NLR, PLR ve CRP) tespit edildi. NLR ve PLR, AP ile ilişkili ciddi komplikasyonları tahmin etmek için önemli olduğu görüldü.

TARTIŞMA: Bu çalışma AP'de NLR ve PLR'nin arttığını göstermiştir. Ayrıca kan NLR ve PLR değerleri, AP ile bağlantılı hastalık şiddetini ve olumsuz sonuçları tahmin edebilir ve hastalık ciddiyetini tahmin etmek için yardımcı bir belirteç olarak kullanılabilir.

Anahtar sözcükler: Akut pankreatit; BISAP; modifiye Glaskow; MPV; NLR; PLR; RDW.

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