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Original Article

Can mean platelet volume (MPV) and platelet/lymphocyte ratio (PLR) be an early predictor for severe acute pancreatitis?

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Abstract

Aim: Investigating serum mean platelet volume (MPV) and platelet/lymphocyte ratio (PLR) levels in acute pancreatitis (AP) patients, and evaluating whether MPV and PLR estimate AP disease severity efficiently at early phase.

Material and Methods: One hundred and forty patients diagnosed with AP and 49 healthy controls (HC) have been included in the study. Ranson criteria, revised Atlanta criteria and Balthazar scores of AP patients have been found from hospital registration systems, and mild-severe AP patients have been separated. MPV and PLR levels at admission were checked for the patients.

Results: While no difference was determined between AP group and HC group with regard to MPV levels (p:0.998), PLR levels was determined to be higher in AP group than HC group (p<0.001). No difference was determined between mild-severe AP patients with regard to MPV according to Ranson criteria, revised Atlanta criteria and Balthazar scoring (p:0.355; p:0.276; p:0.634, respectively). PLR was determined to be higher in severe AP group according to revised Atlanta criteria (p:0.023). However, no relation was determined between PLR and Ranson and Balthazar scoring (p:0.311, p:0.415, respectively). Upon grouping AP patients as biliary and non-biliary according to their etiology, MPV was determined to be lower in non-biliary AP patients (p:0.034). There was no difference between groups with regard to PLR (p:0.0772).

Conclusion: MPV is not suitable for differentiating mild-severe AP patients at early period. PLR may be used as a supporting test in estimating severe AP patients at admission.

Keywords: Acute; mild; MPV; pancreatitis; PLR; severe

INTRODUCTION

Acute pancreatitis (AP) is an inflammatory disease of the pancreas with a quite variable clinical course that develops in the result of the auto-digestion of pancreas by the activation of pancreas enzymes in the pancreas (1). AP can be presented from mild forms with only temporary abdominal symptoms to the severe cases with multiple organ failure that require observation in intensive care (2). AP etiology is fairly variable, and its most common cause is gallbladder stone and alcohol (3). Regardless of etiology, there are similar cascade events in the onset of AP, and it is not known whether edematous or necrotizing pancreatitis will be developed at the start.

In order to determine the prognosis and treatment plan in AP patients, determining disease severity is essential at the start. Since patients with severe course will have higher morbidity and mortality, it is important to differentiate those at early phase. While there are scoring systems with numerous parameters and laboratory markers used for this purpose in AP patients, it is very difficult to determine the prognosis. These parameters are not suitable for the evaluation of patients during application (4). There is still a need for noninvasive, specific, sensitive and simple laboratory markers that can be easily used in daily practices.

There is often an inflammatory condition in AP that is accompanied by thrombosis and bleeding disorders (5). The Platelet Activating Factor (PAF), which is a pro-inflammatory mediator released mostly from vascular endothelium and contributes in local tissue damage and bleeding, poses primary significance for inflammation in AP. PAF activates platelets and neutrophils, increases capillary permeability and causes hypovolemia and edema development (6). Tissue damage and release of

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inflammatory mediators results in an increase in platelet activation (7). The changes in platelet production, activation and function cause changes in Mean Platelet Volume (MPV). As an indicator of platelet function, MPV is easily determined by using automated blood count equipment. MPV has been examined in various proinflammatory and prothrombotic clinical cases (6). There are many articles showing that increases in MPV increases thrombosis risk (8,9). Furthermore, MPV is also increased in inflammatory conditions such as inflammatory bowel disease (10), acute appendicitis (11) and acute cholecystitis (12). There are a small number of studies performed to investigate the relation between MPV and AP, and there are conflicts among the results of those (13-16).

PLR is generally recognized as an indicator of immunoreactivity, and it can simply be calculated by routine peripheral blood tests. There are several studies investigating the prognostic importance of PLR in AP (17-19).

The purpose of this study is to examine the power of MPV and PLR to determine early phase severe AP patients by investigating its relation with clinical, radiological and biochemical parameters used in daily practice to determine disease severity.

MATERIAL and METHODS

Patients diagnosed with AP by using clinical, laboratory and imaging methods, who have applied to emergency department of Okmeydanı Training and Research Hospital in Sağlık Bilimleri University between January 2017 and December 2018, have been included in this study. Extensive demographic, radiographic and laboratory data was collected retrospectively from hospital records of all patients. At least two of the following three symptoms were the sought out criteria for AP diagnosis: 1. Severe and spontaneous abdominal pain spreading to the back that suggests AP, 2. Serum amylase and lipase levels being above 3 times of normal level, 3. Symptoms determined with imaging methods (including abdominal ultrasonography and computed tomography) suggesting AP.

Patients with known chronic pancreatitis and malignant pancreatic disease, heart failure, peripheral vascular disease, hematologic disorder, known acute or chronic inflammatory disease, cancer and chronic liver disease, patients with a history of using anticoagulants, antiaggregants or contraceptives, and patients below age 18 have been excluded from the study.

In order to measure the disease severity, Ranson criteria (20), the 2012 revision of Atlanta classification criteria (21) and computed tomography severity index (CTSI) were used, Balthazar scoring system was performed according to CT (22), and CRP was used as a biochemical marker.

According to Ranson scoring, patients with a score between 0-3 were recognized as mild AP, and those with a score between 4-11 were recognized as severe AP.

Patients were separated in three groups consisting of mild, moderate and severe pancreatitis according to 2012 revision of Atlanta classification criteria (21). Since the fundamental purpose of this study is to differentiate severe AP patients at early phase, mild and moderate AP patients have been grouped together under one group. Their symptoms have been compared with severe AP patients.

In the scales defined by Balthazar et al (22), the grading was based on the degree of necrosis, the presence of inflammation and fluid collections. The severity of pancreatitis was categorized accordingly; mild (score, 0-3), moderate (4-6 points), or severe (7-10 points)

Platelet number, MPV and lymphocyte levels have been documented from the blood count results of the patient at application. PLR was calculated as the ratio of absolute platelet count to absolute lymphocyte count. CRP levels determined at 48th hour after their application to the hospital have been scanned from patient files.

A control group has been selected from healthy individuals who have applied to our hospital for routine check-up, who had no known history of acute or chronic inflammatory disease, and had no history of malignant disease, or history of drug use.

The study was performed in compliance with Helsinki Declaration, and it has been approved by Local Ethics Committee.

Statistical Analysis

The normality of the data in the study was checked with Shapiro Wilk and one sample Kolmogorov Smirnov tests, histogram, Q-Q plots and box plots. Variables with normal distribution were presented as Mean (Mean) ± Standard Deviation (SD), and variables that did not have normal distribution were presented as Median, minimum (Min) and maximum (Max.). In the comparison of continuous variables between two independent groups, variables with normal distribution were analyzed with t test and those that did not have normal distribution were analyzed with Mann Whitney U test. Covariance analysis was performed between groups with different age variables. Variables including 3 or more independent categories that did not have normal distribution or meet parametric test requirements were compared with Kruskal Wallis one-way analysis. Multiple comparisons were not performed since there was no difference. The relation between quantitative variables was examined with Spearman correlation. ROC analysis was performed to determine cut-point values for PLR and MPV. Sensitivity, Specificity, Positive Predictive Value (PPV), Negative Predictive Value (NPV) and Accuracy levels of the tests were presented with 95% confidence intervals. The limit of significance was taken as $p<0.05$ bidirectionally. Analyses were performed by using the NCSS 10 (2015. Kaysville, Utah, USA) software program.

RESULTS

In this study, a total of 140 patients was included in AP patient group consisting of 76 females (54.3%) and 64 males (45.7%) with an average age of 52±15.5, and a total of 49 individuals were included in a healthy control (HC) group consisting of 30 females (61,2%) and 19 males (38.8%) with an average age of 46±12.9. Laboratory data of AP and HC groups that were included in the study is summarized in Table 1.

There was a statistically significant difference with regard to age, neutrophil, lymphocyte and PLR levels between AP and HC groups (p=0.007; p<0.001; p<0.001 and p<0.001, respectively).

Age variable was statistically different between the groups when correction was performed according to age (i.e. covariance analysis), neutrophil, lymphocyte and PLR levels were still different between the groups (p<0.05). Neutrophil and PLR levels in AP group were statistically significantly higher compared to HC group (p<0.001), and lymphocyte levels were lower in a statistically significant level (p<0.001). No statistically significant difference was determined between AP group and HC group with regard to PLT and MPV levels (p>0.05).

Table 1. Demographic data and comparison of AP and HC groups

Variables	AP Group (n=140) Mean ± SD / Median (Min-Max)	HC Group (n=49) Mean ± SD / Median (Min-Max)	P
Age (Years)[a]	54 (19-76)	46 (24-75)	**0.007***
Gender (F/M)	76 (53.3%)/64(45.7%)	30(61.7)/19(38.8%)	
Platelet(/mm^3 x1000/µL)[a]	247 (119-560)	248 (146-363)	0.568
Neutrophil (*10^3/µL)[a]	8.45 (2.06-29.00)	3.67 (1.63-6.21)	**<0.001***
Lymphocyte (*10^3/µL)[a]	1.65 (0.04-4.68)	2.21 (1.26-4.66)	**<0.001***
MPV (fL)[b]	10.4 ±1.0	10.4 ±1.0	0.998
PLR (%)[a]	154.1 (53.8-600.0)	116.2 (43.3-200.7)	**<0.001***

[a]Statistical analysis was performed by using Mann-Whitney U test for variables, and [b] Statistical analysis was performed by using independent samples t-test for variables. *p<0.05 denotes a statistically significant difference
MPV: Mean Platelet Volume; PLR: Platelet /Lymphocyte Ratio

AP patients were separated in two groups consisting of biliary and non-biliary groups according to their etiology, and MPV and PLR values of these groups were individually compared with HC group. The groups were corrected with regard to age (i.e. covariance analysis was performed) since there was an age difference between the groups (Table 2). PLR level was determined to be statistically significantly higher in both biliary and non-biliary AP group compared to HC group (p<0.001). However, there was no difference in MPV. (p>0.05) (Table 2).

Table 2. Comparison of MPV and PLR levels of Biliary and Nonbiliary AP patients with HC group

Variables	HC (n=49) Mean ± SD / Median (Min-Max)	Biliary AP (n=71) Mean ± SD / Median (Min-Max)	Nonbiliary AP (n=69) Mean ± SD / Median (Min-Max)	P[1]	P[2]
MPV (fL)[a]	10.4 ±1.0	10.6 ± 1.0	10.2 ± 1.0	0.338	0.314
PLR (%)[b]	116.2 (43.3-200.7)	161.0 (60.5-560.0)	151.4 (53.8-600.0)	**<0.001***	**<0.001***

[a]Statistical analysis was performed by using independent samples t-test for variables, and [b]Statistical analysis was performed by using Mann-Whitney U test for variables. *p<0.05 denotes a statistically significant difference
p[1] is the "p" value determined in result of the statistical comparison of HC and Biliary AP group variables; and
p[2] is the "p" value determined in result of the statistical comparison of HC and nonbiliary AP group variables

AP patients were compared with regard to MPV and PLR by grouping under biliary and non-biliary groups according to their etiology, ≤150 mg/L and >150 mg/L groups according to CRP levels at the 48th hour, as mild and severe AP patients according to Ranson criteria, and as mild-moderate and severe AP patients according to revised Atlanta criteria (Table 3).

MPV was determined to be statistically significantly lower in the AP group with >150 mg/L CRP level compared to the AP group with ≤150 mg/L CRP level (p=0.046).

Table 3. The distribution and intergroup comparisons of MPV and PLR variables of AP patients according to etiology, CRP levels, Ranson and Atlanta criteria

CRP	Variables	CRP (≤150 mg/L; n=105) Mean ± SD / Median (Min-Max)	CRP (>150 mg/L; n=34) Mean ± SD / Median (Min-Max)	P
	MPV (fL)[a]	10.5 ± 1.0	10.1 ± 1.1	**0.046***
	PLR (%)[b]	148.3 (53.8-560.0)	191.8 (58.6-600.0)	**0.014***
Etiology	Variables	Biliary patients group (n=71) Mean ± SD / Median (Min-Max)	Nonbiliary patients group (n=69) Mean ± SD / Median (Min-Max)	P
	MPV (fL)[a]	10.6 ± 1.0	10.1 ± 1.0	**0.034***
	PLR (%)[b]	161.0 (60.5-560.0)	151.4 (53.8-600.0)	0.772
Ranson	Variables	Mild (n=109) Mean ± SD / Median (Min-Max)	Severe (n=31) Mean ± SD / Median (Min-Max)	P
	MPV (fL)[a]	10.4 ±1.0	10.2 ± 1.2	0.355
	PLR (%)[b]	151.4 (58.6-600.0)	166.7 (53.8-560.0)	0.311
Atlanta	Variables	Mild-Moderate (n=111) Mean ± SD / Median (Min-Max)	Severe (n=29) Mean ± SD / Median (Min-Max)	P
	MPV (fL)[a]	10.4 ± 1.0	10.1 ±1.3	0.276
	PLR (%)[b]	151.0 (58.6-540.0)	188.4 (53.8-600.0)	**0.023***

[a]Statistical analysis was performed by using independent samples t-test for variables, and
[b]Statistical analysis was performed by using Mann-Whitney U test for variables. *p<0.05 denotes a statistically significant difference

PLR was determined to be statistically significantly higher in AP group with >150 mg/L CRP level (p=0.014).

MPV was determined to be statistically significantly lower in non-biliary AP group compared to biliary AP group (p=0.034), and no statistically significant difference was determined between PLR variables of both groups (p>0.05).

No statistically significant difference was determined between mild AP group and severe AP group with regard to MPV and PLR according to Ranson criteria (p>0.05).

PLR was determined to be statistically significantly higher in severe AP group compared to mild-moderate AP group according to revised Atlanta classification (p=0.023), and no statistically significant difference was determined between MPV levels of both groups (p>0.05).

AP patients was grouped as mild, moderate and severe patients according to their Balthazar score, and compared with regard to MPV and PLR (Table 4).

No statistically significant difference was determined among AP patients grouped as mild, moderate and severe according to Balthazar score with regard to MPV and PLR (p>0.05).

For testing the usability of PLR as a determinant test in AP diagnosis, and determining diagnostic test performance parameters and applicable cut-points belonging to parameters used in activity scoring, AP/HC, CRP and Revised Atlanta criteria were determined as state variables and ROC analysis was performed.

In the result of ROC analysis, it was determined for PLR in AP and HC that the diagnostic threshold value was "123" for determining sick individuals, diagnostic threshold value was "153" for determining for >150 CRP at 48th hour, and the diagnostic threshold value was "151" for severe AP according to Revised Atlanta criteria. By using determined threshold values for PLR, Sensitivity, Specificity, Positive Predictive Value (PPV), Negative Predictive Value (NPV), Accuracy and Standard Error (SE), and Area under the curve

Table 4. Distribution and comparison of MPV and PLR levels in AP patients grouped according to Balthazar score

Balthazar	Variables	Mild (n=67) Mean ± SD / Median (Min-Max)	Moderate (n=30) Mean ± SD / Median (Min-Max)	Severe (n=15) Mean ± SD / Median (Min-Max)	p
	MPV (fL)[a]	10.3 ±1.0	10.4 ±1.0	10.1 ±1.3	0.636
	PLR (%)[b]	166.4 ±43.5	190.08 ±119.2	235.71 ± 162.6	0.415

p values were determined with Kruskal Wallis test

(AUC) with P value demonstrated under 95% Confidence Interval (CI) in Figure 1,2,3.

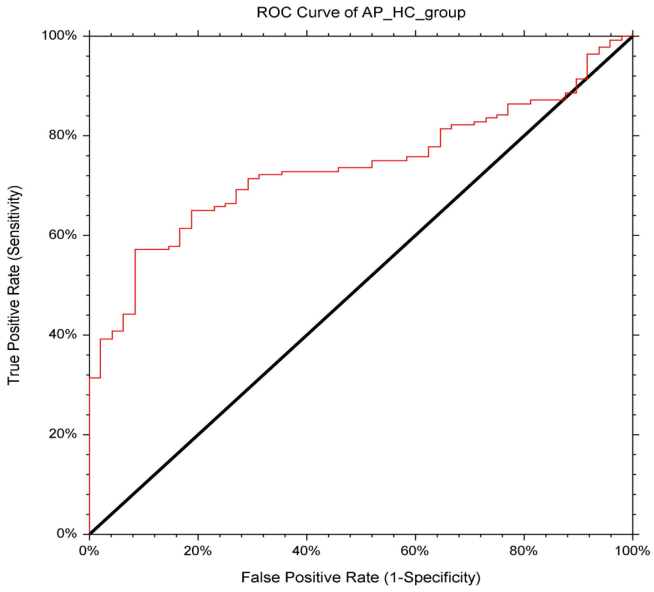

Figure 1. ROC curve graph of PLR variable according to AP-HC group

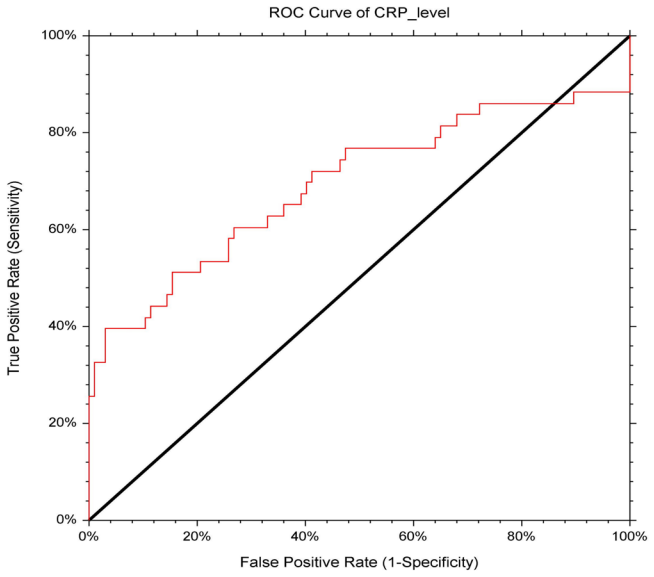

Figure 2. ROC curve graph of PLR variable according to CRP level

The relation between MPV, PLR and Hospitalization Period variables in AP patients was examined with Spearman correlation analysis. In non-biliary AP group, a statistically significant positive weak correlation was determined between hospitalization period and PLR (r=0.239; p=0.049) and a statistically significant negative weak correlation was determined between MPV and PLR (r=-0.256; p=0.035), and no statistically significant correlation was determined between hospitalization period and MPV (p>0.05) No statistically significant correlation was determined between MPV, PLR and hospitalization periods in the biliary acute pancreatitis patient group (p>0.05).

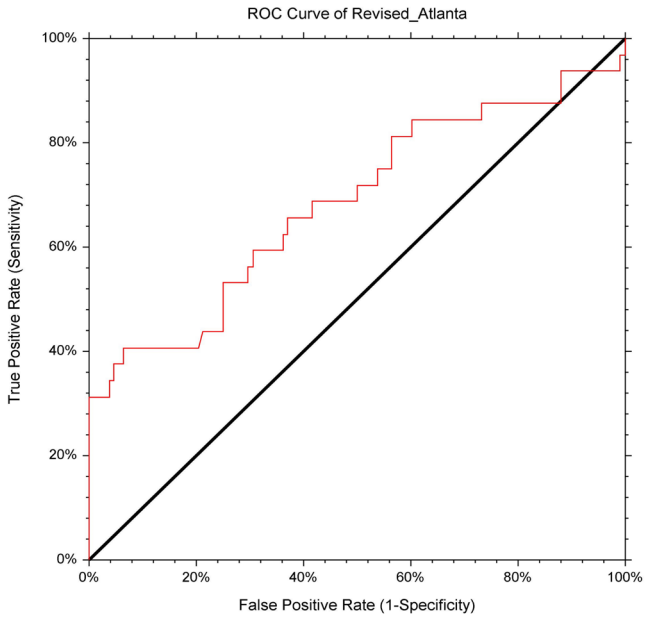

Figure 3. ROC curve graph of PLR variable according to Revised Atlanta criteria

DISCUSSION

AP is one of the most common gastrointestinal emergencies. While mortality rate is around 1% in all AP cases, this rate may reach 20%-30% in severe acute pancreatitis cases (23). For this reason, being able to detect severe AP at early phase and to manage treatment and complications are quite important for deciding whether hospitalization is needed.

AP pathophysiology is not completely elucidated in our day. The triggering mechanism in AP is the activation of trypsinogen in the pancreas. Active trypsin also activates coagulation and fibrinolysis along with complement system and kallikrein-kinin cascade. Increased inflammatory cytokines in the environment are considered to be responsible for systemic manifestations and complications. Cytokines cause an increase in adhesion molecules locally or systemically, and then those trigger the inflammatory cascade by leukocyte migration, complement activation, and the production of phospholipase A2, NO and oxygen radicals (24). Furthermore, in an inflammation atmosphere like AP, neutrophils extend the tissue destruction of inflammatory cytokines (IL-6,IL-8 and TNF alpha) by the activation of a range of proteolytic enzymes (myeloperoxidase, elestase, collagenase) and free oxygen radicals. Lymphocytes are increased following the initial stress and then decreased within the first 24 hours, and lymphopenia is developed (25). In a study performed by Penzili and colleagues, lymphopenia has been reported in AP patients on day 1, continuing into day 3 and 5 (26). In our study, lymphocyte levels checked at the admission of AP patients were lower compared to HC (p <0.001). Coagulation anomalies and changes in platelet number have been determined to be associated with disease severity in AP (27). Much of

the cytokines determined to play an important role in AP pathogenesis can affect MPV. In light of these results, our purpose was to investigate whether MPV and PLR is a parameter that can estimate severe AP in early phase, as an indicator of platelet function.

There is a small number of studies in literature that investigate the relation between PLR and AP. No statistical difference was determined between AP and control group with regard to PLR in a study performed by Ilhan et al. on pregnant AP patients (19). However, this study was performed on a special patient group, and physiological changes caused by pregnancy may have affected PLR results. In the study of Kaplan and colleagues, it was demonstrated that there was a relation between PLR and AP severity (17). They have reported that there is a correlation between PLR and Ranson, Atlanta and BISAP scores, and that taking PLR> 342.31 as cut-off value had 73.3% sensitivity and 99.2% specificity for demonstrating disease severity.

In another study performed on biliary and alcoholic AP patients (18), it has been reported that PLR was higher in biliary AP group compared to AP group due to alcohol, PLR was correlated with revised Atlanta scoring, Ranson and computed tomography severity index (CTSI) in biliary AP group, and that PLR was higher in severe AP patients. In this study, they have attributed the reason why PLR was low to thrombocytopenia, disrupted platelet production, and alcohol-related chronic liver disease, resulting in decreased hepatic synthesis of thrombopoietin, in the group with AP due to alcohol. Similar to our study, PLR levels of patients at application have been taken in this study.

In our study, PLR values of AP group was statistically higher than HC group ($p<0.001$). Upon performing ROC analysis in order to measure the diagnostic value of PLR in AP patients, cut off value was >123 at 95% confidence interval, sensitivity was determined as 72% and specificity was determined as 69%. In addition, PLR was determined to be statistically higher in severe AP patients compared to mild-moderate AP patients with regard to revised Atlanta score. In order to investigate the diagnostic value of PLR for differentiating severe AP patients from mild-moderate AP patients at application, according to revised Atlanta scoring, the most suitable cut-off value was >151 at 95% confidence interval, and sensitivity was determined as 69% and specificity as 62% upon performing ROC analysis. There was no difference in severe AP patients with regard to PLR according to Ranson and Balthazar scoring. No statistical difference was determined between the groups with regard to PLR upon grouping as biliary and non-biliary AP according to etiology. In the correlation analysis between hospitalization periods and PLR, a positive correlation was determined for PLR with hospitalization period in the AP group of non-biliary etiology. That means hospitalization period is prolonged with increasing PLR in AP patients with non-biliary etiology.

There is a small number of studies investigating the relation of MPV in AP patients. However, there are conflicting results between these studies. In a study performed by Beyazıt and colleagues (16), it was determined that MPV value was significantly reduced in AP groups compared to control group. They have determined that severe AP patients had lower MPV values compared to mild AP patients according to modified Glasgow prognostic score. They have reported decreased MPV level with poor prognosis, but also that MPV did not have any superiority against other inflammatory markers for determining prognosis. Meanwhile, higher MPV levels have been suggested in AP patients compared to control group in the study performed by Akbal and colleagues (14). In a study performed by Mimidis et al. (28), lower MPV values have been reported in early phase AP patients compared to AP patients in remission. Increased levels of tumor necrosis factor-α (TNF-α), interleukin-1 (IL-1), IL-6 and monocyte chemotactic protein-1 (MCP-1) have been demonstrated in AP. Among those, IL-6 has been suggested to be the main factor responsible for the decrease in MPV levels (28). Different results have been obtained in these studies that were similar in character.

In our study, no difference was determined between AP and HC with regard to MPV, and also no difference was determined with regard to MPV between mild and severe AP groups according to Ranson criteria, revised Atlanta criteria and Balthazar score that were used to determine disease severity. In our study, only MPV was lower in patients with CRP >150 levels at the 48th hour compared to patients with CRP≤150 levels. The results of the study performed by Kefeli et al. (29) supports our results; no statistically significant difference was shown among early phase AP patients, AP remission and control groups with regard to MPV levels. Early stage MPV was the levels measured at the application of patients in this study.

Platelets do not only control thrombosis and hemostasis in AP patients, they also seem to determine the inflammatory process. It is considered that consumption of large platelets in inflammation area in AP may explain the decrease in MPV level (6). The reason why there was no difference between MPV and control in our study and the study of Kefeli et al. may be explained with the fact that inflammation was not set completely since MPV level was taken from the complete blood count values at the application of AP patients to the hospital. Since the purpose of our study is to investigate MPV as an early predictor for severe AP, first MPV values measured at the application to the hospital have been evaluated. In light of these results, it may be suggested to use MPV values measured at least 1 day after hospital admission or at the onset of symptoms for MPV examinations in AP patients.

CRP is an acute phase reactant produced by the liver against IL-1 and 2, and it has been reported in a study performed by Lei and colleagues (15) that one of the most beneficial serum biochemical markers used to estimate AP severity and progression despite the delayed increase of CRP in AP patients. In a study investigating the relation

between AP severity and CRP level at hour 0, 24, 48 and 72 (30), they have reported that CRP levels above 150 in the first 48 hours could be used as a cheap and safe test for determining AP with severe course (sensitivity 80%, specificity 76%). CRP is still the most useful one among biochemical markers used for determining AP severity and complications in daily practices, and it is used commonly. Its largest disadvantage is that it does not give an early peak immediately after symptoms, and it is delayed by 48-72 hours. In light of this information, we have examined 48th hour CRP levels in order to investigate the relationship between PLR measured at early phase with CRP levels at the 48th Hour. We have grouped the patients under CRP > 150 and ≤150 groups. PLR values were determined to be higher in AP group with CRP >150 than the group with CRP ≤150 levels. The most suitable threshold value of PLR measured at early stage for differentiating patients with CRP >150 levels at 48th hour was >155 in 95% confidence interval, and sensitivity and specificity were determined as 71% and 67%, respectively. It was reported in a study demonstrating the relation between PLR and CRP in AP patients that there was a positive correlation between CRP and PLR (17).

This study has some limitations. The first one is there was a small number of patients participating in this study and it was a retrospective study. The second one is AP severity was compared with MPV and PLR levels at hospital admission in this study. Since the inflammation and clinical status have not completely set down yet at hospital admission, MPV and PLR levels may have been different than expected. If a comparison were to be made with subsequent and remission MPV and PLR levels of the patient, the value of MPV and particularly PLR in estimating severe AP patients may have been emphasized more. Thirdly, we did not do any comparison with biochemical markers such as urinary trypsinogen activation peptide (TAP) that are used to determine AP severity at patient admission . Despite these limitations, this study also has strengths. This study is one of the rare studies that evaluates MPV and PLR together in the same patient group and at patient admission, and it is the first study investigating the strength of PLR to predict CRP levels above 150 at 48th hour.

CONCLUSION

In conclusion, we did not determine a relation between MPV measured at patient admission and severe AP. However, we have found a relation between PLR and AP severity. Nevertheless, due to low PLR sensitivity and specificity, it may be used as a supporting parameter that can be easily calculated and interpreted in emergency conditions without requiring any additional cost at early stage, even though it cannot take the place of recognized scoring systems that are used in routine practices. In order for these parameters to be recommended for routine use in daily practices, there is a need for prospective studies in the future that contains a higher number of patients.

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Ethical approval: In the meeting of the Ethics Committee of Okmeydani Training and Research Hospital, Faculty of Health Sciences, on 05/03/2019, it was found ethically appropriate with the decision numbered 48670771-514.10.

REFERENCES

1. Albert KR, Slutsky A, Ranieri M, et al. Clinical Critical Care Medicine. Pancreatitis; Marshall JC. Chapter 2006;48:525-30,
2. Otsuki M, Takeda K, Matsuno S, et al. Criteria for the diagnosis and severity stratification of acute pancreatitis. World Journal of Gastroenterology: WJG 2013;19:5798.
3. Halangk W, Lerch MM. Early events in acute pancreatitis. Gastroenterol Clin N Am 2004;33:717-31.
4. Pavlidis TE, Pavlidis ET, Sakantamis AK. Advances in prognostic factors in acute pancreatitis: a mini-review. Hepatobiliary Pancreat Dis Int 2010;9:482-6.
5. Mimidis K, Papadopoulos V, Kotsianidis J, et al. Alterations of platelet function, number and indexes during acute pancreatitis. Pancreatology 2004;4:22-7.
6. Gasparyan AY, Ayvazyan L, Mikhailidis DP, et al.Mean platelet volume: a link between thrombosis and inflammation? Curr Pharm Des 2011;17:47-58.
7. Kerekes L, Arkossy P, Altorjay I, et al. Evaluation of hemostatic changes and blood antioxidant capacity in acute and chronic pancreatitis. Hepatogastroenterology 2001;48:1746-9.
8. Chu SG, Becker RC, Berger PB, et al. Mean platelet volume as a predictor of cardiovascular risk: a systematic review and meta-analysis. J Thromb Haemost 2010;8:148-56.
9. Balcik OS, Bilen S, Ulusoy EK, et al. Thrombopoietin and mean platelet volume in patients with ischemic stroke. Clin Appl Thromb Hemost 2001;19:92-5.
10. Kapsoritakis AN, Koukourakis MI, Sfiridaki A, et al. Mean platelet volume: a useful marker of inflammatory bowel disease activity. Am J Gastroenterol 2001;96: 776-81.
11. Fan Z, Pan J, Zhang Y, et al. Mean Platelet Volume and Platelet Distribution Width as Markers in the Diagnosis of Acute Gangrenous Appendicitis. Dis Markers 2015;2015:542013.
12. Sayit AT, Gunbey PH, Terzi Y. Is the Mean Platelet Volume in Patients with Acute Cholecystitis an Inflammatory Marker? J Clin Diagn Res 2015;9:5-7.
13. Okuturlar Y, Soylu A, Dogan H, et al. Mean platelet volume in patients with biliary and non-biliary acute pancreatitis. Int J Clin Exp Pathol 2015;8: 2051-6.

14. Akbal E, Demirci S, Kocak E, et al. Alterations of platelet function and coagulation parameters during acute pancreatitis. Blood Coagul Fibrinolysis 2013;24: 243-6.
15. Lei JJ, Zhou L, Liu Q, et al. Can mean platelet volume play a role in evaluating the severity of acute pancreatitis? World J Gastroenterol 2017;23:2404-13.
16. Beyazit Y, Sayilir A, Torun S, et al. Mean platelet volume as an indicator of disease severity in patients with acute pancreatitis. Clin Res Hepatol Gastroenterol 2012;36:162-8.
17. Kaplan M, Ates I, Oztas E, et al.A New Marker to Determine Prognosis of Acute Pancreatitis: PLR and NLR Combination J Med Biochem 2018;37:21-30.
18. Cho SK, Jung S, Lee KJ, et al. Neutrophil to lymphocyte ratio and platelet to lymphocyte ratio can predict the severity of gallstone pancreatitis. BMC Gastroenterol 2018;18:18.
19. Ilhan M, Ilhan G , Gok AF, et al.Evaluation of neutrophil-lymphocyte ratio, platelet lymphocyte ratio and red blood cell distrtribution with-platelet ratio as early predictor of acute pancreatitis in pregnancy. J Matern Fetal Neunatal Med 2016;29:1476-80.
20. Ranson JH, Rifkind KM, Roses DF, et al. Objective early identification of severe acute pancreatitis. The American J Gastroenterology 1974;61:443.
21. Banks PA, Bollen TL, Dervenis C. Classification of acute pancreatitis-2012: revision of the Atlanta classification and definitions by international consensus. Gut 2013;62:102.
22. Balthazar EJ, Robinson DL, Megibow AJ, et al. Acute pancreatitis: value of CT in establishing prognosis. Radiology 1990;174: 331-6.
23. Whitcomb DC. Clinical practice. Acute pancreatitis. The New England J Med2006;354:2142.
24. Elfar M, Gaber LW, Sabek O, et al. The inflammatory cascade in acute pancreatitis: relevance to clinical disease. Surg Clin North Am 2007;87:1325-40.
25. de Jager CP, van Wijk PT, Mathoera RB, et al. Lymphocytopenia and neutrophil-lymphocyte count ratio predict bacteremia better than conventional infection markers in an emergency care unit. Crit Care 2010;14:192.
26. Pezzilli R, Billi P, Beltrandi E, et al. Impaired lymphocyte proliferation in human acute pancreatitis. Digestion 1997;58(5):431-6.
27. Kakafika A, Papadopoulos V, Mimidis K, et al.Coagulation, platelets, and acute pancreatitis. Pancreas 2007;34:15-20.
28. Mimidis K, Papadopoulos V, Kartasis Z, et al. Assessment of platelet adhesiveness and aggregation in mild acute pancreatitis using the PFA-100 system. JOP 2004;5:132-7.
29. Kefeli A, Basyigit S, Ozgür Yeniova A, et al.Platelet Number and Indexes during Acute Pancreatitis. Euroasian J Hepatogastroenterol 2014;4:67-9.
30. Beger HG, Warshaw AL, Larvin M, et al. Assessment of clinical severity and prognosis. In: The Pancreas, (Eds), Blackwell Science. Oxford 1998;489.