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Original Article

Prognostic significance of mean platelet volume to platelet count ratio in pediatric patients with acute kidney injury

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ABSTRACT

Background. Mean platelet volume (MPV), which is regarded as a marker of thrombocyte function and activation, is related to increased morbidity and mortality. In critically ill patients, the ratio of MPV to platelets can independently predict adverse outcomes. This study aimed to investigate the prognostic value of the mean platelet volume/platelet count ratio (MPR) for mortality in children with acute kidney injury (AKI).

Methods. In this retrospective study, patients hospitalized in the pediatric intensive care unit (PICU) between March 2020 and June 2022 were evaluated. Patients between 1 month and 18 years of age with AKI were enrolled. Clinical and laboratory data were compared between survivors and non-survivors. The MPR ratio was calculated on the first and third days of admission to the intensive care unit. A multiple logistic regression analysis was used to determine the association between MPR and mortality. ROC curves were used for the prediction performance of the logistic regression models and cut-off values of the thrombocyte indices.

Results. Sixty-three children with AKI were included in the study. The total mortality rate was 34.9% (n=22). MPR ratios were significantly higher in the non-survivors at admission (p=0.042) and at the 72nd hour (p=0.003). In the multiple logistic regression analysis, thrombocyte counts and MPR_{72h} ratio were found to be independent risk parameters for adverse outcomes in children with AKI.

Conclusions. MPR is an inexpensive and practical marker that may predict the outcome of children with AKI.

Key words: acute kidney injury, mortality, mean platelet volume, platelet count, mean platelet volume platelet count ratio, children.

Acute kidney injury (AKI) is a complex medical condition defined as a sudden deterioration of renal function, particularly in critically ill patients, such as sepsis, shock, trauma, major surgical operations, and the utilization of nephrotoxic medications.[1] The incidence of AKI varies from 1% to 82% in pediatric populations, while its incidence is 10% in patients admitted to the intensive care unit.[2] Despite improvements in the management of renal diseases, AKI has a high morbidity and mortality rate. It has been shown that AKI is related to 40-60% of mortality in the intensive care setting.[3,4] Nevertheless, mortality rates are considerably high, especially in dialysis-requiring AKI.[5] Therefore, early recognition of AKI or the factors indicating the highest risk of developing AKI is important in order to initiate appropriate treatment options. The development of AKI is the result of the activation of inflammation and coagulation following acute injury. Thrombocytes have a substantial role in the coagulation processes, and it has been indicated that activation of thrombocytes aggravates renal injury.[6] Although the adhesion molecule P-selectin is regarded as the "gold standard" marker of thrombocyte activation that is expressed from activated endothelium and thrombocytes cause the initial attachment of leukocytes to

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the inflamed vascular endothelium, it can't be broadly preferred in clinical practice because of the price and requirement of laboratory facilities.[7,8] In routine blood counts, mean platelet volume (MPV) is measured, which is accepted as a significant marker for the evaluation of thrombocyte function and activity.[9] Larger thrombocytes secrete more thromboxane-A2, β-thromboglobulin, and adhesion molecules to be more metabolically and enzymatically active.[10] A study showed that during renal ischemia-reperfusion injury, necrotic cell-derived DNA leads to the activation of thrombocytes, thrombocyte-granulocyte interaction, and following neutrophil extracellular trap formation, resulting in a further increase in kidney inflammation and tissue damage.[11] In recent years, many new studies investigating the understanding of the pathophysiology of AKI have led to the discovery of new biomarkers. However, the use of these biomarkers in clinical practice is quite limited. Thrombocyte activation and high inflammatory parameters in sepsis have important effects on the development of AKI.[12] Several studies have shown an inverse correlation between thrombocyte counts and MPV in seriously sick patients and suggested that the combination of thrombocyte counts and MPV may be clinically more important than thrombocyte counts or MPV alone.[13,14] Therefore, it is thought that the change in MPV/PLT count ratio (MPR) in pediatric patients may be related to AKI. The goal of this study was to determine the prognostic significance of the MPR for pediatric patients with AKI.

Material and Methods

Patient population

We conducted this retrospective, observational single-center research from March 2020 to May 2022 to analyze the association between MPR and AKI in patients at Afyonkarahisar Health Sciences University Faculty of Medicine Hospital Pediatric Intensive Care Unit (PICU). This study was approved by Ethical Committee of Afyonkarahisar Health Sciences University Faculty of Medicine (date: 01.07.2022, no: 2022/8). AKI was defined according to the Kidney Disease: Improving Global Outcomes 2012 (KDIGO) criteria as an increase in initial serum creatinine (SCr) of 0.3 mg/dl within 48 hours or a 1.5-fold increase in the initial SCr level within seven days or urine output (UOP) of < 0.5 ml/kg/hour for six hours.[15] Patients aged 1 month to 18 years old with AKI, length of hospital stay ≥ 72 h, and complete records were enrolled in the study. Mortality was defined as death prior to 28 days after admission.

Exclusion criteria

Children who received a platelet transfusion in the first 72 hours of their admission to the PICU, had chronic renal failure, diabetes mellitus, hematological or neoplastic disease, connective tissue disease, acute or chronic active inflammatory diseases, hospitalized for less than 72 hours, patients taking anticoagulant or antiaggregant drugs, and had missing data were excluded from the study.

Collection of blood samples

The patients' data were acquired from the electronic medical record system of the hospital. Blood specimens were gathered in tubes containing ethylene diamine tetraacetic acid (EDTA) and hemoglobin value, thrombocyte count, and MPV levels were analyzed within a maximum of 60 minutes after sampling. Demographic data, hemodynamic variables, laboratory values at the time of admission, and clinical outcomes on day 28 were recorded. Pediatric risk of mortality score III (PRISM III), Pediatric logistic organ dysfunction (PELOD, thrombocyte counts, and MPV levels were enrolled. In addition, for each patient MPR [(MPV value/platelet count/10^3) ×100] values were calculated separately for admission and at 72 hours.[16]

Statistical analysis

Statistical analysis was performed by SPSS Statistics 22 software (IBM, Armonk, NY, USA).[17] The patients were divided into two groups based on the outcome: survivors and non-survivors. The normality of the variables was assessed through a combination of visual methods, such as histograms and Q-Q plots, as well as analytical approaches including the Kolmogorov-Smirnov and Shapiro-Wilk tests. If the normal distribution assumption was satisfied mean ± SD was given; otherwise median (interquartile range - IQR) was given for continuous variables. Differentiations in continuous parameters were compared by the Mann-Whitney U test and independent samples t-test. Categorical variables were compared using the Pearson chi-square test, chi-square test with continuity correction, Fisher exact test, or Fisher-Freeman-Halton exact test, depending on the size of the cross-tables and the status of expected values less than 5.

Spearman's correlation analysis was used to determine the relationship among MPR_{adm}, MPR_{72h}, PLT_{adm}, PLT_{72h}, $MPV_{adm,}$ MPV_{72h}, PRISM III score, PELOD score, white blood cell (WBC), C-reactive protein (CRP), and serum albumin. As the MPR values were not normally distributed, the admission diagnosis and the MPR values were compared with the Kruskal-Wallis test.

Logistic regression analysis was performed to evaluate the association between the risk factors and mortality by calculating the odds ratios (OR), adjusted odds ratios (AOR), and 95% confidence intervals (CI). For multiple logistic regression, all possible factors identified with univariate analysis ($p<0.25$) were included in the model to detect independent predictors for outcome. Multicollinearity was assessed by calculating the variance inflation factor (VIF). Variables with values less than 5 were considered to have no significant similarity. Multiple logistic regression models with backward elimination were performed to analyze the association between thrombocyte indices and mortality. Hosmer Lemeshow statistics and Nagelkerke R square were used to check how well the logistic regression model fits the data. MedCalc for Windows, version 19.6 (MedCalc Software, Ostend, Belgium)[18] was used to plot receiver operating characteristic (ROC) curves, calculate the area under the ROC curve (AUC), and compare the prediction performance of the logistic regression models and cut-off values of the thrombocyte parameters. A cut-off value for the variables was calculated by the Youden Index. A p value < 0.05 was accepted as statistically significant.

Results

Eighty-seven AKI patients were admitted to the PICU during the research period. Twenty-four children who had connective tissue disease, diabetes mellitus, chronic renal failure, hematologic disease, or thrombocyte transfusion were excluded because they did not meet the study criteria. Hence, 63 children were finally enrolled. There were 33 male (52.4%) and 30 female (47.6%) cases. The children were allocated into two groups, survivors and non-survivors. Of these 63 patients with AKI, 41 were in the survivor group, while 22 were in the non-survivor group. The total mortality rate was 34.9%. Sepsis was the most common admission diagnosis (n=25, 39.6%), followed by cardiovascular disease (17.4%) and respiratory infection (14.3%). There was no significant difference between the survivor and non-survivor groups in terms of age, gender, and vital signs (Table I). Fluid overload was higher in non-survivors but not statistically significant (p=0.434). The stages of AKI did not show statistically significant differences in terms of mortality (p=0.130). The non-survivor group had an inconsiderably longer mechanical ventilation day (p=0.293) and the length of PICU days was insignificantly higher in the survivor group (p=0.158). The comparison of white blood cell count, hemoglobin level, pH, base deficit, lactate, liver function tests, and albumin

Table I. Demographics, clinical characteristics, and laboratory variables of survivors and non-survivors

Variables	Survivors (n=41)	Non-survivors (n=22)	p value
Age, months*	61 (142.25)	65.5 (164)	0.965[&]
Female gender, n (%)	23 (56.1)	7 (31.8)	0.115[Φ]
Respiratory rate, /min	36.95±13.50	36.36±12.43	0.866[†]
Heart rate, /min	141.31±35.70	147.95±36.61	0.488[†]
Systolic blood pressure, mmHg	89.41±15.22	86.59±19.24	0.525[†]
Most common four-admission diagnosis, n (%)			
Sepsis	12 (29.3)	13 (59.1)	0.097[¥]
Respiratory infections	8 (19.5)	1 (4.5)	
Cardiovascular disease	7 (17.1)	4 (18.2)	
Trauma	3 (7.3)	2 (9.1)	
Others	11 (26.8)	2 (9.1)	
Fluid overload, n (%)	4 (9.8)	4 (18.1)	0.434[ŧ]
Duration of mechanical ventilation, days*	3 (7.5)	4 (12.25)	0.293[&]
Length of PICU stay, days*	6 (17.5)	5 (10.75)	0.158[&]
AKI stage, n (%)			
Stage 1	8 (19.5)	1 (4.5)	0.130[‡]
Stage 2	18 (43.9)	8 (36.4)	
Stage 3	15 (36.6)	13 (59.1)	
Hgb, g/dL	11.66±1.62	11.38±1.06	0.328[†]
WBC_{adm}, $\times 10^3/\mu L$	10.16±5.68	12.46±5.87	0.136[†]
PLT_{adm}, $\times 10^3/\mu L$	160.29±51.47	125.54±36.38	0.003[†]
PLT_{72h}, $\times 10^3/\mu L$	141.09±46.35	108.04±28.49	0.001[†]
MPV_{adm}, fL	10.97±0.80	11.11±0.63	0.474[†]
MPV_{72h}, fL	11.54±0.57	11.84±0.45	0.027[†]
MPR_{adm}	7.82±3,45	9.7±3,36	0.042[†]
MPR_{72h}	9.13±3.36	11.73±3.21	0.003[†]
AST (U/L)*	63 (68)	54.5 (36.25)	0.199[&]
ALT (U/L)*	44 (63)	57 (67.5)	0.349[&]
PRISM III score	16.04±8.1	21.45±7.62	0.012[†]
PELOD score	18.12±14.43	28.95±11.84	0.004[†]
pH	7.34±0.04	7.33±0.05	0.562[†]
Base deficit, mmol/L	-2.30 ± 1.76	-2.51 ± 2.13	0.676[†]
$Lactate_{adm}$, mmol/L*	1.8 (1.25)	1.8 (1.55)	0.994[&]
$Albumin_{adm}$, g/dL	3.56±0.57	3.53±0.48	0.808[†]
CRP_{adm}, mg/dL*	21 (8.5)	18 (8.25)	0.269[&]

ALT: alanine aminotransferase; AST: aspartate aminotransferase; CRP: C-reactive protein; Hgb: hemoglobin; Length of PICU stay: length of pediatric intensive care unit stay; MPR: MPV/PLT ratio; MPV: mean platelet volume; PELOD: pediatric logistic organ dysfunction; PLT: platelet; PRISM III score: pediatric risk of mortality III score; WBC: white blood cell.
* Median (interquartile range) values, [&]: Mann-Whitney U test, [†]: Independent samples t test, [¥]: Fisher-Freeman-Halton exact test, [ŧ]: Fisher exact test, [Φ]: Chi square test with continuity correction, [‡]: Pearson chi square test.

levels between the two groups are presented in Table I. The non-survivors had a significantly higher PELOD and PRISM III score than the survivors (28.95±11.84 vs 18.12±14.43, p=0.004; 21.45±7.62 vs 16.04±8.10, p=0.012, respectively). While MPV values were similar between the two groups at admission, MPV_{72h} values were significantly higher in non-survivors

(MPV_{adm}: 11.11±0.63 vs 10.97±0.80; p=0.474; MPV_{72h}: 11.84±0.45 vs 11.54±0.57; p=0.027). Thrombocyte counts were significantly lower in non-survivors at admission and 72nd hours (PLT_{adm}(×10³/μL): 125.54±36.38 vs 160.29±51.47, p=0.003; PLT_{72h}(×10³/μL): 108.04±28.49 vs 141.09±46.35; p=0.001). Non-survivors exhibited a significantly higher MPR ratio than survivors at admission, and 72nd hours ($MPR_{adm:}$ 9.7±3.36 vs 7.82±3.45, p=0.042; MPR_{72h}: 11.73±3.21 vs 9.13±3.36; p=0.003, respectively).

Despite conducting correlation analysis between MPV_{adm}, MPV_{72h}, MPR_{adm}, MPR_{72h} and platelet counts, WBC, CRP, and albumin levels with the PRISM III score, no significant correlations were observed, as indicated in Table II. The relationship between admission diagnoses and MRP values was assessed using the Kruskal-Wallis test, which indicated no statistically significant impact on MRP values (MPR_{adm}: p=0.656; $MPR_{72h:}$ p=0.820).

Four variables (gender, PRISM III, length of PICU stay and WBC) in univariate analysis with a p-value < 0.25 and thrombocyte indices (PLT_{adm}, PLT_{72h}, MPV_{adm}, MPV_{72h}, MPR_{adm} and MPR_{72h}) were included in the multiple logistic regression analysis (Table III). PELOD was not included in the regression model due to the high correlation with PRISM III (r: 0.78, p<0.001). The variables MPR_{adm} and MPR_{72h}, derived from the PLT and MPV values on the relevant day, were added separately to the regression model in order to avoid multicollinearity issues. AKI stage and admission diagnosis were not included in the models due to an insufficient number of patients for analysis. Multiple logistic regression analysis demonstrated that both PLT_{adm} (AOR: 0.981, 95%CI (0.967-0.995); p=0.009) and PLT_{72h} (AOR: 0.972, 95% CI (0.945-1.000); p=0.046) were independent risk factors for mortality. Each increase of 1×10³/μL in PLT_{adm} and PLT_{72h} was associated with a 0.981 fold and 0.972-fold decrease in the risk of mortality, respectively. Increased MPR_{adm} (AOR: 1.184, 95% CI (0.991-1.414); p=0.063) was not significantly associated with mortality. MPR_{72h} (AOR: 1.537, 95% CI (1.081-2.184); p=0.017) was an independent risk factor for mortality in patients with AKI. Each increase in MPR_{72h} associated with a 1.537-fold increase in the risk of mortality. The discriminative ability of models was shown with the ROC curves for each model (Table IV).

Table III. Univariate analyses with binary logistic regression of risk factor of mortality

Variables	Univariate analysis		
	OR	95% CI	p value
Gender	2.74	0.92-8.1	0.070
PRISM III	1.01	1.02-1.18	0.018
AST	0.99	0.98-1.0	0.291
WBC	1.07	0.98-1.18	0.139
Length of PICU stay	0.99	0.96-1.02	0.635
PLT_{adm}	0.98	0.97-0.99	0.010
PLT_{72h}	0.97	0.96-0.99	0.007
MPV_{adm}	1.30	0.64-2.64	0.468
MPV_{72}	2.9	1.04-8.22	0.042
MPR_{adm}	1.0	1.0-1.03	0.051
MRP72	1.27	1.07-1.52	0.006

MPR: MPV/PLT ratio; MPV: mean platelet volume; OR: Odds ratio; PICU: Pediatric intensive care unit; PLT: platelet count; PRISM III score: Pediatric risk of mortality III score.

Table II. Correlation between MPV_{72h}, MPR_{adm}, MPR_{72h}, platelet counts and other variables

Variables	MPV_{adm}		MPV_{72h}		PLT_{adm}		PLT_{72h}		MPR_{adm}		MPR_{72h}	
	r	p	r	p	r	p	r	p	r	p	r	p
WBC	-0.35	0.78	-0.23	0.86	-0.20	0.11	-0.10	0.42	0.19	0.13	0.12	0.35
CRP	0.19	0.14	0.17	0.17	0.16	0.21	0.14	0.20	-0.13	0.31	-0.13	0.32
Albumin	0.01	0.93	-0.63	0.6	0.07	0.56	-0.03	0.79	-0.08	0.53	0.025	0.84
PRISM III	-0.06	0.62	0.17	0.18	0.08	0.49	0.14	0.26	-0.10	0.43	-0.10	0.40

CRP: C-reactive protein; MPR: MPV/PLT ratio; MPV: mean platelet volume; PRISM III score: pediatric risk of mortality III score; WBC: white blood cell.

Table IV. Multiple logistic regression analysis and the model discriminative ability by area under the ROC curve

	AOR	95% CI	p value	AUC (95% CI)	p value
Model 1 –Binary logistic regression for PLT_{adm}					
Constant	0.451		0.563		
PLT_{adm}	0.981	0.967-0.995	0.009	0.834 (0.719-0.916)	<0.001
Variables Included in the Model: PLT_{adm}					
Confounding factors: Gender, PRISMIII, WBC					
Model Summary: Hosmer and Lemeshov Test χ^2=4.832; p=0.775; Nagelkerke R^2=0.375					
Model 2 –Binary logistic regression for PLT_{72h} and MPV_{72h}					
Constant	0.714		0.816		
PLT_{72h}	0.972	0.945-1.000	0.046	0.882 (0.777-0.950)	<0.001
Variables Included in the Model: $PLT_{72h,}$ MPV_{72h}					
Elimination methods: Backward Wald					
Confounding factors: Gender, PRISMIII, WBC, $PLT_{adm,}$ MPV_{adm}					
Model Summary: Hosmer and Lemeshov Test χ^2=13,					
.758; p=0.088; Nagelkerke R^2=0.438					
Model 3 –Binary logistic regression for MPR_{adm}					
Constant	0.008		<0.001		
MPR_{adm}	1.184	0.991-1.414	0.063	0.813 (0.694-0.900)	<0.001
Variables included in the model : MPR_{adm}					
Confounding factors: Gender, PRISMIII, WBC					
Model Summary: Hosmer and Lemeshov Test χ^2=9.703; p=0.286; Nagelkerke R^2=0.291					
Model-4: Binary logistic regression analysis for MPR_{72h}					
Constant	0.001		<0.001		
MPR_{72h}	1.537	1.081-2.184	0.017	0.856 (0.745-0.932)	<0.001
Variables included in the model : MPR_{72h}					
Confounding factors: Gender, PRISMIII, WBC, MPRadm					
Elimination methods: Backward Wald					
Model Summary: Hosmer and Lemeshov Test χ^2=8.036; p=0.430; Nagelkerke R^2=0.413					

AOR: Adjusted odds ratio; AUC: area under the curve; CI: confidence interval; MPR: MPV/PLT ratio; MPV: mean platelet volume; PLT: platelet count; PRISM III score: Pediatric risk of mortality III score; WBC: White blood cell count.

An analysis using ROC curves for each of the variables was performed, showing the cut-off point of each one with greater specificity and sensitivity. The cut off levels for PLT_{adm} and PLT_{72h} were 79 $\times 10^3/\mu L$ (sensitivity 77.3%, specificity 82.9) and 76 $\times 10^3/\mu L$ (sensitivity 86.3%, specificity 85.3), respectively. The threshold level of MPR_{72h} was >8.35 (sensitivity 72.7%, specificity 90.2%) (Fig. 1).

Discussion

The current research was a retrospective clinical study that evaluated the MPR ratio as a prognostic factor in pediatric patients with AKI. The primary outcomes of this research were that the MPR ratio was significantly higher in non-survivors and revealed to be an independent risk factor for AKI patients, even after adjusting for acceptable parameters. Specifically, we

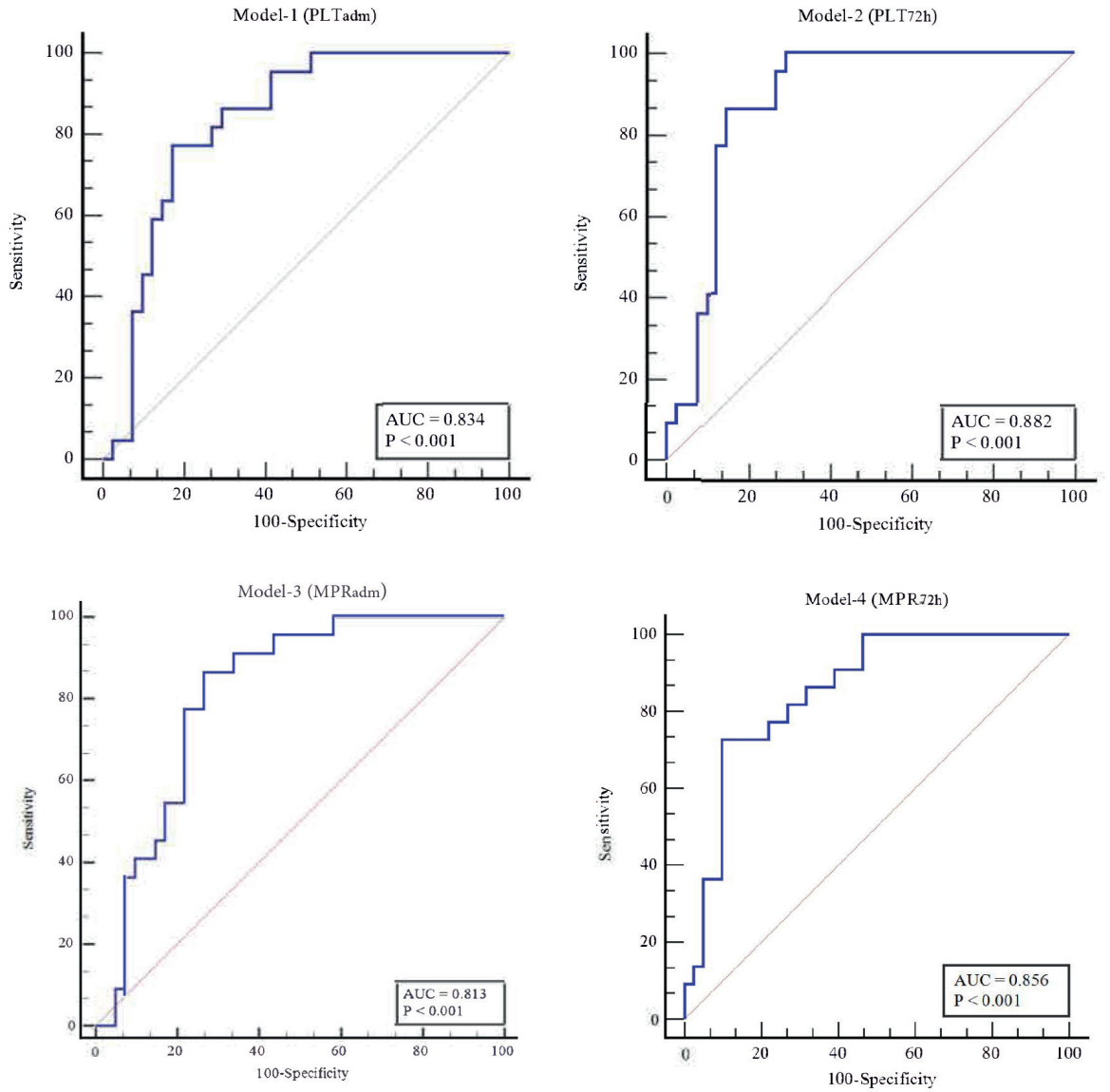

Fig. 1. The ROC curve of the logistic regression model with thrombocyte indices (PLT_{adm}, PLT_{72h}, MPR_{adm},MPR_{72h}). AUC: area under the curve; MPR: MPV/PLT ratio; PLT: platelet count.

should closely monitor AKI patients with a high MPR rate due to their elevated risk of mortality. According to our current knowledge, this is the first study to investigate the association between the MPR ratio and prognosis in children with AKI.

Despite advancements associated with its pathogenesis and management, AKI is still an independent risk factor related to mortality in critically ill patients.[19] Several investigations have suggested that the activation of platelets aggravates kidney damage.[6] Some studies have shown that thrombotic and inflammatory conditions might alter the platelet volume, larger platelets are more reactive and these alterations are related to an increase in morbidity and mortality in patients with various illnesses.[20,21] Although the mechanism of platelet volume control is not clearly known,

it is known that thrombopoietin stimulates megakaryopoiesis and results in increased ploidy and megakaryocyte size.[22] According to a study, MPV values positively correlated with thrombocyte activation, and higher MPV was a risk factor for mortality in cardiovascular diseases.[20] In another study, the authors found that among patients with chronic kidney disease, MPV levels were higher in diabetic patients than in non-diabetic patients.[23] In the current research, we excluded the majority of chronic and inflammatory diseases due to their potential impact on thrombocyte indexes. Clinical characteristics of non-survivors and survivors were compared, and MPV_{72h} level was found to be higher in non-survivors, however, MPV_{adm} was not statistically different between groups. Further analyses have shown that MPV measurements are not a risk factor for mortality. Additionally, we did not observe any association between platelet indices and laboratory markers or diagnoses at admission.

Thrombocytopenia is an independent risk factor and negative prognostic indicator in children with sepsis.[24] Activated thrombocytes interact with monocytes in the bloodstream, which causes the consumption of thrombocytes in circulation, signifying the relationship between activation of thrombocytes and suppressed thrombocyte counts during infection.[25] In a recent study of patients with rhabdomyolysis-induced AKI, low platelet count and myoglobin level were found to be independent risk parameters for kidney injury, and platelet numbers were superior to myoglobin for predicting the risk of kidney injury.[26] Research has shown that in patients with AKI receiving continuous renal replacement therapy, non-survivors had a lower platelet count.[27] Similarly, in the current study, among the non-survivors the thrombocyte count was found lower. Recent investigations recommend that the combination of thrombocyte count and MPV will present clinically more significant results than thrombocyte count or MPV alone.[9,14] Several studies have shown that total thrombocyte count was inversely associated with MPV levels.[28,29] A study hypothesized that the MPV/platelet count ratio could be a more beneficial predictor with higher sensitivity and specificity to detect deep vein thrombosis than MPV alone.[13] Research evaluating the effects of the MPV/PLT ratio on mortality in patients with pediatric septic shock presented that MPV/PLT ratios were statistically higher in the non-survivor group.[9] Although the pathophysiological mechanisms of the relationship between high MPR values and worse outcomes remain unclear, many different mechanisms have been demonstrated to explain the effect of increased inflammatory conditions on low platelet counts and high MPV levels.[10,30] Aligned with previous research findings, the present study revealed that MPV values were not statistically significant for mortality in AKI. However, lower thrombocyte counts and a higher MPR rates especially on the third day, were found to be significantly associated with mortality.

This study had several limitations. Primarily, although the medical data of all patients were collected with a high degree of precision from medical records, control of confounding parameters may be insufficient due to the retrospective design of the study. Secondly, the study's short-time calculation of MPR rates instead of long-time calculations was an important limitation. Third, the effects of drugs on platelet indices may be a factor causing bias in study results. Furthermore, the observational research design of this study did not allow for the investigation of metabolic or molecular mechanisms.

In conclusion, AKI is a common complication in critically ill patients, with adverse effects in the short and long term. Predicting and defining AKI with biomarkers is essential to reduce AKI-related mortality and morbidity. Thrombocyte counts and their indices are inexpensive and available in the complete blood count, which is usually used to evaluate the hematologic status of hospitalized patients. In this current study, the prognosis of AKI patients and the role of MPR in mortality prediction were analyzed.

The results show that MPR may be beneficial in predicting the short-term outcome of AKI. Therefore, we propose the calculation of MPR as a means to determine the prognosis of pediatric patients with AKI. Larger prospective multicenter studies are required to confirm the usefulness of the MPR as a predictive marker in children with AKI.

Ethical approval

The study was approved by Ethical Committee of Afyonkarahisar Health Sciences University Faculty of Medicine (date: 01.07.2022, number: 2022-08).

Author contribution

The authors confirm contribution to the paper as follows: study conception and design: KÇ; data collection: MÇ; analysis and interpretation of results: KÇ, MÇ; draft manuscript preparation: KÇ, MÇ. All authors reviewed the results and approved the final version of the manuscript.

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Conflict of interest

The authors declare that there is no conflict of interest.

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