RESEARCH ARTICLE

Association between the mean platelet volume-to-platelet count ratio and neurological outcomes in patients with out-of-hospital cardiac arrest

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Abstract

Objective: To evaluate the effect of platelet activity on neurological outcomes in patients experiencing cardiac arrest.

Method: The prospective, observational study was conducted from January 1 to December 31, 2023, after approval from the ethics review board of Chonnam National University Hospital, Gwang-ju, South Korea, and comprised patients of either gender aged >18 years who had out-of-hospital cardiac arrest. Serial mean platelet volume, platelet count, and mean platelet volume-to-platelet count ratio were noted at admission and on days 1, 2 and 3 after the return of spontaneous circulation. The primary endpoint was neurological outcome at 3 months. A poor neurological outcome was defined as cerebral performance categories 3, 4 and 5, while categories 1 and 2 were taken as a good neurological outcome. Data was analysed using SPSS 18.

Results: Of the 86 patients with median age 62.0 years (interquartile range: 51.8-70.0 years), 61(75.5%) were males. Overall, 57(66.3%) patients had suffered cardiac arrest, while 30(34.9%) had shockable rhythms at sudden collapse. The median time between collapse and return of spontaneous circulation was 32.5 min (interquartile range: 21.8-44.0 minutes). Of the total, 62(72.1%) patients had a poor neurological outcome. The mean platelet volume-to-platelet count ratio at day 3 (adjusted odds ratio: 0.789; 95% confidence interval: 0.655-0.951; *p*=0.013) was independently associated with poor neurological outcomes.

Conclusion: After return of spontaneous circulation, the mean platelet volume-to-platelet count ratio at day 3 may be a useful element for assessing the neurological outcomes in patients who having suffered out-of-hospital cardiac arrest and undergone targetted temperature management.

Keywords: Mean platelet volume, Platelet count, Out-of-hospital cardiac arrest, Targeted temperature management.

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Introduction

Out-of-hospital cardiac arrest (OHCA) is considerably associated with mortality and neurological impairment.[1] Among OHCA patients, 29.7% achieved the return of spontaneous circulation (ROSC) and 8.8% survived before being discharged from hospital in a study.[2] Of these 8.8% patients, only 10-30% had good neurological outcomes.[2] Current guidelines recommend delaying neurological prognosis assessment until 72 hours after ROSC in unconscious patients having suffered cardiac arrest.[3] However, families in hospitals want to know the patients' survival and neurological prognosis as quickly as possible. Therefore, various factors related to the neurological prognosis of these patients have been studied, but accurate predictions remain challenging. Therefore, current guidelines suggest using various tools simultaneously to help predict the patient's prognosis.[3]

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Trends in platelet count (PLT) and related factors are associated with the prognosis of critical diseases, such as sepsis, trauma and myocardial infarction (MI).[4-6] A reperfusion injury caused by ROSC following whole-body ischaemia during cardiac arrest causes systemic inflammatory responses and sepsis-like syndrome.[7] As ischaemia and reperfusion persist, tissues fail to receive adequate oxygen supply, and various body mechanisms become affected.[8] One of these mechanisms is the coagulation pathway, which occurs because of blood stasis and endothelial cell damage in the body in sepsis-like syndrome.[8,9] Disseminated intravascular coagulation may result from this unusual change in coagulation; consequently, microvascular alterations, organ failure, and death occur.[8,9]

The inflammatory response caused by sepsis-like syndrome changes platelet size, which can be detected by evaluating the mean platelet volume (MPV) via complete blood count (CBC) analysis.[10,11] Depending on the balance between creation and extinction, MPV can vary. Therefore, many studies have shown the relationship between MPV and prognosis in various diseases.[12,13] Additionally, MPV increases in patients who have OHCA and poor neurological outcomes.[14] This increase is associated with sepsis-like syndrome during ROSC after ischaemia.[14]

To our knowledge, studies have yet to determine whether the MPV-to-PLT ratio (MPR) could help assess disease severity and neurological prognosis in OHCA patients who undergo targetted temperature management (TTM). The current study was planned to fill the gap in literature by examining the effect of MPR on neurological outcomes in such patients. It was hypothesised that an increase in MPV and a decrease in PLT in the first 72 hours after admission would be related to poor neurological outcomes in these patients.

Patients and Methods

The prospective, observational study was conducted from January 1 to December 31, 2023, after approval from the ethics review board of Chonnam National University Hospital, Gwang-ju, South Korea, and comprised OHCA patients of either gender aged >18 years, and underwent TTM. Written informed consent was obtained from the patients or their next of kin.

The sample size was calculated based on the effect size reported earlier[14] using G*Power v3.1.7.[15] Power was expressed as 1–β, where β was a type II error; typically 80%. The effect size was 0.636 and power was 90% at alpha (α) value of 0.05. The participants were selected using a non-probability purposive sampling technique. Those excluded were patients not meeting the age criterion, patients who did not complete TTM because of either hospital transfer or death, and patients who did not undergo blood sampling.

Data collected from electronic medical records included age, gender pre-existing illness, bystander cardio-pulmonary resuscitation (CPR), witnessed collapse, first on-scene monitored rhythm, interval from collapse to ROSC, laboratory findings after ROSC, including partial pressure of carbon dioxide ($PaCO_2$), partial pressure of oxygen (PaO_2), lactate level and glucose level. Within 1 day of admission, sequential organ failure assessment (SOFA) scores were calculated.[16]

MPVs and PLTs were measured at admission and on days 1, 2, and 3 after ROSC. MPV was divided by PLT to calculate MPRs at each time point. Neurological outcome was estimated at 3 months after ROSC through phone interviews by using the cerebral performance category (CPC) scale.[17] The primary endpoint was neurological outcome. A poor neurological outcome was defined as CPC categories 3-5, while categories 1-2 were taken as a good neurological outcome.

Data was analysed using SPSS 18 and MedCalc 19. Categorical variables were reported as frequencies and percentages, while continuous variables were expressed as medians with interquartile range (IQR) after data normality was checked using Shapiro-Wilk test. Categorical group data was analysed using chi-square test. Continuous intergroup data was compared using Mann-Whitney U test. Mixed-model analysis was conducted to compare MPV, PLT and MPR with neurological outcome groups. Post-hoc analysis was performed by introducing pairwise Mann-Whitney U test with Bonferroni correction for poor outcomes. The association of MPV, PLT and MPR for poor outcome was examined via multivariable logistic analysis. Variables with $p<0.20$ were included in the multivariable regression model after univariable comparisons. A backward stepwise approach was used to set the final adjusted regression model, sequentially removing variables with a threshold of $p>0.10$. Shockable rhythm and interval from cardiac arrest to ROSC were selected as adjusted variables. MPVs, PLTs, and MPRs at admission and on days 1, 2 and 3 after ROSC were included in the final model and analysed separately. Model calibration was evaluated using Hosmer-Lemeshow goodness-of-fit C-statistics, with $p<0.05$ indicating good calibration. Logistic regression analysis was performed, and results were presented as 95% confidence interval (CI) and adjusted odds ratio (aOR). The predictive performances of MPVs, PLTs and MPRs on T0, T1, T2 and T3 for poor neurological outcomes were assessed with the help of receiver operating characteristic (ROC) curves by examining the area under the curve (AUC). Statistical significance was set at two-sided $p<0.05$.

Results

Of the 112 OHCA patients, 20(17.85%) died before TTM, 5(4.46%) were aged <18 years, and complete data was not available for 1(0.89%). As such, the final sample had 86(76.80%) patients with median age 62.0 years (IQR: 51.8-70.0 years), and 61(75.5%) of them were males. Overall, 57(66.3%) patients had suffered cardiac arrest, while 30(34.9%) had shockable rhythms at sudden collapse. The median time between collapse and ROSC was 32.5 minutes (IQR: 21.8-44.0 minutes). Of the total, 62(72.09%) patients had a poor neurological outcome, while 24(27.90%) had a good neurological outcome. The poor outcome group had higher proportions of non-shockable rhythm, higher proportions of cardiac aetiology, and longer intervals from collapse to ROSC than the good outcome group, and after ROSC, the lactate level, $PaCO_2$ and SOFA score of the poor outcome group were higher than those of the good outcome group (Table 1).

Neurological outcomes post-ROSC showed that while PLT ($p<0.001$) and MPR ($p<0.001$) decreased significantly over time, MPV was not significantly different ($p>0.05$). No significant interactions were observed between the

Table-1: Comparison of baseline characteristics according to neurological outcomes at 3 months.

Variables	Total (n = 86)	Good (n = 24)	Poor (n = 62)	*p*-value
Demographics				
Age (years)	62.0 (51.8–70.0)	59.0 (46.5–69.8)	62.5 (52.8–70.3)	0.405
Male, n (%)	61 (70.9)	17 (70.8)	44 (71.0)	>0.999
Pre-existing illness, n (%)				
Coronary artery disease	7 (8.1)	4 (16.7)	3 (4.8)	0.174
Congestive heart failure	5 (5.8)	2 (8.3)	3 (4.8)	0.914
Hypertension	43 (50.0)	13 (54.2)	30 (48.4)	0.810
Diabetes	23 (26.7)	6 (25.0)	17 (27.4)	>0.999
Renal impairment	13 (15.1)	3 (12.5)	10 (16.1)	0.932
Cerebrovascular accident	6 (7.0)	0 (0.0)	6 (9.7)	0.268
Liver cirrhosis	3 (3.5)	0 (0.0)	3 (4.8)	0.659
Cardiac arrest characteristics				
Witnessed collapse, n (%)	57 (66.3)	19 (79.2)	38 (61.3)	0.187
Bystander CPR, n (%)	58 (67.4)	17 (70.8)	41 (66.1)	0.872
Shockable rhythm, n (%)	30 (34.9)	17 (70.8)	13 (21.0)	<0.001
Cardiac aetiology, n (%)	43 (50.0)	18 (75.0)	25 (40.3)	0.008
Time to ROSC, min	32.5 (21.8–44.0)	19.5 (13.3–25.8)	37.5 (27.8–47.3)	<0.001
After ROSC				
Lactate, mmol/L	8.3 (4.6–11.6)	4.8 (3.4–8.5)	9.4 (5.3–12.9)	0.002
Glucose, mg/dL	252 (193–316)	261 (181–305)	252 (199–325)	0.408
PaO_2, mmHg	185 (118–233)	198 (126–321)	180 (113–224)	0.336
$PaCO_2$, mmHg	45 (34–66)	36 (31–42)	54 (40–69)	<0.001
SOFA score	11 (9–13)	10 (8–12)	12 (10–14)	0.006

CPR: Cardiopulmonary resuscitation, ROSC: Return of spontaneous circulation, PaO_2: Partial pressure of oxygen, $PaCO_2$: partial pressure of carbon dioxide, SOFA: Sequential organ failure assessment.

Table-2: Comparison of MPV, PLR and MPR after ROSC according to neurological outcomes at 3 months.

Variables	Total (n = 86)	Good (n = 24)	Poor (n = 62)	*p*-value
MPV, fL				
At admission	9.8 (9.1–10.5)	9.7 (9.1–10.4)	9.9 (9.1–10.6)	0.449
On day 1 after ROSC	9.8 (9.1–10.6)	9.6 (9.1–10.2)	10.0 (9.0–10.7)	0.260
On day 2 after ROSC	10.2 (9.5–11.1)	9.9 (9.3–10.4)	10.4 (9.7–11.2)	0.023
On day 3 after ROSC	10.5 (9.9–11.0)	10.1 (8.9–10.6)	10.7 (10.1–11.1)	0.004
PLT, $\times 10^9$/L				
At admission	211 (161–256)	234 (202–286)	194 (152–245)	0.010
On day 1 after ROSC	153 (115–190)	171 (146–203)	147 (107–174)	0.015
On day 2 after ROSC	141 (96–175)	165 (151–196)	126 (91–167)	< 0.001
On day 3 after ROSC	125 (84–156)	151 (131–175)	109 (77–145)	< 0.001
MPR				
At admission	21.9 (16.3–26.2)	23.2 (20.2–31.0)	20.7 (15.1–25.3)	0.025
On day 1 after ROSC	16.0 (11.8–19.3)	18.0 (15.2–21.2)	15.2 (10.2–18.0)	0.024
On day 2 after ROSC	13.6 (9.5–17.8)	17.0 (14.3–20.2)	11.8 (9.0–16.7)	< 0.001
On day 3 after ROSC	12.0 (8.2–15.8)	16.2 (13.1–18.7)	10.3 (7.4–14.1)	< 0.001

ROSC: Return of spontaneous circulation, MPV: Mean platelet volume, PLT: Platelet count, MPR: Mean platelet volume-to-platelet count ratio.

neurological outcome groups and elapsed time for MPV, PLT and MPR (p>0.05). Subgroup analysis for MPV, PLT and MPR values was also done at T0, T1, T2 and T3 (Table 2).

After adjustments were made for confounders, MPV on day 3 (aOR, 2.033; 95% CI, 1.028–4.022) was found to be independently associated with poor prognosis (Table 3). Additionally, MPR on day 3 (aOR, 0.789; 95% CI, 0.655–0.951) was independently related to poor outcomes. PLT at

Table-3: Multivariable logistic regression analysis of MPV, PLT and MPR after ROSC for poor neurological outcomes at 3 months.

Variable	Adjusted OR (95% CI)[a]	*p*-value	Hosmer-Lemeshow test χ^2	Hosmer-Lemeshow test *p*-value
MPV, fL				
At admission	1.162 (0.573–2.359)	0.677	6.742	0.565
On day 1 after ROSC	0.972 (0.863–1.095)	0.638	6.933	0.544
On day 2 after ROSC	1.623 (0.853–3.088)	0.140	6.682	0.571
On day 3 after ROSC	2.033 (1.028–4.022)	0.041	3.474	0.901
PLT, $\times 10^9$/L				
At admission	0.996 (0.985–1.006)	0.401	6.742	0.565
On day 1 after ROSC	0.995 (0.980–1.009)	0.469	8.534	0.383
On day 2 after ROSC	0.991 (0.976–1.005)	0.194	10.481	0.233
On day 3 after ROSC	0.981 (0.962–1.001)	0.059	8.369	0.398
MPR				
At admission	0.965 (0.888–1.049)	0.402	6.379	0.605
On day 1 after ROSC	0.976 (0.877–1.086)	0.655	6.133	0.632
On day 2 after ROSC	0.903 (0.796–1.025)	0.115	8.824	0.357
On day 3 after ROSC	0.789 (0.655–0.951)	0.013	9.622	0.293

Each MPV, PLT, and MPR variable was individually entered into the final model and analysed separately but was not adjusted for other variables; [a] Adjusted for shockable rhythm and interval from collapse to ROSC; MPV: Mean platelet volume, PLT: Platelet count, MPR: Mean platelet volume-to-platelet count ratio, ROSC: Return of spontaneous circulation, OR: Odds ratio, CI: Confidence interval.

all time-points was not linked to poor outcomes (p>0.05). All models demonstrated good calibration based on the Hosmer-Lemeshow test results (Table 3).

The AUCs for MPV, PLR and MPR values at day 3 for predicting poor outcomes were 0.701 (95% CI: 0.592-0.795), 0.767 (95% CI: 0.664-0.852) and 0.805 (95% CI: 0.705-0.882), respectively. The AUCs of MPR were not significantly different from those of MPV and PLT (p>0.05).

Discussion

Among patients who had OHCA and TTM after ROSC, those with poor neurological prognosis had higher MPV and lower PLR and MPR than those with good outcomes, in the current prospective, observational study. In terms of neurological outcomes, MPV and MPR on day 3 were associated with poor prognosis. Furthermore, MPR on day 3 exhibited a good performance in predicting poor prognosis.

Thrombocytopenia is associated with the prognosis of critically ill patients.[18,19] In one review article, 8.3-67.6% patients had thrombocytopenia at the time of intensive care unit (ICU) admission, and 13-44.1% had incident thrombocytopenia during ICU stay.18 In another study of OHCA patients, those with poor neurological outcomes after ROSC had lower PLT than patients with good neurological outcomes, and failure to improve platelets within 1 week was associated with poor neurological outcomes.[19] Likewise, in the present study, PLT of patients with a poor neurological outcome was lower than that of the patients with a good neurological outcome after ROSC.

The ischaemia and reperfusion damage observed in post-cardiac arrest syndrome (PCAS) after OHCA is a systemic inflammatory reaction similar to that found in sepsis.[20] In comatose patients who had OHCA and were treated with TTM, systemic inflammation was associated with endothelial activation and damage.[21] For 24 hours, the temperature of all patients with OHCA was decreased to 33°C; although no significant bleeding complications were observed, hypothermia during TTM may cause coagulopathy.[21]

Among blood tests, MPV accurately measures the average platelet size and reflects platelet reactivity.[22] Platelet volume may reflect platelet activity.[22,23] A higher MPV is associated with poor prognosis in patients with arterial and venous thrombosis, cerebrovascular disease, coronary artery disease, chronic inflammatory diseases and sepsis[12,13,24,25] because increased MPV is associated with a higher tendency of mural thrombosis or left ventricular failure.[26] Poor neurological outcomes were observed in patients who suffered from OHCA, received TTM and had elevated MPV.[20] Bro-Jeppesen et al. found that the permeability of the blood-brain barrier is affected during OHCA, and systemic inflammation induced by ischaemic-reperfusion injury may potentially exacerbate brain injury.[20] They also indicated that MPV significantly differed between the groups of patients with good and poor neurological outcomes from the time of hospital admission.[14] However, the current study demonstrated a difference from 48 hours after ROSC because the rate of witnessed collapse and bystander CPR was higher. These factors likely influenced the time until reperfusion injury after ROSC.

However, MPV standardisation becomes difficult because of the time from blood collection to MPV measurement, the anticoagulant used, and the blood analyser used for measurement. MPV can also be affected by several cancers, drugs, autoimmune diseases, general conditions and alcohol. Therefore, the MPR is associated with the prognosis of diseases, such as cancer, infection and MI.[27-29]

Prognostic tools used to predict patient outcomes post-ROSC include neuron-specific enolase (NSE), S100 calcium-binding protein B (S100B), and neuroimaging. NSE levels may increase even with minimal haemolysis, complicating the distinction between brain injury and haemolysis.[30] S100B can be elevated in other conditions, such as trauma, stroke and seizures, potentially leading to false positive results.[31] Neuroimaging is relatively costly and may yield suboptimal results if the patient is immobilised owing to myoclonus or seizure. Additionally, most patients after ROSC require ventilators and high-concentration oxygen, increasing the risk of hypoxia during transport for imaging. However, MPR obtained through CBC is rapid, inexpensive and does not require patient transport. It can also be measured serially to monitor clinical trends. Although the current study showed that MRP on day 3 exhibited good performance for poor prognosis, the evidence is insufficient to support its use as a standalone prognostic marker. Where NSE, S100B, or neuroimaging are unavailable owing to cost or other constraints, MPR can serve as an alternative or adjunctive tool.

In a study on OHCA patients, platelets decreased from the time of admission to 70 hours after TTM, and platelet aggregation levels were below the normal range.[19] In the present study, PLT was also reduced on day 3 after ROSC, and the MPR on day 3 was most statistically significant in explaining poor 3-month neurological outcomes. This finding occurred because, as demonstrated in other studies, systemic inflammatory syndrome during ROSC promotes platelet aggregation and consumption; as a result, PLT decreases during the first 3 days of PCAS and during recovery 4 days after ROSC as the systemic inflammatory response subsides.[19]

The current study has several limitations. Since the study was done at a single centre and was observational in nature, the results are not widely generalisable. Additional multicentre studies are needed to complement the current findings. Also, the sample size was relatively small which has limited the generalisability of the findings. Future large-scale studies are recommended. Besides, interrupted TTM because of transfer to other medical centres or death might have led to selection bias, which may have influenced the findings. Further, the study did not investigate the drugs affecting PLT, like aspirin, antiplatelet medication and glycoprotein IIb/IIIa receptor inhibitors may have induced PLT dysfunction. Lastly, prognosis was only analysed by examining MPR within 72 hours after ROSC. Therefore, the relationship between MPR after 72 hours and neurological prognosis could not be examined.

Conclusion

In terms of predicting poor neurological outcomes in patients who had OHCA, low MPR on day 3 after ROSC was found to be independently associated, and might be useful for assessing the neurological outcome.

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