Acute and Critical Care

Acute and Critical Care 2022 Novemebr 37(4):601-609
https://doi.org/10.4266/acc.2022.00542

| pISSN 2586-6052 | eISSN 2586-6060

Association between C-reactive protein-to-albumin ratio and 6-month mortality in out-of-hospital cardiac arrest

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Background: The inflammatory response that occurs following cardiac arrest can determine the long-term prognosis of patients who survive out-of-hospital cardiac arrest. We evaluated the correlation between C-reactive protein-to-albumin ratio (CAR) following cardiac arrest and long-term mortality.

Methods: The current retrospective observational study examined patients with post-cardiac arrest syndrome (PCAS) treated with targeted temperature management at a single tertiary care hospital. We measured CAR at four time points (at admission and then 24 hours, 48 hours, and 72 hours after) following cardiac arrest. The primary outcome was the patients' 6-month mortality. We performed multivariable and area under the receiver operating characteristic curve (AUC) analyses to investigate the relationship between CAR and 6-month mortality.

Results: Among the 115 patients, 52 (44.1%) died within 6 months. In the multivariable analysis, CAR at 48 hours (odds ratio [OR], 1.130; 95% confidence interval [CI], 1.027–1.244) and 72 hours (OR, 1.241; 95% CI, 1.059–1.455) after cardiac arrest was independently associated with 6-month mortality. The AUCs of CAR at admission and 24, 48, and 72 hours after cardiac arrest for predicting 6-month mortality were 0.583 (95% CI, 0.489–0.673), 0.622 (95% CI, 0.528–0.710), 0.706 (95% CI, 0.615–0.786), and 0.762 (95% CI, 0.675–0.835), respectively.

Conclusions: CAR at 72 hours after cardiac arrest was an independent predictor for long-term mortality in patients with PCAS.

Key Words: albumin; C-reactive protein; cardiac arrest; mortality; prognosis

Original Article

Received: April 20, 2022
Revised: June 28, 2022
Accepted: June 29, 2022

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INTRODUCTION

Post-cardiac arrest syndrome (PCAS) is still often fatal due to complications such as multiorgan failure or neurological damage [1,2]. This outcome is thought to be due to the damage caused by the systemic inflammation that occurs during the whole-body ischemic response that takes place in cardiac arrest. Tissue reperfusion injury, which is defined as an ischemia-reperfusion injury after the return of spontaneous circulation (ROSC), exacerbates the tissue damage [3].

The systemic inflammatory response can be evaluated using various laboratory markers. Several studies showing the statistical significance between laboratory markers and long-

term mortality after out-of-hospital cardiac arrest (OHCA) have been conducted [4-8]. The neutrophil-to-lymphocyte ratio is a representative laboratory marker that has fair performance in predicting long-term mortality and neurological outcomes after OHCA [4]. One previous study revealed that the peak procalcitonin level at 24–48 hours after cardiac arrest can help predict neurological outcomes. The usefulness of these inflammatory markers has primarily focused on values within 48 hours [5]. In addition, C-reactive protein (CRP) levels, which are normally low, increase after ROSC, and many studies have examined their association with mortality and neurologic outcomes [6-8]. Conversely, various recent studies have reported that the CRP-to-albumin ratio (CAR) aided in determining the prognosis in cases of Guillain-Barré syndrome, traumatic brain injury, myocardial infarction, and stroke [9-12]. The patient's body temperature is lowered during targeted temperature management (TTM), and the degree of inflammation decreases accordingly as reported by a study that maintained mild hypothermia for 72 hours [13]. The actual inflammatory response is considered to occur in the rewarming or post-rewarming period after TTM. Thus, the factors that can cause an inflammatory response after cardiac arrest are considered related after 48 hours.

We hypothesized that the mortality of patients with PCAS treated with TTM would be related to CAR beyond 48 hours after cardiac arrest. Therefore, we investigated the CAR at 72 hours after cardiac arrest to determine 6-month mortality and compared it with CARs at admission and 24 hours and 48 hours after cardiac arrest.

KEY MESSAGES

- C-reactive protein-to-albumin ratio (CAR) is an effective marker of a systemic inflammatory response, including post-cardiac arrest syndrome (PCAS).
- Targeted temperature management can affect the systemic inflammatory response in the body by lowering the temperature.
- CAR more than 48 hours after cardiac arrest was an independent predictor for 6-month mortality in patients with PCAS.

MATERIALS AND METHODS

Study Design and Population

The present study was retrospective and observational in design and included patients with PCAS treated with TTM at Chonnam National University Hospital between January 2018 and December 2020. We included patients with PCAS aged ≥18 years who were comatose following TTM. The exclusion criteria were as follows: patients who discontinued TTM due to transfer to other hospitals or passing away, those who underwent TTM with a temperature other than 33°C, those who needed support (such as continuous hemodialysis and/or percutaneous cardiopulmonary support during PCAS care), and those with missing data. This study was approved by the Institutional Review Board of Chonnam National University Hospital (No. CNUH-2021-141). The requirement of informed consent was waived due to the retrospective nature of the present study.

Targeted Temperature Management

We maintained the core body temperature of patients at 33°C for 24 hours. We continued to administer remifentanil and midazolam for sedation during TTM to enhance its efficiency and reduce the brain's metabolism. We observed subclinical seizures in real time using amplitude-integrated electroencephalography.

Data Collection

Data related to the following parameters were obtained from the patients' hospital records: age, sex, underlying disease, first on-scene monitored rhythm, time from sudden cardiac arrest to ROSC, cardiac arrest etiology, witnessed collapse, bystander cardiopulmonary resuscitation (CPR), and calculated the Sequential Organ Failure Assessment (SOFA) score within 24 hours of admission. In addition, serum laboratory results, such as lactate and glucose levels, artery blood gas analysis results (e.g., partial pressure of oxygen [PaO_2] and partial pressure of carbon dioxide [$PaCO_2$] were obtained within 24 hours after admission.

Blood samples for assessing albumin and CRP were taken at admission and again at 24, 48, and 72 hours after cardiac arrest. The high sensitivity nephelometric method (Dade Behring; Marburg, Germany) was used to measure CRP level, which was detected from 0.2 mg/L. CAR was obtained by dividing CRP level by albumin level. Albumin level was determined via enzymatic assay using an automatic analyzer (Hitachi-7600; Hitachi, Tokyo, Japan). We assessed 6-month mortality through telephone interviews with the patients or their caregivers. The primary outcome was 6-month mortality, whereas the secondary outcome was in-hospital mortality.

Statistical Analysis

We presented the categorical variables as frequencies and percentages, whereas continuous variables are shown as the mean±standard deviation or the median and interquartile range, depending on the Shapiro-Wilk test results. The categorical variables of the groups were comparatively analyzed using the chi-square test with a continuity correction in 2×2 tables. Continuous variables were compared between the groups using independent t-tests or Mann-Whitney U-tests. Repeated-measures analysis of variance was used to compare CRP level, albumin level, and CAR between survivors and non-survivors within 72 hours after cardiac arrest. Post-hoc analysis was performed using pairwise Mann-Whitney U-tests with a Bonferroni correction between survivor and non-survivor groups.

We performed a multivariable logistic regression analysis to identify the predictive force of CAR on 6-month or in-hospital mortality. Variables with P-values <0.20 on univariable comparisons were included in the multivariable regression model. We used a backward stepwise approach that sequentially eliminated variables with a threshold of P >0.10 to build a final adjusted regression model. Lastly, the presence of a shockable rhythm and bystander CPR were selected as adjusted variables (Supplementary Tables 1 and 2). CAR values at each time point were included in the final model. The results of the logistic regression analysis are presented as the odds ratio (OR) and 95% confidence interval (CI). We assessed the predictive performance of CAR to determine 6-month or in-hospital mortality using the area under the receiver operating characteristic (ROC) curve (AUC). The comparison of dependent ROC curves was performed using the method proposed by DeLong et al. [14]. All analyses were carried out using PASW version 18.0 (SPSS Inc., Chicago, IL, USA) and MedCalc version 19.0 (MedCalc Software, bvba, Ostend, Belgium). Statistical significance was set at P<0.05 (two-sided).

RESULTS

Patient Characteristics

Among 115 cardiac arrest patients, 52 died within 6 months (44.1%) (Figure 1). The median age of the patients with OHCA was 58.7 years, and 91 male patients (77.1%) were included. In total, 81 collapses (68.6%) were witnessed by bystanders; 52 patients (44.1%) had a shockable rhythm, and the mean value of time from cardiac arrest to ROSC was 23.5 minutes (15.8–39.3 minutes).

Six-month mortality results indicated that non-survivors had a lower incidence of witnessed collapse and bystander CPR, as well as a higher incidence of a non-shockable rhythm and a noncardiac etiology. They also exhibited a prolonged time to ROSC compared with survivors. Non-survivors exhibited increased levels of lactate and $PaCO_2$ after ROSC compared with survivors (Table 1). In-hospital mortality results indicated that non-survivors were older, had lower incidences of a shockable rhythm, and a more prolonged time to ROSC than survivors. Non-survivors exhibited an increased $PaCO_2$ level following ROSC compared with survivors (Table 1).

CRP Level, Albumin Level, and CAR According to 6-Month or In-hospital Mortality

Six-month mortality results revealed that albumin levels at admission and 24, 48, and 72 hours after cardiac arrest were lower in non-survivors than in survivors. CRP level and CAR of non-survivors at 24, 48, and 72 hours after cardiac arrest were higher than those of survivors (Table 2). In-hospital mortality results revealed that albumin levels at admission and at 48and 72 hours after cardiac arrest were lower among non-survivors than among survivors. CRP level and CAR of non-survivors at 48and 72 hours after cardiac arrest were higher than those of survivors (Table 2).

CRP levels and CAR increased, and albumin levels decreased within 72 hours after cardiac arrest (Figure 2). Interactions between both 6-month mortality and in-hospital mortality and changes of CRP level and CAR over time were significant, but those for albumin level was not significant (Figure 2). Post-hoc

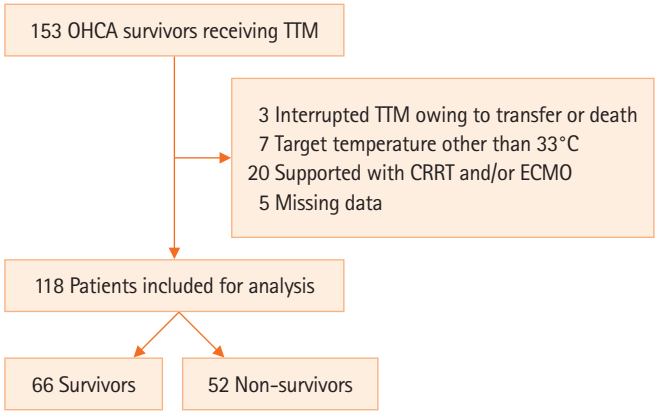

Figure 1. Schematic diagram showing the number of patients in the present study. OHCA: out-of-hospital cardiac arrest; TTM: target temperature management; CRRT: continuous renal replacement therapy; ECMO: extracorporeal membrane oxygenation.

Table 1. Baseline characteristics by 6-month prognosis

Variable	Total (n=118)	6-Month mortality			In-hospital mortality		
		Survivor (n=66)	Non-survivor (n=52)	P-value	Survivor (n=78)	Non-survivor (n=40)	P-value
Demographics							
Age (yr)	58.7±15.3	56.3±14.5	61.8±15.9	0.063	56.5±14.6	63.0±16.1	0.029
Male	91 (77.1)	53 (80.3)	38 (73.1)	0.480	61 (78.2)	30 (75.0)	0.872
Pre-existing illness							
Coronary artery disease	18 (15.3)	8 (12.1)	10 (19.2)	0.419	10 (12.8)	8 (20.0)	0.449
Hypertension	45 (38.1)	23 (34.8)	22 (42.3)	0.524	27 (34.6)	18 (45.0)	0.369
Diabetes	33 (28.0)	14 (21.2)	19 (36.5)	0.102	18 (23.1)	15 (37.5)	0.151
Renal impairment	2 (1.7)	1 (1.5)	1 (1.9)	1.000	2 (2.6)	0	0.789
Cerebrovascular accident	7 (5.9)	2 (3.0)	5 (9.6)	0.267	4 (5.1)	3 (7.5)	0.917
Cardiac arrest characteristics							
Witnessed collapse	81 (68.6)	52 (78.8)	29 (55.8)	0.013	56 (71.8)	25 (62.5)	0.412
Bystander CPR	79 (66.9)	51 (77.3)	28 (53.8)	0.013	57 (73.1)	22 (55.0)	0.077
Shockable rhythm	52 (44.1)	43 (65.2)	9 (17.3)	<0.001	46 (59.0)	6 (15.0)	<0.001
Cardiac etiology	81 (68.6)	52 (78.8)	29 (55.8)	0.013	58 (74.4)	23 (57.5)	0.097
Time to ROSC (min)	23.5 (15.8–39.3)	20.0 (14.0–30.0)	29.0 (20.5–44.8)	0.002	20.0 (14.0–33.5)	29.0 (22.0–44.8)	0.002
Clinical characteristics after ROSC							
Lactate (mmol/L)	7.0 (4.7–10.3)	6.5 (4.1–9.3)	8.6 (5.8–11.7)	0.040	6.6 (4.2–9.4)	8.8 (5.8–11.5)	0.062
Glucose (mg/dl)	262 (198–326)	254 (198–307)	277 (193–350)	0.367	254 (191–308)	288 (205–359)	0.196
PaO_2 (mm Hg)	128 (89–211)	116 (83–211)	152 (97–222)	0.102	116 (84–199)	162 (97–238)	0.099
$PaCO_2$ (mm Hg)	43 (33–60)	37 (32–46)	53 (35–68)	0.002	38 (33–50)	54 (35–68)	0.011
SOFA score	11 (9–12)	10 (8–12)	11 (9–12)	0.104	10 (8–12)	11 (9–12)	0.063

Values are presented as mean±standard deviation, number (%), or median (interquartile range).
CPR: cardiopulmonary resuscitation; ROSC: restoration of spontaneous circulation; PaO_2: partial pressure of oxygen; $PaCO_2$: partial pressure of carbon dioxide; SOFA: Sequential Organ Failure Assessment.

Table 2. CRP level, albumin level, and CAR according to 6-month mortality or in-hospital mortality

Variable	Total (n=118)	6-Month mortality			In-hospital mortality		
		Survivor (n=66)	Non-survivor (n=52)	P-value	Survivor (n=78)	Non-survivor (n=40)	P-value
At admission							
CRP (mg/dl)	0.2 (0.1–0.6)	0.2 (0.0–0.4)	0.2 (0.1–0.6)	0.264	0.2 (0.0–0.5)	0.2 (0.1–0.7)	0.370
Albumin (g/dl)	3.6 (3.3–3.9)	3.7 (3.4–4.1)	3.5 (3.1–3.8)	0.002	3.7 (3.4–4.0)	3.4 (3.0–3.6)	<0.001
CAR	0.0 (0.0–0.1)	0.0 (0.0–0.1)	0.1 (0.0–0.2)	0.171	0.0 (0.0–0.1)	0.1 (0.0–0.3)	0.239
At 24 hours after CA							
CRP (mg/dl)	6.1 (3.1–9.1)	5.7 (2.3–7.8)	6.7 (4.5–11.4)	0.021	5.9 (2.8–8.4)	6.7 (4.7–12.2)	0.067
Albumin (g/dl)	3.2 (2.9–3.5)	3.3 (3.0–3.6)	3.1 (2.8–3.5)	0.020	3.3 (3.0–3.5)	3.1 (2.8–3.5)	0.051
CAR	1.9 (0.9–3.0)	1.7 (0.8–2.7)	2.2 (1.3–3.6)	0.024	1.8 (0.9–2.7)	2.1 (1.3–3.8)	0.094
At 48 hours after CA							
CRP (mg/dl)	13.1 (8.7–18.6)	10.8 (8.2–15.5)	16.3 (12.2–21.8)	<0.001	11.4 (8.4–16.3)	16.8 (12.2–21.8)	0.004
Albumin (g/dl)	3.0 (2.7–3.3)	3.2 (2.9–3.4)	2.8 (2.6–3.1)	<0.001	3.1 (2.8–3.4)	2.8 (2.6–3.1)	<0.001
CAR	4.5 (2.9–6.5)	3.6 (2.6–5.4)	5.6 (3.7–8.1)	<0.001	3.9 (2.6–5.6)	5.9 (3.6–8.6)	<0.001
At 72 hours after CA							
CRP (mg/dl)	12.0 (8.2–17.5)	9.4 (7.1–14.0)	15.6 (11.3–25.0)	<0.001	10.4 (7.3–15.3)	15.8 (11.4–26.4)	<0.001
Albumin (g/dl)	3.0 (2.7–3.2)	3.2 (2.9–3.3)	2.7 (2.6–3.0)	<0.001	3.1 (2.8–3.3)	2.7 (2.5–3.1)	<0.001
CAR	4.1 (2.6–6.1)	3.1 (2.3–4.7)	5.6 (4.0–8.5)	<0.001	3.4 (2.4–5.1)	5.9 (4.0–9.1)	<0.001

Values are presented as median (interquartile range).
CRP: C-reactive protein; CAR: C-reactive protein-to-albumin ratio; CA: cardiac arrest.

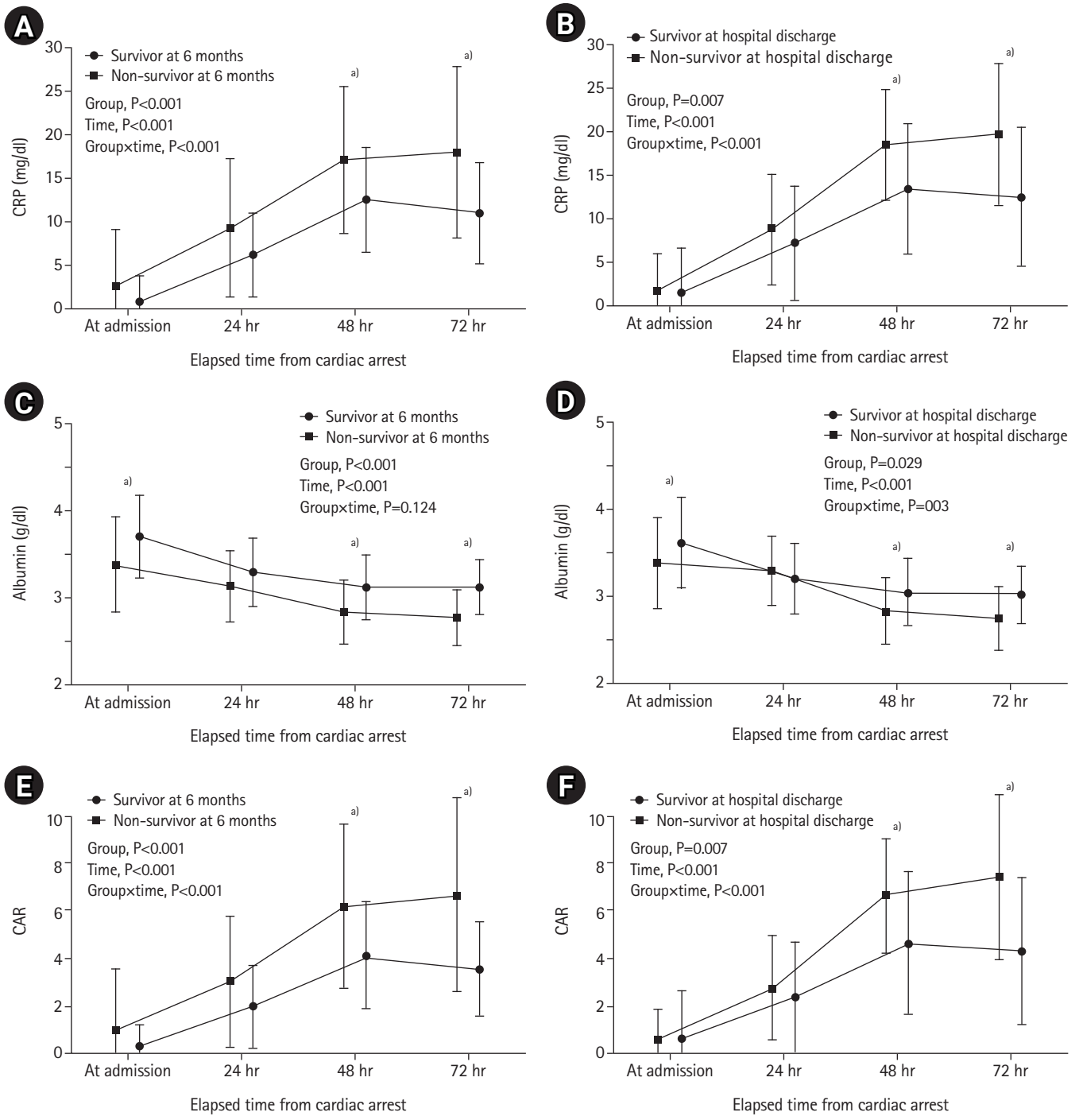

Figure 2. Repeated-measures analysis of variance of C-reactive protein (CRP), albumin, and C-reactive protein-to-albumin ratio (CAR) for 6-month mortality and in-hospital mortality 72 hours after cardiac arrest. (A) CRP level and 6-month mortality, (B) CRP level and in-hospital mortality, (C) albumin level and 6-month mortality, (D) albumin level and in-hospital mortality, (E) CAR and 6-month mortality, and (F) CAR and in-hospital mortality. a) P<0.013.

analysis showed that albumin level was different at admission and at 48 and 72 hours after cardiac arrest between survivors and non-survivors irrespective of in-hospital and 6-month mortality (Figure 2). Post-hoc analysis showed that CRP level and CAR were different at 48 and 72 hours after cardiac arrest between survivors and non-survivors irrespective of in-hospital and 6-month mortality (Figure 2).

Prognostic Value of the CAR for 6-Month Mortality

After confounders were adjusted for, the CARs at 48 hours (OR, 1.291; 95% CI, 1.071–1.556) and 72 hours (OR, 1.515; 95% CI, 1.211–1.894) after cardiac arrest were independently associated with 6-month mortality (Table 3). Moreover, the CARs at 48 hours (OR, 1.181; 95% CI, 1.017–1.372) and 72 h (OR, 1.299; 95% CI, 1.101–1.533) after cardiac arrest were independently associated with in-hospital mortality (Table 3).

The AUCs of CAR at admission and 24, 48, and 72 hours after cardiac arrest for predicting 6-month mortality were 0.583 (95% CI, 0.489–0.673), 0.622 (95% CI, 0.528–0.710), 0.706 (95% CI, 0.615–0.786), and 0.762 (95% CI, 0.675–0.835), respectively (Table 4). Moreover, the AUCs of the CAR at admission and at 24, 48, and 72 hours after cardiac arrest for predicting in-hospital mortality were 0.576 (95% CI, 0.482–0.667), 0.595 (95% CI, 0.501–0.684), 0.687 (95% CI, 0.595–0.769), and 0.735 (95% CI, 0.645–0.812), respectively (Table 4). The AUC of the CAR 72 hours after cardiac arrest differed significantly from that at admission and at 24 hours after cardiac arrest but not from that observed 48 hours after cardiac arrest for predicting 6-month mortality or in-hospital mortality.

DISCUSSION

The primary finding in this study was the association between the CAR at 48 and 72 hours after cardiac arrest and 6-month mortality in the PCAS patient group. The CAR at 72 hours after cardiac arrest showed the highest performance for predicting 6-month mortality. Elevated levels of CRP were associated with 1-year mortality in acute cerebral infarction along with ischemic inflammatory response [15]. Systemic inflammation also occurs in patients with PCAS and ischemic injury. During cardiac arrest, the inflammatory response increases vascular permeability and destroys the blood-brain barrier (BBB), causing multiorgan ischemia, including in the brain [3,16]. CRP is an inflammatory biomarker that may be correlated with

Table 3. Multivariable logistic regression analysis of the ability of the CAR to predict 6-month mortality or in-hospital mortality

Variable	Adjusted OR (95% CI)[a]	P-value
6-Month mortality		
CAR at admission	1.112 (0.865–1.429)	0.408
CAR at 24 hours after cardiac arrest	1.136 (0.918–1.405)	0.242
CAR at 48 hours after cardiac arrest	1.291 (1.071–1.556)	0.007
CAR at 72 hours after cardiac arrest	1.515 (1.211–1.894)	<0.001
In-hospital mortality		
CAR at admission	1.079 (0.873–1.334)	0.482
CAR at 24 hours after cardiac arrest	1.069 (0.896–1.276)	0.458
CAR at 48 hours after cardiac arrest	1.181 (1.017–1.372)	0.029
CAR at 72 hours after cardiac arrest	1.299 (1.101–1.533)	0.002

Each variable was individually entered into the final model and analyzed separately.
CAR: C-reactive protein-to-albumin ratio; OR: odds ratio; CI: confidence interval.
a) Adjusted for bystander C-reactive protein and shockable rhythm.

Table 4. ROC analysis results of the CAR to predict 6-month mortality and in-hospital mortality

Variable	Cutoff	Sensitivity	Specificity	AUC	P-value
6-Month mortality					
CAR at admission	>0.17	26.92	90.91	0.583 (0.489–0.673)	0.116
CAR at 24 hours after cardiac arrest	>1.79	65.38	57.58	0.622 (0.528–0.710)	0.020
CAR at 48 hours after cardiac arrest	>4.14	73.08	63.64	0.706 (0.615–0.786)	<0.001
CAR at 72 hours after cardiac arrest	>4.73	69.23	77.27	0.762 (0.675–0.835)	<0.001
In-hospital mortality					
CAR at admission	>0.17	30.00	89.74	0.576 (0.482–0.667)	0.174
CAR at 24 hours after cardiac arrest	>3.66	30.00	92.31	0.595 (0.501–0.684)	0.097
CAR at 48 hours after cardiac arrest	>5.70	52.50	78.21	0.687 (0.595–0.769)	<0.001
CAR at 72 hours after cardiac arrest	>5.90	50.00	87.18	0.735 (0.645–0.812)	<0.001

ROC: receiver operating characteristic; CAR: C-reactive protein-to-albumin ratio; AUC: area under the ROC curve.

the severity of hypoxic brain damage following cardiac arrest. Engel et al. [5] reported that increased CRP levels after cardiac arrest are correlated with the SOFA scores of day 1 and three-month neurological outcomes. In another study, CRP at admission was associated with 30-day mortality in patients with PCAS [17]; however, CRP on admission was not associated with in-hospital or 6-month mortality in the present study. The reason for this difference may be the difference in the frequency of withdrawal of life-sustaining therapy (WLST). Unlike in Europe and the United States, WLST is rarely implemented for patients with PCAS in Korea. In a previous study [17], more patients had witnesses of their collapse (83.1% vs. 68.6%) and shockable rhythms (53.3% vs. 44.1%) compared with the present study. Thirty-day mortality (41.5%) in the previous study was higher than in-hospital mortality (33.9%) and similar to 6-month mortality (44.1%) in the present study. In one retrospective study, the increase in CRP levels in the TTM group was inhibited during cooling compared to the no-TTM group, and the difference gradually decreased after cooling [18]. In another retrospective study, the group with a poor neurologic outcome exhibited higher CRP levels at 48 and 72 hours than the group with a good neurologic outcome [19]. Thus, the inflammatory response is suppressed during TTM. After rewarming, the metabolism is restored, and the inflammatory response is activated, which results in increased CRP levels.

Hypoxia due to cardiac arrest causes increased the vascular permeability and impaired the BBB [20]. In an experimental study, hypoxic brain injury after cardiac arrest provoked BBB disruption and edema 24 hours after ROSC [21]. Increased vascular permeability leads to the loss of serum albumin, which results in reduced albumin levels following ischemic injury. In a previous study, albumin levels <3.5 g/dl were associated with in-hospital mortality and neurologic outcome [22]. In the present study, the albumin levels of non-survivors at each time point were usually <3.5 g/dl. Since high CRP levels and low albumin levels were associated with a poor prognosis in patients with PCAS, the CAR as calculated using CRP and albumin levels in the present study appears to reflect the severity of PCAS patients well.

CAR at admission is associated with in-hospital mortality of patients resuscitated from OHCA [23]. In this study [23], in-hospital mortality was 57.8% (59/102), which was higher than that reported by other studies, including our study [24,25]. In addition, data on TTM in previous studies have been lacking [23]. We postulated that TTM would delay the inflammatory response. Bisschops et al. [13] reported that mild hypothermia for 72 hours after cardiac arrest was correlated with a lowered inflammatory response. Several mechanisms are likely involved here. TTM contributes to a reduction in the white blood cell count and activity, which in turn prolongs the impairment of neutrophil function [13]. In an experimental study, inflammatory cytokines and gene expression were largely downregulated in the hypothermia-treated heart compared to the normothermic heart at 48 hours after TTM [26]. During *in vivo* experiments, neutrophil and monocyte chemotaxis, migration, phagocytosis, and oxidative metabolism were markedly decreased at 29°C compared with 37°C [27].

The present study has several limitations. First, it was retrospective in design and was conducted at a single center. Therefore, its results cannot be generalized immediately to the overall population. Additional prospective multicenter studies are needed to complement our research results. Second, other inflammatory markers (such as cytokines and chemokines) were not investigated in this study. Further studies that include these inflammatory markers will be needed in the future. Third, drugs such as vasopressors are generally used to improve cerebral perfusion after cardiac arrest and prevent secondary ischemic injury. In addition, norepinephrine increases the production of pro- and anti-inflammatory cytokines, but the effects of vasopressors (including norepinephrine) were not sufficiently considered in this study. Fourth, we did not investigate whether albumin was replaced according to albumin level or whether the replaced albumin affected the clinical outcomes of patients with PCAS.

In conclusion, the CAR 72 hours after cardiac arrest was related to 6-month mortality in the PCAS patient group and exhibited the best performance for predicting 6-month mortality. The CAR obtained 72 hours after cardiac arrest was an independent predictor of long-term mortality in the PCAS patient group.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

FUNDING

None.

ACKNOWLEDGMENTS

None.

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AUTHOR CONTRIBUTIONS

Conceptualization: JHL. Data curation: HHK, JHL. Formal analysis: DHL. Methodology: JHL, DHL. Project administration: JHL. Visualization: BKL. Writing–original draft: HHK, JHL. Writing–review & editing: all authors.

SUPPLEMENTARY MATERIALS

Supplementary materials can be found via https://doi.org/10.4266/acc.2022.00542.

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