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ORIGINAL ARTICLE

The C-Reactive Protein to Albumin Ratio Predicts Acute Kidney Injury in Patients With ST-Segment Elevation Myocardial Infarction Undergoing Primary Percutaneous Coronary Intervention

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Background The relationship between acute kidney injury (AKI) and C-reactive protein (CRP) and albumin has been previously demonstrated in patients with ST elevation myocardial infarction (STEMI) treated with primary percutaneous coronary intervention (pPCI). However, to our knowledge, CRP to albumin ratio (CAR), a newly introduced inflammation-based risk score, has not yet been studied. In this study, we aimed to investigate the possible relationship between the CAR and AKI.

Method A total of 815 consecutive STEMI patients treated with pPCI were reviewed.

Results One hundred ten 110 (13.5%) patients developed AKI in the study population. The subjects were divided into two groups according to AKI development. The in-hospital mortality rate was higher in patients with AKI than those without AKI (15.5% vs. 1.3%; $p < 0.001$). The patients with AKI had significantly higher mean value of CRP and CAR (0.29 [0.16–0.50] vs. 0.55 [0.37–1.05]; $p < 0.001$) and lower mean levels of albumin than those without AKI. Age, diabetes mellitus, haematocrit, left ventricular ejection fraction, hypotension, and CAR (Odds ratio [OR]2.307, 95% confidence interval [CI] 1.397–3.809, $p = 0.001$) were independent predictors of AKI.

Conclusion The CAR may be a useful inflammation-based risk score to predict AKI development in STEMI patients treated with pPCI.

Keywords C-Reactive protein • Albumin • C-Reactive protein to albumin ratio • Acute kidney injury • ST elevation myocardial infarction • Primary percutaneous coronary intervention

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Introduction

Acute kidney injury (AKI) is described as an abrupt deterioration in renal function associated with the administration of contrast media, frequently encountered as a complication of percutaneous coronary intervention (PCI) [1]. Post-PCI AKI is observed in at least 2% of patients with normal baseline creatinine levels; whereas this prevalence rate increases to 20%-30% in patients with creatinine levels >2 mg/dL after the PCI [2,3]. Furthermore, the rate of AKI development was significantly higher in acute myocardial infarction (AMI) patients treated with emergency PCI than in those who underwent elective procedure [2,4,5]. As a common complication of PCI, AKI is associated with higher mortality and morbidity, longer hospital duration, and increased health care costs [6]. Given the poor prognostic implications, the ability to predict AKI development becomes a crucial step.

The pathophysiology of AKI has not been fully elucidated. The development of AKI has been speculated to be influenced by changes in renal haemodynamics because of the effects of contrast medium. The potential underlying causes include improper balance between the increased vasoconstrictors (i.e., endothelin and adenosine) and vasodilators (i.e., nitric oxide and prostaglandin), increase oxidative stress and oxygen-free radicals, direct cellular toxicity, cellular necrosis, and interstitial inflammation [7]. However, AKI does not seem to be associated with a cascade triggered only by contrast medium injection. Further, the basal inflammatory status is associated with AKI. Studies conducted on these bases have found significant associations between C-reactive protein (CRP) and AKI [8–10]. Similarly, the level of serum albumin (SA), a negative acute phase protein in acute inflammation, has been associated with AKI [11].

C-reactive protein to albumin ratio is believed to reflect the stability between the CRP and albumin levels in determining the prognoses of critical diseases and malignancies and is identified as an inflammation-based prognostic score [12,13]. The superiority of CAR to CRP and albumin levels in reflecting systemic inflammation has prompted an investigation regarding the possible relationship between CAR and AKI. In this study, we aimed to assess whether CAR is associated with AKI development in STEMI patients treated with primary PCI (pPCI).

Methods

Study Population

This was a retrospective study conducted between April 2012 and June 2015. A total of 968 consecutive STEMI patients who had undergone pPCI in Kafkas University were enrolled. The STEMI was diagnosed based on the electrocardiography (ECG) conducted on admission as well as the clinical symptoms and findings. Patients with chest pain that continued for >30 minutes (within 12 hours of presentation) and the presence of new or presumed new ST-segment elevation at the J-point in ≥2 contiguous leads of ≥0.2 mV in leads V1, V2, or V3 and ≥0.1 mV in other leads were diagnosed with STEMI. The marked ST-segment depression that was maximal in leads V1 through V3, without ST-segment elevation in other leads, was designated as posterior-wall MI and also included in the STEMI study population [14]. We excluded the patients who were referred to emergency coronary artery bypass graft surgery (CABG) or with a history of elective CABG surgery (n = 59), treated with only medical therapy (n = 22), undergoing haemodialysis (n = 12), had known malignancies (n = 11), had febrile conditions (n = 9), had autoimmune disorders (n = 4), or had incomplete medical files or records (n = 34). A total of 815 patients met the eligibility criteria and were enrolled in the study. The research protocols were as per the Declaration of Helsinki; the study was approved by the local ethical board.

Laboratory Measurements

The blood samples were collected from all patients for measurement of the baseline values of albumin, creatinine, glucose, and CRP at the time of hospital admission prior to pPCI. Serum creatinine, CRP, and SA levels were determined using an automatic biochemical analyser (Roche Diagnostics Cobas 8000 c502, Indianapolis, IN, USA). The normal values for CRP level and SA levels were 0–19 mg/L and 3.5–5.5 g/dL, respectively, in the hospital. The CAR was calculated by dividing the serum CRP level by the SA level, then, multiplying the result by 10 [11]. The estimated glomerular filtration rate (eGFR) was estimated according to the modification of diet in renal disease (MDRD) formula. Acute kidney injury was defined as an increase of 25% or 0.5 mg/dL in the creatinine level compared to that at baseline within 48 hours. In addition, left ventricular ejection fraction (LVEF) was calculated in accordance with Simpson's method in all patients within the first 24 hours of admission.

Coronary Angiography

All patients underwent coronary angiography via femoral artery and all procedures were performed by experienced interventional cardiologists. The balloon angioplasty and/or stent implantation were performed for treating the culprit lesions. Nonionic, low-osmolality contrast medium was used for all patients. The patients were hydrated with normal saline for 18–24 hours postoperatively. The patients in the emergency department received a single loading dose of 300 mg aspirin and 600 mg clopidogrel before the coronary angiography. The unfractionated heparin (per 40–70 U/kg with intravenously) was given to reach an activated clotting time of 200–250 seconds during the pPCI. The use of glycoprotein IIb/IIIa inhibitors as well as the decision regarding the stent type (drug eluting or bare metal) was left to the discretion of the interventional cardiologist.

Statistical Analyses

Windows SPSS 21.0 was used to conduct the statistical analyses (SPSS Inc., Armonk, NY, USA). Normally distributed continuous variables are presented as mean ± standard

deviation values and analysed using Student t test or analysis of variance; those with non-normal distribution are presented as median (25th–75th) values and analysed using Mann–Whitney or the Wilcoxon rank-sum test. Categorical variables were described as absolute values and percentages, and analysed by the chi-square or Fisher exact test. Multivariable logistic regression analyses were performed for all the variables that were statistically different in the univariate analysis to identify the independent risk factors of AKI. Receiver operating characteristic (ROC) curve analysis was conducted, and the Youden index was used to determine the best cut-off value for the CAR for predicting AKI in STEMI. The area under the curve (AUC) values between the CAR, SA, and CRP were compared using the MedCalc statistical software (MedCalc Software demo, version 11.4, Ostend, Belgium). The Kaplan-Meier method was used to create event-free survival curves using the log-rank test. P value <0.05 was considered statistically significant.

Results

Among the 815 STEMI patients enrolled in the study (the mean age 57 ± 12 years, 18% were women), the incidence of

Table 1 The demographic, clinical, laboratory and coronary angiographic characteristics of all patients, patients with CIN and without CIN and with p value.

	Contrast-induced nephropathy						
	All patients (n:815)		Patients without CIN (n:705)		Patients with CIN (n:110)		P value
Age, years	57	±12	56	±12	64	±12	<0.001
Male Gender, n (%)	147	(18.0)	120	(17.0)	27	(24.5)	0.056
Diabetes mellitus, n (%)	191	(23.4)	149	(21.1)	42	(38.2)	<0.001
Hypertension, n (%)	339	(41.6)	274	(38.9)	65	(59.1)	<0.001
Dyslipidaemia, n (%)	361	(44.3)	324	(46.0)	37	(33.6)	0.016
Family history, n (%)	195	(23.9)	170	(24.1)	25	(22.7)	0.751
Smoking, n (%)	452	(55.5)	409	(58.0)	43	(39.1)	<0.001
SBP, (mm Hg)	132	±32	132	±28	134	±48	0.636
DBP, (mm Hg)	78	±19	78	±17	77	±28	0.947
Heart rate (bpm)	77	±17	77	±15	77	±22	0.105
WBC Count, (10*3/μl)	12.3	±3.8	12.2	±3.6	13.2	±5.2	0.194
Haematocrit, (%)	41.0	±5.4	41.4	±5.1	38.2	±5.9	<0.001
Peak CK-MB, (U/L)	178	99-318	167	96-298	283	145-452	<0.001
Baseline creatinine, (mg/dl)	0.91	±0.3	0.88	±0.2	1.15	±0.5	<0.001
eGFR, (ml/min)	88.96	±24.9	91.30	±22.3	73.92	±33.8	<0.001
Peak creatinine, (mg/dl)	0.95	0.81-1.13	0.90	0.80-1.08	1.50	1.18-2.47	<0.001
Increase of creatinine, (%)	10.00	0.00-16.67	9.59	0.00-16.30	48.38	35.92-66.67	<0.001
Serum albumin, (g/dl)	3.74	±0.5	3.78	±0.5	3.50	±0.4	<0.001
C-Reactive protein, (mg/dl)	12.30	6.44-20.70	11.20	6.00-18.50	19.15	12.40-32.50	<0.001
Crp/albumin ratio	0.32	0.17-0.56	0.29	0.16-0.50	0.55	.037-1.05	<0.001
LVEF (%)	46.64	±8.3	47.45	±8.0	41.45	±8.7	<0.001
Killip class > 1 on admission, n (%)	127	(15.6)	91	(12.9)	36	(32.7)	<0.001
Pain to balloon time, min	140	80-240	140	80-230	200	90-300	<0.001
IRA of LAD, n (%)	426	(52.3)	369	(52.3)	57	(51.8)	0.919
Multi-vessel disease, n (%)	317	(38.9)	439	(62.3)	59	(53.6)	0.084
LMCA disease, n (%)	10	(1.2)	9	(1.3)	1	(0.9)	<0.001
Cardiogenic shock on admission, n (%)	37	(4.5)	19	(2.7)	18	(16.4)	<0.001
Out-of-hospital cardiac arrest, n (%)	18	(2.2)	12	(1.7)	6	(5.5)	0.013
Hypotension, n (%)	64	(7.9)	37	(5.2)	27	(24.5)	<0.001
Intra-aortic balloon pump use, n (%)	49	(6.0)	29	(4.1)	20	(18.2)	<0.001
Contrast media, (mL)	271	±72.5	260.1	±63.0	337.8	±91.2	<0.001
Death, n (%)	26	(3.2)	9	(1.3)	17	(15.5)	<0.001
Length of hospital stay, days	5	±3	5	±3	7	±5	<0.001

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure; eGFR, estimated glomerular filtration rate; WBC, white blood cell; CK-MB, creatinine kinase-myocardial band; LVEF, left ventricular ejection fraction; IRA, infarct related artery; LAD, left anterior descending; LMCA, left main coronary artery.

AKI was 13.5% (n = 110). Haemodialysis treatment was applied to eight patients with AKI and only one patient required chronic renal replacement therapy during the follow-up. The demographic, clinical, laboratory and angiographic characteristics of the individual groups are listed in Table 1, along with the comparison of the groups with and without AKI. The patients with AKI were older and had a higher frequency of diabetes mellitus, hypertension, dyslipidaemia, Killip class> 1 and cardiogenic shock on admission, out-of-hospital cardiac arrest, hypotension, and intra-aortic balloon pump usage than patients without AKI ($p < 0.05$, for all). The CAR (0.29 [0.16–0.50] vs. 0.55 [0.37–1.05]; $p < 0.001$), CRP, peak creatinine kinase myocardial band (CK-MB), and total ischaemic time were higher in the AKI group, while, the SA and haematocrit level, eGFR, and LVEF values were lower in the AKI group ($p < 0.001$). The in-hospital mortality rate was higher in patients with AKI (17 patients out of 110 patients, 14.5%,) than those without AKI (9 patients out of 705 patients, 1.3%) ($p < 0.001$ Figure 1).

The CAR values of the patients ranged from 0.020 to 2.72 (median: 0.323). The study population was divided into three groups based on the CAR tertiles as follows: patients with CAR<209 composed the lower tertile group (n = 271), those with 0.209≤CAR≤0.457 composed the intermediate group (n = 272), and those with CAR>0.457 composed the higher tertile group (n = 72). With the increasing CAR tertiles, there was an increase in the age, total ischaemic time, heart rate, CRP and peak CK-MB values, baseline creatinine, prevalence of hypertension, Killip class >1 on admission, left anterior descending artery (LAD) as the infarct related artery, cardiogenic shock, hypotension, and intra-aortic balloon pump usage, while there was a decrease in the eGFR and LVEF values. Furthermore, the statistically significant increase in the development of AKI was observed as the CAR tertile increased (4.4% vs. 10.7% vs. 25.4%, respectively; $p < 0.001$ for each comparison) (Table 2).

To identify the independent predictors of AKI development, multivariate regression analyses with a stepwise backward model were performed using the variables that showed marginal association with AKI in the univariate analyses; these variables included age, diabetes mellitus, hypertension, smoking, dyslipidaemia, haematocrit, peak CK-MB, CAR, LVEF, Killip class > 1 on admission, pain to ischaemia time, left main coronary artery (LMCA) disease, cardiogenic shock on admission, out-of-hospital cardiac arrest, hypotension, intra-aortic balloon pump (IABP) usage, the amount of contrast media usage. Age (Odds ratio [OR] 1.044, 95% confidence interval [CI] 1.023–1.065, $p < 0.001$), diabetes mellitus (OR 1.708, 95% CI 1.053–2.769, $p = 0.030$), haematocrit (OR 0.950, 95% CI 0.911–0.990, $p = 0.016$), LVEF (OR 0.957, 95% CI 0.928–0.986, $p = 0.004$), hypotension (OR: 3.087, 95% CI 1.624–5.871, $p \leq 0.001$), and CAR (OR 2.307, 95% CI 1.397–3.809, $p \leq 0.001$) were found to be independent predictors of AKI (Table 3; model 1). To exclude patients presenting with cardiogenic shock, multivariate regression analysis was repeated using the variables that showed marginal association with AKI in the univariate analyses including age, diabetes mellitus, hypertension, smoking, dyslipidaemia,

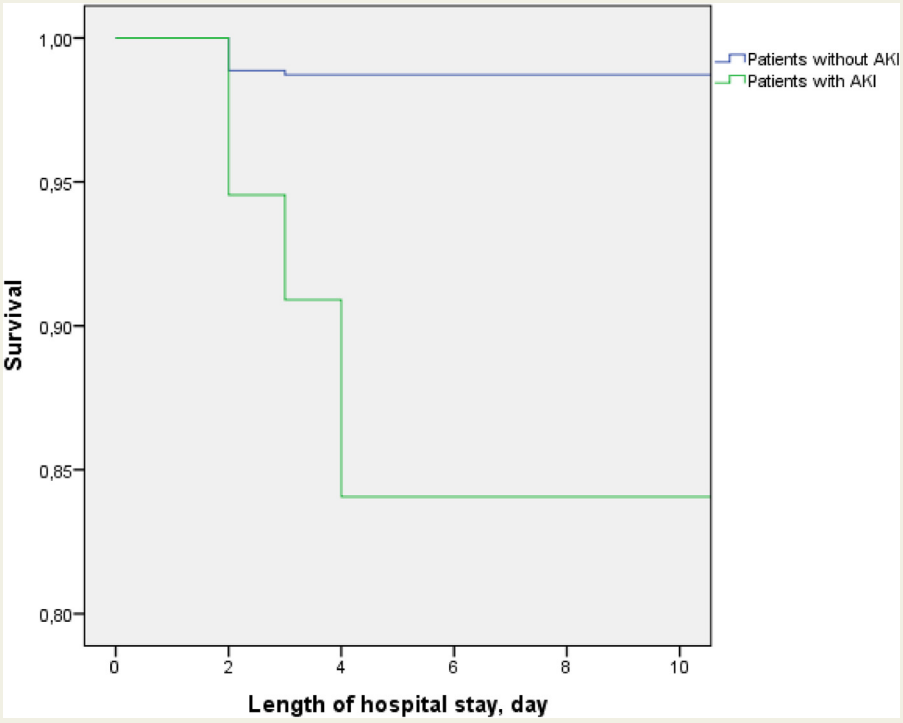

Figure 1 The overall survival comparison curves between the patients with and without acute kidney injury.

Table 2 The demographic, clinical, laboratory and coronary angiographic characteristics of CAR tertiles with p value.

	CRP/albumin ratio with tertiles						
	<0.209 (n:271)		0.209-0.457 (n:272)		>0.4577 (n:272)		P value
Age, years	54	±11	57	±12	60	±12	<0.001
Male gender, n (%)	42	(15.5)	50	(18.4)	55	(20.2)	0.354
Diabetes mellitus, n (%)	58	(21.4)	64	(23.5)	69	(25.4)	0.552
Hypertension, n (%)	95	(35.1)	117	(43.0)	127	(46.7)	0.019
Dyslipidaemia, n (%)	120	(44.3)	133	(48.9)	108	(39.7)	0.098
Family history, n (%)	55	(20.3)	77	(28.3)	63	(23.2)	0.086
Smoking, n (%)	162	(59.8)	149	(54.8)	141	(51.8)	0.171
SBP, mm Hg	130	±27	135	±29	132	±37	0.190
DBP, mm Hg	78	±16	79	±17	78	±23	0.265
Heart rate, bpm	76	±15	76	±15	79	±19	<0.001
WBC count, $10^3/\mu l$	12.0	±3.3	11.9	±3.2	13.1	±4.8	0.089
Haematocrit, n (%)	41.3	±5.0	41.3	±5.1	40.5	±6.0	0.188
Peak CK-MB, U/L	118	76-197	177	114-277	288	149-423	<0.001
Baseline creatinine, mg/dl	0.85	±0.2	0.88	±0.2	1.00	±0.4	<0.001
eGFR, ml/min	95.57	±23.9	89.68	±21.5	81.64	±27.0	<0.001
Peak creatinine, mg/dl	0.90	0.80-1.00	0.96	0.85-1.10	1.01	0.90-1.30	<0.001
Increase of creatinine (%)	11.11	1.09-20.00	13.25	3.47-24.64	11.11	1.18-20.00	<0.001
Serum albumin, g/dl	3.84	±0.3	3.77	±0.4	3.61	±0.6	<0.001
C-Reactive protein, mg/dl	4.93	3.44-6.44	12.30	9.65-14.35	25.64	20.00-40.45	<0.001
CRP/albumin ratio	0.13	0.09-0.17	0.32	0.26-0.38	0.71	0.56-1.11	<0.001
LVEF, %	49.94	±6.8	47.80	±7.2	42.19	±8.8	<0.001
Killip class >1 on admission, n (%)	25.00	(9.2)	39.00	(14.3)	63.00	(23.2)	<0.001
Pain to balloon time, min	120	75-180	150	80-230	195	110-300	<0.001
IRA of LAD, n (%)	131	(48.3)	136	(50.0)	159	(58.5)	0.041
Multi-vessel disease, n (%)	94	(34.7)	105	(38.6)	118	(43.4)	0.115
LMCA disease, n (%)	4	(1.5)	1	(0.4)	5	(1.8)	0.268
Cardiogenic shock on admission, n (%)	5	(1.8)	3	(1.1)	29	(10.7)	<0.001
Out-of-hospital cardiac arrest, n (%)	2	(0.7)	4	(1.5)	12	(4.4)	0.009
Hypotension, n (%)	9	(3.3)	14	(5.1)	41	(15.1)	<0.001
Intra-aortic balloon pump use	5	(1.8)	5	(1.8)	39	(14.3)	<0.001
Contrast media (mL)	259.5	±65.3	266.0	±67.8	286.2	±80.8	<0.001
Acute kidney injury, n (%)	12	(4.4)	29	(10.7)	69	(25.4)	<0.001
Haemodialysis requirement, n (%)	1	(0.4)	1	(0.4)	6	(2.2)	0.043
Death, n (%)	3	(1.1)	3	(1.1)	20	(7.4)	<0.001
Length of hospital stay, day	5	±3	5	±3	6	±3	<0.001

Abbreviations: SBP, systolic blood pressure; DBP, diastolic blood pressure eGFR, estimated glomerular filtration rate; WBC, white blood cell; CK-MB, creatinine kinase-myocardial band; LVEF, left ventricular ejection fraction; IRA, infarct related artery; LAD, left anterior descending; LMCA, left main coronary artery; CRP, C-reactive protein.

systolic blood pressure, haematocrit, peak CK-MB, CAR, LVEF, Killip class > 1 on admission, pain to balloon time, hypotension, IABP usage, the amount of contrast media usage. The age, systolic blood pressure (SBP), haematocrit, CAR (OR: 3.076; %95 CI: 1.679–5.636), hypotension, and contrast media amount were found to be independent predictors of acute renal failure (Table 3; model 2).

To determine whether CAR was better than CRP and albumin levels in predicting AKI, their ROC curves were compared. The AUC of CAR (AUC 0.724, 95% CI 0.675–0.772) was significantly higher than that of CRP (AUC 0.705, 95% CI 0.658–0.753) and albumin level (AUC 0.679, 95% CI 0.623–0.734) ($p < 0.001$ for each comparison). The AUC values of CRP and albumin were similar. The best cut-off value of CAR for predicting AKI was ≥0.369 with a sensitivity of 75.4% and a specificity of 60.7% (Figure 2).

Discussion

Our study demonstrated that, in patients with STEMI, who had undergone pPCI, the CAR was associated with the

Table 3 Univariate and multivariate logistic regression analysis of demographic, clinical, laboratory and coronary angiographic characteristics for prediction of acute kidney injury.

	Model 1*					
	Univariate analysis			Multivariate analysis		
	Odds ratio	95% C.I.	P value	Odds ratio	95% C.I.	P value
Age	1.064	1.046-1.083	<0.001	1.044	1.023-1.065	<0.001
Diabetes mellitus	2.305	1.507-3.525	<0.001	1.708	1.053-2.769	0.030
Haematocrit	0.900	0.868-0.933	<0.001	0.950	0.911-0.990	0.016
LVEF	0.917	0.894-0.940	<0.001	0.957	0.928-0.986	0.004
Hypotension	5.873	3.402-10.139	<0.001	3.087	1.624-5.871	<0.001
CRP/albumin ratio	4.183	2.772-6.312	<0.001	2.307	1.397-3.809	<0.001
	Model 2**					
	Univariate analysis			Multivariate analysis		
	Odds ratio	95% C.I.	P value	Odds ratio	95% C.I.	P value
Age	1.035	1.009-1.062	<0.001	1.033	1.008-1.058	0.008
SBP	1.023	1.002-1.044	<0.001	1.014	1.006-1.023	<0.001
Haematocrit	0.948	0.901-0.997	<0.001	0.946	0.901-0.994	0.029
CRP/albumin ratio	2.986	1.598-5.582	<0.001	3.076	1.679-5.636	<0.001
Hypotension	10.125	2.795-36.675	<0.001	10.459	3.261-33.554	<0.001
The amount of contrast media	1.014	1.010-1.017	<0.001	1.014	1.010-1.017	<0.001

Abbreviations: SBP, systolic blood pressure; LVEF, left ventricular ejection fraction; CI, confidence interval; CRP, C-reactive protein.

*All patients included in model 1.

**Patients without cardiogenic shock were analysed in Model.

occurrence of AKI and, also, it was an independent predictor of AKI development. Moreover, AKI development and the elevated CAR values were associated with in-hospital mortality. Furthermore, the CAR predicted AKI more accurately than either CRP or SA.

Many studies have demonstrated the association of AKI with short- and long-term adverse clinical outcomes including, longer hospitalisation, recurrent cardiovascular events, progression to chronic renal disease (CKD), and higher in-hospital mortality [2,15–17]. In our study, AKI developed in 13.5% of the patients. The mortality rate was 14.5% during the hospitalisation, and the length of hospital stay was longer in AKI patients, a consistent finding with the previous data [18,19]. Given the poor prognostic implications of AKI, several demographic, clinical, and laboratory characteristics have been established to identify individuals who are at an increased risk of AKI. In accordance with the results of previous studies [1,20], in our study, patients with AKI were older, had a higher frequency of diabetes mellitus and hypertension, had signs and symptoms of cardiogenic shock and Killip class> 1 on admission, had a higher frequency of hypotension, and greater need of intra-aortic balloon pump usage than those without AKI. In addition, in patients with AKI, SA, haematocrit, eGFR, and LVEF were lower than in those without AKI.

C-reactive protein is considered a useful and objective stratification tool for active inflammatory process. The previous studies have shown a significant relationship between the elevated CRP levels and the occurrence of AKI [10,21]. In the present study, CRP was also significantly higher in the AKI group. CRP is a potent chemo attractant that can mediate the enhanced expression of adhesion molecules, potentiate plasminogen activator inhibitor-1, and diminish nitric oxide production. The increase in CRP levels may result in endothelial dysfunction via inducing vasoconstrictive, pro-thrombotic, and pro-inflammatory pathways [22,23]. The activation of platelet and coagulation systems may reduce blood flow to the kidneys resulting in reducing oxygen delivery [24,25]. Serum albumin, the most abundant circulating protein, has a prominent anti-oxidant activity [26]. The level of SA is inversely associated with the degree of inflammation in the body. The inflammation may cause the decreased albumin synthesis [27]. The decrease in the SA level (hypoalbuminaemia) may increase blood viscosity and disrupt the endothelial function [28]. In our study, SA levels were significantly lower in patients with AKI. These findings may suggest that the basal inflammatory status may be a major determinant of AKI development.

The response of acute phase reactants to each inflammatory status may not be at similar degree. By merging albumin and CRP into a single index, as an inflammation-based prognostic score in diseases where inflammation plays an important role, provides the stability amongst the CRP and

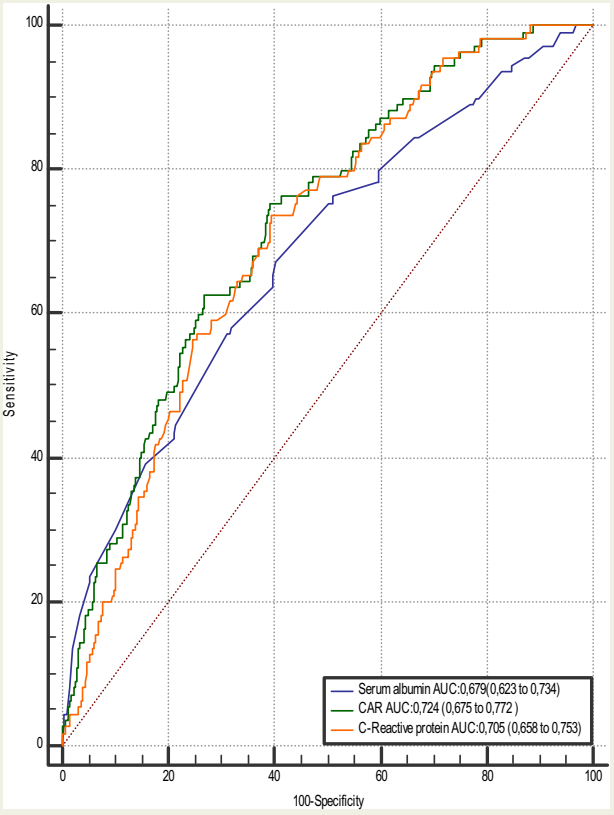

Figure 2 The ROC curves of CAR, C-reactive protein, and albumin for acute kidney injury.
Abbreviations: ROC, receiver operating characteristic; CAR, C-reactive protein/albumin ratio.

albumin levels. A study which evaluates the prognostic impact of CAR in patients with critically ill medical patients has demonstrated that CAR has a favourable prognostic value in the elderly, especially in those with acute exacerbations of chronic disease [13]. Furthermore, increased CAR values at intensive care unit discharge have been found to provide more consistent results than CRP values in predicting long-term mortality of septic patients [29]. Similar results have been observed in patients with hepatocellular carcinoma [12]. In accordance with these studies, in-hospital mortality of our patients located in a higher CAR tertile was significantly higher than those located in a lower CAR tertile. The CAR was significantly higher in AKI patients and was an independent predictor of AKI development. Furthermore, a comparison of the ROC curves of CAR with those of CRP and albumin showed that CAR had a superior predictive ability than either CRP or albumin alone for predicting the AKI development. This superiority of CAR to CRP and album can be explained as follows: Firstly, the changes in CRP and albumin levels in response to inflammation may vary widely between individuals and clinical conditions. Secondly, the proportion of CRP to albumin, which both have a reverse response to each other with respect to inflammation, has increased the diagnostic value compared to CRP and albumin alone.

Conclusion

In conclusion, considering the relationship between AKI and poor outcomes, identifying individuals at an increased risk of AKI is a vital step. In accordance with previous studies which emphasise the effect of inflammation on AKI development, CAR is found to be an independent predictor of AKI in STEMI patients who underwent pPCI independently of cardiogenic shock on admission. Also, the present study suggests that AKI may be associated not only with basal inflammatory status, but also with contrast medium amount and subsequent changes in renal haemodynamics.

Limitations

This was a retrospective study. Therefore, the risk of bias cannot be ruled out although we attempted to adjust for the confounding factors. This study included only STEMI patients, so further study is needed in patients with other acute coronary syndromes and stable coronary artery disease.

Conflict of Interest

Both authors declare that they have no conflict of interest.

Ethical Approval

The research protocols were as per the Declaration of Helsinki; the study was approved by the local ethical board.

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Disclosures

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