Original Article

Association of eosinophil-to-monocyte ratio with 1-month and long-term all-cause mortality in patients with ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention

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Background: To determine the relationship between eosinophil-to-monocyte ratio (EMR) on admission and one-month and long-term all-cause mortality in patients with ST-elevation myocardial infarction (STEMI) undergoing primary percutaneous coronary intervention (P-PCI).

Methods: A total of 426 consecutive STEMI patients treated with P-PCI were enrolled and categorized in terms of tertiles of EMR on admission between September 2015 and October 2017. Final follow-up for long-term outcomes was January 2017.

Results: As EMR decreased, all-cause mortality at 1 month (mean, 29.5±3.5 days) and at mean 14.1± 7.8 months follow-up increased (P=0.012, P=0.003, respectively). Kaplan-Meier survival curve analysis showed EMR was associated with 1-month and long-term all-cause mortality (P=0.048, P=0.015, respectively). In multivariate Cox proportional hazards analysis, EMR was independently associated with one-month and long-term mortality (hazard ratio =0.097; 95% CI, 0.010–0.899; P=0.04; hazard ration =0.176; 95% CI, 0.045–0.694; P=0.013). The area under the curve of EMR for the prediction of 1-month and long-term total mortality in receiver operating characteristic analysis was 0.789 (95% CI, 0.658–0.921; P=0.003) and 0.752 (95% CI, 0.619–0.884; P=0.001), respectively.

Conclusions: EMR on admission was independently correlated with 1-month and long-term all-cause mortality in STEMI patients undergoing P-PCI, suggesting EMR as a potential simple, useful, and inexpensive index for risk stratification of STEMI patients.

Keywords: ST-segment elevation myocardial infarction; primary percutaneous coronary intervention; eosinophil-to-monocyte ratio (EMR); mortality

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Introduction

ST-segment elevation myocardial infarction (STEMI) is a severe type of coronary artery disease (CAD) with high morbidity and mortality (1). Inflammatory and immunological responses play an irreplaceable role in the pathogenesis of STEMI (2,3). Leukocytes including neutrophils (4), monocytes (5), and lymphocytes (6) are indispensable inflammatory cells and are associated with atherosclerosis development and progression, plaque rupture, vascular dysfunction and left ventricular remodeling in STEMI patients (7,8). Recently, neutrophil-to-lymphocyte ratio (NLR) and lymphocyte-to-monocyte ratio (LMR) were found to be independent predictive factors of the severity and clinical prognosis of STEMI (9-12). Nevertheless, the association of these individual indexes with cardiovascular diseases has been inconsistent because of their weak specificity (13). There are reports that eosinophils were also inflammatory cells and were able to regulate the inflammatory progress (14). We are not sure whether eosinophil-to-monocyte ratio (EMR) has the similar predictive value as NLR and LMR. This present study assessed the relationship between EMR and one-month and long-term all-cause mortality in STEMI patients undergoing primary percutaneous coronary intervention (P-PCI).

Methods

Study population

As shown in *Figure 1*, a total of 510 consecutive patients with STEMI who received P-PCI between September 2015 and October 2017 at our center were retrospectively recorded. 84 were excluded because of the following criteria: presence of cancer (n=9), or rheumatic or allergic disease (n=4); death within 12 h (n=3); confirmed infection (n=8); unsuccessful angiography (n=1); presence of coronary thrombus from atrial fibrillation (n=3); thrombolysis before angiography (n=4); and missing data (n=52). The left 426 patients (aged 64.7±11.7 years; 81% men) were included in our analysis and categorized according to tertiles of EMR on admission into: T1 group (EMR <0.06, n=141), T2 group (0.06≤ EMR ≤0.18, n=140), and T3 group (EMR >0.18, n=145).

The study protocol was approved by our institutional ethics committee (Ethic Approval: Bro12-030). All patients provided informed consent.

Study definitions

STEMI was defined as typical chest pain lasting >30 minutes but <12 h with documented ST-segment elevation ≥1 mm in ≥2 contiguous leads or new left bundle branch block and elevated myocardial infarction markers (15). One-month and long-term all-cause mortality, defined as death from any reason during the observational period (29.5±3.5 days and 14.1±7.8 months, respectively) post P-PCI, were primary study outcomes. Major adverse cardiac event (MACE) included all-cause mortality, target vessel revascularization (TVR), and myocardial reinfarction. TVR was defined as any kind of revascularization of the initial target vessel, including coronary artery bypass graft (CABG) surgery and PCI. Myocardial reinfarction was recorded according to the third universal definition (16).

Data collection

The following demographic and clinical data were recorded: age, sex, blood pressure, medical history, chest pain duration and Killip class. The follow-up data were achieved by telephone. Blood samples were drawn on admission for the measurement of leucocytes, cardiac troponin T (cTNT), serum creatinine (Scr) and C-reactive protein (CRP) using a Sysmex XS 500i auto analyzer (Sysmex, Kobe, Japan). The blood samples were drawn again on the next morning for reviewing latter indexes and measuring serum lipid levels. All patients finished ultrasonic cardiogram (UCG) testing and the first result was recorded. The value of EMR was calculated by dividing eosinophil count by monocyte count.

Coronary angiography

All patients received dual antiplatelet therapy with aspirin (300 mg) and clopidogrel (300 mg) or ticagrelor (180 mg) before PCI. The statin and beta-blocker agents were prescribed to all the individuals if without contraindication. A team of at least two experienced cardiologists performed PCIs. The choices of approach, technique, and equipment were left to the operator. The interventional success was defined as the achievement of <20% diameter stenosis with Thrombolysis in Myocardial Infarction (TIMI) 3 flow in the target vessel. All angiographic and procedural details were judged and recorded by the operators and assistants who were blind to the study design and subsequent data analysis. All cases received standard perioperative hospital care.

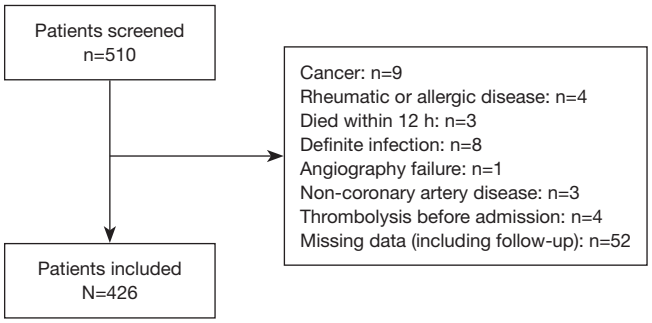

Figure 1 Patient selection flow diagram.

Statistical analysis

Continuous variables were presented as mean ± standard deviation (SD) when normally distributed as per the Kolmogorov-Smirnov test or otherwise as median (interquartile range). Categorical variables were expressed as number (%). Continuous variables were compared by the Mann-Whitney *U* test and categorical variables were compared by Mantel-Haenszel test. The relationship between EMR and other variables was assessed using Spearman bivariate correlation analysis. Univariate analysis was applied to evaluate the effect of different variables on the all-cause mortality. Suspected variables or variables with P<0.1 in univariate analysis were included into multivariate Cox proportional hazards model. Collinearity diagnostics was finished before the multivariable analysis, whose results were presented as hazard ratios (HR) and 95% confidence intervals (95% CI). Survival curves were analyzed using Kaplan-Meier estimation and they were compared using the log-rank test. Receiver operating characteristic curve analysis was performed to determine the best cut-off value for predicting all-cause mortality and the sensitivity as well as the specificity of EMR. P value <0.05 was considered statistically significant. All data analyses were conducted by SPSS 22.0 software (IBM SPSS Statistics, USA).

Results

Patient baseline and clinical characteristics

The baseline characteristics of the study population were provided in *Tables 1*,*2*. Compared with the T2 and T3 group, the T1 group had a higher level of high density lipoprotein-cholesterol (HDL-C), creatine kinase-myocardial band (CK-MB), cTNT, and neutrophil count, whereas a lower level of N-terminal pro b-type natriuretic peptide (NT-proBNP), hemoglobin, eosinophil count, and lymphocyte count. Besides, there was a higher proportion of patients with symptom-to-hospital time ≤3 h in the T1 group. There was no statistically significant difference in terms of other analyzed demographic, laboratory, and angiographic features.

EMR was negatively correlated with cTNT, CK-MB, CRP, and NT-proBNP levels, and neutrophil, leukocyte counts. However, EMR had a positive and strong correlation with lymphocyte and eosinophil count, a positive and weak correlation with left ventricular ejection fraction (LVEF), and hemoglobin and triglyceride levels (*Table 3*).

Clinical outcomes and EMR

One-month and long-time all-cause mortality rate was 4.25% and 7.09% in the T1 group, 2.14% and 2.9% in the T2 group, and 0% and 0.7% in the T3 group [P (1-month) =0.012; P (long-term) =0.003, *Table 4*]. Other outcomes like TVR and myocardial reinfarction were comparable among groups, as were in-hospital cost and duration.

In multivariate Cox proportional hazards analysis to determine the correlation factors of all-cause mortality, EMR (hazard ratio =0.097; 95% CI, 0.010–0.899; P=0.04), age, Scr, platelet count, and Killip class were independently associated with one-month mortality (*Table 5*). In addition, EMR (hazard ratio =0.176; 95% CI, 0.045–0.694; P=0.013), age, Scr, platelet count, Killip class, and Gensini score were independently correlated with long-term mortality (*Table 6*). As several laboratory parameters such as CRP (r=–0.162; P=0.001), cTNT (r=–0.435; P<0.001), CK-MB (r=–0.47; P<0.001), eosinophil count (r=0.951; P<0.001), NT-pro BNP (r=–0.219; P<0.001), leukocyte (r=0.259; P<0.001), hemoglobin level (r=0.161; P=0.001) correlated with EMR, they were not included into multivariate analysis.

Both Kaplan-Meier survival curves for 1-month and long-term all-cause mortality (*Figure 2*) showed significant difference among groups. Apparently, the T3 group had a higher 1-month and long-term survival rate. In receiver operating characteristic curve analysis, the area under the curve of EMR for the prediction of one-month and long-term mortality was 0.789 (95% CI, 0.658–0.921) and 0.752 (95% CI, 0.619–0.884) (*Figure 3*).

Discussion

Our study demonstrated the association of EMR on admission and the 1-month and long-term total mortality in

Table 1 Demographic and laboratory characteristics

Variables	Tertile of EMR on admission			P value
	T1 (<0.06; n=141)	T2 (0.06–0.18; n=140)	T3 (>0.18; n=145)	
Age, years	65 [59–73]	62.5 [56–73.5]	64 [57.5–73]	0.675
Female	35 (24.82)	22 (15.71)	24 (16.55)	0.077
Diabetes	29 (20.6)	43 (30.7)	36 (24.8)	0.416
Hypertension	90 (63.8)	87 (62.1)	80 (55.2)	0.134
Hyperlipidemia	25 (17.7)	22 (15.7)	27 (18.6)	0.839
Current smoking	50 (35.5)	69 (49.3)	68 (46.9)	0.044
Stroke	9 (6.4)	9 (6.43)	15 (10.3)	354
Family history of CVD	1 (0.71)	5 (3.57)	3 (2.07)	0.431
Previous MI	3 (2.13)	5 (3.57)	5 (3.45)	0.519
Previous PCI	8 (5.67)	7 (5)	9 (6.21)	0.843
Previous CABG	0	0	2 (1.38)	0.087
LVEF, %	50 [45–57]	52 [46.25–58]	52 [47.5–59]	0.081
Valve calcification	40 (28.4)	32 (22.9)	33 (22.8)	0.274
Admission SBP, mmHg	119 [105–134]	120 [106.5–136.75]	120 [109–140]	0.481
Admission DBP, mmHg	70 [64–81]	74 [66–83]	72 [65–84]	0.285
Total cholesterol, mmol/L	4.34 [3.75–4.97]	4.3 [3.65–4.91]	4.34 [3.9–5]	0.397
Triglyceride, mmol/L	1.18 [0.85–1.5]	1.27 [0.95–1.85]	1.21 [0.91–2.02]	0.031
HDL, mmol/L	1.01 [0.92–1.22]	1 [0.83–1.12]	1 [0.88–1.15]	0.023
LDL, mmol/L	2.64 [2.09–3.14]	2.63 [2.08–3.09]	2.64 [2.30–3.08]	0.595
C–reactive protein, mg/L	8 [5.3–14.85]	7 [3.5–10.9]	7 [3–10.8]	0.029
Scr, μmol/L	72 [60–83.5]	73 [60–82]	75 [64.5–91]	0.091
cTNT, ng/mL	0.39 [0.1–0.96]	0.11 [0.04–0.34]	0.05 [0.02–0.14]	<0.001
CKMB, U/L	48 [26–126]	26 [17–39.5]	19 [12–30.5]	<0.001
NT–proBNP, pg/mL	194.5 [104.4–628.85]	165 [68.98–319.15]	165 [43.45–241.75]	<0.001
Hemoglobin, g/L	142 [128–151.5]	145 [135–154.75]	145 [135–155]	0.012
Leukocyte count, 10^9/L	11.26 [9.35–13.29]	10.42 [8.67–12.05]	10.36 [8.69–11.61]	<0.001
Neutrophil count, 10^9/L	9.5 [7.65–11]	7.8 [6.03–9.4]	7.7 [6.1–9.1]	<0.001
Eosinophil count, 10^9/L	0.01 [0–0.02]	0.05 [0.04–0.07]	0.06 [0.04–0.08]	<0.001
Lymphocyte count, 10^9/L	1.2 [0.9–1.55]	1.6 [1.2–2.28]	1.6 [1.2–2.3]	<0.001
Monocyte count, 10^9/L	0.46 [0.33–0.72]	0.5 [0.4–0.67]	0.51 [0.4–0.67]	0.325
Platelet count, 10^9/L	205 [165–248]	208 [171.75–246.5]	206 [171–247]	0.805
Mean platelet volume, fL	10.5 [10–11.2]	10.5 [10.1–11.1]	10.5 [10.1–11.1]	0.879
Thrombocytocrit, %	0.22 [0.18–0.26]	0.22 [0.18–0.25]	0.22 [0.18–0.25]	0.839
Platelet distribution width, %	11.8 [10.7–12.7]	11.6 [10.8–13.0]	11.7 [10.9–13.1]	0.991

Data are presented as median (interquartile range) or number (percentage). CVD, cardiovascular disease; MI, myocardial infraction; PCI, percutaneous coronary intervention; CABG, coronary artery bypass graft; HDL-C, high density lipoprotein-cholesterol; LDL-C, low density lipoprotein-cholesterol; LVEF, left ventricular ejection fraction; SBP, systolic blood pressure; DBP, diastolic blood pressure; Scr, Serum creatinine; cTNT, cardiac troponin T; CKMB, creatine kinase-myocardial band; NT-proBNP, N-terminal pro B-type natriuretic peptide.

Table 2 Angiographic and procedural characteristics

Variables	Tertile of EMR on admission			P value
	T1 (<0.06; n=141)	T2 (0.06-0.18; n=140)	T3 (>0.18; n=145)	
Symptom to hospital time ≤3 h	11 (7.8)	31 (22.1)	44 (30.3)	<0.001
Killip class >2	7 (4.96)	4 (2.86)	3 (2.07)	0.171
Anterior myocardial infarction	62 (44.0)	51 (36.4)	54 (37.2)	0.247
Chronic total occlusion	13 (9.15)	13 (9.3)	13 (8.97)	0.94
Multi-vessel disease	111 (78.72)	104 (74.29)	112 (77.24)	0.772
Left main artery lesion	2 (1.42)	3 (2.1)	2 (2.1)	0.687
Visually thrombus	103 (73.0)	110 (78.6)	115 (79.3)	0.211
Gensini score	68 [46–88]	63 [43–82.75]	63 [43–82]	0.514
Use of DES or PTCA	2 (1.42)	8 (5.71)	5 (3.45)	0.36
Stent diameters, mm*	3 [2.75–3.5]	3.13 [2.84–3.5]	3.13 [2.8–3.5]	0.361
Stent length, mm*	28 [23–33]	28 [23–32]	28 [23–32]	0.452
No reflow	7 (4.96)	6 (4.29)	7 (4.83)	0.958
Medication at discharge				
β-blocker	134 (95.0)	137 (97.9)	136 (93.8)	0.602
Statin	141 (100.0)	139 (99.3)	145 (100.0)	0.991
ACEI/ARB	135 (95.7)	136 (97.1)	138 (95.2)	0.8

Data are presented as median [interquartile range] and number (percentage). *n=411. DES, drug eluting stent; PTCA, percutaneous transluminal coronary angioplasty; ACEI/ARB, angiotensin converting enzyme inhibition/angiotensin II receptor blockers.

Table 3 Correlation between EMR and cardiac biomarkers and hematological parameters.

Variables	EMR	
	R	P value
C-reactive protein, mg/L	–0.162	0.001
cTNT, ng/mL	–0.435	<0.001
CKMB, U/L	–0.47	<0.001
NT-proBNP, pg/mL	–0.219	<0.001
Hemoglobin, g/L	0.161	0.001
Leukocyte, 10^9/L	–0.259	<0.001
Neurophil, 10^9/L	–0.474	<0.001
Lymphocyte, 10^9/L	0.477	<0.001
Triglyceride, mmol/L	0.099	0.042
Eosinophil, 10^9/L	0.951	<0.001
LVEF	0.12	0.013

cTNT, cardiac troponin T; CKMB, creatine kinase-myocardial band; NT-proBNP, N-terminal pro B-type natriuretic peptide; LVEF, left ventricular ejection fraction.

patients with STEMI who were treated with P-PCI. To the best of our knowledge, this is the first study to introduce EMR and prove the lower EMR was associated with higher one-month and long-term mortality in STEMI patients who underwent P-PCI.

In the previous studies, researchers have accumulated lots of evidence to illustrate the relationship between monocytes and pathological process of STEMI. First, monocytes are key cells in the process of atherosclerosis. The dysfunction of endothelial cells will lead to the recruitment of inflammatory cells in the arterial wall (17). After the recruitment, some monocytes can be activated into macrophages. The monocytes and macrophages subsequently release more inflammatory factors such as interleukin-6 (IL-6), tumor necrosis factor alpha (TNF-α) attracting more inflammatory cells (18). Several clinical studies have showed that elevated monocytes were associated with left ventricular dysfunction in STEMI patients, which suggests that monocytes might affect the process of left ventricular remodeling (19). Erkol *et al.* also found that inhibiting monocytes gathering to infarcted

Table 4 One-month and long-term follow-ups of the study patients

Variables	Tertile of EMR on admission			P value
	T1 (<0.06; n=141)	T2 (0.06-0.18; n=140)	T3 (>0.18; n=145)	
Long-term MACE	15 (10.64)	9 (6.43)	5 (3.45)	0.016
Long-term mortality	10 (7.09)	4 (2.9)	1 (0.7)	0.003
Long-term TVR	1 (0.71)	1 (0.71)	1 (0.69)	0.984
Long-term myocardial reinfarction	4 (2.84)	4 (2.9)	3 (2.1)	0.681
Short-term MACE	8 (5.67)	3 (2.14)	0	0.003
Short-term mortality	6 (4.25)	3 (2.14)	0	0.012
Short-term myocardial reinfarction	2 (1.42)	0	0	0.081
In-hospital cost, 10^4 RMB	5.62 [4.82–7.09]	5.26 [4.48–6.97]	5.17 [4.45–6.87]	0.08
In-hospital duration, days	8 [6–9]	7 [6–9]	7 [6–9]	0.528

Data are presented as median [interquartile range] and number (percentage). MACE, major adverse cardiac event; TVR, target vessel revascularization.

Table 5 Univariate and multivariate Cox proportional hazards analysis for 1-month mortality

Variables	Univariate analysis			Multivariate analysis		
	Unadjusted HR	P	95 CI	Adjusted HR	P	95 CI
Age, years	1.114	0.003	1.038–1.196	1.109	0.008	1.027–1.196
Female	0.293	0.067	0.079–1.091			
Diabetes	2.382	0.196	0.640–8.869			
Hypertension	0.817	0.763	0.219–3.042			
Hyperlipidemia	0.592	0.621	0.074–4.734			
Stroke	0.679	0.715	0.085–5.426			
Previous PCI	2.104	0.483	0.263–16.823			
Killip class >2	43.400	<0.001	11.639–162.05	11.385	0.002	2.430–53.332
Platelet, 10^9/L	1.012	0.016	1.002–1.021	1.012	0.004	1.004–1.021
Scr, μmol/L	1.011	<0.001	1.006–1.016	1.009	0.032	1.001–1.018
Admission SBP, mmHg	0.962	<0.001	0.942–0.983			
Symptom to hospital time ≤3 h	1.129	0.880	0.235–5.435			
Anterior myocardial infarction	1.247	0.742	0.335–4.646			
Left main artery lesion	0.048	0.781	0–970			
Chronic total occlusion	0.043	0.534	0–874.98			
Multi-vessel disease	1.065	0.937	0.221–5.126			
Gensini score	1.001	0.882	0.983–1.020			
EMR value ≥0.102 (median)	0.120	0.046	0.015–0.961	0.097	0.04	0.010–0.899

HR, hazard ratio; CI, confidence interval; PCI, percutaneous coronary intervention; Scr, Serum creatinine; SBP, systolic blood pressure; EMR, eosinophil-to-monocyte ratio.

Table 6 Univariate and multivariate Cox proportional hazards analysis for long-term mortality

Variables	Univariate analysis			Multivariate analysis		
	Unadjusted HR	P	95 CI	Adjusted HR	P	95 CI
Age, years	1.087	0.001	1.033–1.144	1.10	0.002	1.035–1.17
Female	0.346	0.044	0.124–0.977			
Diabetes	1.467	0.484	0.501–4.293			
Hypertension	0.982	0.972	0.349–2.758			
Hyperlipidemia	0.738	0.689	0.166–3.269			
Stroke	0.579	0.09	0.307–1.089			
Previous PCI	2.750	0.18	0.620–12.21			
Killip class >2	28.120	<0.001	9.860–80.200	11.796	<0.001	3.576–38.912
Platelet, 10^9/L	1.01	0.018	1.002–1.018	1.011	0.004	1.003–1.018
Scr, μmol/L	1.009	<0.001	1.005–1.014	1.009	0.011	1.002–1.016
Admission SBP, mmHg	0.977	0.023	0.957–0.997			
Symptom to hospital time ≤3 h	0.612	0.519	0.138–2.714			
Anterior myocardial infarction	1.010	0.985	0.359–2.839			
Left main artery lesion	8.910	0.004	2.003–39.624			
Chronic total occlusion	1.558	0.56	0.351–6.912			
Multi-vessel disease	1.217	0.761	0.344–4.312			
Gensini score	1.014	0.021	1.002–1.026	1.016	0.018	1.003–1.029
EMR value ≥0.102 (median)	0.246	0.03	0.069–0.872	0.176	0.013	0.045–0.694

HR, hazard ratio; CI, confidence interval; PCI, percutaneous coronary intervention; Scr, serum creatinine; SBP, systolic blood pressure; EMR, eosinophil-to-monocyte ratio.

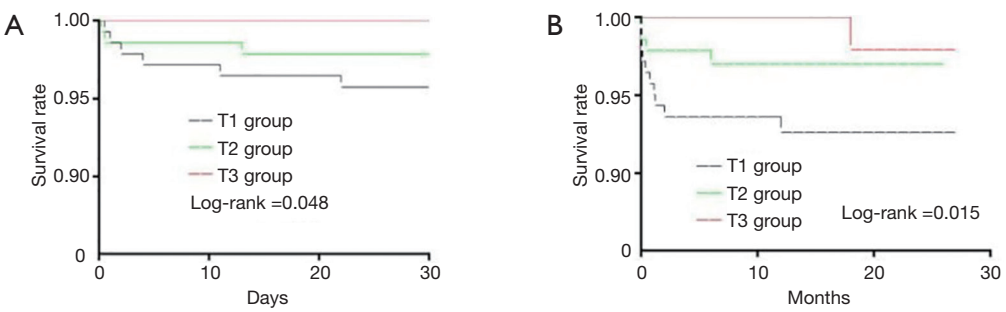

Figure 2 Kaplan-Meier survival curve for 1-month (A) and long-term all-cause mortality according to tertile of eosinophil-to-monocyte ratio on admission.

myocardium might improve ventricular function (20).

An emerging finding of eosinophils is its effect on regulating the inflammation progress. Eosinophils act an important role in the initiation, progression, and rupture of thrombus. Eosinophils help platelets adhering to the injured vessel wall (21), and they can release immunosuppressive cytokines like IL-10, IL-4, and IL-13 which are suggested to modulate the inflammatory response (22). Atherosclerotic plaques rupture, a basic inflammatory pathogenesis of STEMI, involves the infiltration of eosinophils into the

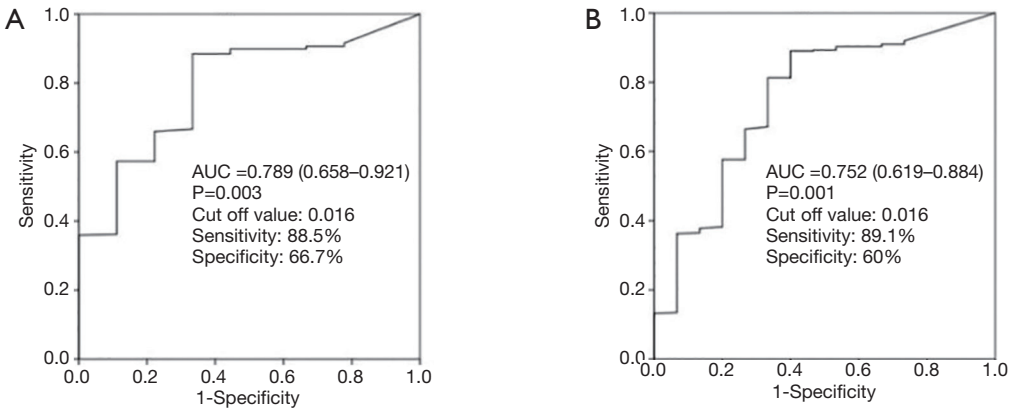

Figure 3 Receiver operator characteristic curves of eosinophil-to-monocyte ratio for predicting 1-month (A) and long-term all-cause mortality (B).

infarcted myocardium (14). In contrast to other inflammatory parameters, eosinophil was seldom discussed. Jiang found that circulatory eosinophils reflected the extent of myocardial infarction (23). Besides, eosinophils were found to be a useful biomarker for risk stratification of CAD patients and predicting 6-month mortality (24).

As the single inflammatory cell is unable to summarize the overall systematic inflammation, new indexes were proposed by combining different subtypes of the leukocyte. NLR (25), LMR (9,26) and eosinophil to leukocyte ratio (27) were reported to be independently associated with MACEs in STEMI patients with P-PCI. EMR is a totally new parameter. We found that a lower EMR was associated with a higher risk after STEMI, which may result from an increased monocyte count or a decreased eosinophil count. However, in our study, the monocyte count was comparable among the three groups and EMR values positively and strongly correlated with eosinophil counts. In this case, we speculated that a lower EMR may represent a reduction in eosinophil count. The low eosinophil count may due to the infiltration of eosinophils into the infarcted myocardium and coronary thrombi, which resulted in the decrease in peripheral circulating eosinophils (14). Besides, an increase in the cortisol concentration caused by an acute stress response to STEMI may also lower the peripheral eosinophil count (27). Both eosinophils and monocytes are inflammation and immune cells and related with immunosuppressive cytokines. Eosinophils were found to be able to lower the viability or activation of T cells (28).

In the present study, we demonstrated that a lower EMR on admission was associated with higher one-month and long-term mortality in patients with STEMI who underwent primary PCI. Besides, EMR was negatively related with cTNT and CK-MB. Thus, to some extent, EMR might have the potentiality to indicate the infarct size of the myocardium. Moreover, the Scr level on admission was independent risk factor of long-term total mortality which was in accord with the previous studies (29). Similarly, we also notice the relationship between the platelet count and 1-month as well as long-term mortality. Age, another parameter which was suggested to be associated with both 1-month and long-term mortality, was already widely used as a predictive factor in various prognosis analysis models (30).

Conclusions

We demonstrated that a lower EMR on admission was associated with higher 1-month and long-term mortality in patients with STEMI who underwent primary PCI. EMR could be a simple, useful, and inexpensive marker for risk stratification of STEMI patients.

Limitations

First, it was a single-center observational with inherent bias. Second, patients with very severe disease who died before undergoing coronary artery angiography were missed. Third, some patients were poorly compliant with use of prescribed medications. Larger-scale, prospective, and randomized clinical trials are required to confirm our findings.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

Ethical Statement: The study protocol was approved by our institutional ethics committee (Ethic Approval: Bro12-030). All patients provided informed consent.

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