Journal of Inflammation Research

Dovepress
Taylor & Francis Group

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ORIGINAL RESEARCH

Associations Between Albumin/Neutrophil-to-Lymphocyte Ratio Score and New-Onset Atrial Fibrillation in Patients with Acute Myocardial Infarction Undergoing PCI

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Background: Inflammation was associated with the increased risk of atrial fibrillation (AF). As a novel inflammatory indicator, albumin/neutrophil-to-lymphocyte ratio score (ANS) has been demonstrated to associate with coronary artery disease. However, the relationship between ANS and new onset atrial fibrillation (NOAF) in patients with acute myocardial infarction (AMI) underwent PCI was not determined.

Methods: A total of 2410 AMI patients underwent PCI were consecutively included between March 2020 and December 2023. Patients were divided into NOAF group and control group according to the occurrence of NOAF during hospitalization. The ANS was calculated and analyzed, so as to determine its predictive value in the presence of NOAF in AMI patients after PCI.

Results: In total, 88 (3.7%) individuals developed NOAF during hospitalization. We found that NOAF was associated with older age, greater LA, higher NT-proBNP, ANS and Killip ≥ 2. The ANS exhibited an accurately predictive value for the NOAF (area under the curve [AUC], 0.695; 95% CI, 0.649–0.740, P < 0.001). Moreover, when divided into three groups according to the tertile of ANS, patients in tertile 1 (lowest in ANS) showed a 2.214-fold increased risk of NOAF in comparison to those in the tertile 3 (HR, 2.214; 95% CI 1.804–5.101; P = 0.029).

Conclusion: ANS is a robust tool for the prediction of NOAF in AMI patients underwent PCI. Therefore ANS could be used for risk prediction and optimal management for NOAF in AMI patients after PCI.

Keywords: albumin/neutrophil-to-lymphocyte ratio score, new-onset atrial fibrillation, acute myocardial infarction, PCI

Introduction

Atrial fibrillation (AF) has been suggested as one of the most commonly encountered heart rhythm disorders in patients with acute myocardial infarction (AMI). In earlier years, the incidence of new onset atrial fibrillation (NOAF) varied from 6% to 21% in different clinical studies with varied comorbidities.[1] Due to the episodes of AF are frequently silent, so the incidence of NOAF reported are more likely to be underestimated.[2] The occurrence of AF post AMI may in turn aggravate myocardial ischemia and heart failure. Moreover, the additional anticoagulant therapy may bring in an increased risk of bleeding events.[3] Accumulating studies have suggested that AF post AMI is related to a worse short term as well as long-term prognosis.[4–7] The pathogenesis of NOAF in AMI patients is still quite complex and undetermined, nonetheless, the risk factors including advanced age, female sex and cardiac dysfunction may play a role.[8] The main mechanisms of NOAF in AMI patients are attributed to hemodynamic changes, thromboembolism, and secondary inflammatory responses.[8–10] Accumulating studies demonstrate the close association between inflammatory responses and AF, suggesting that the occurrence of AF is a consequence of the necrosis and fibrosis caused by inflammatory processes, which bring in trial dysrhythmias directly through fluctuations in membrane potential.[11,12] As

Received: 30 October 2024
Accepted: 6 December 2024
Published: 3 January 2025

the most widely used inflammatory indicator, CRP was suggested to associate with AF coincided in patients receiving coronary artery bypass surgery.[13] Nonetheless, the exact etiological for the presence of AF in AMI patients following PCI is still not determined.

The neutrophil-to-lymphocyte ratio (NLR) included the two different immune responses, the innate immunity mainly from neutrophils, and the adaptive immune response from the lymphocytes.[14] As an new indicator for the assessment of local or systemic inflammatory status, neutrophil-to-lymphocyte ratio (NLR) has been widely discussed in the cardiovascular diseases. The ENGAGE AF- TIMI 48 trial revealed that increased NLR was associated with increased risk of bleeding, cardiovascular events, and mortality in patients with AF.[15] A more recent study from China suggested that NLR was a valuable predictor for NOAF in AMI patients.[16] As the most abundant protein in human, the albumin possesses antioxidant and anti-inflammatory effects, inhibiting platelet aggregation and activation, thereby influencing plasma viscosity.[17] Accumulating studies had proven that a decreased albumin level was associated with a poor prognosis in patients with coronary artery disease after PCI.[18,19] Recently, a newly developed inflammatory indicator albumin/NLR score (ANS) has been suggested, which displayed a prognostic value in patients with colorectal cancer.[20] Dr Chen et al discovered that ANS could be used as a risk prediction tool for the screening of the patients with suspected or subclinical coronary artery disease.[21] Given the inflammatory effect in the development of cardiovascular disease and the inflammation in the development of AF, we speculated that ANS may also relate to the presence of NOAF in AMI patients. Therefore, in this study, we aimed to explore the relationship between ANS and occurrence of NOAF in AMI patients.

Methods

Study Population

We consecutively enrolled 2410 AMI patients receiving PCI from March 2020 to December 2023 in this study, including 1543 non-ST segment elevation myocardial infarction (NSTEMI) and 867 ST segment elevation myocardial infarction (STEMI). The study flowchart and the exclusion criteria were shown in Figure 1. STEMI and NSTEMI were diagnosed according to the relevant guidelines of the European Society of Cardiology.[22,23] All the patients with STEMI received primary PCI according to the relevant guidelines. The patients with NSTEMI were also received optimal PCI according to the risk stratification. NOAF was defined as AF episodes lasting more than 30s, as recorded by electrocardiogram (ECG), bedside telemetry or electrocardiographic continuous monitoring during hospitalization.[8] This study was approved by the Ethics Committee of The General Hospital of Ningxia Medical University according to the principles of the Declaration of Helsinki, and the informed consent of all patients was obtained.

Clinical and Laboratory Data Assessments

After admission, we obtained the data from the Hospital Information System (HIS) of our hospital. The baseline characteristics of the studied patients including age, gender, body mass index (BMI) were recorded. The complications including hypertension, diabetes, stroke, smoking and drinking status, previous PCI, family history of CAD and current drug use were also acquired in detail. The laboratory test including the blood routine test, liver and renal examinations,

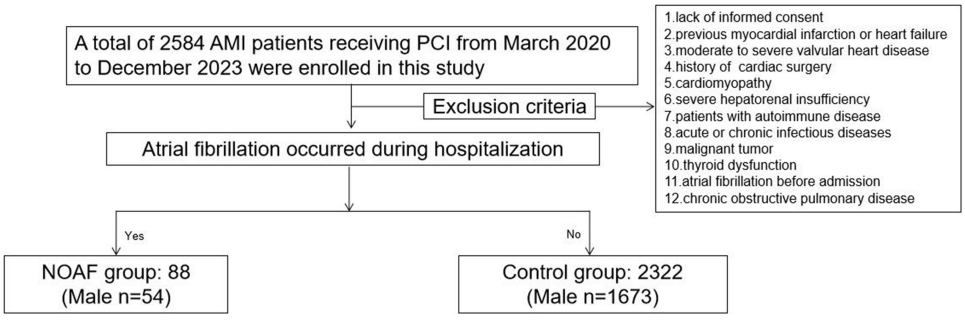

Figure 1 The study flowchart.

lipid Profile, uric acid, N-Terminal pro-brain natriuretic peptide (NTproBNP) and cardiac troponin I were also recorded. The echocardiography was performed on admission or after primary PCI. ANS was calculated as albumin-to-NLR ratio.

Interventional Procedures

All the individuals received the coronary angiography via the radial artery or femoral route. The culprit vessel was determined by the experienced interventional cardiologists according the revelant guidelines. Multivessel disease was defined as at least two main vessels have stenosis ≥50%.[8] The lesion characteristics and procedural characteristics were collected and analyzed. All patients received informed consent before the procedure.

Statistical Analysis

Statistical analysis was performed using SPSS version 20.0 (IBM, USA). The Kolmogorov–Smirnov test was performed to determine the distribution characteristics of continuous variables. The continuous variables were expressed as mean and standard deviation, or median and interquartile range, which were compared by Student *t* test or Mann–Whitney test as appropriate. Categorical variables were expressed as rates or percentages, which were compared using the chi-square test or Fisher's exact test between NOAF group and the control group. Univariate logistic regression was used to determine the factors associated with NOAF. We performed a multicollinearity test on potential risk factors determined by the univariate analysis and the parameters with $p< 0.1$ between the two groups. The selected variables with a variance expansion factor (VIF) < 3 were then analyzed in the multivariate analysis so as to determine independent risk factors for NOAF in AMI patients after PCI. The receiver operating characteristic (ROC) curves were performed to investigate the predictive value of ANS for NOAF in AMI patients after PCI. Pearson or Spearman correlation analysis, as appropriate, was performed to investigate the correlation between ANS and the NOAF risk factors. All tests were two-sided, and the statistical significance was set at $p < 0.05$.

Results

Baseline and Clinical Characteristics

A total of 2410 AMI patients receiving PCI from March 2020 to December 2023 were in this study, including 1543 NSTEMI and 867 STEMI patients. NOAF occurred in 88 individuals, accounting for 3.7% (88/2410) of the patients. The baseline and clinical characteristics, medications and echocardiographic parameters were displayed in Table 1. There were no significant differences between NOAF and the controls with regard to gender, current smoking, alcohol use, Diabetes Mellitus, hypertension, previous Stroke, previous PCI, family history of CAD, and BMI ($p > 0.05$) (Table 1). Compared with the controls, patients with NOAF tended to be older and more likely to have a higher Killip ≥ 2, left atrial (LA), and left ventricular end systolic volume (LVESV) ($p < 0.05$) (Table 1). Patients with NOAF had a lower left ventricular ejection fraction (LVEF) ($p < 0.05$) (Table 1). The Laboratory parameters were displayed in Table 2. There were no significant differences between NOAF and the controls in terms of white blood cell (WBC) count, neutrophil count, lymphocyte count, monocyte count, fasting blood glucose (FBG), uric acid, total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG) and cardiac troponin I (TnI) ($p > 0.05$) (Table 2). However, patients with NOAF had an increased creatinine, N-terminal pro B-type natriuretic peptide (NT-proBNP), NLR and ANS, while a decreased albumin (ALB) ($p < 0.05$) (Table 2).

Angiographic and Procedural Characteristics of the Studied Patients

Angiographic and procedural characteristics of the patients in the two groups were displayed in Table 3. The infarction-related arteries were comparable between the two groups ($p > 0.05$) (Table 3). No significant differences were observed in terms of multi-vessel disease, coronary spontaneous reperfusion, coronary vessel diameter, the stent length and the incidence of slow flow or no reflow ($p > 0.05$) (Table 3).

Association of the Markers with the Risk of NOAF

We selected numerous indicators in the univariate regression analysis, including the parameters with statistical significance between the two groups and the parameters with a p value <0.1, so as to make the best possible to include the factors

 https://doi.org/10.2147/JIR.S500743

Table 1 Clinical Characteristics of Study Population

Variables	NOAF Group (n=88)	Control Group (n=2322)	P-value
Age(years)	69.5(63.3,75.0)	62.0(53.0,70.0)	<0.001
Gender(male), n(%)	54(61.4%)	1673(72.0%)	0.04
Current smoker, n(%)	40(45.5%)	1246(53.7%)	0.16
Alcohol use, n(%)	24(27.3%)	697(30.0%)	0.64
Diabetes Mellitus, n(%)	31(35.2%)	610(26.3%)	0.07
Hypertension, n(%)	48(54.5%)	1027(44.2%)	0.06
Previous Stroke, n(%)	17(19.3%)	326(14.0%)	0.16
Previous PCI, n(%)	14(15.9%)	204(15.4%)	0.88
Family history of CAD, n(%)	12(13.6%)	298(12.8%)	0.75
BMI (kg/m^2)	24.5(21.5,27.1)	25.0(22.9,27.3)	0.34
Killip≥2	18(20.5%)	133(10.1%)	0.006
Clinical presentation			
NSTEMI	54(61.4%)	1489(64.1%)	0.66
STEMI	34(38.6%)	833(35.9%)	
Medication history			
ACEI/ARB/ARNI, n (%)	62(70.5%)	1630(70.2%)	1
B-blocker, n (%)	52(59.1%)	1486(64.0%)	0.37
Statines, n (%)	87(98.9%)	2301(99.1%)	0.56
Calcium channel blockers, n (%)	20(22.7%)	619(26.7%)	0.46
Spirolactone, n (%)	18(20.5%)	519(22.4%)	0.79
SGLT-2i, n (%)	32(36.4%)	951(41.0%)	0.44
Diuretics, n (%)	19(%)	604(%)	0.68
Echocardiographic analysis			
LA	40.0(38.0,44.0)	39.0(36.0,41.0)	<0.001
LVEDV	100.5(85.0,131.5)	100.0(87.0,118.0)	0.18
LVESV	59.5(46.0,81.8)	54.5(44.0,68.0)	0.02
LVEF(%)	41.0(37.0,45.8)	44.0(39.0,50.0)	0.001

Abbreviations: PCI, percutaneous coronary intervention; CAD, coronary artery disease; BMI, body mass index; ACEI, angiotensin converting enzyme inhibitors; ARB, angiotensin II receptor blocker, ARNI, angiotensin receptor enkephalinase inhibitor; SGLT-2i, sodium-glucose co-transporter type-2 inhibitors; LA, left atrium; LVEDV, left ventricular end diastolic volume; LVESV, left ventricular end systolic volume; LVEF, left ventricular ejection fraction.

associated with NOAF. The univariate regression analysis suggested that age, LA, LVEF, NT-proBNP/100, ANS, and Killip ≥ 2 were associated with NOAF in AMI patients after PCI ($p < 0.05$) (Table 4). The multicollinearity analysis was performed to determine the indicators for a further multivariate analysis with a variance inflation factor (VIF)>3. Consequently, we selected age, LA, LVEF, NT-proBNP/100, ANS, and Killip ≥ 2 for further multivariate analysis

Table 2 Laboratory Analysis of Study Population

Variables	NOAF Group (n=88)	Control Group (n=2322)	P-value
WBC count (109/L)	8.4(6.9,10.1)	8.1(6.6,9.9)	0.34
Neutrophil count, (109/L)	6.0(4.7,7.7)	5.6(4.2,7.4)	0.14
Lymphocyte count, (109/L)	1.6(1.2,1.8)	1.6(1.2,2.1)	0.051
Monocyte count, (109/L)	0.6(0.4,0.8)	0.6(0.4,0.7)	0.10
FBG, mmol/L	6.5±2.1	6.6±2.3	0.52
Cr, mmol/L	74.5(64.3,94.4)	69.9(59.2,82.0)	0.002
Uric acid, mmol/L	345.5(278.0,446.3)	333.0(271.0,401.0)	0.16
ALB, g/L	36.5(33.4,39.4)	38.4(35.8,40.8)	<0.001
TC, mmol/L	4.3(3.7,5.0)	4.5(3.8,5.2)	0.13
TG, mmol/L	1.3(0.8,1.6)	1.4(1.0,2.1)	0.10
LDL-C, mmol/L	2.8(2.1,3.4)	2.9(2.3,3.4)	0.43
HDL-C, mmol/L	1.0(0.8,1.2)	1.0(0.8,1.1)	0.18
NT-proBNP, pg/mL	4315.5±6425.3	1885.8±3529.6	<0.001
cTnI, (ug/L)	2.5 (0.5–13.8)	2.6 (0.6–13.8)	0.82
NLR	3.7(2.7,5.8)	3.3(2.4,4.7)	0.008
ANLR	9.1(4.4,12.6)	10.2(4.7,14.9)	0.023

Abbreviations: WBC, white blood cell; FBG, fasting blood glucose; Cr, creatinine; ALB, albumin; TC, total cholesterol; TG, triglyceride; LDL-C, lowdensity lipoprotein cholesterol; HDL-C, high-density lipoprotein cholesterol; NT-proBNP, N-terminal proB-type natriuretic peptide; cTnI, cardiac troponin I; NLR, neutrophil-to-lymphocyte ratio; ANLRS, albumin/neutrophil-to-lymphocyte ratio score.

Table 3 Angiographic Characteristics of the Studied Patients

Variables	NOAF Group (n=88)	Control Group (n=2322)	P-value
Infarction-related artery			
LAD, n(%)	26(29.5%)	653(28.1%)	0.81
LCX, n(%)	16(18.2%)	378(16.3%)	
RCA, n(%)	46(52.3%)	1291(55.6%)	
Multivessel disease	21(23.9%)	512(22.0%)	0.70
Spontaneous coronary reperfusion	17(19.3%)	498(21.4%)	0.69
Reference diameter, mm	3.2±0.5	3.3±0.5	0.48
Maximal stent length, mm	35.4±16.7	34.0±15.4	0.60

Abbreviations: LAD, left anterior descending artery; LCX, left circumflex artery; RCA, right coronary artery.

https://doi.org/10.2147/JIR.S500743

Table 4 Univariate and Stepwise Multivariate Logistic Regression Analysis of BUs

	Univariate Analysis			Multivariate Analysis		
	OR	**95% CI**	**P value**	**OR**	**95% CI**	**P value**
Female	1.623	1.047–2.517	0.030	1.015	0.622–1.656	0.935
Age	1.060	1.039–1.081	0.001	1.044	1.022–1.066	<0.001
Diabetes Mellitus	0.777	0.442–1.366	0.288			
Hypertension	0.715	0.391–1.307	0.276			
LA	1.127	1.073–1.184	<0.001	1.095	1.038–1.154	0.001
LVEF	0.970	0.949–0.991	0.006	0.996	0.968–1.025	0.996
NT-proBNP/100	1.008	1.005–1.011	<0.001	1.004	1.001–1.009	0.020
ANLRS	0.894	0.875–0.974	0.002	0.887	0.871–0.970	0.042
Tertile 3	Reference					
Tertile 2	0.924	0.892–1.019	0.126			
Tertile 1	2.214	1.804–5.101	0.029			
Killip≥2	1.870	1.405–2.89	<0.001	1.632	1.173–2.273	0.006
Cr	1.002	1.000–1.004	0.158			

Abbreviations: LA, left atrium; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal proB-type natriuretic peptide; ANLRS, albumin/neutrophil-to-lymphocyte ratio score; Cr, creatinine.

Table 5 Collinearity Diagnostics for the Variables Included in the Multivariate Logistic Regression Analysis

	Tolerance	**VIF**
Age	0.915	1.093
LA	0.946	1.057
LVEF	0.877	1.140
NT-proBNP/100	0.575	1.739
ANLR	0.961	1.041
Cr	0.668	1.498

Abbreviations: VIF, variance inflation factor; LA, left atrium; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal proB-type natriuretic peptide; ANLRS, albumin/neutrophil-to-lymphocyte ratio score; Cr, creatinine.

(Tables 4 and 5). After multiple factors were included, we found that a lower ANS was a predictor for the presence of NOAF in AMI patients after PCI (odds ratio [OR], 0.887; 95% confidence interval [CI], 0.871–0.970, P = 0.042) (Table 4). Moreover, when divided into three groups according to the tertile of ANS, patients in tertile 1 (lowest in ANS) showed a 2.214-fold increased incidence of NOAF in comparison to those in the tertile 3 (HR, 2.214; 95% CI 1.804–5.101; P = 0.029). In addition, we also discovered that age (OR, 1.047; 95% CI, 1.025–1.069, P < 0.001), LA (OR, 1.099; 95% CI, 1.044–1.157, P < 0.001), NT-proBNP (OR, 1.005; 95% CI, 1.000–1.009, P = 0.020), Killip ≥ 2 (OR, 1.635; 95% CI, 1.171–2.283, P = 0.004) were independent predictors for NOAF in AMI patients underwent PCI (Table 4).

The ROC analysis suggested that ANS provided an accurate predictive value for NOAF (AUC, 0.695; 95% CI, 0.649–0.740, P < 0.001). An ANS value of <11.8 distinguished NOAF with 53.1% sensitivity and 67.1% specificity. The ANS had a significantly superior predictive value than age (AUC, 0.581; 95% CI, 0.521–0.642), LA (AUC, 0.617; 95% CI, 0.556–0.678) or NT-proBNP (AUC, 0.679; 95% CI, 0.620–0.739) (Figure 2).

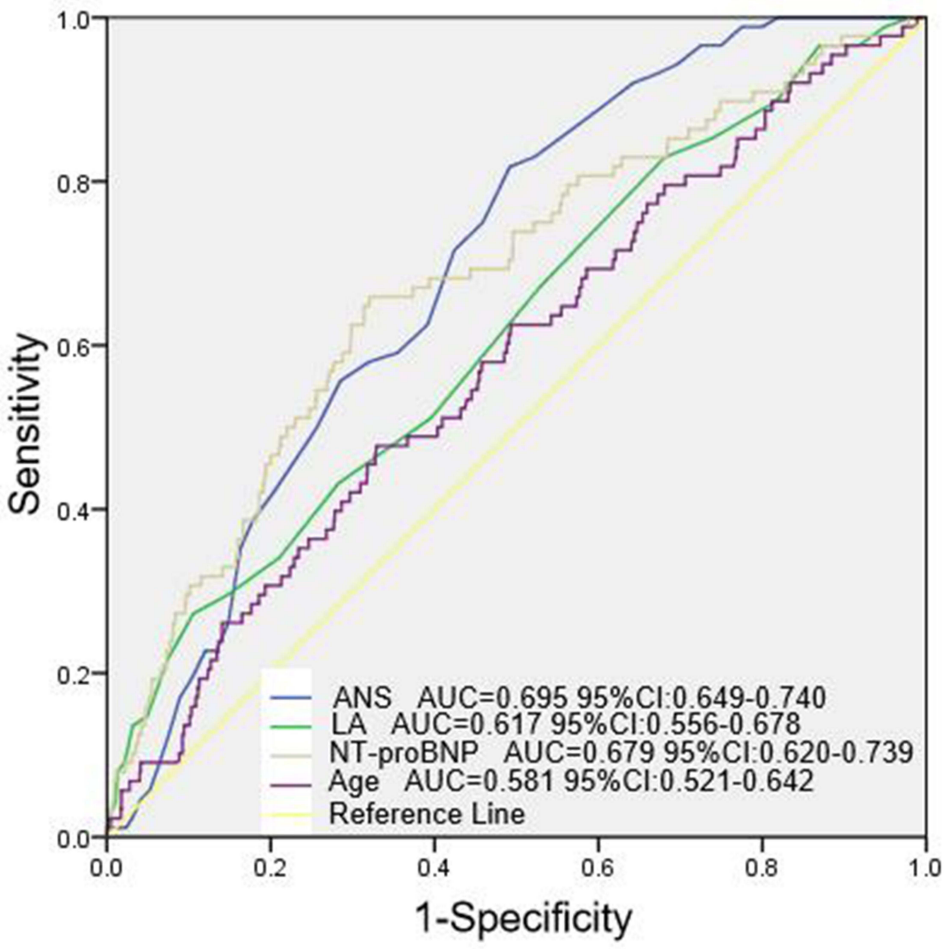

Figure 2 Receiver operating characteristic (ROC) curves for assessing the predictive value of the indicators for the presence of NOAF.

Correlation Between the ANS and Risk Factors for NOAF

To explore potential relationships between ANS and risk factors for NOAF, a Spearman correlation test was performed. We discovered that the ANS correlated well with age ($r=-0.124$; $p < 0.001$), TNT-proBNP ($r=-0.161$; $p < 0.001$), creatinine ($r=-0.088$; $p < 0.001$), LA ($r=-0.049$; $p = 0.016$) and LVEF ($r = 0.152$; $p < 0.001$) in AMI patients after PCI (Table 6).

Table 6 Correlation Between the ANLRS and Other Variables

Variables	Coefficient	P-value
Age	−0.124	<0.001
Uric acid	−0.012	0.578
NT-proBNP	−0.161	<0.001
Creatinine	−0.088	<0.001
LA	−0.049	0.016
LVEF	0.152	<0.001

Abbreviations: NT-proBNP, N-terminal proB-type natriuretic peptide; Cr, creatinine; LA, left atrium; LVEF, left ventricular ejection fraction.

https://doi.org/10.2147/JIR.S500743

Discussion

In the present study, we investigated the potential predictive factors for NOAF in AMI patients after PCI. We discovered that NOAF was independently associated with older age, greater LA size, higher NT-proBNP, ANS and Killip ≥ 2. Moreover, when divided into three groups according to the tertile of ANS, patients in tertile 1 (lowest in ANS) showed a 2.214-fold increased risk of NOAF in comparison to those in the tertile 3.

NOAF is quite a common arrhythmia in patients with AMI underwent PCI. In the earlier years, due to a low incidence of primary PCI, the incidence of NOAF reported varied from 6% to 21% in different clinical studies with varied comorbidities.[1] However, with the development of international therapy, especially for the guideline based optimal management of AMI, the incidence of NOAF reported was decreased significantly. Now the incidence of NOAF reported varied from 3.1% to 16.7% in AMI patients after PCI.[24–27] NOAF after AMI carries a substantial future risk a prolonged hospitalization and a poor prognosis.[8,28–30] Moreover, Guenancia C et al suggested that patients diagnosed NOAF during AMI had an increased risk of recurrence of AF during long term follow-up.[31] In addition, a recent study demonstrated that NOAF during AMI is a more aggravating diagnosis than other pre-existing types of AF.[32] So NOAF during AMI should not be considered as a benign phenomenon. Therefore, early identification of patients at high risk of NOAF underwent PCI is clinically important for providing preventive measures and optimal managements.

The etiology of NOAF in AMI is quite complex and undetermined. The coronary blood flow disorders, embolic effects, atrial ischemia or infarction, local and systemic inflammation, and hormone activation were reported to associate with the presence of NOAF in AMI.[8,33] However, these factors serving as the triggering factors of NOAF could bring in a structural and electrical remodeling of the atrium, which contributes to the occurrence of NOAF. In clinical practice, it is more important to determine the predictive factors for NOAF, so as to improve the management of these patients. However, the predictors for NOAF varied greatly and quite complex, with some indicators not easily acquired. So, it is quite necessary for us to explore the potential easily acquired indicators for the prediction of NOAF in AMI after PCI.

Previous studies have demonstrated the association between advanced age and the presence of NOAF in AMI patients after PCI.[8,34] Similarly, in the present study, we also discovered that patients with NOAF tended to be older and an advanced age is related to an increased risk of NOAF in AMI patients after PCI. It had been reported that a large LA size reflected a poor left ventricle diastolic dysfunction, which is associated with elevated LV filling pressures.[35] The enlargement of LA represents a structural and electrophysiological atrial remodeling, which was involved in the occurrence and development of AF.[36] Previous studies had demonstrated that a larger LA size was a reliable predictor for NOAF in the general population[37] and recurrence post cardioversion[38] and AF ablation.[39] In the present study, we discovered that a large LA size was associated with an increased risk of NOAF in AMI patients after PCI, which aligns with the previous study.[34,40–42]

The NT-proBNP was a well established biomarker for the assessment of LA strain, which was also been proven to relate to the presence of NOAF in the general population.[43,44] In our study, we also discovered that an elevated NT-proBNP level was linked to an increased risk of NOAF in AMI patients after PCI. The Killips class was widely used in the clinical practice to assess the cardiac function during the acute phase of AMI. A high Killips class represents a larger infarction size, which brings in an acute increase in ventricular filling pressure and triggers NOAF.[45] Moreover, NOAF in turn may further bring in the deterioration of cardiac function.[45] Similar to previous studies, we also found that high Killips class was the independent predictor for the occurrence of NOAF in AMI patients after PCI.[34,45]

The local or systemic inflammation during the acute phase of AMI is critical for cardiac repair and cardiac remodeling.[46] It is well established that inflammation is associated with the initiation and development of AF in AMI patients.[47] Notably, the local inflammatory reaction changes were also observed in atrial tissues in patients with AF.[48] The inflammatory response further leads to the endothelial dysfunction, which may stimulate the synthesis and release of various inflammatory factors.[49] As an indicator for the assessment of local or systemic inflammatory status, NLR has been proven to associate with increased risk of bleeding, cardiovascular events, and mortality in patients with AF.[15] A more recent study from China suggested that NLR was a valuable predictor of NOAF in AMI patients.[16] As the most abundant protein in human, the albumin possesses antioxidant and anti-inflammatory effects, inhibiting platelet

aggregation and activation, thereby influencing plasma viscosity.[17] The ARIC Study suggested that serum albumin level is independently inverse associated with AF in a linear pattern, although the causal effect was unclear.[50] Recently, a newly developed inflammatory indicator albumin/NLR score (ANS) has been suggested, which displayed a prognostic value in patients with colorectal cancer.[20] Dr Chen et al discovered that ANS could be used as a risk prediction tool for the screening of the patients with suspected or subclinical coronary artery disease.[21] However, the relationship between ANS and NOAF in AMI patients was not discussed. In the present study, we discovered that patients with NOAF had a high ANS level and multivariate analysis showed that ANS was an independent predictor for the occurrence of NOAF in AMI patients after PCI. We suggested that ANS value <11.8 distinguished NOAF with 53.1% sensitivity and 67.1% specificity. Moreover, ANS showed a better predictive value in the presence of NOAF than other indicators including age, LA size, or NT-proBNP. This is the first study to investigate the relationship between ANS and NOAF in AMI patients. As an easily acquired and calculated indicator in the clinical practice, ANS may provide help in the early identification of high-risk populations. Moreover, ANS could provide new targets for the primary prevention of NOAF in AMI patients after PCI.

This study had some limitations. First, although the sample size is quite large; however, the present study is a single-center study. Second, the NOAF was recorded by ECG, bedside telemetry or electrocardiographic continuous monitoring during hospitalization; however, we could not exclude the patients with silent paroxysmal AF previously, which may overestimate the incidence of NOAF in this study. Third, we only reported the occurrence of NOAF during hospitalization, the follow-up after discharge was not performed, which may underestimate the incidence of NOAF. Fourth, although we tried to include all the possible indicators associated with NOAF, still some may get escaped, which may affect the results. We also did not include the golden standard for the assessment of inflammation, such as high sensitivity C reactive protein. Fifth, we did not perform a nomogram so to acquire a predictive model for NOAF. We also did not perform an external validation in other populations. Sixth, from the prospective of statistical efficacy, the ratio of NOAF group to control group was about 1:26, we admitted this quite significant disequilibrium may reduce the statistical efficacy. Finally, although patients with a lower ANS tended to have an increased risk of NOAF; however, whether anti-inflammatory therapy could reduce this risk was not discussed in this study.

Conclusion

In the era of modern guidelines based revascularization, NOAF is still a frequent arrhythmia in AMI. The ANS, age, LA size, or NT-proBNP were independent predictors for NOAF in AMI patients underwent PCI. ANS showed a better predictive value than other indicators. As an easily acquired and calculated indicator in the clinical practice, ANS may provide help in the early identification of high-risk populations and the primary prevention of NOAF in AMI patients after PCI. However, whether anti-inflammatory could reduce the incidence of NOAF needs further investigations.

Abbreviations

AMI, acute myocardial infarction; NOAF, new-onset atrial fibrillation; NLR, Neutrophil-to-lymphocyte ratio; ANS, Albumin/Neutrophil-to-lymphocyte ratio score.

Data Sharing Statement

The data supporting the conclusions of this article will be made available by the corresponding author upon reasonable requests.

Acknowledgments

We express our gratitude to all the staff contributing to this study.

Funding

There is no funding to report.

Disclosure

The authors report no conflicts of interest in this work.

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