BMC Cardiovascular Disorders

RESEARCH

Open Access

Microcirculatory dysfunction in patients with acute anterior myocardial infarction combined with new complete right bundle branch block

Hong Liu[1,2†], Yu Yuan[1†], Yu Dong[1,2], Ying Yang[1,2], Xitong Yang[1,2], Biao Sun[1,2], Lilan Ma[1,2], Tao Li[1,2] and Xin-Hua Wu[1,2*]

Abstract

Objective This study sought to investigate clinical characteristics of acute anterior ST-segment elevation myocardial infarction (STEMI) patients complicated by new complete right bundle branch block (CRBBB) and evaluate the occurrence of microcirculatory dysfunction post-percutaneous coronary intervention (PCI).

Methods Retrospective analysis was conducted on 261 patients with acute anterior STEMI, differentiating 40 with concurrent new CRBBB (CRBBB group) from 221 without (no-CRBBB group). Data on demographics and hospitalization were collected, and clinical features and prognoses were compared. Post-PCI microcirculatory function was further characterized using coronary angiography-derived index of microcirculatory resistance (caIMR), thrombolysis in myocardial infarction (TIMI) grade flow, corrected TIMI flow frame count (CTFC) of the infarct-related artery, and ST segment regression in electrocardiograph (STR).

Results Age, Killip class, GLUC, TG, HDL, BUN, GFR, AST, ALT, WBC, TNI at admission significantly differed between groups ($P<0.05$). Incidences of in-hospital major adverse cardiovascular events and LVEF showed significant disparities ($P<0.05$). The CRBBB group exhibited higher CaIMR, lower TIMI flow, and STR ($P<0.05$). Multivariate analysis indicated TIMI ≤ grade 2 (OR = 6.833, 95% CI: 1.009 ~ 46.287, $P=0.049$), STR ≥ 50% (OR = 0.176, 95% CI: 0.051 ~ 0.606, $P=0.006$), CTFC (OR = 1.079, 95% CI: 1.009 ~ 1.155, $P=0.027$), and caIMR (OR = 1.120, 95% CI: 1.059 ~ 1.185, $P<0.001$) were independently linked to new onset of CRBBB. Complicated of new CRBBB was strongly associated with elevated CaIMR in anterior STEMI patients. (OR = 5.065, 95% CI:1.793–14.308, $P=0.002$).

Conclusion In patients with acute anterior STEMI, those with new CRBBB are at an increased likelihood of experiencing microcirculatory dysfunction.

Keywords Acute anterior ST-elevation myocardial infarction, New onset complete right bundle branch block, Microcirculatory dysfunction

†Hong Liu and Yu Yuan contributed equally to this work and are the co-first authors.

*Correspondence:
Xin-Hua Wu
13987286915@139.com

[1]Department of Cardiology, The first Affiliated Hospital of Dali University, Yunnan, China
[2]Yunnan Trans-plateau Cardiovascular Disease of Prevention and Treatment Research Center, Yunnan, China

Introduction

Acute anterior wall ST-segment elevation myocardial infarction (STEMI) is predominantly caused by the occlusion of the left anterior descending artery (LAD) and its branches [1]. Bundle branch block (BBB) is commonly associated with acute myocardial infarction due to its impact on blood supply. The right bundle branch, which is primarily perfused by the septal branch of the LAD, is more delicate and lacks branches, making it more susceptible to damage compared to the left bundle branch [2]. This anatomical difference suggests that the occurrence of complete right BBB (CRBBB) may coincide with anterior wall STEMI. The accumulating evidence suggests that BBB detected on admission electrocardiograph (ECG) is associated with higher long-term mortality in patients with acute myocardial infarction (AMI) compared to those without BBB [3, 4]. In patients with anterior wall STEMI, RBBB, as opposed to new onset left bundle branch block (LBBB), is persistently associated with adverse clinical outcomes, indicating that CRBBB may function as a more robust independent predictor of in-hospital mortality in anterior wall STEMI [5, 6].

Primary percutaneous coronary intervention (PPCI) is the recommended treatment according to clinical guidelines for STEMI, offering early mechanical reperfusion of the epicardial coronary artery. Nevertheless, clinical and animal studies indicate that a deficiency in coronary microvasculature restoration is associated with poorer outcomes, with this phenomenon occurring in approximately half of cases post-reperfusion therapy [7]. This is attributed to various factors, including reperfusion injury [8], slow-flow/no-reflow phenomenon (SF/NR) [9], microvascular obstruction [10], intramyocardial hemorrhage [11], Immune-Inflammation reaction [12] and coronary microvascular dysfunction [13]. The index of microcirculatory resistance (IMR) has emerged as a reliable indicator for evaluating microcirculatory dysfunction [14]. It is unclear whether the poor prognosis associated with acute anterior myocardial infarction complicated by new CRBBB is related to microcirculatory dysfunction. In this study, we employed the angiography-derived index of microcirculatory resistance (CaIMR) as a novel and relatively simple method to assess the microcirculatory function in anterior STEMI patients complicated by new CRBBB.

Materials and methods

2.1 Study Design and Population: A total of 261 patients were recruited from the Department of Cardiology at the First Affiliated Hospital of Dali University for undergoing drug-eluting stent implantation during primary percutaneous coronary intervention (PCI) or within 2–24 h post-thrombolysis PCI (Fig. 1). Patients between 18 and 75 ages with acute anterior STEMI confirmed by ECG and angiography, admitted within 12 h of symptom onset, were included. Exclusions: prior BBB, prior CABG, or incomplete information. Demographic data, laboratory results at admission, interventional details, and indices of microcirculatory dysfunction for all patients were retrospectively assessed. The study was granted ethical

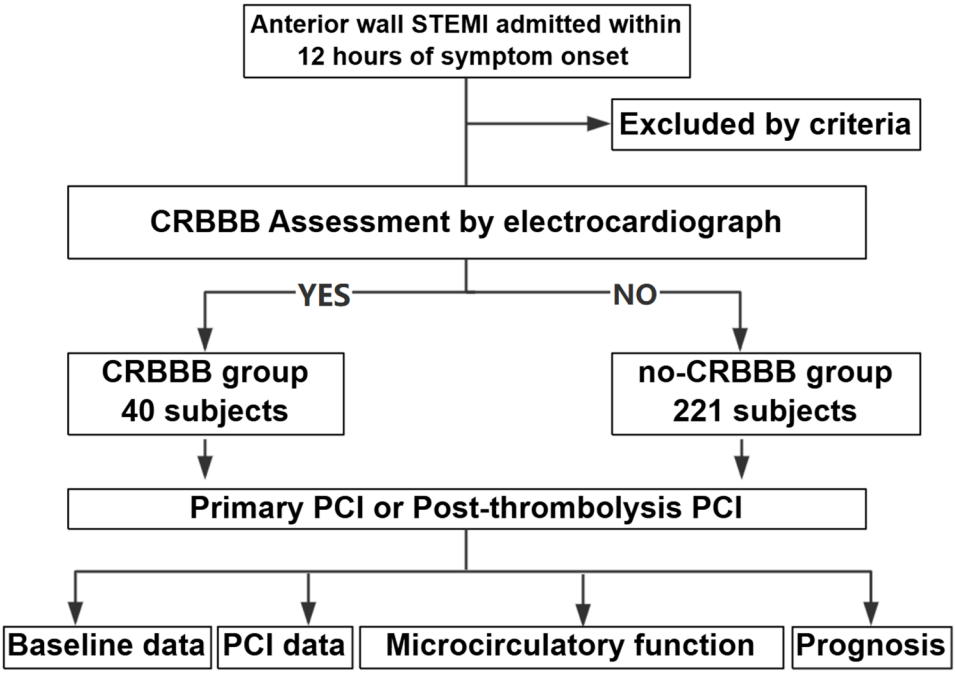

Fig. 1 Flowchart of study design

approval by the institutional ethics committee of the First Affiliated Hospital of Dali University, all participant provided written informed consent to participate in this study.

2.2 CaIMR, thrombolysis in myocardial infarction (TIMI) grade, corrected TIMI frame count (CTFC), and ST-segment regression (STR) were utilized to assess microcirculatory function. These assessments were performed in the culprit vessel immediately following successful PCI. CaIMR was assessed using the Rainmed Fractional Flow Reserve system, as previously described [15]. Briefly, a 3D model of the coronary artery along the target vessel was developed. Computational pressure-flow dynamics were employed to assess angiography-derived fractional flow reserve (angio-FFR). Estimated hyperemic pressure (Ph) was computed as mean arterial pressure (MAP) × 0.2 when MAP ≥ 95 mmHg or MAP × 0.15 when MAP < 95 mmHg. Resting flow velocity during diastole ($V_{diastole}$) was obtained using the TIMI frame count method. CaIMR was subsequently calculated as Ph × angio-FFR × (vessel length/K × $V_{diastole}$). Vessel length was defined as the distance from the inlet to the distal segment of the target vessel, and K was a constant used to normalize the difference between resting and hyperemic flow velocities. t is widely accepted that CaIMR values < 25 indicate normal coronary microcirculatory blood flow, while values > 40 suggest compromised flow [16].

2.3 Statistical Analysis: Statistical analyses were conducted using SPSS version 25.0. Normally distributed quantitative data were reported as the mean ± standard deviation (x ± s) and compared between groups using the t-test. Non-normally distributed data were described as the median and interquartile range [M (P25, P75)] and compared using the Mann-Whitney U test. Categorical and ordinal data were tabulated as the number of cases (n) and percentage (%) and compared using the chi-square test or Fisher's exact probability test. Variables with a *P*-value < 0.05 were considered for inclusion in binary logistic regression for multifactorial analysis. Receiver operating characteristic (ROC) analysis was employed to evaluate the predictive capacity for CaIMR values>40 in anterior wall STEMI. Statistical significance was defined at $P < 0.05$ for all results.

Results

3.1 Among the 261 consecutive patients with acute anterior wall STEMI, 15.3% (40/261) presented with new onset CRBBB and 84.7% (221/261) manifested without CRBBB. The CRBBB group exhibited a significantly older patient population compared to the no-CRBBB cohort ($P = 0.001$). Admission Killip class was predominantly class I-II in the no-CRBBB group, while it was majority of class III-IV in the CRBBB group ($P < 0.001$). Although the CRBBB group exhibited lower admission blood pressure and higher heart rate compared to the no-CRBBB group, these differences were not statistically significant ($P > 0.05$). No significant differences were found between the two groups regarding history of hypertension, diabetes, hyperlipidemia, coronary heart disease, chronic kidney disease, cerebrovascular disease, gout, gender, family history of cardiovascular disease, smoking, alcohol use, or PCI modality ($P > 0.05$). However, there were significant differences in baseline laboratory values such as glucose (GLUC), triglyceride (TG), blood urea nitrogen (BUN), glomerular filtration rate (GFR), high density lipoprotein (HDL), aspartate aminotransferase (AST), alanine aminotransferase (ALT), white blood cell (WBC), and troponin I (TNI) ($P < 0.05$). These data are presented in Table 1.

3.2 Interventional therapy data revealed that the median time from first medical contact to wire time (FMC2W) and total ischemic time were longer in the CRBBB group, although these differences did not reach statistical significance. In the CRBBB group, 97.5% of the infarction-related arteries (IRA) were proximal left anterior descending (LAD) arteries, while in the no-CRBBB group, 68.8% of the IRA were proximal LAD and 29.9% were mid LAD, representing a statistically significant difference ($P < 0.001$). There were no significant differences between the two groups in terms of TIMI grade, presence of thrombus, number of diseased vessel branches of the IRA before PCI ($P > 0.05$). Additionally, a comparison of intraoperative medications revealed a higher rate of dopamine use in the CRBBB group compared to the no-CRBBB group ($P < 0.05$). However, no statistically significant differences were found in the use of other medications, including atropine, norepinephrine, tirofiban, nitroglycerin, sodium nitroprusside, adenosine, and nicorandil ($P > 0.05$). These findings are presented in Table 2.

3.3 The CRBBB group exhibited a higher incidence of in-hospital major adverse cardiovascular events (MACEs), including cardiac death, recurrent myocardial infarction, acute left heart dysfunction, cardiogenic shock, complete atrioventricular block, ventricular fibrillation, and persistent ventricular tachycardia, compared to the no-CRBBB group. Additionally, the median left ventricular ejection fraction (LVEF) was significantly lower in the CRBBB group than in the no-CRBBB group ($P < 0.05$). Although the mean left ventricular end-diastolic diameter (LVEDD) was greater in the CRBBB group, the difference did not reach statistical significance compared to the no-CRBBB group ($P > 0.05$). Furthermore, there was a statistically significant difference in the use of vasoactive drugs (dopamine or norepinephrine) and the duration of use post-PCI between the two groups ($P < 0.05$). These results are summarized in Table 3.

Table 1 Baseline characteristics

Variables	no-CRBBB (*n* = 221)	CRBBB (*n* = 40)	X^2/t/Z	*P* value
Age(years)	59.36 ± 10.59	65.35 ± 10.85	-3.278	0.001*
Men(%)	185(83.7)	31(77.5)	0.916	0.339
Family history of CVD(%)	17(7.7)	1(2.5)	0.728	0.393
Smoking(%)	145(65.6)	22(55)	1.655	0.198
Alcohol(%)	52(23.5)	11(27.5)	0.292	0.589
Systolic BP(mmHg)	124.37 ± 17.65	118.83 ± 20.52	1.782	0.076
Diastolic BP(mmHg)	80.77 ± 13.43	78.25 ± 13.68	1.091	0.276
Heart rate(bpm)	80.31 ± 14.49	83.13 ± 17.83	-0.943	0.350
GLUC(mmol/L)	6.08(5.23,7.41)	7.14(5.77,11.40)	-2.915	0.004*
TC(mmol/L)	4.97(4.30,5.69)	4.59(3.83,5.60)	-1.377	0.168
TG(mmol/L)	1.63(1.2,2.59)	1.44(1.03,1.92)	-2.068	0.039*
HDL(mmol/L)	1.08(0.93,1.28)	1.23(1.01,1.52)	-2.526	0.012*
LDL(mmol/L)	2.90 ± 0.87	2.80 ± 1.07	0.660	0.510
Lp(a)(mmol/L)	20.5(8.8,38.05)	14.75(8.5,27.78)	-1.054	0.292
UA(umol/L)	345(287.5,409)	345.5(257,467.25)	-0.748	0.455
BUN(mmol/L)	5.19(4.30,6.51)	5.52(4.7,7.64)	-1.988	0.047*
Scr(umol/L)	78(65,90)	82(67.25,108.5)	-1.408	0.159
GFR(ml/min/1.73m^2)	93.59(75.75,115.07)	80.07(62.63,107.01)	-2.198	0.028*
AST(U/L)	185(87.5,312)	280(182.75,534.25)	-3.653	<0.001*
ALT(U/L)	48(32.5,73)	67(41,105.5)	-2.636	0.008*
WBC(10 ~ 9/L)	11.13(9.21,12.89)	13.09(9.53,17.66)	-2.908	0.004*
RBC(10 ~ 12/L)	4.83 ± 0.61	4.87 ± 0.71	-0.395	0.693
HB(g/L)	149.19 ± 18.28	150.13 ± 21.38	-0.291	0.771
PLT(10 ~ 9/L)	211.04 ± 67.44	215.93 ± 64.36	-0.424	0.672
BNP(pig/mL)	102(44.85,233.5)	100.65(40.9,244.75)	-0.153	0.879
D-D(ng/mL)	473(101,1180)	487.5(114,1505)	-0.761	0.446
CK-MB(ng/mL)	80(43.2,80)	80(59.6,80)	-1.722	0.085
MYO(ng/mL)	495(262.5,500)	500(286,500)	-0.972	0.331
Post-PCI TNI(ng/mL)	27.9(8.12,30)	30(25.23,30)	-2.235	0.025*
Killip class(%)				
I	146(66.1)	7(17.5)	95.104	<0.001*
II	56(25.3)	4(10)		
III	4(1.8)	11(27.5)		
IV	15(6.8)	18(45)		
Admission medication				
Aspirin(%)	221(100)	40(100)		
Clopidogrel (%)	113(51.8)	20(50)	0.017	0.895
Ticagrelor (%)	108(48.9)	20(50)	0.017	0.895
Statins (%)	221(100)	40(100)		
Heparin (%)	113(51.8)	20(50)	0.017	0.895

- Indicates Fisher's exact test; *Indicates the difference was statistically significant

CVD, cardiovascular disease; GLUC, glucose; TC, total cholesterol; TG, triglyceride; HDL, high density lipoprotein; LDL, low density lipoprotein; Lp(a), Lipoprotein (a); UA, uric acid; BUN, blood urea nitrogen; Scr, serum creatinine; GFR, glomerular filtration rate; AST, aspartate aminotransferase; ALT, alanine aminotransferase; WBC, white blood cell; RBC, red blood cell; HB, hemoglobin; PLT, platelet; BNP, B-type natriuretic peptide; D-D, D-dimer; CK-MB, Creatine kinas-MB isoenzyme; MYO, myoglobin; TNI, troponin I

3.4 Evaluation of microcirculatory function post-PCI revealed that the CRBBB group had a higher percentage of patients with TIMI ≤ grade 2 and ST-segment regression (STR) < 50% in the IRA compared to the no-CRBBB group. Furthermore, the caIMR was significantly higher in the CRBBB group than in the no-CRBBB group ($P < 0.05$). However, the median corrected TIMI frame count (CTFC) was not statistically different between the two groups ($P > 0.05$). These findings are detailed in Table 4.

3.5 Multivariate analysis was conducted to assess the microcirculatory function after PCI, with new CRBBB as the dependent variable. Binary logistic regression was utilized to adjust for confounding factors. The results

Table 2 Analysis of interventional therapy

Variables	no-CRBBB (*n* = 221)	CRBBB (*n* = 40)	X^2	*P* value
PCI modality(%)				
Primary PCI	108(48.9)	20(50)	0.017	0.895
PCI after thrombolysis	113(51.1)	20(50)	-1.806	0.071
FMC2W(min)	248(157.5,367)	305.5(208,448.8)	-0.821	0.412
ischemic time(min)	440(318,674.5)	490.5(342.3,682.5)		
infarct-related arteries(%)				
proximal LAD	152(68.8)	39(97.5)	17.761	<0.001*
mid LAD	66(29.9)	1(2.5)		
distal LAD	2(0.9)	0(0)		
right coronary artery	1(0.5)	0(0)		
thrombus(%)	21(9.5)	5(12.5)	0.087	0.767
number of diseased vessel branches(%)				
1	95(43)	17(42.5)	0.029	0.986
2	68(30.8)	12(30)		
3	58(26.2)	11(27.5)		
stent diameter(mm)	3(2.75,3)	3(2.75,3.5)	-1.912	0.056
stent length(mm)	24(18,33)	23(18,33)	-1.070	0.285
Intraoperative medication(%)				
atropine	16(7.2)	2(5)	0.031	0.861
dopamine	22(10)	9(22.5)	3.965	0.046*
norepinephrine	19(8.6)	8(20)	3.598	0.058
tirofiban	87(39.4)	12(30)	1.262	0.261
nitroglycerin	77(34.8)	16(40)	0.393	0.531
sodium nitroprusside	26(11.8)	8(20)	2.027	0.154
adenosine	2(0.9)	1(2.5)	-	0.394
nicorandil	1(0.5)	2(5)	-	0.062

-Indicates Fisher's exact test; *Indicates the difference was statistically significant

Table 3 Analysis of prognosis

Variables	no-CRBBB (*n* = 221)	CRBBB (*n* = 40)	X^2/t/Z	*P* value
in-hospital major adverse cardiovascular events(%)	84(38)	29(72.5)	16.412	<0.001*
LVEF(%)	60(52.5,67)	55(44.25,61)	-2.644	0.008*
LVEDD(mm)	48.84 ± 6.70	49.46 ± 4.08	-0.794	0.429
the use of vasoactive drugs after PCI(%)	39(17.6)	13(32.5)	4.683	0.030*
the duration of vasoactive drugs use after PCI(day)	0(0,0)	0(0,2)	-2.345	0.019*

*Indicates the difference was statistically significant.

Table 4 Analysis of the microcirculatory function post-PCI

Variables	no-CRBBB (*n* = 221)	CRBBB (*n* = 40)	X^2/Z	*P* value
TIMI grade(%)				
≤ 2	19(8.6)	10(25)	7.640	0.006*
3	202(91.4)	30(75)		
STR(%)				
≥ 50%	181(81.9)	14(35)	39.434	<0.001*
<50%	40(18.1)	26(65)		
CTFC(frames)	21.18(15.29,27.06)	24.71(15.88,32.65)	-1.622	0.105
caIMR	29.5(23.8,35)	40.25(33.35,49.68)	-5.650	<0.001*

*Indicates the difference was statistically significant.

demonstrated that, in addition to admission Killip classification, GLUC, HDL, and the location of IRA, TIMI grade ≤ 2 (OR = 6.833, 95% CI: 1.009 ~ 46.287, $P = 0.049$), STR ≥ 50% (OR = 0.176, 95% CI: 0.051 ~ 0.606, $P = 0.006$), CTFC (OR = 1.079, 95% CI: 1.009 ~ 1.155, $P = 0.027$), and caIMR (OR = 1.120, 95% CI: 1.059 ~ 1.185, $P < 0.001$) were independently associated with the presence of new CRBBB in patients with acute anterior STEMI. These associations are presented in Table 5.

3.6 The multivariate logistic regression analysis identified CaIMR > 40 as an independent risk factor for adverse outcomes in patients with acute anterior STEMI(Table 6). Data showed that new-onset CRBBB was strongly associated with elevated CaIMR (OR = 5.065, 95%

Table 5 Multivariate analysis of new-onset CRBBB in anterior STEMI

Variables	B value	Standard error of b-value	Wald's chi-square value	*P* value	OR value	95%CI
Age(years)	0.064	0.035	3.403	0.065	1.066	0.996 ~ 1.141
Killip class	0.859	0.274	9.846	0.002*	2.360	1.380 ~ 4.035
GLUC(mmol/L)	0.241	0.107	5.094	0.024*	1.272	1.032 ~ 1.569
TG(mmol/L)	-0.446	0.410	1.181	0.277	0.640	0.286 ~ 1.431
HDL(mmol/L)	1.878	0.953	3.882	0.049*	6.540	1.010 ~ 42.348
BUN(mmol/L)	-0.356	0.225	2.508	0.113	0.701	0.451 ~ 1.088
GFR(ml/min/1.73m2)	-0.012	0.012	1.095	0.295	0.988	0.966 ~ 1.011
AST(U/L)	0.001	0.002	0.177	0.674	1.001	0.997 ~ 1.005
ALT(U/L)	0.006	0.011	0.312	0.577	1.006	0.985 ~ 1.028
WBC(10 ~ 9/L)	0.176	0.095	3.476	0.062	1.193	0.991 ~ 1.436
TNI(ng/mL)	-0.020	0.036	0.295	0.587	0.981	0.914 ~ 1.052
PART of IRA	-4.149	1.646	6.357	0.012*	0.016	0.001 ~ 0.397
Dopamine use	-1.234	1.138	1.178	0.278	0.291	0.031 ~ 2.705
Post PCI TIMI ≤ grade 2	1.922	0.976	3.876	0.049*	6.833	1.009 ~ 46.287
STR ≥ 50%	-1.740	0.632	7.583	0.006*	0.176	0.051 ~ 0.606
CTFC(frames)	0.076	0.035	4.913	0.027*	1.079	1.009 ~ 1.155
caIMR	0.114	0.029	15.603	<0.001*	1.120	1.059 ~ 1.185

*Indicates the difference was statistically significant

Table 6 Multivariate analysis of caimr > 40 in anterior STEMI

Variables	B value	Standard error of b-value	Wald's chi-square value	*P* value	OR value	95%CI
new-onset of CRBBB	1.622	0.530	9.347	0.002*	5.065	1.793 ~ 14.308
Age(years)	0.007	0.017	0.161	0.688	1.007	0.974 ~ 1.041
Killip class	-0.028	0.195	0.021	0.885	0.972	0.663 ~ 1.426
Pre-PCI TIMI ≤ grade 2	0.047	0.140	0.113	0.737	1.048	0.796 ~ 1.380
IRA	-0.063	0.391	0.026	0.871	0.939	0.436 ~ 2.020
sodium nitroprusside	0.146	0.547	0.065	0.799	1.157	0.375 ~ 3.567
Post-PCI TIMI ≤ grade 2	-1.600	0.543	8.685	0.003*	0.202	0.070 ~ 0.585
STR ≥ 50%	-0.742	0.395	3.529	0.060	0.476	0.220 ~ 1.033
CTFC(frames)	0.018	0.020	0.797	0.372	1.018	0.979 ~ 1.058

CI:1.793–14.308, $P = 0.002$), post-PCI TIMI flow grade inversely correlated with CaIMR > 40 (OR = 0.202, 95% CI:0.070–0.585, $P = 0.003$). Age, Killip class, pre-PCI TIMI flow grade, infarct-related artery location, sodium nitroprusside use, and CTFC showed no statistically significant associations ($P > 0.05$ for all). CRBBB demonstrated moderate predictive value for CaIMR > 40 (AUC = 66.8%, $P < 0.001$; 95% CI:0.577–0.760). Post-PCI TIMI flow had limited discriminative capacity (AUC = 36.5%, $P = 0.003$; 95% CI:0.272–0.458).(Fig. 2).

Discussions

The incidence of new-onset CRBBB in AMI has shown that although less common, it is significantly associated with a poorer prognosis [17]. Furthermore, there are suggestions that new-onset CRBBB be considered as an indication for reperfusion therapy in future guidelines [18]. Nonetheless, the mechanisms underlying the poorer prognosis associated with newly developed CRBBB have yet to be fully elucidated. Our study has identified a high incidence of microcirculatory dysfunction post-PCI in anterior wall STEMI patients with new-onset CRBBB, potentially contributing to their poorer prognosis.

Within our study cohort comprising 261 patients with acute anterior STEMI, we detected 40 patients with new CRBBB, yielding an incidence rate of 15.3%. This incidence rate is consistent with the 10.9% reported during the thrombolytic era [19], and the range of 4.3–15.7% observed in the stent era [20–23], but notably higher than the 1.8% reported in a large study [5]. Patients with CRBBB tended to be older and displayed increased WBC counts, elevated fasting blood glucose levels, and higher serum levels of AST and ALT, along with decreased GFR. These findings imply that CRBBB patients may suffer larger infarct sizes and a more robust inflammatory response, which could lead to hemodynamic instability, as well as renal and liver dysfunction. Notably, there was study suggested that new-onset CRBBB may present with different clinical manifestations, such as increased dyspnea [24] rather than chest pain. Moreover, our findings indicate that the prolonged time from FMC2W in the new CRBBB group may correlate with more severe cardiac dysfunction and microcirculatory dysfunction

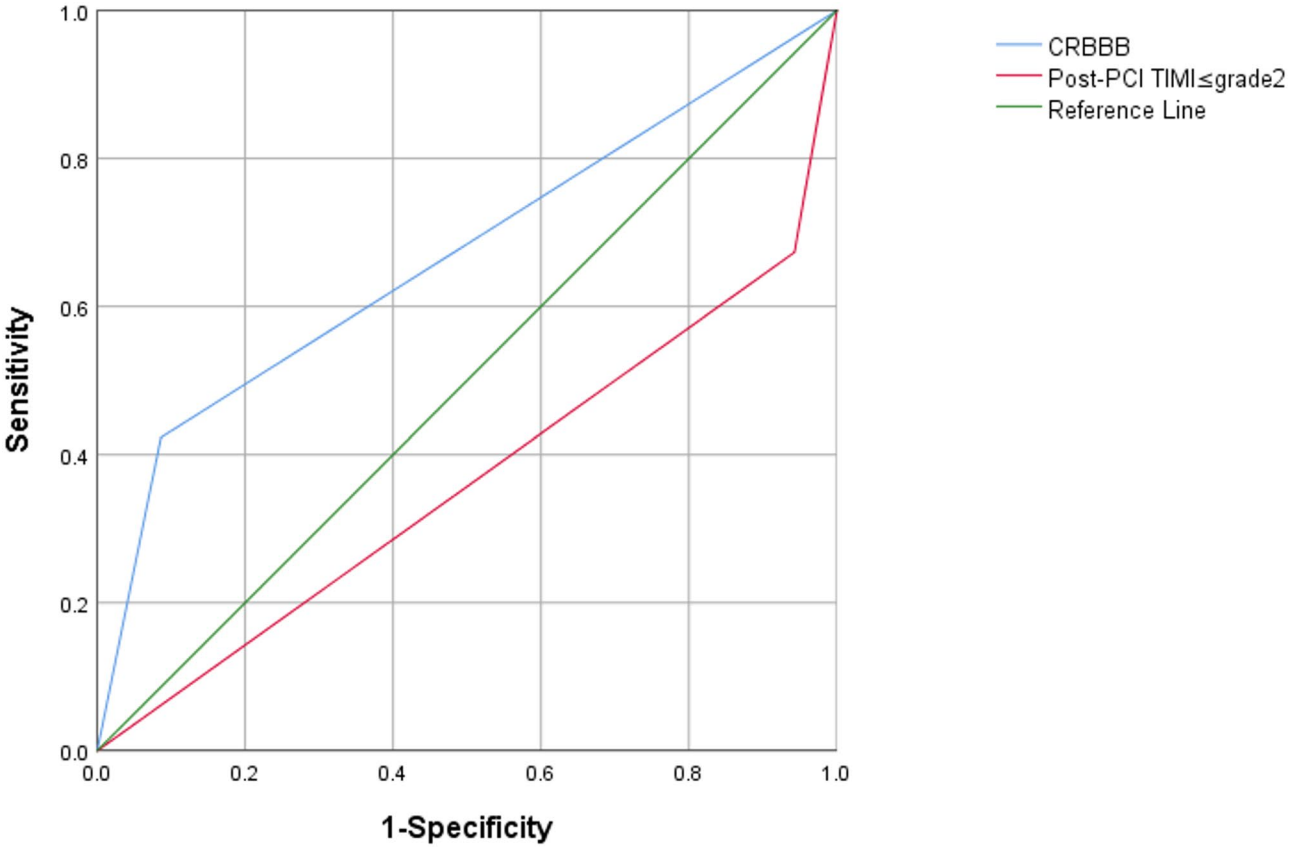

Fig. 2 Predictive value of ROC curve for *CaIMR > 40*

in patients with acute anterior STEMI and new CRBBB. The early identification of high-risk patients and prompt intervention should be emphasized.

Moreover, our study indicated that the IRA was predominantly the proximal LAD artery in 97.5% of CRBBB patients, which corresponds to the typical blood supply characteristics of the bundle branches [25]. Since the LAD provides a substantial portion of the myocardium in the left heart, anterior wall infarction can readily impair contractile function and lead to hemodynamic instability. Proximal LAD occlusion may induce a "dual injury" phenomenon—direct microcirculatory impairment (via plaque embolization or reperfusion injury) and ischemic disruption of the RBB. Our findings further demonstrate that patients in the CRBBB group predominantly presented with Killip class III or IV upon admission, in contrast to the predominantly Killip class I or II in the no-CRBBB group. Admission Killip classification was independently correlated with the development of new CRBBB in acute anterior STEMI patients after adjusting for confounders (OR = 2.360, 95% CI: 1.380–4.035, $P = 0.002$), suggesting poorer cardiac function in CRBBB patients. Furthermore, the incidence of in-hospital adverse cardiovascular events, the rate of post-operative vasoactive drug use, and the duration of use were significantly higher in the CRBBB group, whereas the median left ventricular ejection fraction (LVEF) was lower compared to the non-CRBBB group, reflecting poorer cardiac function and prognosis. Reactive oxygen species (ROS) and pro-inflammatory cytokines (e.g., IL-6, TNF-α) released during ischemia-reperfusion not only exacerbate microvascular endothelial damage but may also directly inhibit sodium channel function in Purkinje fibers, explaining the association between elevated CaIMR and RBBB incidence. A study of interest reported that new-onset RBBB and ventricular fibrillation in STEMI are independently associated and exhibit particularities based on the duration of the conduction disturbance and/or the primary or secondary nature of the arrhythmia [26]. However, in our study, we observed the same fatal arrhythmias incidence and attributed poor long-term prognosis to cardiac insufficiency and microcirculatory dysfunction.

In our study, we employed caIMR, TIMI grade, CTFC, and ST-segment regression to assess microcirculatory function. The analysis demonstrated significant disparities in caIMR, TIMI grade, and STR between the CRBBB and non-CRBBB cohorts. Notably, following adjustment for confounders, caIMR, TIMI grade ≤ 2, STR ≥ 50%, and CTFC were identified as independent correlates of

the development of new CRBBB in patients with acute anterior STEMI. These data suggest a robust association between the occurrence of new CRBBB and the development of microcirculatory dysfunction in STEMI patients. To the best of our knowledge, this study provides the initial evidence of coronary microcirculatory dysfunction in STEMI patients who have developed new CRBBB. Nevertheless, the etiology of this presentation—whether attributable to the CRBBB itself or the elevated Killip grade observed in these patients—remains ambiguous, given that cardiac insufficiency frequently precipitates coronary microcirculatory dysfunction [27]. Interestingly, we found an inverse relationship between post-PCI TIMI flow and CaIMR, which underscores the limitations of conventional angiographic metrics in capturing microcirculatory impairment, advocating for adjunctive CaIMR assessment.

The coronary microcirculation consists of a network of small vessels tasked with delivering oxygen and nutrients to the myocardium. The mechanisms underpinning microcirculatory dysfunction are multifaceted and not entirely elucidated; however, they may encompass microvascular embolism, endothelial dysfunction, platelet aggregation, leukocyte activation, inflammatory processes, and micro thrombosis [28]. Myocardial infarction can induce swelling of cardiomyocytes and microvascular endothelial cells within the ischemic area, leading to microvessel occlusion and compromised microvascular endothelial cell integrity, which in turn promotes micro thrombosis [29]. Microcirculatory dysfunction may also reflect periprocedural myocardial injury (PMI), a phenomenon frequently observed in PPCI procedures and predominantly attributed to ischemia-reperfusion injury. This pathophysiological cascade has been robustly linked to heightened risks of long-term adverse cardiovascular outcomes, including heart failure progression and recurrent infarction [30]. Future investigations should employ longitudinal assessments to delineate the relative contributions of pre-existing microvascular impairment versus iatrogenic procedural damage in this high-risk cohort.

In this study, we employed the CaIMR—a fractional flow reserve (FFR)-based metric validated for its prognostic utility in predicting long-term LVEF impairment and adverse clinical outcomes [31]. Critically, the elevated CaIMR values observed in patients with new-onset CRBBB underscore the necessity for tailored mitigation strategies to address coronary microcirculatory dysfunction in this high-risk STEMI subgroup, such as potential methods for identifying high-risk patients [32] and interventions encompass the use of pharmacological agents [33, 34], thrombus aspiration [35] and deferred stenting strategy [36], remote ischemic preconditioning [37], as well as intra-aortic balloon pump support [38]. Moreover, the conduct of clinical trials specifically designed to target microcirculatory dysfunction may offer the potential to improve long-term prognostic outcomes in patients with newly developed CRBBB.

Limitation: Given the high mortality risk associated with anterior AMI, particularly when complicated by new-onset CRBBB, prompt ECG interpretation is critical for interventional cardiologists to assess disease severity and guide timely revascularization decisions. therefore, it should be noted that interventional cardiologists evaluating post-procedural parameters in this study were not blinded to patients' ECG findings, which may introduce potential measurement bias in outcome assessments. While this single-center cohort provided statistically robust preliminary insights into the association between CRBBB and microcirculatory dysfunction, but did not analyze subgroups with LBBB or atypical RBBB. Future studies should systematically expand sample sizes by multicenter prospective studies in different type of BBB and employ animal models to test the "electro-microvascular coupling" hypothesis.

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12872-025-04872-9.

Supplementary Material 1

Acknowledgements

We gratefully thank the patients and families for their involvement in this study.

Author contributions

Hong Liu and Yu Yuan designed, performed, analyzed and interpreted most research. Yu Dong, Ying Yang, Biao Sun, Lilan Ma, Tao Li established the clinical discovery cohort and participated in the data collection. Xitong Yang participated in data analysis, figure preparation. Xin-Hua Wu proposed and developed the concept, conceived and supervised the study, and revised the manuscript.

Funding

This work was supported by grants from Project of National Natural Science Foundation of China(82460066), High-level scientific and technological talents and innovation team Program of Yunnan Province (No.202305AS350027), Yunnan Young Academic and Technical Reserve Talents program (No.202405AC350021), Key Research and development Project of Yunnan Province (No.202103AC100004).

Data availability

Data is provided within the supplementary information files.

Declarations

Competing interests

The authors declare no competing interests.

Received: 13 June 2024 / Accepted: 19 May 2025
Published online: 29 May 2025

References

1. Klein A, Baranchuk A. Identification of high-risk patients based on electrocardiogram during acute anterior ST-elevation myocardial infarction: the qRBBB pattern. Can J Cardiol. 2020;36(11):1708–9.
2. Lévy S. Bundle branch blocks and/or hemiblocks complicating acute myocardial ischemia or infarction. J Interv Card Electrophysiol. 2018;52(3):287–92.
3. Rector G, Triska J, Ajene G, Wang A, Haddadin F, Jabri A, Alam M, Birnbaum Y. Right bundle branch and bifascicular blocks: insensitive prognostic indicators for acute myocardial infarction. Curr Probl Cardiol. 2023;48(1):101418.
4. Schmitz T, Wein B, Methe H, Linseisen J, Heier M, Peters A, Meisinger C. Association between admission ECG changes and long-term mortality in patients with an incidental myocardial infarction: results from the KORA myocardial infarction registry. Eur J Intern Med. 2022;100:69–76.
5. Shrivastav R, Perimbeti S, Casso-Dominguez A, Jneid H, Kwan T, Tamis-Holland JE. In hospital outcomes of patients with right bundle branch block and anterior wall ST-segment elevation myocardial infarction (From a nationwide study using the national inpatient sample). Am J Cardiol. 2021;140:20–4.
6. Yang Y, Wang J, Wu B, Xu Y, Tang L, Jiang H, Wang B, Zhu T. New permanent bundle-branch block and long-term prognosis of patients with new onset ST-elevation myocardial infarction who underwent percutaneous coronary intervention. Front Physiol. 2022;13:892623.
7. Konijnenberg LSF, Damman P, Duncker DJ, Kloner RA, Nijveldt R, van Geuns RM, Berry C, Riksen NP, Escaned J, van Royen N. Pathophysiology and diagnosis of coronary microvascular dysfunction in ST-elevation myocardial infarction. Cardiovasc Res. 2020;116(4):787–805.
8. Ibáñez B, Heusch G, Ovize M, Van de Werf F. Evolving therapies for myocardial ischemia/reperfusion injury. J Am Coll Cardiol. 2015;65(14):1454–71.
9. Annibali G, Scrocca I, Aranzulla TC, Meliga E, Maiellaro F, Musumeci G. No-reflow phenomenon: a contemporary review. J Clin Med. 2022;11(8).
10. Robbers LF, Eerenberg ES, Teunissen PF, Jansen MF, Hollander MR, Horrevoets AJ, Knaapen P, Nijveldt R, Heymans MW, Levi MM, et al. Magnetic resonance imaging-defined areas of microvascular obstruction after acute myocardial infarction represent microvascular destruction and haemorrhage. Eur Heart J. 2013;34(30):2346–53.
11. Carrick D, Haig C, Ahmed N, Rauhalammi S, Clerfond G, Carberry J, Mordi I, McEntegart M, Petrie MC, Eteiba H et al. Temporal evolution of myocardial hemorrhage and edema in patients after acute ST-segment elevation myocardial infarction: pathophysiological insights and clinical implications. J Am Heart Assoc. 2016;5(2).
12. Karakayali M, Altunova M, Yakisan T, Aslan S, Omar T, Artac I, Ilis D, Arslan A, Cagin Z, Karabag Y, et al. The relationship between the systemic immune-inflammation index and ischemia with non-obstructive coronary arteries in patients undergoing coronary angiography. Arq Bras Cardiol. 2024;121(2):e20230540.
13. Camici PG, Crea F. Coronary microvascular dysfunction. N Engl J Med. 2007;356(8):830–40.
14. Choi KH, Dai N, Li Y, Kim J, Shin D, Lee SH, Joh HS, Kim HK, Jeon KH, Ha SJ, et al. Functional coronary angiography-derived index of microcirculatory resistance in patients with ST-segment elevation myocardial infarction. JACC Cardiovasc Interv. 2021;14(15):1670–84.
15. Li J, Gong Y, Wang W, Yang Q, Liu B, Lu Y, Xu Y, Huo Y, Yi T, Liu J, et al. Accuracy of computational pressure-fluid dynamics applied to coronary angiography to derive fractional flow reserve: FLASH FFR. Cardiovasc Res. 2020;116(7):1349–56.
16. Ai H, Feng Y, Gong Y, Zheng B, Jin Q, Zhang HP, Sun F, Li J, Chen Y, Huo Y, et al. Coronary angiography-derived index of microvascular resistance. Front Physiol. 2020;11:605356.
17. Xiang L, Zhong A, You T, Chen J, Xu W, Shi M. Prognostic significance of right bundle branch block for patients with acute myocardial infarction: a systematic review and meta-analysis. Med Sci Monit. 2016;22:998–1004.
18. Widimsky P, Rohác F, Stásek J, Kala P, Rokyta R, Kuzmanov B, Jakl M, Poloczek M, Kanovsky J, Bernat I, et al. Primary angioplasty in acute myocardial infarction with right bundle branch block: should new onset right bundle branch block be added to future guidelines as an indication for reperfusion therapy? Eur Heart J. 2012;33(1):86–95.
19. Melgarejo-Moreno A, Galcerá-Tomás J, Garciá-Alberola A, Valdés-Chavarri M, Castillo-Soria FJ, Mira-Sánchez E, Gil-Sánchez J, Allegue-Gallego J. Incidence, clinical characteristics, and prognostic significance of right bundle-branch block in acute myocardial infarction: a study in the thrombolytic era. Circulation. 1997;96(4):1139–44.
20. Figueroa-Triana JF, Mora-Pabón G, Quitian-Moreno J, Álvarez-Gaviria M, Idrovo C, Cabrera JS, Peñuela JAR, Caballero Y, Naranjo M. Acute myocardial infarction with right bundle branch block at presentation: prevalence and mortality. J Electrocardiol. 2021;66:38–42.
21. Kleemann T, Juenger C, Gitt AK, Schiele R, Schneider S, Senges J, Darius H, Seidl K. Incidence and clinical impact of right bundle branch block in patients with acute myocardial infarction: ST elevation myocardial infarction versus non-ST elevation myocardial infarction. Am Heart J. 2008;156(2):256–61.
22. Vivas D, Pérez-Vizcayno MJ, Hernández-Antolín R, Fernández-Ortiz A, Bañuelos C, Escaned J, Jiménez-Quevedo P, De Agustín JA, Núñez-Gil I, González-Ferrer JJ, et al. Prognostic implications of bundle branch block in patients undergoing primary coronary angioplasty in the stent era. Am J Cardiol. 2010;105(9):1276–83.
23. Tomoda H, Aoki N. Right bundle branch block in acute myocardial infarction treated by primary coronary angioplasty and stenting. Angiology. 2005;56(2):131–6.
24. Alkindi F, El-Menyar A, Rafie I, Arabi A, Al Suwaidi J, Singh R, Albinali H, Gehani AA. Clinical presentations and outcomes in patients presenting with acute cardiac events and right bundle branch block. Angiology. 2020;71(3):256–62.
25. Arslan U, Balcioğlu S, Tavil Y, Ozdemir M, Cengel A. Clinical and angiographic importance of right bundle branch block in the setting of acute anterior myocardial infarction. Anadolu Kardiyol Derg. 2008;8(2):123–7.
26. Galcerá-Jornet E, Consuegra-Sánchez L, Galcerá-Tomás J, Melgarejo-Moreno A, Gimeno-Blanes JR, Jaulent-Huertas L, Wasniewski S, de Gea-García J, Vicente-Gilabert M, Padilla-Serrano A. Association between new-onset right bundle branch block and primary or secondary ventricular fibrillation in ST-segment elevation myocardial infarction. Eur Heart J Acute Cardiovasc Care. 2021;10(8):918–25.
27. Toya T, Nagatomo Y, Ikegami Y, Masaki N, Adachi T. Coronary microvascular dysfunction in heart failure patients. Front Cardiovasc Med. 2023;10:1153994.
28. Sagris M, Theofilis P, Antonopoulos AS, Oikonomou E, Paschaliori C, Galiatsatos N, Tsioufis K, Tousoulis D. Inflammation in coronary microvascular dysfunction. Int J Mol Sci. 2021;22(24).
29. Herrmann J, Kaski JC, Lerman A. Coronary microvascular dysfunction in the clinical setting: from mystery to reality. Eur Heart J. 2012;33(22):2771–b2782.
30. Armillotta M, Bergamaschi L, Paolisso P, Belmonte M, Angeli F, Sansonetti A, Stefanizzi A, Bertolini D, Bodega F, Amicone S, et al. Prognostic relevance of type 4a myocardial infarction and periprocedural myocardial injury in patients with non-ST-segment-elevation myocardial infarction. Circulation. 2025;151(11):760–72.
31. Canton L, Suma N, Amicone S, Impellizzeri A, Bodega F, Marinelli V, Ciarlantini M, Casuso M, Bavuso L, Belà R, et al. Clinical impact of multimodality assessment of myocardial viability. Echocardiography. 2024;41(7):e15854.
32. Karakayali M, Omar T, Artac I, Ilis D, Arslan A, Altunova M, Cagin Z, Karabag Y, Karakoyun S, Rencuzogullari I. The prognostic value of HALP score in predicting in-hospital mortality in patients with ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention. Coron Artery Dis. 2023;34(7):483–8.
33. Ito N, Nanto S, Doi Y, Kurozumi Y, Natsukawa T, Shibata H, Morita M, Kawata A, Tsuruoka A, Sawano H, et al. Beneficial effects of intracoronary nicorandil on microvascular dysfunction after primary percutaneous coronary intervention: demonstration of its superiority to nitroglycerin in a cross-over study. Cardiovasc Drugs Ther. 2013;27(4):279–87.
34. Rehan R, Virk S, Wong CCY, Passam F, Layland J, Keech A, Yong A, White HD, Fearon W, Ng M. Intracoronary thrombolysis in ST-elevation myocardial infarction: a systematic review and meta-analysis. Heart. 2024;110(15):988–96.
35. Silva-Orrego P, Colombo P, Bigi R, Gregori D, Delgado A, Salvade P, Oreglia J, Orrico P, de Biase A, Piccalò G, et al. Thrombus aspiration before primary angioplasty improves myocardial reperfusion in acute myocardial infarction: the DEAR-MI (Dethrombosis to enhance acute reperfusion in myocardial Infarction) study. J Am Coll Cardiol. 2006;48(8):1552–9.
36. Carrick D, Oldroyd KG, McEntegart M, Haig C, Petrie MC, Eteiba H, Hood S, Owens C, Watkins S, Layland J, et al. A randomized trial of deferred stenting versus immediate stenting to prevent no- or slow-reflow in acute ST-segment elevation myocardial infarction (DEFER-STEMI). J Am Coll Cardiol. 2014;63(20):2088–98.
37. Lau JK, Roy P, Javadzadegan A, Moshfegh A, Fearon WF, Ng M, Lowe H, Brieger D, Kritharides L, Yong AS. Remote ischemic preconditioning acutely improves coronary microcirculatory function. J Am Heart Assoc. 2018;7(19):e009058.

38. De Silva K, Lumley M, Kailey B, Alastruey J, Guilcher A, Asrress KN, Plein S, Marber M, Redwood S, Perera D. Coronary and microvascular physiology during intra-aortic balloon counterpulsation. JACC Cardiovasc Interv. 2014;7(6):631–40.

Publisher's note