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Case Report

Poor Outcome of Right Bundle Branch Block Coexist with ST-Elevation Myocardial Infarction

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ABSTRACT

Background : The incidence of new-onset right bundle branch block (RBBB) coexistence with ST-elevation myocardial infarction (STEMI) has been associated with higher in-hospital mortality than those without RBBB.
Case : We present three cases of new-onset RBBB that coexist with STEMI. Case I: a 64 years old male presented Killip I STEMI inferior-anterior with RBBB as new-onset. Rescue percutaneous coronary intervention (PCI) after failed thrombolytic was performed. New-onset atrial fibrillation (AF) with rapid ventricular response worsened his hemodynamic profile, leading to cardiogenic shock. Case II: an 80 years old male presented Killip IV late-onset anterior STEMI with new-onset RBBB. Cardiogenic shock got worsened after PCI. Case III: a 65 years old male presented Killip II extensive anterior STEMI with new-onset RBBB who underwent a primary PCI. After PCI, there was recurrent ventricular tachycardia (VT), worsening cardiogenic shock, and transient AV block.
Discussion : The right bundle branch blood supply is mainly provided by a septal branch of the left descending artery (LAD). Therefore, it may indicate proximal LAD occlusion and extensive infarction. Thus, catastrophic events may occur, including acute heart failure, AV block, malignant ventricular arrhythmia, new-onset AF, and mostly cardiogenic shock, despite initiating reperfusion being performed without delay once the diagnosis is confirmed.
Conclusion : New RBBB suggests a poor short-term prognosis due to its complication. Higher mortality is mostly linked to worsening cardiogenic shock.

1. Introduction

One of the primary causes of death in developed countries is coronary artery disease (CAD).[1] Despite the fact that mortality rates have fallen in recent decades due to significant advances in treatment, they have stabilized in 3–5% of cases.[2] Myocardial infarction (MI) is defined as damage or death to the heart muscle caused by a blockage in the blood flow to that area. Myocardial infarction is divided into two categories based on ST-segment changes: ST-elevation myocardial infarction (STEMI) and non-ST elevation myocardial infarction (NSTEMI) (NSTEMI). Complications kill 15% of individuals with acute myocardial infarction, with STEMI death rates exceeding NSTEMI rates.[3]

ST-elevation myocardial infarction is characterized as a new J-point ST elevation in at least two contiguous leads of ≥ 1.5 mm (0.15 mV) in females and ≥ 2 mm (0.2 mV) in males in V2-V3 leads and/or ≥ 1 mm (0.1 mV) in other contiguous leads.[4] ST-elevation myocardial infarction is an indicator of immediate reperfusion treatment.[5] However, a condition induced by severe blockage of the coronary area does not always manifest itself with conventional ST-elevation ECG findings, necessitating resource mobilization for primary percutaneous coronary intervention (PCI) or immediate thrombolytic therapy. STEMI equivalents are the names given to these conditions.

According to earlier guidelines, patients with new or suspected new left bundle branch block (LBBB) should be treated as if they had a STEMI, with urgent reperfusion therapy.[6] This advice has been expanded in subsequent guidelines for patients with right bundle branch block (RBBB0) [6]. Patients with Right Bundle Branch Block (RBBB) have a worse prognosis than those without RBBB after an ST-Elevation Myocardial Infarction (STEMI). However, with fewer available clinical studies and a lower level of evidence, the evidence for RBBB is less rigorous. We present three examples of new-onset RBBB in conjunction with STEMI in this paper.

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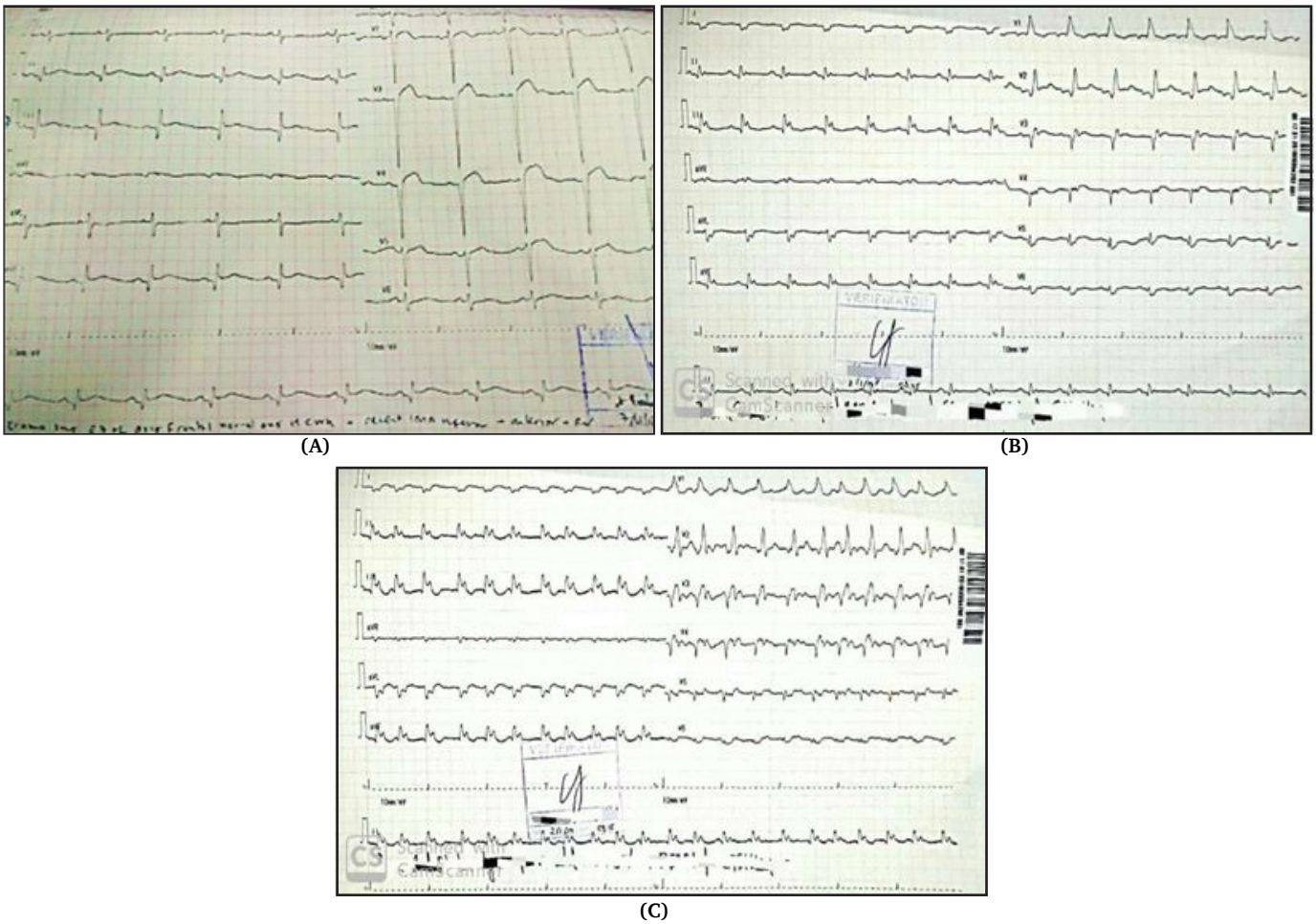


Figure 1. (A) First ECG, one month before admission. (B) Second admission ECG, new-onset RBBB occurred. (C) New-onset AF with rapid ventricular response occurred after rescue PCI.

2. Case Series

Case 1

A 64 years old male patient was referred to the Soetomo General Hospital due to failed thrombolytic. He admitted typical chest pain and diaphoresis 7 hours prior to admission. He had dyslipidemia and type II diabetes. He reported a late-onset inferior-anterior STEMI post-primary PCI one month before admission (Figure 1A). Coronary angiography revealed a three-vessel disease with implanted DES from proximal to mid LAD. On admission, he was diaphoretic with a 6/10 pain intensity. Hemodynamic profile was unstable with blood pressure (BP) about 110/60 mmHg supported by norepinephrine 50 nano/kgBW/min and dopamine 7mcg/kgBW/min, the heart rate (HR) 122 bpm regular, the respiratory rate (RR) 25/min, with oxygen saturation (SpO2) 99% without any O2 support. Lung and heart auscultation were normal. The electrocardiogram (ECG) performed sinus rhythm 100 bpm with trifasicular block (new-onset RBBB, left posterior fascicular block (LPFB), and first-degree AV block) (Figure 1B).

The high-sensitivity troponin I level increased to 16.67ng/L (N<0.02 ng/L) with random blood sugar was 400 mg/dL. Rescue PCI came up with normal right coronary artery (RCA) and left circumflex artery (LCx), patent old stent at proximal to the mid of left descending artery (LAD), and high thrombus burden at distal LAD. DES was implanted at distal LAD after thrombus aspiration with TIMI flow II. Transthoracic Echocardiography (TTE) revealed ejection fraction by Teich was 42% with hypokinetic on anterior, anteroseptal, inferoseptal, and inferior walls. His hemodynamic profile was pulse capillary wedge pressure (PCWP) 16.78 mmHg, systemic vascular resistance (SVR) 1661.054 dynes.sec/cm5, cardiac output (CO) 2.97 L/min, cardiac index (CI) 1.60 L/min2, Est RAP 15 mmHg. We continued the therapy with continuous infusion of furosemide, dual antiplatelet (DAPT), high-intensity statin, norepinephrine, and dopamine.

The patient was then transferred to the Cardiovascular Care Unit (CVCU). New-onset AF with rapid ventricular response occurred after rescue PCI (Figure 1C). 150 joule of cardioversion was performed, but the rhythm failed to convert. We decided to administer intravenous amiodarone 150 mg bolus, followed by 300 mg for 6 hours and 600 mg for 18 hours with up-titration of dopamine and norepinephrine pump. The hemodynamic got worsened gradually. Finally, the patient pronounced death due to refractory cardiogenic shock.

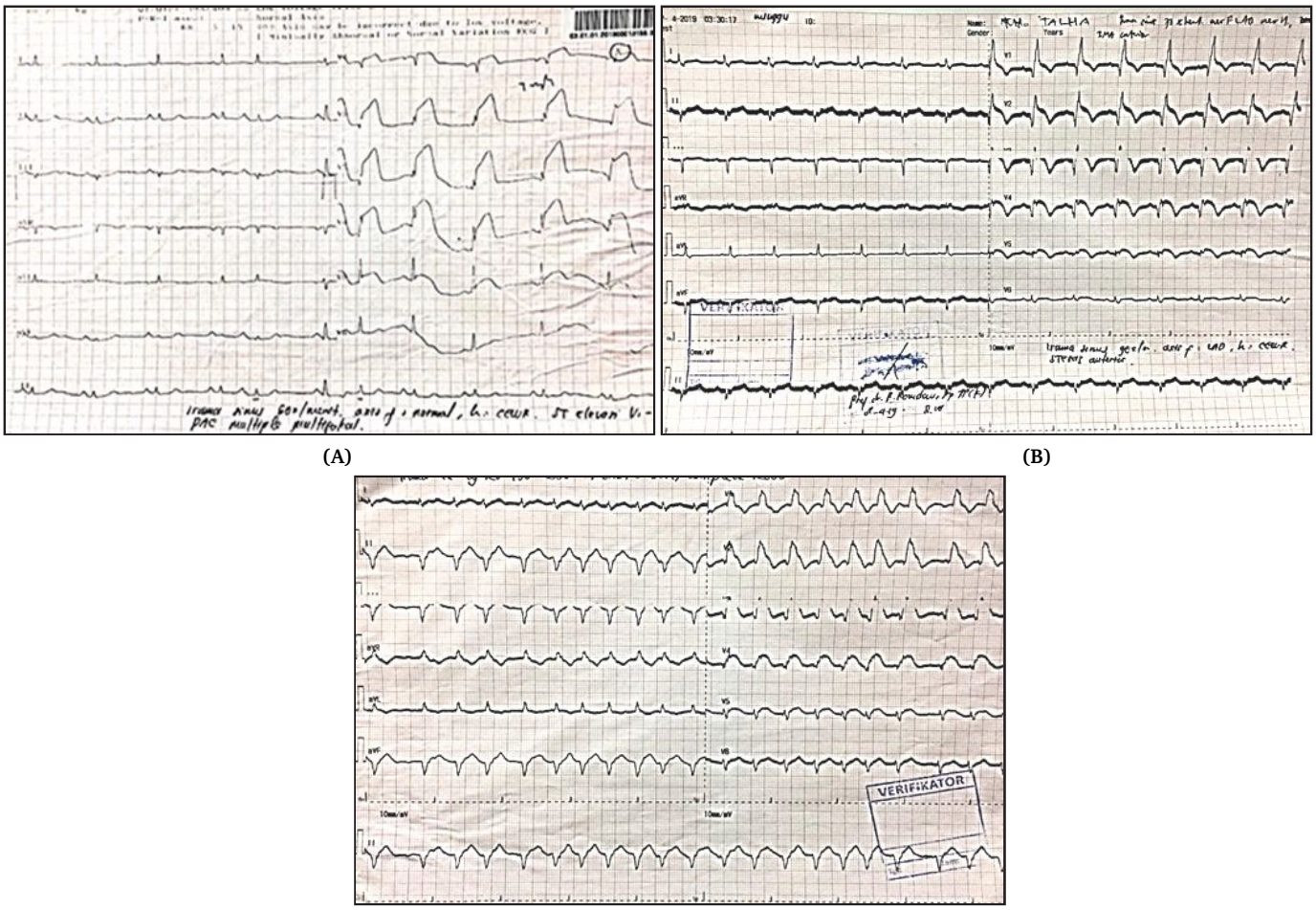
Case 2

An 80 years old male with a history of heavy cigarette smoking and hypertension presented with Killip I late-onset anterior STEMI and a new-onset RBBB. He had typical chest pain from 2 days prior to admission. He had 2/10 pain intensity. BP was 120/70 mmHg, HR 80 bpm regular, RR rate 25/min, with SpO2 99%. There were no rales and wheezing from lung auscultation. The ECG revealed sinus rhythm 60 bpm, ST elevation at V1-V6 lead with multiple premature arterial complexes (PACs) (Figure 2A). The high-sensitivity troponin I level increased to >50 ng/L (N<0.02 ng/L) with random blood sugar was 127 mg/dL. Teich's ejection fraction was 50% with hypokinetic on

anterior, anteroseptal, and inferoseptal walls. His hemodynamic profile performed PCWP 12.88 mmHg, SVR 1780 dynes.sec/cm5, CO 3.49 L/min, CI 2.10 L/min2, Est RAP 10 mmHg. We continued the therapy with DAPT, high-intensity statin, ACE inhibitor, and beta-blocker. On the second day, the patient experienced new-onset RBBB, and the hemodynamic became unstable (Figure 2B).

The BP decreased gradually, and we decided to administer intravenous dopamine and dobutamine. The patient underwent primary PCI due to 70% osteal lesion of RCA and 99% osteal lesion of LAD. The final result was no flow after stent implantation at osteal to proximal LAD.

Hypotension followed by cardiac arrest occurred during the procedure. Cardiopulmonary Resuscitation (CPR) was initiated. The patient gradually recovered his heart rate and blood pressure supported by norepinephrine 200 nano/kgBW/min, dopamine 10 mcg/kgBW/min, and dobutamine 10 mcg/kgBW/min. New-onset AF with rapid ventricular response worsened cardiogenic shock at CVCU (Figure 2C). We performed 150-joule cardioversion, but the rhythm could not be converted to sinus. Intravenous amiodarone 150 mg was administered with an up-titration of dobutamine, dopamine, and norepinephrine. The patient had a recurrent cardiac arrest and failed to achieve the Return of Spontaneous Consciousness (ROSC).



(A) (B)

(C)

Figure 2. (A) First ECG at Emergency Room (ER). (B) Second ECG, new-onset RBBB has occurred. (C) Second ECG, new-onset AF occurred.

Case 3

A 65 years old male with a history of heavy cigarette smoking was referred to the Soetomo General Hospital with Killip II anterior-extensive STEMI and new-onset RBBB. He reported typical chest pain and shortness of breath 9 hours before admission. He had no history of cardiovascular risk factors. On admission, BP was 110/80 mmHg, HR was 84/min, RR was 24/min, with SpO2 99% via nasal cannula. There were rales from lung auscultation. The previous hospital's ECG revealed sinus rhythm 94 bpm with anterior-extensive STEMI (Figure 3A). On admission, the ECG revealed to sinus rhythm 90 bpm, extensive anterior STEMI with new onset of RBBB (Figure 3B). The high-sensitivity troponin I level increased to 60,735 ng/L (N<0.02 ng/L) with random blood sugar was 147 mg/dL. Primary PCI was performed with the result of single-vessel disease with total occlusion at proximal LAD. DES was implanted at proximal LAD with TIMI flow 2. The patient was transferred to CVCU with stable hemodynamic.

Transthoracic echocardiography (TTE) revealed ejection fraction by Teich was 48% with hypokinetic on anterior, anteroseptal, and septal walls. His hemodynamic profile performed PCWP 18,02 mmHg, SVR 1757 dynes.sec/cm5, CO 3,26 L/min, CI 2,22 L/min2, Est RAP 10 mmHg. We continue the therapy with continuous infusion of furosemide, DAPT, high-intensity statin, and ACE inhibitor. On the second day, the patient experienced non-sustained VT and developed to pulseless sustained VT (Figure 3C). 360-joule defibrillation followed by CPR was performed. The ECG converted to sinus with transient AV block, and the patient had ROSC. We administered intravenous lidocaine 1,5 mg/kg bolus and followed by 2 mg/min. The patient rapidly became hemodynamically unstable. We decided to administer up-titration intravenous dopamine and norepinephrine. Finally, the patient was pronounced to death due to refractory cardiogenic shock.

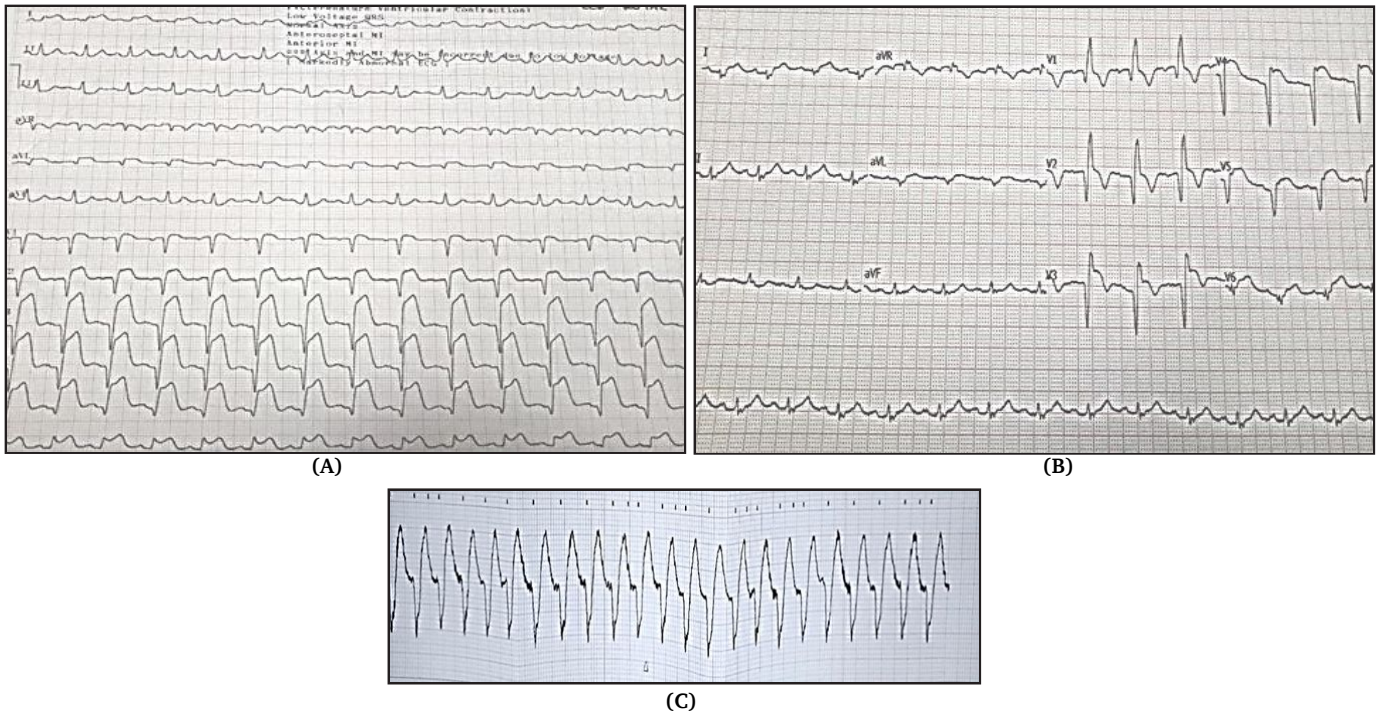


Figure 3. (A) First ECG from the previous hospital. (B) New-onset RBBB occurred on admission. (C) Pulseless sustained VT.

3. Discussion

The link between myocardial infarction caused by a substantial occlusion and ECG abnormalities other than STEMI and STEMI equivalents is being researched. Development of a new RBBB in a clinical situation of chest discomfort or an equivalent is one of the circumstances. RBBB was not regarded a diagnostic criterion for ACS on its own, unlike LBBB.[7] The link between acute myocardial infarction and new or old RBBB was investigated in a meta-analysis by Hazem et al. They came to the conclusion that people with bundle branch block had a higher risk of all-cause mortality in the 30-day follow-up period than those without.[8]

The new-onset RBBB is associated with proximal LAD blockage and a large region of infarction. The right bundle branch and the left bundle branch's anterior fascicle are perfused by the septal perforator arteries of the LAD in 90% of cases, and their obstruction causes RBBB. RBBB can cause a variety of fascicular or AV nodal blockages.[9] QRS prolongation varies on whether the right-bundle conduction is entirely blocked and whether other parts of the conduction system are also impaired.[10] Severe heart failure, AV block, malignant ventricular arrhythmias, and cardiogenic shock are all possible complications of this condition.

The link between new-onset RBBB and acute myocardial infarction is highlighted in our case studies. Wang et al. examined the prognostic efficacy of new-onset RBBB in acute MI using a meta-analysis of five trials (874 patients). Patients with acute MI who have new-onset RBBB have a higher risk of cardiogenic shock and ventricular arrhythmia, but not of cardiac failure, according to the findings.[10] Patients with bundle branch block, particularly those with LBBB, exhibited worse baseline features, according to another study by Timoteo et al.[11] They also had increased all-cause mortality and worse outcomes, particularly RBBB, in long-term follow-up. It is necessary to be aware of these patients and to pay special attention to them. In terms of pharmacological treatment and invasive technique, they must be treated at least as aggressively as individuals with normal QRS duration .[11] According to a study conducted by Iwasaki et al., new-onset persistent RBBB is one of the most important independent risk factors for in-hospital adverse events.[12]

Due to the multiple studies on the association between RBBB and acute myocardial infarction, a revised concept from The Management of STEMI ESC Guidelines 2017 suggested early reperfusion in STEMI with RBBB as new-onset.[6] The goals are to minimize the duration of RBBB and recover LAD and septal branch blood flow. However, these serial cases showed that poor clinical outcomes remain the same despite revascularization. These RBBB presences with varying Killip degrees classification were related to TIMI flow less than three as a final result.

As a result, we recommend more independent investigations in the appropriate clinical environment focused on RBBB and its relationship to myocardial infarction and coronary heart disease. This approach will help researchers learn more about the impact of acute and chronic new-onset RBBB, as well as the implications for myocardial infarction prognosis, outcome, and therapy.

4. Conclusion

New-onset RBBB in the presence of ischemic symptoms should alert the possibility of a catastrophic coronary occlusion, which necessitates rapid intervention and is linked to a higher risk of short-term mortality. This higher mortality may be due to the presence of new-onset AF with the rapid ventricular response, AHF, malignant ventricular arrhythmia, and mostly cardiogenic shock. These serial cases suggested that new-onset RBBB could be a poor prognosis in STEMI, even though revascularization was performed without delay once the diagnosis was confirmed. The poor clinical outcomes were associated with TIMI flow less than 3. As a result, it is reasonable to reexamine and reconsider its relevance in the era of reperfusion therapy. New-onset RBBB coexists with STEMI should be listed in the prospective registry to evaluate its prognostic value role.

5. Declarations

5.1. Ethics Approval and Consent to participate

Not applicable.

5.2. Consent for publication
Not applicable.

5.3. Availability of data and materials
Data used in our study were presented in the main text.

5.4. Competing interests
Not applicable.

5.5. Funding source
Not applicable.

5.6. Authors contributions
Idea/concept: ADA, IPD. Design: ADA, AA. Control/supervision: AA, AKR. Data collection/processing: ADA, IPD, EPBM. Analysis/interpretation: IPD, EPBM, AKR. Literature review: ADA, IPD. Writing the article: ADA, IOPD, EPBM. Critical review: AA, AKR. All authors have critically reviewed and approved the final draft and are responsible for the content and similarity index of the manuscript.

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