

Case report: Posterior myocardial infarction in presence of right bundle branch block: an old concept with new findings

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Background

The diagnosis of acute ischaemic coronary syndromes in presence of an intra-ventricular conduction disturbance represents a clinical challenge. In the cardiac segmentation model the posterior wall is replaced by the basal inferior segment. However, in the clinical scenario of acute coronary syndrome the concept of posterior myocardial infarction (PMI) endures. The association of a PMI and right bundle branch block (RBBB) is a rare condition characterised by broad R waves and ventricular repolarization disorders in right precordial leads in both entities, which could lead to misinterpretation and delay in reperfusion therapy.

Case Summary

We describe a case report of a 74-year-old man with acute chest pain and an electrocardiogram with broad R waves, a 4 mm ST-segment downsloping (excessively discordant) in right precordial leads, RBBB, and ST-segment elevation in posterior leads. There was resolution of ST-segment downsloping in right precordial leads after percutaneous coronary intervention and stenting of the circumflex artery, with disturbance of the repolarization process only attributable to RBBB.

Discussion

Patients with acute chest pain with RBBB and a ST segment with an excessive downsloping (out of proportion of what is expected in isolated RBBB) suggest PMI with occlusion of the circumflex coronary artery.

Keywords

Posterior myocardial infarction • Bundle branch block • Right bundle branch block • Case report

Learning points

- In acute myocardial ischaemia with right bundle branch block the excessive discordance of the ST segment in V1–V2 suggests the possibility of an associated posterior myocardial infarction.
- ST-segment elevation in posterior leads and the resolution of ST-segment depression following angioplasty support this theory.

Introduction

Controversy exists regarding the existence of posterior myocardial infarction (PMI). It has been suggested that the electrocardiographic findings described in this entity correspond to infarction of the lateral wall (due to the oblique arrangement of the heart) and in the segmentation model, the posterior wall is replaced by the basal inferior segment.^{1,2} Other authors describe the posterior wall in the isolated heart.³ However, in the American Heart Association (AHA)/American College of Cardiology Foundation (ACCF)/Heart Rhythm

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Society (HRS) standardization recommendations of acute ischaemia⁴ and in the 2017 European Society of Cardiology (ESC) guidelines of acute myocardial infarction with ST-segment elevation (STEMI) the definition of PMI is maintained and recommends immediate reperfusion.⁵ There are few reports of the association of PMI and right bundle branch block (RBBB);⁶ this represents a challenge in diagnosis due to the finding of broad R waves and ventricular repolarization disorders in right precordial leads in both entities.

Timeline

Events	
Initial presentation at the emergency room	<ul style="list-style-type: none"> • 3-h chest pain • Respiratory failure and need of advance air-way support • Electrocardiogram with broad R waves, 4 mm ST-segment downsloping in right precordial leads, right bundle branch block (RBBB), and ST-segment elevation in posterior leads • Coronary angiography with total thrombotic occlusion in the proximal segment of the circumflex artery • Bare metal stent was placed • The patient developed cardiogenic shock and intra-aortic balloon pump was placed; norepinephrine, vasopressin, and dobutamine were administered with clinical improvement
48 h post-arrival	<ul style="list-style-type: none"> • Electrocardiogram with disturbance of repolarization only attributable to RBBB • The patient developed acute renal failure and haemodialysis was initiated
After 5 days.	<ul style="list-style-type: none"> • During a haemodialysis session, he developed sustained ventricular tachycardia with degeneration in asystole • CPR was initiated, there wasn't return of spontaneous circulation • Patient decease

Case presentation

A 74-year-old man with Type 2 diabetes and hypercholesterolaemia arrived to the emergency room with 3-h chest pain that began after emotional stress. The patient was dyspnoeic with SaO₂ 75%, HR 130 b.p.m., and BP 130/80 mmHg. Jugular venous engorgement and diffuse pulmonary rales were found. The electrocardiogram (ECG) showed broad R waves, 4 mm ST-segment downsloping with RBBB in the right precordial leads. Posterior leads showed pathological Q waves and STEMI and there wasn't ST elevation in V4R–V6R (Figure 1). Laboratory studies reported: troponin I 2.5 ng/mL (0.01–0.04 ng/mL), CKMB 70.6 ng/dL (0.06–6.3 ng/dL), and NT-proBNP 4075 pg/mL (15–125 pg/mL). The initial management included oxygen, aspirin 300 mg, clopidogrel 600 mg, unfractionated heparin

4000 IU, atorvastatin 80 mg, and furosemide 60 mg. The patient required intubation. Coronary angiography was performed with total thrombotic occlusion TIMI V of the proximal segment of the circumflex artery (CX). Left main artery, left anterior descending artery, and right coronary artery had significant chronic lesions. Percutaneous coronary intervention (PCI) and stenting was done in CX with final flow TIMI 3 (Figure 2), we decide to treat only the culprit vessel because the other arteries present chronic lesions with severe calcification. After PCI the patient developed cardiogenic shock and norepinephrine 0.5 mcg/kg/min, vasopressin 0.02 U/min and dobutamine 10 mcg/kg/min were administered and an intra-aortic balloon pump was placed with clinical improvement. The ECG post-PCI showed RBBB with resolution of the ST-segment downsloping with persistence of acuminated T waves in right precordial leads; the ECG performed 48 h after arrival showed alteration of the process of repolarization attributable to RBBB (Figure 3). The patient developed acute renal failure and haemodialysis was initiated. On his 5th day at the coronary unit, he developed hypotension during a haemodialysis session attributable to sustained ventricular tachycardia with degeneration in asystole. Advance cardiovascular life support was provided but there wasn't return of spontaneous circulation.

Discussion

Ventricular repolarization in right bundle branch block

Right bundle branch block is an intra-ventricular conduction delay or block at the right bundle branch and thus depolarization takes place through the unspecific muscle generating a 'jumping wave' phenomenon through the interventricular septum towards the right ventricle mass (Figure 4A). The repolarization vector (which at this point is predominant at the interventricular septum) moves away from V1 to V2 accordantly to the dipole theory (Figure 4B). Thus, there is a negative T wave in those leads (rule of discordance).⁷

Myocardial infarction in presence of right bundle branch block

The AHA/ACC/HRS establish that the ECG diagnosis of myocardial infarction (MI) does not modify in the presence of RBBB or fascicular blocks,⁸ but the diagnosis of MI in this situation is challenging.⁶ There are several findings that suggest MI in the presence of RBBB⁹:

- *MI of the mid-interventricular septum:* R wave disappears in V1–V2 (with new onset of QR or qR complex) and there is no Q wave in V5–V6.
- *MI of the inferior interventricular septum:* the vectors that run through the mid and low septum disappear, with the appearance of QR or QS complex in V3–V4.
- *MI of the left ventricle free wall:* it is manifested by the presence of qrS or QrS in v5–v6, with q or Q and S always notched.

MI with QR or qR in V1–V2 with STEMI suggest occlusion of left anterior descending artery proximal to the first septal branch.¹⁰ The 2017 ESC guidelines for the management of STEMI recognise that in the presence of RBBB and ischaemic symptoms immediate reperfusion strategy must be established,⁵ based on a study that showed that patients with STEMI, RBBB was present in 6.3% of cases and mortality was higher than in patients with left bundle branch block (LBBB).¹¹

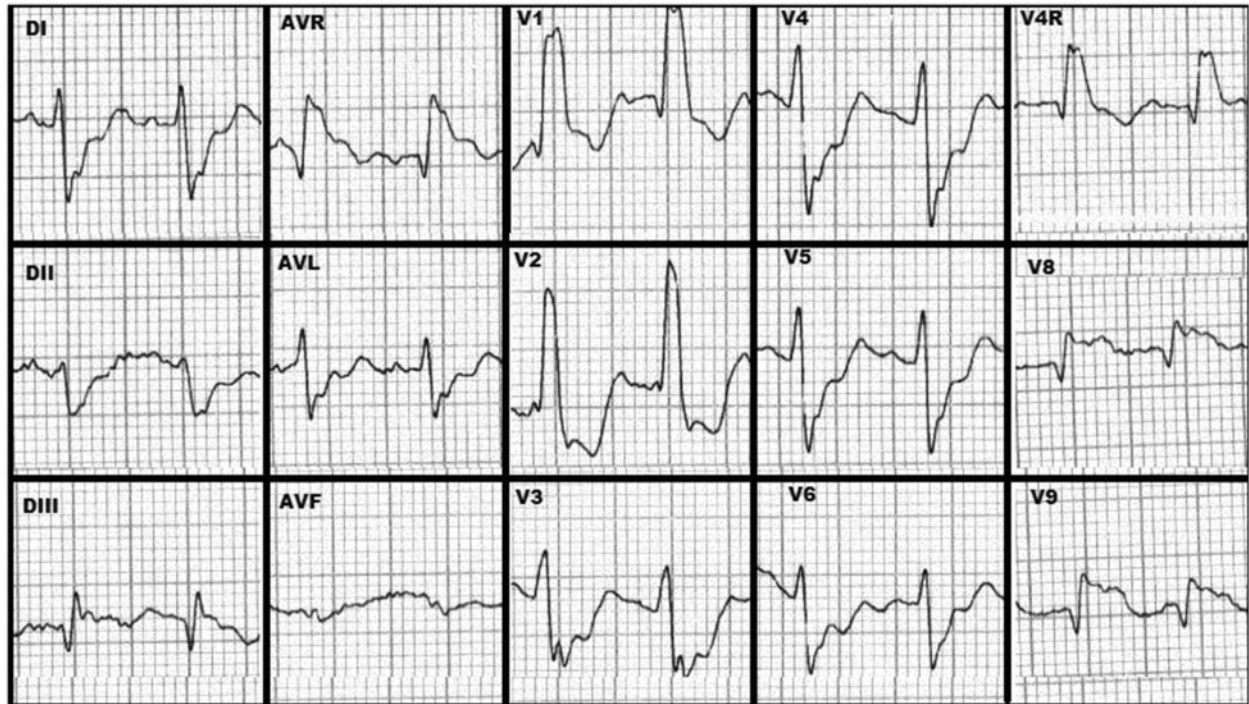


Figure 1 A 12 lead electrocardiogram at patient admission.

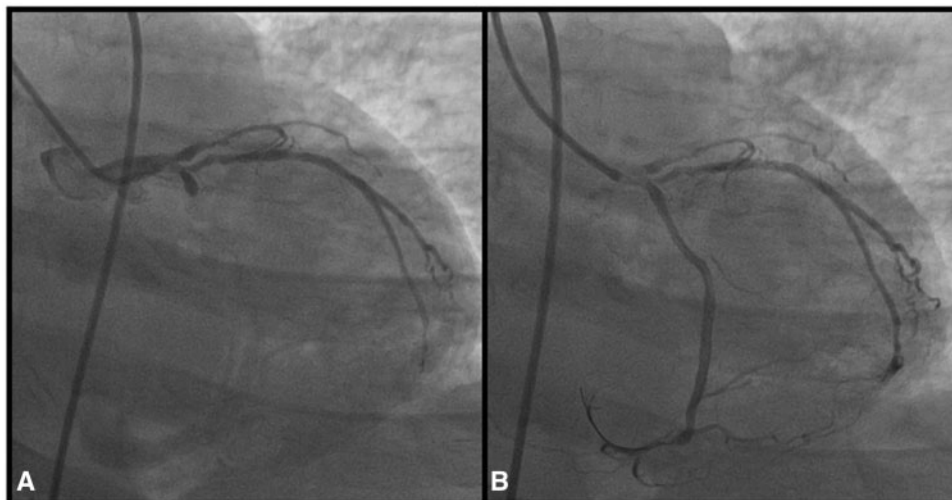


Figure 2 Coronary angiography pre- and post-percutaneous coronary intervention.

The electrocardiogram in posterior myocardial infarction

In the horizontal plane the vector of the 'electrically inactive area' is directed towards right precordial leads (V1–V2), the vector of the 'injured area' points towards the infarction (V7–V9) and the vector of the 'ischaemic area' is also directed forward (V1–V2), this is

expressed in the electrocardiogram by the presence of broad R waves, concave depression of ST segment and broad, symmetric T waves in right precordial leads¹²; the so-called 'mirror image' of the registry of the posterior leads (V7, V8, and V9) (Figure 5). When PMI occurs in absence of inferior infarction, the culprit vessel is usually the CX.¹³ There is lower proportion of STEMI (less than 50%) in this

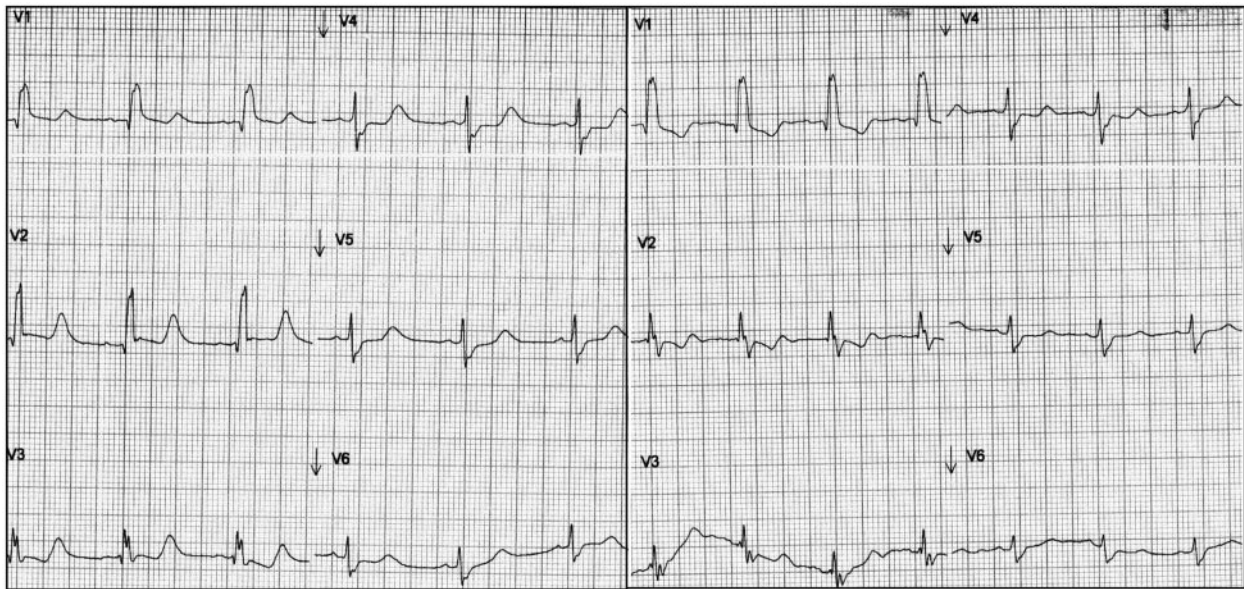


Figure 3 Electrocardiograms performed immediately and at 48 h after successful percutaneous coronary intervention.

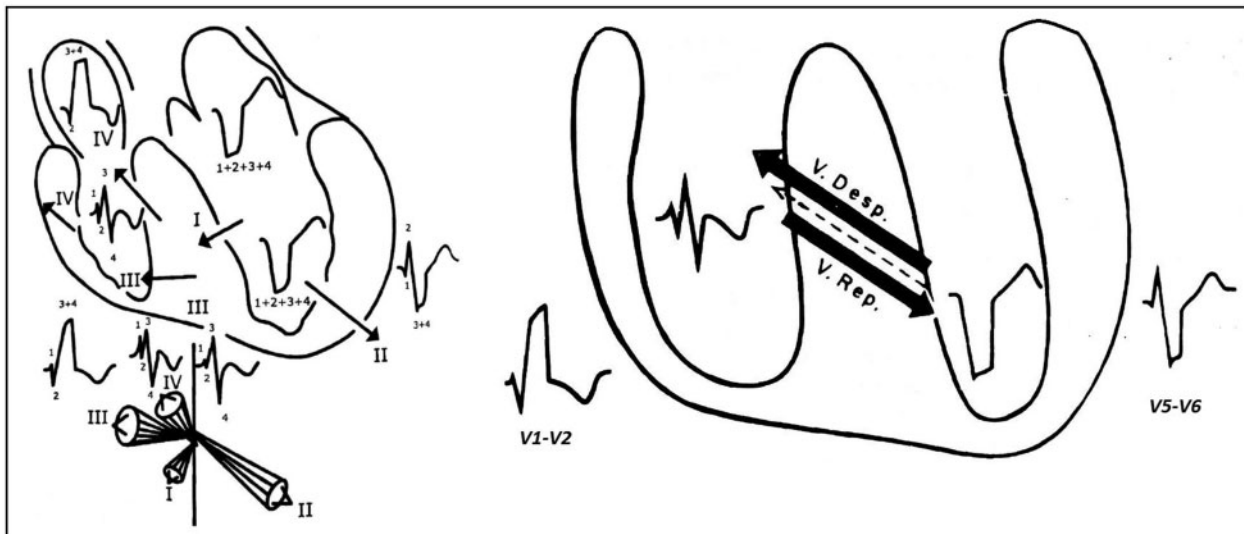


Figure 4 (A) The process of ventricular depolarization in the presence of right bundle branch block. (B) The process of ventricular repolarization in the presence of right bundle branch block. Reproduced from de Micheli et al.⁷

context compared to the occlusion of the left anterior or right coronary artery; 25% had ST-segment depression and 36% did not present electrocardiographic change.^{14,15} In clinical practice, this causes an underestimation of the acute artery occlusion and a delay in revascularization therapy.

Posterior myocardial infarction in the presence of right bundle branch block

In the presence of acute chest pain with an ischaemic pattern and a new or pre-existing RBBB, the excessive discordance (depression) of the ST segment in V1 and V2 suggests the possibility of an associated

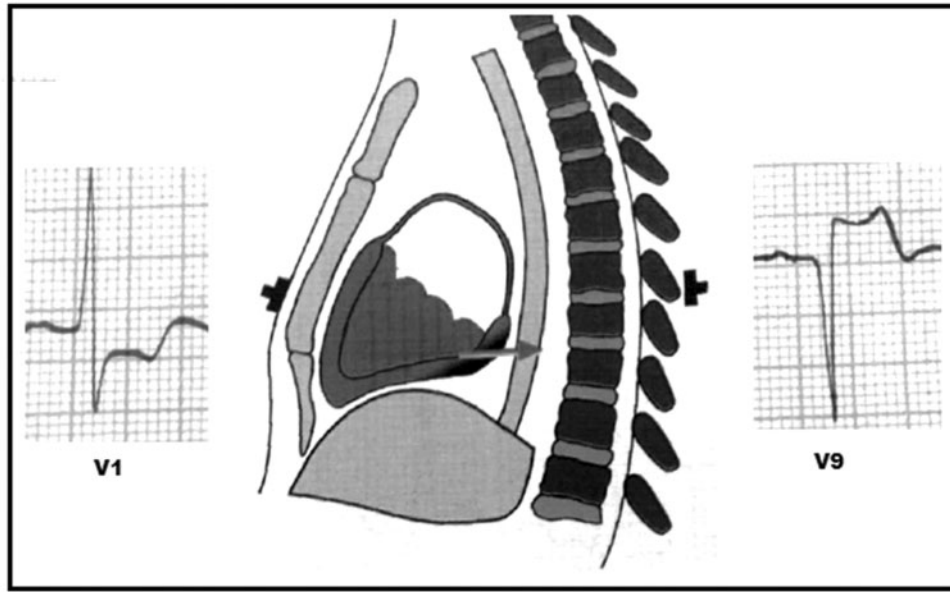


Figure 5 The vector of the 'injured area' points towards the infarction, with ST-segment depression in V1–V2 and ST-segment elevation in posterior leads (V7–V9). Reproduced from Castellano et al.¹⁶

PMI (corresponding to the mirror image of the 'injured area'). In this patient the ST segment depression of 4 mm in V2 (out of proportion to what would be expected in the isolated RBBB), the STEMI greater than 0.5 mm in posterior leads and the resolution of ST-segment depression following angioplasty in the context of total thrombotic occlusion of the CX, support this theory.

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Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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