

Isolated right ventricular myocardial infarction: a case report

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Background

Isolated right ventricular myocardial infarction (RVMI) due to a recessive right coronary artery (RCA) occlusion is a rare presentation. It is typically caused by right ventricle (RV) branch occlusion complicating percutaneous coronary intervention. We report a case of an isolated RVMI due to flush RCA occlusion presenting via our primary percutaneous coronary intervention ST-elevation myocardial infarction pathway.

Case summary

A 61-year-old female smoker with a history of hypercholesterolaemia presented via the primary percutaneous coronary intervention pathway with sudden onset of shortness of breath, dizziness, and chest pain while walking. Transradial coronary angiography revealed a normal left main coronary artery, large left anterior descending artery that wrapped around the apex and dominant left circumflex artery with the non-obstructive disease. The RCA was not selectively entered despite multiple attempts. The left ventriculogram showed normal left ventricle (LV) systolic function. She was in cardiogenic shock with a persistent ectopic atrial rhythm with retrograde p-waves and stabilized with intravenous dobutamine thus avoiding the need for a transcutaneous venous pacing system. A computed tomography pulmonary angiogram demonstrated no evidence of pulmonary embolism while an urgent cardiac gated computed tomography revealed a recessive RCA with ostial occlusive lesion. A cardiac magnetic resonance imaging confirmed RV free wall infarction. She was managed conservatively and discharged to her local district general hospital after 5th day of hospitalization at the tertiary centre.

Discussion

This case describes a relatively rare myocardial infarction presentation that can present with many disease mimics which can require as in this case, a multi-modality imaging approach to establish the diagnosis.

Keywords

Case report • Ectopic atrial rhythm • Hypoxic respiratory failure • Cardiogenic shock • Isolated right ventricular myocardial infarction • Recessive right coronary artery occlusion

Learning points

- Isolated right ventricular myocardial infarction is a rare presentation typically caused by right ventricle branch occlusion complicating percutaneous coronary intervention or presenting as an acute coronary syndrome.
- The use of multi-modality imaging approach is encouraged where possible to help establish its diagnosis.

Introduction

Isolated right ventricular free wall myocardial infarction (MI) due to a recessive right coronary artery (RCA) occlusion occurs in 3% of all

MIs.¹ It is typically caused by the right ventricle (RV) branch occlusion complicating percutaneous coronary intervention (PCI).^{2,3} We report a case of an isolated RVMI due to flush RCA occlusion presenting via our primary PCI ST-elevation myocardial infarction pathway.

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Timeline

Timeline	Event
25 January 2020	Patient presented via primary percutaneous coronary intervention (pPCI) pathway, an invasive coronary angiography was performed which showed no significant stenosis in the dominant left coronary arterial system however the right coronary artery was not selectively entered despite multiple attempts using a variety of diagnostic catheters.
25 January 2020	Beside transthoracic echocardiography showed normal left ventricular systolic function, dilated right ventricle with severe systolic impairment and signs suggestive of right-sided pressure overload.
25 January 2020	Urgent computed tomography pulmonary angiography demonstrated no evidence of pulmonary angiography.
28 January 2020	Departmental agitated saline transthoracic echocardiography revealed the presence of patent foramen ovale.
29 January 2020	Cardiac gated computed tomography revealed an ostial occlusive lesion in the recessive right coronary artery.
31 January 2020	Cardiac magnetic resonance imaging performed confirmed the presence of an isolated right ventricular myocardial infarction with no evidence of haemodynamically significant shunt.
31 January 2020	Patient was discharged to her local district general hospital for her ongoing care.
05 February 2020	Multi-disciplinary device team meeting was held following a 5 s sinus pause (nocturnal) on Day 10 post-index presentation, a consensus was made not to implant a permanent pacing system as there was not enough indication.
06 February 2020	Patient was enrolled into cardiac rehabilitation programme and discharged home on dual antiplatelet therapy.

Case presentation

A 61-year-old female smoker with history of hypercholesterolaemia presented via the pPCI pathway with sudden onset of shortness of breath, dizziness and chest pain while walking. Her electrocardiogram (ECG) revealed ST elevation inferiorly and V1–V3 with transient right bundle branch block (RBBB) and junctional bradycardia (Figure 1). She was loaded with Aspirin 300 mg, Ticagrelor 180 mg orally on presentation, and then regular doses of Aspirin 75 mg daily,

Ticagrelor 90 mg b.i.d. with prophylactic dose Enoxaparin 40 mg daily in line with our hospital's pPCI protocol.

Transradial coronary angiography revealed a normal left main coronary artery, large left anterior descending artery that wrapped around the apex and a dominant left circumflex artery with non-obstructive disease (Supplementary material online, Videos S1 and S7). The RCA was not visualized and subsequently engaged despite multiple attempts using a variety of diagnostic catheters (Supplementary material online, Video S2). A left ventriculogram showed normal left ventricle (LV) systolic function (Supplementary material online, Video S3). Physical examination revealed an elevated jugular venous pressure, clear lung fields, normal heart sounds with a pansystolic murmur, and signs of hypoperfusion.

Her National Early Warning Score (NEWS) was 10 based on respiratory rate of 27/min, oxygen saturation of 88% (room air), temperature 35.9°C, blood pressure 98/54 mmHg, and pulse rate of 53 beats/min with signs of hypoperfusion and oliguria (initial serum lactate of 2.5 mmol/L, urine output of <0.5 mL/kg/h within the first 4 h, respectively). She was started on oxygen therapy (4 L/min via nasal cannula) and intravenous dobutamine at a rate of 2.5 µg/kg/min and repeat haemodynamic observation after 30 min revealed a NEWS score of 4 based on respiratory rate of 20 per minute, oxygen saturation of 94% (on oxygen), temperature 36.6°C, blood pressure 120/67 mmHg, and pulse rate of 69 beats/min.

She was in cardiogenic shock with a persistent ectopic atrial rhythm with retrograde p-waves indicative of ventriculoatrial conduction, present (Figure 2), but stabilized with intravenous dobutamine thus avoiding the need for a transcutaneous venous pacing system. Using Fridericia correction, her QTc 479 ms which was prolonged (Figure 2) became normalized upon discharge. A bedside transthoracic echocardiography (TTE) revealed a normal LV size and function (ejection fraction > 55%), leftward systolic septal wall motion suggestive of right-sided pressure overload and moderate RV dilatation (basal RV diameter of 44 mm). Right ventricular systolic function was severely impaired with a tricuspid annular plane systolic excursion (TAPSE) of 5 mm and free wall akinesis. There was severe tricuspid regurgitation (TR) with evidence of flow reversal in the hepatic veins and the estimated RV systolic pressure, based on the TR gradient and estimated right atrial pressure, was 45.76 mmHg (right atrial pressure 10 mmHg) (Supplementary material online, Videos S8 and S9).

Admission bloods revealed a normocytic anaemia with a haemoglobin 109 g/L (normal range 115–165 g/L), raised white cell count $16.2 \times 10^9/L$ (normal range $4.0\text{--}10.0 \times 10^9/L$) and C-reactive protein 55.1 mg/L (normal value <5 mg/L). High sensitivity cardiac Troponin T level was elevated from 46 to 1998 (normal value <14 ng/L), with a N-terminal pro B-type natriuretic peptide level of 3550 ng/L (normal value <263 ng/L).

Arterial blood gas analysis showed a pH 7.51 (normal range 7.35–7.45), pO_2 7.0 kPa (normal range 10–14 kPa), pCO_2 4.20 kPa (normal range 4.5–6 kPa), lactate 1.4 mmol/L (normal range 0.5–2.2 mmol/L), HCO_3^- 27.8 mmol/L (normal range 22–26 mmol/L) with an oxygen saturation 94.4% on 4 L of oxygen via nasal cannula indicating hypoxic respiratory failure with alkalosis.

The differential diagnoses included massive pulmonary embolism (PE), bradycardia with haemodynamic disturbance, right ventricular myocarditis, and isolated right ventricular myocardial infarction (RVMI).

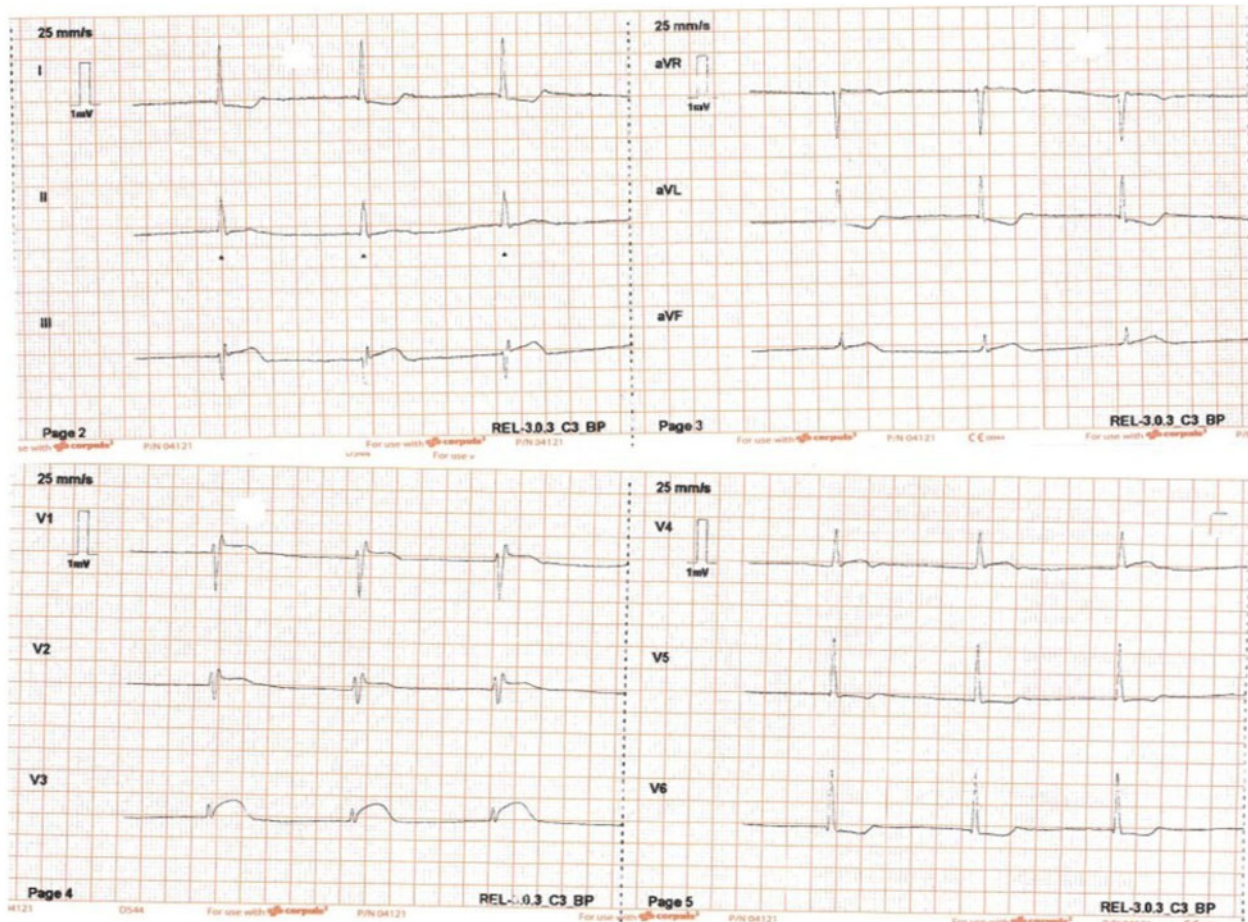


Figure 1 Presenting electrocardiogram (ECG) with inferior and V1–V3 ST-segment elevation with right bundle branch block (RBBB) and junctional bradycardia thereby triggering the primary percutaneous coronary intervention pathway.

Her chest radiograph was normal, computed tomography pulmonary angiogram demonstrated no evidence of PE, an urgent cardiac gated computed tomography (*Figure 3, Supplementary material online, Video S4*) revealed a recessive RCA with ostial occlusive lesion. A cardiac MRI was performed once the patient had stabilized demonstrated a normal RV end-diastolic volume (78 mL/m^2), an elevated RV end-systolic volume (42 mL/m^2) with an ejection fraction of 46% with marked regional wall motion abnormality along the RV free wall coupled with extensive late gadolinium contrast enhancement, consistent with infarction (*Figure 4A, B, Supplementary material online, Videos S5 and S6*). A departmental agitated saline TTE study, performed prior to the MRI, revealed the presence of a patent foramen ovale but this was not haemodynamically significant on MRI. Left ventricular systolic function was normal across all imaging modalities.

She was managed conservatively and discharged to her local district general hospital (DGH) after 5 days in our tertiary centre. She had a nocturnal sinus pause lasting 5 s on telemetry at Day 10 post-index presentation. Multi-disciplinary device team discussion felt there was not enough of an indication to proceed to a permanent pacing system. She was enrolled in a cardiac rehabilitation programme and discharged home on Aspirin, Ticagrelor (b.i.d. for

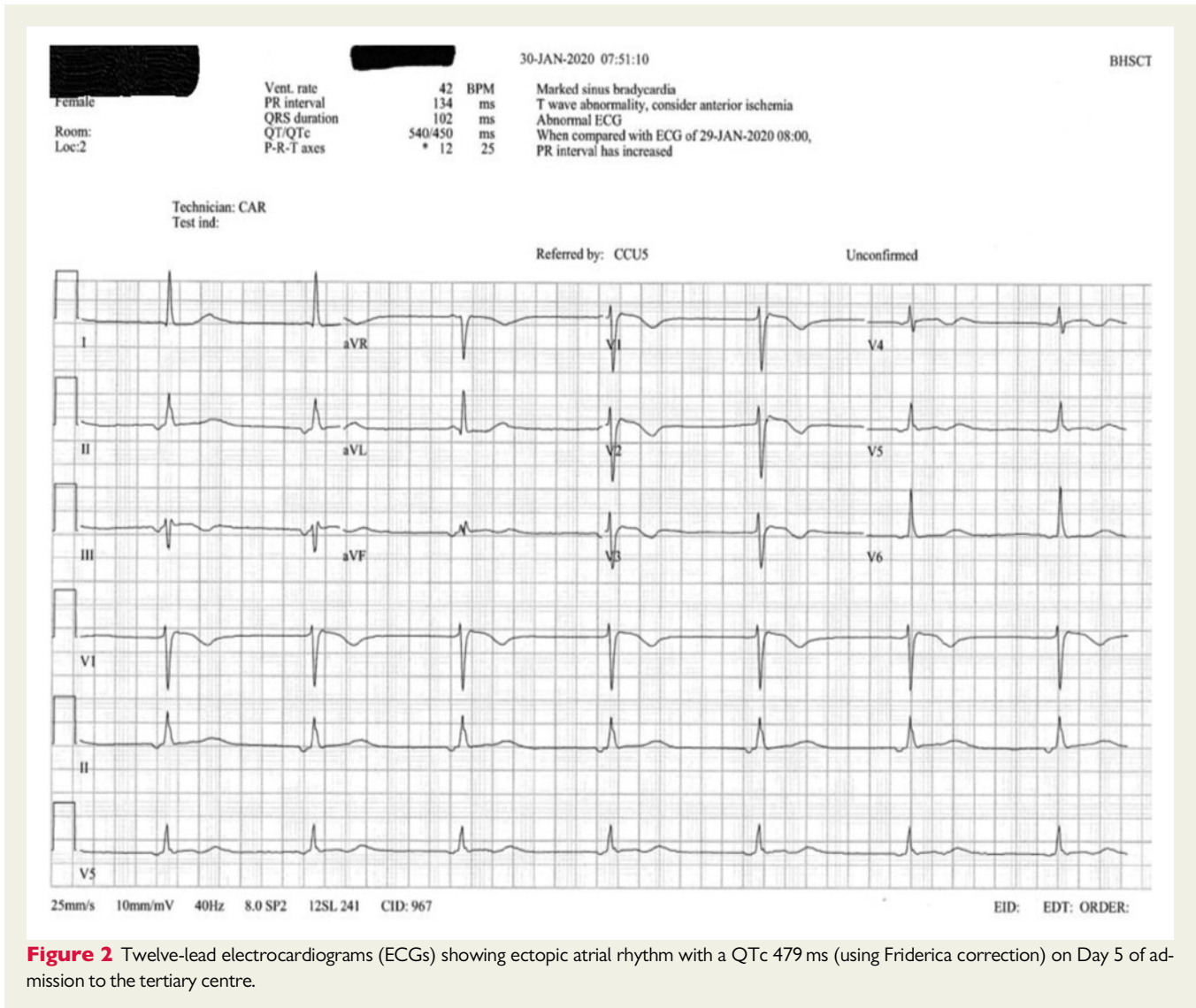
12 months), Atorvastatin, Spironolactone, and Lansoprazole with plan for an outpatient follow-up.

Discussion

Isolated RVMI due to a recessive right coronary artery (RCA) occlusion occurs in 3% of all MIs¹ and its incidence rate is not yet established. It is typically caused by right ventricle (RV) branch occlusion complicating percutaneous intervention^{2,3} but may also occur in the presence of significant RV hypertrophy and normal coronary artery;⁴ however, it was due to flush occlusion of recessive RCA in this case.

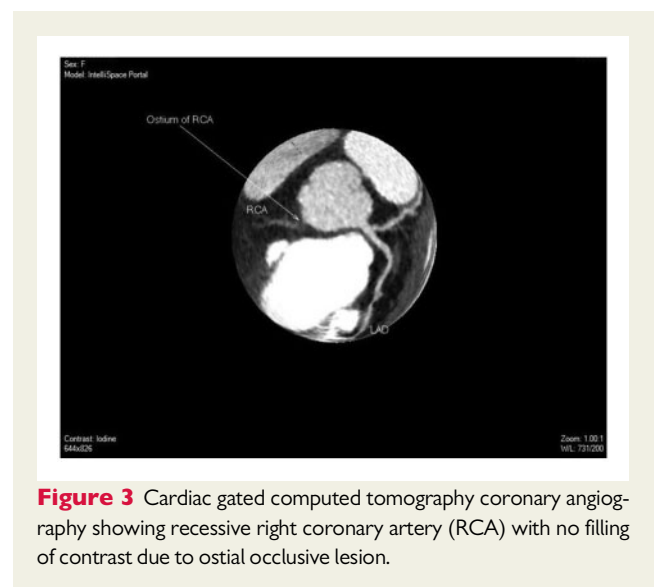
The ECG changes in RVMI mostly demonstrates ST-segment elevation in inferior leads, V1–V2, and right precordial leads (V3R and V4R),^{2,3} but there have also been reports of RVMI associated with V1–V4, as evidenced in this case, or V5 ST-segment elevation mimicking an anterior MI.^{5,6}

Right ventricular myocardial infarction can be associated with bradyarrhythmia, hypotension, and cardiogenic shock⁷ due to significant reduction in the preload causing hypotension in the absence of congestive cardiac failure,⁸ as seen in this case. Ticagrelor a potent P2Y12 inhibitor is recommended as first-line antithrombotic agent in



addition to Aspirin in acute coronary syndrome; however, it may exert a deleterious effect in patients already presenting with symptomatic bradycardia causing atrioventricular block published in few case reports.^{9,10}

While acute massive PE may rarely be complicated by bradycardia,¹¹ it typically presents with shock, RV dysfunction, hypoxaemia, and tachycardia. Computed tomography pulmonary angiogram excluded PE in this case. Isolated RV myocarditis is another differential diagnosis and is usually preceded by a viral prodrome^{12,13} which was absent in this case, and the cardiac MRI findings were typical of myocardial infarction as opposed to inflammation. Conversely in a chronic presentation, Arrhythmogenic right ventricular cardiomyopathy (ARVC) or a haemodynamically significant atrial septal defect (ASD) with Eisenmenger's physiology are possible differential diagnoses for a dilated and dysfunctional right heart. ARVC would not usually present with this clinical picture as it typically presents with palpitations, syncope, ventricular tachycardia, and sudden cardiac death¹⁴ and subsequent investigations revealed RVMI as the cause of the presentation as opposed to fulfilment of task force criteria for



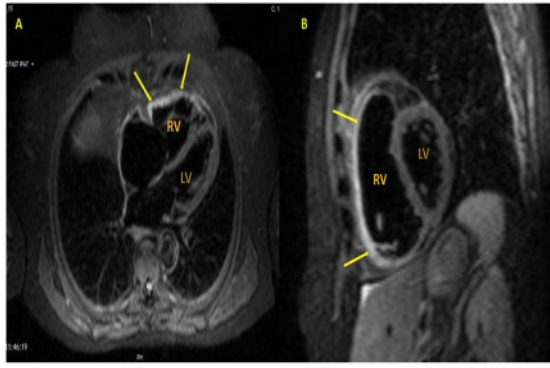


Figure 4 (A, B) Cardiac magnetic resonance imaging (MRI) showing extensive late gadolinium contrast enhancement (yellow arrows), consistent with infarction.

ARVC. In ASD with Eisenmenger's physiology patients usually present with exercise intolerance, breathlessness, arrhythmia, and cyanosis in the 4th decade of life,¹⁵ but assuredly clinical examination, TTE, and cardiac MRI excluded this possibility.

The management of RVMI includes intravenous fluid resuscitation, inotropes, atropine and, where indicated, pacing for acute conduction disturbance,⁸ alongside the restoration of coronary flow if possible. This case report is an example of a rare MI presentation that can present with many disease mimics which can require, as in this case, a multi-modality imaging approach to establish the diagnosis.

Lead author biography



Dr Adeogo Akinwale Olusan is a specialist trainee in cardiology in Northern Ireland, United Kingdom. He is a member of the Royal College of Physicians of the United Kingdom (MRCPUK) and London (MRCP London) and a certified member of the British Society of Echocardiography (BSE). He also holds a Master of Science (MSc) degree and Diploma of Imperial College

(DIC) in Preventive Cardiology. He has a special interest in cardiovascular interventions and has represented the Northern Ireland Deanery in a number of national and international cardiovascular meetings.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

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 guidelines.

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