

CASE REPORT

Isolated right ventricular infarction presenting with anterior ST-segment elevation: A case for careful assessment of right ventricular branch occlusion

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KEYWORDS

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Abstract We describe the case of a 61-year-old man who presented with chest pain and ST-segment elevation in the anterior precordial leads (V1–V5) due to proximal occlusion of the right ventricular branch of a codominant right coronary artery. Primary coronary angioplasty and stenting of this branch was performed resulting in resolution of the chest pain and ST-segment elevation. Our description illustrates a case of isolated right ventricular infarction as an uncommon but important differential diagnosis of anterior ST-segment elevation. In addition, it highlights the value of careful review of the angiographic images in this context, as the culprit lesion may be a right ventricular branch occlusion. To the best of our knowledge, this is the first reported case of primary percutaneous coronary intervention treatment of a right ventricular branch occlusion causing isolated acute right ventricular myocardial infarction and anterior ST-segment elevation.

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PALAVRAS-CHAVE

Enfarte do miocárdio;
Ventrículo direito;
Angioplastia;
Stent

Enfarte isolado do ventrículo direito simulando enfarte da parede anterior: atenção ao ramo ventricular direito!

Resumo Descreve-se o caso de um homem de 61 anos que se apresentou com dor torácica típica e supradesnívelamento do segmento ST nas derivações precordiais (V1–V5). A coronarografia de urgência revelou a oclusão proximal de um ramo ventricular da artéria coronária direita. Foi realizada angioplastia primária deste ramo com implantação de *stent* metálico convencional, resultando na resolução da dor torácica e da elevação ST. A presente descrição ilustra um caso de enfarte isolado do ventrículo direito como um diagnóstico diferencial pouco comum mas importante de elevação ST nas derivações precordiais. O presente caso realça ainda a importância de uma cuidadosa revisão das imagens angiográficas neste contexto, uma vez que

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a lesão culpada pode ser a oclusão de um ramo ventricular direito. Segundo a nossa pesquisa, o presente caso trata-se da primeira descrição de intervenção coronária percutânea primária de um ramo ventricular direito causando enfarte isolado do ventrículo directo e elevação ST nas derivações precordiais.
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Introduction

The presence of ST-segment elevation in the anterior precordial leads in patients with acute coronary syndromes usually indicates left anterior descending coronary artery occlusion. However, anterior ST-segment elevation has also

been described in right coronary artery (RCA) occlusion and is thought to signify right ventricular myocardial infarction (RVMI). We describe a case of isolated RVMI presenting with anterior ST-segment elevation due to proximal occlusion of a right ventricular branch that was treated by primary angioplasty.

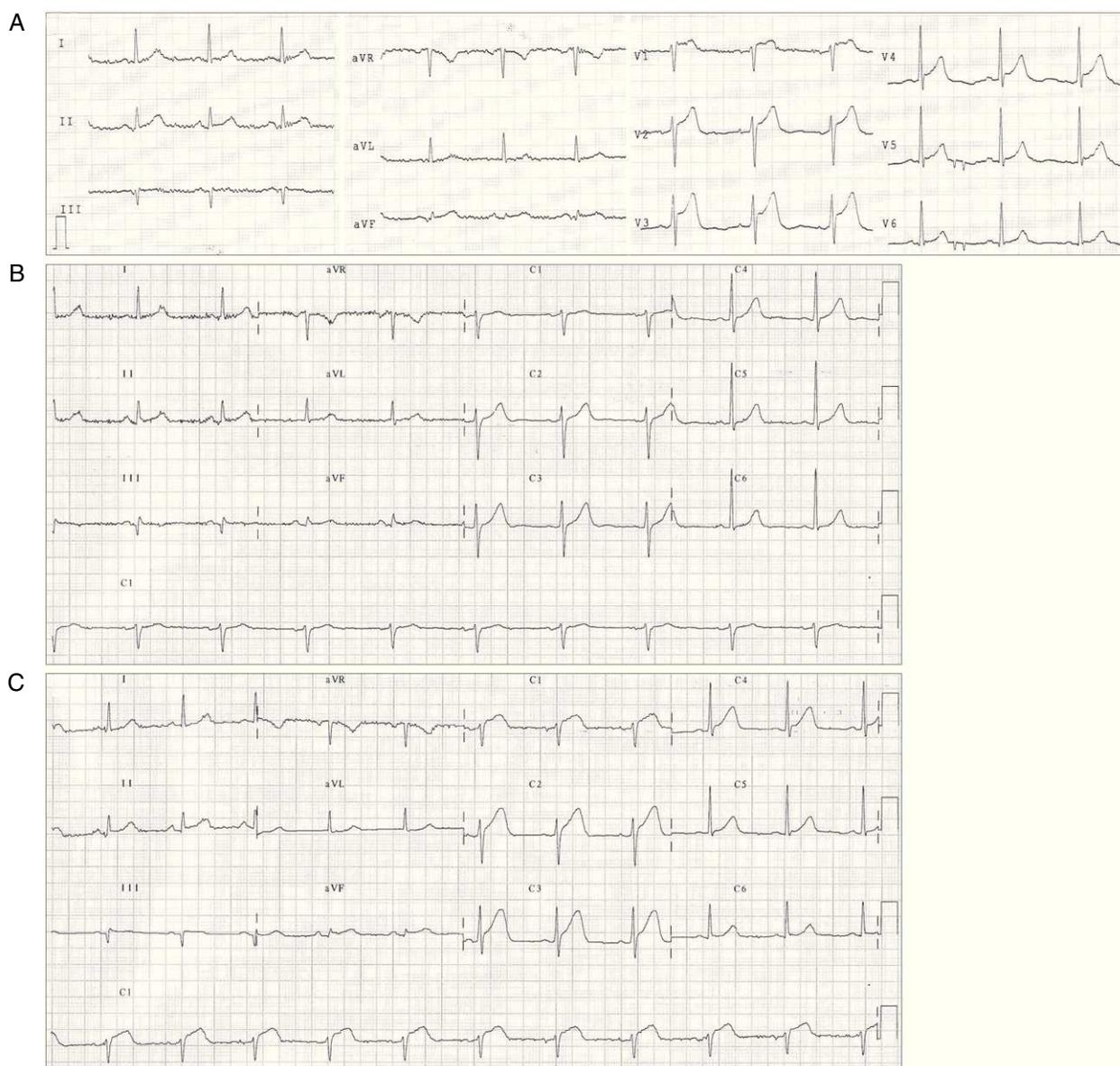


Figure 1 (A) Initial ECG showing ST-segment elevation in the anterior precordial leads. (B) Post-coronary angioplasty ECG showing normal sinus rhythm with resolution of ST-segment elevation and no anterior Q waves. (C) Acute stent thrombosis causing new ST-segment elevation in leads V1-V4 and slight ST-segment elevation in leads II, III, and aVF.



Figure 2 Left coronary angiogram showing no significant obstruction in the left anterior descending coronary artery (left anterior oblique view with cranial angulation).

Case report

A 61-year-old man with no history of cardiac disease presented to a community hospital reporting typical chest pain 3 hours in duration with more severe intensity in the last 30 minutes. The initial standard 12-lead electrocardiogram (ECG) showed ST-segment elevation in leads V1–V5 and slight ST-segment elevation in the inferior leads (Fig. 1A). The patient was given 5000 U of unfractionated heparin IV, 600 mg clopidogrel oral loading dose and 300 mg oral aspirin, and was transferred to our hospital for urgent catheterization. He arrived at our cath lab hemodynamically stable, with persistent chest pain. Coronary angiography, performed four and a half hours after symptom onset, showed a normal left coronary artery (Fig. 2) and patent main vessel of the codominant RCA. Left ventriculography was normal. Careful review of the coronary angiogram revealed proximal

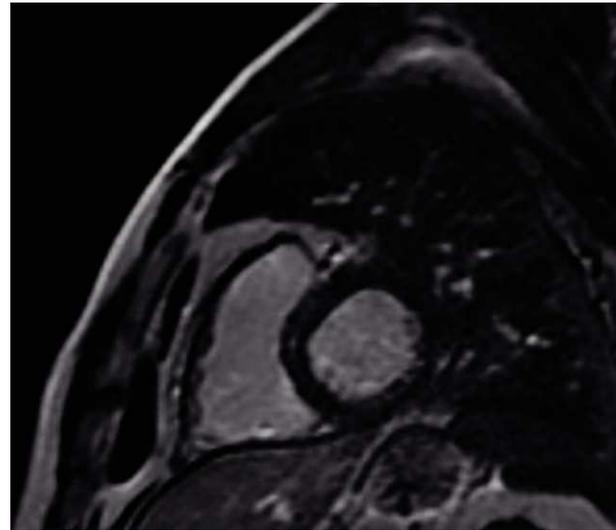


Figure 4 Contrast-enhanced magnetic resonance imaging demonstrating absence of delayed hyperenhancement in the right or left ventricles.

occlusion of the right ventricular (RV) branch of the RCA (Fig. 3A and B). The RV branch occlusion was managed with balloon dilation and stenting (2.0 mm × 12.0 mm bare-metal stent at 16 atm, Multi-Link Mini Vision®, Abbott Vascular, USA), resulting in resolution of the chest pain and ST-segment elevation (Figs. 1B and 3C). One hour after the procedure the patient experienced a new episode of chest discomfort and ST-segment changes similar to the initial ECG (re-elevation in leads V1–V4 and slight elevation in leads II, III, and aVF) (Fig. 1C). Emergency coronary angiography demonstrated acute stent thrombosis, which was probably related to previous inadequate stent covering of the proximal portion of the coronary plaque. Abciximab infusion was initiated, thrombus aspiration was performed and another proximal and overlapped bare-metal stent (2.25 mm × 12.0 mm at 12 atm, Multi-Link Mini Vision®, Abbott Vascular, USA) was implanted, again resulting in resolution of chest pain and ST-segment elevation. The peak troponin I level was 5.2 ng/ml. The patient recovered without sequelae and was asymptomatic and fully active one

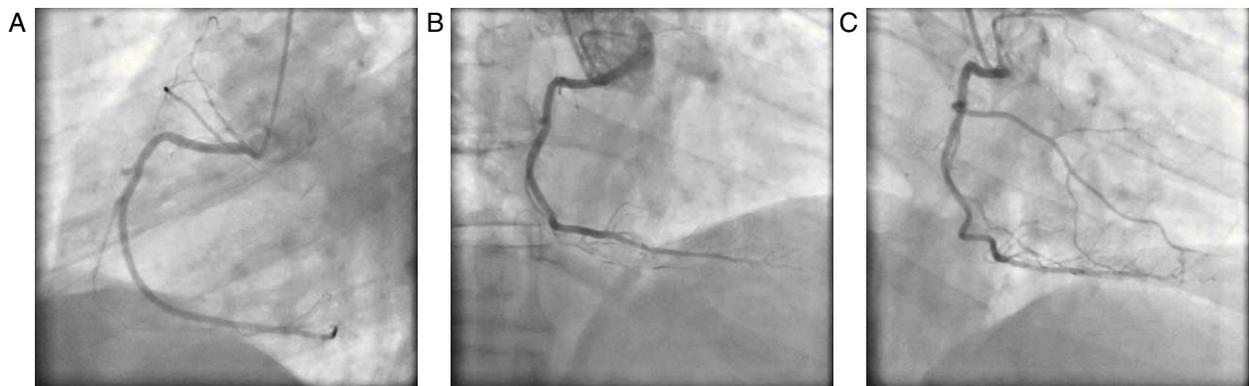


Figure 3 Right coronary angiogram (A, left anterior oblique view; B, right anterior oblique view) demonstrating total occlusion of the right ventricular branch. (C) Angiogram of the right coronary artery (right anterior oblique view) after stenting of the right ventricular branch with TIMI 3 flow.

month later. Contrast-enhanced magnetic resonance imaging (MRI) performed 30 days after presentation showed normal left ventricular function and normal right ventricular size and motion. No area of delayed hyperenhancement was visualized (Fig. 4).

Discussion

Isolated RVMI is uncommon, with autopsy series indicating that it occurs in less than 3% of all patients with myocardial infarction.¹ It has been described essentially in two anatomical contexts: occlusion of a nondominant RCA,² and loss of large RV branches during coronary angioplasty of the RCA.³ There have been few reports of anterior ST-segment elevation caused by isolated RVMI due to RV branch occlusion in the absence of percutaneous coronary intervention.⁴

Although it is usually considered that occlusion of a small nondominant right coronary artery is not associated with significant consequences, there are reports of cases presenting with sudden cardiac death.⁵ Importantly, there are also reports of sudden cardiac death in which autopsy confirmed the occlusion of a branch of the RCA causing isolated RVMI.⁶ Our case, to the best of our knowledge, is the first one reported of primary percutaneous coronary intervention treatment of an RV branch occlusion causing isolated acute RVMI and anterior ST-segment elevation. Acute stent thrombosis with recurrence of chest pain and anterior ST-segment re-elevation was successfully managed percutaneously. Revision of images of the first intervention showed that the proximal portion of the unstable coronary plaque was not covered by the stent. Another stent was implanted, proximal and overlapped to the previous one, to cover all the unstable plaque. Minimal myocardial necrosis was demonstrated by troponin assay and follow-up contrast-enhanced MRI showed no areas of delayed hyperenhancement in the right or left ventricles.

When isolated RVMI occurs, the ECG may show an acute anterior ST-segment elevation pattern (leads V1 through V5) and right ST-segment elevation (leads V3R through V6R, which were not performed in our case). However, isolated ST-segment elevation due to RVMI is uncommon in clinical practice. Since RVMI usually occurs with simultaneous left ventricular inferior infarction, the electrical current of injury from the left ventricle dominates the RV electrical forces. The typical ECG changes in this setting are ST-segment elevation in the inferior leads with concomitant ST-segment depression in the precordial leads if the left ventricular posterior wall is involved.⁷

Certain electrocardiographic features have been suggested which may help differentiate ST-segment elevation secondary to isolated RVMI from left anterior descending artery territory infarction. However, these features are not pathognomonic and it may be impossible to make this distinction on the basis of electrocardiography alone. The

absence of Q-wave development in the anterior precordial leads has been reported as favoring the diagnosis of RVMI,⁷ as was observed in our case. Nevertheless, other suggested features such as progressive reduction in ST-segment elevation across the precordial leads⁷ were not seen in this case. In addition, an electrocardiographic criterion described by Lopez-Sendon et al.⁸ in the differential diagnosis of RVMI and anteroseptal MI is the relationship between ST-segment elevation in V4R–V3. In most patients with RVMI, ST-segment elevation in V4R is higher than that found in V1–V3.

Conclusion

Our description clearly illustrates a case of isolated RVMI as an uncommon but important differential diagnosis of anterior ST-segment elevation and highlights the value of careful review of angiographic images, as the culprit lesion may be an RV branch occlusion. Primary percutaneous intervention of these branch vessels is possible and enables complete resolution of chest pain and ST-segment changes, minimization of the damage to the RV myocardium, and avoidance of malignant arrhythmias.

Conflicts of interest

The authors have no conflicts of interest to declare.

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