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A patient with simultaneous anterior and inferior ST-segment elevation after percutaneous coronary intervention

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ABSTRACT

A patient who presented with acute inferior-right ventricular (RV) ST-segment elevation (STE) myocardial infarction (MI) is described. Coronary angiography showed a mid-right coronary artery (RCA) occlusion and high-grade proximal left anterior descending (LAD) artery stenoses. Electrocardiography (ECG) after stent angioplasty to the RCA showed new STE in leads V1-V6. Whereas STE pattern recognition was misleading, ECG analysis using vector concepts enabled exclusion of anterior MI due to proximal LAD artery occlusion and recognition of the RV origin of this ECG picture. The ability of the ECG to "capture" RV dilation that enabled the manifestation of this ECG picture is highlighted.

Case presentation

A 59-year-old diabetic male presented with chest pain of two-hour duration without signs of acute left or right-sided heart failure. His blood pressure and oxygen saturation were 120/70 mmHg and 98% at atmospheric air. Admission electrocardiography (ECG) (Fig. 1A) showed sinus rhythm, >0.1 mV ST-segment elevation (STE) at the J point in leads II, aVF and III, reciprocal ST-segment depression in leads aVL and I and 0.1 mV STE in the additional right-sided leads (V3R through V6R). Emergency coronary angiography showed acute occlusion of a dominant right coronary artery (RCA) in the mid segment just beyond the origin of a right ventricular branch (RV) and two high grade (>70%) stenoses in the proximal left anterior descending (LAD) artery. Percutaneous coronary intervention (PCI) to the RCA was subsequently performed with implantation of two 3.0 mm × 33 mm drug-eluting stents. In final angiogram of the RCA a Thrombolysis In Myocardial Infarction (TIMI) grade 3 flow and a TIMI myocardial perfusion grade 2 were shown along with occlusion of the stent-jailed RV and acute marginal branches and embolic occlusion of a posterior left ventricular branch. During PCI the patient developed recurrent episodes of ventricular fibrillation and was successfully managed with immediate defibrillation, potassium replenishment therapy and intubation. Because of persistent hypotension (systolic blood pressure < 90 mmHg) after cardiac arrest, despite fluid resuscitation, vasopressor and inotropic therapy with noradrenaline and dobutamine respectively, we instituted mechanical circulatory support via intra-aortic balloon counterpulsation. Yet, an ECG recorded about 1 h after PCI, showed persistent STE in leads II, aVF and III and new STE in leads V1 through V6 (Fig. 1B). What would you do next?

Discussion

Right ventricular myocardial infarction (RVMI) is common during acute inferior myocardial infarction (MI) and portends an increased risk of mortality and morbidity thereby warranting early and accurate diagnosis [1]. In clinical practice, RVMI is diagnosed with a high degree of accuracy (>80%) by the presence of ≥0.1 mV STE in lead V4R [2]; yet, it may rarely yield precordial STE, mostly in leads V1-V3 [1,3,4]. The development and extend of such STE depends on the ratio of the magnitude of the coexisting electrical forces produced by the concomitant inferior, inferior-lateral MI and RVMI, degree of clockwise rotation of the heart in the horizontal plane and body geometry. Most RVMI cases presenting with precordial STE are associated with absent or minimal inferior or inferior-lateral injury current leaving the RV injury current unopposed, as may happen in spontaneous or iatrogenic occlusion of a RV branch, occlusion of a non- or co-dominant RCA or occlusion of a collateralized or bypassed dominant RCA [4–6]. Yet, in quite exceptional cases, such a distinct ECG presentation of RVMI may occur in the setting of a dominant inferior injury current and is facilitated by RV dilation [4,7]. Acute RV dilation and reduced systolic performance due to ischemia may result in reduced systemic cardiac output and cardiogenic shock because of impairment of left ventricular diastolic filling caused by decreased RV output, elevated intrapericardial pressure and decreased left ventricular compliance secondary to a leftward septal shift which, in addition, causes impairment of left ventricular systolic performance due to distortion of left ventricular cavity geometry [1,8].

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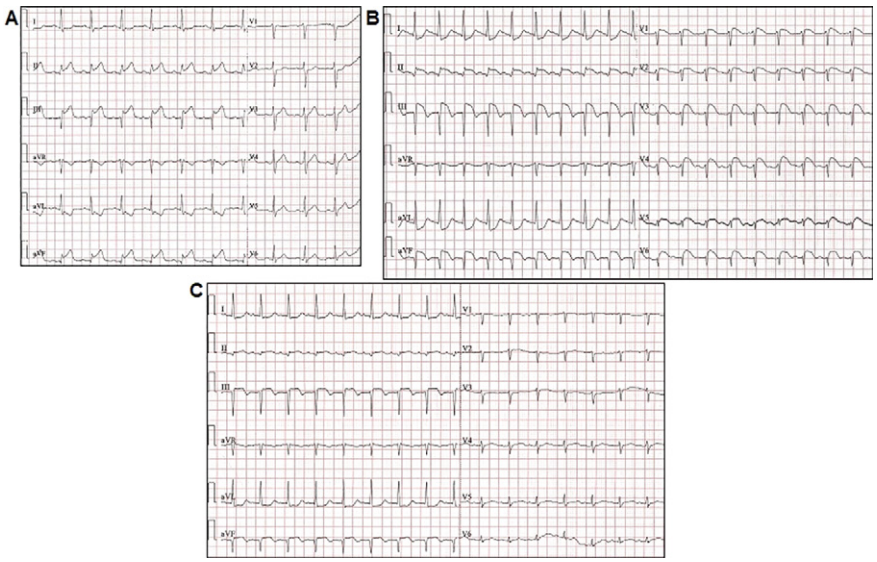

Fig. 1. Twelve-lead electrocardiograms (12-lead ECG). (A) 12-lead ECG on admission showing ST-segment elevation in leads II, aVF and III and reciprocal ST-segment depression in leads aVL and I. (B) 12-lead ECG after percutaneous coronary intervention (PCI) to the right coronary artery showing persistent ST-segment elevation in leads II, aVF and III and new ST-segment elevation in leads V1 through V6; note that leads V5 and V6 have been reversed. (C) 12-lead ECG recorded 24 h after PCI showing complete ST-segment elevation resolution and absence of evolution of Q waves but decreased R wave amplitude in the precordial leads.

Accordingly, knowledge of this distinct ECG presentation of RVMI helps avoid the erroneous diagnosis of anterior or anterior-septal MI and recognize a patient with RV dilation who might be more susceptible to hemodynamic deterioration.

Herein, a standard 12-lead ECG obtained on admission (Fig. 1A) showed STE of greater magnitude in lead III than II and ST-segment depression in lead I indicating a mean spatial ST-segment vector directed inferiorly and about 110° to the right in the frontal plane which suggested the RCA as the culprit artery. Furthermore, occlusion of the proximal RCA and hence concomitant RVMI was suggested by the presence of an isoelectric ST-segment in lead V1 and subtle ST-segment depression in lead V2, suggesting that the inferior-lateral injury current was strong enough to neutralize the RV injury current [9]. Coronary angiography confirmed occlusion of the RCA proximal to an acute marginal branch (Fig. 2A) resulting in inferior MI with concomitant MI of the inferoposterior and part of the lateral RV free wall which was

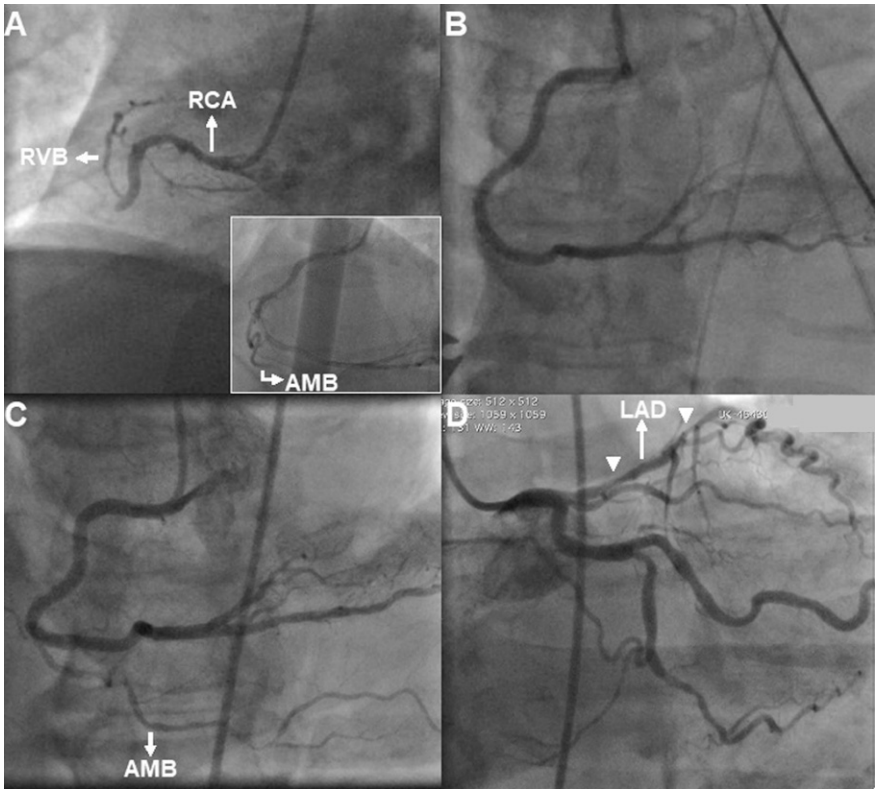

Fig. 2. Coronary artery angiograms. (A) Occluded right coronary artery (RCA) in the mid segment just after the origin of a right ventricular branch (RVB) and before the origin of an acute marginal branch (AMB; embedded panel). (B) Recanalized RCA with occlusion of the stent-jailed RVB and AMB. Planned coronary angiography showing a spontaneously recanalized stent-jailed AMB (C) and a patent left anterior descending (LAD) artery (D) harboring high-grade stenoses (arrowheads).

correctly diagnosed by the demonstration of STE in the additional right-sided leads (V3R through V6R) as proven in autopsy studies [10]. Yet, a post-PCI ECG (Fig. 1B) showed a dome-shaped STE in leads V1-V6 with maximal STE in lead V3 indicating a mean spatial ST vector directed about 40°-50° anteriorly. Although the dome-shaped STE in leads V1-V6 favored a RVMI, the pattern of STE favored an anterior MI where almost exclusively leads V2–V4 show the greatest STE and STE in lead V3 is greater than in V1 [4,6]. In case of RVMI, the highest STE is usually observed in lead V1 or V2 and STE in lead V1 or V2 is greater than or equal to STE in lead V3 with absence of progression or decreased elevation towards more left-sided leads [4–6]. However, post-PCI ECG also showed persistent STE in leads II, aVF and III which was ascribed to embolic occlusion of a PLVB and impaired microvascular reperfusion; the mean spatial ST vector was about +120° to the right in the frontal plane producing ST-segment depression in lead I. Accordingly, the rightward (> + 90°) and anterior mean spatial ST vector was directed towards epicardial injury in the RV [3–6]. If precordial STE was produced by proximal occlusion of the LAD artery due to spontaneous thrombosis and/or spasm which could have been induced by noradrenaline and/or dobutamine, the mean spatial ST vector would have also been directed anteriorly, commonly at 10° to 70°, but to the left at −30° to −90° in the frontal plane producing STE in lead I [3–6].

With the pre- and post-PCI angiographic findings and the above-mentioned ECG considerations in mind, we proceeded to an echocardiogram which showed hypokinesia involving the inferior, inferior-lateral and inferior-septal left ventricular walls, good contraction of the LAD artery-dependent myocardium and a dilated and hypokinetic RV with reduced global systolic function. As RV dilation develops, the RV moves more to the left and causes clockwise rotation of the heart in the horizontal plane; therefore, we confirmed that precordial STE was not a manifestation of anterior MI caused by proximal LAD artery occlusion but a manifestation of RVMI, since the ischemic and dilated RV had a large amount of its free wall directed anteriorly thereby enabling part of its injury current to escape neutralization by the dominant inferior injury current. Therefore, we refrained from performing additional emergency left-sided coronary angiography. Precordial STE resolved completely within 24 h and subsequent ECGs showed absence of evolution of Q waves but decreased R wave amplitude (Fig. 1C). Although, it was initially reported that precordial STE in RVMI does not evolve into decreased R wave amplitude or Q waves, subsequently reported cases of inferior MI with concomitant RVMI producing precordial STE revealed evolution of a QS pattern and/or loss of R waves in leads V1 through V4 or V5; such a pseudoanteroseptal ECG pattern has been ascribed to involvement of the anterior RV free wall [4]. Our patient had an uneventful but extended hospital course. He was gradually weaned off mechanical and pharmacological circulatory support and was extubated without any neurologic sequelae. Pre-discharge coronary angiography showed spontaneous recanalization of the occluded stent-jailed acute marginal branch (Fig. 2B and C) and enabled PCI to the patent but significantly obstructed LAD artery (Fig. 2D).

Conclusion

We have presented a patient with inferior wall STE MI in whom concomitant RVMI resulted in acute RV dilation by virtue of which the RV injury current became manifested by STE in leads V1–V6. Whereas STE pattern recognition was misleading, ECG analysis using vector concepts allowed us to recognize the RV origin of this ECG picture. The ability of the ECG to “capture” the RV structural change, i.e. dilation that enabled the manifestation of this ECG picture is thus highlighted. Awareness of this ECG pattern and the circumstances under which it develops enables correct diagnosis and management by avoiding misinterpretation as a sign of anterior or antero-septal MI and by recognizing a patient with RV dilation who might be more susceptible to hemodynamic deterioration.

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