Case Report

Prominent ST-segment elevation in leads V1–V4 due to isolated right ventricular branch occlusion after primary percutaneous coronary intervention for right coronary artery

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KEYWORDS
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Summary Isolated right ventricular myocardial infarction (RVMI) rarely occurs. It accounts for only 3% of all infarctions. In the literature, there are several reported isolated RVMI cases with precordial ST-segment elevation. We describe a 55-year-old man with prominent ST-segment elevation in leads V1–V4 in whom isolated RVMI developed due to occlusion of the right ventricular branch during primary percutaneous coronary intervention to the right coronary artery for acute inferior myocardial infarction.

Introduction

Isolated right ventricular myocardial infarction (RVMI) infrequently occurs. It accounts for only 3% of all infarctions [1]. Isolated RVMI and an observation of ST elevation in anterior derivations caused by isolated right ventricular branch occlusion is rarely seen.

In the literature, there are several reported isolated RVMI cases with precordial ST-segment elevation [2–7]. We describe a 55-year-old man with prominent ST-segment elevation in leads V1–V4 in whom isolated RVMI developed due to occlusion of the right ventricular branch during primary percutaneous coronary intervention to the right coronary artery for acute inferior myocardial infarction.

Case report

A 55-year-old man with the symptoms of squeezing chest pain and breathlessness lasting for 2 h was admitted to the Rize Training and Research Hospital (Rize, Turkey). He was a smoker and had hyperlipidemia history. His blood pressure was 140/90 mmHg and his heart rate was 78 beats/min, with a regular rhythm. Except for a grade 1/6 apical systolic murmur, his cardiac examination findings were normal and his
Figure 1  Admission electrocardiogram shows prominent ST-segment elevations in the inferior leads (II, III, aVF), and reciprocal ST-segment depressions in the lateral leads (I, aVL) and anterior leads (V1–V3).

Figure 2  Cardiac catheterization and intervention. The patient had 10% stenosis in the left anterior descending (LAD) artery, 50% stenosis in the first diagonal branch, 70% stenosis in the distal left circumflex artery, and 70% stenosis in the third obtuse margin (a). The right coronary artery had a lesion which included the right ventricular branch of the right coronary artery (RCA) (red arrowhead) and just after was totally occluded (b). After the lesion was passed by the floppy guide-wire, RCA flow was achieved and the culprit lesion seen (c). After the stent deployment into the proximal and mid-RCA, control pose disclosed that RCA had Thrombolysis In Myocardial Infarction-3 flow but occlusion of the right ventricular branch had developed (d).
Figure 3  Post-intervention electrocardiograms (ECGs). First post-intervention ECG revealed total ST-segment resolution in the inferior leads, but anterior leads had prominent ST-segment elevations and PR segment depressions in leads V1–V4 (a). Repeated ECGs showed permanent ST elevations (b and c) and 8-h ECG after the percutaneous coronary intervention revealed a total ST-segment resolution in precordial leads V1–V4 (d).

Figure 4  Follow-up and pre-discharge electrocardiogram (ECG). Follow-up (a) and pre-discharge ECG showed no Q waves in the anterior leads (b).
lung fields were clear. Twelve-lead electrocardiogram (ECG) revealed prominent ST-segment elevations in the inferior leads (II, III, aVF), and reciprocal ST-segment depressions in the lateral leads (I, aVL) and anterior leads (V1–V3) (Fig. 1).

Following pretreatment with 300 mg acetylsalicylic acid and 5000 IU unfractionated heparin, he was taken to the catheterization laboratory for primary percutaneous intervention. He had 10% stenosis in the left anterior descending (LAD) artery, 50% stenosis in the first diagonal branch, 70% stenosis in the distal left circumflex artery, and 70% stenosis in the third obtuse margin (Fig. 2a). The RCA had a lesion which included the RV branch of the RCA and just after was totally occluded (Fig. 2b). After the lesion was passed by 0.014 in. floppy guidewire, RCA flow was achieved and the culprit lesion was seen (Fig. 2c). A 50-μg bolus dose of nitroglycerine was administered and after nitrate was given we decided to perform percutaneous coronary intervention by direct stenting. We implanted a 3.0 mm × 28 mm stent into the proximal and mid-RCA. The patient’s chest pain was relieved. Control pose disclosed that RCA had Thrombolysis In Myocardial Infarction-3 flow but occlusion of the RV branch had developed after percutaneous coronary intervention. Because of the acceptable good angiographic results for RCA, we did not attempt any additional intervention for the RV branch (Fig. 2d). Soon after balloon angioplasty, the patient was taken into the coronary care unit. Twelve-lead ECG revealed total ST-segment resolution in the inferior leads, but anterior leads had prominent ST-segment elevations in leads V1–V4 (Fig. 3a).

The patient’s chest pain did not increase and his clinical status was stable. On bedside echocardiographic examination, the segmental wall motions of the left ventricle were found to be slightly hypokinetic in the inferior wall and in all other walls were found to be completely normal despite prominent ST-segment elevations in the precordial leads.

Repeated ECGs were recorded at 30, 60, and 120 min and confirmed permanent ST elevation (Fig. 3b and c) and 8-h ECG after the PCI revealed a total ST-segment resolution in precordial leads V1–V4 (Fig. 3d) and his chest pain was totally alleviated.

Follow-up ECGs showed no Q waves in the anterior leads (Fig. 4a). The patient was prescribed acetylsalicylic acid, clopidogrel, enoxaparin, metoprolol, ramipril, and atorvastatin. The patient was asymptomatic and the in-hospital course was uneventful, and he was discharged from the hospital 4 days later. There was no Q wave in precordial leads on his pre-discharge ECG (Fig. 4b). On repeated control echocardiography, the segmental wall motions of the left ventricle were found to be completely normal except for hypokinesia in the inferior wall.

On admission, the patient’s creatinine kinase level was 288 U/L with an MB fraction of 91 U/L. On follow-up, his peak creatinine kinase level rose to 740 U/L with an MB fraction of 91 U/L. On follow-up third day, creatinine kinase and MB fraction were 336 and 67 U/L, respectively.

However, there are several cases of RVMI presenting with precordial ST-segment elevations on ECG. RVMI is predominantly a complication of inferior myocardial infarction.

RV involvement can be diagnosed by the presence of ST segment elevation of 1 mm or more in lead V4R in the setting of acute inferior myocardial infarction manifested as ST-segment elevations in leads II, III, and aVF. In our case, the patient presented with inferior ST-segment elevations; after a successful primary stent implementation, precordial ST segments were prominently elevated and PR segments were slightly depressed, whereas the ST segments of the left leads returned to isoelectric line. In the control pose, his RCA was seen as patent, but the RV branch was found to be occluded by the implanted stent. Ackel et al. [7] reported ST-segment elevations in leads V1–V3 caused by occlusion of the RV branch of the RCA while performing primary PCI in a case of acute inferior myocardial infarction. However, they did not report any change on PR segments. In our case, slight PR segment depressions were seen in leads V1–V4 as well as ST-segment elevations in these leads. PR segment depression may indicate atrial injury. In our case, there was not any change in PR segments on baseline ECG and it occurred after stent implantation into the mid-portion of the RCA. Additionally, an interesting ECG finding of our case is that precordial ST-segment elevations in leads V3 and V4 were higher than in V1 and V2 in some repeated ECGs. This may be related to a well-developed RV branch but we cannot find an exact explanation of this ECG finding in the literature.

Our case demonstrates that prominent ST segment elevation in leads V1–V4 may be a sign of well-developed RV branch occlusion after successful PCI to a totally occluded RCA. Although the appearance of ST segment elevation in precordial leads V1–V3 is highly suggestive of acute anteroseptal left ventricular infarction secondary to LAD occlusion, it may also represent isolated RVMI due to an occluded RV branch of the RCA.

References