

Proximal complete occlusion of right coronary artery presenting with precordial ST-segment elevation

A case report

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Abstract

Background: It is well known that cardiologists empirically judge the culprit lesion of acute ST-segment elevation myocardial infarction (STEMI) according to the corresponding electrocardiographic leads. However, In addition to the obstruction of left anterior descending (LAD) coronary artery, rare cases with the occlusion of proximal right coronary artery (RCA) and/or isolated right ventricular (RV) branch showed the ST-segment elevation in precordial leads V1–V3 as well.

Case summary: We reported a patient complaining of acute chest pain and suffering ventricular fibrillation (VF) on admission. The electrocardiogram (ECG) showed mild ST-segment elevation in precordial leads V1–V3 and V4R. Bedside echocardiography displayed normal left ventricular ejection fraction and slight RV dilation. Proximal occlusion of nondominant RCA was confirmed by coronary angiography and urgent percutaneous coronary intervention (PCI) to RCA successfully resolved the chest pain and ST-segment elevation.

Conclusion: Undoubtedly, coronary angiography is usually the definite measurement for the diagnosis of culprit lesion. However, bedside echocardiography, ST-segment features in left and right precordial leads, and heart rate will be the additional information for judging ST-segment elevation in precordial leads V1–V3 resulting from occlusion of RCA or LAD.

Abbreviations: ECG = electrocardiogram, LAD = left anterior descending, PCI = percutaneous coronary intervention, RCA = right coronary artery, RV = right ventricular, STEMI = ST-segment elevation myocardial infarction, VF = ventricular fibrillation.

Keywords: case report, catheterization, electrocardiogram, myocardial infarction

1. Introduction

Electrocardiogram (ECG) is the most important noninvasive examination for cardiac ischemia. Complete occlusion of right coronary artery (RCA) usually displays the ST-segment elevation in inferior leads, and ST-segment elevation in precordial leads V1–V3 frequently means the anterior wall or anteroseptal infarction. However, the special electrocardiographic phenomena of proximal tract occlusion of RCA and/or isolated right ventricular (RV) branch occlusion presenting with ST-segment

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elevation in precordial leads V1–V3 were reported.^[1–4] We described a patient with mild ST-segment elevation in precordial leads V1–V3 who suffered from ventricular fibrillation (VF) on admission, but culprit lesion was total obstruction of proximal RCA proved by angiography results.

2. Case presentation

A 64-year-old male with current smoking and hypertension (amlodipine 5 mg once daily) was admitted to our hospital because of acute chest pain accompanied with dizziness, radiated pain of shoulder, and back for 4 hours. His initial blood pressure was 95/60 mm Hg and heart rate was about 60 beats per minute in our emergency department. The patient was diagnosed with chronic gastritis several years ago and irregularly took proton pump inhibitors. He denied surgical history, similar episodes in the past, and any recent chest trauma. On physical examination, there was no cardiac murmur, abnormal breath sounds, jugular vein engorgement, or lower extremities edema.

Admission ECG displayed accelerated junctional escape rhythm and mild ST-segment elevation in precordial leads V1–V3 (Fig. 1A), but without significant ST-segment change in leads II, III, and AVF. Just completion of 12-lead ECG examination, the patient suffered from VF. After immediate cardiopulmonary resuscitation and successful transthoracic defibrillation with biphasic waveforms at 200J, the patient recovered consciousness and his ECG turned into atrial fibrillation. The right precordial leads of V3R and V4R showed moderate ST-segment elevation, especially in right precordial

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Figure 1. (A) Admission ECG displaying mild ST-segment elevation in precordial leads V1–V3, but no significant ST-segment deviation in inferior leads II, III, and AVF. (B) Higher ST-segment elevation in precordial leads V1–V3 after electrical cardioversion. ECG = electrocardiogram.

lead V4R. Noticeably, the degree of ST-segment elevation in precordial leads V1–V3 was more significant after defibrillation than that on admission (Fig. 1B). According to the ECG features, a suspected anteroseptal wall myocardial infarction was firstly considered.

After taking 300 mg of clopidogrel and 300 mg of aspirin, the patient was immediately transferred to catheterization unit for emergency coronary angiography. A total of 5000 units unfractionated heparin were infused through the artery sheath following radial artery puncture. Coronary angiography showed dominant left coronary artery system displaying about 50% luminal stenosis in the middle of left anterior descending (LAD) and mild luminal stenosis at distal tract of left circumflex artery. Surprizingly, proximal total occlusion of nondominant RCA was found (Fig. 2). The patient was treated with thrombus aspiration following administration of tirofiban via intracoronary at a dose of 10 µg/kg. Primary percutaneous coronary intervention (PCI) of RCA was performed with a domestic polylactide polymer-based EXCEL stent (3.0×28) mm) (JW Medical, Weihai, China) at 12 atm. Successful PCI resulted in the resolution of the chest pain and ST-segment elevation.

During in-hospital period, ECG showed sinus rhythm and the patient did not suffer from cardiovascular events. Bedside echocardiography showed normal left ventricular ejection fraction, slight RV dilation, and no pericardial effusion after stent implantation. The present study was approved by the Ethics Committee of Zhejiang Provincial People's Hospital (Hangzhou, Zhejiang, China).

3. Discussion

The acute occlusion of proximal RCA usually showed STsegment elevation in inferior wall leads. However, the bewildering phenomenon of ST-segment elevation in left precordial V1–V3 caused by AMI due to RCA occlusion was observed in clinical practice. Kim et al^[1] reported that proximal occlusion of RCA accompanied with distal collateral circulation from left coronary artery displayed ST-segment elevation in precordial leads. Some cases displaying ST-segment elevation in precordial leads V1 through V3 due to complete occlusion of RV branch were documented.^[3,4] We reported a patient with V1–V3 STsegment elevation caused by proximal obstruction in nondominant RCA without collateral circulation, and he suffered from VF on admission. How can we deduce the real culprit lesion from the ST-segment elevation in left precordial leads?

There are several clues to judge the ST-segment elevation in precordial leads probably caused by occlusion of RCA and/or isolated RV branch. First, as for AMI patients with occlusion of RCA, ST-segment elevation in lead V1 is usually higher than that in precodial leads V2 and V3,^[5,6] and progression reduction in the ST-segment elevation and absence of Q-wave development in precordial leads as the diagnosis of RVMI have been mentioned in previous reports.^[7,8] Second, one of the electrocardiographic

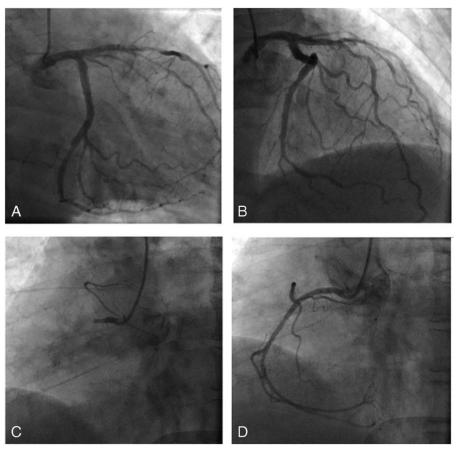


Figure 2. Angiographic pictures of emergency cardiac catheterization. (A) About 50% luminal stenosis in the mid-LAD artery. (B) Mild stenosis at distal tract of LCX. (C) Proximal complete occlusion of RCA. (D) Angiographic finding of RCA following successful PCI. LAD=left anterior descending, LCX=left circumflex artery, PCI=percutaneous coronary intervention, RCA=right coronary artery.

features for patients with RCA occlusion and predominant RV infarction commonly showed the convex or dome-like STsegment elevation in precordial leads,^[9] which was observed in this case. Third, ST-segment elevation of V3R-V4R is usually found in right coronary occlusion, especially in right precordial lead V4R.^[10] Fourth, a fast bedside echocardiography usually displayed the slight dilation with or without local abnormal wall motion of RV.^[11] RV dilation has been identified as a predisposing factor for cardiac clockwise rotation, which may be associated with ST-segment elevation in precordial leads in patients with acute occlusion of RCA. Fifth, coronary artery disease is the most common cause for sinus node dysfunction, and bradycardia is often accompanied with infarction of proximal RCA and/or RV branch.^[12] We noticed the similar phenomenon that admission ECG presented as accelerated junctional escape rhythm. We speculated about the above-mentioned bradycardia rising from intermittent dysfunction of sinus node due to reduced flow to sinus node artery in patients with proximal occlusion of RCA. At last, the nondominant RCA usually do not cause significant posterior infarction, which may reversely result in relative ST-segment elevation in precordial leads V1-V3.^[13]

Although the predictors for VF were quite complicated and uncertain, VF is related with higher in-hospital mortality in patients with ST-segment elevation myocardial infarction (STEMI).^[14,15] It was reported that patients with acute occlusion of LAD had a higher incidence of VF than those with RCA occlusion,^[16,17] which may partly be explained by the fact that the infarct related artery in STEMI was more prevalent in LAD compared to RCA.^[18] The nondominant RCA and no coronary collateral circulation increased the incidence of VF in patients with the occlusion of RCA.^[19,20] We speculated the higher ST-segment elevation in precordial leads V1–V3 of postdefibrillation ECG caused by electrical cardioversion. In fact, the phenomenon of electrical cardioversion leading to slight ST-segment elevation in precordial leads was reported in previous study, and they normalized within several minutes after the electrical shock.^[21] As for the transient atrial fibrillation with slow ventricular response after electrical cardioversion, which may be associated with the cardiac electrical instability and intermittent sinus node dysfunction.

4. Conclusion

It is necessary that emergency PCI to the culprit lesion according to the electrocardiographic guidance in patients with STEMI. However, ECG characteristics are not sufficiently specific in judging the culprit lesion of STEMI. Considering the fact that ECG displaying ST-segment elevation in precordial leads V1–V3 may secondary to proximal occlusion of RCA, we should question the diagnosis when the clinical symptoms are not accordant with the myocardial lesion concluded from ECG. Combined assessment of ST-segment elevation ratio of V1 to V3 in precordial leads, V4R features in right precordial lead, bedside echocardiograhpic examination and bradycardia may be useful for speculating the

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