Precordial ST-Segment Elevation Caused by Proximal Occlusion of a Non-Dominant Right Coronary Artery

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For patients with ST-segment elevation myocardial infarction, primary percutaneous coronary intervention to the culprit lesion via electrocardiographic guidance is essential. We herein report the rare case of a 49-year-old man who presented with ST-segment elevation in the precordial leads, while coronary angiography results indicated total occlusion of the proximal non-dominant right coronary artery. We evaluated its possible pathophysiologic mechanisms and thoroughly discussed isolated right ventricular infarction and its electrocardiography findings.

Key Words: Coronary angiography • Myocardial infarction • Total occlusions

INTRODUCTION

ST-segment elevation in precordial electrocardiogram leads is characteristic of anterior wall or anteroseptal wall infarction. However, it is rarely observed in patients with proximal right coronary occlusion. In the latter scenario, patients may either have ST-segment elevation in the anterior leads alone or in both the anterior and inferior leads. These are reported to be associated with proximal right coronary artery (RCA) occlusion and right ventricular (RV) infarction, and even with isolated RV branch total occlusion. We report a rare case of a patient with ST-elevation myocardial infarction (STEMI) who presented with ST-segment elevation in the precordial V1-V3 leads, but angiography results revealed total occlusion of the proximal RCA. We also discuss the pathophysiologic mechanism.

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CASE REPORT

A 49-year-old previously healthy male smoker was referred to our hospital complaining of chest pains for 2 hours, along with cold sweats and breathlessness. Upon physical examination, the patient showed no hypotension, tachypnea, desaturation, jugular vein engorgement, cardiac murmur, abnormal breath sounds, or peripheral edema. The result of electrocardiography (ECG) earlier performed in another hospital showed hyperacute T waves in leads V1-4 (Figure 1A). The ECG performed in our emergency department then demonstrated an acute anterior wall STEMI with a convex ST-segment elevation over leads V1-3 (Figure 1B). We decided to perform a primary intervention, and administered a 600 mg loading dose of clopidogrel (prasugrel and ticagrelor are not available in Taiwan) and 300 mg of aspirin after 4000 units of intravenous heparin. Coronary angiography results demonstrated left side dominant coronary arteries. Left circumflex artery (LCX) gave rise to left posterior descending artery to

supply to posterior, lateral, wall and inferior walls. Total occlusion of the proximal RCA with massive thrombus was observed (Figure 2A). The left coronary artery (LCA) was remarkable for a mid-50% segmental stenosis in the left anterior descending artery (LAD). There was mild atherosclerosis of the LCX. Primary percutaneous transluminal coronary angioplasty of the occluded RCA was performed with repeated manual thrombus aspiration and subsequently a bare metal stent (3.0 × 18 mm) was deployed. Intracoronary tirofiban 25 μg/kg was also prescribed during the procedure. Restoration of thrombolysis in myocardial infarction (TIMI)-3 flow to the target vessel and grade 2 myocardial blush were obtained, with an acceptable angiographic result (Figure 2C). A follow-up electrocardiogram showed complete resolution of the ST-segment elevation over the V1-V3 leads (Figure 1C). The patient's peak troponin I level was 75.06 ng/mL, while peak creatinine phosphokinase (CPK) level with the creatinine kinase muscle-brain fraction (CK-MB) were 1793 IU/L and 208.1 ng/mL (11.6%). The results of echocardiography performed on the same day, after primary percutaneous coronary intervention (PCI), showed an ejection fraction of 53%, an RV fractional area change of 37%, and a tricuspid annular plane systolic excursion of 17.7 mm, without obvious regional wall motion abnormality [wall motion score index (WMSI) = 1].[1] A diagnosis of acute RV MI was made, and the patient was discharged uneventfully on the fourth hospital day.

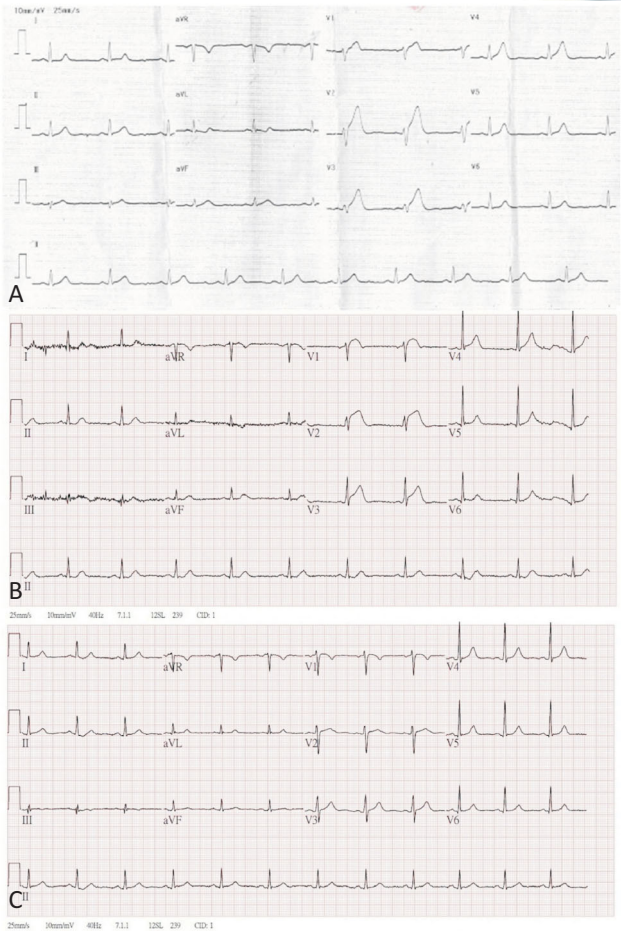

Figure 1. *(A) Electrocardiography (ECG) performed in another hospital showed hyperacute T wave in leads V1-4. (B) ECG obtained at our emergency department showed convex ST-segment elevation over leads V1-3. (C) Follow-up ECG after primary percutaneous coronary intervention showed complete resolution of ST-segment elevation over leads V1-V3.*

DISCUSSION

Typically, the ECG results in RCA occlusion show ST-segment elevation in leads II, III, and aVF. Sometimes, the test results may present concomitant ST elevation in

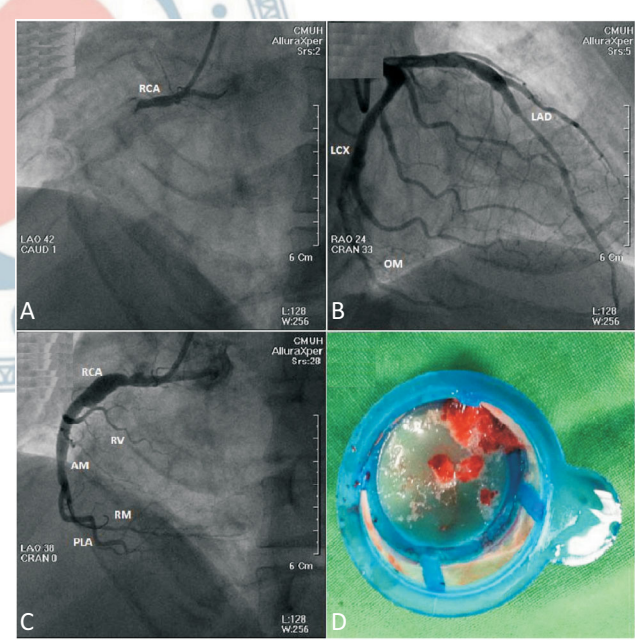

Figure 2. *(A and B) Coronary angiography showed total occlusion of the proximal right coronary artery (RCA) and mid-50% stenosis of the left anterior descending artery. There was mild atherosclerosis of the left circumflex artery. (C) After percutaneous coronary intervention, coronary angiography showed restoration of the RCA with thrombolysis in myocardial infarction (TIMI)-3 flow and grade 2 myocardial blush. (D) Massive thrombus was obtained by repeated manual thrombus aspiration of the RCA.*

the precordial and inferior leads, but rarely in the precordial leads alone. According to previous literature, simultaneous involvement of both the anterior and inferior leads is attributed to occlusion of the proximal RCA,[2] while isolated ST elevation in the precordial leads has been reported to result from isolated RV branch occlusion,[3] proximal RCA occlusion with good collateral flow to the left coronary arteries,[4-6] or proximal RCA occlusion with predominant damage originating from the RV wall rather than the inferior wall.[7] Similarly, a diseased dominant RCA with extreme cardiac counterclockwise rotation was also discussed. In addition to the above electrocardiographic patterns, Nanavati et al. described a totally occluded RCA that presented with a normal electrocardiogram due to the existence of a subendocardial microvascular network.[8] This means that in cases of RCA occlusion, the ECG results may present as ST elevation of the precordial leads or with normal ST segments in the inferior leads instead of the typical II, III, and aVF ST elevation. Our patient presented with isolated ST-segment elevation in V1-3, and coronary angiography results showed proximal RCA total occlusion without collateral arteries from the left coronary arteries. This scenario showed some differencies from previous case reports.

After percutaneous coronary intervention, the RCA was relatively large in the proximal part but short in total length. It only gave off RV marginal branches and small PDA. Because of less RCA territory, the inferior wall was dominantly supplied by the LCA. We attributed V1-V3 ST-segment elevation to RV branch infarction. The typical inferior lead ST-segment elevation may not have been significantly present for this non-dominant RCA. Furthermore, the electrical effect may have been neutralized by the reciprocal change of precordial ST-segment elevation.

Isolated RV infarction is rare, and accounts for less than 3% of all patients with myocardial infarction.[9] A review of prior reports shows that it can occur in any of the following situations: acute loss of RV branch during coronary angioplasty of the RCA,[10,11] occlusion of a nondominant RCA,[12] or acute occlusion of the proximal RCA, with a patent protecting collaterals from other vessels.[4-6] RVMI usually occurs with simultaneous inferior wall infarction. The dominant electric forces generated by the ischemia of the inferior wall suppress the changes caused by the ischemia of the RV. On the other hand, in patients with non-dominant RCA, infarcted RV predominated electric forces and presented ST segment elevation in precordial leads. To the best of our knowledge, this is the first reported case of "anterior ST segment elevation" caused by proximal occlusion of "non-dominant" RCA.

Certain ECG features have been suggested to differentiate causes of precordial ST-segment elevation as either isolated RVMI or LAD territory infarction. The absence of Q-wave development in the anterior leads and progressive reduction in ST-segment elevation across the precordial leads have been reported as favoring the diagnosis of RVMI.[13] Lopez-Sendon et al.[14] described ST-segment elevation in V4R higher than V1-V3 indicated RVMI. Although these ECG features were helpful, they were not sufficiently specific for our purposes. It was impossible to make this distinction on the basis of ECG alone.

CONCLUSIONS

In summary, anterior ST-segment elevation for proximal occlusion of the RCA has been rarely reported, and many underlying mechanisms have been proposed. Early recognition of this scenario and subsequent initiation of the appropriate management may change the outcome of the disease.

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