CASE REPORT

The ECG Pattern of Isolated Right Ventricular Infarction during Percutaneous Coronary Intervention

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Isolated right ventricular infarction (RVI) is a rare event. The electrocardiographic (ECG) pattern of RVI, ST-elevation in lead V_4R and in anterior chest leads V_{1-3} is similar to that of a proximal occlusion of a small, nondominant right coronary artery (RCA). The ECG changes may be misinterpreted as signs of infarction of the anterior wall. This paper describes a case of isolated temporary occlusion of the major side branches of the RCA during percutaneous coronary intervention, recognized by angiography findings and typical ECG changes. This case demonstrates how one might avoid wrong decisions even in the catheterization laboratory by putting attention to the anatomical interpretation of the ECG.

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The right coronary artery (RCA) supplies most of the right ventricular (RV) free wall. The lateral RV free wall is subtended by the RV branches and the inferoposterior RV free wall by the right acute marginal (RAM) branches and partly by the posterior descending branch. Blood supply to the anterior RV free wall is provided by the conus branch of the RCA and the right ventricular branches of the left anterior descending (LAD) coronary artery.[1–4]

It is important to recognize the electrocardiographic (ECG) pattern of RV infarction. Although acute myocardial infarction (MI) involving only the right ventricle is a rare event, it is common (24–52 %) in acute inferior wall MI resulting in higher mortality.[5–7]

CASE REPORT

A 55-year-old previously healthy carpenter was referred to our institution with acute coronary syndrome. The worst pain episode had occurred 1 day before hospital arrival. He was free of chest pain when admitted. Physical examination was normal. The ECG showed inverted T waves in leads II, III, and aVF. Lead V_4R and the precordial leads were within normal limits. Echocardiography showed normal ejection fraction, mild mitral valve regurgitation, and normal diameters of the left and the right ventricles. Maximal troponin T (TnT) was 1.24 μg/L (normal value < 0.03 μg/L). Aspirin, low molecular weight heparin, glycoprotein IIb/IIIa inhibitor, clopidogrel, and betablocker medication was initiated immediately. Coronary angiography performed 36 hours after admission showed a 60% stenosis in the distal part of the LAD coronary artery and a 99% stenosis in the midpart of the small left circumflex coronary artery. There was a 90% stenosis in the midpart of the dominant RCA with 2 major RV side branches. The tight lesion of the RCA was treated by percutaneous coronary intervention (PCI). Continuous 12-lead ECG was recorded at 25 mm/s speed during predilatation and stenting. 15-Lead ECG was recorded at 50 mm/s speed immediately after balloon dilatation and stenting. During the predilatation the balloon was placed distal to the first RV branch and

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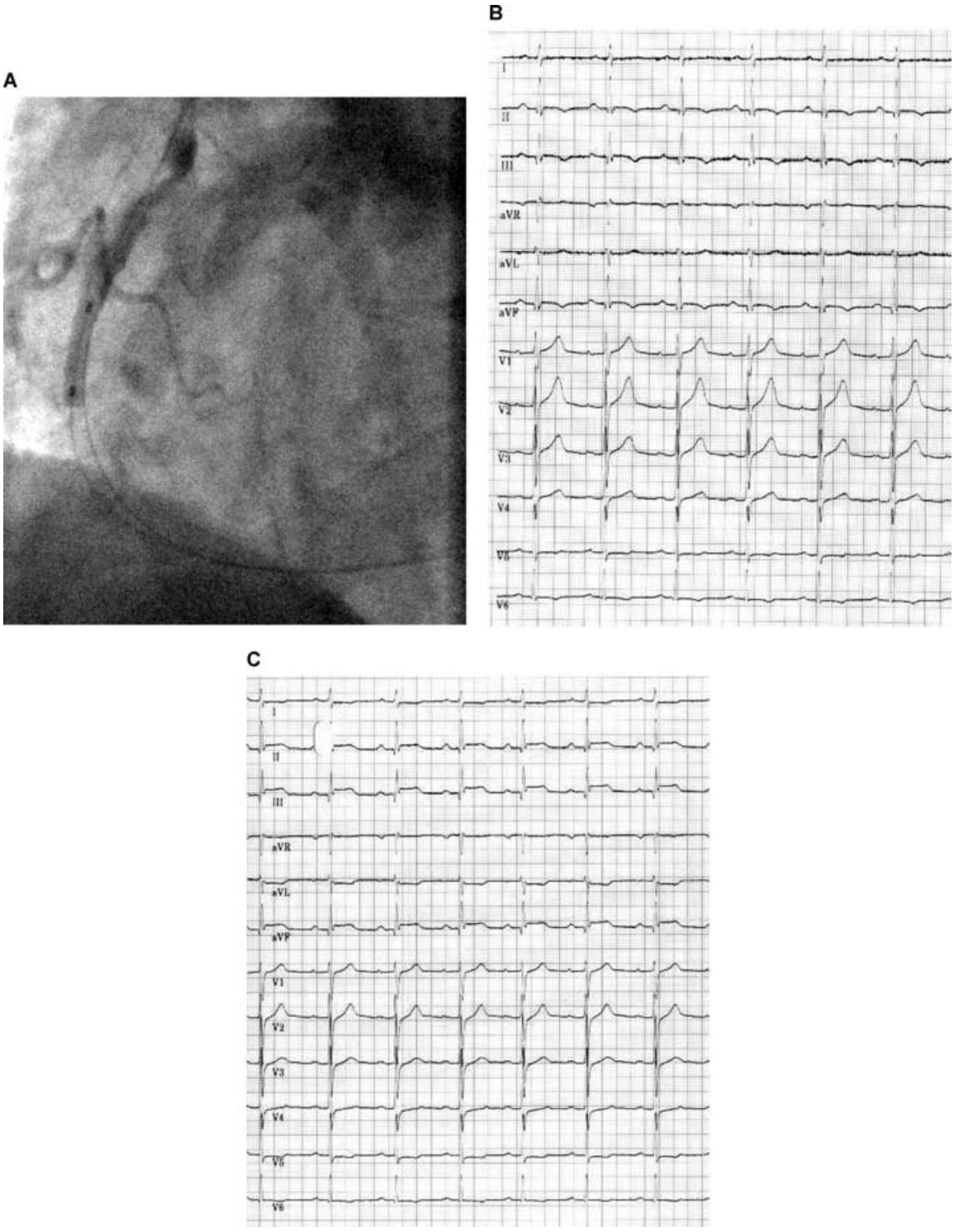

Figure 1. (A) Coronary angiography shows the position of the balloon distal to the right ventricular side branch. The right acute marginal branch is covered by the balloon. (B) The ECG recorded (25 mm/s) before predilatation reveals inverted T waves in leads II, III, aVF, and V_6. (C) The ECG is recorded (25 mm/s) at the end of the predilatation. The balloon was inflated for 1 minute and 28 seconds. The ECG reveals ST segment elevation and pseudonormalization of the inverted T waves in leads II, III, and aVF. Note the ST segment depression and T wave inversion in lead aVL representing reciprocal changes to the ST elevation in LIII.

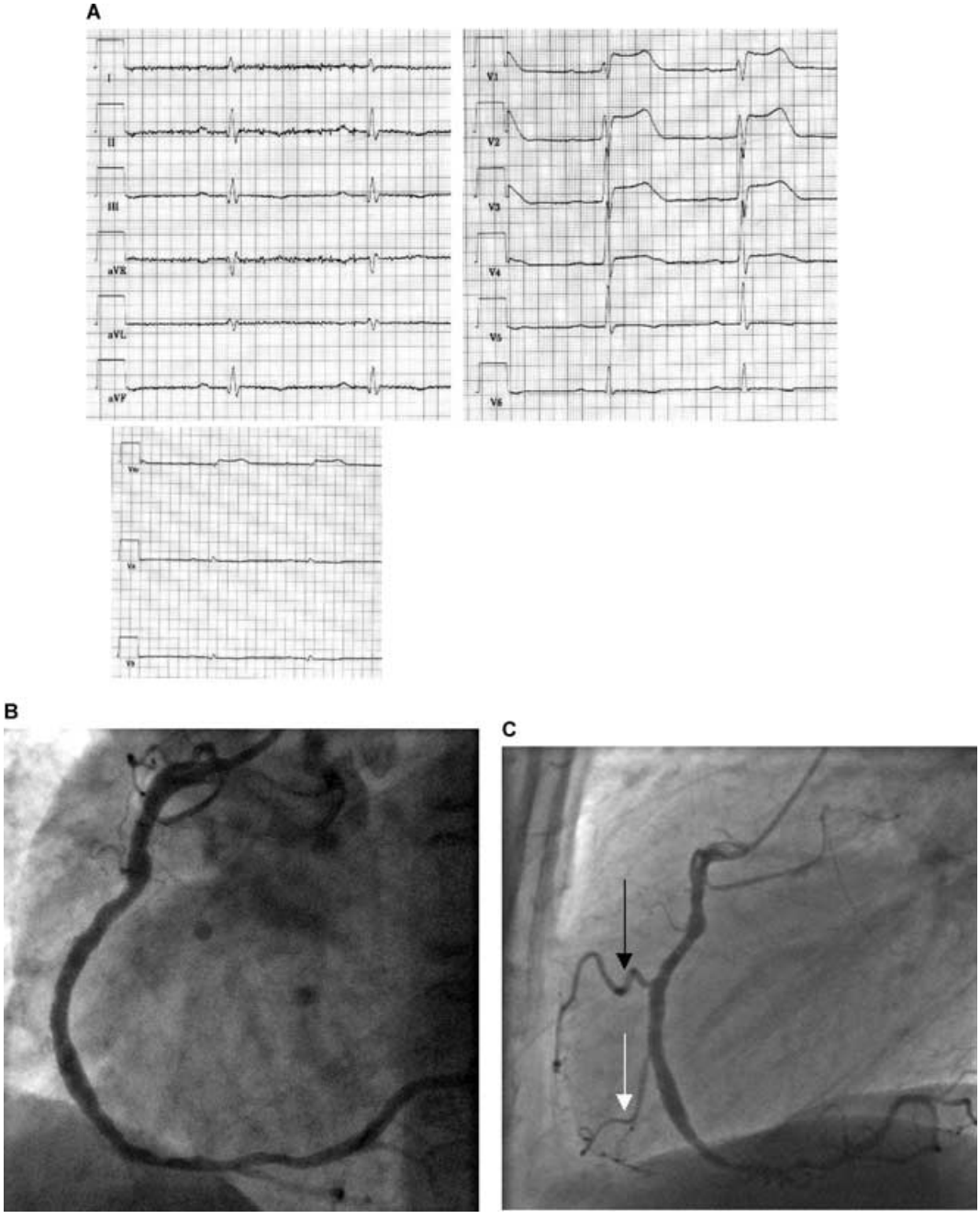

Figure 2. (A) The 15-lead ECG recorded (50 mm/s) during chest pain immediately after stenting of the right coronary artery reveals the ECG pattern of an isolated right ventricular infarction: the ST segment is elevated in leads V_4R, V_1, V_2, V_3, and V_4. The sum of the ST segment elevations in leads V_{1-2} is greater than that in leads V_{2-3}. The ST segments and T waves in leads II, III, and aVF are unchanged. The absence of ST elevation in aVL and of ST depression in leads II, aVF, and III immediately excludes an occlusion in a proximal part of the left anterior descending coronary artery. (B) The angiography shows an open right coronary artery with normal distal flow, but absence of flow in the right ventricular branches. (C) Flow in both major right ventricular side branches improved spontaneously. The right ventricular branch (black arrow) supplies the lateral right ventricular free wall and the inferoposterior right ventricular free wall is subtended by the right acute marginal branch (white arrow).

proximal to the RAM branch (Fig. 1A). Continuous 12-lead ECG recording showed progressive ST-segment elevation and pseudonormalization of the T waves in leads II, III, and aVF, whereas the precordial leads were invariable (Figs. 1B,C). The lesion was stented. To cover the whole diseased segment the stent was positioned proximal to the RV and distal to the RAM branch covering both side branches. Immediately after stenting the patient sensed chest pain. 15-Lead ECG surprisingly showed ST-elevations in precordial leads V_{1-4}. There was also ST-elevation in lead V_4R (Fig. 2A). The ST-segments in LII, LIII, and aVF were isoelectric and the T waves inverted. The ECG pattern was quite different from the one recorded during the first balloon inflation. The angiography revealed the cause of the ECG findings: the RV and RAM branches were occluded, but the right main coronary artery and the stented area were open with normal coronary flow (Fig. 2B). During the next minutes the chest pain abated and both side branches regained flow spontaneously (Fig. 2C). The next ECG showed 100% ST-segment resolution in lead V_4R and 50% ST-segment resolution in the precordial leads. In an ECG recorded 90 minutes later the ST-segment was normal also in the precordial leads. The TnT value 24 hours post PCI was only slightly higher than the initial value (1.35 μg/L vs 1.24 μg/L) and no new Q waves could be seen. The patient was discharged home on the 6th day in clinically stable condition.

DISCUSSION

Recording of lead V_4R during chest pain in patients with acute inferior wall MI distinguishes patients with occlusion of the RCA proximal to the first RV branch from those with a distal RCA occlusion with 90% sensitivity and 91% specificity.[8,9] There have been some reports of angiographically demonstrated isolated RV branch occlusion with concomitant ST-segment elevation in the left precordial leads.[10–12] The recording of RV electrical potentials in the left precordial leads depends on the degree of clockwise rotation of the heart in the horizontal plane and on body geometry.[13] Left precordial ECG manifestations of RV injury are absent in most patients because they are suppressed by dominant forces of the accompanying LV inferior wall injury in cases with proximal occlusion of the RCA.[14] This opponent injury current is absent in isolated occlusion of the RV and/or RAM branches. Sometimes a proximal occlusion of a small, nondominant RCA produces a similar ECG pattern as in isolated RV infarction, ST-elevation in leads V_4R and $V_{1-3,}$ because of absence of opponent posterior injury current.[15] Those ECG changes may be misinterpreted as signs of infarction of the anterior wall. This emphasizes the importance of recording right-sided chest leads in patients with acute myocardial ischemia. In LAD occlusion, the maximal ST segment elevation is almost exclusively in leads V_{2-4} and ST elevation is higher in lead V_3 than in V_1.[16]

We present a patient with isolated temporary occlusion of the major side branches of the RCA, which supply most of the RV free wall. This ECG pattern contains ST elevation in leads V_4R and V_{1-3} without ST elevation in inferior leads II, III, and aVF. It is important to take into account the anatomical information in the ECG to make right decisions about the culprit lesion in the catheterization laboratory.[15] A patient with the ECG pattern described in this article might be mistakenly regarded as suffering from anterior MI caused by an occlusion of the LAD. Accordingly, a marginally significant lesion in the LAD could be treated by PCI if considered as the culprit lesion. Also unnecessary additional left-sided coronary angiography because of chest pain and precordial ST elevations during a procedure could be the consequence. Even with the information gained from angiography, ECG might give additional information aiding in clinical decision making.

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