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Case Report Open Access

Rescue Thrombolysis Followed by Salvage Percutaneous Coronary Intervention for the Treatment of Inferior ST Elevation Myocardial Infarction Combined with Cardiogenic Shock in a Patient with an Anomalous Right Coronary Artery Origin

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Abstract

We report a case of rescue thrombolysis followed by salvage percutaneous coronary intervention (PCI) for the treatment of inferior ST elevation myocardial infarction (STEMI) combined with cardiogenic shock in a patient with an anomalous right coronary artery (RCA) origin. When it is not feasible to perform primary PCI due to the anomalous origin of the RCA, rescue thrombolysis can be an optional treatment strategy that may allow sufficient time to perform facilitated PCI in a STEMI patient with hemodynamic instability.

Keywords: Thrombolytic therapy; Percutaneous coronary intervention; Coronary vessel anomalies; Myocardial infarction; Cardiogenic shock

Introduction

Performing primary percutaneous coronary intervention (PCI) in a ST elevation myocardial infarction (STEMI) patient with hemodynamic instability may be challenging since conventional diagnostic and guiding catheters are generally designed to approach coronary arteries with normal origins [1-3]. In this case, thrombolytic therapy can be an alternative revascularization strategy for the treatment of patient with STEMI to gain time. However, some of the STEMI patients who receive thrombolytic therapy fail to achieve patency of the infarct-related coronary artery [4]. Under these circumstances, performing salvage PCI to restore coronary blood flow soon after the thrombolysis fails seems to be necessary [5]. Herein, we report a case of rescue thrombolysis followed by salvage PCI for the treatment of inferior STEMI combined cardiogenic shock in a patient with anomalous right coronary artery (RCA) origin. When it is not feasible to perform primary PCI due to the anomalous origin of the RCA, rescue thrombolysis can be an optional treatment strategy that may allow sufficient time to perform facilitated PCI in a STEMI patient with hemodynamic instability.

Case Report

A 39-year-old male patient was admitted to our emergency department with sudden onset of severe chest pain for 30 minutes. He had no relevant past medical history. Physical examination revealed a heart rate of 45 beats/min and blood pressure of 78/45 mmHg, which indicated cardiogenic shock. Auscultation showed clear lung sounds, bradycardia and no murmur or gallop. Initial 12-lead electrocardiography revealed ST segment elevation in the inferior leads with complete atrioventricular block (CAVB) suggestive of inferior wall STEMI (Figure 1a). Intravenous volume infusion followed by aspirin, clopidogrel loading and intravenous heparinization was performed immediately. Fortunately, the heart rate as well as the blood pressure increased to the normal range (blood pressure 114/76 mmHg and heart rate 81 beats/min). Follow-up electrocardiography revealed resolution of the CAVB but the ST segment elevation still remained in the inferior leads (Figure 1b). So, he was transferred to the catheterization laboratory for primary PCI. Because of the hemodynamic instability, the right common femoral vein was punctured and a transvenous temporary pacemaker was prepared in case of CAVB development. Then, the right common femoral artery was punctured to perform invasive coronary angiography. The left coronary artery was approached by a 5F Judkins left 4 diagnostic catheters and it revealed no significant luminal narrowing at the left anterior descending coronary artery and the left circumflex coronary artery. An attempt was made to engage the RCA with a 5F Judkins right 4 diagnostic catheter but this failed. We could not find the RCA ostium. So, we immediately performed an aortogram with a 5F pigtail catheter and found that the RCA was originated from the left sinus of Valsalva- below the sinotubular plane between the midline and origin of the left coronary artery. We tried to engage the RCA with a 6F Amplatzer 1 guide catheter but it was not feasible. At that time, the patient suffered more severe chest pain, and his blood pressure got lower. Intravenous fluids were administered followed by an intravenous dobutamine infusion. We knew that we did not have much time. We could have re-tried RCA engagement using an Amplatzer catheter, but we could not be sure of having success with the RCA engagement right away. So, we decided to perform rescue thrombolysis in order to gain time. Thrombolytic therapy was administered with Alteplase, the recombinant tissue plasminogen activator (t-PA) (total dose 100 mg; bolus of 15 mg IV, followed by 50 mg for 30 minutes and 35 mg for the next 60 minutes). ST segment resolution was noted during the t-PA administration. The patient's chest symptom resolved as well and the blood pressure rose slowly. Accelerated idioventricular rhythm was present for about 10 minutes during the t-PA infusion. After rescue thrombolysis; we planned to perform adjunctive PCI (after 3 hours, but

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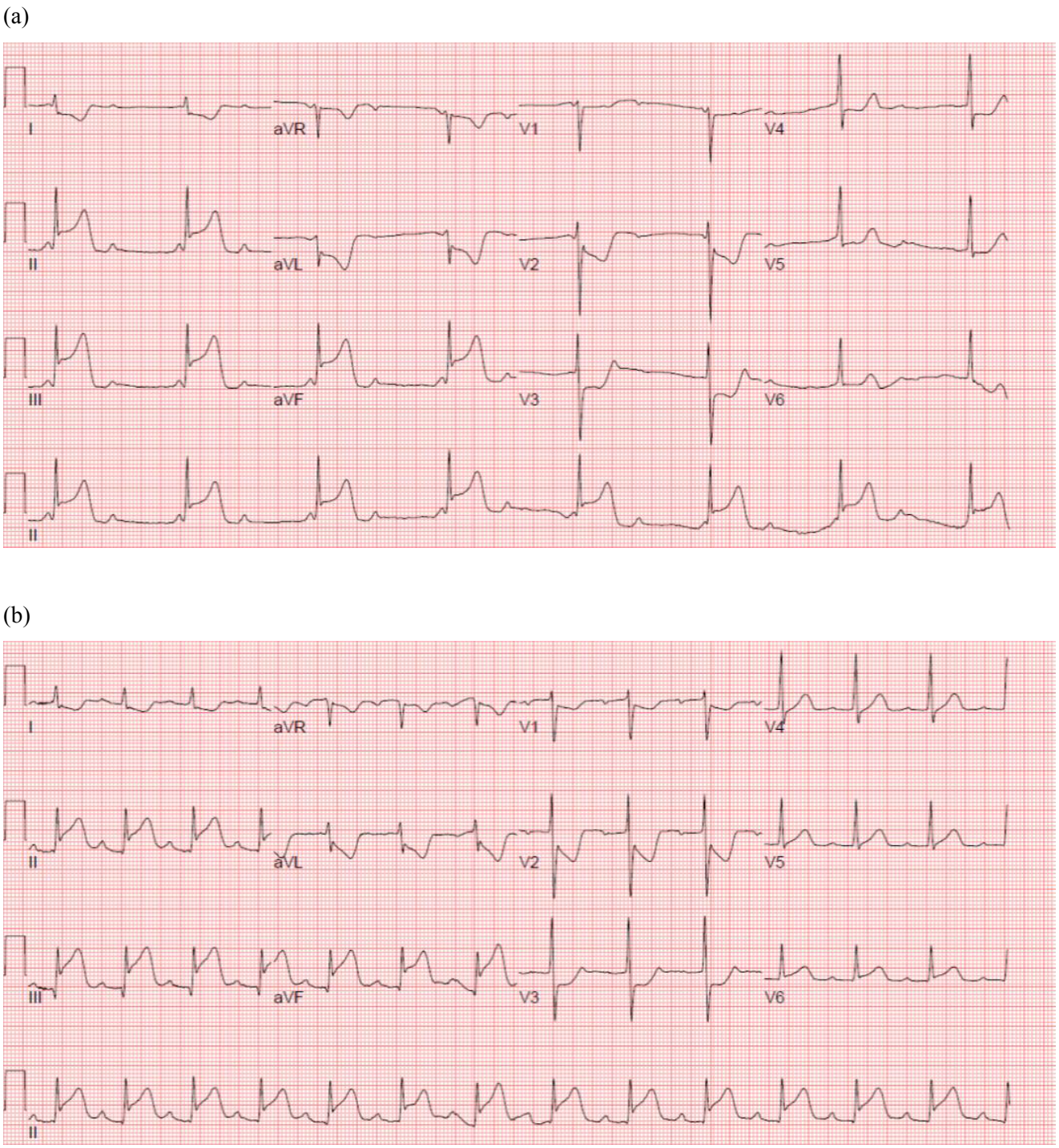
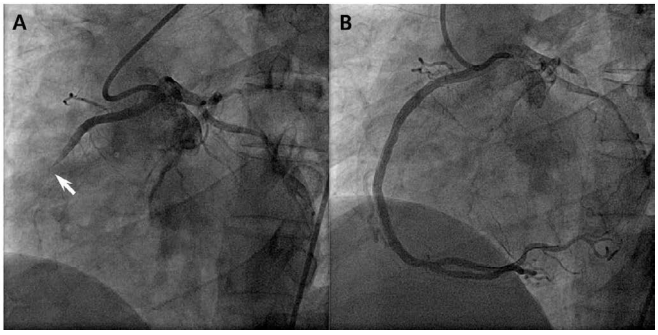


Figure 1: Initial electrocardiography of the patient revealed (a) complete atrioventricular block (CAVB) with marked ST segment elevation in the inferior leads and (b) resolution of the CAVB but ST segment elevation still remained in the inferior leads after intravenous fluid infusion followed by aspirin, clopidogrel loading and intravenous heparinization.

within 24 hours after the thrombolysis). However, about 10 minutes after the rescue thrombolysis, the patient suffered from chest pain again, and ST segment elevation was noted on electrocardiographic monitoring. So, we decided to perform facilitated PCI, since the RCA was thought to be re-occluded. We tried to engage the RCA with a 6F Amplatzer 1 guide catheter, and it engaged successfully. An RCA angiogram revealed total occlusion of the mid-RCA with Thrombolysis in Myocardial Infarction (TIMI) grade 0 flow (Figure 2a). We introduced a 0.014 inch J-curved Fielder wire and passed the lesion. Predilatation was performed with an Ikazuchi 2.5×15 mm balloon at the mid-RCA. Two Resolute Integrity stents 3.0×26 mm and 3.0×18 mm were implanted sequentially at the mid-RCA. The final RCA angiogram showed revascularization of the infarct-related RCA with TIMI grade 3 flow (Figure 2b). The patient's chest symptom as well as



Arrow indicates totally occluded lesion of the anomalous RCA.

Figure 2: Angiographic images of the anomalous right coronary artery (RCA) (a) before and (b) after facilitated PCI (left anterior oblique view).

the ST segment elevation on electrocardiographic monitoring resolved completely. The patient's blood pressure and heart rate were stable and he was sent to the intensive care unit for further management. Initial laboratory analysis revealed that the creatine kinase (CK) was 114 U/L (normal values: 24-170 U/L), CK-MB, 1.4 mcg/mL (normal values: 0-6.6 mcg/mL), troponin I, 0.017 ng/mL (normal values: <0.028 ng/mL), and brain natriuretic peptide, 10.0 pg/mL (normal values: 0-100 pg/mL). Transthoracic echocardiography was performed 6 hours after the facilitated PCI. Transthoracic echocardiography revealed reduced left ventricular (LV) systolic function with an ejection fraction of 47%. Regional wall motion abnormalities were also noted, showing akinesis of the inferior wall from the basal segment to the apical segment, the posterolateral wall from the mid-LV segment to the apical segment, and severe hypokinesis of the inferoseptal wall from the basal segment to the apical segment. During admission, the peak levels of cardiac biomarkers were CK >2000 U/L (normal values: 24-170 U/L); CK-MB 87.0 mcg/mL (normal values: 0-6.6 mcg/mL), and troponin I>50.0 ng/mL (normal values: <0.028 ng/mL). Fasting glucose level was 129 mg/dL and post-prandial glucose level was 265 mg/dL. In addition, the glycated hemoglobin level was 7.5%. So, under the diagnosis of new onset type 2 diabetes, we started anti-diabetic medication with biguanides. Lipid profiles consisting of total cholesterol, triglycerides, high density lipoprotein cholesterol, and low density lipoprotein cholesterol were 217, 151, 45, and 137 mg/dL, respectively. Therefore, we also started lipid lowering therapy with atorvastatin 40 mg. The patient was stable during the rest of his admission, and afterwards, he was discharged with medications.

Discussion

Along with primary PCI, thrombolytic therapy is an alternative revascularization strategy for the treatment of patients with STEMI. However, 30% to 50% of STEMI patients fail to achieve patency of the infarct-related coronary artery at 90 min after the onset of thrombolytic therapy [4]. Under these circumstances, facilitated PCI may play a crucial role inrestoring the blood flow of the re-occluded infarct-related coronary artery. A prior study demonstrated that salvage PCI performed early after failed thrombolysis seems to be as effective and safe as primary PCI [5]. Moreover, another compelling study revealed the virtue of salvage PCI as a treatment strategy since it is beneficial when compared to repeat thrombolysis as well as conservative therapy, in terms of prognosis [6]. Therefore, performing salvage PCI to restore coronary blood flow soon afterthe thrombolysis fails seems to be necessary. When performing salvages PCI, bleeding complications are the main concern. However, earlier studies revealed that thebleeding complication rate in patients undergoing salvage PCI was comparable to those undergoing primary PCI or delayed PCI after thrombolysis [6,7].

Although coronary arteries of anomalous origin are uncommon, with an incidence of 0.2-1.2% of patients who undergo PCI, they may be challenging since conventional diagnostic and guiding catheters are generally designed to approach coronary arteries with normal origins [1-3]. An anomalous origin of the RCA from the left sinus of Valsalva has been reported in 6-27% of patients with coronary anomalies [8]. Intriguingly, a previous study suggested that the presence of this anomaly may be associated with an increased predisposition to developing coronary atherosclerotic disease [9].

We report a case of rescue thrombolysis followed by salvage PCI for the treatment of inferior STEMI combined with cardiogenic shock in a patient with an anomalous RCA origin. Time does not matter for performing an 'elective PCI' for treating the patient with ananomalous RCA origin since the patient is hemodynamically stable during the procedure. However, time matters for performing a 'primary PCI' for treating a patient with an anomalous RCA origin presenting with STEMI and cardiogenic shock, due to the hemodynamic instability. Our case illustrates that rescue thrombolysis can be an optional treatment strategy that may allow sufficient time for the performance of facilitated PCI, when it is not feasible to perform primary PCI due to an anomalous origin of a coronary artery.

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