Primary Angioplasty with AngioJet Thrombectomy in a Patient with a Single Coronary Ostium and Acute ST Elevation Myocardial Infarction: Technical Considerations

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INTRODUCTION

A solitary coronary ostium in the aorta, without other major congenital cardiovascular anomalies, is very rare, and can be clinically and technically challenging when myocardial infarction occurs. This report documents a 51-year-old man with an anomaly of three coronary arteries arising from a solitary coronary ostium in the right sinus of Valsalva who underwent primary percutaneous coronary intervention for acute inferior wall ST segment elevation myocardial infarction. Our case demonstrate that when coronary arterial ostia are difficult to identify, an aortogram should be performed as early as possible to delineate the locations of coronary arterial ostia. A 2.0-cm left Amplatz guiding catheter and extrasupport wire were used to obtain optimal support. AngioJet thrombectomy and cutting balloon angioplasty were then successfully performed in this case.

Key Words: Myocardial infarction • Coronary angioplasty • Single coronary artery • AngioJet thrombectomy

CASE REPORT

A 51-year-old male smoker with no family history of coronary artery disease was brought to our emergency room after one hour of sudden onset, substernal chest pain. He denied any previous history of angina. The chest pain occurred just after dinner. He was alert on arrival. Blood pressure was 94/66 mmHg and heart beat was regular at 90 bpm, without conduction delays. An electrocardiogram (ECG) revealed ST segment elevation in leads II, III, and aVF. There was no ST segment elevation at V3R or V4R. Ventricular tachycardia (VT) caused this patient to lose consciousness, but he was successfully resuscitated by cardioversion (200 Joules). At the same time, the patient was endotracheally intubated and mechanically ventilated. Biochemistry re-
vealed glucose levels of 118 mg/dL, creatinine of 1.2 mg/dL, Na\(^+\) of 142 mEq/L, K\(^+\) of 3.3 mEq/L, CPK of 125 U/L, CK-MB of 13 U/L and Troponin-I of 0.096 ng/mL. Echocardiography demonstrated severe hypokinesis of the inferior, posterior and lateral left ventricular (LV) walls, with an estimated left ventricle ejection fraction of 38%. The subject was transferred for emergent cardiac catheterization.

The left and right common femoral arteries were simultaneously accessed for intervention and intra-aortic balloon counterpulsation (IABP), respectively. Initially, a 6 Fr Judkins left-4 cm (JL4) catheter failed to engage the left coronary artery. Right coronary artery (RCA) angiography by a 7 Fr Judkins right-4 cm (JR4) catheter was also unsuccessful; aortography was then performed. In true anteroposterior and left lateral projections, aortograms revealed a single main coronary trunk originating from an abnormal RSV. A left anterior descending (LAD) coronary artery arose from the main trunk, turned left and then passed along the anterior wall of the left ventricle (LV). A circumflex (LCX) coronary artery, originating from the main trunk, turned toward the right at its start and then rotated clockwise to the left and passed along the lateral wall of the LV. It also demonstrated intracoronary thrombus, with total occlusion in the proximal right coronary artery (RCA), originating from the main trunk.

A 7 Fr Amplatz left 2-cm (AL2) catheter was inserted into the RSV. It was intended to face the main trunk orifice, but failed to enter the ostium because of the trunk’s abnormal location. Selective coronary engagement of the main trunk was achieved through this catheter by inserting a 0.014\(^\prime\)/0.178\(^\prime\), 180-cm tracker wire to the LCX. The entire occlusion lesion at the proximal RCA was bridged by a 0.014\(^\prime\)/0.300-cm tracker wire. The RCA vessel size was about 4.0 mm, with a thrombus-containing lesion in the proximal segment (Figures 1-A).

At first, AngioJet thrombectomy was performed twice for the entire occlusion lesion to prevent distal embolization. A critical and heavily calcified lesion was displayed at the proximal RCA after thrombectomy (Figures 1-B). We chose cutting balloon angioplasty to dilate this calcified critical lesion. A 4.0\(^\prime\)/0.10-mm cutting balloon was inflated to 6 atm for 40 seconds after pre-dilation by 1.5\(^\prime\)/0.20-mm and 2.0\(^\prime\)/0.20-mm Ryujin balloons. Finally, the residual proximal RCA lesion was of less than 20% stenosis, with TIMI-3 anterograde flow and grade 3 myocardial blush (Figures 1-C). There were three VT attacks during intervention, but were all successfully resuscitated by cardioversion (100 Joules). Door-to-balloon time was 2 hours and 49 minutes; the time from symptom onset to reperfusion was 3 hours and 52 minutes.

Peak CPK and CK-MB values were 5550 U/L and 478 U/L, respectively, at twelve hours after symptom onset. Subsequent electrocardiography revealed pathological Q-waves in leads II, III, and aVF. The patient’s post-infarction course was stable. He was successfully

![Figure 1](image1.png)

**Figure 1.** Coronary angiograms taken before percutaneous coronary intervention (PCI) reveals a single main trunk originating from abnormal right sinus of Valsalva (RSV); RCA, LAD and LCX are all arising from the main trunk. A left anterior oblique (LAO) projection (60 degrees) with cranial angulation of 20 degrees (A) shows total occlusion in the proximal RCA portion (arrow). Coronary angiograms taken after AngioJet thrombectomy reveals diffuse calcified lesions (arrow) in the proximal RCA (B). Coronary angiograms taken after PCI with AngioJet thrombectomy and cutting balloon angioplasty reveals the residual proximal RCA lesion (arrow) with less than 20% stenosis (C).
weaned off the IABP and ventilator on his third hospital day. Coronary computerized tomography (CT) and arteriogram revealed a single coronary artery originating from the RSV, and separated into three coronary arteries. The LAD arising from the main trunk passed between the main pulmonary trunk and the aorta; the LCX originated from the main trunk on a retro-aortic course (Figure 2-A). A smaller and abnormal RCS was also revealed by CT in an unusual location (Figure 2-B), more anterior and leftward-turned than normal. The subject was discharged on his eighth day in the hospital. Five months later, angiography revealed mild coronary atherosclerosis only.

DISCUSSION

Anomalous coronary arteries of aortic origin are rare but potentially fatal, with incidence reported at 0.3-0.8% in angiographic intervention patients. Anomalies can be in the vessel’s origin, course or terminus. A left coronary artery originating from the pulmonary trunk is the most common congenital coronary ostial anomaly; a single coronary ostium in the right sinus of Valsalva (RCS) is the rarest.

Our patient had a single coronary artery arising from the RCS. This rare anomaly occurred less than 0.02% by coronary angiograms survey. Young patients (< 20 years) with a single coronary artery frequently have associated abnormalities such as great vessel transposition or coronary arterial fistulae. Older patients, such as our subject, have a low incidence of associated abnormalities. According to methods proposed by Lipton et al., our case would be classified as Type R-III, where the LAD and LCX arise separately from the proximal RCA and the LAD passes between the aorta and the pulmonary artery while the LCX passes behind the aorta.

Several case reports have discussed balloon angioplasty for anomalous coronary arteries. Although these cases were usually challenging, success was achieved through proper attention to anatomical details such as orifice configuration, exit angulation from the aorta, anomalous arterial route and atherosclerotic lesion site. A key factor in balloon angioplasty for anomalous arteries is guiding catheter support. The Amplatz catheter curve is optimal for this coronary anomaly, where the solitary coronary artery is initially superior and leftward-oriented to the usual RCA ostium in the RCS. A 2.0-cm left Amplatz guiding catheter provided optimal support in this case and allowed unencumbered passage of the instrument. A stiffer coronary guidewire also helped guide the catheter into a coaxial position for the anomalous artery.

The presence of thrombi in the coronary arteries raises considerably the risks of distal embolization, “no-reflow” or other embolic complications during PCI. AngioJet thrombectomy had a higher success rate and lower complication rate than balloon angioplasty for the treat-
ment of thrombus-containing lesions. Prior studies have demonstrated that AngioJet thrombectomy is safe and effective in patients with AMI. The AngioJet is currently indicated in patients with moderate to large thrombus-containing native vessels or SVGs, prior to definitive therapy by balloon PCI and stent. The AngioJet should not be used in small (< 2 mm) vessels because of perforation risks. However, a recent meta-analysis of randomized trials has demonstrated that, among AMI patients treated with PCI, the use of adjunctive mechanical devices to prevent distal embolization is associated with better myocardial perfusion rates and less distal embolization but no benefit for 30-day mortality. In one recently published study evaluating the feasibility of AngioJet thrombectomy in AMI patients, the AngioJet catheter was positioned across the target lesion and into the distal coronary artery before thrombectomy activation. This may have paradoxically raised the probability of distal embolization.

The “controlled” injury by cutting balloon angioplasty supposedly reduces the occurrence of severe dissections and may result in a reduction of restenosis. In this case, a tight and heavily calcified lesion was demonstrated after thrombectomy. We used cutting balloon angioplasty, and follow-up angiography revealed only mild atherosclerosis five months after the procedure.

Door-to-balloon time (169 minutes) was longer than usual and primarily due to a long door-to-door time (116 minutes). A lot of time was spent on informing this patient’s family members and on resuscitation. The time from aortogram to AngioJet thrombectomy was only 23 minutes. To speed up reperfusion, aortograms should be performed as early as possible when coronary ostia are hard to engage with conventional devices and anomalous coronary arteries are strongly suspected.

REFERENCES