Spontaneous Recanalization of the Obstructed Right Coronary Artery Caused by Blunt Chest Trauma
A Case Report

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Summary
Blunt chest trauma can cause a wide variety of injuries including acute myocardial infarction (AMI). Although AMI due to coronary artery dissection caused by blunt chest trauma is very rare, it is associated with high morbidity and mortality. In the vast majority of patients with AMI, primary percutaneous coronary interventions (PCI) are performed to recanalize obstructed arteries, but PCI carries a substantial risk of hemorrhagic complications in the acute phase of trauma. We report a case of AMI due to right coronary artery (RCA) dissection caused by blunt chest trauma. The totally obstructed RCA was spontaneously recanalized with medical therapy. We could avoid primary PCI in the acute phase of blunt chest trauma because electrocardiogram showed early reperfusion signs. We performed an elective PCI in the subacute phase when the risk of bleeding subsided. Since the risk of severe hemorrhagic complications is greater in the acute phase of blunt chest trauma as compared with the late phase, deferring emergency PCI is reasonable if signs of recanalization are observed.

Key words: Coronary artery dissection, Percutaneous coronary intervention

Blunt chest trauma can cause a wide variety of injury including bone fractures, vascular injuries, and cardiac injuries. Of these injuries, acute myocardial infarction (AMI) due to coronary artery dissection caused by blunt chest trauma is a rare but life-threatening complication with high morbidity and mortality. In most reported cases of AMI caused by blunt chest trauma, primary percutaneous coronary interventions (PCI) were performed to recanalize obstructed arteries. However, since multiple injuries were not uncommon in traffic injury, the patients have a substantial risk of life-threatening complications including intracranial hemorrhage, which may hamper early coronary invasive intervention.

We report a case of AMI due to right coronary artery (RCA) dissection caused by blunt chest trauma. The totally obstructed RCA was spontaneously recanalized with medical therapy. We successfully avoided primary PCI in the acute phase of blunt chest trauma and performed elective PCI in the subacute phase when the hemorrhagic risk decreased.

Case Report
A 51-year-old female was brought to a community hospital due to blunt chest trauma caused by a collision between a bicycle and a pedestrian. The patient was a bicycle rider. Her past medical history was not significant except for dyslipidemia that was found at a previous health examination. She did not have other coronary risk factors, and her total cholesterol levels at admission were normal (176 mg/dL). Although she was hemodynamically stable, her electrocardiogram (ECG) showed ST-segment elevation in leads V1-2 and AV-junctional rhythm (Figure 1). She was transferred to our hospital with a tentative diagnosis of acute ST-elevation myocardial infarction (STEMI). On admission to our hospital, however, she became hypotensive with a systolic blood pressure of 60 mmHg and bradycardic with a rate of 44 beats/minute, subsequently complicated with a ventricular fibrillation requiring defibrillation. Transthoracic echocardiography revealed preserved left ventricular wall motion and reduced right ventricular wall motion. Her ECG showed ST-segment resolution and terminal T wave inversion in leads V1-2 and AV-junctional rhythm (Figure 1). She was transferred to our hospital with a tentative diagnosis of acute ST-elevation myocardial infarction (STEMI). On admission to our hospital, however, she became hypotensive with a systolic blood pressure of 60 mmHg and bradycardic with a rate of 44 beats/minute, subsequently complicated with a ventricular fibrillation requiring defibrillation. Transthoracic echocardiography revealed preserved left ventricular wall motion and reduced right ventricular wall motion. Her ECG showed ST-segment resolution and terminal T wave inversion in leads V1-2 and AV-junctional rhythm (Figure 2). Coronary computed tomography angiography (CCTA) showed the fractured sternum and an occlusion of the proximal segment of RCA (Figure 3A). We considered that this patient had acute right ventricular infarction with minor left ventricular myocardial damage. We initiated intravenous heparin...
and dobutamine, and prescribed oral aspirin 100 mg per day.

On the second day of admission (12 hours after the hospital arrival), we performed coronary angiography, which showed an obstruction of the proximal RCA (Figure 4A) and a Rentrop grade 2 collateral from the left coronary artery (LCA) to the RCA (Figure 4B). Since she was asymptomatic and her vital signs were stable, we did not perform PCI to the obstructed RCA. Peak CK and CK-MB were 1436 mg/dL and 115 mg/dL, respectively.

Figure 1. Initial twelve-lead electrocardiogram. AV-junctional rhythm. ST-segment elevations in leads V1-2 were observed.

Figure 2. Twelve-lead electrocardiogram after ventricular fibrillation. AV-junctional rhythm. ST-segment resolution and terminal T wave inversion in lead V1-2 were observed.
At day 7, intravenous heparin was discontinued when hemothorax was noted. At day 10, we performed cardiac MRI for the detection of late gadolinium enhancement (LGE) and ischemia provoked by adenosine (Figure 5). While there was no LGE, there was perfusion defect during adenosine stress, suggesting inferior wall ischemia. Follow-up CCTA suggested improvement in the RCA flow at day 11 (Figure 3B). On the following day, a follow-up echocardiography showed no segmental asyn-ergy in left ventricular wall motion, and the ejection fraction measured by the modified Simpson method was 76%. We decided to perform PCI on the RCA. A coronary angiography at day 16 showed the recanalized RCA with a focal dissection, confirmed by intravascular ultrasound.

Figure 3. Findings of coronary computed tomography angiography (CCTA). A: CCTA at day 1. The CCTA showed the fractured sternum (arrow head) and an occlusion of the proximal segment of RCA (arrow). B: CCTA at day 11. The CCTA showed the recanalized RCA.

Figure 4. Coronary angiogram at day 2. A total obstruction of the proximal RCA (A) and a Rentrop grade 2 collateral circulation from the left coronary artery to the RCA (B) was shown. Collateral flows (dotted circle) were weakly visible from the left anterior descending artery (LAD) septal branch to the distal segment of ativoventricular branches (arrows) and the distal segment of right ventricular branch (arrow heads).
Figure 5. Cardiac MRI performed at day 10. A: Two chamber view. There was no late gadolinium enhancement (LGE). B: Short axis view. There was no LGE. C: Short axis view. Perfusion MRI during adenosine stress. There was perfusion defect in the inferior wall (arrows). D: Short axis view. Perfusion MRI at rest. There was no defect.

Figure 6. Findings of intravascular ultrasound (IVUS) and optical frequency domain imaging (OFDI). IVUS showed an intimal flap (arrow in A), and OFDI also showed an intimal flap (arrow in B).

(IVUS) (Figure 6A) and optical frequency domain imaging (OFDI) (Figure 6B). A 3.5 × 18 mm Biolimus-eluting stent was implanted to cover the dissection (Figure 7A and B). Her clinical course was uneventful after the successful PCI. At day 18, we performed cardiac MRI for follow-up. There was no perfusion defect during adenosine stress. At day 19, she ambulated home at discharge from hospital. Follow-up angiographies at 1 month and 6
months after the discharge from the hospital did not show any restenosis. A follow-up echocardiography at 6 months showed normal left ventricular wall motion (ejection fraction 69%). Her plasma brain natriuretic peptide (BNP) level was normal (15.1 pg/mL).

Discussion

We reported a case of the obstructed RCA caused by blunt chest trauma. The obstructed RCA was spontaneously recanalized only after the initiation of aspirin and intravenous heparin. Since PCI requires substantial anticoagulation (activated coagulation time > 250 seconds) as well as dual antiplatelet therapy, primary PCI during the acute phase of blunt chest trauma has a potential risk of life-threatening hemorrhagic complications. Our case suggests primary PCI to the obstructed coronary artery can be deferred under certain conditions during the acute phase of blunt chest trauma.

The most important factor for deferring primary PCI was a sign of recanalization. Her electrocardiogram after ventricular fibrillation showed ST-segment resolution and T wave inversion, which are known for signs of early reperfusion. Moreover, her vital signs were stabilized after starting administration of intravenous dobutamine. Although an early recanalization was not confirmed by the emergent CCTA, ST-segment elevation was no longer observed in her ECG (Figure 2). Therefore, those signs of recanalization may be more important when making a decision whether or not to perform primary PCI, compared with CCTA findings of coronary obstruction.

In the current patient, the presence of fair collateral flow (Rentrop Grade 2) from the left coronary artery might affect ST resolution and T wave inversion. In the atherosclerotic coronary stenosis, the growth of collateral flow is associated with moderate to high intensity exercise. On the other hand, the mechanism of the collateral flow growth in the non-atherosclerotic coronary stenosis/occlusion was not well understood. Since the patient did not have coronary risk factors, there was low possibility that a sufficient collateral flow had existed before the blunt chest trauma. Because post mortem angiographic studies showed that the collateral circulation was observed in all human subjects irrespective of coronary artery stenosis, the rapid growth of the collateral flow might happen after the sudden occlusion of the right coronary artery. Although previous studies reported that the natural growth of collateral artery required several weeks (at least 2 weeks), Kyriakidis, et al. showed that mechanical balloon occlusion to the recipient artery rapidly increased the collateral flow from the donor artery. Furthermore, recent advance of PCI to chronic total occlusions revealed the presence of occult good collateral that was only visible by super selective injection by microcatheter. While an exact mechanism of the rapid growth of the collateral flow in this patient was unknown, the process of non-atherosclerotic occlusion might be associated with the rapid growth of the collateral flow.

In conclusion, acute coronary artery occlusion caused by blunt chest trauma was successfully treated with an early non-invasive strategy. Since a risk of severe hemorrhagic complications is greater in the acute phase of blunt chest trauma as compared with the late phase, deferring emergency PCI can be a reasonable option if signs of recanalization are observed.

Disclosures

Conflicts of interest: Dr. Sakakura has received speaking honoraria from Abbott Vascular, Boston Scientific, Medtronic Cardiovascular, Terumo, OrbusNeich, Daiichi-Sankyo, Sanofi, and NIPRO; has served as a proctor for Rotablator for Boston Scientific; and has served as a consultant for Abbott Vascular and Boston Scientific. Prof. Fujita served as a consultant for Mehergen Group Holdings, Inc.
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