Coronary Malperfusion due to Flap Suffocation after Acute Type A Dissection Surgery

Susumu Isoda, MD, Motohiko Osako, MD, Tamizo Kimura, MD, Yuji Mashiko, MD, Nozomu Yamanaka, PhD, Shingo Nakamura, PhD, and Tadaaki Maehara, MD

A 24-year-old man presented with chest pain. He was diagnosed as having a type A acute aortic dissection and an annulo-aortic aneurysm. After emergency surgery for an aortic root replacement, his electrocardiogram showed ST-segment depression and T-wave inversion. Echocardiography showed asyn-ergy of the left ventricle without coronary ostial pathology. Heart catheterization revealed no coronary stenosis, but the true lumen of the residual ascending aorta had extreme diastolic narrowing due to flap suffocation. This resulted in coronary malperfusion. The pullback pressure curve confirmed the mechanism. The patient underwent a surgical re-intervention for a total arch repair, which diminished the coronary malperfusion. At a follow-up appointment four years and four months later, the patient was doing well.

Keywords: acute aortic dissection, coronary malperfusion

Introduction

An acute type A aortic dissection sometimes causes coronary artery malperfusion. Occlusion of the true lumen of the coronary artery is the typical cause of acute coronary malperfusion. However, diastolic occlusion of the true lumen of the ascending aorta can also cause hypoperfusion of the aortic root and coronary arteries. Here, we present a case of acute coronary malperfusion due to flap suffocation at the distal anastomosis of an aortic root replacement, which occurred after surgery for an acute type A dissection.

Case

A 23-year-old man had a sudden onset of acute chest pain and was transferred to an outlying hospital. He underwent computed tomography and was diagnosed as having a type A acute aortic dissection and annulo-aortic ectasia. He was transferred to our institution seven hours after the onset of the symptom. His height was 189 cm and body weight was 65 kg. Blood pressure was 132/46 mmHg, and pulse rate was 107 beats per min. Auscultation of the chest at the third intercostal space of the left sternal border revealed a to-and-fro murmur with Levine grade 3/6. Preoperative laboratory data showed no remarkable changes.

An electrocardiogram showed a regular sinus rate of 91 beats per minute, an axis of 97 degrees, and no left ventricular high-voltage or ST segment abnormality. Echocardiography indicated adequate wall motion with a left ventricular diastolic diameter of 53.4 mm and a systolic diameter of 37.5 mm. Moderate aortic valve regurgitation was noted. The aortic valve annulus was 25 mm. Pericardial effusion was not observed. The chest X-ray showed cardiomegaly with a left fourth arch protrusion and costothoracic ratio of 55.5%.
Computed tomography revealed dilatation of the ascending aorta with a diameter of 6 cm. The Valsalva sinus had a diameter of 65 mm. An intimal tear was apparent at the ascending aorta. The dissecting space of the aorta was thrombosed from the arch to the descending aorta, and the dissection extended from the aortic root to the level of the diaphragm. An emergent operation was performed seven hours after the patient’s arrival.

The chest was opened through a median sternotomy, and the pericardium was entered. The pericardial effusion was mildly bloody. The dilated, ascending aorta was pear-shaped, and the aortic root was bluish. Cardiopulmonary bypass with hypothermia at 25°C was initiated; blood was sent to the right axillary artery and right femoral artery, and then drained from the right atrium. The distal ascending aorta appeared healthy, and its caliber, approximately 24 mm, was not enlarged. We decided to avoid an open distal anastomosis. The distal ascending aorta was cross clamped in a non-dilated zone with an atraumatic Fogarty clamp. Retrograde blood cardioplegia was administered.

On opening the aorta, we found a longitudinal intimal tear of the ascending aorta. The dissection ended proximally at the ostium of each coronary artery. Antegrade cardioplegia was selectively administered.

Coronary buttons were formed. The dissection of coronary artery was repaired using gelatin-resorcin-formalin (GRF) glue. Using a 28-mm tubular graft (Hemashield; Maquet Japan K.K., Tokyo, Japan) and a 25-mm CarboMedics mechanical valve (Japan Lifeline Co, Tokyo, Japan), the composite graft was anastomosed to the aortic valve annulus with 2-0 braided polyester sutures (Johnson & Johnson K.K., Tokyo, Japan).

Both coronary buttons were directly sutured to the holes of the composite graft that was formed by a heat cutter and reinforced with Teflon felt. The proximal end of the aortic cross clamp, which was placed on the distal ascending aorta, was the distal anastomotic site. Where the half circle of the aorta was dissected at the distal stump, the intima and adventitia were adhered with GRF glue. The aortic wall was sandwiched by inner and outer Teflon felt sheets. A distal aortic anastomosis was formed using 4-0 polypropylene sutures. The distal ascending aorta was smaller than the graft at the anastomosis site. The cross clamp was removed after controlled reperfusion. The patient was weaned from cardiopulmonary bypass without incident. His chest was closed in the usual fashion.

The patient’s postoperative course was uneventful, initially. He was extubated on postoperative day (POD) 1.

On POD 2, ionotropic support was increased and the patient’s condition deteriorated because of congestive heart failure. His blood pressure was 86/61 mmHg and pulmonary arterial pressure was 41/21 mmHg. The cardiac index was 2.1 L/min/m² and SvO₂ was 51%. Electrocardiography showed ST segment deceleration and T-wave inversion at II, III, aVF, V₄, V₅, and V₆. Echocardiography revealed akinesia at the anteroseptal wall and severe hypokinesis of the left ventricular lateral wall. Ostial stenosis or occlusion of left or right coronary artery was not noted.

On POD 3, the patient underwent a heart catheterization study. It revealed no stenosis of the left or right coronary artery, normal mechanical valve motion, and no significant regurgitation. The ascending aortic graft showed no kinking, but the distal ascending aorta was dissected from the distal anastomosis to the arch. The diameter of the distal ascending aorta was enlarged, which seemed to be caused by the new dissection at the distal ascending aorta and arch. Consequently, the distal anastomosis site revealed to be somehow stenotic since the graft was larger than the distal stump, which was small in the first surgery. The calculated cross-sectional area at the distal anastomosis from aortography was 1.7 cm². During diastole, the dissecting flap closed the true lumen of the aorta and extremely diminished the coronary flow (Fig. 1).

The pressure pullback curve showed an aortic root pressure of 132/23 mmHg and aortic arch pressure of 90/61 mmHg (Fig. 2). A systolic pressure gradient (42 mmHg) can be caused by a rather small anastomosis alone, since a large volume of catecholamine administration should provide systolic high-flow through the anastomosis. However, diastolic hypotension at the aortic root is difficult to explain with only a small anastomosis, since regurgitant flow through the anastomosis is only a coronary flow, which is estimated at 0.5 L/min with a competent aortic valve. Diastolic hypotension at the aortic root caused the coronary malperfusion. Pressure at the aortic root was the same as the left ventricular pressure, and the dissecting flap appeared to be functioning as an aortic valve. A stent graft to open the dissecting flap closure was initially attempted but was unsuccessful.

An emergency surgery for a total arch replacement was performed. The patient’s chest was opened through a median sternotomy. The distal ascending aorta and arch were dilated and bluish. Cardiopulmonary bypass with deep hypothermia at 20°C was initiated with blood sent to the right femoral artery and drained from the right
**Fig. 1** Aortography after the aortic root replacement. The arrows indicate the intimal flap position (A) during systole and (B) during diastole. Intimal flap movement causes narrowing of the true lumen.

**Fig. 2** The pullback pressure curve study after the aortic root replacement. The blood pressure at the aortic root was 140/20 mmHg, and the pressure at the aortic arch was 90/50 mmHg. Coronary perfusion at the aortic arch is markedly diminished.
atrium. The aortic graft was cross clamped. Retrograde and antegrade blood cardioplegia were administered. Under circulatory arrest at a nasopharyngeal temperature of 20°C, the aorta was opened, and selective cerebral circulation was initiated. Multiple entries were observed at the origins of the innominate artery, the left carotid artery, and the left subclavian artery. The dissecting flap extended from the ascending aorta to the arch. Flap suffocation apparently occurred when the dissecting luminal pressure pushed the flap, causing closure of the true lumen during diastole. An eighteen millimeter graft was inserted into the descending aorta as the elephant trunk. The total arch was replaced with a 24-mm four-branched graft (Hemashield).

The patient was weaned uneventfully from cardiopulmonary bypass. His postoperative course was somewhat eventful, although he had residual heart failure and respiratory failure. After the second surgery, eleven days passed before the patient could be extubated, and then three more days passed before he could be discharged from the intensive care unit (ICU). His hospital stay was 49 days. The patient was discharged without any morbidity.

A histologic examination of the aortic wall specimen showed cystic medial necrosis. The patient now works in business and has been in good condition for four years and four months since the surgery.

**Discussion**

The incidence of coronary involvement in a type A aortic dissection is reportedly 6% to 11%. The usual mechanism may be a dissection that has progressed into the coronary arteries, thereby resulting in narrowing or obstruction of the true lumen of the coronary arteries. It is unusual to have extreme diastolic narrowing of the true lumen of the aortic root occurring with the proximal invagination of the cylindrical flap. Diastolic intussusception of the tubular flap affects the degree of blood flow obstruction in the coronary arteries.

In our patient, a similar mechanism of coronary malperfusion occurred after an aortic root replacement. A post-operative dissection at the residual ascending aorta and arch (where the cross-clamped region may have been damaged) caused extreme diastolic narrowing of the true aortic lumen. This resulted in diastolic coronary malperfusion. We could not find a similar postoperative course in the medical literature.

Echocardiography indicated asynergy of the left ventricle with possible myocardial ischemia; however, it did not show any coronary ostial pathology. After a heart catheterization, the patient was diagnosed as having coronary malperfusion due to flap suffocation (Fig. 1).

The pressure pullback curve in our patient demonstrated that an unusual mechanism was causing coronary malperfusion (Fig. 2). Although criticism may indicate the effect of anastomosis size, it is difficult to explain marked diastolic hypotension only with the anastomosis size. We could not find a similar presentation of a pressure pullback curve in the medical literature. The intimal flap functioned as an aortic valve and sequential surgery corrected the problem.

**Conclusion**

We experienced a case of coronary malperfusion due to flap suffocation after type A dissection surgery. The mechanism of the malperfusion, which was revealed by the pressure pullback curve, has not been previously reported in the medical literature.

**References**