Subacute Type A Aortic Dissection with 10% of Ejection Fraction

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Abstract
A 36-year-old male suffered acute type A aortic dissection complicated with Ejection Fraction (EF) of less than 10% without coronary dissection and valvular disease. We decided to manage the heart failure first and when EF improved to 25% one week later, we performed ascending aorta replacement as this was subacute aortic dissection. Although central venous arterial extracorporeal membrane oxygenation support was needed for one day and the patient had embolic stroke on postoperative day 9, the patient was discharged to the rehabilitation center on postoperative day 22. The symptom of stroke was almost resolved at 6-month follow-up.

Keywords: Subacute type A aortic dissection; Low ejection fraction; CT

Introduction
Preoperative ventricular dysfunction is a significant risk factor of acute aortic dissection repair. There is no report about subacute type A dissection with low Ejection Fraction (EF). We report our management of subacute type A aortic dissection in a young patient who had severe ventricular dysfunction due to non-ischemic cardiomyopathy.

Case Presentation
A 36-year-old male patient with no significant medical history presented with shortness of breath and epigastric pain to the emergency room at an outside hospital. Heart rate was sinus rhythm 100 bpm, blood pressure was 174/135 mmHg. The patient was alert without any neurological symptoms. A Computed Tomography scan (CT scan) showed aortic dissection beginning at the sinotubular junction to the take-off of the left common carotid artery (Figure 1). All head vessels were intact. This aortic dissection was subacute as the onset of the symptom was three weeks ago. The patient was directly transferred to the Operating Room (OR). A transesophageal echocardiogram in the OR revealed bilateral ventricular dysfunction with ejection fraction of less than 10% and no valvular disease. The patient’s precise past medical history concerning cardiac dysfunction was unclear. Then we brought the patient to the hybrid room to perform coronary angiogram, which revealed intact coronary artery with left dominance. As the patient was hemodynamically stable, this aortic dissection was subacute, and coronary artery was intact. We decided to treat this patient medically first to optimize the patient’s condition and assess this unknown cardiomyopathy. The patient was transferred to the intensive care unit and carefully monitored while controlling blood pressure with β-blocker. One week later, the ventricular function improved to EF of 25%. EF was still low but the cardiac function was recovering. Therefore, we determined to operate this patient.

Arterial cannula was directly placed in the proximal aortic arch with a guide wire technique and dual venous cannula in the right atrium. The patient was cooled down to 34 Celsius. Initially cold blood retrograde cardioplegia was given and ascending aorta was clamped. Ascending aorta was transected and ostial cardioplegia was given. Dissection flap was fibrotic, which seemed to be chronic. The intimal tear was found in the proximal ascending aorta. Ascending aorta was replaced with 26 mm Gelweave™ graft (Terumo, MI, USA). After weaning from bypass, the patient went into supra ventricular tachycardia. It was cardioverted, however, the patient became hemodynamically unstable requiring cardiopulmonary bypass. Central cannulation Venous-Arterial Extracorporeal Membrane Oxygenation (V-A ECMO) was initiated to decompress the heart for the recovery. As the chest was left open, arterial cannula in the proximal aortic arch and venous cannula in the right atrium were used for V-A ECMO. The bypass time was 206 minutes and the cross-clamp time was 79 min without circulatory arrest. On Postoperative Day One (POD 1), ECMO was successfully explanted and the chest was closed. The patient was extubated on POD 2 and transferred to step down unit on POD 6. EF improved to 45%. On POD 9, the patient suffered right sided weakness...
Preoperative CT scan. Intimal flap begins at the sinotubular junction extended to the origin of the left common carotid artery. A larger entry in the proximal intimal flap could be seen. Right innominate artery was perfused from false lumen, left carotid artery and left subclavian artery was from true lumen. All aortic arch branches, left and right coronary ostium were intact. The mild left ventricular hypertrophy was noticed.

And aphagia. CT angiogram of the head showed left middle cerebral artery occlusion without dissection in the left carotid artery. An interventional radiologist emergently performed embolectomy through left carotid artery. The neurological function improved and the patient was discharged to the rehabilitation center on POD 22. A CT scan 5 months after the surgery showed residual aortic dissection in the aortic arch without aneurysmal dilatation. All aortic arch branches were intact. There was no dissection in the descending aorta. Right innominate artery was perfused from false lumen, left carotid artery and left subclavian artery was from true lumen.

Discussion

Without surgical intervention, the mortality rate in type A aortic dissection in the acute phase (within 2 weeks) is from 57% to 74% [1,2]. After 2 weeks, however, the incidence of mortality is significantly lower than that of the acute phase. As most patient with acute type A aortic dissection undergo immediate surgical repair, the data about the natural history and the appropriate timing of surgical intervention of subacute/chronic type A dissection are limited. Our patient had subacute type A aortic dissection and cardiomyopathy with 10% of ejection fraction. Low ejection fraction is a risk factor for high mortality and morbidities in aortic surgery [3]. Coronary artery dissection is a well-known cause of cardiogenic shock in type A aortic dissection. In a case of ischemic cardiomyopathy induced by coronary artery dissection, immediate intervention for reperfusion of the coronary artery is beneficial. In our case, EF was less than 10% and coronary artery was intact and we expected that immediate surgery could severely harm the cardiac function. We did not know this etiology of the cardiac dysfunction because of lack of exact medical history and whether this was reversible or not. As this aortic dissection was subacute, we decided to prioritize assessing this cardiac dysfunction and one week later, EF recovered to 25% with blood pressure control with β-blocker and a decision was made to proceed to the surgery. To minimize the CPB time and avoid circulatory arrest, we simply performed ascending aorta replacement under cross clamp on the distal ascending aorta to minimize the length of pump time to preserve the heart. Eventually short-term ECMO support was required, however ECMO was successfully removed on the following day and EF improved to 40% on POD 4. Although the prognosis of patients who need short-term ECMO support for post cardiotomy shock is promising, the outcome of the patients with prolonged ECMO support are disenchanting. Prolonged ECMO support is related to the complications including thromboembolic events, bleeding, infection, and end-organ failure [4]. The patient had embolic stroke of the left middle cerebral artery on POD 9. This was caused by thrombus in the left middle cerebral artery. There was no dissection in the left carotid artery but the intimal flap was close to the left common carotid artery, which might have been contributed to this thrombus formation. There might be an argument if more aggressive approach such as partial arch replacement under circulatory arrest to exclude all dissected aorta should have been done. Considering the risk from heart failure and the timing and type of surgical repair, we choose the safest procedure to minimize the invasiveness of surgical intervention to preserve the heart function. We could save this patient’s life and the heart function also significantly improved. The symptom of stroke was almost resolved at 6-month follow-up.

References