

Progression of Left Coronary Artery Dissection during and after Aortic Replacement in Acute Type A Aortic Dissection: A Case Report

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A 69-year-old man was transferred to our hospital with a diagnosis of acute type A aortic dissection. In the emergent operation, the dissection was found to extend to the orifice of the left coronary artery, but not to the coronary artery itself. The false lumen was closed using glue and sutures with felt strips, and graft replacement of the ascending aorta was performed. However, signs of myocardial ischemia were present after the operation, and the patient's condition continued to be unstable, even though intraaortic balloon pumping was initiated. A coronary angiogram and intravascular ultrasound performed three hours after the operation revealed a left main trunk stenosis due to pulsatile compression of the false lumen, which was caused by the extension of dissection. A coronary artery stent was subsequently deployed in the left main trunk. The patient was discharged four weeks later in a stable condition, although with segmental asynergy of wall motion, due to myocardial damage. (Ann Thorac Cardiovasc Surg 2007; 13: 209–212)

Key words: aortic dissection, myocardial infarction, surgery

Introduction

When acute aortic dissection (AAD), extends proximally and involves the coronary orifice, it is complicated by myocardial malperfusion and subsequent myocardial infarction. One of the major causes of death in AAD is myocardial damage,^{1,2} particularly damage arising from the total obstruction of the left coronary artery ostium. In most AAD cases accompanied by myocardial ischemia, signs of ischemia are present prior to the operation.² We report here an interesting case of AAD in which coronary artery malperfusion progressed during and after operation.

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Case Report

A 69-year-old man had a sudden syncopal attack and was admitted to a hospital. A computed tomography (CT) scan showed no cerebral lesion and revealed acute dissection of the ascending aorta. He was then transferred to our institute for surgical treatment. Although he regained consciousness upon admission, his blood pressure was still low, around 60–70 mmHg in spite of catecholamine infusion. An enhanced chest CT scan indicated a clotted false lumen localized to the ascending aorta and a massive pericardial fluid space (Fig. 1). Since cardiac tamponade, due to oozing from the false lumen, was thought to be the cause of the patient's low blood pressure, an emergent operation was undertaken.

Surgery was performed with the patient under total anesthesia and using a median sternotomy. Opening the pericardium relieved the cardiac tamponade and resulted in immediate and excessive recovery of the blood pressure, to 150 mmHg. Shortly afterwards, the QRS waveform on the electrocardiogram (ECG) monitor became

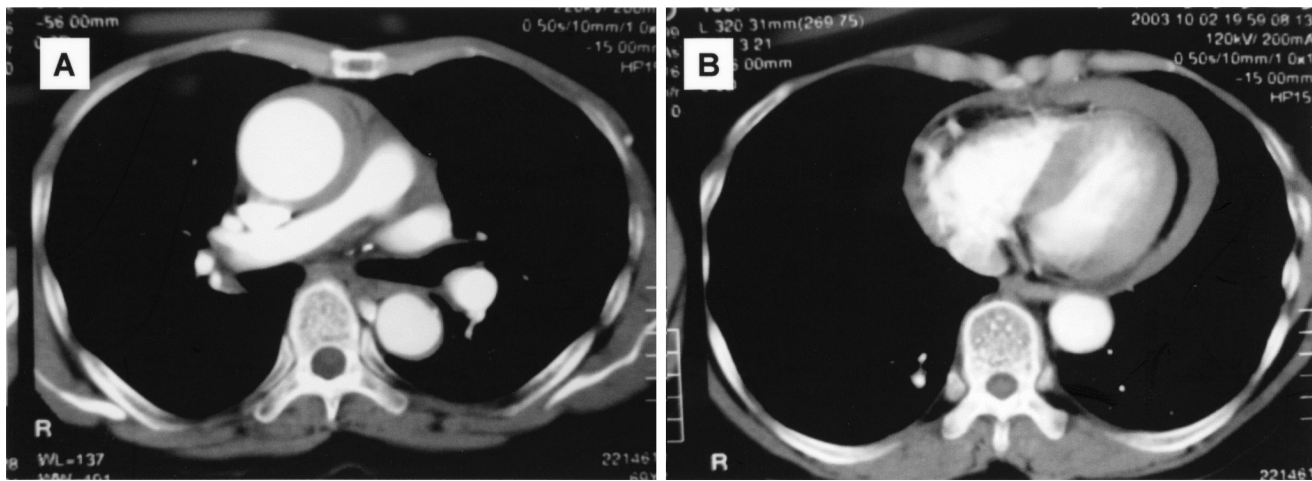


Fig. 1. Chest computed tomography showed (A) aortic dissection in the ascending aorta and (B) massive collected pericardial effusion.

wider, and it soon changed to indicate ventricular tachycardia and, subsequently, ventricular fibrillation. Direct cardiac massage (CPB) was immediately performed until a cardiopulmonary bypass (CPB) was initiated.

Under extracorporeal cardiopulmonary support, cardiac arrest was introduced with cross-clamping of the ascending aorta and retrograde cardioplegia infusion. The aortic root was incised, and an intimal tear was observed just beneath the Valsalva sinus. The dissection seemed to extend to the orifice of the left coronary artery, but not to the left coronary artery itself. Therefore, the proximal stump was reinforced using GRF glue in the false lumen and two felt strips on both the outside and inside of the aorta, and no additional procedure was performed affecting the coronary artery. We then carried out proximal anastomosis to a 22-mm woven Dacron graft, and core cooling was started. At a core temperature of 25°C, systemic circulatory arrest was introduced and a distal point for the suture line was decided upon, just proximal to the brachiocephalic artery origin.

The distal stump was trimmed and reinforced in the same fashion as the proximal stump and anastomosed to the graft. Systemic circulation was then re-started and the whole body warmed. The heart soon started beating spontaneously; however, ST segment elevation was observed on the ECG monitor. Since neither nitroglycerin nor administration of an inotropic agent improved the ST segment abnormality, an intraaortic balloon pump (IABP) was inserted and pumping was started. With the support of the IABP and an inotropic agent, the CPB was terminated and the patient was transferred to the ICU.

Even after admission to the ICU, the patient's blood

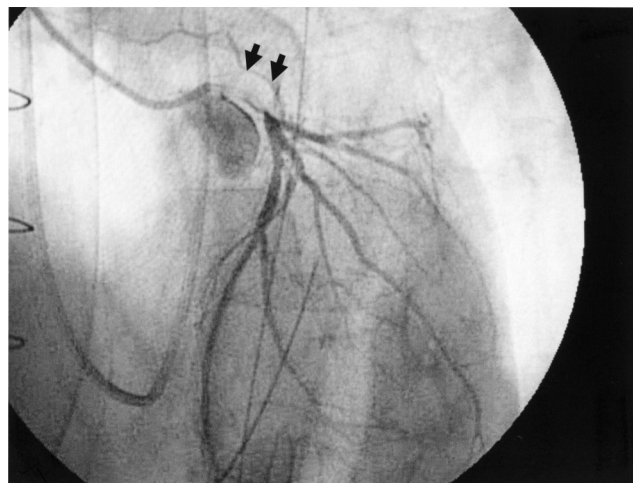


Fig. 2. The coronary angiogram demonstrated eccentric stenosis (arrows) and delay of the contrast medium in the left main trunk.

pressure and cardiac output remained low. The ECG suggested broad myocardial damage or ischemia, and transthoracic echocardiography showed asynergy in the anteroseptal and lateral areas. These results suggested coronary artery malperfusion, probably caused by progression of the aortic dissection into the left main trunk (LMT) of the coronary artery, and emergent coronary angiography (CAG) was performed three hours after the operation.

The CAG revealed an eccentric 90% stenosis of the LMT (Fig. 2), and intravascular ultrasound (IVUS) imaging showed that the stenosis was caused by pulsatile compression of the false lumen at the same site (Fig. 3). Given the circumstances during and after the operation,

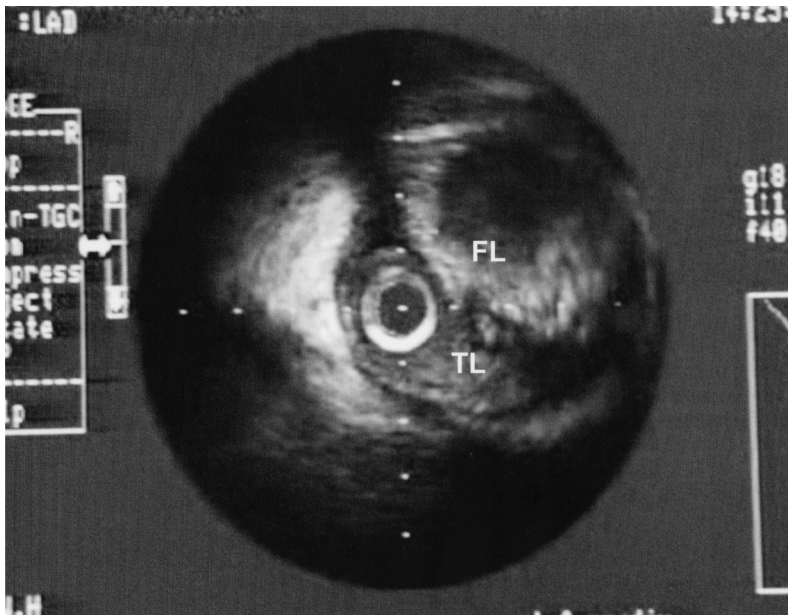


Fig. 3. Intravascular ultrasound demonstrated pulsatile compression from outside the coronary artery, indicating a false lumen was present. TL, true lumen; FL, false lumen.

it was speculated that the blood inflow into the false lumen occurred after recirculation to the proximal aorta owing to a small cut in the intima or a needle hole, and that dissection then progressed proximally to the left coronary artery ostium and into the LMT itself. Subsequently, the enlarged false lumen compressed the LMT, ultimately causing ischemia. A titanium coronary arterial stent was introduced at the lesion site, and the stenosis was successfully relieved. The postoperative course after catheter intervention was absolutely stable and uneventful, although echocardiography indicated asynergy of the left ventricular anterior wall as a result of perioperative myocardial damage. The patient was discharged from the hospital in a stable condition, about four weeks after the surgical repair.

Discussion

In clinical situations, AAD is often accompanied by organ malperfusion, which strongly affects the severity of the disease.³⁾ Once end-organ ischemia has taken place with AAD, surgical intervention to relieve the compression by the false lumen is needed as soon as possible to salvage the organ. In particular, myocardial ischemia caused by the disturbance of a coronary artery ostium by a false lumen is a severe and often lethal complication of AAD.^{1,2)} Therefore, coronary artery ischemia and concomitant coronary artery bypass grafting (CABG) have been reported as significant risk factors for postoperative

mortality.⁴⁾

This case had two points at issue. First, the myocardial ischemia occurred after opening the chest, and the patient showed no previous sign of it except for the syncope prior to the operation. In general, myocardial ischemia due to AAD is obvious soon after its onset, and some cases of AAD present with myocardial infarction. Thus, it is essential to keep in mind that a coronary artery disorder can happen, even after pericardiotomy, and therefore immediate establishment of CPB is important.

Second, the coronary artery dissection progressed after the surgical procedure was finished, even though the coronary artery orifice was carefully observed from inside the aorta and no additional procedure was deemed necessary, because the coronary artery itself showed no dissection.

Treatment of coronary artery dissection can vary with the grade of severity. Neri and colleagues²⁾ classified coronary malperfusion in aortic dissection into three main types, according to the extent of the coronary artery dissection: type A, ostial dissection; type B, dissection with a coronary false lumen; and type C, circumferential detachment with an inner cylinder intussusception. In the case of types B and C, it is not difficult to make the decision to undertake concomitant CABG. However, for a type A coronary dissection, as in this case, the coronary artery ostium seems intact because the intimal tear and the enlarged false lumen are closed using GRF glue and sutures. However, a fragile intima might easily be damaged

by needle holes, and arterial blood flow into the false lumen could re-occur if the management of false lumen is inadequate. Therefore, Neri et al. suggested repairing the coronary ostium or grafting a coronary artery bypass, even in type A cases.²⁾

In this case, myocardial ischemia should be suspected when circulatory instability was observed after the termination of CPB, and decision to carry out additional CABG was necessary without delay. Even after careful inspection of the inside of aorta and stump reinforcement, coronary artery dissection could occur, and immediate response is indispensable if myocardial ischemic sign is observed.

Once coronary malperfusion has occurred, myocardial salvage depends on how quickly it is recognized. However, it is not easy to estimate coronary artery disturbance, especially during surgery. Transesophageal echocardiography is reported to be useful for diagnosing AAD and coronary ostial disturbance, even during the operation.^{5,6)} The use of preoperative CAG to assess the presence of a concomitant coronary lesion, including coronary artery dissection, is still controversial. Most patients undergoing this operation are critically ill and there is little time to perform a CAG. On the other hand, Penn and colleagues reported that the determination of coronary status with a CAG did not impact on survival in patients undergoing emergency aortic surgery.⁷⁾

In conclusion, myocardial ischemia is possible whenever we deal with Stanford type A AAD, and an immediate decision is required to add bypass grafting if needed.

Aggressive treatment for coronary artery ostial dissection might be a positive factor in avoiding subsequent myocardial ischemia.

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