Successful Surgical Repair of Delayed Chronic Type A Dissection after Previous Coronary Artery Bypass Grafting

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This reports a case of a 68-year-old woman who had undergone coronary artery bypass 5 years previously. Magnetic resonance angiography (MRA) revealed that the ascending aorta was dilated to about 8 cm in diameter, with type A dissection, and with a patent left internal thoracic artery (LITA) graft to the left anterior descending artery (LAD). Angiography at the ascending aorta did not reveal a coronary artery, nor did it show the sequential saphenous vein graft (SVG) to the obtuse marginal and posterolateral branches. Although the risk of surgical treatment via repeat median sternotomy was very high, we successfully performed the reoperation using profound hypothermic circulatory arrest. The dissection in the mediastinum was facilitated by a sternum retractor for ITA-graft dissection, intraoperative surface echocardiography, and ultrasonic scalpel, with a widely opened bilateral pleural cavity. Furthermore, assuming that most of the myocardium was maintained by perfusion from the in-situ, patent, ITA graft, it was thought that cardioplegia was not necessary during profound hypothermic circulation. (Ann Thorac Cardiovasc Surg 2006; 12: 293–6)

Key words: reoperation, repeat median sternotomy, type A dissection, cardioplegia

Introduction

Delayed chronic type A dissection of the aorta after previous cardiac surgery (e.g. coronary artery bypass grafting (CABG) and aortic valve replacement) has been reported with variable etiology and with high surgical mortality.1–6) Recently we successfully undertook the surgical treatment of a patient with type A dissection who had received previous CABG and poor left ventricular function (LVF) under hypothermic circulatory arrest without cardioplegia.

Case

A 68-year-old woman was admitted with chest discomfort and dyspnea. She had undergone triple CABG (left internal thoracic artery (LITA) to left anterior descending artery (LAD), and sequential saphenous vein graft (SVG) to the obtuse marginal branch and the posterolateral branch), 5 years previously at our hospital, and had undergone hemodialysis for chronic renal failure for 8 years. Chest X-ray film demonstrated moderate cardiomegaly and dilated mediastinum. Magnetic resonance angiography (MRA) revealed that the ascending aorta was dilated aneurysmally to about 8 cm in diameter, with type A dissection and with a patent LITA graft to the LAD (Fig. 1). Angiography at the ascending aorta failed to reveal the left or right coronary arteries or the SVG, which seemed to diverge from the pseudolumen. The LITA graft had excellent patency (Fig. 2), but LVF was depressed, with an ejection fraction of 24%. Myocardial single-photon emission computed tomography (SPECT) showed ischemia in the inferoposterior area. The perfusion of the
heart seemed to be maintained almost solely via the LITA graft. Because it was thought that the risk of rupture of the aneurysm was higher than that of surgical repair, we planned the replacement of the ascending aorta and coronary artery bypass to the inferoposterior area.

**Operation**

After the satisfactory induction of general anesthesia, the left femoral artery and femoral vein were exposed, taped, and prepared with purse-string stitches for cannulation.

A secondary midline skin incision was made on the previous scar, followed by repeat sternotomy, with little trouble. Mediastinal adhesions were tremendously thick and heavy. We performed careful dissection to the right pleural cavity and opened it widely. Next, we executed a careful and painstaking dissection by retracting the left side of the sternum, using a sternum retractor (Jostra AG, Hirrlingen, Germany), for dissection of the ITA graft. After the left pleural cavity was entered, the chest was opened widely. The mediastinum became *en bloc* with a patent LITA graft and a huge ascending aortic aneurysm

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**Fig. 1.** Preoperative magnetic resonance angiography (MRA) (T2-weighted images).
The ascending aorta was dilated to about 8 cm in diameter, with dissection, and with a patent left internal thoracic artery (LITA). The arrow and asterisks indicate the patent LITA and the pseudolumen of dissection, respectively.

**Fig. 2.** Preoperative angiography of the left internal thoracic artery (LITA) graft.
The LITA graft to the left anterior descending artery (LAD) had excellent patency.
(about 8 cm). We were able to dissect safely the LITA and the ascending aorta using an ultrasonic scalpel (Harmonic Scalpel, Ethicon Endo-Surgery Inc., Cincinnati, OH, USA) with intraoperative surface echocardiography for detecting the position of the patent LITA graft. The SVG was collapsed with a scar like appearance. The flow rate from the LITA graft to the LAD, measured using a 2.5 MHz Doppler flow probe (Butterfly Flowmeter, Medi-Stim Inc., Oslo, Norway), was good (87 ml/min). We planned bicalve venous cannulation so that retrograde cerebral perfusion was obtained via the superior vena cava (SVC), but the wall of the SVC was very weak and had been frequently lacerated. Therefore, under full heparinization (300 unit/kg), cardiopulmonary bypass (CPB) was started — initially with the arterial infusion cannula in the left femoral artery and a single, 2-stage venous cannula in the low right atrium. After the vent tube was inserted in the left ventricle via the right upper pulmonary vein, core cooling was initiated. While cooling proceeded we dissected around the heart, and the distal end of the SVG was anastomosed to the posterolateral branch of the left circumflex coronary artery. When a nasopharyngeal temperature of 18°C was reached, circulatory arrest was initiated and the ascending aorta was cut open. The patient’s head was covered with an ice pack during core cooling. An intimal tear was identified in the anterior side of the proximal ascending aorta. We did not perform direct infusion of cardioplegia to the orifice of the coronary artery, because there was a severely calcified atherosclerotic lesion around the coronary orifices and preoperative angiogram showed that both orifices of native coronary artery were occluded. The ascending aorta was transected just at the brachiocephalic take-off, obliquely, on the lesser curvature of the arch, just beyond the intimal tear. After the distal stump was reinforced with a strip of ePTFE felt on the outside of the aorta, the beveled end of a piece of 30-mm woven Dacron prosthesis (UB Shield Graft™, Ube Medical Co., Ltd., Tokyo, Japan) was anastomosed with huge aneurysm, a patent ITA graft from the previous cardiac surgery is in agreement with the reported prevalence of 6–9%.2,5) Apparently, postoperative findings could not clarify the etiology of the dissection. The early hospital mortality in late type A dissection of the aorta after previous cardiac surgery is high, exceeding that of primary type A dissections by almost 2-fold.41 On the other hand, the low incidence of perforation after previous cardiac surgery is in agreement with the reported prevalence of 6–9%.2,5) Apparently, postoperative pericardial scarring and adhesions may, in part, explain this condition. Though the aortic dissection was chronic in our case, the diameter of the ascending aorta was 80 mm with dissection. We judged the risk of aneurysm rupture to be higher than that of surgical repair.

Even so, the surgical risk in this case was very high because of the combination of repeat median sternotomy with huge aneurysm, a patent ITA graft from the previous CABG, depressed LVF, and chronic renal failure with ongoing hemodialysis. In particular, if the LITA graft was damaged, it would very likely be fatal because this graft was anastomosed using a 3-0 polypropylene running suture. The proximal end of the SVG was anastomosed to a hole made on the ascending prosthesis with a 6-0 running polypropylene suture. De-airing of the left heart was carried out via the aortic root catheter, followed by declamping of the graft. The heart spontaneously resumed beating into ventricular fibrillation, underwent cardioversion into a slow rhythm, and was then paced. About 30 min after declamping the aorta, the hemodynamics stabilized with good left ventricular contraction. CPB was withdrawn uneventfully. After sufficient hemostasis was achieved, the chest was closed.

On the next postoperative day, the patient regained consciousness and the endotracheal tube was extubated on the second postoperative day. Continuous hemodiafiltration was performed for 4 days, followed by hemodialysis 3 times a week. Postoperative MRA showed that aortic dissection had disappeared and a patent SVG was identified. 201-thallium myocardial SPECT showed that the perfusion in the territory of SVG and the inferoposterior wall had been improved.

**Discussion**

Aortic dissection has previously been reported as a complication originating from cross-clamp injury,39 or from intimal tears at the suture line of a CABG41 or from the site of cannulation.50 In the present case, the intimal tear was identified in the anterior of the ascending aorta, which was compatible with the site of the suture line of the SVG or the site of antegrade cardioplegia infusion, but intraoperative findings could not clarify the etiology of the dissection. The early hospital mortality in late type A dissection of the aorta after previous cardiac surgery is high, exceeding that of primary type A dissections by almost 2-fold.41 On the other hand, the low incidence of perforation after previous cardiac surgery is in agreement with the reported prevalence of 6–9%.2,5) Apparently, postoperative pericardial scarring and adhesions may, in part, explain this condition. Though the aortic dissection was chronic in our case, the diameter of the ascending aorta was 80 mm with dissection. We judged the risk of aneurysm rupture to be higher than that of surgical repair.
was the most important artery supplying the myocardium in this patient. The repeat median sternotomy and mediastinal dissection were performed using safe and effective methods. After the right pleural cavity was entered and opened widely, the left side of the sternum was lifted using a sternum retractor for ITA harvest, dissected using a harmonic scalpel and the left pleural cavity was opened widely. This method of dissection yields a comparatively good surgical field. In addition, intraoperative surface echocardiography was very useful for dissection of the patent ITA graft from the adherent heart and ascending aorta. We believe that this is an efficacious approach for dissection of the mediastinum for redo CABG via repeat median sternotomy, in the presence of a patent graft.

Most commonly, profound hypothermic circulatory arrest was performed with intermittent antegrade cardioplegia or continuous retrocardioplegia, or both. In our case, however, the calcified atherosclerosis (including fibrin deposits) around the coronary orifices forced us to abandon both direct antegrade cardioplegia. We believed that the patient’s heart was being maintained not by the native coronary artery but by the LITA, because the native coronary artery could not be identified by preoperative angiography, and myocardial SPECT showed severe hypoperfusion except for the territory of the LITA graft. In the present case, it is unclear whether retrograde cardioplegia or perfusion via the LITA was preferable for myocardial protection, but we consider either to be better than antegrade cardioplegia alone. On the other hand, it is not known whether retrograde cardioplegia alone is able to perfuse enough of the territory of the LITA. Because, retrograde perfusion from the coronary sinus is not drained when the LITA graft is clamped, In addition, it may cause excessive edema of the heart. We thought that the low temperature of the perfusate (18°C) meant that perfusion via the LITA alone was sufficient to protect the myocardium while the heart was arrested. In a case such as this one, if most of the myocardium is maintained by perfusion from an in-situ patent ITA graft, myocardial protection may be sufficient if perfused via the LITA, under profound hypothermic circulation. Though deep hypothermia circulatory arrest time was comparatively long with 48 min, it was lucky for us not to have caused a central neurological complication. Covering the head with an ice pack may have contributed to prevention of central neurological complication.7)

References