Successful Treatment of Acute Dissection of the Donor Aorta After Orthotopic Heart Transplantation

Ali Kubilay Korkut, a Francis Wellens, MD, a Luc Foubert, PhD, DSc, b and Marc Goethals, MD c

Acute aortic dissection is one of the rare aortic complications that occur after orthotopic heart transplantation. We report the second case of successful surgical treatment of aortic dissection confined to the donor aorta in a recipient of an orthotopic cardiac allograft. A 68-year-old patient was admitted with chest pain and shortness of breath 7 years after orthotopic heart transplantation. He previously had undergone twice coronary artery bypass grafting. Echocardiography revealed acute dissection of the donor aorta. The patient underwent urgent Bentall procedure with a prosthetic conduit. The post-operative course was uneventful. The heart donor was a 40-year-old man with known arterial hypertension and who had received long-term ergotamine tartrate therapy for migraine. This case demonstrates that heart-transplant recipients with arterial hypertension and donor-related risk factors are prone to aortic complications and require careful follow-up. J Heart Lung Transplant 2003;22:701–704.

Heart transplantation (HTX) is standard treatment for patients with end-stage cardiomyopathy and offers 1- and 5-year survival rates >85% and >75%, respectively. Aortic dissection is one of the fatal complications that occur after HTX. In this case report, we describe a patient with acute dissection of the donor aorta several years after undergoing 2 coronary artery bypass surgeries and orthotopic HTX.

CASE REPORT

A 68-year-old man with acute Stanford type-A dissection of the donor aorta underwent a Bentall procedure. Previously, the patient had undergone 2 coronary artery bypass surgeries, in 1978 and 1993. He experienced end-stage heart failure and 2 years later underwent orthotopic HTX with Lower-Shumway technique. The length of the donor aorta extended 3 cm above the commissural edge. End-to-end anastomosis was performed using Blalock 4-0 Prolene suture. The donor was a 40-year-old man with hypertension who died of cerebral bleeding. The donor had received ergotamine tartrate for migraine. In the immediate post-transplant period, the recipient received azathioprine, cyclosporine, and prednisone as immunosuppressive therapy. Azathioprine was stopped at the 52nd month. A recent, complete checkup revealed no major problems after radical prostatectomy for non-disseminated prostate cancer. Echocardiography showed that the left ventricular ejection fraction was 52% ± 10%, and an endomyocardial biopsy specimen showed minimal active rejection, standard grading IA. The blood pressure was controlled with 5 mg/day amlodipine, 20 mg/day furosemide, and 20 mg/day lisinopril. The patient experienced mild renal insufficiency caused by nephroangiosclerosis and cyclosporine toxicity. Blood creatinine levels were between 2.5 and 3 mg/dl, and blood urea nitrogen levels were between 90 and 110 mg/dl.

Seven years after HTX, the patient was re-admitted for sudden onset of severe back pain and severe systemic hypertension. An early diastolic murmur
was present. Electrocardiography showed signs of old inferior myocardial infarction and non-specific T-wave abnormalities. Transthoracic echocardiography revealed the presence of an aortic dissection flap, severe aortic valve insufficiency 3/4 because of prolapse of the non-coronary cusp, mitral valve insufficiency 2/4, tricuspid valve insufficiency 2/4, and pulmonary hypertension.

Transeosophageal echocardiographic evaluation was performed during surgery and confirmed severe aortic valve insufficiency (3/4) and dissection flap in the ascending aorta (Figure 1). The aortic arch and the descending aorta were normal. Based on this clinical and echocardiographic picture, the patient underwent urgent surgical repair.

After median sternotomy, the ascending aorta was cannulated through the aortic arch, and the patient was cooled to 27°C. The aorta was cross-clamped and aortotomy performed. Dissection was present below the anastomosis between donor and recipient aorta, and also involved the non-coronary cusp (Figure 2). The aortic root was replaced with a bioprosthetic 25-mm stentless porcine aortic conduit (No-React Stentless Aortic Valve Conduit, model NR-2000C, Sheltigh; Millburn, NJ). The patient had no operative complications and was extubated on the second day and discharged on 15th post-operative day. Cyclosporine (2 mg/kg/day) and prednisone (5 mg/day) were continued as immunosuppressive therapy, and a calcium antagonist was used to control blood pressure. Histology of the donor aorta showed fibrosis of the tunica intima and atrophy of the tunica media.

**DISCUSSION**

Aortic dissection is a rare and fatal complication after HTX. Several risk factors are associated with aortic dissection. Hypertension and immunosuppressive agents, especially glucocorticosteroids, are the most common predisposing factors for aortic dissection after HTX.

Acute aortic rupture in the very early phase after HTX can be related to weakness of the aortic tissue and technical mistakes.
Many patients referred for HTX have undergone previous open-heart surgery, and incorrect surgical dissection can damage the native aortic wall. A large number of patients have end-stage ischemic heart disease, which often is associated with arterial hypertension, diabetes, hypercholesterolemia, and tobacco use and as a consequence atherosclerosis of the native arterial system. In addition, the donor may have had an undiagnosed connective tissue disorder, missed easily in the “fast-track” donor evaluation. Nevertheless, hypertensive episodes play a major role in aortic dissection in the late phase. In our patient, blood pressure was controlled with angiotensin-converting enzyme inhibitors and diuretics.

Aortic dilatation and aortic aneurysm formation can be pre-disposing factors for acute aortic dissection. Dilatation and development of abdominal aortic aneurysm are more common in patients >55 years old, with pre-transplant ischemic cardiomyopathy, and with pre-operative ejection fraction <20%, as described by Bull et al. These authors suggest an association between improved hemodynamics (ejection fraction) and development of increased aortic diameter after heart transplantation. Although this study focused on the infrarenal aorta, the mechanism also could play a role in the pathophysiology of thoracic aneurysm or aortic dissection formation.

Hypertension is a serious problem in 50% to 90% of patients treated with cyclosporine. In rare cases, aortic dissection after renal transplantation has been reported after long-term use of immunosuppressive agents.

The site of the aortic anastomosis in HTX is a potential source of early and late complications. Discrepancy in tissue quality and size between donor and recipient’s aorta are responsible for compliance mismatch that causes a difference in wall tension. Difference in wall tension acts as a critical stress factor at the anastomosis. It also suggests that the length of both donor and recipient aorta has to be kept as short as possible to reconstruct a short and streamlined ascending aorta.

**FIGURE 2** Dissection is located below the anastomosis site, between the donor heart and the recipient aorta, and involved the non-coronary cusp. F, flap; NCC, non-coronary cusp.
Other authors believe atherosclerosis is coincidental rather than causative of aortic dissection. However, aortic dissection of an arteriosclerotic aneurysm increases the risk of rupture. Several risk factors commonly associated with the development of atherosclerosis are identified frequently in recipients, such as receiving long-term immunosuppression, especially glucocorticosteroids. Because of the fragility of the aortic tissue, surgical repair of aortic dissection is hazardous in patients with atherosclerosis receiving long-term steroid therapy.

Successful repair of acute dissection of the donor aorta after orthotopic HTX has been reported in only 1 case in the literature. Complications in the donor aorta in the late phase after HTX can be related to several donor-related factors. The donor in our case died of cerebral bleeding. He had stress-dependent hypertension. He also had migraines and received ergotamine tartrate therapy for a number of years. Garnier et al reported the unusual case of a 50-year-old woman with high blood pressure who experienced spontaneous dissection of the cervicocephalic, renal, and hepatic arteries and of the descending aorta. She had been taking ergotamine tartrate for 10 years for migraine. In our patient, rupture of the donor aorta in the late phase after HTX may have been related to weakness of the donor aortic tissue caused by the use of ergotamine in the donor. Ergotamine also may play a direct role in fibrosis of the aorta.

In conclusion, all transplant recipients with hypertension are candidates for aortic complications. The site of the aortic anastomosis in HTX deserves particular attention as a potential source of early and late complications. These patients require careful surveillance and medical treatment for hypertension and atherosclerosis. Chronic diseases, such as hypertension in the donor, or long-term drug therapy in the donor may cause weakness of the aortic tissue. In addition to these donor-related factors, inconsistency of tissue quality and size between the donor’s and the recipient’s aorta, technical failure, arterial hypertension, and immunosuppressive agents may cause serious complications in the donor aorta after HTX. Complications related to the donor aorta are seldom reported in the literature. To prevent these complications, careful investigation of the donor is necessary because chronic diseases and drugs used by donors for several years may affect recipients even late after transplantation.

REFERENCES