Recipient Type A Aortic Dissection After Orthotopic Heart Transplantation

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Abstract

Very few aortic dissections have been published following a heart transplant. Most of these have been reported as donor aortic dissections. Recipient aortic dissection is extremely rare. Here, surgical treatment of chronic Stanford type A recipient aortic dissection in a 40-year-old patient who underwent an orthotopic heart transplant 10 years ago will be discussed in the light of the literature.

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Introduction

Donor and/or recipient aortic dissection following heart transplantation (HTx) was published in very small numbers of articles (1-7). As a result of the preparation of recipients, donor care,

anesthesia and surgical techniques, improvements in immunosuppressive therapy, the survival of HTx patients are increasing day by day, thus increasing the incidence of late complications (1). Incompatibility between donor and the recipient aorta may cause infection, technical errors, impaired hemodynamic flow dynamics or hypertension. In rare cases, these conditions can cause aortic aneurysm and/or dissection. Type A chronic aortic dissection can cause cerebrovascular events, malperfusion in visceral organs, aortic valve failure, cardiac ischemia or tamponade (2,3,6). The frequency of this rare complication was reported between 1-2% (5,6). In this case, we presented a 40-year old male patient with recipient aortic dissection and its surgical treatment.

Case Report

A forty-year-old male patient was diagnosed with dilated cardiomyopathy and treated by heart transplantation 10 years ago. Donor was a 15-year-old female. After HTx, he developed hypertension and diabetes mellitus (DM). The patient has recently admitted to our clinic with developing chest pain and dyspnea. In his first surgery, it was noted that the mismatch between the recipient and donor aorta was compensated by plication of the recipient aorta. The patient's immunosuppressive regimen was cyclosporin, mycophenolate mofetil and steroids. The patient was checked regularly for ten years, and no signs of aortic pathology were detected. However, for the last month he described shortness of breath and chest pain. In echocardiography, left ventricular and valvular functions were normal. Ascending aortic diameter was reported as 75 mm. In thorax computed tomography (CT) angiography, the donor aorta was seen normally to the suture line level. Dissection begins from the native ascending aorta (beginning from the suture line), extends through the iliac arteries (Figure 1). All vessels were originated from the true lumen except the left renal and celiac arteries. There was a reentry at renal artery level. Coronary CT angiography was performed, and coronary arteries were found to be normal. There was no problem in the blood tests except for mild urea and creatinine elevation. Cyclosporin levels were within the desired limits.

In the operation, subclavian arterial and right femoral venous cannulation was performed. Redo sternotomy was performed without entering the cardiopulmonary bypass (CPB). There were tight adhesions on the aneurysm, vena cava superior and the right atrium side. There was almost no adhesion around the inferior vena cava, diaphragmatic and lateral face. The adhesions were dissected. The second venous cannula from the superior vena cava, retrograde canula and vent canula was placed. Ascending aorta was found to be very wide and about 8 cm in diameter.

In the heart transplant surgery, teflon reinforced stitches was noticed within the antero-lateral part of the anastomosis line. The brachiocephalic artery has been turned and, cross clamp has been placed, then aneurysm/dissection sac opened. The dissection seen from the beginning of the suture line, and the native aorta and the donor aorta were seen to be separated from each other. The donor aorta was normal. False lumen was seen moving towards the large curvature (Figure 2). Blood cardioplegia from the coronary ostia was given and the heart was arrested. Afterwards, cardiac protection was continued with retrograde cardioplegia plus intermittent antegrade perfusion at 25 minutes. The patient was cooled to 28 C°. The proximal side was trimmed to the donor aorta. By using 28 mm Dacron graft and 4/0 polypropylene sutures, proximal anastomosis was made. The donor tissue was intact, and no reinforcement was needed (Figure 3).

Arcus branches were inspected. It was all originated from the true lumen. The tissues were intact. There was a slight flow from the false lumen. Both lumens were combined to generate the true lumen and reinforced with teflon felt and fibrin sealant (Tisseel, Baxter) (Figure 4). Distal anastomosis was made with 4/0 polypropylene sutures and reinforced with teflon felts as standard (Figure 5). The cross clamp was removed, following the removal of the air from the proximal side. The cross-clamping time was 59 minutes, the selective brain perfusion time was 25 minutes, and the CPB time was 102 minutes. After decamping, the heart started in sinus rhythm spontaneously. The patient was heated and CPB terminated without the need for inotropic support. The postoperative period of the patient went smoothly. Immediately after the extubation, the immunosuppressive regimen was initiated. The patient was closely monitored from the cardiac point of view and no problems were encountered. The patient was discharged on the 10th day. A month later, the thorax CT showed that the false lumen was completely thrombosed.

Discussion

Post-HTx dissection is rare and requires urgent surgical intervention with high mortality (1). Ruptures that occur in the acute period are dramatic and are due to tension in the suture line or aortic mismatch (3). Proper adjustment and good alignment of the donor aorta will reduce the tension at the suture line (5). These patients present with shortness of breath, chest pain, back pain and dry cough. (5) However, patients may be asymptomatic due to denervation. The development of aneurysm or tear in the donor aorta prevents the dissection from spreading towards the recipient aorta, and neurological problems may not be seen (3,5).

Our patient had similar symptoms. If dissection occurs in donor tissue, it can usually be detected in control

echo or autopsies (4,5). Lang et al have detected the incidence in childhood as 0.9%. This rate is almost similar in adult patients. They stated that diagnosis is difficult due to cardiac denervation (6). The situation is in the form of painless dissection. There is no or minimal mediastinal bleeding due to adhesions (3,6). The cause of dissection is not clear (3). However, cross clamping trauma, de-airing needle location, connective tissue diseases such as Marfan syndrome (2), donor and recipient aortic mismatch, candida infection, secondary hypertension due to postoperative immunosuppressive therapy (1,2,4), technical reasons, mediastinal heart trauma, past percutaneous coronary intervention, or invasive interventions (4,6) can be the cause. Frequent antibiotic use, central catheter insertion, total parenteral nutrition are the most common causes of candida infections (3,4,5). Donor hearts harvested from Marfanoid, and Loeys-Dietze donors, the risk of dissection of the donor aorta increases (5,6).

In our patient, dissection was found to be caused by the anastomosis line. Although, dissection was reported mostly in the donor aorta (2,4) in the literature, recipient aortic dissection was present in our case. We think that the dissection of the suture line prevents it from progressing to the donor aorta.

Although, no hypertension was present on the pre-transplant history. The patient has used ACE inhibitor and Ca channel blocker in the last five years for hypertension possibly due to immunosuppressive therapy. DM in addition to hypertension can increase the risk even more. Hypertension may trigger the development of dissection. Furthermore, a mismatch was reported between the donor and the recipient's aorta during the patient's initial surgery. This was the second risk factor. Iatrogenic intimal trauma was ruled out. Because our patient did not undergo angiography or invasive intervention before. Past infections and mycotic causes may cause pseudoaneurysms. They are larger and develop in the ascending aorta (3-5,8).

Coppola et al detected aortic dissection in two postmortem cases. In these patients, they reported that the tear was in both donor and recipient aorta and was caused by cross-clamping (8). Postmortem studies or pathological examination of the extracted aneurysm tissue can be beneficial illuminating in terms of etiology. Our patient did not have any known connective tissue disease.

Donor aortic dissections are more likely to be retained as the retrograde that develops downwards, and aortic insufficiency is a fatal complication of such acute aortic dissections. It may require Bentall and Cabrol procedure or valve protecting procedure such as David (2,3,5,7,8-12). The Bentall procedure after HTX was first performed by Schellemans et al (5). Since dissection developed from the anastomosis line to the distal in our patient, the donor aorta was intact and there was no aortic insufficiency. There was no need to interfere with the valve. Leone et al applied the Bentall procedure and the Elephant Trunk technique together in such cases (8). Supra-coronary ascending aorta and hemi arcus replacement may be sufficient in cases like ours. (13) Indeed, in our early thorax CT, we found that the false lumen was completely thrombosed. Following HTx, ascending aortic aneurysms developing in the native aorta were also be reported. Ascending and hemi arch replacement were performed for the treatment (14).

We recommend that the donor aorta should be shortened properly during the trimming procedure so that the aortic anastomosis line is not stretched. Infection is also a major cause. Hypertension and DM due to immunosuppressive therapy must be treated sufficiently. Such patients require rapid and aggressive surgical treatment for optimal long-term results. As in other dissection cases, cardiac team should be prepared for all kinds of scenarios.

Conclusion

Type A aortic dissection after heart transplantation could occur in recipient aorta. Surgical management can be performed successfully. The requirements for the prevention of dissection during both intraoperative and postoperative periods are of utmost importance.

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Figure 1: Dissection beginning from the suture line (arrow) Figure 2: Intraoperative image of aortic dissection starting from greater curvature. Figure 3: Intact donor aorta and suture line Figure 4: Both lumens were combined to generate the true lumen and reinforced with teflon felt and fibrin sealant Figure 5: Distal anastomosis