A rare cause of right heart failure after heart transplantation: right coronary cusp thrombosis

Hatice Soner Kemal,1 Serkan Ertugay,2 Çağatay Engin,2 Sanem Nalbantgil,1 Tahir Yağdı,2 Mustafa Özbaran2

Departments of 1Cardiology and 2Cardiovascular Surgery, Medical Faculty of Ege University, İzmir, Turkey

ABSTRACT

Right heart failure and pump thrombosis are potentially life-threatening complications and major causes of morbidity following left ventricular assist device implantation. Herein, we report a case of 47-year-old male patient who had successful heart transplantation 20 months after left ventricular assist device implantation. The device-supported period was uneventful during follow-up, but right coronary cusp thrombosis occluding the right coronary artery resulted in right heart failure 11 days after heart transplantation.

Keywords: Right heart failure; thrombosis; transplantation; ventricular assist device.

ÖZ


Anahtar sözcükler: Sağ kalp yetmezliği; trombüs; nakil; ventrikül destek cihazı.

Since 1967, heart transplantation has become the treatment of choice for patients with end-stage heart failure.1 Native aortic root thrombosis2 after left ventricular assist device (LVAD) implantation3,4 and left atrial thrombus after heart transplantation5 have been previously reported in the literature, but herein, we report an unusual case of right coronary artery occlusion after heart transplant surgery that was caused by right coronary cusp thrombosis. In addition, the occlusion was also accompanied by left atrial thrombus.

CASE REPORT

A 47-year-old male patient with diabetes mellitus and end-stage heart failure due to ischemic heart disease underwent elective Heart Mate II™ LVAD implantation (Thoratec Inc., Pleasanton, CA, USA) as bridge to heart transplantation. The postoperative period was uneventful, and the patient was discharged on warfarin, acetylsalicylic acid (300 mg/day) and conventional heart failure therapy. During the follow-up period, the patient had a driveline infection and was treated with antibiotics (levofloxacin). He was rehospitalized a few months later because of a high fever, nausea, and vomiting and was diagnosed with a pocket infection. However, after prolonged treatment with broad-spectrum antibiotics, the clinical signs of infection disappeared. The patient then received an orthotopic heart transplant 20 months after the LVAD had been implanted. The donor was 50 years old and had no known cardiovascular risk factors. The surgeon assessed the donor heart carefully before proceeding with the procedure and detected only smooth fatty plaques of the aorta. Methylprednisolone and mycophenolate mofetil were utilized for the patient’s early immunosuppressive
therapy, and tacrolimus was added on the postoperative first day. No immediate postoperative complications were seen, and echocardiography showed normal valve competence and systolic function in both ventricles. On the 11th postoperative day, the patient developed a sudden onset of tachycardia, and electrocardiography revealed atrial fibrillation with a rapid ventricular response. Transthoracic echocardiography (TTE) was then performed, but no pathological findings were visualized. The patient was anticoagulated and administered an amiodarone infusion. In addition, we observed normal sinus rhythm with a left bundle branch block pattern. After approximately an hour, the patient’s hemodynamics deteriorated sharply, which led to bradycardia and hypotension. Immediate inotropic support was started, and repeat TTE revealed a hyperechoic mass on the right coronary cusp via the parasternal short-axis view (Figure 1). After this finding, the patient was transferred to the operating room where intraoperative transesophageal echocardiography confirmed the presence of the thrombus on the aortic valve that extended from the right sinus of Valsalva into the right coronary artery as well as a thrombus just above the posterior mitral valve that extended to the base of the left atrium. The patient then underwent successful aortic root exploration, and the thrombus on the right coronary cusp was removed (Figures 2a-c). Because of an adequate retrograde flow from the right coronary artery, additional coronary bypass surgery was not performed. Subsequently, the left atrial thrombus was excised via a small left atrial incision. However, we were unable to wean the patient off of cardiopulmonary bypass due to right ventricle failure. We then performed right femoral artery and vein cannulation along with venoarterial extracorporeal membrane oxygenation, which allowed us to wean the patient from the pump. Unfortunately, the patient’s right heart function did not improve after this procedure, and he died as a result of sepsis on the 21st day after the heart transplant while being followed up in the intensive care unit.

DISCUSSION
Virchow’s triad is still present after cardiac transplantations, and factors that cause a hypercoagulable state, disruption to the flow, or endothelial injury can cause thrombus formation.

**Figure 1.** Short-axis transthoracic echocardiographic image showing the hyperechoic mass on the right coronary cusp.

**Figure 2.** Intraoperative view of (a) the thrombus located at the right coronary cusp and (b) the removal of the thrombus material from the right coronary artery. (c) Macroscopic view showing the removed thrombus material.
In the case of our patient, the pocket infection before the heart transplant may have been a predisposing factor. It is also possible that the bradycardia could have caused blood stasis on the aortic cusps, but the patient had been anticoagulated. A clotting disorder, for example heparin-induced thrombocytopenia, might also have played a role, but since the patient’s thrombocyte levels were within the normal range, we ruled this out as a possibility. Prior to the LVAD implantation, the patient had been evaluated for procoagulant conditions (i.e., protein C or S deficiency, factor V deficiency, and antiphospholipid disorders), but no abnormalities were detected. Furthermore, he had no thrombotic events during the LVAD support.

Native aortic valve thrombi, especially those not associated with an abnormal aortic valve or infectious endocarditis, are very rare and also extremely challenging to treat when accompanied by acute myocardial infarction. However, in our case, the tricuspid aortic valve was not damaged and was functioning properly.

Thrombolytic therapy is a treatment option for patients without a history of recent major surgery. Surgical removal is safe, but it requires aortic cross-clamping, which may cause a deterioration in ventricular functions. In cases in which a residual thrombus in the coronary artery is suspected, a saphenous graft bypass can be performed.

In conclusion, unfortunately, after investigating this rare case of post-transplant right coronary artery occlusion caused by right coronary cusp thrombosis that resulted in late right heart failure, we could find no reason why this occurred. Therefore, we recommend that further research be conducted to identify the cause of this anomaly to ensure the survival of other patients who might have this condition.

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**REFERENCES**