

Emergency percutaneous coronary intervention for left main trunk thrombus following orthotopic heart transplantation

Masaki Tsuji^{1*}, Daisuke Nitta¹, Shun Minatsuki¹, Hisataka Maki¹, Yumiko Hosoya¹, Eisuke Amiya¹, Masaru Hatano^{1,2}, Eiki Takimoto¹, Osamu Kinoshita³, Kan Nawata³, Minoru Ono³ and Issei Komuro¹

¹Department of Cardiovascular Medicine, Graduate School of Medicine, The University of Tokyo, Hongo 7-3-1, Bunkyo-ku, Tokyo 113-8655, Japan; ²Department of Therapeutic Strategy for Heart Failure, The University of Tokyo, Tokyo, Japan; ³Department of Cardiac Surgery, Graduate School of Medicine, The University of Tokyo, Tokyo, Japan

Abstract

Heart transplantation (HTx) is the gold standard therapy to improve quality and quantity of life in end-stage heart failure patients. However, recipients are at risk of experiencing allograft rejection and post-transplant complications, in the acute as well as chronic phase. A 43-year-old man with a history of left ventricular non-compaction underwent orthotopic HTx. On Day 7, transthoracic echocardiography showed a sudden decrease in cardiac function with hypokinesis in a left ventricular anterior wall distribution. Coronary angiography revealed a large thrombus in the left main trunk. With intra-aortic balloon pump support, emergency percutaneous coronary intervention was performed. Endomyocardial biopsy showed no rejection. A left main trunk thrombus is rare in the early phase after HTx, but it can be a life-threatening complication. Transthoracic echocardiography is well known to be important in the management of heart transplant recipients, and coronary angiography as well as myocardial biopsy should be considered when left ventricular wall motion is impaired.

Keywords Heart transplantation; Left main trunk thrombosis; Percutaneous coronary intervention

Received: 8 June 2018; Accepted: 27 January 2019

*Correspondence to: Masaki Tsuji, Department of Cardiovascular Medicine, Graduate School of Medicine, The University of Tokyo, Hongo 7-3-1, Bunkyo-ku, Tokyo 113-8655, Japan. Tel: +81 3 3815 5411; Fax: +81 3 5800 9142. Email: mtsuji-cib@umin.ac.jp

Introduction

Heart transplantation (HTx) is the gold standard therapy to improve quality and quantity of life in end-stage heart failure patients.¹ However, recipients are at risk of experiencing allograft rejection and post-transplant complications, in the acute as well as chronic phase. Here, we report an uncommon case of left main trunk (LMT) thrombosis in the acute phase of HTx.

Case report

A 43-year-old man with a history of left ventricular (LV) non-compaction received an implantable LV assist device as part

of a bridge-to-transplant strategy due to advanced heart failure. Four years later, he underwent orthotopic HTx using the modified bicaval technique.

The heart was obtained from donation after brain death in his or her 30s. The donor heart had a normal LV ejection fraction on transthoracic echocardiography (TTE). The total ischaemic time was approximately 4 h.

On removal of the cardiac implantable electronic device lead with an excimer laser after separation from cardiopulmonary bypass, a considerable amount of bleeding occurred from the subclavian vein, which resulted in haemorrhagic shock. Internal cardiac massage and direct current defibrillation to treat ventricular fibrillation were needed, achieving haemodynamic stability. We transfused 20 units of packed red blood cells, 30 units of platelet concentrates, and 40 units of fresh-frozen plasma during and after the operation but did

not transfuse any activated factor concentrates. After the operation, the patient was transferred to the intensive care unit in a stable condition.

Dobutamine, noradrenaline, and dopamine were started at rates of 5.6, 0.18, and 4.2 $\mu\text{g}/\text{kg}/\text{min}$. Noradrenaline and dopamine administrations were reduced gradually and discontinued on post-operative Days 1 and 5. Dobutamine administration was reduced gradually and continued at a rate of 1.9 $\mu\text{g}/\text{kg}/\text{min}$.

An immunosuppressive regimen of cyclosporine, mycophenolate, and prednisolone was initiated without induction therapy. Low-dose aspirin was also started. Daily TTE showed normal contraction until Day 6; however, hypokinesia in an LV anterior wall distribution was noted on Day 7. Electrocardiography showed a normal sinus rhythm (98 beats/min), and ST-segment change was not observed (*Figure 1*). Renal dysfunction with a creatinine level of 1.67 mg/dL (normal, <1.07 mg/dL) was noted, but emergent catheterization was performed. Right heart catheterization indicated a mean pulmonary capillary wedge pressure of 20 mmHg and a cardiac index of 1.78 L/min/m². Coronary angiography showed a thrombus-like large filling defect in the LMT (*Figure 2A and B*; Supporting Information, *Movie S1*). Thus, we decided to perform percutaneous coronary intervention (PCI) with intra-aortic balloon pump support. Intravascular ultrasound revealed a low echogenic thrombus (*Figure 3A and B*). Aspiration thrombectomy was attempted, but few clots were aspirated. After debulking with an excimer laser, a 3.5 \times 18 mm everolimus-eluting stent was successfully deployed at the LMT–left anterior descending artery lesion (*Figure 2C and D*;

Supporting Information, *Movie S2*). Endomyocardial biopsy was negative for cellular and antibody-mediated rejection.

After PCI, his haemodynamic condition stabilized, and the intra-aortic balloon pump was removed on the next day. The maximum creatine kinase-MB level was 17 U/L (normal, <12 U/L). TTE revealed normal LV systolic function. He was discharged on Day 30. Six months after PCI, follow-up coronary angiography showed no re-stenosis at the stent site.

Discussion

An LMT thrombus is a life-threatening condition that has a severe clinical presentation involving cardiogenic shock or sudden cardiac death.² Acute coronary syndrome (ACS) due to acute thrombus formation is rare in the early phase after HTx.

Coronary artery disease after HTx is often related to allograft vasculopathy, which is an accelerated arteriosclerosis characterized by diffuse, longitudinal, and concentric proliferation of intimal smooth muscles and the extracellular matrix in the remote phase.³ However, ACS in the early phase after HTx is associated with the surgical procedure, especially the biatrial anastomosis technique, tissue embolization, a post-surgical coronary air embolus, thrombosis, and coronary artery disease of the donor heart.⁴

The organ donor in the present case had a low risk of coronary artery disease. Additionally, the recipient had no history of coagulation abnormality, and heparin-induced thrombocytopenia antibody was negative.

Figure 1 Twelve-lead electrocardiography on the day of catheterization shows a normal sinus rhythm (98 beats/min) and no ST-segment change.

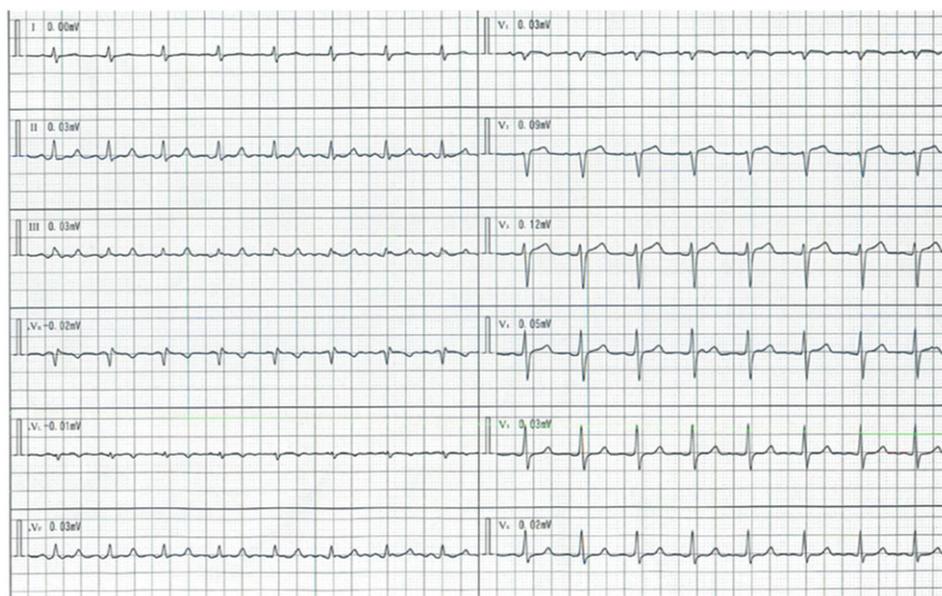


Figure 2 Coronary angiography before percutaneous coronary intervention. A thrombus-like large filling defect is seen in the left main coronary artery (arrow). (A) Right anterior oblique cranial view and (B) left anterior oblique caudal view. Coronary angiography after percutaneous coronary intervention at the left main trunk with a drug-eluting stent. (C) Right anterior oblique cranial view and (D) left anterior oblique caudal view.

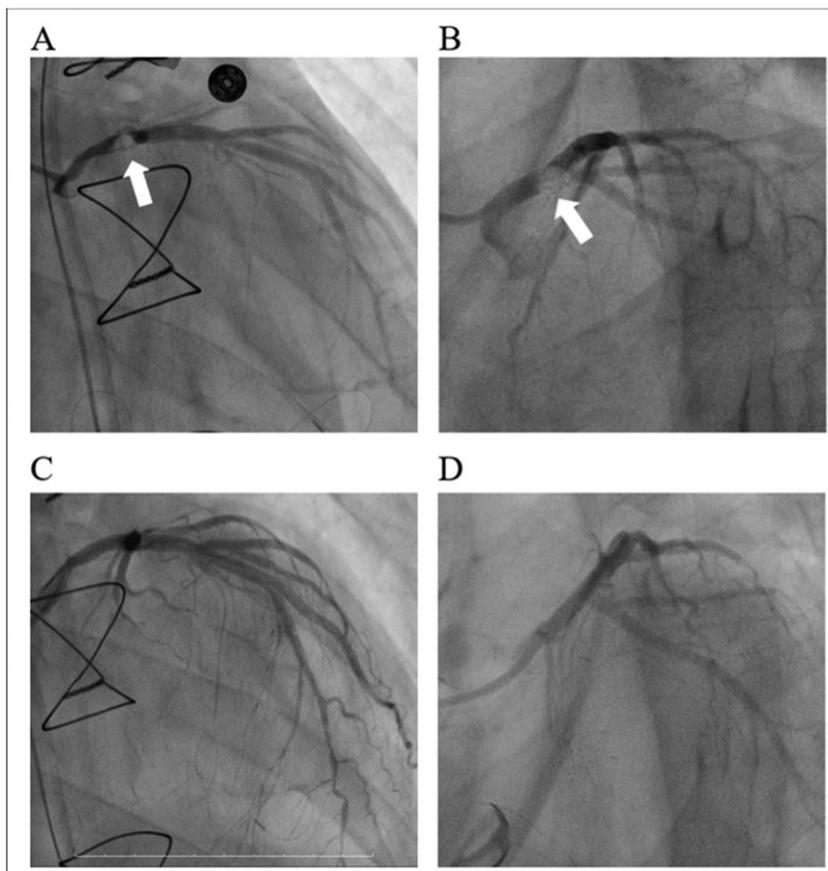
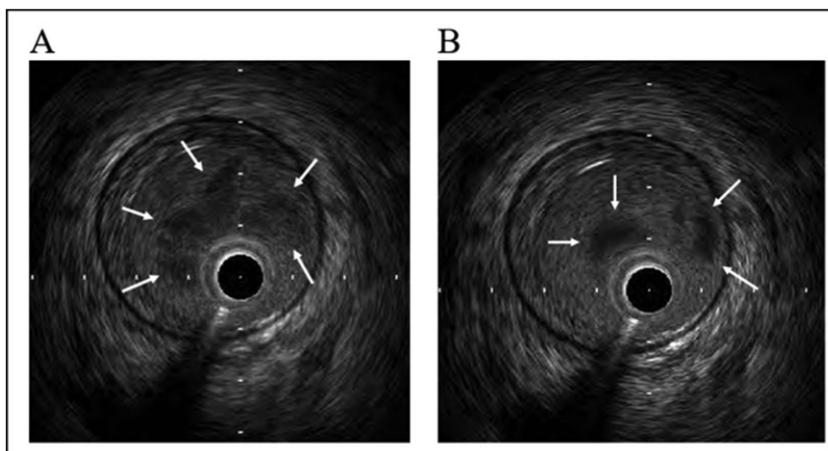


Figure 3 Intravascular ultrasound of the left main trunk shows a low echogenic lesion suggestive of a thrombus (arrow). (A) Proximal lesion and (B) distal lesion.



Several causes of LMT thrombus formation are possible. First, a small thrombus might have remained after flushing and removing blood from the donor heart. Development of a thrombus within a few days after the operation might have led to a coronary embolism.

Second, haemorrhagic shock occurred during cardiac device lead removal, and it is known that significant deceleration in blood flow under hypotension increases the risk of thrombosis and embolism formation. Although haemostasis was achieved with compression and transfusion, this is an extremely unusual complication. There might have been a thrombus in the coronary artery at the time of bleeding, and ventricular fibrillation might have occurred owing to myocardial ischaemia triggered by hypovolaemia.

Third, coronary vasospasm has been described in heart transplant recipients,⁵ and it is considered as a cause of thrombosis. Administration of vasopressors or inotropic catecholamine agents is one of the risk factors of coronary vasospasm.⁶ Coronary angiography was performed without intracoronary infusion of nitrates in our case, and there was no coronary artery spasm. However, as the spasm provocation test was not performed, coronary spasm could not be ruled out completely.

Antibody-mediated rejection was considered in the differential diagnosis, which could lead to vascular injury, coagulation, and graft dysfunction in the form of reduced cardiac function. However, histological, immunopathological, and se-

rological findings characteristic of antibody-mediated rejection were not noted in our case.

The presentation of ACS in heart transplant recipients is atypical because of autonomic denervation of the transplanted heart.⁷ In our case, as the condition was asymptomatic and the electrocardiogram was normal, diagnosis was difficult. TTE is a useful non-invasive modality to detect LV dysfunction. It is important to perform coronary angiography as well as myocardial biopsy for diagnosis when TTE shows regionally impaired LV wall motion in heart transplant recipients.

Conflict of interest

None declared.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Movie S1. Pre-percutaneous coronary intervention cine loop with a thrombus in the left main coronary artery.

Movie S2. Post-percutaneous coronary intervention cine loop with an implanted drug-eluting stent.

References

1. Lund LH, Edwards LB, Dipchand AI, Goldfarb S, Kucheryavaya AY, Levvey BJ, Meiser B, Rossano JW, Yusen RD, Stehlik J. The registry of the International Society for Heart and Lung Transplantation: thirty-third adult heart transplantation report—2016; focus theme: primary diagnostic indications for transplant. *J Heart Lung Transplant* 2016; **35**: 1158–1169.
2. De Luca G, Suryapranata H, Thomas K, van't Hof AW, de Boer MJ, Hoorntje JC, Zijlstra F. Outcome in patients treated with primary angioplasty for acute myocardial infarction due to left main coronary artery occlusion. *Am J Cardiol* 2003; **91**: 235–238.
3. Chih S, Chong AY, Mielniczuk LM, Bhatt DL, Beanlands RS. Allograft vasculopathy: the Achilles' heel of heart transplantation. *J Am Coll Cardiol* 2016; **68**: 80–91.
4. Rossignol DA, Kipreos B, Akosah K, Mohanty PK. Accelerated transplant coronary artery disease and massive silent acute myocardial infarction in a heart transplant patient—a case report and brief review of literature. *Angiology* 1999; **50**: 947–953.
5. Boffa GM, Livi U, Grassi G, Casarotto D, Isabella G, Cardaioli P, Panfili M, Chioin R. Angiographic presentation of coronary artery spasm in heart transplant recipients. *Int J Cardiol* 2000; **73**: 67–74.
6. Kawano H, Fujii H, Motoyama T, Kugiyama K, Ogawa H, Yasue H. Myocardial ischemia due to coronary artery spasm during dobutamine stress echocardiography. *Am J Cardiol* 2000; **85**: 26–30.
7. Di Cori A, Petronio AS, Gemignani C, Zucchelli G, Di Bello V, Mariani M. Symptomatic acute myocardial infarction in a cardiac transplant recipient successfully treated with primary coronary angioplasty: evidence of prognostic importance of chest pain after cardiac transplantation. *J Heart Lung Transplant* 2005; **24**: 1146–1149.