

Early thrombus formation in patient with HeartWare left ventricular assist device presenting with acute heart failure



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Heart failure is one of the leading causes of mortality and morbidity in the world. Heart transplantation is still the gold standard therapy despite emerging treatment options. Due to the limited number of available donors, the use of ventricular assist devices has increased. However, increasing incidences of complications are observed with using these devices. In this article, surgical treatment of a huge mobile thrombus formation in an inflow cannula due to ineffective anticoagulation in a 59-year-old man who received a HeartWare ventricular assist device because of ischemic cardiomyopathy is presented.

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Introduction

The use of a left ventricular assist device (LVAD) in the management of advanced heart failure has grown in recent years. Thrombosis of LVADs is a potentially lethal complication, as it usually results either in the need for urgent transplantation, LVAD replacement, or death. Device thrombosis incidence is 0.02 events per patient per year [1,2]. Subtherapeutic anticoagulation is the most common cause of device thrombosis. Early diagnosis and treatment of

LVAD is important. Device thrombosis is clinically suspected in the presence of hemolysis, elevated lactate dehydrogenase (LDH), worsening heart failure, and/or device malfunction (power spike, low-flow alarms) [3]. We present a case with inflow cannula thrombus, due to subtherapeutic anticoagulation which required surgical treatment.

Case report

A 59-year-old man with a history of ischemic cardiomyopathy with an operation of coronary artery bypass grafting in 2004, presented with

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end-stage heart failure requiring implantation of a continuous-flow LVAD for bridging therapy for cardiac transplantation, and received a HeartWare System, Framingham, MA, USA, LVAD 15 months ago. The patient presented to the cardiology department with “low flow” alarms for 2 days.

At presentation, he was dyspneic and anxious. Physical examination showed blood pressure of 90/60 mmHg and a heart rate of 69 beats/min. Cardiovascular system examination showed apical 3/6 systolic murmur and S3. Respiratory system examination showed rales reaching mid lung zones. Electrocardiography showed atrial fibrillation 73 beats/min, Q waves in inferior derivations, and loss of R wave progression in anterior derivations. The device’s settings were checked. The pump’s flow rate was an average of 3.5 L/min and the power consumed was an average of 3.5 watts at the patient’s 468 days of monitoring. On presentation, while the pump revved 2600 rpm, the flow rate was between 1 and 1.8 L/min, and the power was an average of 2.5 w. When the log file was examined, there had been a sudden drop in flow and power 2 days ago. The patient’s LDH value was 1680 unit/L and the international normalized ratio value was found 2.7 at admission. Although the patient’s international normalized ratio was in the range of therapeutic values, he had a history of irregular use of anticoagulants in the previous 2 weeks. Transthoracic echocardiography was performed, and the aortic valve opening with every cardiac cycle and low flow velocity through the inflow cannula suggesting inadequate unloading of the left ventricle was seen. Transesophageal echocardiography was then performed. Gastric views showed a mobile huge thrombus formation in the inflow cannula (Fig. 1). Because of the mobile,

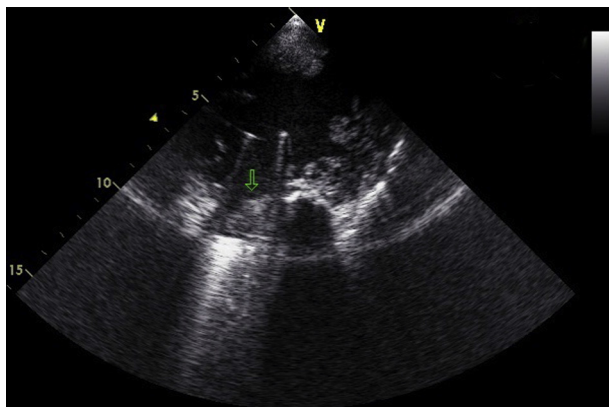


Figure 1. A huge thrombus in the inflow cannula.

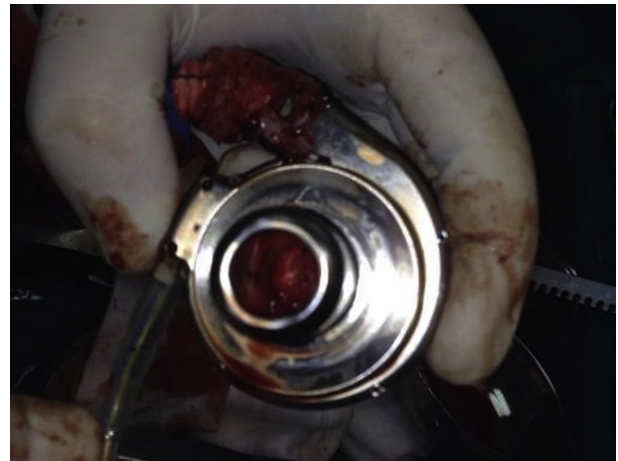


Figure 2. Thrombus formation in the inflow cannula surgically extracted.

huge thrombus, and unstable condition, surgery was preferred to thrombolytic therapy. The patient was referred to the cardiac surgery department immediately. Anterolateral thoracotomy incision was performed from the fifth intercostal space and the thrombus was detected in the extracted pump’s inflow cannula (Fig. 2). Pump replacement was performed successfully by using a minimal thoracotomy technique. After the operation, the pump’s flow rate was observed at 3.2 L/min. In the postoperative follow-up periods, the patient’s hemodynamics showed a stable pump flow rate of an average of 3.4 L/min and there were no findings suggestive of obstruction in the outflow graft. Because the patient’s body mass index was 21.1, a 3.4 L/min pump flow was considered to be sufficient for the patient and he was discharged 2 weeks later.

Discussion

Heart failure is one of the leading health problems because of increasing incidence and prevalence worldwide. In 2010, more than 40 million patients were living with heart failure. According to 1990 data a 14% increase has been observed. Globally, 38% of heart failures were caused by ischemic heart disease. The pattern varies by region. Ischemic heart disease was responsible for 61% of heart failure in North America and 50% in Western Europe [4].

Heart transplantation is the gold standard treatment for patients with advanced heart failure. Due to donor limitation, mechanical circulatory support is being used increasingly to treat patients with advanced heart failure as bridge to transplant therapy or destination therapy. First-generation

pulsatile flow pumps have been used as a bridge to transplant therapy. Pulsatile flow pumps have been replaced with continuous-flow rotary pumps. Continuous-flow rotary pumps have been demonstrated as adequate hemodynamic support and cause less hemolysis; in addition these pumps have helped to improve patients' life quality, due to their small size [5].

Widespread usage of mechanical support devices have brought a number of problems. These problems lead to device dysfunctions, one of which is a low pump alarm caused by various mechanisms of cannula obstruction. These include partial inlet occlusion by adjacent myocardial trabeculations, cannula angulation into the myocardium, or other cannula malposition caused by left ventricular underfilling, inlet or outlet kinking, and cannula thrombus [6,7]. Echocardiography is mostly an ideal tool to detect these problems. It is noninvasive, widely available, and can be performed at the bedside.

LVAD thrombosis is related to increased morbidity and mortality, usually requiring replacement of the device and requires a high level of clinical suspicion. In a LVAD patient, if there are new signs or symptoms of heart failure, changes in the functional capacity, an increase in LDH levels (a diagnostic marker of hemolysis), and changes in LVAD parameters, device thrombosis should be suspected [3]. Additionally, when used in conjunction with elevated LDH levels, echocardiographic "Ramp test" is highly sensitive and specific in the determination of LVAD thrombosis. When there is device thrombosis, partial or total occlusion, there may be a sudden drop in power and flow. In addition, power and current can be increased in parallel with the gradual formation of thrombus [8]. In our case, the patient had worsening heart failure and elevated LDH levels, a sudden drop in device power, and flow. We suspected LVAD thrombosis and performed transesophageal echocardiography and detected a huge mobile thrombus in the inflow cannula. As we saw the inflow cannula thrombus on the echocardiographic examination, a Ramp test was not performed. Different strategies are used in the treatment of continuous and axial flow LVAD thrombosis, including systemic or intraventricular thrombolytics or surgical replacement. Intraventricular thrombolytics were used successfully with the axial-flow pumps with a low risk of hemorrhagic events [2,3,9]. However, pump replacement despite high morbidity and mortality

can be performed in unstable patients or those who have contraindication for thrombolytic therapy.

Successful treatment of continuous flow LVAD thrombosis has been noted with thrombolysis despite the risk of catastrophic hemorrhagic events, especially intracranial bleeds. In our clinic, the first approach in hemodynamically stable patients with pump thrombus is thrombolytic therapy. However, in this case, the patient was hemodynamically unstable. So, as the first approach we preferred pump replacement because the thrombus was large and mobile, the symptoms of heart failure were serious, and severe hypotension was accompanied despite inotropes. After pump replacement, the patient clinically recovered and was discharged 2 weeks later.

Conclusion

LVAD thrombosis is a serious problem and treatment must be started immediately. Despite high risks, surgical treatment is an option for LVAD thrombosis in unstable patients.

References

- [1] Slaughter MS, Rogers JG, Milano CA, Russell SD, Conte JV, Feldman D, et al. Advanced heart failure treated with continuous-flow left ventricular assist device. *N Engl J Med* 2009;361:2241–51.
- [2] Lindenfeld J, Keebler ME. Left ventricular assist device thrombosis: another piece of the puzzle? *JACC Heart Fail* 2015;3:154–8.
- [3] Birati EY, Rame JE. Diagnosis and management of LVAD thrombosis. *Curr Treat Options Cardiovasc Med* 2015;17:361.
- [4] Forouzanfar MH, Moran A, Phillips D, Mensah G, Ezzati M, Naghavi M, et al. Prevalence of heart failure by cause in 21 regions: Global burden of diseases, injuries and risk factors-2010 study. *J Am Coll Cardiol* 2013;61(10_S). [http://dx.doi.org/10.1016/S0735-1097\(13\)60786-X](http://dx.doi.org/10.1016/S0735-1097(13)60786-X).
- [5] Neragi-Miandoab S. A ventricular assist device as a bridge to recovery, decision making, or transplantation in patients with advanced cardiac failure. *Surg Today* 2012;42:917–26.
- [6] Scalia GM, McCarthy PM, Savage RM, Smedira NG, Thomas JD. Clinical utility of echocardiography in the management of implantable ventricular assist devices. *J Am Soc Echocardiogr* 2000;13:754–63.
- [7] Catena E, Milazzo F. Echocardiography and cardiac assist devices. *Minerva Cardioangiol* 2007;55:247–65.
- [8] Stulak JM, Sharma S, Maltais S. Management of pump thrombosis in patients with left ventricular assist devices. *Am J Cardiovasc Drugs* 2015;15:89–94.
- [9] Rothenburger M, Wilhelm MJ, Hamel D, Schmidt C, Tjan TD, Böcker D, et al. Treatment of thrombus formation associated with the MicroMed DeBakey VAD using recombinant tissue plasminogen activator. *Circulation* 2002;106:S189–92.