Early Aortic Root and Left Main Coronary Artery Thrombosis after Left Ventricular Assist Device Implantation

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Abstract

We describe a case of 39 years old male, underwent continuous flow LVAD implantation (HeartWare) HVAD as destination therapy (DT) for severe left ventricular dysfunction, moderate right ventricular dysfunction and severe pulmonary hypertension, presented 3rd day post-operatively with polymorphic ventricular tachycardia (VT storm) alternating with VF before aborting to bradycardia and complete heart block. Coronary angiography revealed a big LM coronary artery thrombus extended distally to LCX and LAD arteries and pedunculating into the aortic root proximally, despite full coverage with antiplatelet and anticoagulant therapy. In conclusion, the “take-home message” is: we should be “thrombus minded” for early diagnosis and management of post LVAD implantation aortic root and LM coronary artery thrombosis and it is necessary in improving post-operative outcome. Also, high risk surgical interference in such sick patients can be avoided by early interventional management.

Keywords: Thrombosis; LVAD implantation; Heart failure; Heart transplantation

Introduction

In the modern era, the use of surgically implanted left ventricular assist devices (LVADs) as a bridge therapy to either recovery or cardiac transplantation has given a hope at the end of the HF course of disease. More recently, the use of LVADs as “DT” has further illuminated a future for patients with end-stage HF [1].

Today, LVADs have evolved from large, bulky pulsatile systems to smaller, compact, fully implantable continuous flow (CF) pumps.

These CF-LVADs use rotodynamic pumps to transfer kinetic energy from a circulating impeller to the bloodstream, thereby generating forward flow [1,2].

CF-LVADs can be divided into two categories: axial-flow and centrifugal-flow pumps. The physiology of axial versus centrifugal flow pumps was reviewed by Starling et al. in 2013. In both cases, blood is pulled into the impeller of the pump via an inlet cannula connected to the left ventricular apex and delivered to the systemic circulation via an outflow cannula connected to either the ascending or descending aorta [2-4].

LVAD thrombosis is a rare but well-described adverse event following this surgery and it develops generally on the device itself, because the original texturing inside the pump facilitates thrombus formation. Starling et al. summarized the rate of device thrombosis among 837 patients across three implantation centers in USA, and Kirklin et al. described data reported to the Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) on the rate of device thrombosis among 6910 patients from 2011 till April 2014. But all these big studies were done on Heart Mate2 devices (Thoratec) [4,5] and very few were done on (HeartWare) HVAD devices.

Figure 1: Coronary angiography: A) Thrombus in aoric root and LM+LCX. B) Aspiration thrombectomy. C) Patent coronaries.
Left main coronary artery thrombosis and aortic root thrombosis are rarely described, but in our case; thrombus had occurred early with the newer generation (HeartWare) HVAD device despite full anticoagulation and antithrombotic coverage.

Case Report

39 years old man was diagnosed with severe dilated cardiomyopathy since 2003 with recurrent admissions due to decompensated congestive heart failure. Dual Chamber ICD was inserted in 2012, but removed after few months due to infection and thrombosis. He is a known diabetic on insulin therapy, heavy smoker with liver cardiac cirrhosis and has severe chronic kidney disease with baseline creatinine around 200 mmol/L. Echocardiography showed severe LV dysfunction with EF less than 20% and RV moderate to severe dysfunction with systolic PAP pressure 55-65 mmHg. He remained in NYHA IIIb heart failure despite optimum medical treatment, so the decision was LVAD implantation as DT. After complete preoperative work up for LVAD surgery; (HeartWare) HVAD device implantations was done without complications and the patient was shifted to cardiovascular-ICU with minimal inotropic support, then extubated in the same night 10 hours post operatively. Heparin infusion (60-90 PTT protocol) was started 8 hours post-surgery. Next day, he was ambulated outside the bed and started taking orally.

Figure 2: Photograph showing the clots after successful suction thrombectomy.

His post-operative course was uneventful, except from frequent PVCs which are common in such LVAD cases. Apart from PVCs he had smooth post-operative course until 3rd day, when he developed intermittent polymorphic ventricular tachycardia (VT storm) which required frequent DC shocks and amiodarone IV boluses followed by infusion, plus lidocaine boluses and infusion without satisfactory response. Later on, VT was aborted to bradycardia and 3rd degree heart block. His laboratory results showed INR=6.4, PTT=77.9, PT=41.7, LDH=240 IU/L, TropT=0.635 mg/L (mildly increased) and normal creatinine kinase=77 U/L, CK-MB=13 U/L, serum creatinine=213 mmol/L, AST=14 U/L, Alkaline phosphatase=214 U/L, K=4.8 mmol/L, Na=137 mmol/L, Cl=98 mmol/L, and plasma lactate=1 U/L. The LVAD machine readings were: flow=4.5 L/min, RPM=2400, energy power=2.7 watt. He was on oral aspirin 162 mg and clopidogrel 75 mg daily (warfarin was withheld due to high INR).

The bedside echocardiography was done which didn't show any LV, LA or LVAD thrombus, with well-functioning LVAD machine and severe LV and RV dysfunction.

So the decision was to shift him to the cath lab for diagnostic coronary angiography, which showed big aortic root clot occluding LM coronary artery and extending in to LAD and LCX coronary artery (Figure 1).

Suction thrombectomy was done successfully from LM, LAD and LCX Coronary arteries and from the aortic root using right femoral access using 8 French Export catheter. At the end of the procedure, a TIMI III flow was achieved with good results and patent left coronary system (Figures 1 and 2). The patient was shifted back to cardiovascular surgery-ICU in a better shape and more stable hemodynamically with normal sinus rhythm, alternating with paroxysmal AF and PVCs.

On the next day, the patient required external RVAD implantation to support his failing RV, which was removed 6 days later when the RV started to recover. Internal medicine and hematology consultation was sought to exclude inherent or acquired causes of hypercoagulability, and the results were negative for hyperhomocysteinemia, antiphospholipid antibodies, protein C and protein S deficiencies, or factor V leiden mutations. So the probable explanation for his clot formation was stasis of blood in the aortic root due to laminar continuous flow from the LVAD and hypercoagulable state of chronic renal impairment. The likely explanation for no initial significant rise of cardiac enzymes is, a) early diagnosis and management of thrombus, b) no stress on the myocardium as LVAD takes nearly the entire burden, and c) severely myopathic and scarred myocardium (EF<20%). There was a significant rise in the cardiac enzymes only after suction thrombectomy, with the peak about 6 hours. At the end of his post-operative course, his recovery was satisfactory and was shifted to post-cardiac surgery recovery ward to complete his routine post-operative care.

Discussion

Since the last two years, across major LVAD centers in USA, there was a greater incidence of LVAD-related thrombosis as compared to the previous years. Studies prompted further meticulous data, which established that rates of so-called “LVAD pump thrombosis” had jumped over several years from 1%-2% to 6%-8% [4].

Thromboembolic complications, such as device thrombosis and cerebrovascular accident (CVA), can be devastating. According to INTERMACS, 11% of patients on device support at 1-year have a new CVA [5].

Starling et al. documented a sudden spike in HeartMate2 (Thoratec) pump thrombosis started from 2011. Kirklin et al. and Starling et al. reports confirmed this spike as well as a dramatic shift to early pump thrombosis with HeartMate2 (Thoratec) device more than the new HeartWare (HVAD), which was approved for BTT in November 2012 [1-3].

In a study done by Saeed et al. in 2014 on the role of aortic valve opening and thrombotic events with HeartMate2 (Thoratec) devices, he concluded that the thrombus formation in the dilated areas like aortic root sinuses of valsalva and carotid bulbs post CF-LVAD implantation is due to stasis of blood in these areas due to continuous flow mechanism itself and this laminar flow can be accentuated if the aortic valve (AV) is not opening regularly. So he concluded that
preserving AV opening may be helpful in reducing stasis in such dilated areas and reducing chances of thrombus formation and embolization by giving semi-pulsatile flow [5].

Freid et al. documented the 1st case of LM and aortic root thrombosis complicated by ST elevation myocardial infarction (STEMI) in a 46 years old male on the 2nd day post HeartMate2 (Thoratec) implantation as DT who had to undergo urgent CABG surgery plus surgical removal of his aortic root thrombus [6]. In our case, the patient was younger (39 years old male) and received (HeartWare) HV AD device as DT and successfully managed by the interventional cardiologist, who was able to aspirate the thrombus from his coronaries and from aortic root as well (Figures 1 and 2).

Freed et al. described the 1st case of aortic root and left main coronary thrombus in (HeartWare) HVAD in a case of 60-year-old female who presented with a non-ST elevation myocardial infarction 3 months post placement of (HeartWare) HVAD as BTT. She stopped to take anticoagulant three days before her presentation with acute anterior wall MI, was treated medically with thrombolytic therapy and underwent successful orthotopic heart transplantation 6 months later [7]. This was different from our case, where we had a 39 years old male developing thrombus in his aortic root and extending in to LM three days post (HeartWare) HVAD implantation as DT and was anti-coagulated to supra therapeutic levels (INR reached >6).

Conclusion

We think that our case is unique according to the best of our knowledge for the following reasons:

• This is a first documented case of a patient having LM and aortic root thrombus after (HeartWare) HVAD implantation as a DT (the previous case reports were BTT). This is a youngest documented patient to have early complication with CF-LVAD despite full antiplatelet and anticoagulation coverage, and after exclusion of any blood clotting diseases. His presentation with polymorphic VT then complete heart block without obvious cardiac enzyme leak and his other laboratory results were not compatible with the thrombus formation.

• In a nutshell, early diagnosis and management of aortic root and LM coronary artery thrombosis post LVAD implantations is necessary in improving post-operative outcome. Moreover, high risk surgical interference in such sick patients can be avoided by early interventional management.

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