



Angiodysplasia Coagulation and Intraventricular Thrombolysis in Patient with Left Ventricular Assist Device Thrombosis: The Combined Strategy of Management of this Rare Patient

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Abstract

The Left Ventricular Assist Devices (LVAD) is considered a good way to improve survival and quality of life in patients with end stage left ventricular failure compared to the optimal medical therapy alone. Long-term survival of patients receiving LVADs may be complicated by different cardiac (heart failure and arrhythmia) and extra-cardiac diseases: Bleeding, infections, and thrombosis, many of which could require different intervention. Endoventricular thrombolysis may have a pivotal role in LVAD thrombosis. The coexisting risk of angiodysplasia gastrointestinal bleeding, often elevated in patient with this type of non pulsatile ventricular assistance device should be considered by single case.

Keywords: Left ventricular assist devices; Heart failure; HeartWare™; Thrombosis; Thrombolysis; Angiodysplasia

Introduction

The news Left Ventricular Assist Devices (LVAD) are considered a good way to improve survival and quality of life in patients with end stage left ventricular failure compared to the optimal medical therapy alone [1,2]. HeartWare™ (HeartWare International Inc, Framingham, Mass) is a third generation Left Ventricular Assist Device that generates up to 10 L/min of continue flow. The device inflow cannula is implanted in the heart apex, and the outflow cannula is implanted in ascending aorta through median sternotomy. The risk of pump thrombosis associated with the HeartWare™ LVAD is unknown [3-5]. We describe the management of a thrombotic HeartWare™ obstruction using intraventricular thrombolysis in patient with angiodysplasia.

Case Presentation

A 68 year old man (weight 62.7 Kg, height 160 cm) presented with a history of Chronic Obstructive Pulmonary Disease and duodenal ulcer. In 2001 was admitted for anterior acute myocardial infarction that require systemic thrombolysis and rescue PCI that failed to treat the culprit lesion. In 2011 episode of cardiogenic shock with evidence at echocardiogram of severe left ventricular dysfunction (Ejection Fraction, EF 20%), good right ventricular function, moderate mitral insufficiency, elevate left ventricular filling pressure and severe pulmonary artery systolic pressure (70 mmHg). After an ineffective attempt of stabilization with medical therapy, his hemodynamic condition persisted severely compromised (INTERMACS LEVEL II). Therefore, we proceeded to implant LVAD HeartWare™ as "Destination Therapy" in consideration of age and comorbidity conditions. After LVAD implantation, the patient's symptoms of dyspnea and fatigue improved, and he required only routine follow-up visits to the outpatient Heart Failure Clinic for

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Figure 1: Evidence of colon Angiodysplasia in pt with LVAD.

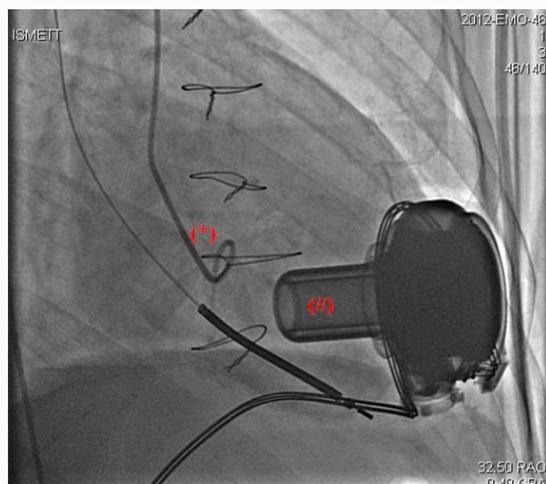


Figure 2: Right angle oblique plane cineangiographic image showing a 6F pigtail catheter (*) in close proximity to the HeartWare™ LVAD inflow cannula (#).

clinical and laboratory evaluation. After six months from discharge, since the patient experienced an episode of severe epistaxis, the range of anticoagulant therapy was reduced. A few days later he was urgently readmitted for acute episode of “right” heart failure with peripheral edemas, ascitis, and dyspnea. The arterial pressure recorded by invasive monitoring was 85 mmHg/60 mmHg (mean 68 mmHg). The estimated LVAD flow was more than 10 L/min, and power at 4.1 W, suggestive of LVAD thrombosis. The patient’s usual parameters at that speed and hematocrit level were a flow of 5.6 L/min, and power at 3.2 W. The patient was admitted to the Cardiothoracic Care Unit and started furosemide intravenous (i.v.) to treat fluid overloaded. In order to avoid thromboembolic events, i.v unfractionated heparin was initiated, with a target activated partial thromboplastin time of 50s, taking into consideration the fact that the incidence of thromboembolism ranges between 10% and 30% in this pool of patients, with rapid normalization of LVAD parameter. After eight days episode of melena and evidence of anemia. Laboratory tests revealed Hemoglobin (Hb) 8 g/dL, Hematocrit (Ht) 27%, WBC 8640/mL, serum urea 72 mg%, serum creatinine 2.2 mg% and N-terminal pro-brain natriuretic peptide 8334 pg/mL. The patient was transfused. Since colonoscopy showed a picture of ischemic colitis, we decided to discontinue his antithrombotic regimen with acetylsalicylate. After ten days new evidence of LVAD thrombosis occurs with clinical manifestations of low cardiac output and severe anemia. Therefore we performed enteroscopy the showed two angiodysplasia of colon

(Figure 1). It was decided a combined strategy made by angiodysplasia argon plasma coagulation and intraventricular thrombolysis (Figure 2) with Alteplase 30 mg. After 30 min of drug infusion we observed LVAD parameters normalization. Unfortunately in the days following a new episode of massive gastrointestinal bleeding required discontinuation of anticoagulant therapy causing another event of thrombosis of the VAD and exitus of the patient.

Discussion

Non-pulsatile flow may increase the development of Gastro-Intestinal (GI) angiodysplasia. Angiodysplasia, the most common vascular lesion of the GI tract, is a degenerative lesion of previously healthy blood vessels found most commonly in the cecum and proximal ascending colon. The vessel walls are thin, with little or no smooth muscle, and the vessels are ectatic and thin [6]. An association between aortic stenosis and chronic GI bleeding was first reported in 1958 and has come to be known as Heyde’s syndrome [7]. The narrow pulse pressure that occurs in aortic stenosis and in non pulsatile devices may increase intraluminal pressure and dilate mucosal veins, in 2002 [8], Warkentin et al. [9] proposed that this bleeding disorder may be explained by acquired type IIA von Willebrand’s syndrome, a deficiency of High Molecular-Weight (HMW) multimers of von Willebrand Factor (vWF). In severe aortic stenosis, accelerated breakdown of the large vWF molecule by its natural enzyme occurs in areas of high shear stress near the stenotic valve. Further studies found that after aortic valve replacement, HMW vWF multimers levels rise and GI bleeding stops [10]. The continuous impeller mechanism of the rotary LVAD pump may result in vWF deformation, proteolysis, and ultimately deficiency of HMW vWF multimers. Patients with preexisting GI angiodysplasia would be at risk for GI bleeding under these conditions. Decreasing non pulsatile device flow could result in pulsatile blood flow and potentially decrease vWF deformation and proteolysis. Restoration of HMW multimers of vWF levels in this way could prevent or resolve GI bleeding. The thrombosis of a ventricular assist device (0.02 events/patient/year) represents a potentially fatal complication. Management of device thrombosis includes device replacement or thrombolysis. Pump replacement is associated with high morbidity and mortality. Successful use of a glycoprotein IIB-IIIa inhibitor for the management of cf-LVAD thrombosis has been reported [11,12]; however, continuous infusion for 4 days was required for resolution.

Conclusion

Left ventricular assist device thrombosis is an uncommon complication that leads to pump failure and adversely affects outcomes after implantation. Diagnosis and treatment of this entity presents a significant challenge. Management of device thrombosis includes few therapeutic options. Despite being the definitive therapy, pump replacement is associated with significant morbidity and mortality. Different strategies are used in the treatment of axial flow LVAD thrombosis, including systemic or intraventricular thrombolysis. Intraventricular thrombolysis was used successfully with the axial flow pumps with a low risk of hemorrhagic events. The data on managing device thrombosis with these new centrifugal pumps are lacking. As in our case, the decision was made to treat the patient with a percutaneous approach based on the reduced risk of local therapy compared with systemic application. Our experience suggests that in the majority of case endoventricular thrombolysis may have a pivotal role, but the coexisting risk of gastrointestinal bleeding, often elevated in patient with this type of non pulsatile ventricular assistance device,

should however be considered by single case.

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