Deactivation of left ventricular assist device (LVAD) after recovery of cardiac function: a case report

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Abstract:

Recovery of heart function during support with durable LVAD is uncommon, and there are few reports of cases that address the issue of eliminating the LVAD without the need for a heart transplant. Radical surgical removal of the LVAD may cause distortion of left ventricular cavity and thus affect its function, in addition to the associated risks of the operation. Innovative ways to de-activate the LVAD, relying mainly on implantation of vascular plugs in the outflow graft have been used. Few reports have shown the success of this method. In this case report, we review the story of a young patient with advanced heart failure, who underwent LVAD implantation, and after 6-month, there was a dramatic improvement of heart function that enabled successful de-activation of the device.

Introduction:

The use of continuous flow left ventricular assist device (LVAD) is considered a cornerstone in the treatment of chronic heart failure in the advanced stages, and in some cases of acute heart failure. Studies in this regard have shown significant improvement in survival and quality of life compared to patients on optimum medical therapy alone.[1,2,3]

LVAD can be used as a bridge to recovery, bridge to heart transplantation and as a destination therapy. In few patients with the diagnosis Dilated Cardiomyopathy

(DCM) supported with long term LVAD, there has been reported cases of left ventricular recovery leading to removal of the mechanical support. However, the relapse rate into severe left ventricular failure is significant.[4]

We describe here a case with DCM supported by LVAD with left ventricular recovery and discontinuation of the mechanical support.

Case report:

A twenty-three-year-old male suffering from DCM and severe heart failure referred to our centre. Despite optimal medical treatment, the patient had repeated hospital admissions. There was a history of smoking and drug abuse.

Evaluation of cardiac function using echocardiography showed severe impairment of left ventricular function, ejection fraction (EF) < 15%, and the left ventricle end diastolic diameter (LVEDD) was 7.4 cm with moderate mitral and tricuspid valve regurgitation. Cardiac computed tomography (CT) revealed no coronary artery disease. Cardiac Magnetic Resonance Imaging (CMRI) confirmed severe LV enlargement and poor LVEF=9% and RVEF=16%.

Baseline right heart catheterization performed after clinical stabilization, showed normal right sided filling pressure (6 mm Hg), moderate pulmonary hypertension (mean pulmonary artery pressure 32 mm Hg), increased pulmonary artery wedge pressure (PAWp) 24 mm Hg, very low cardiac output (CI 1.6 l/min/m2), and calculated pulmonary vascular resistance (PVR) was 3.2 WU.

The case was discussed in a multidisciplinary team meeting. With a view to three hospitalizations within period of 6 weeks, intolerance to ACE inhibitors and beta blockers due to hypotension, frequent systolic blood pressure <90 mm Hg, previous

inotropic support and serum sodium <133 mEq/L, NYHA class IIIB, Intermacs stage 4(frequent flyer), the consensus was for proceeding with LVAD implantation.

Surgical implantation was performed using the Heartmate 3 (Abbott) device on cardiopulmonary bypass and beating heart with all measures for preserving the right ventricular function.

Intraoperative Trans-esophageal echocardiography (TEE) confirmed optimal positioning of the inflow cannula inside the left ventricle(Figures 2,3). Cardiac biopsy specimens reveal endocardial thickening by fibrosis, interstitial fibrosis and cardiac myocytes hypertrophy consisting with the diagnosis of DCM.

The patient was discharged home in stable condition with regular follow-up. During the follow up, a recovery of the functional capacity and end organ function were seen. However, after a couple of months the heartmate device started to have increasing rates of “low flow” alarms without any symptoms. These alarms became more frequent due to the recovery and remodelling of the left ventricle effecting the inflow cannula pointing towards and “touching” the antero-lateral surface of LV with a subsequent “sucdown event” (figure 3). TTE confirmed a recovery of the LV with LVEF on 40-45% and LVEDD=4.2 cm.

Right heart catheterization showed normal hemodynamic based on normal cardiac output and cardiac index obtained by “Fick + Thermo dilution methods” and performed under multiple conditions with simultaneous transthoracic echocardiography assessment of the LV. Repeated myocardial biopsy showed un-change findings.

Having very frequent low flow alarms, recovered LV function, normal fillings pressures and normal cardiac index, it was decided to discontinue the support. Initially, test-occlusion of the outflow graft was performed while monitoring the hemodynamics, then occlusion staring near to the LVAD pump outflow orifice and placing Vascular Plugs, two of Amplatzer vascular plug II size 20mmx16mm and two Amplatzer VSD muscular device size 12mm with additional attention to position of the last plug release within the conduit and before the anastomosis site to the Ascending Aorta to avoid protrusion into the aorta. Angiograms were performed at regular intervals and only trace blood flow was seen within the graft (figure 4). The LVDA drive line was exposed through its tunnel and shortened, caped and buried into the subcutaneous tissue.

The patient was discharged from hospital with unchanged LVEF (45%) and remained stable in the first 2 months post discontinuation period and was kept on anticoagulation (warfarin) to prevent thrombus formation inside the left ventricular apex.

Discussion:

The standard method of explanting the continuous flow LVAD devices is a complete removal of the device and closure of the left ventricular apex with a potential risk of left ventricular geometry distortion.

In order to decrease the left ventricular distortion after LVAD explanation, Cohn and associates reported a technique of felt plug insertion into the sewing ring of LVAD.[5] Schmitto and Potapov reported a successful less-invasive explanation of device using platinum plug in inflow cannula tract.[6,7]

For the same goal, another group presented a technique based on the transecting the inflow graft of the LVAD without the need to perform left ventricular dissection.[8]

Pettit et al. de-activated the Heartware LVAD by placing a percutaneous vascular plug at the proximal and distal ends of the outflow graft.[9]Others have successfully de-activated the Heartmate II LVAD by placing vascular plug only at the distal end of outflow graft.[10,11]

Percutaneous deactivation of the LVAD may allow rapid and durable correction of retrograde outflow graft flow and can also be used to deactivate the LVAD in inoperable patients. However, the need to anticoagulation is still required, but its period is still not defined. The effect of the device remaining within the pericardium and its long-term consequences is still unclear.

Conclusion:

Global experience is accumulating about the effectiveness of catheter based de-activation of the LVAD. It provides an alternative intervention to the radical surgical approach.

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Dr. Ulf Kjellman appointed by (Abbott) for a proctorship for the HeartMate III device.

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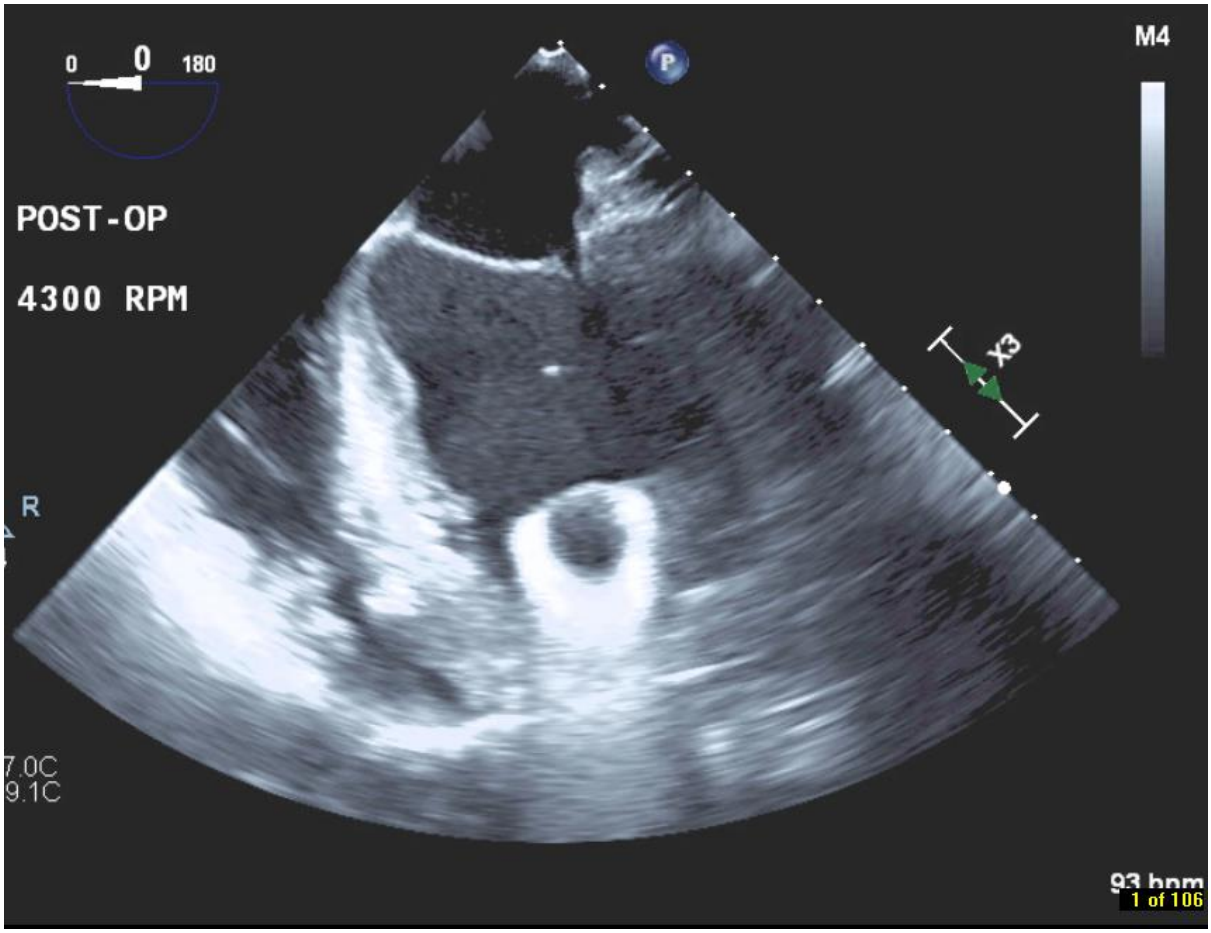

Figure1: A post-operative tran-esophageal echocardiography showing the relation of LVAD internal orifice in relation to the left ventricular cavity.

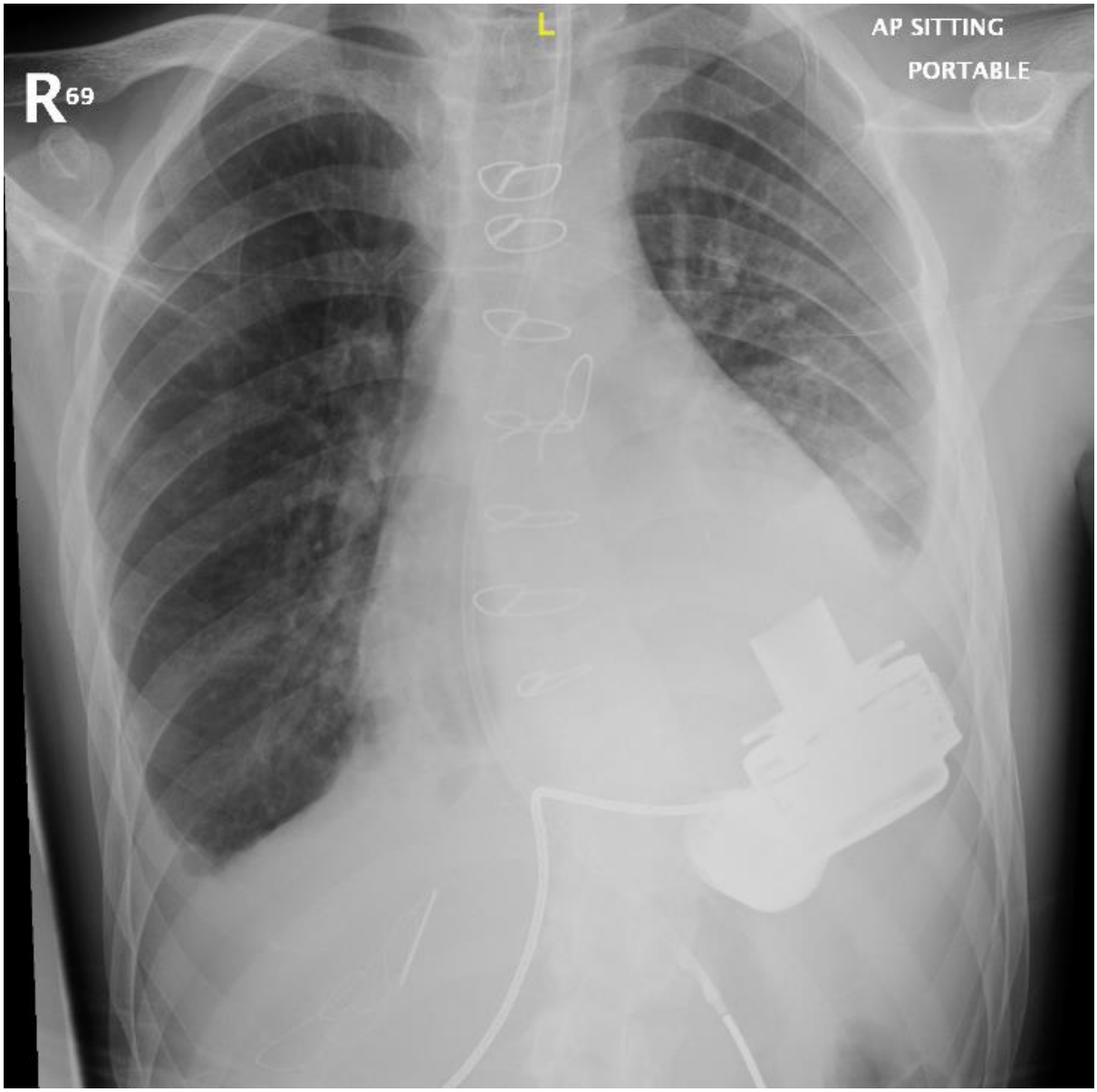

Figure2: Anterior posterior chest XR 5 days after the operation, note the distance between the inner cannula of the LVAD and the anterior wall of left ventricle.

Figure3: Coronal section of cardiac CT 6 months after the procedure, showing the improvement in the LV dimensions and collision of the anterolateral wall of the left ventricle with the inner cannula of the LVAD.

Figure4: Left anterior lateral view of the heart after vascular plug deployment.

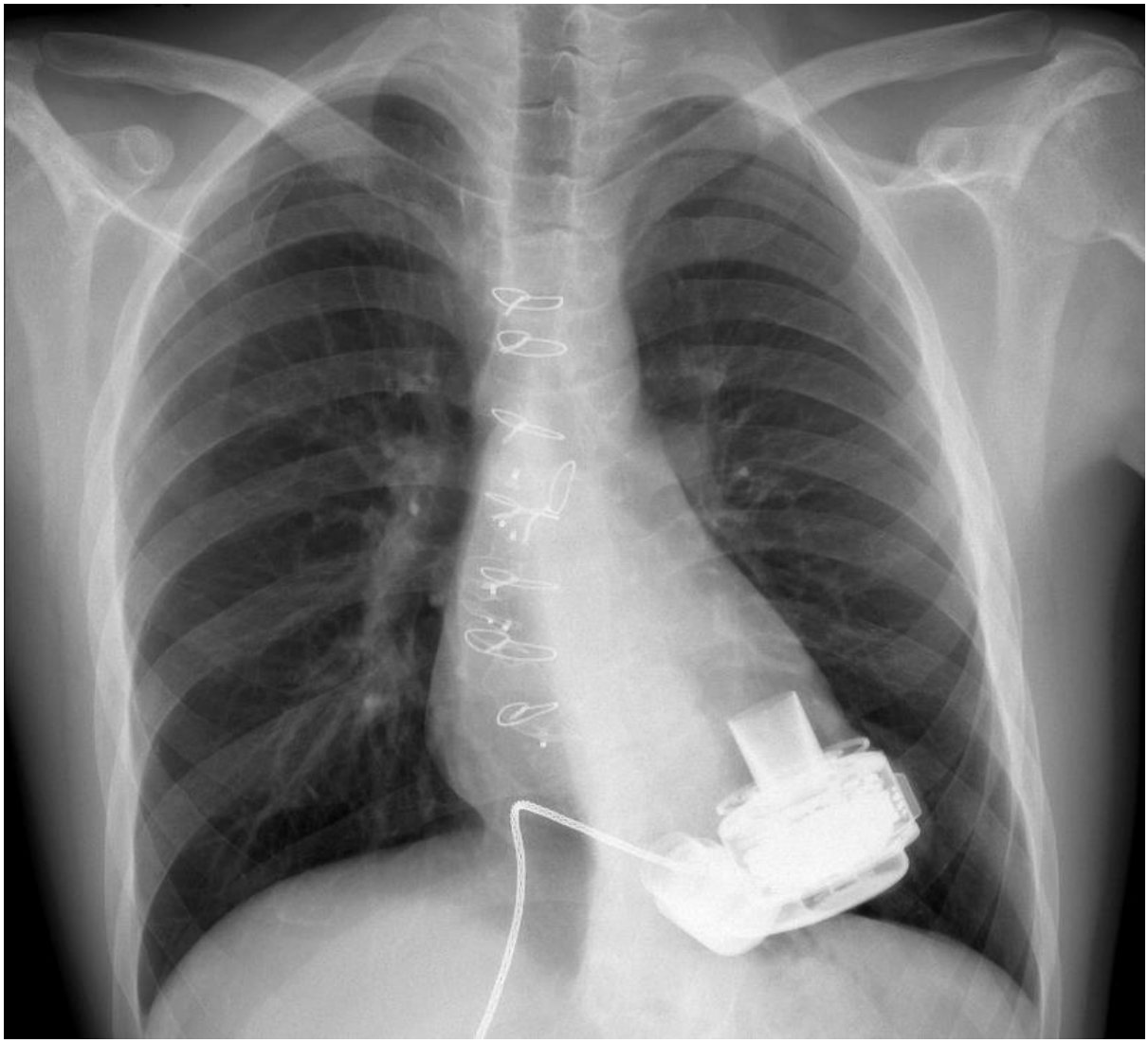

Figure5: A posterior anterior image of the chest a month after the procedure showing the normal dimensions of the heart.