

Letter to the Editor

Limited changes in severe functional mitral regurgitation and pulmonary hypertension after left ventricular assist device implantation: A clue to consider concurrent mitral correction?

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A 50-year-old Caucasian man underwent evaluation for left ventricular assist device (LVAD) implantation. He had a 15-year history of heart failure (HF) due to idiopathic dilated cardiomyopathy and permanent atrial fibrillation with several admissions for worsening congestive HF despite cardiac resynchronization therapy. Echocardiography showed severe dilation of the left ventricle (LV): end-diastolic diameter (EDD) 88 mm, end-diastolic volume (EDV) 350 ml with LV ejection fraction [EF] of 27%. Functional severe mitral regurgitation (MR) was associated with a huge left atrium (LA volume of 700 ml), and moderately decreased right ventricular (RV) function (tricuspid annular plane excursion [TAPSE] of 15 mm) with a moderate tricuspid regurgitation. Right heart catheterization (RHC) data are presented in the table. An increased pulmonary artery pressure (PAP: 65/28, mean 44 mm Hg) was observed. Pulmonary hypertension (PH) was only partially reverted with high dose of sodium nitroprussiate: mean PAP of 27 mm Hg. On the day of the RHC, pro-BNP level was markedly increased: 7660 ng/l and creatinine level was 2.3 mg/dl. The patient was implanted with a continuous-flow (CF) HeartMate II LVAD as bridge-to-candidacy indication. The early post-operative period was characterized by a prolonged state of pulmonary congestion, persistence of PH and difficult weaning from inhaled nitroxide. The patient gradually improved

and was discharged on post-operative day 20. Five months after LVAD implant, the patient was in functional class NYHA IIa without new admission for worsening HF. Echocardiographic assessment showed a persistently closed aortic valve, a mild reduction in LV dimensions (EDD 80 mm, EDV 328 ml), EF of 22% with a huge LA of 710 ml of volume and the persistence of severe functional MR due to posterior leaflet motion restriction associated with anterior leaflet pseudoprolapse secondary to papillary displacement and annular dilation. The RV was mildly dilated with significantly decreased TAPSE of 6 mm, but with only mild-to-moderate dysfunction by visual assessment (Fig. 1A–C and Videos 1–3 as online materials) and moderate tricuspid regurgitation. The MR volume was 108 ml, with LVAD speed of 9200 rpm, systolic systemic blood pressure (SBP) of 93 mm Hg, and heart rate (HR) of 85 beats per minute (bpm). The MR volume remained at 106 ml (Fig. 1D–F and Videos 4–5 as online materials) with increased LVAD speed at 10,000 rpm, mean at SBP 105 mm Hg, and HR at 90 bpm [1]. RHC showed (see Table 1) persistently increased PAP of 39/20, mean 30 mm Hg at the speed of 9200 rpm, which remained slightly increased at 10,000 rpm. On the day of the RHC pro-BNP level was 965 ng/l and creatinine level 1.4 mg/dl. The patient is currently on LVAD support as destination therapy.

LVAD therapy successfully decreases pulmonary hypertension, even in patients with “fixed” PH, allowing candidacy for heart transplantation, and among bridge-to-transplant candidates, the presence of pre-implant fixed PH does not reduce post-transplant survival [2]. The severity of the functional MR can affect the PH. Concomitant surgical procedures associated with LVAD implantation are burdened by increased operative mortality compared with isolated LVAD implantation [3]. Since LV overload is a major determinant of the severity of functional MR, and LVAD unloads the LV by promoting forward flow, MR is generally markedly reduced after LVAD implant. However, when left chambers are extremely dilated as in the reported case (Fig. 1G), LVAD unloading may not be sufficient to counteract functional MR. The CF-LVAD obtained a relative hemodynamic improvement, with persistent low output and PH, despite an appreciable clinical improvement and the preoperative evidence of reversible PVR-I. The outflow cannula was correctly positioned in the middle of the LV (Fig. 1D and Video 6 as online material). Increasing LVAD speed did not translate into further reduction of MR, although a slight improvement of the hemodynamic profile was observed, suggesting that in this case, due to extreme chamber dilation, MR was no more only “functional” but had a

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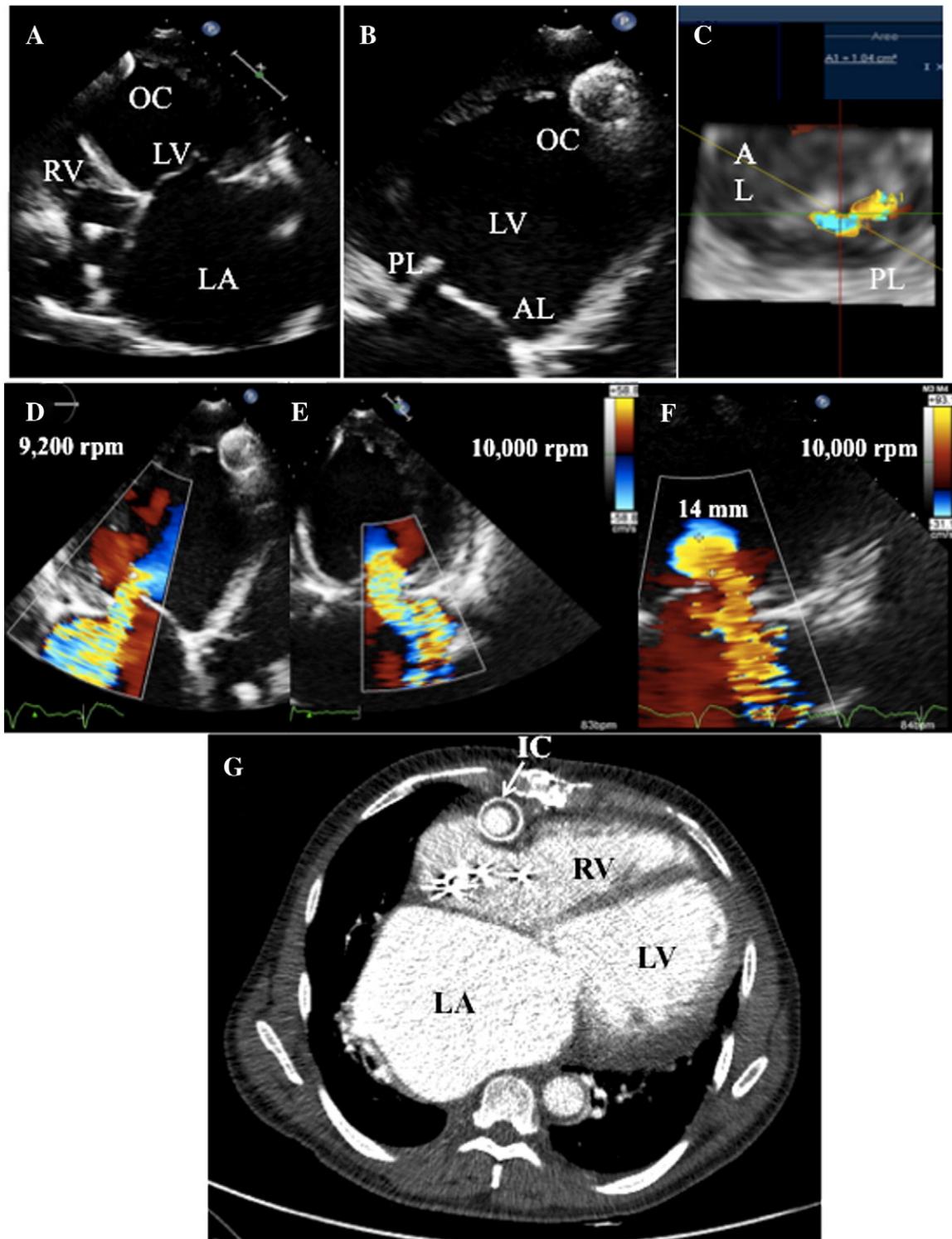


Fig. 1. Echocardiographic assessment after left ventricular assist device (LVAD) implantation: (A) the apical 4-chamber view showed persistently increased dimensions of the left ventricle (LV) and of the left atrium (LA) with mildly dilated right ventricle (RV). (B) The apical 3-chamber view showed the “fixed” posterior leaflet (PL) motion associated with anterior leaflet (AL) pseudoprolapse secondary to papillary displacement and annular dilatation (antero-posterior diameter 39 mm). (C) 3D image that shows the regurgitant orifice area (1.04 cm²) of the mitral valve. The outflow cannula (OC) was correctly positioned in the middle of the LV, excluding technical problem as possible cause of unsatisfactory LV unloading. Color Doppler images of the severe mitral regurgitation (MR) at LVAD speed of 9200 rpm (D), which remained unchanged at LVAD speed of 10,000 rpm (E). Quantification of the MR through the calculation of effective regurgitant orifice area (EROA) by the proximal isovelocity surface area (PISA) method showed a regurgitant volume of 106 ml at LVAD of 10,000 rpm (F). (G) Computerized tomography scan with contrast agent of the chest of the patient after 5 months since the implant showed extremely dilated left ventricular chambers. IC = inflow cannula of the left ventricular assist device. Artifacts and poor quality of the image are due to the presence of the device.

“fixed” anatomical substrate that could be corrected only with a surgical procedure, i.e. mitral valve repair or replacement with biological prosthesis. In conclusion, in similar case, when functional MR is associated with extremely dilated left chambers, concurrent correction of MR could be

considered at the time of LVAD implant, although an increased operative mortality could be expected [3]. It could decrease pulmonary congestion, achieving a better hemodynamic profile at the time of the subsequent heart transplantation. Some experience reports effective unloading of

Table 1

Right heart catheterizations before and after left ventricular assist device (LVAD) implant. CO = cardiac output; CI = cardiac index; PAP = pulmonary artery pressure; PCWP = pulmonary capillary wedge pressure; PVR-I = indexed pulmonary vascular resistance.

	Systemic systolic pressure (mm Hg)	CO (l/min)	CI (l/min/m ²)	Right atrium pressure (mm Hg)	Systolic PAP (mm Hg)	Diastolic PAP (mm Hg)	Mean PAP (mm Hg)	PCWP (mm Hg)	PVR-I (WU/m ²)
Pre-LVAD implant	100	1.8	1.1	14	65	28	44	31	11.8
Pre-LVAD implant + 3 µg/kg/min SNP	95	3.6	2.1	4	38	17	27	16	5.0
LVAD at 9200 rpm	100	3.2	1.8	16	39	20	30	19	6.0
LVAD at 10,000 rpm	93	3.6	2.1	12	37	15	26	18	3.8

LV in similar cases with the use of pulsatile LVAD, although it represents today a higher risk therapeutic option as compared to CF devices [4]. Alternatively, we speculate that surgical ventricular restoration, performed concomitantly to LVAD implantation, could improve LV unloading [5]. Cavity reshaping could reduce ventricular volumes and achieve a more physiologic position of papillary muscles of the mitral valve, both contributing to a further decrease of MR.

Supplementary data to this article can be found online at <http://dx.doi.org/10.1016/j.ijcard.2013.03.098>.

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