A 46-year-old nonsmoking male patient without significant medical history was referred to our intensive care unit for cardiogenic shock. Progressive dyspnea developed during the past 2 weeks. Heart rate was at 120 beats per minute and blood pressure at 80/60 mm Hg at admission. Physical examination revealed cool extremities with bilateral leg edema, jugular venous distension, systolic murmur, and S3 gallop. ECG showed sinus tachycardia without ST segment abnormality. Chest radiograph showed cardiomegaly and bilateral alveolar infiltrates. Bedside echocardiography revealed a dilated left ventricle (LV) at 80 mm and a poor LV function (LV ejection fraction measured at 10% and forward stroke volume at 27 mL) without valvular disease. Right ventricular (RV) function was normal (tricuspid annular plane systolic excursion at 22 mm and peak tricuspid tissue Doppler S wave at 12 mm/s). Renal and liver dysfunctions were found on laboratory data. Coronary angiography was normal. Cardiac MRI showed a dilated cardiomyopathy without perfusion abnormality or late gadolinium enhancement and a normal RV. The use of loop diuretic and inotropic support produced significant improvement in symptoms, hemodynamic status, and end-organ function. Because the patient could not be weaned from inotropic agents (INTERMACS [Intergroup Registry for Mechanically Assisted Circulatory Support] score at 3) and the risk to develop postoperative RV failure was low (MICHIGAN score at 0),1 LV assist device (LVAD Heartmate II, Thoratec Corporation, Pleasanton, CA) as a bridge-to-transplant was implanted under femoral cardiopulmonary bypass support. After sternotomy and usual field preparation, the pump was set below the left rectus muscle, anterior to the posterior rectus sheath without diaphragmatic section. The inflow cannula pointed posteriorly toward the mitral valve without angulation toward the septum or LV free wall, along LV axis. The outflow graft was pulled tight after precise estimation of length for attachment to the ascending aorta in an end-to-side fashion as described by Thoratec.2 Ischemic time was 32 minutes, and on-pump time was 68 minutes. The sealed outflow band relief collar was fixed just before sternal closure (Figure 1). Weaning from cardiopulmonary bypass support offered no difficulties without any inotropic support. Concomitantly, we proceeded to LVAD progressive start. Immediate postoperative transesophageal echocardiography displayed a normal RV function and a preserved native cardiac function. Acute hypotension and poor LVAD flow unresponsive to careful fluid resuscitation developed a few minutes later requiring high doses of inotropic therapy. Control transesophageal echocardiography was performed and showed an unexpected anterior tricuspid leaflet prolapse with severe regurgitation associated with an extrinsic compression of the RV free wall (Figures 2 and 3; Movies I–III in the online-only Data Supplement). Chest reopening revealed that the sealed outflow band relief collar was involved in the RV compression phenomenon and helped restore LVAD normal function. Removal of this component along with new positioning of the aortic cannula readily resulted in hemodynamic improvement, RV decompression, and complete resolution of tricuspid valve dysfunction as noted on transesophageal echocardiography (Figure 4; Movie IV in the online-only Data Supplement). Weaning from inotropic treatments was then possible. Postoperative course was simple. The patient is ambulatory at 3-month follow-up, awaiting heart transplant. Severe functional tricuspid regurgitation after LVAD implantation usually develops as a consequence of RV and tricuspid annular dilation and severe leftward shifting of the interventricular septum. This latter condition occurs in patients with excessive pulmonary vascular resistances and may be improved by the lowering of the LVAD pump speed, inotropic and nitric oxide support, and eventually needs right-sided mechanical circulatory support. In our patient, extrinsic RV compression by the device occurred at sternal closure and induced tricuspid leaflet prolapse and thereby severe acute tricuspid regurgitation compromising LVAD filling and output. Both the sealed outflow band relief collar and the outflow conduit passing over the RV have probably been the cause of compression of RV free wall, displacing the anterior papillary muscle of the tricuspid
valve and causing prolapse. To the best of our knowledge, this is the first case of acute reversible nontraumatic tricuspid regurgitation in the era of LVAD implantation. Repositioning the aortic cannula and removal of the sealed outflow band relief collar restored both RV and LVAD function. The purpose of this article is to highlight an unusual cause of reversible RV failure following LVAD implantation.

**Disclosures**

None.

**References**

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