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Regression of end-stage heart failure by left ventricular assist devices

Andrei-Dumitru MARGULESCU, MD

Department of Cardiology and Internal Medicine,
University Hospital of Bucharest, Romania

Non-ischemic dilative cardiomiopathy is associated with extensive remo-deling process of the left ventricle. This remodeling phenomenon leads to LV dilatation, increased concentricity, decreased systolic function and other structural changes that ultimately leads to overt systolic heart failure. The remodeling process can be delayed by several drugs, like angiotensin II converting enzime inhibitors (ACEI), betablokers (i.e. carvedilol), low dose spironolactone, and angiotensin II receptor blockers (ARB, also known as sartans). Several series also described reversed remodeling with prolonged unloading of the left ventricle with the use of a left ventricular assist device (LVAD). However, previous data showed that in less than one fifth of these patients, the LV functional improvement was good enough to permit explantation of LVAD.

A series of 15 patients was recently published where an English medical team evaluated if the use of LVAD together with maximal medical therapy in patients with end-stage heart failure due to non-ischemic dilative cardiomiopathy would lead to sustained improvement of the LV function and reduction of the severity of heart failure. Criteria for LVAD implantation included refractory heart failure, despite maximal medical therapy including inotropes, and signs of developing or ongoing multiple organ failure secondary to heart failure. All the patients included in the study received a LVAD.

After implantation, medical therapy known to improve remodeling was initiated with 100 mg daily of carvedilol, 40 mg daily of lisinopril, 25 mg daily of spironolactone, and 100 mg daily of losartan. After maximal decrease of the LV end-diastolic diameter (LVED), carvedilol was replaced with bisoprolol (a selective beta-1 blocker) and clenbuterol (a beta-2 agonist) was added. The LVAD was explanted if strict criteria would have been met, including a LVED < 60 mm and a LV ejection fraction (LVEF) $> 45\%$. At the beginning of the study the mean LVED of the patients was 69 mm and the mean LVEF was 12%. The patients were followed for a mean of 4 years after LVAD explantation. This could be achieved in 11 patients out of the total of 15, after a mean time of 320 days.

After explantation another two patients died, one of cancer. Therefore, for the survivors (9 out of 15), the survival rate after explantation was 100% at 1 year and 88.9% at 4 years. A

single surviving patient developed reccurrent heart failure, probably due to an episode of heavy alcohol abuse. In the case of the surviving patients, after 59 months, the mean LVED was 59 mm and the mean LVEF was 64%.

In **conclusion,** this series of patients shows that, in selected patients, combined therapy between LVAD and maximal treatment known to inhibit cardiac remodeling can induce sustained improvement of stage D, refractory heart failure and reversal of LV remodeling.

Comment on the paper:

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