

1053

Use of selexipag for pulmonary hypertension in a patient with durable LVAD: Is there a role for outpatient prostacyclin analogues in patients with LVAD and pulmonary hypertension?

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Background: Pulmonary hypertension (PH) group 2 is a frequent complication of advanced left ventricular (LV) failure. It has been demonstrated that LVAD implantation can be an effective treatment of PH group 2 in patients with advanced HF. Pulmonary vascular resistance (PVR) reduction in LVAD recipients might enable heart transplant eligibility, reduce morbidity due to chronic right ventricular (RV) dysfunction and prevent late RV failure. To date, the long-term use of pulmonary vasodilators in patients with durable LVAD in an outpatient setting for treatment of RV dysfunction remains uncertain. Limited data suggest a role of phosphodiesterase-5 (PDE-5) inhibitor and endothelin receptor antagonist (ERA) in patients with LVAD and elevated PVR who are candidates for a heart transplant. No studies have evaluated the use of prostacyclin analogs for outpatient treatment of PH in patients with implanted durable LVAD. We report a case of a patient with implanted HM2 LVAD treated with the combination of sildenafil and selexipag with the goal of optimizing RV function and reconsideration of transplant eligibility.

Methods: A 69-year-old male with a history of stage D, NYHA IV ischemic cardiomyopathy admitted for symptomatic hypotension due to low output cardiogenic shock requiring milrinone. He developed a cardiac arrest with pulseless ventricular tachycardia and subsequent worsening of cardiogenic shock. Left (LHC) and Right (RHC) cardiac catheterization revealed obstructive lesions in left anterior descending, right coronary artery and first obtuse marginal branch requiring placement of 4 stents and biventricular failure that required temporary mechanical circulatory support (MCS) with LV Impella device. Due to failure of optimal medical therapy and inability to wean off percutaneous MCS, the patient underwent implantation of HM2 LVAD as destination therapy (DT). His post-op course was complicated by worsening RV failure that required prolonged inotropic support with the inability to increase the LVAD pump speed above 8400 rpm and therefore increased risk of pump thrombosis. He was discharged to the rehab facility one month later, remaining on the same pump speed, off pharmacologic inotropic support.

Results: 40 days after discharge patient was deemed ineligible for a heart transplant due to elevated PVR. Repeated RHC at that time, while on sildenafil 80mg TID and LVAD pump speed of 8400 rpm, when compared to the initial RHC, showed decrease in RVEDP from 23 to 15mmHg, mean PA from 42 to 34 mmHg, PCWP from 25 to 13 mmHg, PVR of from 6.46 to 5.25 WU, while TPG and DPG were 21 and 7 mmHg, respectively (table 1). Treatment with selexipag was initiated with the goal to reduce PVR, improve RV function and achieve optimal pump speed to decrease the risk of pump thrombosis and to reevaluate the patient's transplant eligibility after 6 months of treatment. The patient remained on selexipag for the next 6 months with the highest dose being 400 mcg BID. Repeat TTE demonstrated a mild decrease in PASP to 48.4 from the RHC value of 55 mmHg, improvement in RV function from severe to mild hypokinesis. The LVAD pump speed was increased to 8800 rpm and was well tolerated. Up to this date, the patient remained symptom-free without new hospitalizations.

Conclusions: Addition of selexipag to sildenafil and LVAD produced a small reduction in PASP, improved RV contractility and achieved higher LVAD pump speed, without observed side effects. Selexipag, if studied in a larger scale trial, could serve as a useful addition to the armamentarium for treatment of CPC-PH and RV dysfunction in patients with LVAD with or without transplant consideration.



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