Central Sleep Apnea Interfering with Adequate Left Ventricular Filling in a Patient with Left Ventricular Assist Device

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CASE REPORTS

Central sleep apnea is common in patients with advanced heart failure. Apneic episodes are associated with hypoxemia, hypercapnia, and neurohumoral activation resulting in a rise in pulmonary vascular resistance. This case report describes a patient with a left ventricular assist device implanted for severe heart failure in whom unrecognized central sleep apnea resulted in under-filling of the left ventricle and a reduction in

left ventricular assist device inflow.

Keywords: Central sleep apnea, heart failure, left ventricular assist device

Citation: Schaffer SA; Bercovitch RS; Ross HJ; Rao V. Central Sleep apnea interfering with adequate left ventricular filling in a patient with left ventricular assist device. *J Clin Sleep Med* 2013;9(2):161-162.

Central sleep disordered breathing including central sleep apnea (CSA) and Cheyne-Stokes respiration (CSR) is a common finding in heart failure patients, with reported prevalences ranging between 25% and 75%.¹ Medical optimization, cardiac resynchronization therapy, and heart transplantation have all been shown to improve the CSA of heart failure.²

Mechanical circulatory support is being used increasingly in cases of advanced heart failure both as a bridge to transplantation and more recently as destination therapy. Left ventricular assist devices (LVAD) are associated with improved hemodynamic parameters, end-organ perfusion, and reduction in neurohumoral activation. However, the impact of LVADs on sleep disordered breathing is incompletely understood with conflicting results. A case series of heart failure patients with CSR showed persistent sleep disordered breathing after LVAD implantation despite improved hemodynamics.³ A case of a patient receiving biventricular mechanical support demonstrated resolution of CSA on a follow-up polysomnography (PSG), with later recurrence post-transplant.⁴

REPORT OF CASE

A 36-year-old male with a dilated cardiomyopathy following anthracycline-based chemotherapy for treatment of Hodgkin lymphoma was referred to our hospital for transplant assessment. His symptoms had progressed to NYHA functional class IV despite maximal medical therapy. On echocardiography the left ventricle (LV) was severely dilated with an ejection fraction of less than 20%. The right ventricle was mildly dilated with moderate systolic dysfunction.

Given the patient's decompensated state, he was admitted for tailored inotropic therapy with invasive hemodynamic monitoring. Right heart catheterization demonstrated pulmonary hypertension with a pulmonary arterial (PA) pressure of 68/43, right

atrial pressure of 13, and pulmonary artery wedge pressure of 20 mm Hg. The transpulmonary gradient was elevated at 31 mm Hg with minimal reversibility despite diuresis and a course of milrinone. Attempts to wean inotropes resulted in immediate recurrence of pulmonary congestion and rest dyspnea. Not eligible for transplantation candidacy due to elevated pulmonary vascular resistance, he underwent HeartMate 2 LVAD (Thoratec Corporation, Pleasanton, CA) implantation. There were no signs of right heart failure in the immediate postoperative period and the patient was easily liberated from inotropes.

Initial pump speed was set at 8,800 rotations per minute (RPM) with adequate flows. Transthoracic echocardiography confirmed adequate cannula positioning and left ventricular decompression without significant septal shift. Daily review of the LVAD event recordings demonstrated nocturnal drops of pump speed to 8,000 RPM and pulsatility index (PI) to < 4.0 with low flow alarms. Daytime readings remained within acceptable limits, with PI ranging between 5.4-5.9 and calculated flows 4.5-5.5 L/minute.

A review of his sleep history revealed a past diagnosis of CSA. Polysomnography performed 4 years earlier demonstrated an apnea-hypopnea index of 32.1 events per hour. Events were predominantly central apneas (index 21 events/h) with Cheyne-Stokes respirations. Mild snoring was noted, but there were no obstructive apneas. Continuous positive airway pressure (CPAP) at 6 cm H₂O abolished the central apneas. However, the patient stopped using his CPAP machine several months prior to current admission due to mask discomfort. With the re-introduction of nocturnal CPAP, there was normalization of pump speed, flow and pulsatility (**Figure 1**).

DISCUSSION

This case demonstrates the potential impact of sleep disordered breathing on LVAD function. Pulsatility index is a calcu-

Figure 1—Left ventricular assist device settings and readings at baseline, nocturnally with alarms and following the initiation of positive airway therapy

RPM, revolutions per minute; CPAP, continuous positive airway pressure.

lated measure of LVAD blood flow variability over a 1-second interval. This value reflects the degree of the left heart unloading and preload. In the setting of hypovolemia or right heart failure, the left heart becomes under-filled, and the pulsatility of blood flow across the LVAD drops, reflected as a low PI. In this case the patient was not hypovolemic, with persistent jugular venous distension and peripheral edema well into his recovery phase.

Cheyne-Stokes respiration is a type of central sleep apnea commonly seen in patients with heart failure. The mechanism of CSR is thought to include high ventilatory drive, lower apneic threshold during sleep, and long circulatory time. CPAP is effective in reducing CSR, possibly through increased PCO_2 during sleep, although improvement in hemodynamics may also play a role.⁵ CPAP has a myriad of potential beneficial effects on the myopathic heart, including reduction of LV afterload, increased LV ejection fraction, reduced myocardial oxygen consumption, increased RV systolic and diastolic function, and reduced PA pressures.² These improvements are mainly due to the direct effects of increased intrathoracic pressure. However, in patients with CHF and CSR, additional benefits may be from prevention of the adverse hemodynamic effects of sleep apnea.²

Sleep apnea is known to cause nocturnal elevations in PA pressure, possibly due to intermittent hypoxemia.⁶ In our case,

we postulate that the presence of CSA resulted in a rise in pulmonary vascular resistance interfering with normal pulmonary venous return and LVAD inflow. During wakefulness, right ventricular function was sufficient to maintain pulmonary blood flow and left ventricular filling. In the setting of apnea and increased pulmonary vascular resistance, the impaired RV was no longer capable of maintaining adequate output, leaving the LV under-filled and susceptible to the PI events. We cannot exclude the possibility that the effects of CPAP were from direct hemodynamic benefits of increased intrathoracic pressure. However, since LVAD dysfunction was strictly nocturnal, this suggests that the effects of CPAP were due to treatment of CSR.

Given the high prevalence of sleep disordered breathing in advanced heart failure, central sleep apnea should be considered in patients with unexplained PI events during sleep. In those without a prior diagnosis of sleep apnea, PSG should be performed to establish the diagnosis and test interventions aimed at reducing the frequency and severity of apneic episodes.

REFERENCES

1. Javaheri S. Sleep disorders in systolic heart failure: A prospective study of 100 male patients. The final report. *Int J Cardiol* 2006;106:21-8.
2. Arzt M, Bradley TD. Treatment of sleep apnea in heart failure. *Am J Respir Crit Care Med* 2006;173:1300-8.
3. Padeletti M, Henriquez A, Mancini D, Basner R. Persistence of Cheyne-Stokes breathing after left ventricular assist device implantation in patients with acutely decompensated end-stage heart failure. *J Heart Lung Transplant* 2007;26:742-4.
4. Vermes E, Fonkoua H, Kirsch M, et al. Resolution of sleep-disordered breathing with a biventricular assist device and recurrence after heart transplantation. *J Clin Sleep Med* 2009;5:248-50.
5. Eckert DJ, Jordan AS, Merchia P, Malhotra A. Central sleep apnea: Pathophysiology and treatment. *Chest* 2007;131:595-607.
6. Tilkian AG, Guilleminault C, Schroeder JS, Lehrman KL, Simmons FB, Dement WC. Hemodynamics in sleep-induced apnea. Studies during wakefulness and sleep. *Ann Intern Med* 1976;85:714-9.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication October, 2011

Submitted in final revised form May, 2012

Accepted for publication May, 2012

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DISCLOSURE STATEMENT

This was not an industry supported study. Dr. V. Rao is on the Scientific Advisory Board for Terumo Inc. The other authors have indicated no financial conflicts of interest.