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CASE REPORT

Resolution of Sleep-Disordered Breathing with a Biventricular Assist Device and Recurrence after Heart Transplantation

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Sleep-disordered breathing (SDB) is common in chronic heart failure. Both obstructive sleep apnea syndrome (OSAS) and central sleep apnea with periodic Cheyne-Stokes respiration (CSA-CSR) can occur. CSA-CSR is believed to correlate with heart function. Little information exists about the impact of mechanical assist devices and heart transplantation on SDB in patients with end-stage heart failure. Here, we describe, for the first time, the effects on SDB of a biventricular external assist device and of heart transplantation used successively in the same patient.

Keywords: Sleep-disordered breathing, chronic heart failure, ventricular assist device, heart transplantation

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Sleep disordered breathing (SDB) is common in patients with chronic heart failure (CHF) and may cause a further deterioration in heart function.[1,2] One study has shown that the apnea/hypopnea index (AHI) correlated with heart function (peak exercise oxygen consumption and left ventricular ejection fraction).[3]

Mechanical assist devices are effective in patients with end-stage heart failure and are awaiting transplantation.[4] They return hemodynamic parameters to normal, thereby improving end-organ function. However, little information is available about the impact of mechanical assist device treatment on SDB.[5] Likewise, the effects of heart transplantation on SDB are not well understood.[6,7] Here, we report, for the first time, the effects on SDB of mechanical assist-device therapy and heart transplantation used successively in a patient with end-stage heart failure.

REPORT OF CASE

A 52-year-old man was referred to our department in May 2005 for evaluation prior to heart transplantation. He had a BMI of 29.0 kg/m^2 and was a former smoker without pulmonary disease. In 1993, he experienced extensive anterior myocardial infarction, which led to CHF. At the time of the evaluation in 2005, despite optimal medication therapy (bisoprolol, losartan, furosemide, and spironolactone) and a biventricular pacemaker, he had severe symptoms (NYHA class IV). Coronary angiography showed severe inoperable arterial lesions. Echocardiography findings consisted of left ventricular dilation (end-diastolic diameter, 70 mm), severe left ventricular dysfunction (ejection fraction 20%), and severe systolic pulmonary hypertension (85 mm Hg) that was confirmed by right heart catheterization. He was listed for heart transplantation on May 23, 2005.

As part of his pretransplant evaluation, overnight polysomnography was performed in the laboratory (Embla N7000, ResMed, Lyon, France). We used standard criteria to assess sleep parameters including the AHI[8] for all recordings. The recordings showed both central sleep apneas with Cheyne Stokes respiration (CSA-CSR) and obstructive apneas, as well as inspiratory flow limitations and occasional snoring episodes.This mixed sleep apnea syndrome (SAS) was moderate, with an AHI of 21/h, a transcutaneous nocturnal oxygen (SpO_2) desaturation index (ODI) of 21/h, 193 minutes spent with SpO_2 below 90%, and a mean SpO_2 of 72% (Figure 1, panel A). Because he reported daytime sleepiness and had an Epworth sleepiness score of 13, nocturnal adaptive servoventilation was initiated. As expected,[9] this treatment improved both the CSA-CSR and the obstructive apneas, decreasing the AHI to less than 5/h.

A few weeks later, a biventricular external assist device (Thoratec, Berkeley, CA) was implanted and the nocturnal ventilation was stopped. Resting left ventricular assist device flow averaged 4.5 L/min and right ventricular assist device flow 3.8 L/min. Echocardiography with the device in place showed a decrease in left ventricular end-diastolic diameter to 55 mm with no change in left ventricular ejection fraction. Bisoprolol and spironolactone were started. One month after implantation, attended cardiorespiratory polysomnography (Embletta, Res-

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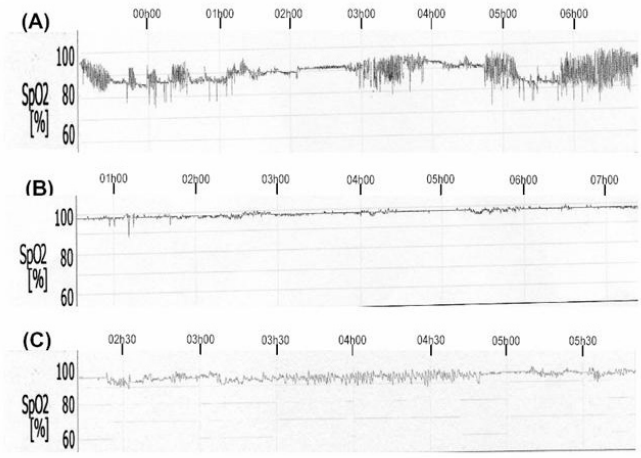

Figure 1—Nocturnal oximetry values before assist device implantation (panel A), immediately after assist device implantation (panel B), and 1 year after heart transplantation (panel C). Time is indicated above each trace (hh:min) and transcutaneous oxygen saturation (SpO_2) is shown on the left (%).

Med, Lyon, France) was performed, as presence of the assist device precluded in-laboratory polysomnography. AHI was 5/h. BMI was unchanged at 29.0 kg/m^2 (Figure 1, panel B).

Heart transplantation was performed 2 months later. After several postoperative complications, the patient was discharged from the ICU 2 months after the transplantation procedure. His treatment consisted of cyclosporine, prednisolone, mycophenolate mofetil, and amlodipine. Echocardiography showed good graft function (ejection fraction, 60%). Eight weeks later, despite substantial weight loss (BMI, 23.6 kg/m^2), cardiorespiratory polysomnography (Embletta, ResMed, Lyon, France) showed mild obstructive SAS (AHI, 13/h), with no nocturnal oxygen desaturation. One year later, the patient had gained weight (BMI, 28.1 kg/m^2) and had a left ventricular ejection fraction of 50%. Nocturnal polysomnography using the same device as before showed moderate obstructive SAS with an AHI of 29/h, an ODI of 28/h, 2 minutes spent with $SpO_2 < 90\%$, and a minimum SpO_2 of 87% (Figure 1, panel C). The patient refused nocturnal ventilation. A mandibular advancement splint was offered.

DISCUSSION

We describe for the first time, the impact of mechanical assist device treatment followed by heart transplantation on mixed SAS in a patient with CHF. The central and obstructive apneas resolved with the assist device but the obstructive apneas recurred after heart transplantation.

Our report is in contrast to a previous study reporting the persistence of central sleep apnea with Cheyne-Stokes respiration (CSA-CSR) after left-ventricular assist device implantation in 3 patients with end-stage heart failure.[5] However, our patient received a biventricular external assist device, whereas the patients in the earlier report received monoventricular assist devices.[5] The effect of heart transplantation on SAS is controversial. Complete resolution of CSA-CSR has been reported after heart transplantation.[10] More recently, OSA was found in 25%[6] to 36%[11] of heart transplant recipients. In most cases, weight gain was the identified risk factor for OSA, as with our patient.

Both OSA and CSA-CSR may occur in patients with CHF, either separately or in combination, as illustrated by our case report. Whereas OSA is believed to be a cardiovascular risk factor per se.[12] CSA-CSR may be a marker for CHF severity and prognosis.[13] CSA-CSR is common among patients with CHF.[1,14] The proportion of OSA versus CSA may change throughout the night[15] or from night to night. Thus, in a study of four consecutive nights done using the same recording device as in our patient, a shift from CSA to OSA or vice versa occurred in 8 (42%) of 19 men with CHF.[16]

Several lines of evidence suggest that OSA and CSA-CSR may share common pathophysiological mechanisms.[13] CHF is widely thought to cause CSA-CSR via oscillations of the central nervous system respiratory output, but central apneas have been found to be accompanied by upper airway occlusion.[17] Mechanical assist device therapy, by improving heart function, may influence upper airway patency via changes in ventilatory drive and circulation time, decreased edema, and modifications in neurohumoral control. These effects may have contributed to the resolution of the central and obstructive sleep apneas in our patient. The recurrence of OSA 4 months after the heart transplantation was probably due to weight gain. Whatever the mechanism involved, SDB should be treated, as it may cause transplant failure in heart transplant recipients.[18,19]

In conclusion, patients with CHF should be evaluated for SDB both after mechanical assist device implantation and after heart transplantation. Effective treatment is crucial.

DISCLOSURE STATEMENT

This was not an industry supported study. The authors have indicated no financial conflicts of interest.

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