CASE REPORT

Termination of Recurrent Ventricular Tachycardia by Continuous Positive Airway Pressure Ventilation

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We report a case of hypertrophic cardiomyopathy with recurrent ventricular tachycardia that resolved after initiating continuous positive airway pressure therapy.

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CASE PRESENTATION

A 72-year-old man with hypertrophic cardiomyopathy was admitted to the hospital for congestive heart failure (CHF) and frequent ventricular arrhythmias. His initial presentation, 5 years prior, was for sustained ventricular tachycardia (VT), for which an implantable cardioverter defibrillator (ICD) was placed. Medications included amiodarone and carvedilol, which were subsequently changed to sotalol and carvedilol as patient developed signs of interstitial pneumonia.

ICD interrogation on the day of admission revealed 162 episodes of nonsustained ventricular tachycardia (NSVT) and two episodes of sustained VT terminated by antitachycardia pacing. On admission, the temperature was 36.7°C, the blood pressure 104/58, the pulse 59 beats per minute, the respiratory rate 14 breaths per minute, and the oxygen saturation rate 96% while the patient was breathing ambient air. Physical examination was significant for elevated jugular venous pressure and trace bilateral leg edema. Inpatient therapy consisted of increased dose of carvedilol and sotalol as

well as diuretics. Symptoms improved and the patient was discharged. However, despite resolution of CHF, frequent NSVT persisted for 3 months after discharge. Importantly, a higher incidence of NSVT was noted at night (Fig. 1). Furthermore,

Figure 1. Twenty-four hour electrocardiogram monitoring showing runs of nonsustained ventricular tachycardia at different rates and morphology.

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Figure 2. Suppression of nonsustained ventricular tachycardia as detected by the implantable cardioverter defibrillator after initiation of continuous positive airway pressure ventilation.

a history of snoring and daytime somnolence prompted overnight polysomnography. This test confirmed severe central and obstructive sleep apnea syndrome.

Continuous positive airway pressure ventilation (CPAP) was initiated. After beginning CPAP therapy, an immediate decrement in NSVT was detected. In fact, since initiation of CPAP, no further ventricular arrhythmias were noted (Fig. 2).

DISCUSSION

We present a case of frequent ventricular ectopy, which resolved after initiation of CPAP. Patients with sleep apnea have an increased incidence of NSVT,¹ particularly during sleep.² In addition, patients with sleep apnea have a higher incidence of sudden cardiac arrest during sleep.³ Possible mechanisms include sympathetic surges⁴ related to hypercapnea, hypoxia,⁵ and nighttime repetitive arousal. Additionally, increased transmural ventricular pressure resulting from acutely elevated

systemic blood pressure and pulmonary hypertension may trigger arrhythmias in sleep apnea.

Sympathetic nervous system activity and associated ventricular ectopy in patients with sleep apnea can be reduced through the use of CPAP.⁶ Additionally, CPAP improves long-term cardiovascular outcomes mainly by reducing sudden cardiac death at night.⁷ The presence of an ICD in the reported patient uniquely illustrates the link between initiation of CPAP and resolution of life-threatening ventricular arrhythmias.

CONCLUSION

The present case demonstrates that sleep apnea can be the trigger for ventricular arrhythmias in susceptible patients. CPAP can reduce the incidence of these arrhythmias. Thus, clinicians should diligently screen patients with arrhythmias for signs and symptoms of sleep apnea and consider CPAP as adjunctive therapy for treatment of arrhythmias.

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