



The National Healthy Sleep Awareness Project involves a partnership between the American Academy of Sleep Medicine, Center for Disease Control and Sleep Research Society. The long term goal of the project is to promote improved sleep health in the United States. The project will increase public awareness of the importance of healthy sleep. It also will promote the treatment and prevention of sleep disorders.

Atrial Fibrillation and Sleep Apnea: A Case Report

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CASE

A 57 year-old man with a prior history of hypertension, hyperlipidemia, paroxysmal atrial fibrillation and gout presented to the emergency room with shortness of breath and palpitations. Examination demonstrated obesity (body mass index of 34.6 kg/m²), congested throat (Mallampati 4 score), irregular tachycardia, normal breath sounds, and no peripheral edema. Electrocardiogram confirmed the presence of atrial fibrillation with rapid ventricular response of 134 beats/min.

For the past 4 years prior to admission, he had recurrences of paroxysms of atrial fibrillation and has been under the care of a cardiologist. He has been taking metoprolol 25 mg BID, valsartan 160 mg, simvastatin 40 mg and aspirin 81 mg. He had previously declined systemic anticoagulation. Prior episodes were usually short-lived and were, on other occasions, triggered by stress or alcohol consumption. Diltiazem and metoprolol were used to acutely manage his symptoms; on one occasion, he required cardioversion. With recurring episodes, a catheter pulmonary vein isolation was proposed, but the patient declined. Echocardiograms showed left ventricular hypertrophy, but no valvular heart disease.

DISCUSSION

Atrial fibrillation (AF) is the most common serious arrhythmia. Its risk factors include advanced age, hypertensive heart disease, coronary artery disease and, less commonly in the United States, rheumatic heart disease. Atrial fibrillation leads to decrease in cardiac output and is associated with cardiomyopathy, heart failure, and secondary valvular changes. Also, it leads to *in situ* thrombosis within the atria, with a risk of systemic embolism including stroke.

Presence of obstructive sleep apnea (OSA) and central sleep apnea (CSA)

increase the risk of atrial fibrillation and this increased risk has been documented in a number of studies. For instance, in the Sleep Heart Health Study, compared to patients without sleep-disordered breathing, patients with severe obstructive sleep apnea had a four-fold increase in risk of atrial fibrillation, after adjustment for usual confounders.¹ It is thought that repeated, intermittent episodes of hypoxia and reoxygenation are responsible for this association between sleep-disordered breathing and arrhythmogenesis.^{2,3} Also, among patients <65 years of age, the risk of incident (new onset) atrial fibrillation was higher with coincident OSA, and increased with deeper desaturations at night.⁴ Taken from another angle, among patients with atrial fibrillation, there is a higher prevalence of obstructive sleep apnea, ranging from 30 to 80%.⁵

There is some evidence that treatment of OSA may reduce the risk of recurrence of atrial fibrillation. In a study by Kanagala et al., the risk of recurrence of AF over the 12 months of observation was higher in patients with untreated OSA than in matched patients with OSA controlled by continuous positive airway pressure (CPAP).⁶ In another study, among sleep apnea patients who underwent a catheter pulmonary vein isolation (ablation), the risk of recurrence of atrial fibrillation was higher in those without treatment than in those on CPAP (63% versus 28%). The risk of recurrence of AF among treated OSA patients was in fact, similar to the risk of recurrence in patients without OSA.⁷

Obstructive sleep apnea may also increase the risk of other arrhythmias. Patients with OSA have a higher risk of ventricular ectopy and ventricular arrhythmias. Based on the systematic review of literature, OSA was identified as a risk factor of ventricular tachycardia, ventricular fibrillation and implantable cardioverter-defibrillator shocks in patients with and without heart failure.^{1,8,9} Whether CPAP can improve this risk is an area of active investigation; at this point, there are insufficient data to demonstrate such an improvement.

In summary, OSA may be a modifiable risk factor for recurrent atrial fibrillation after cardioversion or ablation. Obstructive sleep apnea, which, if left untreated, may worsen cardiovascular outcomes. Clinical features suggestive of obstructive sleep apnea, such as history of snoring, witnessed apneas, obesity, excessive soft tissue in the upper airway should be systematically sought in patients presenting with atrial fibrillation, especially if they are accompanied by excessive daytime sleepiness and concurrent systemic hypertension. If these clinical features are present, a sleep medicine referral should be considered. In chronic care of patients with atrial fibrillation, in addition to typical rhythm- and rate-controlling

therapies and anticoagulation, treatment of concurrent obstructive sleep apnea is expected to result in an improved likelihood of remaining in the sinus rhythm and possibly, prevention of complications of this arrhythmia. Board certified sleep medicine physicians can help determine the appropriate diagnostic test(s), and provide long-term management with CPAP or other appropriate treatment modalities.

CASE FOLLOW-UP

Following the control of the ventricular rate with metoprolol, the patient's AF converted spontaneously to sinus rhythm. The consulting cardiologist recommended an outpatient catheter ablation and sleep evaluation. In addition to a comprehensive sleep evaluation, his polysomnogram demonstrated severe obstructive sleep apnea with an apnea-hypopnea index of 32, nadir oxygen saturation of 77% and high sleep fragmentation. CPAP applied during the same night (split night protocol) was successful in controlling airway obstruction at 12 cmH₂O, with mild residual central apnea that improved in the later part of the study. While it took several weeks for the patient to get used to his home CPAP unit, he is now successfully using his CPAP at home, with good symptomatic improvement and adequate control of OSA based on the adherence data monitor.

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