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Case report

Continuous positive airway pressure therapy converted atrial fibrillation in a patient with obstructive sleep apnea

Vichai Senthong, MD[a], Jarin Chindaprasirt, MD[a], Pattarapong Makarawate, MD[a], Panita Limpawattana, MD[a], Akkaranee Timinkul, PhD[b,c], Aonchuma Domthong, MD[a], Somchit Chumjan, RN[d], Verajit Chotmongkol, MD[a], Noppadol Aekphachaisawat, MSc[e], Kittisak Sawanyawisuth, MD, PhD[a,f,*]

[a] Department of Medicine, Faculty of Medicine, Khon Kaen University, Khon Kaen 40002, Thailand
[b] Faculty of Associated Medical Sciences and Back, Neck and Joint Pain Research Group, Khon Kaen University, Khon Kaen 40002, Thailand
[c] Back, Neck and Other Joint Pain Research Group, Khon Kaen University, Khon Kaen 40002, Thailand
[d] Outpatient Section, Srinagarind Hospital, Faculty of Medicine, Khon Kaen University, Khon Kaen 40002, Thailand
[e] Central Library, Silpakorn University, Bangkok 10200, Thailand
[f] The Research and Training Center for Enhancing Quality of Life of Working-Age People, Khon Kaen University, Khon Kaen 40002, Thailand

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ABSTRACT

Background: Obstructive sleep apnea (OSA) is one of the possible causes of atrial fibrillation (AF). Continuous positive airway pressure (CPAP) therapy may lower the recurrence rate of AF after cardioversion to normal sinus rhythm. We report a case of AF caused by OSA and successfully converted by CPAP therapy.
Case: A 65-year-old man presented with AF of unidentified causes. After severe OSA was diagnosed, he was treated with CPAP for 2 months and his cardiac rhythm returned to sinus rhythm without any antiarrhythmic drugs or cardioversion.
Conclusion: AF caused by OSA may be converted to sinus rhythm by CPAP treatment.

1. Introduction

Atrial fibrillation (AF) is a common cardiac arrhythmia and may be caused by obstructive sleep apnea (OSA). A previous report showed that AF occurred in 4.8% of the sleep-disordered breathing (SDB) group ($N=228$), whereas only 0.9% of those without SDB presented with AF ($p=0.003$) [1]. Other common causes of AF include hyperthyroidism, hypertension, and valvular heart disease, particularly mitral stenosis [2]. AF can be a contributing factor for various diseases such as cerebral or systemic embolism [3]. Conversion of AF is crucial to reduce cardiovascular morbidity and mortality [4,5].

OSA is a risk factor for hypertension and stroke in particular [6,7]. Continuous positive airway pressure (CPAP) treatment reduces deaths and cardiovascular events, and improves hypertension control [8,9]. The role of CPAP therapy on the treatment of cardiac arrhythmia is still limited. Here, we reported a case of OSA-induced AF and successful AF conversion by CPAP therapy.

2. Case history

A 65-year-old Thai man presented with dizziness. His physical examination was unremarkable except for an irregular pulse. His electrocardiography (EKG) result was compatible with AF (Fig. 1). He underwent laboratory examinations for causes of AF, including echocardiography (Table 1) and thyroid function test. The left atrium (LA) size was 32 mm. All investigations were normal. He was sent to a sleep clinic because of a history of snoring, daytime somnolence (Epworth Sleepiness Scale score of 18/24), morning headache, and AF. Physical examination revealed a body mass index of 30 kg/m^2, neck circumference of 42 cm, Mallampati class 4, no tonsillar enlargement, no retrognathia, and no torus. His sleep was monitored using a portable monitoring device (Embletta)®. The apnea-hypopnea index (AHI) was 63.6 times/h (supine AHI, 64.5 h^{-1}; non-supine AHI, 63.5 h^{-1}). Details of AHI values are provided in Table 2. Other polysomnographic data were as follows:

Total recording time: 8 h 30 min (510 min).
Position: supine 81.9 min (16.4%), non-supine 417.8 min (83.6%); upright time: 2.4 min (0.5%); movement time: 8.0 min (1.6%).
Oxygen saturation: mean oxygen saturation, 92.4%; oxygen desaturation events (OD), 162 (19.4 h^{-1}); lowest oxygen saturation, 83.0%; oxygen saturation $<90\%$, 60.4 min (13.4%).

* Corresponding author at: Department of Medicine, Faculty of Medicine, Khon Kaen University, Khon Kaen 40002, Thailand. Tel.: +66 43 363664; fax: +66 43 348399.
E-mail address: kittisak@kku.ac.th (K. Sawanyawisuth).

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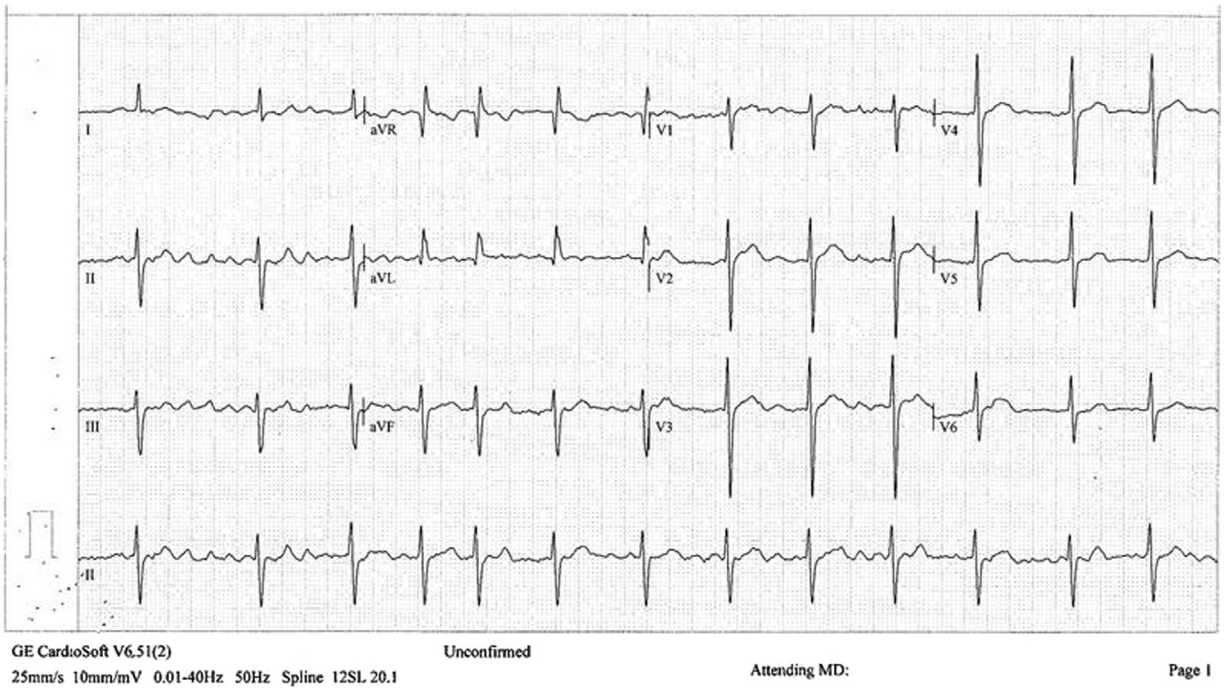

Fig. 1. Electrocardiogram showing atrial fibrillation before continuous positive airway pressure treatment.

Table 1
Echocardiographic findings by M-mode.

Variables	Values
Interventricular septal diameter, diastolic	14.00 mm
Left ventricular end diastolic diameter	47.00 mm
Posterior wall thickness diameter, diastolic	10.00 mm
Interventricular septal diameter, systolic	18.00 mm
Left ventricular end systolic diameter	23.00 mm
Posterior wall thickness, systolic	17.00 mm
Left ventricular mass	215.32 g
Left ventricular mass index	107.12 g/m^2
Relative wall thickness	0.43
End diastolic volume (Teich)	102.36 mL
End systolic volume (Teich)	18.00 mL
Ejection fraction (Teich)	82.42%
Aorta (AO)	34.00 mm
Left atrium (LA)	32.00 mm
LA/AO	0.94

Snoring time: 275.4 min (55.1%), number of snoring episodes: 355, mean snoring episode duration: 0.8 min, longest snoring episode: 10.2 min.
Plethysmogram: autonomic arousal 150 times (18.0 h^{-1}).
Heart rate (HR): total mean HR, 59.8 (53–121) bpm; supine HR, 59.8 (53–71) bpm; non-supine HR, 59.8 (54–121).

CPAP therapy was introduced, and treatment was successful. A CPAP pressure of 10 cm H_2O reduced the AHI to 3.2 h^{-1}. The patient had good compliance with CPAP therapy and reported refreshed sleep. At the 2 months follow-up examination, his pulse rate became regular with an EKG finding of a normal sinus rhythm (Fig. 2). Unfortunately, he developed left facial palsy due to squamous cell carcinoma of the inner ear 6 months later. He stopped complying with CPAP therapy owing to poor mask fitting. His cardiac rhythm returned to AF at 2 months after he stopped using CPAP. He died of brain metastasis.

3. Discussion

The condition of this patient was diagnosed as OSA-induced AF because no other possible cause was identified. OSA has been shown to be independently associated with AF development [10]. The prevalence rate of OSA-induced AF may be high as 50% [2]. There are several underlying mechanisms. Intermittent desaturation at night is the main mechanism of cardiac arrhythmia caused by OSA [1,11]. Nighttime hypoxemia from OSA causes atrial remodeling and dilatation, conduction abnormalities, vagal tone hyperfunction, pulmonary vasoconstriction/hypertension, or increase in inflammatory markers [11–16].

This case shows that effective CPAP treatment with good compliance may convert AF to normal sinus rhythm. CPAP therapy resolves all underlying mechanisms of AF induced by OSA. To our knowledge, there is no previous report on using CPAP to convert the AF cardiac rhythm to sinus rhythm in an OSA patient, without cardioversion or antiarrhythmic drugs. Previously, CPAP therapy was shown to reduce the recurrence rate of AF after successful cardioversion to sinus rhythm at the 12 months follow-up examination [17]. Unfortunately, the present patient was able to comply with the CPAP therapy for only a short period. Therefore, the long-term effects of CPAP therapy on cardiac arrhythmia were not evaluated in this patient.

Although attended polysomnography, which allows evaluation of sleep stages, was not performed in this patient, the portable sleep study was approved for the diagnosis of OSA in patients at risk [18,19]. Pro-brain natriuretic peptide and high-sensitivity C-reactive protein levels were not measured because of the normal echocardiogram and the absence of clinical features of heart failure, coronary artery disease, or other structural heart diseases. We believed that the AF in this patient was persistent and converted by CPAP therapy. The clinical symptoms of OSA and palpitations were also improved after CPAP therapy without any other treatments for AF. When the patient quit CPAP therapy, the AF recurred for >1 month, suggesting persistent AF. Therefore, CPAP therapy may convert AF to sinus rhythm in an OSA patient if