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Managing Sleep Apnea Early Prevents the Development of Heart Failure

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Abstract

The relationship between Obstructive Sleep Apnea (OSA) and Heart Failure (HF) is frequently observed in clinical practice. But abolition of coexisting OSA by Continuous Positive Airway Pressure (CPAP) in patients with HF has not been proved to the improvement of long-term outcomes. The strategy for the prevention of cardiovascular damage is the focus and diagnosis of OSA early. From this case, we can understand the pathophysiologic progress of OSA on cardiovascular system, and identify the important of diagnosis and interfering OSA before damaging cardiovascular system seriously.

Keywords: Obstructive sleep apnea; Central sleep apnea; Hypopnea

Introduction

The relationship between Obstructive Sleep Apnea (OSA) and Heart Failure (HF) is frequently observed in clinical practice. OSA results from upper airway collapse. In patients with OSA, on the one hand, blood pressure is frequently elevated as a result of sympathetic nervous system over activation, on the other hand, the negative intrathoracic pressure exaggerate during obstructive apneas, both increases Left Ventricular (LV) after load, reduces cardiac output, and may promote the progression of HF. Usually when OSA patients presented, he had been associated with cardiac insufficiency [1]. In patients with HF, abolition of coexisting OSA by Continuous Positive Airway Pressure (CPAP) have not been proved to contribute to the improvement of long-term outcomes [2], but the significance of identifying and managing sleep apnea early should be more emphasized to prevent the development or progression of HF.

Case Presentation

A 60-year old man presented for evaluation of chest tightness and short of breath for 4 months in July 2003. The review of systems showed that he snored for 20-year, and was diagnosed with sleep disorder in 1992 (Table 1), but he did not accepted CPAP treatment those years for doubting the effect of CPAP. Medical records of a decade were shown in Table1. In 2001, his Blood Pressure (BP) began to rise and he was prescript with valsartan 80mg qd to control BP to the normal, then he stop valsartan arbitrarily after one year being afraid of adverse reactions shown in package insert. Being doubted with Coronary Heart Disease (CHD) in that year, he got a coronary arteriography examination and found no coronary arterial atherosclerosis. He had no evidence of diabetes melites, chronic bronchitis, Chronic Obstructive Pulmonary Disease (COPD), and other diseases. He had been diagnosed with chronic heart dysfunction two months before and his medication list at initial appointment included digoxin 0.125mg qod, Benazepril 5mg qd, hydrochlorothiazide 50mg qd, and antisterone 40mg qd, and keep the symptom stable. Until that month, he felt symptom increased after getting cold. After admitted to our inpatients department, we found that his vital signs were stable, his BMI was 32.11Kg/m^2 and BP was 130/80mmHg. Physical examination indicated mild engorgement of the neck veins, wet sound in double lungs, expanded heart to the left and edema of both lower limbs. ECG tracing showed ST segment decrease on left ventricular leads and premature ventricular contraction. Chest radiographs suggested double pulmonary congestion. UCG revealed dilatation of the Left Ventricle (LV) and diffuse hypokinesis with the following measurements: LV Diastolic Dimension (Dd) of 81 mm, systolic dimsion (Ds) of 70mm, and an Ejection Fraction (EF) of 38%. Abdominal B-type ultrasound scan reminded hepatomegaly, but there is no ascites. Sleep test shown the mixted apnea during sleep (Figure 1). At first, he was given combination of furosemidum 20mg IV bid with oral antisterone 40mg qd, digoxin 0.125mg qod and valsartan 80mg qd. After 6 days, he felt better, and all of the signs disappeared including engorgement of the neck veins, rales in lungs and edema of lower limbs. When titrated to achieve dry weight, he was given furosemidum 20mg qod, antisterone 40mg qd, digoxin 0.125mg qod, valsartan 80mg qd, carvedilol 3.125mg bid. Combining serious sleep apnea with AHI reaching 44.6 by sleep test (Table 1), he was treated by Positive End Expiratory Pressure (PEEP) with 4cm H_2O pressure, and then CPAP with 8±2 cm H_2O pressure. At last he was discharged to outpatient department and followed by us, and taking medication and using CPAP with 10±3 cm H_2O pressure for next 10 years, and kept his heart function stably. Although admitted to our hospital for worsen of heart function several times, he was recovery from heart dysfunction eventually, but in 2014, he dead of sudden death at home.

Discussion

This case report illustrates the clinical feature of OSA patient: a middle-aged man began to disease, one of the earliest Sleep-Disordered Breathing (SDB) is given priority to low ventilation, with the development of the SDB appeared OSA and Central Sleep Apnea (CSA) gradually. When CSA happened, it meant the occurrence of cardiac insufficiency. It took about 10 years to progress from SDB to cardiac dysfunction. With CPAP treatment for OSA in this HF patient, the heart function is relatively stable for 10 years.

There are fewer studies in which the effects of therapy of HF on OSA have been tested, but effects of OSA treatment on HF have

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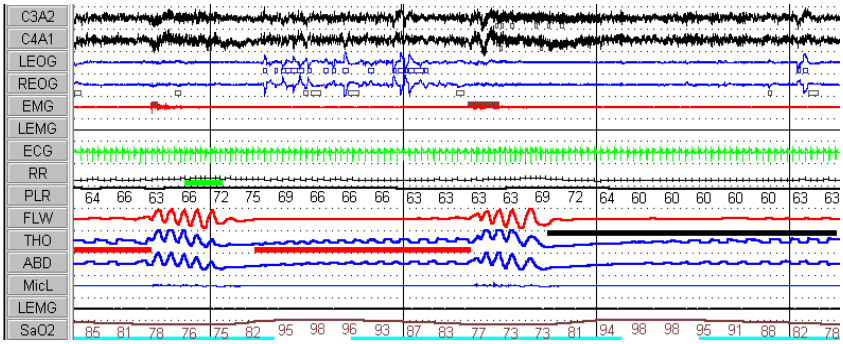

Figure 1: Sleep Test by PSG.

Table 1: Clinical datum during 1992-2003.

Year	Diagnosis	PSG			UCG			Holter
		AHI	TYPES	SaO2%	LVDs	LVDd	EF	
1992	PVC	63.4	HO	53				PVC
1998	Frequent Ventricular Arrhythmias	45.5	HO+OSA	59	38	52	46	BPVC
2001	Frequent Ventricular Arrhythmias Hypertension Cardiac Dilatation Dyslipidemia	52.7	HO+OSA+CSA	51	48	69	49	B/TPVC
2003	Frequent Ventricular Arrhythmias PVC B/TPVC VT PAF Hypertension Cardiac Dysfunction Cardiac Dilatation Dyslipidemia Diabete melites	44.6	OSA+HO+CSA	54	70	81	40	PVC B/TPVC VT PAF

BPVC: Bigeminy Premature Ventricular Contraction; TPVC: Trigeminy Premature Ventricular Contraction; PAF: Paroxysmal Atrial Fibrillation; VT: Ventricular Tachycardia; HO: Hypopnea; OSA: Obstructive Sleep Apnea; CSA: Central Sleep Apnea; UCG: Ultrasonic Cardiogram; LV: Left Ventricle; Dd: Diastolic dimension; Ds: Systolic dimension; EF: Ejection Fraction.

been studied. Theoretically CPAP alleviates OSA, abolishes negative intrathoracic pressure swings, reduced sympathetic vasoconstrictor activity and reduces nocturnal BP and HR, increased baroreflex sensitivity, resulting in reduced LV afterload [1]. But the differences in trial design, methodologies, patient characteristics, and the type of CPAP employed made some small sample randomized trial discrepancies in their results. Although promising, these results are not conclusive due to the nonrandomized nature of the studies and their small sample sizes. In such patients, indications for treating OSA have not been clearly defined [3,4]. Especially the recently published SAVE study remind us that prescribing CPAP with the sole purpose of reducing future cardiovascular events in asymptomatic patients with OSA and established cardiovascular disease cannot be recommended, although excluded patients with overt HF and patients with Cheyne-Stokes respiration in this study [5].

But we know it is a long time development from SDB to heart dysfunction if not confusing other factors such as diabetes mellitus. This natural process is about 10-year shown in our case. That means we have enough time to prevent heart construction and function ingravescence from damaging of SDB especially OSA on. So, an improved understanding of those mechanisms of OSA on cardiovascular disease could lead to improved strategies for the prevention of myocardial damage as well as early diagnosis of OSA. So, we can say that it is much more important intervening SDB before heart dysfunction rather than caring the effect of CPAP on the OSA patients with established cardiovascular diseases.

Conclusions

SDB especially OSA has adverse cardiovascular effects and is associated with reduced survival in patients with HF. Although small scale randomized trials demonstrate that treating OSA in HF patients improves cardiovascular function, such trials have not established whether treating OSA reduces mortality, which means large-scale randomized trials are needed. Accordingly, opportunities abound to advance knowledge in this field and to improve the outlook for the many patients suffer from OSA. The strategy for the prevention of cardiovascular damage including heart dysfunction is the focus and diagnosis of OSA early, which relay on the understanding to the pathophysiologic mechanism of OSA on cardiovascular system. We call all cardiovascular doctors to pay attention to the existence of OSA during their clinical practice and interfere OSA before damaging cardiovascular system seriously.

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