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ORIGINAL ARTICLE

Can continuous positive air way pressure reverse carotid artery atherosclerosis in obstructive sleep apnea?



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KEYWORDS

Continuous positive airway pressure;
Obstructive sleep apnea;
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Carotid

Abstract *Background:* Data from epidemiological studies and randomized clinical trials strongly suggest that obstructive sleep apnea (OSA) is associated with elevated risk of cardiovascular events. Although OSA and cardiovascular diseases share many risk factors, studies have demonstrated that OSA is an independent risk factor of arterial hypertension and atherosclerosis.

Objective: To determine the impact of treatment with continuous positive airway pressure (CPAP) on carotid artery intima-media thickness (CIMT) in patients with OSA.

Methods: 40 newly diagnosed OSA patients were assigned into two groups. Conservative treatment group (CT, $n = 20$) which refused CPAP treatment, and CPAP group (CPAP, $n = 20$) which received CPAP treatment. CIMT was determined at baseline and after 6 months.

Results: Mean follow-up time was 6.1 ± 2.1 months. At baseline, all measurements were similar in both groups and did not change significantly in CT group after 6 months. In contrast, a significant change occurred in CIMT in CPAP group ($8 (20)$ vs. $-115 (10) \mu\text{m}$, $p = 0.03$) from baseline.

Conclusion: CPAP significantly reduced CIMT in OSA patients.

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Introduction

OSA is a common sleep related breathing disorder characterized by repetitive upper airway collapse during sleep resulting in intermittent hypoxia, sleep fragmentation and sympathetic

over-activity. The condition affects all age groups and is prevalent across different populations globally [1].

Although OSA and cardiovascular diseases share many risk factors, studies have demonstrated that OSA is an independent risk factor of arterial hypertension and atherosclerosis [2].

The mechanisms of this relationship are incompletely understood. However, the evidence suggests that intermittent hypoxia and the arousal response are likely the main pathophysiologic factors associated with oscillation of systemic and pulmonary arterial blood pressures, heart rate, and cardiac function. These factors expose the heart and circulation to a

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cascade of noxious stimuli that, over time, may initiate or contribute to the progression of most cardiovascular disorders [3].

In everyday practice, attention is usually paid to atherosclerotic plaques in the vessels, typical of advanced atherosclerosis with organ dysfunction. However, atherosclerosis is a chronic, progressive, pathological process with a long asymptomatic subclinical phase [4]. In recent years, CIMT has been well accepted as a non-invasive tool which may predict the likelihood of acute coronary events and stroke in asymptomatic healthy subjects [4,5]. Therefore, assessment of CIMT as an early predictor for atherosclerotic changes seems to be a much better approach.

Ultrasonography—a noninvasive, quick, and reproducible technique—can be used to evaluate the atherosclerotic process at an early stage. CIMT correlates well with anatomic measurements and is a marker of both structural vascular damages and in prediction of the cardiovascular risk in asymptomatic healthy subjects [2]. Epidemiological and interventional studies frequently use CIMT as a surrogate marker for subclinical or early atherosclerosis [3].

CPAP is the treatment of choice for patients with symptomatic OSA, as it has been shown to improve daytime sleepiness, alertness and quality of life and to decrease blood pressure [6–8]. Whether CPAP treatment is effective in counteracting the autonomic imbalance and increased arterial stiffness in patients with OSA remains a matter of debate [9].

Aim of the present study

The aim of the present study is to evaluate the effect of CPAP vs. conservative treatment on CIMT over a period of 6 months in OSA patients.

Patients and methods

The present study was conducted as prospective observational study for the treatment effects on CIMT in 40 newly diagnosed OSA patients. OSA was defined by an overnight portable sleep study showing apnea–hypopnea index (AHI) ≥ 5 /h of sleep plus excessive daytime sleepiness or two of the following symptoms: choking or gasping during sleep, recurrent awakenings from sleep, unrefreshed sleep, daytime fatigue, and impaired concentration [10,11].

The exclusion criteria included patients known to have coronary artery disease or receiving lipid-lowering agents for dyslipidemia or previously diagnosed to have OSA.

Sleep assessment

Overnight diagnostic portable sleep study (Embletta gold, level III) was performed for every subject recording chest and abdominal wall movement by inductance plethysmography, airflow measured by a nasal pressure transducer [11,12]. Apnea was defined as cessation of airflow – or its reduction by 90% – for > 10 s and hypopnea as a reduction of airflow of $\geq 30\%$ for > 10 s plus an oxygen desaturation of $> 3\%$. Following confirmation of OSA, all patients were arranged to undergo overnight autoCPAP titration. All patients were given a basic CPAP education program by a respiratory technician supplemented by education booklet [11,12]. The technician would fit a comfortable CPAP mask from a wide range of selection

for every patient, who was then given a short trial of CPAP therapy with the Autoset (ResMed, Sydney, Australia) CPAP device for 3 days. Following the overnight autoCPAP titration study, each patient was interviewed and invited to participate in the serial CIMT study.

Conservative treatment group (CT, $n = 20$)

After confirmation of significant OSA and completion of overnight autoCPAP titration, 20 patients who were not keen to start CPAP yet were encouraged to (a) avoid sleep deprivation by having sufficient hours of sleep every night; (b) sleep in lateral positions; (c) avoid sedatives and alcohol consumption; and (d) lose weight by exercise and diet [13]. This group was labeled as conservative treatment group (CT).

CPAP group (CPAP, $n = 20$)

In addition to the usual advice as given to CT group, 20 patients had agreed to commence CPAP treatment for > 5 h/night after completing an overnight autoCPAP titration. They were subsequently prescribed CPAP device with a time counter recording machine run time. The CPAP pressure for each patient was set at the minimum pressure needed to abolish snoring, obstructive respiratory events, and airflow limitation for 95% of the night as determined by the overnight AutoSet CPAP titration study [11,12]. This group was labeled as CPAP group (CPAP).

Carotid artery intima-media thickness (CIMT)

CIMT was measured at baseline and 6 months for patients in both groups. The patients were followed up at the Respiratory clinic monthly whereas objective CPAP usage was measured from the time counter for CPAP group. CIMT was assessed by B-mode ultrasound scanning with compound and harmonic imaging to reduce the near field artifacts [14]. Bilateral CIMT measurements were obtained at the distal 10 mm of common carotid artery [15,16]. The CIMT was defined as the distance between the leading edge of the luminal echo to that of the media/adventitia echo (only the intima “echogenic layer” and the media “echo-poor layer” are included), and analyzed with a computerized edge-detection system. Carotid artery IMT was assessed by B-mode ultrasound scanning with a 12-MHz linear phase array transducer. Carotid IMT measurement by ultrasound was done using a semiautomated border detection program with validated accuracy. Three end-diastolic frames were selected, digitized, and analyzed for the mean IMT, and the average reading from these 3 frames was calculated for both right and left carotid arteries. The sole carotid scan operator was blinded to the clinical treatment status of the studied subjects and was not involved in the clinical assessment. Blood pressure (BP) was measured in the right arm after at least 15 min of rest using a standard sphygmomanometer before PSG and at clinic visits at 6 months.

Statistical analysis

The sample size was estimated by the Power Analysis and Sample Size for Windows software (PASS 2000, NCSS,

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