Case Report

Therapeutic Efficacy of Continuous Positive Airway Pressure in Obstructive Sleep Apnea Patients with Acute Aortic Dissection: a Case Report

Suguru Yamashita, Tomotaka Dohi, Koji Narui, and Shin-ichi Momomura

Cardiovascular Center, Toranomon Hospital, Tokyo, Japan

The coexistence of obstructive sleep apnea (OSA) may impose an additional risk on aortic dissection due to the possible increase in aortic transmural pressure. Thus, effective treatment for OSA, such as noninvasive positive pressure ventilation (NPPV), is thought to decrease the risk in patients with aortic dissection. We experienced one case of an OSA patient with aortic dissection who was successfully treated with continuous positive airway pressure (CPAP), resting and antihypertensive therapy. Few reports of this kind are available in the medical literature.

A 55-year old Japanese man with sudden chest and back pain was admitted to this hospital. Acute aortic dissection De Bakey type 3b was observed by radiography and the patient was treated successfully.

In cases with a high likelihood of OSA with aortic dissection, application of CPAP treatment should be considered promptly along with resting and antihypertensive therapy, except if there are complications such as comorbidities or withholding of consent.

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Key words; Obstructive sleep apnea, Noninvasive positive pressure ventilation, Continuous positive airway pressure, Acute aortic dissection

Introduction

There is increasing evidence that noninvasive positive pressure ventilation (NPPV) treatment such as continuous positive airway pressure (CPAP) reduces systemic blood pressure in patients with obstructive sleep apnea (OSA). It has been reported that OSA closely relates to the development of aortic dissection due to obstructive episodes of the upper airways with a marked increase in transmural pressure of the aorta wall¹⁾. Thus, demonstration of the efficacy and usefulness of CPAP treatment in stable cardiovascular and respiratory conditions for acute cardiovascular disease, such as aortic dissection, is needed.

Case Presentation

The patient was a 55-year-old man with main complaints of sudden chest and back pain. Although hypertension and hyperlipidemia had been identified for about five years, he had never received treatment. One evening on his way home, he suddenly experienced chest and back discomfort and was brought to our hospital by ambulance. In the ambulance, the discomfort progressed to severe pain in the same area. When he was admitted to our hospital, his face was contorted. Regarding physical findings, his height was 171 cm and weight was 89 kg. Because his body mass index (BMI) score was 30.3, he was considered to be obese. High mean blood pressure in his right arm, at 143 mmHg, was indicated. Hemochemical findings were all within normal limits, although the white-cell count was slightly high at 11,400/ μ L. Although the patient complained of extreme chest and back pain, the electrocardiogram was normal and ultrasonic cardiography showed no evidence of wall motion abnor-

Address for correspondence: Suguru Yamashita, Cardiovascular Center, Toranomon Hospital, 2-2-2 Toranomon, Minato-ku, Tokyo, 105-8470, Japan

E-mail: y0304@muse.ocn.ne.jp

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malinity in the left ventricle or severe aortic valve regurgitation. Thus, acute aortic dissection was suspected based on the typical symptoms and further examinations were performed. The origin of dissection was shown to be the aortic arch, just distal to the left subclavian artery to the bifurcation of the common iliac artery by thoracic and abdominal computed tomography (CT) scan using intravenous contrast (**Fig. 1**). Acute aortic dissection De Bakey type 3b was diagnosed. Accordingly, medical treatment was prescribed. Continuous 24-hour antihypertensive agent infusion, calcium channel blocker and β -blockers, was performed. Furthermore, OSA was highly suspected according to his physical findings and medical interview, including his thick neck circumference, mandibular retraction and daytime sleepiness. He also displayed breathing interruptions and snoring, which seemed to be associated with temporary complete obstruction of the upper airway and a subsequent decrease in oxygen saturation. Thus, CPAP treatment was initiated the first night after obtaining informed consent. After emergency admission, a slight increase in transient left pleural effusion was observed by chest X-ray film and a gradual increase in the inflammation value, indicating C-reactive protein and white blood cell count, was observed by blood examination; however, improvement was observed with proper mean blood pressure around 100 mmHg, urine volume and respiratory control (**Fig. 2**). With improvement in activity, treatment was shifted from continuous infusion to the oral administration of antihypertensive agents, such as calcium channel blocker, β -blockers and an angiotensin receptor blocker. In general, sufficient mean blood pressure could be maintained, around 100 mmHg, which ensured sufficient urine volume. CPAP was continued at a fixed 12 centimeters H₂O, which is the least amount to stop any snoring, and was adjusted by his bed-side every night while sleeping. The pressure was adjusted while referring to the cardiorespiratory monitor and the effect on pulseoximetry. The patient was given supplementary oxygen, especially during sleep, to maintain oxygen saturation above 90%. We postponed definite prevention of upper airway narrowing for cost effectiveness, etc; therefore, fixed CPAP instead of auto-titrating CPAP was chosen. The medical course at the hospital was very satisfactory and uncomplicated, except for phlebitis. Thus, the patient could be discharged less than one month later. We are confident that CPAP treatment is effective in the management of the acute phase of cardiovascular diseases, such as aortic dissection.

After discharge, the patient was admitted to the

Fig. 1. Contrast-enhanced computed tomography scan.

From a sagittal computed tomography scan taken on arrival, the origin of dissection was identified as the aortic arch, just distal to the left subclavian artery to the bifurcation of the common iliac artery.

sleep center of this hospital and polysomnography examination was performed. Minimum oxygen saturation during sleep was 75%, and the mean durations of apnea and hypopnea were 21.1 seconds and 23.8 seconds, respectively. According to a 2001 consensus report by a task force of the American Academy of Sleep Medicine, apnea is defined as the condition in which the patient stops breathing for more than ten seconds while sleeping, and hypopnea is defined as an abnormal respiratory event characterized by a 30% or more reduction in airflow, accompanied by oxyhemoglobin desaturation of at least 4%²⁾. Severe OSA was diagnosed due to very high apnea hypopnea index (AHI) and arousal index (AI) values as expected in room air. In addition, CPAP titration was examined and demonstrated that AHI and AI could be improved and deep sleep could be increased (**Table 1**). According to the results of an auto-titrating CPAP study, the lower and upper pressure limits were set at 4 and 12 cm H₂O as recommended. Thereafter, the patient received auto-titrating CPAP. Polysomnography with a fixed CPAP of 12 cm H₂O was not performed since auto-titrating CPAP has better acceptance and adherence than fixed CPAP.

Fig. 2. Clinical course of this patient.

Mean blood pressure (MBP) is defined as follows:

$MBP = \text{diastolic blood pressure} + (\text{systolic blood pressure} - \text{diastolic blood pressure})/3$

CPAP: Continuous positive airway pressure

Table 1. Results of Polysomnography

	Room Air	On CPAP
Sleep stage 1	76.7%	19.9%
Sleep stage 2	17.5%	43.2%
Sleep stage 3 + 4	0.0%	20.3%
REM	5.8%	16.6%
Arousal Index	41.4	18.2
AHI	30.5	6.9

REM: Rapid eye movement, AHI: Apnea hypopnea index

Discussion

Thoracic aortic dissection is an uncommon but potentially fatal disease with catastrophic complications, such as ischemic heart disease, obstructive pulmonary disease, and renal dysfunction. An investigation based on autopsies by S. Svensjo, *et al.* reported that the incidence of thoracic aorta rupture was 0.9 per 100,000 for men and 1.0 per 100,000 for women; the incidence of thoracic aorta dissection was 3.2 per 100,000 for both sexes³. R. Young, *et al.* reported that the incidences of rupture of dissecting aneurysms of the thoracic aorta and the entire aorta were both 75%⁴. Once rupture occurred, it became the direct cause of death in 41.2% of cases⁴; therefore, if acute aortic dissection is diagnosed, the necessary treatment must be provided immediately.

There is ample evidence confirming that resting and reducing blood pressure are important in the medical treatment of aortic dissection⁵. It is also a well-established concept that CPAP treatment significantly reduces blood pressure in OSA patients⁶⁻⁸. In this case, OSA itself was thought to contribute to the initiation and progression of aortic dissection because OSA resulted in systemic hypertension, an unstable respiratory state and unrest with repetitive arousal, etc. According to these symptoms, immediate treatment or relief of OSA in this patient suffering from aortic dissection, especially during acute management, was considered necessary. CPAP treatment was initiated and resulted in good control of the cardiovascular and respiratory conditions.

On the other hand, we could not find a prevalence of OSA in patients with aortic dissection or the rate of aortic dissection in patients with OSA; however, M. Kohler, *et al.* reported that the prevalence of OSA in patients with Marfan's syndrome was considerably higher than in matched control subjects⁹. He also suggested that OSA could be a risk factor for aortic root dilatation. Twenty of 61 (32.8%) patients with Marfan's syndrome had an apnea/hypopnea index (AHI) greater than 5 and 3 of 26 (11.5%) matched control patients had an AHI greater than 5 (*p* value 0.04). A possible underlying pathophysiological mechanism seems to be post-apnea reflex sympathetic activation and the subsequent marked increase

in blood pressure, producing shear stresses in the blood vessel walls, which are thought to cause direct vascular wall damage.

Additionally, P. A. Cistulli, *et al.* reported that two patients with Marfan's syndrome with coexisting OSA were treated with CPAP to attenuate aortic root dilatation¹⁰. They explained that OSA is characterized by repeated obstructed inspiratory efforts which generate progressively larger intrathoracic pressure swings until apnea is terminated. These rapid, repetitive, and major changes in intrathoracic pressure, coupled with the hemodynamic changes just outlined, could conceivably increase the risk of progressive dilatation in the presence of a weakened aortic wall. Consequently, CPAP treatment may decrease the risk of aortic dilatation and subsequent rupture with Marfan's syndrome with coexisting OSA. Based on these underlying pathophysiological mechanisms, CPAP seems to show some additional effect in the care of acute aortic dissection patients, especially those with coexisting OSA. At present, CPAP treatment for underlying OSA could not be shown to alter the clinical course after the onset of aortic dissection. We could only speculate that CPAP and conventional antihypertensive therapy would be more effective than antihypertensive therapy alone, since OSA may be related to the development of aortic dissection through several mechanisms. In the future, examination of whether treatment of OSA is involved in aortic diameter diminution is necessary. In connection, Serizawa, *et al.* recently suggested that OSA may contribute to increased thoracic aortic size through mechanical stress on the aortic wall due to repeated episodes of apnea and hypopnea, independently of systemic hypertension¹¹.

Moreover, G. Sampol, *et al.*, showed that patients with aortic dissection ($n = 19$) had a significantly larger AHI than patients with matched age, sex, BMI, blood pressure and neck circumference ($n = 19$), and that the number of aortic dissection patients with severe OSA, with an AHI greater than 30/hr, was significantly larger than that of the control group¹. He speculated that the coexistence of OSA may impose an additional risk of aortic dissection in predisposed patients or may result in worsening development due to the increase in aortic transmural pressure.

Conclusion

In patients with aortic dissection and where OSA is highly suspected, application of CPAP treatment should be considered as soon as possible, in addition to resting and antihypertensive therapy, unless there are contraindications, such as comorbidities or withholding of consent.

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