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Case Report

A daytime normotensive patient with nocturnal hypoxia-induced hypertension and severe obstructive sleep apnea

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ABSTRACT

This is the case of a 60-year-old male. He had no past medical history at a regular medical check-up. According to findings at the regular medical check-up, he was obese (body mass index, 32.8 kg/m^2), and had short neck, small jaw, and low soft palate; therefore, it was suspected that he may have sleep-disordered breathing. Blood pressure (BP) at the medical check-up was 121/80 mmHg, and the results of electrocardiogram and chest radiography were normal. Blood test data at the medical check-up indicated abnormality of lipid metabolism and hyperuricemia. No other abnormalities were found. It became clear that he became sleepy during daytime at an additional medical interview. Accordingly, he was diagnosed as having severe obstructive sleep apnea (OSA) with apnea–hypopnea index 65.3/h and arousal index 64.4/h by polysomnography. The oxygen-triggered nocturnal BP monitoring that was conducted at home around the same time indicated remarkable hypoxia-induced hypertension (Day 1: hypoxia-peak nocturnal BP 181/117 mmHg, Day 2: hypoxia-peak nocturnal BP 204/137 mmHg). The patient recognized the risk of OSA by visualizing the hypoxia-induced hypertension; therefore, introduction of continuous positive airway pressure (CPAP) therapy for severe OSA was smooth. As the results of CPAP therapy, we could confirm disappearance of hypoxia-induced hypertension.

<**Learning objective:** An office worker without cardiovascular disease was diagnosed as having severe obstructive sleep apnea. Remarkable hypoxia-induced nocturnal hypertension was identified by oxygen-triggered blood pressure (BP) monitoring. It was considered that nocturnal hypertension, which was not able to be recorded by previous ambulatory blood pressure monitoring, was recorded by the oxygen-triggered BP monitoring method.>

Introduction

Obstructive sleep apnea (OSA) is closely related to hypertension and abnormal daily blood pressure (BP) fluctuation [1]. The oxygen-triggered nocturnal BP monitoring is a method to measure BP including the following two functions: 1) an oxygen-trigger function that measures BP when oxygen saturation decreases by 10% from the baseline; and 2) a fixed-interval function that measures BP at a fixed interval, e.g. at 2:00, 3:00, and 4:00 am.

This method records BP surge caused by nocturnal hypoxia which cannot be measured by existing ambulatory BP monitoring (ABPM) [2,3].

Case report

The patient was a 60-year-old male office worker on the fast track. He had no specific medical history or treatment history. The patient was suspected to have OSA because he was obese with body mass index 32.8 kg/m^2 (weight 97.1 kg, height 1.72 m) with short neck, small jaw, and low soft palate. He had no history of smoking and was a social drinker. BP at the medical check-up was

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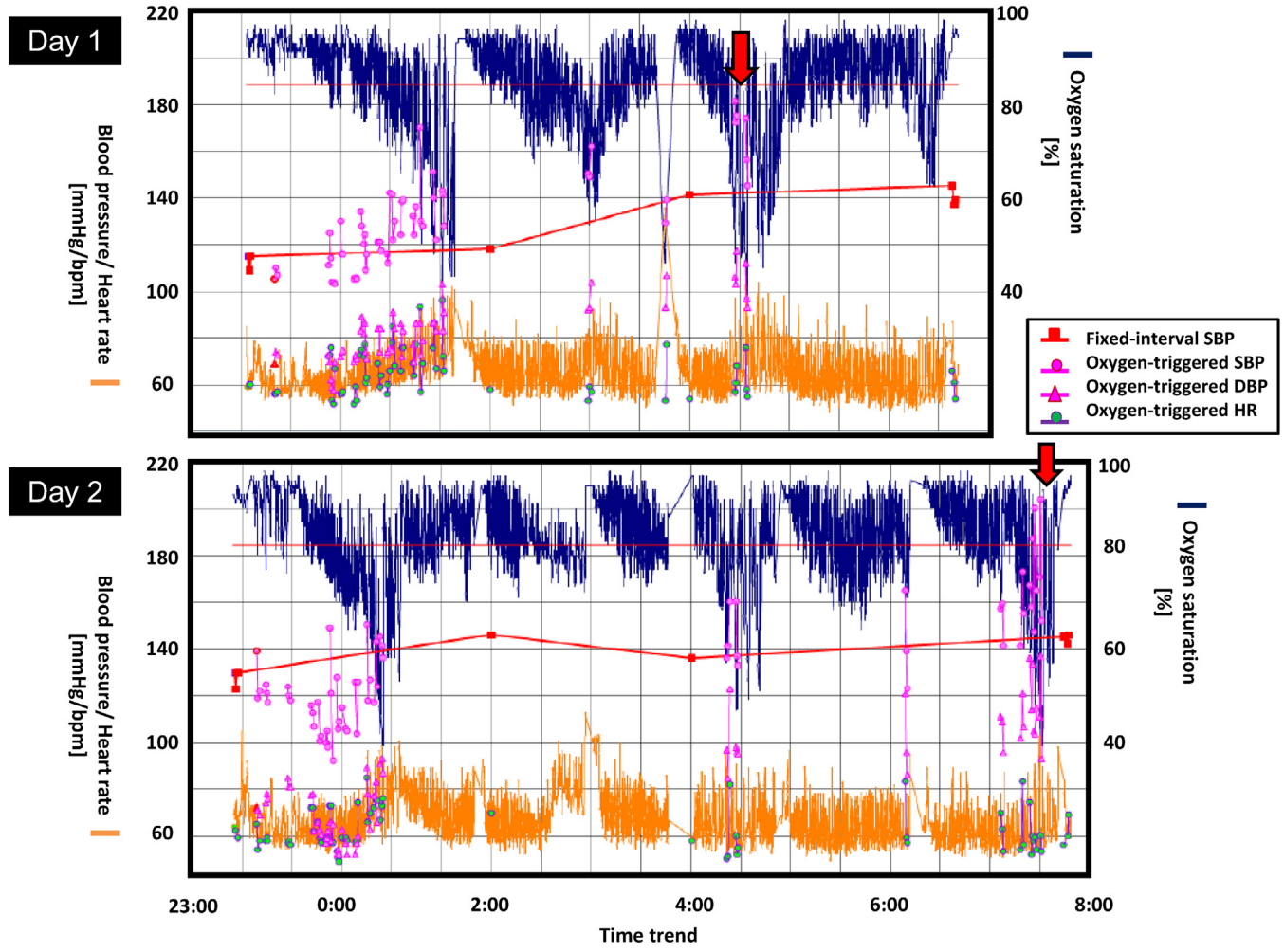

Fig. 1. The triggered nocturnal blood pressure (BP) monitoring before continuous positive airway pressure treatment.
The hypoxia-peak BP was 181/117 mmHg (red arrow) on day 1 and 204/137 mmHg (red arrow) on day 2. The mean fixed-interval BP was 142/91 mmHg on day 1 and 141/103 mmHg on day 2. The triggered BPs were measured when this patient's oxygen saturation fell below 84% on day 1 and 82% on day 2. The triggered BPs were measured 56 times on day 1 and 63 times on day 2.
SBP, systolic blood pressure; DBP, diastolic blood pressure; HR, heart rate.

121/80 mmHg, and the results of electrocardiogram and chest radiography were normal. Blood test data indicated abnormality of lipid metabolism (high-density lipoprotein cholesterol 32 mg/dL, low-density lipoprotein cholesterol 107 mg/dL, triglycerides 186 mg/dL) and hyperuricemia (8.2 mg/dL). No other abnormalities were found in the blood test. At an additional appointment after the check-up, his Epworth Sleepiness Scale was 20 points; therefore, polysomnography (PSG) was performed to provide a diagnosis of OSA. The apnea–hypopnea index was 65.3/h, apnea index was 52.1/h, slow-wave sleep was 0.0%, and arousal index was 64.4/h. Furthermore, endocrine and renovascular hypertension were negative. BP was also measured at home by oxygen-triggered BP monitoring for two nights at approximately the same time as BP measured on the day of PSG. On the first night, evening BP 116/66 mmHg, morning BP 140/102 mmHg, mean BP of 2 am and 4 am 142/91 mmHg, hypoxia-peak BP 181/117 mmHg, and HR 96 bpm; and the results of the second night measurement were evening BP 127/86 mmHg, morning BP 144/107 mmHg, mean BP of 2 am and 4 am 141/103 mmHg, hypoxia-peak BP 204/137 mmHg, and HR 85 bpm (Fig. 1). We told the patient that he should be indicated for continuous positive airway pressure (CPAP) therapy for severe OSA; however, he hesitated to introduce the therapy at first. Then, we showed him the increased nocturnal blood pressure from the oxygen-triggered BP monitoring record at home, and explained that CPAP therapy was strongly recommended to prevent cardiovascular diseases, and as a result we succeeded in introducing CPAP therapy. The patient used CPAP for 4 h and 52 min on average for about four months. After that, the oxygen-triggered BP monitoring at home indicated suppression of the remarkable hypoxia-induced hypertension although nocturnal hypertension (average nocturnal BP at 2, 3, and 4 am ≥120/70 mmHg) continued (Fig. 2). The results of the first night measurement were evening BP 150/103 mmHg, morning BP 146/102 mmHg, and mean BP of 2 am, 3 am, and 4 am 134/89 mmHg; and the results of the second night measurement were evening BP 143/99 mmHg, morning BP 130/95 mmHg, and mean BP of 2 am, 3 am, and 4 am 130/87 mmHg.

Discussion

Obstructive sleep apnea and hypertension

Despite regular medical check-ups, hypertension had not been diagnosed in this office worker for several decades. Patients with nocturnal hypertension are at high risk of developing cardiovascular diseases in spite of well-controlled BP during the daytime

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