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Clinical Case

Recurrence of stroke caused by nocturnal hypoxia-induced blood pressure surge in a young adult male with severe obstructive sleep apnea syndrome

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Abstract

Obstructive sleep apnea syndrome (OSAS) causes resistant hypertension and a hypopnea-related nocturnal blood pressure (BP) surge. This could lead to an increase of not only the nocturnal BP level but also nocturnal BP variability, both of which increase an individual's cardiovascular risk. We recently developed a trigger sleep BP monitoring method that initiates BP measurement when an individual's oxygen desaturation falls below a variable threshold, and we demonstrated that it can detect a BP surge during apnea episodes. We here report the case of a 36-year-old man with severe OSAS who experienced the recurrence of stroke due to nocturnal hypoxia and a nocturnal BP surge measured by this trigger sleep BP monitoring device. A nocturnal BP surge during sleep in OSAS patients could be a strong trigger of cardiovascular events. J Am Soc Hypertens 2016;10(3):201–204.

Keywords: Nocturnal blood pressure surge; obstructive sleep apnea syndrome.

Introduction

Obstructive sleep apnea syndrome (OSAS) is the most frequent cause of resistant hypertension, and OSAS increases the risks of stroke, heart failure, and premature death.[1–4] Clinical studies have demonstrated that nocturnal blood pressure (BP) and sleep BP variability are more important for predicting future cardiovascular events compared to awake BP and awake BP variability.[5,6] However, the existing strategies to assess BP variability including the BP surge associated with sleep apnea episodes in OSAS patients during sleep have been difficult to use clinically because neither previous home BP monitoring nor ambulatory BP monitoring could detect the peak sleep BP or the nocturnal BP surge. We developed a trigger sleep BP monitoring (TSP) method based on the automated fixed-interval measurement function with an additional oxygen-triggered function that indicates BP measurement when an individual's oxygen desaturation falls below a set variable threshold continuously monitored by pulse oximetry.[3,7,8] Here, we describe the case of an adult male who showed a prominent BP surge during sleep as measured by this TSP system and then had a recurrence of stroke. Our findings suggest that cardiovascular events in hypertensive OSAS patients with hypoxia-triggered nocturnal BP surges cannot be completely prevented even if their awake BP is well controlled.

Case Report

A 36-year-old Japanese male who had been diagnosed with hypertension at the approximate age of 10 years was referred to our hospital by his primary care physician for the further evaluation and treatment of high BP. He had

Conflicts of interest: The authors declare that there are no conflicts of interest related to this study.

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been given medication to treat hypertension because he experienced a lacunar infarction of the right corona radiate at the age of 32 years. However, after that, his condition was also complicated by a left putaminal hemorrhage at age 35 years. He also had some smoking history but did not drink alcohol to excess. His physical examination at the first visit revealed high BP (147/87 mm Hg) and overweight (body mass index, 32.9 kg/m^2). The other results of the physical examination were normal. The routine laboratory examination of blood showed dyslipidemia and hyperuricemia. His endocrinologic data, which could indicate secondary hypertension, were within normal limits. The electrocardiography results were normal, and echocardiography indicated concentric remodeling.

After the initial examination, we treated the patient with the daily morning administration of telmisartan 40 mg, amlodipine 5 mg, and bisoprolol 2.5 mg. One month later, his clinic BP values reduced to around the normal value (<140/90 mm Hg), but his early-morning home BP values remained high (156/114 mm Hg). We added bedtime dosing of amlodipine 5 mg, but the early-morning BP only slightly decreased.

We performed full polysomnography to examine the patient's possible sleep apnea syndrome (SAS) complication, because of his severe daytime sleepiness. The examination findings revealed severe OSAS as measured by his apnea hypopnea index of 75.5. To evaluate the patient's nocturnal BP and hypoxia-related nocturnal BP surge, we also performed an overnight TSP on three different discontinuous days (Figure 1). The trigger function of the TSP revealed almost the same night BP surges by hypoxic episodes. The level of systolic BP (SBP) surges peaked above 200 mm Hg, and the early-morning BP values were also very high compared with the clinic BP values. The patients attempted continuous positive airway pressure (CPAP) under our supervision, but he could not continue because of his lack of tolerance. One month later, he awoke one night during sleep because of a severe headache and right partial paralysis about an hour and a half after going to bed. He was brought to another emergency department and diagnosed with left putaminal hemorrhage (Figure 2). After conservative treatment and discharge, he made an effort and accordingly lost 20 kg of body weight. We then again evaluated his nocturnal BP and hypoxia-related nocturnal BP surge by overnight TSP for a comparison with his baseline values, and the results demonstrated a reduced nocturnal SBP surge (peaked to 155 mm Hg).

Discussion

Although in this patient's case the clinical BP had been almost within normal limits after intensive medical

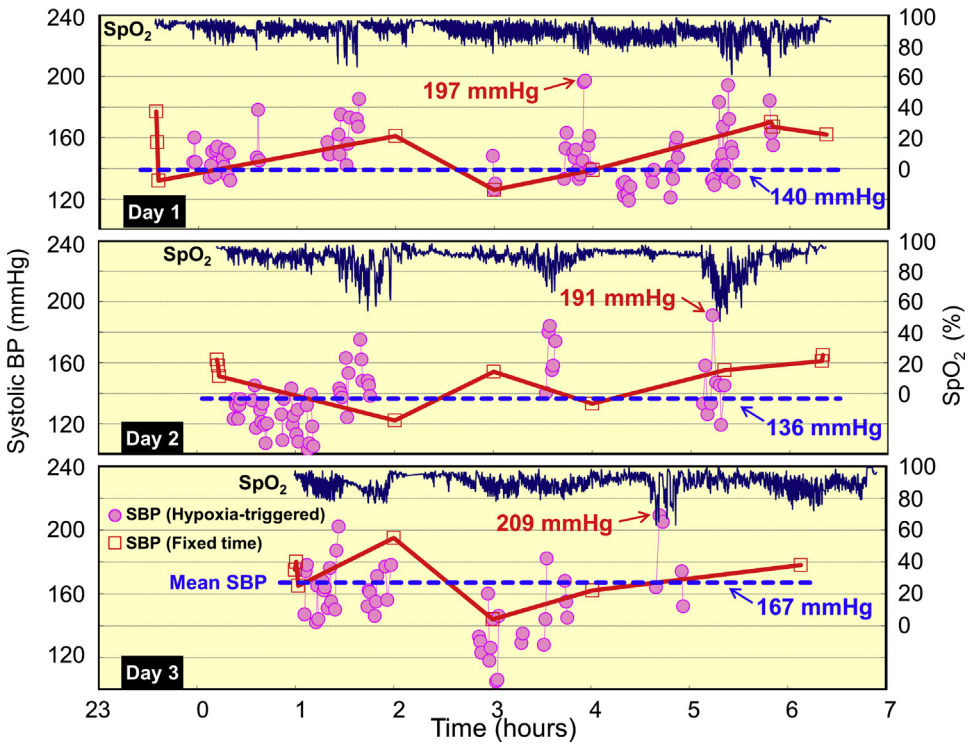

Figure 1. Time-trend data of the oxygen saturation and sleep SBP data on three different discontinuous days shown by the trigger sleep BP (TSP) monitoring method. Closed circles represent SBP measured by an oxygen-triggered function. Open boxes represent SBPs measured by the fixed-point function. The hypoxia-related nocturnal SBP surges were observed on all three days. SBP, systolic blood pressure; SpO_2, oxygen saturation monitored by pulse oximetry.