Sleep Apnea Patient with Sustained Ventricular Tachycardia During Polysomnography

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Standard in-lab polysomnography (PSG) is a relatively safe, common diagnostic procedure. Few reports have flagged cardiac events during PSG, but the recent study shows that adverse events during a PSG were uncommon. Here, we present a patient with obstructive sleep apnea who had a medical emergency (sustained ventricular tachycardia) during PSG.

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INTRODUCTION

Polysomnography (PSG) is a standard diagnostic instrument for recording and analyzing multiple physiologic parameters associated with sleep, breathing, movements, and heart rate and rhythm, in patients with diverse sleep complaints. The primary diagnostic intent of PSG involves monitoring of sleeping patients, since most patients have sleep-related disorder complaints. PSG is a common, relatively safe procedure, performed in various medical settings. A few reports emphasized that during PSG, cardiac events occurred in 18–48% of patients with obstructive sleep apnea (OSA).^{1,2} However, a recent large retrospective study revealed that adverse events during a PSG were relatively uncommon.³ Ventricular tachycardia is regular and fast heart rate that results from abnormal electrical activity in the ventricles of the heart. Ventricular tachycardia may lead to cardiac arrest, and eventually sudden death.⁴ Here, we report an OSA patient who had an episode of ventricular tachycardia and cardiac arrest during PSG, and we further discuss the adverse events of PSG and methods of promoting the safety of the patient during PSG.

CASE REPORT

A 48-year-old male visited the sleep clinic for the first time, with complaints of daily loud snoring and apnea. He presented with multiple medical problems, including hypertension, congestive heart failure, diabetes mellitus with complications, a chronic renal disease involving hemodialysis thrice a week, and history of intracranial hemorrhage. His chief complaints included difficulty falling asleep and frequent awakenings. Upon awakening, he felt unrefreshed, but denied any daytime sleepiness (Epworth Sleepiness Scale, 6). He had never smoked or drank alcohol. His physical examination revealed mild pitting edema in both lower extremities. His vital sign was stable, and his body mass index was 21.3 kg/m². A PSG was ordered to evaluate OSA, which was conducted one month later. He had received hemodialysis one day before the PSG study. On PSG, the patient had difficulty in falling asleep as usual. He complained of discomfort in both legs, along with accompanying lower back pain. The patient had taken one tablet of acetaminophen on his own. PSG showed a very short duration of sleep, highly fragmented

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sleep, and severe OSA (apnea-hypopnea index, 77/h). While struggling to sleep, he showed sudden generalized tonic posture with flexed right arm, and his head bent backwards. Electrocardiography (ECG) showed sustained wide complex ventricular tachycardia (Fig. 1). Cardiopulmonary resuscitation and defibrillation were initiated. Laboratory workup revealed hyperkalemia of 7.5 meq/L. He was admitted to the intensive care unit, and discharged after 46 days.

DISCUSSION

The American Academy of Sleep Medicine (AASM) proposed a single modified ECG lead II, using a body electrode placement in parallel to the right shoulder and left hip.⁵ A single lead II ECG is placed after determination of heart rate, recognition of rhythm, and detection of ectopy. In an adult, sinus tachycardia is defined as a sustained heart rate > 90 beats per minutes (bpm), and wide or narrow complex tachycardia as longer or less than 120 msec QRS duration. Wide-complex tachycardia should be scored for rhythms lasting minimum three consecutive beats at a rate of > 100 bpm, with QRS duration > 120 msec and the duration longer than

thirty seconds of tachycardia as sustained.⁶ The event shown in the current case qualifies as a sustained wide complex tachycardia, as the QRS is about 320 msec and the rate is 210 bpm, lasting over 30 seconds (Fig. 1).

Various electrophysiological signals obtained by PSG resemble those performed in inpatient environments or emergency centers. However, most sleep centers tend to evaluate patients who are considered medically stable without acute medical illness, and most sleep studies are attended by sleep technologist, and not nurses or physicians. Furthermore, PSG may be performed in facilities of various settings equipped for ill or physically challenged patients and environments, such as inside a hospital, those that are adjacent to and in acute care settings, or those operated as independent testing facilities not attached to any other medical assistance service.³

In a multicenter study, the incidence of adverse events occurring during PSG was reported as 0.35% of 16000 PSGs.⁷ Adverse events were defined as an episode identified by the technician, which occurred at the time of sleep study or during scoring PSG after sleep study, and requiring medical attention by the physician. Most adverse events were arrhythmias, half being ventricular in origin, and the arrhythmias were often linked with

Fig. 1. A: A 60-second epoch from overnight polysomnography. B: A 10-second window view of the ECG abnormality. Sustained monomorphic ventricular tachycardia is wide complex and without P-wave. ECG: electrocardiography.

sleep disordered breathings. The observed mortality rate was 0.006%. By contrast, a recent retrospective review of 36141 PSGs revealed that adverse events during a PSG were relatively uncommon (1 event/623 PSGs), and chest pain without arrhythmia and falls were the most common. It was concluded that the previous emphasis on the cardiac arrhythmias might be overstated, and improving patient safety should be sought by fall reduction during PSG.³

In the current case, some important issues need to be addressed for properly managing the patient. First, the patient had multiple co-morbidities such as hypertension, congestive heart failure, diabetes mellitus with complications, a chronic renal disease with hemodialysis, and a history of intracranial hemorrhage. Since this was the patient's first visit to the sleep clinic, a baseline workup such as routine laboratories, ECG, a chest X-ray was warranted. However, the patient denied workup because he was regularly receiving hemodialysis and had a check up in another hospital. Moreover, PSG was performed one month after the first visit. Therefore, the physician should have additionally warned the sleep technician to pay close attention to the patient. Second, the sleep technician treated the patient as medically stable, with sleep apnea. The overall level of care given to the patient by the sleep technician was not different from that given to other patients. He essentially observed the signals of sleep state, respiration, saturation and movements, and not signals from the ECG. Third, the sleep technician had thought the patient had seizure or syncope, and therefore the cardiac emergency had not been notified earlier. Fortunately, after the ventricular tachycardia was noticed, the cardiopulmonary resuscitation and defibrillation was successfully conducted since the study room was located near the intensive care unit and ward.

Although serious adverse events rarely occur during a PSG, sleep studies are usually evaluated for individuals having comorbid conditions such as hypertension, coronary artery disease, diabetes mellitus, and stroke.⁸ AASM recommends that cardiac events during PSG should be reported, including sinus tachycardia or bradycardia, asystole, wide or narrow complex tachycardia, atrial fibrillation, heart block and ectopic beats, if clinically significant.⁹ Regarding the influence of cardiac-related events, the prompt recognition and proper response to cardiac arrhythmias in real time require adequately trained and attentive sleep staff and suitable monitoring facilities. All members of the sleep center must have an ability to identify and treat common cardiac arrhythmias.⁶

The patient had shown a highly fragmented sleep and severe OSA during short sleep time; these disturbances might be significantly related and could have contributed to the cardiac event. Chronic severe sleep apnea and associated fragmented sleep adversely affects the conduction of the heart, leading to fatal arrhythmias.¹⁰ Repetitive apneas, hypoxemia, hypercapnia, adrenergic activation and increased cardiac wall stress induce conductive tachycardia-bradycardia oscillations and ultimately arrhythmia

events, in the absence of any significant abnormalities in the conduction system.¹⁰⁻¹² Moreover, a patient with heart failure or ischemic, or hypertrophic heart may be more susceptible.¹¹ In this case, ventricular tachycardia caused by hyperkalemia was reasonable, but OSA should be regarded as one of the hidden contributors.

Accreditation of the sleep center should aim to enhance quality and safety of patient care.^{8,9} The settlement of the Korean sleep accreditation standards should be endeavored. Accreditation should define a minimum level of sleep centers and a quality of monitoring equipment. Also, detailed sleep technologist, physician training and job descriptions should be described, as well as minimum guidelines should be followed, to enhance the safety of patients.⁹ Critical pathway managing unexpected urgent patients during PSG should also be established.

In patients without significant medical comorbidities and those with a high likelihood of having sleep apnea, the home sleep testing with portable devices might be a safe and ancillary option. However, critical events such as cardiac arrhythmias or seizures, might be missed.⁹

Physicians should perform a thorough medical evaluation before the PSG, and pay a high index of caution to patients at risk during PSG. Maintaining devoted physicians and sleep technologists can expect a small number of minor adverse events during PSG.

Conflicts of Interest

The author has no financial conflicts of interest.

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