Idiopathic Central Sleep Apnea Presenting as Cheyne-Stokes Breathing

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A 70-year-old man had complained of excessive daytime sleepiness and fatigue for 3 years. He underwent an overnight polysomnography followed by a multiple sleep latency test which showed central sleep apnea with Cheyne-Stokes breathing (CSB) pattern. Further evaluation including magnetic resonance imaging of the brain could not find any cause of this sleep-disordered breathing. We describe the rare case of idiopathic central sleep apnea presenting as CSB pattern.

Key Words: Central sleep apnea, Cheyne-Stokes breathing, Polysomnography.

Central sleep apnea (CSA) syndromes include those in which respiratory effort is diminished or absent in an intermittent or cyclic fashion due to central nervous system or cardiac dysfunction.¹⁴ These disorders are broken down into the idiopathic form and those with defined underlying pathologic or environmental causes. Cheyne-Stokes breathing (CSB) is characterized by a cyclic fluctuation in breathing with periods of central apneas or hypopneas alternating with periods of hyperpneas in a gradual waxing and waning fashion.¹² It is most commonly noted in congestive cardiac failure and neurologic disorders.¹² But we found central sleep apnea presenting as CSB pattern without specific causes.

Case Report

A 70-year-old male (body mass index: 21.9 kg/m²) visited our sleep disorder clinic for excessive daytime sleepiness and fatigue for 3 years, although he has slept 8-9 hours everyday. Snoring, cataplexy, hypnagogic hallucination, sleep paralysis and sleep attack was denied, but sleep apnea was observed by his wife. He had taken thiazide 1-2 times per week for hypertension and terazocin irregularly for benign prostate hypertrophy for 5 years. An overnight polysomnography (PSG) and a multiple sleep latency test (MSLT) were done after stopping these medication 2 weeks ago.

Physical examination including ear, nose, and throat were normal and neurological examination was normal. Blood tests, including thyroid function tests were normal except for slight elevation in liver enzymes. Arterial blood gas analysis showed pH 7.47, PaCO₂ 37.0 mmHg, PaO₂ 72.9 mmHg, HCO₃⁻ 27.3 mmol/L, Base excess 3.5 mmol/L.

Overnight PSG showed total sleep time of 465 minutes, sleep latency of 6.0 minutes, and sleep efficiency of 97.5%. Sleep architecture for stage 1 sleep was 11.8%, stage 2 sleep was 72.4%, slow wave sleep was 3.3%, REM sleep was 12.5%. Total arousal index was 7.7/hr and apnea hypopnea arousal index was 7.6/hr. Mean SaO₂ was 93.6%, the lowest SaO₂ was 83.3%, and SaO₂ below 90% was 0.3%. Snoring was not observed, and periodic limb movement index was 37.8/hr. PSG also showed cyclic fluctuation in breathing with periods of central apneas or hypopneas alternating with periods of hyperpneas in a gradual waxing and waning fashion (Fig. 1). MSLT was performed 5 times and showed mean sleep latency of 1.5 minutes with one sleep-onset REM period.

We evaluated the patient to find out the cause of CSB. There was no ventricular hypertrophy or dysfunction and ejection...
fraction was 61% on two-dimensional echocardiography. No abnormal findings were found on magnetic resonance imaging of the brain and pulmonary function test except old tuberculosis lesions in computer tomography of the chest.

**Discussion**

The PSG in this case showed cyclic fluctuations in breathing with periods of apneas or hypopneas alternating with periods of hyperpneas in a gradual waxing and waning fashion, with absent or reduced ventilatory effort as well as frequent arousals from sleep at the peak of hyperpnea. But, this CSB pattern in our case is not typical. Whereas the length of the ventilatory-apneic cycle in idiopathic CSA is typically shorter than 45 seconds, it is almost invariably longer than 45 seconds in CSB pattern. The lengths of the apneic cycles in our case were usually less than 45 seconds (Fig. 1). In addition, we did not find the specific causes of CSB pattern and so we entitled it 'idiopathic CSA presenting CSB'.

It is a technical limitation that we didn’t use esophageal pressure or end-tidal CO₂ monitoring sensor, but there was no obstructive apnea by nasal pressure sensor during all night monitoring.

The arterial blood gas analysis of the patient showed very mild metabolic alkalosis and respiratory alkalosis. The arterial blood gas analysis was done after pulmonary function test and he had taken thiazide 1-2 times per week for hypertension. Considering his age and intermittent diuretic use, it was not absolutely normal, but not abnormal either. The PSG were done after stopping thiazide 2 weeks ago because acute use of thiazide might have an effect on PSG and MSLT. So the cessation of thiazide had little effect on the respiratory pattern.

Although PaCO₂ during sleep was not checked, there was not sustained oxygen desaturation during sleep that was unexplained by discrete apnea and hypopneas on PSG. Physical examination including body mass index and neurological examination was normal. This case could be distinguished from sleep related hypoventilation/hypoxicemic syndrome.

Although the CSB pathophysiology of congestive heart failure can be explained with high ventilatory drive, small difference between the apneic threshold and sleeping eucapnic PaCO₂, long circulation time, and increase in reflex stimulation of breathing, the CSB pathophysiology of brain vessel diseases and renal failure are not yet clearly defined. This case might also have ventilatory instability although the cause of ventilatory instability was not detected. A previous case report described a 61-year-old male who presented with apparent idiopathic central sleep apnea but after 4 years developed features of autonomic, cerebellar and extrapyramidal dysfunction consistent with a diagnosis of multiple system atrophy. We believe that fur-
ther studies will be needed to discern whether idiopathic central sleep apnea presenting as CSB pattern is the first symptom of certain diseases or independent disease.

Although this case showed the classical PSG findings of CSB during sleep, the neurologist, cardiologist, and pulmonologist could not find any specific cause for CSB. So we concluded that this was an idiopathic central sleep apnea case presenting as CSB pattern.

REFERENCES