Rapid eye movement dependent central apnea with periodic leg movements

Melike Yüceege, Hikmet Fırat, Mutlu Kuyucu[1], Sadık Ardıç

Department of Respiratory and Sleep Medicine, Dışkapı Y. B. Educational and Research Hospital, Ankara,
[1]Department of Neurology, Urfa Medigap Hospital, Urfa, Turkey

Abstract

Central sleep apnea is a period of at least 10 s without airflow, during which no ventilatory effort is present. Most of the central apneas occur in Non-Rapid eye movement (NREM) sleep. Central apnea occuring in Rapid eye movement (REM) sleep is extremely rare. We present our patient who had a diagnosis of obstructive sleep apnea in another sleep center since 2003. His Auto Continuous Positive Airway Pressure (CPAP) machine was disrupted so he admitted to our center to renew his machine and for daytime sleepiness while using his machine. The polysomnography revealed central apneas ending with respiratory arousals and periodic leg movements in rapid eye movement (REM) stage. We found no cause for central apneas. The patient benefited from servo ventilator therapy. We present this case as an unusual form of central apnea with the review of the literatures. Even the patients diagnosed as obstructive sleep apnea should be analyzed carefully. The diagnosis and the therapeutic approach may change in the favor of the patient.

Key Words

Central apnea, periodic leg movements, REM sleep

For correspondence:
Dr. Melike Yüceege, Dışkapı Y.B. Educational and Research Hospital Respiratory and Sleep Medicine Clinic, Ankara, Turkey.
E-mail: melikebanuy@yahoo.com.tr

Ann Indian Acad Neurol 2013;16:154-6

Introduction

Central sleep apnea (CSA) is a period of at least 10 s without airflow, during which no ventilatory effort is present. Most of the central apneas occur in Non Rapid eye movement (NREM) sleep. Central apnea occuring in rapid eye movement (REM) sleep is so rare.[1] One case report has been reported with central apneas in rapid eye movement (REM) sleep so far.[2] We present this case as an unusual form of central apnea with the review of the literatures.

Case Report

A 47-year-old man was admitted to our sleep center with snoring, witnessed apnea and day-time sleepiness. He also reported leg jerks during sleep and had a diagnosis of obstructive sleep apnea in 2003 in another hospital. He had been given Auto-CPAP therapy but his machine was disrupted and hence was admitted to renew his machine. He declared that he had day-time sleepiness while using his previous machine. His body mass index was 28.6 kg/m^2 and his physical examination findings of heart and lung were normal. Ear-nose-throat examination revealed slightly larger tongue and a long uvula. Routine hematologic and biochemistrical tests revealed normal findings. The Epworth day-time sleepiness score was 24/24. Further tests were performed because of central apneas. Neurologic examination, kranial magnetic resonance imaging revealed normal findings. The eye examination was in normal ranges; there was no optic neutitis in eye examination, electrophysiologic tests (pattern visual evoked potential) were found to be normal in both sides. Echocardiologic examination revealed normal findings. Blood Borrelia Burgdoferi Immunoglobuline M (IgM) and Immunoglobuline G (IgG) levels were normal in range. Anti Cytomegalovirus (CMV) Immunoglobuline M, Anti Rubella Immunoglobuline M, Antitoxoplasma Immunoglobuline M and Herpes Simplex were negative. Anti Cytomegalovirus Immunoglobuline G and Anti Rubella Immunoglobuline G were above the normal limits.

Polysomnography (Compumedics®, Melbourne, Victoria, Australia) was performed in Dışkapı Y. B. Educational and Research Hospital Sleep Department. The apnea-hypopnea index (AHI) was 28 and periodic leg movement index was 26. All the events were in REM stage and central apneas created most of the respiratory events; 192/192 apneas were central and 15 hypopneas were detected. Periodic leg movements followed the respiratory

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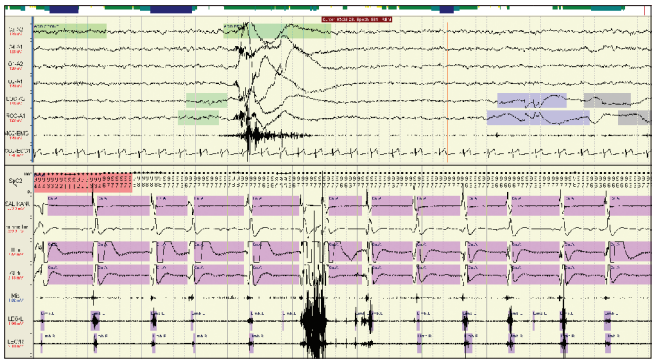

Figure 1: Central apneas ending with arousal and leg movements (30 s epoch upper part, 5 min epoch lower part)

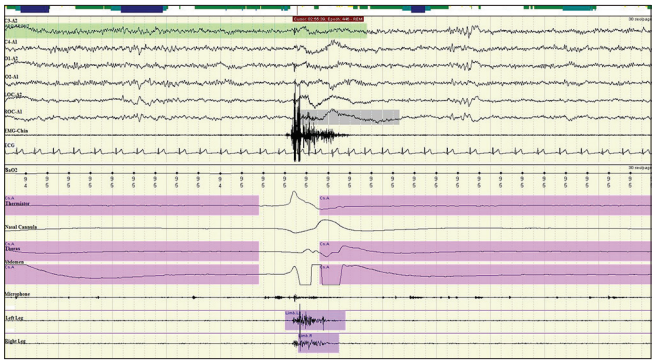

Figure 2: Two central apneas in REM stage ending with leg movement (30 s epoch)

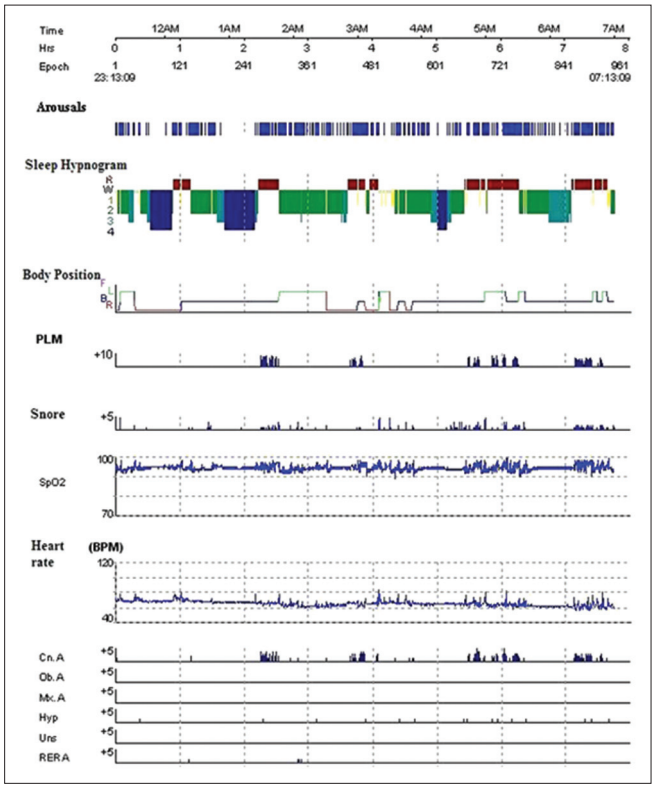

Figure 3: Histogram of the patient (whole night)

arousals and central apneas in REM stage [Figures 1 and 2]. His histogram demonstrated central apneas in REM stage with periodic leg movements [Figure 3]. Piezo electric was used as effort sensor type. The duration of oxygen saturation <90% were nearly 0 min, most of the events caused only 2% desaturation.

Mean transcutaneous carbon dioxide ($TcCO_2$) was 35.8 mmHg (min. 32.2 and max. 45.5 mmHg). His apneas were unresponsive to noninvasive ventilator titration with continuous positive airway pressure (CPAP), bilevel positive airway pressure (BPAP) and BPAP-S/T; so servoventilator was used on the second titration night. After 2 nights' of non-invasive mechanical ventilator titration (Weinman®, Germany), he was given servoventilator (Maximum IPAP: Inspiratuar Positive Airway Pressure: 20 cmH_2O, Min EPAP : Expiratuar Positive Airway Pressure: 7 cm H_2O and EEPAP: End Expiratuar Positive Airway Pressure: max 12 cm H_2O). The apnea-hypopnea index in polysomnography with the servoventilator was 5,5 and periodic leg movements had disappeared.

The patient reported that his day-time sleepiness had also disappeared after using the device. He is still under our control periodically.

Discussion

Our patient had central apneas in REM stage with no known cause. This is the second reported case with central apneas in REM stage. In the first case,[2] the patient with central apneas in REM stage had elevated levels of Borrelia Burgdoferi in blood with no proven neuroborreliosis. She had no periodic leg movements in polysomnography and the severity of the central apneas were reduced spontaneously by waiting, due to the patients refusals of further evaluation during that period. In our case, the patient had central apnea and periodic leg movements in REM sleep, probably since 2003 and had used Auto-CPAP until the disruption of his machine.

Most patients have overlap of obstructive sleep apnea (OSA) and central sleep apnea (CSA). CSA Syndrome is considered primary diagnosis when >50% of apneas are scored as central in origin.[3] Central apneas are classified as:

- High altitude periodic breathing
- Idiopathic CSA
- Narcotic induced central apnea
- Cheyne stokes breathing
- Obesity hypoventilation syndrome (OHS) (hypercapneic CSA)
- Complex sleep apnea.

Both hypercapnia and hypocapnia cause central apnea. There is further reduction in responsiveness of chemoreceptors in REM sleep. In hypercapnic central apnea, there is impaired central drive and/or impaired respiratory motor control. Cheyne Stokes Breathing and idiopathic CSA cause non-hypercapnic apnea. Lesions of brain stem – tumors, trauma induced lesions, congenital central hypoventilation syndrome (Ondine's curse), long-term use of opioids, OHS cause hypercapnia via impaired central drive.

Disorders of autonomic system, damage to brain stem (respiratory centers) by post-polio syndrome, tumor, infection, hemorrhage, encephalitis, and interruption of neural pathways

from medullary respiratory centers to ventilatory muscles are some of the neurologic causes of central apnea.

In neuromuscular disorders and chest wall syndromes, there is impaired respiratory muscle control. In cheyne stokes breathing, there is cyclic crescendo-decrescendo respiratory effort and airflow during wakefulness and sleep, without upper airway obstruction

Idiopathic central apnea may occur as distinct events or repetitive cyclical pattern and the duration of cycle usually 20-40 s with less severe O_2 desaturations. There are arousals at termination of apnea. The events are mainly in stage N1 and N2 sleep and there is usually elevated hypercapnic ventilatory response.

Our patient had central apneas in REM stage with no known cause. Mean $TcCO_2$ level was 35.8 mmHg, in normal ranges (min. 32, max. 45 mmHg). He had no cardiac insufficiency, neurologic disease or eye disease. The viral antibodies including Borrelia were negative. All of the periodic leg movements followed a respiratory arousal due to central apnea. All of the events were in REM stage and the patient did not wake-up in most of the events.

The titration was successfully done with servo ventilator, which is one of the choices in central apneas.

Even if the diagnosed patients as "obstructive sleep apnea" with daytime sleepiness should be examined carefully, the therapeutic approach may vary. Central apneas may lead to frequent night-time awakenings leading to excessive day-time sleepiness and increased risk of cerebrovascular adverse outcomes.

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How to cite this article: Yüceege M, Firat H, Kuyucu M, Ardlç S. Rapid eye movement dependent central apnea with periodic leg movements. Ann Indian Acad Neurol 2013;16:154-6.
Received: 04-01-13, **Revised:** 05-02-13, **Accepted:** 03-02-13

Source of Support: Nil, **Conflict of Interest:** Nil