LETTER TO THE EDITOR

A Patient with Obese Hypoventilation Syndrome with Ventilator-associated Pneumonia: Brain Mapping and Polysomnography Outcomes

Obese hypoventilation syndrome is also historically described as the Pickwickian syndrome. Patients may clinically present themselves with symptoms such as excessive daytime sleepiness, fatigue, or morning headaches, which are similar to the symptoms of obstructive sleep apnea-hypopnea syndrome. Ventilator-associated pneumonia is the most common nosocomial infection in patients receiving mechanical ventilation.

A 65-year-old male patient was admitted because of breathlessness and decreased responsiveness in the previous 3 hours. The patient also complained of insomnia, confusion, and restlessness. Past history showed lower lobe consolidation of the lung and multinodular goiter. The patient was later diagnosed as having hypoventilation syndrome and carbon dioxide (CO2) narcosis. Two years previously, the patient underwent an operation. He was inserted with a metal tracheostomy tube. He had a known case of diabetes mellitus since the previous 6 months and received 500 mg of metformin daily. He had a morbid body mass index of 42.8 kg/m². The results of atrial blood gases were as follows: pH, 7.37; arterial partial pressure of oxygen (PaO2), 130 mm Hg; arterial partial pressure of carbon dioxide (PaCO2), 47.7 mm Hg; and oxygen saturation (SaO2), 96.5%. The patient had normal findings in the complete blood counts, renal and liver function, serum glucose, electrolytes, ammonia, erythrocyte sedimentation rate, and thyroid function tests. He was treated intravenously every 6 hours with 250 mg of cefotaxime. From his personal history, he was a nonsmoker and nonalcoholic. From his past medication history, he was taking daily bactrim (100 mg tablet), diltiazem (30 mg), and pantoprazole (40 mg). On the 4th day of admission, the patient was referred to the metabolic ward of the SRM Medical College Hospital and Research Centre (Tamil Nadu, India) for an overnight polysomnography (PSG) study that was performed simultaneously with brain mapping. The sleep study was performed on two consecutive nights. The total PSG recording time was 6 hours 20 minutes, total sleep time was 217 minutes, sleep latency was 30.5 minutes, wake after sleep onset was 42.7 minutes, sleep efficiency was 59.9%, stage 1 (N1) sleep was 104.7 minutes, stage 2 (N2) sleep was 3.1 minutes, stage 3 (N3) sleep was 1.6 minutes, rapid eye movement (REM) sleep was 5.9 minutes, and wake was 101.8 minutes (Figure 1). The apnea/hypopnea duration index was 23.2. Hypopnea episodes and desaturation (1–3%) episodes were frequent (Figure 2). Sinus tachycardia was detected on his overnight electrocardiogram. From the brain mapping results, 10–11 Hz low amplitude alpha-frequency waves were in the occipital and posterior parietal regions in response to eye opening. Predominant delta waves ranged 4–6 Hz amplitude. Photostimulation and hypoventilation responses were unremarkable. No spikes or sharp waves were observed.

The patient received an overnight PSG, conducted by the guidelines published by the American Academy of Sleep Medicine (Darien, Illinois, USA). The PSG findings consisted of continuous recordings of central and occipital electroencephalograms (EEGs), bilateral electro-oculograms, submental and bilateral tibial electromyograms, and an electrocardiogram. The nasal and oral airflows were measured using thermocouple sensors and pressure transducer airflow monitoring devices. Body positioning was verified by infrared video recording.

It is unclear why only 10%–15% of patients with obstructive sleep apnea-hypopnea develop hypoventilation, although obstructive sleep apnea-hypopnea syndrome has been postulated as a
cause of depressed ventilator response and hypoventilation.\textsuperscript{4} Nocturnal continuous positive airway pressure therapy (applied by nasal mask) is usually effective for treating concurrent obstructive sleep apnea/hypopnea syndrome. Brain mapping provides a continuous measure of cortical function with excellent time resolution. It is also relatively inexpensive, noninvasive, and safe, compared to newer brain imaging techniques. Hypoventilation could not produce any epileptic form changes or clinical seizures in the EEGs of the patient.

The PSG findings showed that this patient had a shortened sleep time, reduced sleep efficiency, increased sleep latency, increased REM latency, and reduced REM sleep. Quantitative electroencephalography (QEEG) displayed relatively normal electrical activity of the brain. Therefore, we need to draw clinicians’ attention to the importance of sleep complaints and parameters regarding prognosis in obese hypoventilation syndrome with ventilator-associated pneumonia cases.

References

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