

Case report of new-onset obstructive sleep apnea after carbon monoxide poisoning

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Abstract

Obstructive sleep apnea (OSA) is characterized by repetitive intermittent oxygen desaturation during sleep. Carbon monoxide poisoning (COP) is the second most common cause of death among non-medicinal poisonings, and oxygen therapy is the current standard of treatment for COP. We herein report a case of a 50-year-old woman diagnosed with severe OSA associated with COP. Both the OSA and COP gradually resolved by automatic continuous positive airway pressure (CPAP) therapy. New OSA symptoms appeared following the development of delayed encephalopathy after acute COP (DEACMP) 3 weeks later. Severe OSA was diagnosed 76 days after COP with an apnea–hypopnea index of 66 events/hour, and CPAP therapy was immediately administered. The patient’s DEACMP symptoms and OSA both improved with CPAP therapy (her apnea–hypopnea index decreased to 32.4 and 16.5 events/hour at 161 and 204 days after COP, respectively). To our knowledge, this is the first case report of OSA caused by COP based on the occurrence and disappearance of OSA symptoms and laboratory findings associated with the emergence and improvement of DEACMP.

Keywords

Carbon monoxide poisoning, obstructive sleep apnea, continuous positive airway pressure, delayed encephalopathy after acute carbon monoxide poisoning, apnea–hypopnea index, polysomnography

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Introduction

Carbon monoxide poisoning (COP) is the second most common cause of death among non-medicinal poisonings in the United States.¹ The number of patients with COP seen in emergency rooms is also a few times higher than that of all other instances of poisoning during winter in some cold areas of China. An extensive literature review revealed no other cases of exacerbation or the presence of obstructive sleep apnea (OSA) caused by COP. We herein report a case of OSA caused by COP that resolved with the improvement of delayed encephalopathy after acute COP (DEACMP) and continuous positive airway pressure (CPAP) therapy.

Case report

A 50-year-old woman, who was one of three survivors of a COP event that left her in a coma and another dead, was referred to the emergency department of a local hospital after approximately 12 hours of exposure to a coal-burning stove. After she had been unconscious for 3 days in the hospital, she gradually began to recognize her surroundings, talk, and eat. Apart from routine medical treatment, she also received hyperbaric therapy after regaining consciousness. Three weeks later, she rapidly exhibited typical symptoms of DEACMP, including disorientation, apathy, dyskinesia, increased muscular tension, slurred speech, slowed responses, marked global cognitive impairment, abnormal behavior, and double incontinence. Magnetic resonance imaging (MRI) conducted 25 days after COP revealed toxic encephalopathy in the basal ganglia, central and frontal parietal cortex, and white matter as well as ischemia in the bilateral semiovale center and under the frontal parietal cortex.

During this period, the patient's family members noticed that she had newly developed loud snoring and witnessed apnea during sleep. Because a polysomnography (PSG) examination room was not available, she underwent PSG 76 days after COP. Although her severe DEACMP symptoms had somewhat improved by that time, she still had slow reactions and speech, increased muscular tension, and loud snoring. As shown in Figure 1(a), the patient had severe OSA with an apnea-hypopnea index (AHI) of 66 events/hour. During a total sleep time of nearly 6 hours, she had 189 obstructive apnea events (longest event was 87 s), 33 hypopnea events (longest event was 40 s), 155 mixed apnea events (longest event was 98 s), and 15 central apnea events (longest event was 27 s). Moreover, the patient developed frequent and severe oxygen desaturation (oxygen desaturation index of 50.4 episodes/hour, lowest peripheral blood oxygen saturation of 63%).

The patient immediately began to use auto-CPAP therapy during sleep. Under CPAP therapy, she had more stabilized sleep without snoring or apnea, and the improvement of her overall DEACMP symptoms was more substantial. The patient underwent a second and third PSG examination without CPAP 161 and 204 days after COP, during which her AHI dropped to 32.4 and 16.5 events/hour, respectively (Figure 1(b) and (c)).

The patient continued using CPAP for 7 months with good adherence and effective treatment reported by the memory card. Her loud snoring and witnessed apnea disappeared; therefore, she discontinued CPAP therapy. The patient had no excess weight gain and did not use any sedative medications after COP or during the period of PSG and CPAP therapy. Throughout the 10-month follow-up, she had no significant sequelae and was nearly back to her normal life.

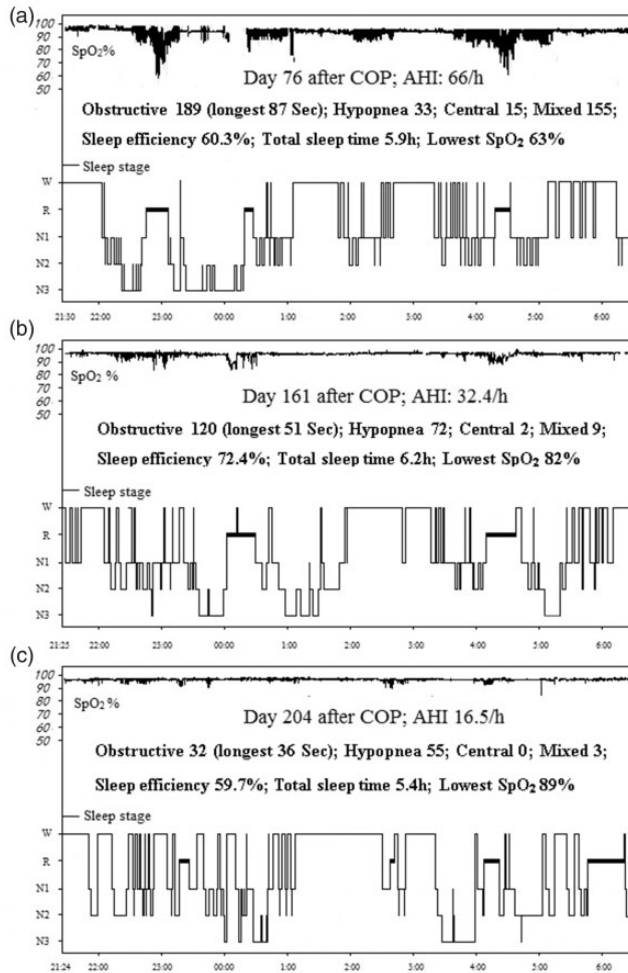


Figure 1. SpO₂ (upper) and sleep histogram (lower) for three polysomnographic recordings. (a) Night of diagnosis (76 days after COP). (b, c) Repeated polysomnographic examination (161 and 204 days after COP, detected without CPAP). COP, carbon monoxide poisoning; AHI, apnea-hypopnea index; SpO₂, peripheral blood oxygen saturation.

Discussion

To our knowledge, this is the first case report of OSA caused by COP based on the occurrence and disappearance of OSA symptoms and laboratory findings associated with the emergence and improvement of DEACMP.

COP-induced diffuse hypoxic-ischemic encephalopathy may cause neuronal necrosis and apoptotic death leading to diffuse

brain atrophy including areas such as the basal ganglia and thalamus. Moreover, white matter demyelination is also a sign of delayed neuropsychiatric syndrome that commonly involves areas such as the periventricular white matter and centrum semi-ovale.² We speculate that two factors may have accounted for the COP-induced OSA in our patient. First, the impairment of brain regions shown in the MRI

examination may have affected the pathway between the cortex and brain stem respiratory control center. Second, although MRI showed no abnormality in the region of the brain stem, there may have also been some undetectable pathological changes in certain neuron cells or nuclei that influenced the control of the pharyngeal dilator muscles, especially the genioglossus. However, we were unable to identify the exact mechanism of COP-induced OSA in this case. Furthermore, severe COP-induced brain impairment is diffuse and persistent. In this case, the AHI was still 66 and 32.4 events/hour at nearly 2 and 4 months, respectively, after treatment for DEACMP. Oxygen therapy is the current standard of treatment for COP.^{3,4} The recurrent intermittent hypoxia and disrupted sleep induced by OSA events are central pathological issues for patients with OSA. Therefore, timely identification and specific management of severe intermittent hypoxia induced by OSA is considerably beneficial for these patients.

Additionally, our patient's sleep efficiency was still very low (60%) because of her long wakeful episodes in the latter half of the night during approximately 9-hour recordings at 2 and 6 months after DEACMP. PSG showed low overnight sleep efficiency, which normally reflects a circadian disturbance induced by organic and functional brain impairments.⁵ These findings indicate that COP-induced OSA and an objectively determined poor amount of sleep may require a more extensive and longer period of CPAP and other therapies. Moreover, CPAP therapy not only improves OSA or related symptoms but also COP. In the present case, the improvement of the patient's overall DEACMP symptoms became more substantial after CPAP therapy. Previous studies have also indicated that CPAP can be an effective treatment alternative to

conventional oxygen therapy in patients with COP.⁶⁻⁸

A clear limitation in this report is that the patient did not undergo an overnight PSG examination before COP to fully confirm that she did not have pre-existing OSA. However, her family denied that she had OSA symptoms before COP. In addition, because the interval between the presence of OSA symptoms after DEACMP and the first PSG examination was 2 months, we were unable to determine whether OSA was even more severe during the immediate presence of DEACMP symptoms compared with 2 months later.

Conclusion

This unusual case suggests that clinicians should screen for possible OSA in association with COP. Further clinical research concerning the prevalence of OSA and effects of CPAP treatment in patients with COP is needed. Timely identification and specific management of intermittent hypoxia induced by OSA would be considerably beneficial for patients with COP. In addition, that this patient was on the waiting list for nearly 2 months prior to receiving PSG and beginning CPAP after DEACMP indicates that the use of a portable device to perform the examination might be considered if a PSG room is not available.

Ethics

Written consent for treatment and publication was obtained from the patient described in this case report. The patient's details have been de-identified. The ethics review committee waived the requirement for ethics approval because of the nature of the study (case report).

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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