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Severe Obesity Hypoventilation Syndrome Treated with Auto-CPAP

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Summary

Obesity hypoventilation syndrome (OHS) is a serious medical condition that remains undiagnosed in seriously ill hospitalized patients. Significant improvement of daytime hypercapnia can be achieved with positive airway pressure (PAP) therapy. Bi-level PAP is generally employed with the goal of improving ventilation. A 50-year-old woman with OHS and severe hypercapnia was successfully treated with an auto titrating continuous PAP (Auto-CPAP) device. The major role in the pathogenesis of daytime hypercapnia in patients with OHS, is the progressive accumulation of carbon dioxide (CO₂) caused by repetitive obstructive events at night, which can be eliminated with the low cost approach of treating with Auto CPAP.

Background

OHS, an interaction between sleep disordered breathing and obesity-related respiratory impairment leading to chronic daytime hypercapnia, remains under recognized and definitive treatment is often delayed.[1] Treatment of sleep disordered breathing with PAP therapy results in significant improvement of daytime hypercapnia.[2] Auto-CPAP is generally not recommended to treat obesity hypoventilation syndrome.[3,4] We present a patient with OHS and severe daytime hypercapnia who was successfully treated with Auto CPAP.

Case Presentation:

A fifty-year-old morbidly obese (BMI 52) non-smoking female with a history of hypertension and untreated obstructive sleep apnea (OSA) was admitted to the hospital with severe dyspnoea on exertion in July 2012. She was hypoxemic at admission. Arterial blood gas (ABG) on 2 liters of supplemental oxygen revealed a pH of 7.34, pCO₂ of 88 mm Hg and a pO₂ of 79 mm Hg. Serum bicarbonate level was elevated at 44mEq/L. Cardiac enzymes were normal. Brain natriuretic peptide (BNP) was elevated at 2161 pg/ml. D-dimer was normal. Chest x-ray was notable for prominence of main pulmonary artery, suggestive of pulmonary artery hypertension. EKG had non-specific changes. Echocardiogram revealed normal left ventricular systolic and diastolic function with mild pulmonary hypertension. Nuclear medicine stress test was normal. Computed tomography angiogram was normal.

Pulmonary/Sleep medicine was consulted, and a presumptive diagnosis of OHS was made given the patient's morbid obesity, previous history of OSA and hypoventilation on ABG.

She was placed on CPAP at 10 cm water pressure in the hospital and demonstrated significant clinical improvement with relief of shortness of breath. Supplemental oxygen requirements improved from as high as 4 liters per minute via nasal cannula to 2 liters per minutes.

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Investigations:

A polysomnogram with a split night protocol was performed prior to patient being discharged home, revealed an Apnoea-hypopnoea index (AHI) of 41 events per hour with a baseline oxygen saturation of 85%. CPAP titration was suboptimal during the study mainly due to lack of recording time as the study was performed with a split night protocol leaving inadequate time for optimal titration. . Supplemental oxygen was required for non-apneic oxygen desaturation.

Pulmonary function tests revealed severe restrictive defect with severely reduced expiratory reserve volume and normal inspiratory and expiratory muscle pressure. These results were suggestive of morbid obesity.

Treatment:

As the titration study was suboptimal, she was prescribed Auto CPAP with a maximum pressure of 20 and minimum of 12 cm along 2 liters of supplemental oxygen.

Outcome and follow up:

The patient demonstrated excellent compliance with CPAP with complete resolution of sleep apnoea on smart card data download at six week follow up. Shortness of breath resolved, resulting in discontinued use of oxygen with ambulation and at rest. ABG on room air 3 months after use of Auto CPAP revealed a pH of 7.39, pCO₂ of 51mm Hg, and pO₂ of 73mm Hg. Nocturnal oximetry on Auto CPAP did not demonstrate oxygen desaturation, so supplemental oxygen at night was discontinued. Patient did not follow up for two years due to lack of medical insurance, but was compliant with PAP therapy. Upon her return visit in February, 2015, she was clinically doing well. AHI was 1.8 on smart card data download. As the serum bicarbonate was normal at 26 mEq/ml, ABG was not repeated.

Discussion:

OHS is defined as awake hypercapnia (PaCO₂ > 45 mm Hg) in an obese patient (BMI ≥ 30 kg/m²) after other causes that could account for awake hypoventilation, such as lung or neuromuscular disease have been excluded.[2] 90% of patients with OHS have moderate to severe OSA. The remaining 10% have sleep related hypoventilation that is not related to upper airway obstruction.[2] OHS is associated with significant morbidity and mortality.[1,5] However, diagnosis and definitive treatment are often delayed,[1] as shown by our patient, who underwent extensive work-up before the institution of definitive therapy.

Compared with eucapnoeic obese individuals, patients with OHS have altered ventilatory responsiveness to hypoxia and hypercapnia.[6] The role of OSA in the pathogenesis of daytime

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Thomas Jefferson University Hospital, Department of Medicine, Jefferson sleep disorders center. hypercapnia is suggested by its improvement in many cases upon relief of upper airway obstruction with PAP therapy or tracheostomy.[7]

It is unclear whether patients with OHS should be treated with CPAP or Bi-level PAP therapy.[8] Traditionally, Bi-level PAP therapy is used by increasing airway pressure during inspiration (IPAP) and expiration (EPAP) to eliminate obstructive events and increasing pressure support (PS) to improve alveolar ventilation.[9] A recently published randomized control trial of 211 patients with OHS demonstrated significant improvement in clinical symptoms, serum bicarbonate, and PaO₂ levels in patients treated with Bi-level PAP and CPAP compared with a control group, with no significant differences between Bi-level and CPAP groups.[10] An earlier study reported both CPAP and Bi-level PAP therapy to be equally effective in improving daytime hypercapnia in patients with OHS without severe nocturnal desaturation.[8] These two studies support the use of CPAP for treatment of OHS.

For our patient we elected to use Auto-CPAP as opposed to fixed pressure CPAP. Despite not being recommended by current guidelines,[3.4] Auto-CPAP has been shown to be equally efficacious in treatment of OSA.[11] Furthermore, once our patient established effective Auto-CPAP usage, supplemental oxygen was no longer required, suggesting that hypoxemia and the progressive accumulation of CO₂ caused by repetitive obstructive events was responsible for the increased daytime PaCO₂. We believe that the elimination of upper airway obstruction should be the primary goal in improving daytime hypoventilation and weight loss should be the long term goal, when treating patients with OHS. Monitoring to assess compliance with PAP therapy along with relief of symptoms, and documenting improvement of daytime hypercapnia with ABG or serum bicarbonate are important.

Learning points/Take home messages

1. OHS can be successfully diagnosed and treated if clinicians have a high index of suspicion
2. OHS is associated with high morbidity and mortality
3. Treatment of associated OSA can relieve daytime hypercapnia and hypoxemia in many cases
4. OHS in many cases does not require Bi level PAP therapy and can be treated effectively with Auto CPAP

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Keywords:

Auto CPAP, obesity hypoventilation syndrome, obstructive sleep apnea