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BOARD REVIEW CORNER

A Big Problem in the ICU

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A 40-year-old morbidly obese (BMI 47 kg/m^2) man is admitted for the third time this year to the ICU with respiratory failure. He carries the diagnosis of COPD and congestive heart failure. Initial arterial blood gas is pH 7.30, $paCO_2$ 70 mm Hg, and paO_2 60 mm Hg on room air. He is treated with noninvasive positive pressure ventilation, and empirically with bronchodilators, steroids, and furosemide. Over several days he improves and is discharged to the floor.

On the medical floor, efforts are made to understand the cause of the patient's recurrent respiratory failure and to prevent readmission. Echocardiogram shows a normal left ventricular ejection fraction and no evidence of diastolic dysfunction. Pulmonary function testing shows FEV1/FVC ratio of 0.85, and an FVC of 80% of predicted.

The patient is weaned off of oxygen during the day; however, there are profound oxygen desaturations at night. Apneas are noted. The patient had been scheduled on prior discharges for outpatient sleep studies, but had never attended the sleep lab (sometimes due to readmission). An inpatient sleep study is performed that shows severe obstructive sleep apnea with an apnea-hypopnea index (AHI) of 104 events per hour and an oxygen nadir of 50%.

What is the next best step in management?

A. Nocturnal supplemental oxygen
B. Initiation of CPAP/bilevel PAP therapy
C. Initiation of average volume assured pressure support (AVAPS) therapy
D. Respiratory stimulant therapy with progesterones
E. Weight loss

ANSWER: B. Initiation of CPAP/bilevel PAP therapy

Obesity hypoventilation syndrome (OHS) is defined by the triad of obesity, daytime hypoventilation, and sleep disordered breathing. It is a diagnosis of exclusion, and there must be no other potential cause of hypoventilation such as intrinsic lung disease or neuromuscular weakness, as in this case. Patients with extreme obesity are more likely to have OHS. In patients with OSA, the prevalence of OHS is approximately 25% in those with a BMI > 40 kg/m^2.[1] Most OHS patients have severe OSA and classical OSA symptoms with excessive daytime sleepiness.

Diagnosis is based on analysis of an arterial blood gas performed on room air during wakefulness, without supporting evidence of another cardiopulmonary disorder. Serum bicarbonate > 27 mEq/L is suggestive of the diagnosis.[2] Serum hematocrit may be secondarily elevated due to chronic hypoxemia. Frequently, however, such patients come to medical attention after admission for acute on chronic respiratory failure to the hospital or ICU. Such patients are frequently admitted and treated empirically for other cardiopulmonary disorders, often for years, before the diagnosis is made.[3] The delay in diagnosis may be due to difficulties in obtaining timely (e.g., inpatient) polysomnography for the formal diagnosis of OSA. Inpatient portable monitoring is technically feasible, although few centers provide such testing.[4]

The pathophysiology is not completely known and may involve multiple mechanisms, including increased mechanical load on the respiratory system as well as blunted central respiratory drive.[5]

Although patients with OHS hypoventilate, treatment aimed at relieving upper airway obstruction alone is sufficient in most cases in also improving ventilation over time. That is, CPAP therapy that relieves OSA also ameliorates hypoventilation.[6] Bilevel therapy can be considered in those who do not improve despite adequate adherence to PAP therapy, or in patients with substantial hypoventilation (as in this case). Average volume assured pressure support (AVAPS) therapy is a ventilation mode that varies the amount of pressure support to deliver a target tidal volume, which should help support minute ventilation. However, studies to date have not demonstrated substantial benefits over bilevel PAP in objective physiological or subjective outcomes, and AVAPS is considerably more expensive.[7] Oxygen alone would not be expected to improve upper airway patency and might worsen hypercapnia.

Weight loss would be effective to help improve OHS. However, substantial weight loss is difficult and/or takes time, thus this is not the best answer as the next step in management. It is also increasingly recognized that patients who successfully lose weight through bariatric surgery typically have an initial improvement in OSA that recedes with time. The same has been seen in those with OHS who have undergone bariatric surgery.[8] There are limited and conflicting data to recommend medroxyprogesterone or other respiratory stimulants, as these have side effects as well.

CITATION

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