Obesity Hypoventilation Syndrome in the Critically Ill

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KEYWORDS

• Obesity hypoventilation syndrome • Critically ill • Hypercapnic respiratory failure
• Continuous positive airway pressure

KEY POINTS

- Obesity hypoventilation syndrome is a common but underrecognized cause of acute on chronic hypercapnic respiratory failure in the intensive care unit.
- The development of the obesity hypoventilation syndrome is multifactorial and is due to impairments in pulmonary function, ventilatory drive, sleep-disordered breathing, and hormonal regulation.
- Obesity, an awake $Paco_2$ greater than 45 mm Hg, and a serum bicarbonate level higher than 27 mEq/L are key diagnostic indicators of the disease.
- Positive airway pressure (PAP) (continuous PAP and noninvasive ventilation including bilevel PAP and more advanced modes) can successfully treat respiratory failure.
- Weight loss is critical in the management of obesity hypoventilation syndrome.
- Obesity hypoventilation is associated with significant morbidity and mortality.

INTRODUCTION

Obesity hypoventilation syndrome (OHS) is characterized as obesity and daytime hypoventilation in the absence of other causes of hypoventilation such as pulmonary disease, neuromuscular weakness, or chest wall disorders. A common presentation of OHS is in the critically ill patient who presents for acute on chronic hypercapnic respiratory failure. As the proportion of obese and morbidly obese individuals increases, intensive care unit (ICU) providers need heightened awareness of OHS and its complications, prognosis, and treatment modalities. An understanding of the how obesity affects pulmonary function and control of ventilation is needed. Continuous positive

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airway pressure (CPAP), bilevel positive airway pressure (BPAP), and average volume-assured pressure support (AVAPS) are modes of noninvasive ventilation (NIV) used to manage respiratory failure in this population. Long-term strategies to address weight loss are important in the chronic management of the patient with OHS, and pharmacologic therapy plays a significant role in respiratory stimulation. OHS is associated with significant negative outcomes, particularly in the critically ill. This review serves to increase knowledge of the epidemiology, diagnosis, pathophysiology, treatment, and outcomes in patients with OHS.

EPIDEMIOLOGY

The exact prevalence of patients with OHS is unknown. Most studies examining the prevalence of OHS were conducted in sleep laboratories and clinics, estimating that 10% to 20% of patients with obstructive sleep apnea (OSA) have OHS.[1] According to data from the National Health and Nutrition Examination Survey in 2009-2010, 35.7% of United States adults are obese, with rates of obesity climbing fastest among the aging and men.[2] Rates of morbid obesity in the United States are increasing, with 5% of Americans having a body mass index (BMI; calculated as weight in kilograms divided by height in meters squared, ie, kg/m^2) greater than 40.[3] It can be assumed that rates of OHS will increase as the population of obese patients increases. An estimate of the prevalence of OHS in the general adult population of the United States is 1 in 300 to 1 in 600 adults.[4] The prevalence of OHS in hospitalized patients, and particularly the critically ill, is less known. In single study of consecutive adult hospitalized patients with a BMI of 35 or higher, OHS was present in 31%.[5] In the ICU, obese patients represent up to 50% of all patients,[6] and 8% of ICU admissions met criteria for OHS in a study at a single center.[7] However, the underdiagnosis and underreporting of OHS likely underestimates the true prevalence. Ethnic and geographic factors also affect OHS prevalence. For example, the prevalence of OHS was 2.3% among Japanese patients with OSA.[8] However, in a study conducted at a tertiary health care facility in Turkey, 3.4% of patients who underwent arterial blood gas analysis had evidence of hypoventilation, and OHS accounted for 24% of these subjects.[9]

PATHOPHYSIOLOGY

The progression from obesity to OHS is variable and multifactorial.[10,11] The prevalence of OHS correlates with the degree of obesity, severity of sleep-disordered breathing, and restrictive mechanics on lung function tests.[12–14]

Lung Mechanics

With obesity there is reduction in the functional residual capacity (FRC) and expiratory reserve volume (ERV).[15] These lung volumes are further reduced with OHS.[16] The fat distribution can affect lung volumes. A central pattern of obesity is seen with morbid obesity, with higher waist to hip ratio and larger neck circumference.[16] The central adiposity has been shown to correlate with lower forced vital capacity (FVC) and forced expiratory volume in 1 second (FEV_1) independent of the BMI.[17]

Data from anesthetized morbidly obese patients undergoing surgery suggests they have high pleural pressures throughout the chest. There is reduction in respiratory system compliance, primarily from lung compliance rather than chest wall compliance.[18] Compared with normal subjects, eucapnic obese individuals have a 20% lower respiratory system compliance, which drops further to almost 60% lower in OHS subjects[19]: high pleural pressures result in tidal breathing near their residual volume. Low lung volumes affect the respiratory mechanics unfavorably. At this point