Hello, everyone, and welcome today.

I'm going to be talking about the management of obesity hypoventilation syndrome.

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One of the earliest references to obesity hypoventilation syndrome came from Charles Dickens, actually.

He referred to a wonderfully fat boy standing upright on the mat with his eyes closed as if asleep, thereby referring to Pickwickian Syndrome, what we today call obesity hypoventilation syndrome.

What is obesity hypoventilation syndrome?

A diagnosis can be made with a BMI of 30 or greater, which is obesity, in addition to chronic alveolar hypoventilation, which is defined by an arterial pCO2 of more than 45 millimeters of mercury.

These patients usually have some degree of daytime hypoxemia as well, and their arterial p02 generally runs at less than 70 millimeters of mercury.

About 90% of these patients will have obstructive sleep apnea syndrome, although about 10% have central hypoventilation with no discrete respiratory events.

And it's a diagnosis of exclusion.

So there should be no other cause of alveolar hypoventilation to call it obesity hypoventilation syndrome, or OHS.

So why do we care about OHS, or Obesity Hypoventilation Syndrome?

The prevalence of OHS is about 10% to 15% in obstructive sleep apnea patients.

However, when you look at the reverse, about 70% to 90% patients with OHS have obstructive sleep apnea syndrome.

So looking at those prevalence estimates, the prevalence of OHS is about 3.7 out of every 1,000 persons living in the United States.

And there's a dose-response relationship between obesity, as measured by the body mass index, and OHS.

As depicted on this next bar chart, the light gray bars basically depict individuals with simple obesity, and the dark gray bars are individuals with obesity hypoventilation syndrome.

And as you can see, as the BMI goes up the proportion of subjects who have obesity hypoventilation syndrome also goes up.

Individuals with a BMI of 50 or greater, about 40% to 50% of them will have OHS.

The next chart shows you the clinical features of obesity hypoventilation syndrome as seen in a particular study.

In this study, the average age of these individuals was about 52 years.

About 60% of these were men, and their mean body mass index was about 44.

These patients had hypercapnia by definition, so their main arterial pCO2s were 53, and they also had hypoxemia with a mean arterial p02 of about 56 millimeters of mercury.

And they were quite sleepy.

The average Epworth sleepiness score was about 14.

The next graph shows you a hypnogram depicting a rise in arterial pCO2 during REM sleep coinciding with the intermittent episodic hypoxemia that happens with sleep apnea, and is more pronounced during REM sleep.

So obesity can produce hypercapnia by several mechanisms.

These include leptin resistance, which blunts the ventilatory response.

In addition to that, there is decreased respiratory system compliance because of obesity, and thereby increased work of breathing.

It also produces upper airway obstruction, thereby resulting in sleep apnea.

And by all these mechanisms, chronic hypochondriac can result.

OHS is a chronic condition.

So basically it's associated with sleep apnea, with altered respiratory mechanics, leptin and insulin resistance, low-grade inflammation, endothelial dysfunction, which can produce many of the cardiovascular and neurological sequelae that we associate with sleep apnea, excessive daytime sleepiness, and a high risk of hospitalization and death.

As in intensivists, the first presentation of obesity hypoventilation syndrome that we might see might be in the ICU with acute respiratory failure.

A higher prevalence of congestive heart failure, pulmonary hypertension, and diabetes has been reported in OHS patients as compared to sleep apnea patients, who are normocapnic or eucapnic.

There is also a higher prevalence of hospitalizations and ICU admissions in OHS as compared to obese patients who are eucapnic.

And at the end of the day, we care because patients with OHS die faster, as seen by the lower dotted line, as compared to those who have sleep apnea and no obesity hypoventilation syndrome.

A diagnosis of obesity hypoventilation syndrome can be established with an elevated serum bicarbonate level with the caveat that if you do see a serum bicarbonate level of 27 or greater, it's important to confirm the diagnosis with an arterial blood gas.

In addition to that, it's a diagnosis of exclusion, as I mentioned earlier.

So other causes of hypoventilation have to be excluded.

Many of these patients will have waking hypoxemia as seen on a pulse oximetry, and they also have secondary erythrocytosis.

So this constellation of findings should raise suspicion that the patient may not just have sleep apnea but obesity hypoventilation syndrome.

Treatment of OHS revolves around the treatment of sleep apnea, which is often associated with it.

This includes relief of upper airway obstruction with positive airway pressure such as CPAP, or BIPAP, or, ultimately, tracheostomy.

Hypoxemia can be relieved with oxygen.

Alveolar hypoventilation can be treated with respiratory stimulants like almitrine, progesterone, and acetazolamide.

Leptin has been studied in the mouse model but not in human beings.

And then weight loss surgery for morbid obesity would certainly help to improve these manifestations of OHS.

It's very important that obesity hypoventilation should be treated, especially perioperatively.

As untreated sleep apnea, perioperatively can increase the risk of hypercapnic respiratory failure around that time.

So when patients present with acute respiratory failure and are unstable, as defined by respiratory acidosis, mental status change, hemodynamic instability, or multi-organ failure, BIPAP generally can achieve a near-normal pCO2 level fairly quickly.

This is in contrast to the management of chronic stable outpatient OHS where CPAP and BIPAB perform equivalently.

So in conclusion, obesity hypoventilation syndrome is going to be seen increasingly because of the rising obesity epidemic.

It's associated with significant adverse health consequences, and research into treatment approaches other than positive airway pressure is needed for the future.

Thank you for your attention.