# OBESITY HYPOVENTILATION SYNDROME

# (OHS; "PICKWICKIAN SYNDROME")

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## ➢ Definition:

Is defined as the presence of awake alveolar hypoventilation (arterial carbon dioxide tension **[PaCO2] >45 mmHg**) in an obese individual **(body mass index ≥30 kg/m2)** which cannot be attributed to other conditions associated with alveolar hypoventilation (e.g., neuromuscular disorders).

## ➢ Pathogenesis:

- ⇨ Obesity (body mass index [BMI]> 30 kg/m2), in particular, severe obesity (BMI >50 kg/m2), is the major risk factor for OHS.
- ⇨ Obesity induces increased demand on the respiratory system that triggers compensatory mechanisms to maintain adequate ventilation.
- ⇨ It is thought that OHS develops due to the failure of such compensatory mechanisms resulting in hypoventilation and hypercapnia (PaCO2 ≥45 mmHg)
- ⇨ Complex interaction between the following factors is likely involved in the pathogenesis of OHS ⇩
  - Sleep disordered breathing (SDB)
  - Altered pulmonary mechanics
  - Impaired ventilatory control
  - Increased carbon dioxide production

## ➢ Risk Factors:

- Significant increase in waist: hip ratio (i.e., central obesity)
- Reduced lung function due to obesity
- Reduced inspiratory muscle strength
- Severe obstructive sleep apnea (OSA; e.g., apnea hypopnea index >50 events per hour).

## ➢ Clinical Manifestations:

The clinical manifestations of OHS are nonspecific and reflect the manifestations of

- Obesity
- Coexistent obstructive sleep apnea (OSA is present in 90 percent of OHS)
- Or of OHS-related complications (eg, pulmonary hypertension)

### Symptoms and signs

- Patients with OHS are obese (body mass index [BMI] >30 kg/m2)
- hypersomnolent individuals
- Ninety percent have coexisting OSA, which is often severe

**Symptoms and physical findings of OSA include**

- daytime hypersomnolence,
- loud snoring,
- choking during sleep
- fatigue,
- impaired concentration and memory,
- a small oropharynx, and a thick neck

✪ The ten percent of individuals with OHS who have coexistent sleep hypoventilation rather than OSA, present similarly, except witnessed apneas during sleep are less common.

## ➢ Late In The Disease: ( COMPLICATION )

⇨ Severe hypoxemic hypercapnic respiratory failure

While many patients present with chronic stable symptoms or chronic hypercapnic respiratory failure

⇨ Right heart failure from pulmonary hypertension (dyspnea on exertion, elevated jugular venous pressure, hepatomegaly, and pedal edema) and less commonly, facial plethora from polycythemia.

## ➢ DIAGNOSIS:

- ⇨ Polysomnography with continuous nocturnal carbon dioxide monitoring is the gold standard for the evaluation of patients suspected of having obesity hypoventilation syndrome (OHS).
- ⇨ ABG & ECHO
- ⇨ Spirometry : restrictive pattern

## When to suspect obesity hypoventilation?

- ➲ A strong clinical suspicion for OHS is critical for the diagnosis.
- ➲ We typically initiate evaluation in obese patients (body mass index [BMI] >30 kg/m2) with suspected or known OSA (or sleep disordered breathing), particularly those with severe OSA (e.g., apnea hypopnea index >60 events per hour, since the prevalence is high in this population).
- ➲ We also initiate evaluation in obese individuals with or without OSA who have the following clinical features:
  - Unexplained awake room air peripheral saturation (SpO2) ≤94 percent or an overnight nadir saturation <80 percent
  - Unexplained dyspnea on exertion
  - Symptoms and signs of pulmonary hypertension and/or right-sided heart failure (e.g., elevated jugular venous pressure, hepatomegaly, and pedal edema)
  - Facial plethora, which may indicate polycythemia

## ➢ TREATMENT:

For patients with OHS,

- ⇨ Immediate initiation of noninvasive positive airway pressure (PAP) together with lifestyle modifications for weight loss.
- ⇨ For patients with OHS who fail or do not tolerate first line therapies , options include tracheostomy for treatment of sleep disordered breathing and bariatric surgery