SPECIAL ISSUE 2: INVITED ARTICLE

Obesity Hypoventilation Syndrome

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International Journal of Head and Neck Surgery (2019): 10.5005/jp-journals-10001-1368

Case Description

A 67 years male, nonsmoker with BMI 37 kg/m^2, had complaints of progressive breathlessness with morning headaches. His wife gave history of snoring and witnessed choking spells. He had no known pulmonary disease in past. On examination, he looked drowsy but arousable and had crowded oropharynx. His oxygen levels on room air were 90%. On evaluation, blood gas analysis showed pH 7.34, pCO_2 48 mm of Hg and HCO_3 32 mEq/L. Spirometry revealed mild restriction. Echocardiography showed moderate pulmonary hypertension. PSG showed lowest SpO_2 65% and AHI 28/hr.

Obesity hypoventilation syndrome (OHS) or initially known as Pickwickian syndrome was recognized way before OSA in 1969, in history.[1] Hypoventilation in human system can occur due to various causes like sleep related hypoventilation syndrome, primary central hypoventilation, drug related hypoventilation and other medical disorders causing decreased ventilatory drive. With rising concern of obesity across the globe, obesity hypoventilation syndrome (OHS) has surfaced as a worrisome entity. OHS comprises of chronic alveolar hypoventilation in obese patients with accompanying sleep disordered breathing. Among all the forms of sleep disordered breathing, OHS has the worst prognosis with significant morbidity and mortality. OHS primarily is a diagnosis of exclusion in patients with suggestive symptoms.

Figure 1 shows that across the spectrum of sleep disordered breathing, toward left side surgical intervention can be advantageous when performed on right patient at right time. On the contrary, toward right side, option of surgical intervention becomes less prominent with more complications.

Epidemiology

Obesity hypoventilation syndrome presents usually in 5th and 6th decades of life. Unlike OSA, OHS does not present with any prominent gender predisposition. However, similar to OSA, condition presents in an atypical manner in females. No ethnic or racial dominance has been appreciated. Prevalence of OHS is higher in those with morbid obesity (BMI > 40 kg/m^2) or severe grades of OSA. Approximately, 10–20% of patients suffering from OSA get diagnosed with OHS eventually.[3]

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How to cite this article: Pandey A, Prabhudesai P. Obesity Hypoventilation Syndrome. Int J Head Neck Surg 2019;10(2):39–41.

Source of support: Nil

Conflict of interest: None

Pathophysiology

Obesity hypoventilation syndrome is largely a brunt of obesity intermixed with defect in ventilatory drive and upper airway obstruction. Obesity forces OHS patients to breath at low functional residual capacity (FRC) with diminished activity of diaphragm. Defect in ventilator drive is attributable to central leptin resistance. Leptin is a circulating protein released from adipose tissue which inhibits desire to eat more and acts as a respiratory stimulant. Additionally, these patients suffer from sleep disordered breathing. Around 90% of OHS patients demonstrate upper airway obstruction and remaining 10% have sleep hypoventilation. Table 1 depicts the current accepted definition of OHS.

Figure 2 shows constraints in OHS lead to a state of chronic hypoventilation with raised serum bicarbonate (>27 mEq/L), hypoxemia and hypercapnia.[4]

Clinical Presentation

Obese patients with or without underlying OSA presenting with morning headaches, extreme daytime lethargy or fatigability even while doing routine activity, leg swelling and exertional dyspnea should be taken as leads to suspect OHS in them. These patients when examined would appear exhausted and hypoxemic in daytime. A comparative analysis of patients with OHS, OSA and those with obesity is discussed in Table 2.

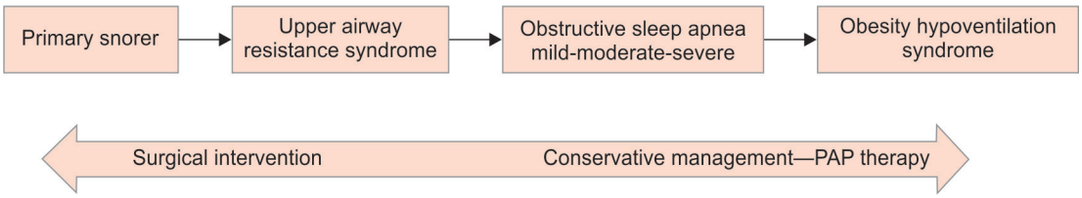


Fig. 1: Spectrum of sleep disordered breathing and therapeutics

Table 1: Definition of obesity hypoventilation syndrome[2]

Criteria	*Description*
Obesity	BMI >30kg/m^2
Chronic alveolar hypoventilation	Daytime awake PCO_2 >45 mm of Hg
Sleep disordered breathing (on polysomnography)	Evidence of OSA or sleep hypoventilation
Absence of other causes of hypoventilation	Severe obstructive airway disease, severe interstitial lung disease, severe neuromuscular disorders, severe hypothyroidism and central alveolar hypoventilation

BMI, body mass index; OSA, obstructive sleep apnea

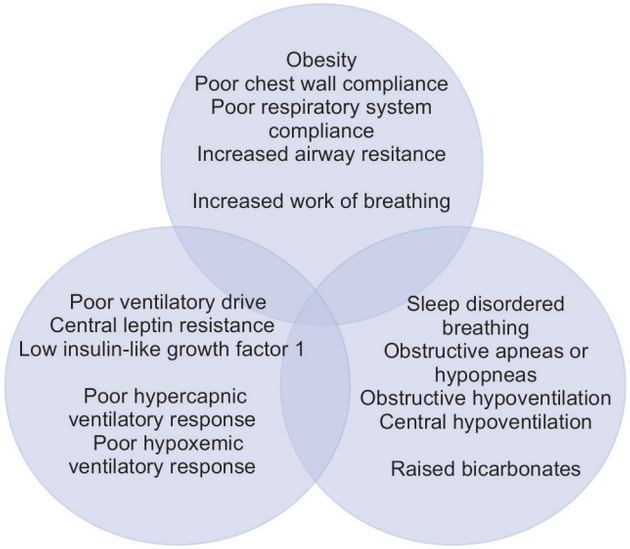
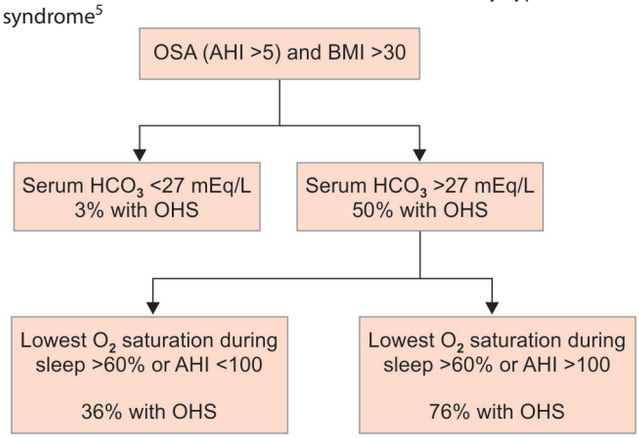


Fig. 2: Pathophysiology of obesity hypoventilation syndrome

Flowchart 1: Serum bicarbonates in evaluation of obesity hypoventilation syndrome[5]



Diagnosis

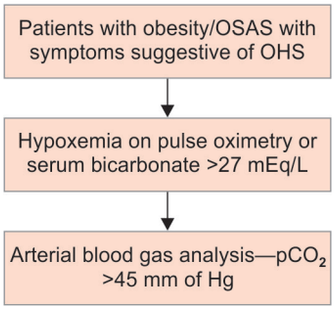
All OSA patients and those with morbid obesity of central distribution, should be carefully evaluated for clinical blinkers for OHS. Pulse oximetry showing awake hypoxemia should be looked for. Also, raised serum bicarbonate levels (>27 mEq/L) can be helpful in predicting chronic hypoventilation as depicted in Flowchart 1. Sensitivity of 92% and specificity of 50% makes this tool an acceptable screening modality for OHS. Subsequently, a blood gas analysis should be performed to confirm hypercapnia and hypoxemia. Sleep study should be performed to establish sleep disordered breathing. Pulmonary function test shows restrictive airway defect due to obesity. All other causes of hypoventilation as listed above should be excluded. It can be really challenging

Table 2: Comparison of patients with obesity, OSA and OHS

	Obesity	*OSA*	*OHS*
Symptoms			
Daytime drowsiness at rest daytime drowsiness	–	++	+++
while work	–	–	+
Exertional dyspnea	–	+	+++
Fatigability	+	++	+++
Morning headache	–	+	+++
Signs			
Facial plethora	–	+	+++
Features of pulmonary hypertension	–	–	+
BMI(kg/m^2)	>30	Normal or increased	>30
Laboratory parameters			
Daytime hypoxemia	–	–	+
Daytime hypercapnia	–	–	+
Serum bicarbonate	Normal	Normal	>27 mEq/L
Polycythemia	–	+	+++
Sleep study	AHI <5	AHI >5	AHI >5 or sleep hypoventilation
Prognosis			
Morbidity and mortality	fair	Increased	Markedly increased
Cardiovascular sequelae	–	+	++

OSA, obstructive sleep apnea; OHS, obesity hyperventilation syndrome

Flowchart 2: Algorithm for diagnosis of OHS



to rule out whether hypoventilation in a patient of OSA is due to accompanying chronic obstructive pulmonary disease (COPD) or OHS. If component of COPD is severe then hypoventilation can be attributable to pulmonary pathology than OHS.

Flowchart 2 shows an algorithm for diagnosis of OHS in suspected patients. For morbidly obese patients without evidence of OSA, Polysomnography study should be done showing AHI > 5/hr or sleep hypoventilation

In our case, patient is obese with moderate OSA on PSG. Raised bicarbonates and daytime pCO_2. He has accompanying pulmonary hypertension and other comorbities for hypoventilation. Thus, is a classical case of OHS.

Treatment

PAP Therapy

Approximately 90% patients with OHS have underlying OSA which needs to be addressed with PAP therapy. Treatment of OHS should be done under guidance of an expert at any time. All patients should receive a CPAP trial first[6] with fixed pressures and a backup rate. If patient stays comfortable with an improvement in oxygenation, daytime PCO_2 levels, bicarbonate levels and excessive daytime sleepiness, then CPAP should be continued on long-term basis. It has been found that 20–50% of OHS patients may not tolerate CPAP where bi-level PAP becomes treatment of choice. Thus, bi-level PAP should be opted for patients who do not tolerate CPAP and those with frank sleep hypoventilation. Long-term bi-level PAP therapy has been found to improve blood gas analysis, excessive daytime sleepiness, overall quality of life and frequency of hospitalization. IPAP takes care of alveolar ventilation and EPAP eliminates upper airway obstruction. Importantly, titration of these pressures needs technical expertise. Another effective mode is average volume assured pressure support (AVAPS), which is increasingly being used to combat ventilator struggle in OHS patients. PAP therapy needs high amount of counseling.

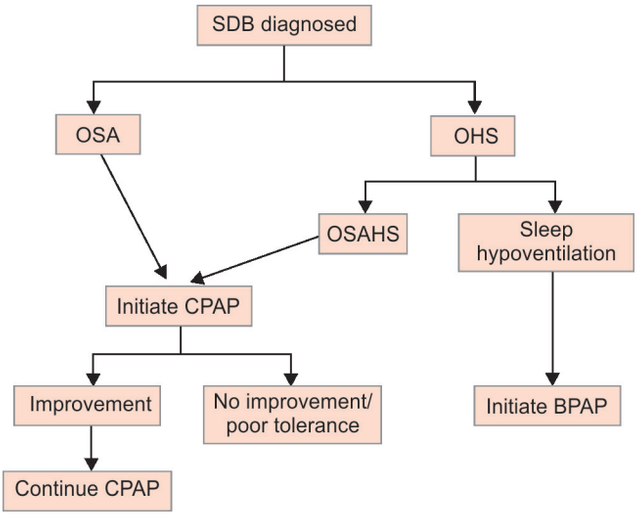
Other Alternatives or Adjunctive Therapies

Oxygen therapy—OHS patients usually suffer from concomitant pulmonary hypertension and thus should be treated with supplemental low flow oxygen support in conjunction to PAP therapy.

Tracheostomy—Those patients who fail to tolerate PAP therapy and have advancing disease require tracheostomy as an end resort followed by invasive PAP therapy.

Respiratory stimulant like medroxyprogesterone or acetazolamide can also be used in patients as an adjunctive therapy in advanced stage disease with an understanding that they do not alter the respiratory mechanics significantly.

Flowchart 3: Algorithm for PAP therapy in SDB



Bariatric surgery—For morbidly obese patient bariatric surgery may help deduct the burden on respiratory system. However, surgical intervention in such patients comes with real high risk.

Weight reduction with diet and exercise should be done in all the patients with OHS. With rising evidence on benefits of pulmonary rehabilitation, OHS patients should also be subjected to rehabilitation. Overall prognosis is worse in patients with OHS amounting to more than 50% mortality with most deaths occurring due to cardiovascular complications.[7]

Flowchart 3 shows an algorithm for evaluation of patients found to have sleep disordered breathing (SDB).

Pearls of Wisdom

- Early detection of OHS from subset of OSA should be tried for.
- Diagnosis of OHS should rely upon arterial blood gas analysis and serum bicarbonate levels.
- Treatment with PAP therapy is the standard line of therapy in current era.
- Prevention of complication is utmost important.
- Prognosis of OHS is worst amongst all the forms of sleep disordered breathing.

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