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Obesity Hypoventilation Syndrome Epidemiology and Diagnosis

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Keywords

Sleep-disordered breathing; Obesity hypoventilation syndrome; Sleep hypoventilation; Obstructive sleep apnea; Obesity; Morbid obesity

INTRODUCTION

The drumbeats have been sounding for some time now—the obesity epidemic has become a pandemic. By 2015, nearly 1 of 3 adults in the world are expected to be overweight (body mass index [BMI] ≥ 25 kg/m²) and almost 1 in 10 adults will be obese (BMI ≥ 30 kg/m²).¹ The health consequences of this “Obesity Era” are becoming more and more apparent. Among the myriad comorbidities associated with obesity is obesity hypoventilation syndrome (OHS), defined as the presence of diurnal alveolar hypoventilation (awake arterial P_{CO2} >45 mm Hg) among obese patients in the absence of other possible causes of hypoventilation.

Patients with OHS have higher rates of hospitalization and health care utilization, greater cardiorespiratory comorbidities, and higher mortality than obese-matched patients with obstructive sleep apnea (OSA) without hypoventilation.^{2,3} In light of these consequences, it seems imperative for clinicians to recognize and treat this condition appropriately, yet OHS remains frequently overlooked.^{4,5}

The aim of this article is to therefore increase clinician awareness of OHS by reviewing diagnostic criteria and current data on disease prevalence.

DEFINITION

Initial reports of OHS by Auchincloss and colleagues⁶ and Bickelmann and colleagues⁷ described patients with obesity, hypersomnolence, secondary erythrocytosis, pulmonary hypertension, and cor pulmonale. Nocturnal observation of these patients led to the first description of OSA⁸ and it was soon noted that a consistent proportion of OSA patients also exhibited daytime hypoventilation.^{9,10} During this period of investigation, considerable variation existed with respect to the definition of hypoventilation and OSA, and to establish standardized descriptions of sleep-breathing disorders, a task force of the American Academy of Sleep Medicine established a definition of OHS as the presence of daytime alveolar hypoventilation (awake, sea-level, arterial $P_{CO_2} > 45$ mm Hg) among patients with BMI ≥ 30 kg/m² in the absence of other causes of hypoventilation.¹¹ This definition also incorporated the observation that, although most patients with OHS had concurrent OSA, approximately 10% had no evidence of nocturnal OSA; these patients developed sleep-related hypoventilation, particularly during REM sleep.^{12,13} This point is relevant because, although the definition suggests a diurnal pathologic abnormality, overnight polysomnography is required to determine the pattern of nocturnal hypoventilation (obstructive or nonobstructive) and to individualize therapy based on an adequate titration study.

DIAGNOSIS

Importantly, OHS is a diagnosis of exclusion, and suspected patients should be evaluated for other possible causes of hypoventilation, such as under-lying obstructive respiratory disease, mechanical respiratory limitations (eg, kyphoscoliosis), neuro-pathic and myopathic conditions, and central causes (eg, cerebrovascular disease, severe hypothyroidism) (Box 1).^{14,15} Furthermore, suspected patients should be screened for factors that can exacerbate hypoventilation, such as the use of sedative-hypnotics, opiates, or alcohol.¹³

OHS is frequently first diagnosed either when an afflicted patient reaches a high state of acuity, in the form of acute-on-chronic hypoventilatory failure, or alternatively, when ambulatory care is escalated to evaluation by pulmonary or sleep specialists.^{2,4,16} Unfortunately, a delay in diagnosis is common; the diagnosis typically occurs during the fifth and sixth decades of life,⁵ and during that delay, OHS patients use more health care resources than comparably obese normocapneic patients.³

Patients with OHS tend to be morbidly obese (BMI ≥ 35 kg/m²), have severe OSA (≥ 30 obstructive respiratory events per hour of sleep), and are typically hypersomnolent. Compared with patients with OSA, patients with OHS are more likely to report dyspnea and manifest cor pulmonale. Table 1 paints the typical portrait of an OHS patient, based on the clinical features¹⁶⁻¹⁹ of a large combined cohort of OHS patients reported in the literature.⁵

Several investigators have suggested incorporating serum bicarbonate (HCO_3^-) levels into the definition of OHS, particularly given that using a single measurement of arterial P_{CO_2} for OHS diagnosis is susceptible to several confounding factors, including the impact of periprocedural patient anxiety leading to hyperventilation.¹⁷ Mokhlesi and colleagues¹⁸ first demonstrated that a venous serum bicarbonate threshold of 27 mEq/L, suggestive of chronic

respiratory acidemia from hypoventilation, could be used for OHS diagnosis. Their data demonstrated that among obese patients with OSA, a serum bicarbonate level less than 27 mEq/L had a 97% negative predictive value for excluding a diagnosis of OHS. Macavei and colleagues¹⁹ assessed earlobe capillary blood gas samples from patients referred to a sleep center and determined that bicarbonate values calculated from the Henderson-Hasselbach formula have similar predictive value. A calculated serum bicarbonate level of 27 mEq/L had a sensitivity of 85% and a specificity of 89% for the diagnosis of OHS among their patient sample.

In addition to blood gas sampling and venous bicarbonate assessments, daytime finger pulse oximetry may be a valuable tool for clinicians in screening for possible OHS. Hypoxemia during wakefulness is not a typical feature of patients with either OSA or obesity. Therefore, abnormal pulse oximetry during wakefulness should prompt an evaluation for OHS among obese OSA patients.¹³

RISK FACTORS

Obesity, and in particular, morbid obesity, is a pre-dominant risk factor for OHS.²⁰ However, although all OHS patients are obese, not all patients with obesity, or even morbid obesity, develop OHS. It is worth noting that there are significant differences between obese patients who develop OHS compared with similarly obese patients without OHS, as summarized in Table 2.²⁰

In the same light, the severity of concurrent OSA also appears to be a significant risk factor for OHS,²⁰ but not all patients with even extremely severe OSA have OHS. In fact, even among patients with an apnea-hypopnea index 100/h and serum bicarbonate 27 mEq/L, 24% do *not* meet criteria for OHS,¹⁸ suggesting nonobstructive pathophysiologic factors for OHS even among these very severe OSA patients.

Male gender does *not* appear to confer a significant risk for OHS, in contrast to uncomplicated OSA.²¹ Similarly, no race-specific or ethnic-specific risk has been demonstrated thus far, although it has been speculated⁵ that African Americans might have a higher risk for OHS simply because of the higher prevalence of morbid obesity in this race compared with others.^{22,23}

PREVALENCE

More than 20 studies regarding the prevalence of OHS have been published since the syndrome was first described. Prevalence estimates for OHS vary significantly across studies, owing partly to differences in sample characteristics, disease definitions, and assessment procedures.

Because most OHS patients have coexisting OSA, most prevalence studies have focused on patients referred to sleep centers for evaluation of sleep-disordered breathing, and a reasonable prevalence estimate among OSA patients has emerged. At least 20 studies have examined this prevalence. Of these, only approximately 10 studies either included coprevalence data for obstructive pulmonary disease^{9,18,19,24-26} or excluded these patients

entirely^{4,27-29} and allow for an assessment of the prevalence of OHS without these patients (Table 3). From these 10 studies, the aggregate prevalence of OHS among OSA patients referred to a sleep disorder center is 17% (range 4%–50%). The considerable range in prevalence reflects varying patient populations across studies. For example, the mean BMI of patients in the 2 studies with the lowest prevalence^{25,28} was 34 kg/m² and was considerably lower than the mean BMI of 59 kg/m² from the study with the highest prevalence.²⁹

The total prevalence of OHS in the general population is unknown but can be estimated using OSA prevalence data, as illustrated by Mokhlesi.⁵ If approximately 6% of the general US population has severe obesity (BMI ≥ 40 kg/m²),²² and if it is conservatively estimated that half of patients with this degree of obesity will have OSA (although some studies actually suggest a prevalence of OSA >70% in patients with this degree of obesity who are referred for bariatric surgery)³⁰⁻³² and that approximately 20% of these OSA patients will have OHS, then the estimated prevalence of OHS can be estimated as roughly 0.6% (approximately 1 in 160 adults in the US population; Fig. 1). This estimate does not take into account the ~10% of OHS patients who do not have significant OSA, but this figure may also overestimate the frequency of OHS in countries where the prevalence of severe obesity is lower.

Several studies have shed light on the increasing prevalence of OHS as the degree of obesity worsens (Fig. 2). Among patients with OSA, the prevalence of OHS at a BMI of 30 to 35 kg/m² is 8% to 11% among non-Asian populations and increases to 18% to 31% at a BMI ≥ 40 kg/m².^{5,18,19,28} At the extreme, Banerjee and colleagues²⁹ evaluated patients referred for overnight polysomnography with a BMI ≥ 50 kg/m² and found an OHS prevalence of 50% in this cohort.

East Asian populations are known to develop OSA at a lower BMI compared with other populations because of cephalometric differences.³³ Therefore, in these populations, OHS may be more prevalent at a lower BMI range than in non-Asian populations. Indeed, the average BMI of 291 OHS patients from 4 studies involving Japanese subjects was 32 kg/m²,^{26,27,34,35} compared with an average BMI of 44 kg/m² from 757 OHS patients from 10 studies involving mostly non-Asian subjects.⁵

Because patients undergoing bariatric surgery are generally more obese, the prevalence of OHS might be expected to be higher among these patients and, indeed, 2 studies support this notion. Sugerman and colleagues³⁶ found that 65% of their bariatric surgery OSA patients had OHS. Domínguez-Cherit and colleagues³⁷ found that 18 of 37 (49%) patients undergoing bariatric surgery in their cohort had either hypercapnia or hypercapnia + hypoxemia; the mean BMI in their study was 50 kg/m². In contrast, however, Lecube and colleagues³⁸ evaluated asymptomatic women referred to a bariatric clinic. Of 66 of these subjects diagnosed with OSA based on limited polygraphy, their study found that only 7 (11%) had OHS; the lower prevalence in the study could be attributed to recruitment of only asymptomatic subjects as well as OSA diagnosis based on limited polygraphy.

Hospitalized patients are also known to have a higher prevalence of OHS and may suffer excess morbidity and mortality because of a lack of appropriate OHS therapy. Of 277 patients with a BMI ≥ 35 kg/m² admitted to an inpatient medical service, Nowbar and colleagues⁴ found that 31% met criteria for OHS. Reinforcing the association with obesity, when a subgroup of patients with BMI greater than 50 kg/m² was examined, OHS was found in 48%. Of particular concern, (though likely representative of many hospitals), effective therapy for hypoventilation was only instituted in 13% of these patients. This lack of recognition may have important consequences: compared with patients with simple obesity, OHS patients in this study were more likely to require intensive care and invasive mechanical ventilation, and strikingly, these patients had nearly twice the mortality 1.5 years after hospital discharge. Marik and Desai³⁹ found that 8% of *all admissions* to a general ICU met criteria for OHS. All OHS patients had been admitted with acute on chronic hypercarbic respiratory failure, and of these patients, nearly 75% were misdiagnosed and treated for obstructive lung disease despite having no evidence of obstruction on pulmonary function testing.

ASSOCIATED MORBIDITY

As already noted, most OHS patients are morbidly obese and have severe OSA.¹⁸ Although obesity⁴⁰ and severe OSA⁴¹ are associated with decreases in quality of life, OHS may contribute to further quality-of-life decrements.⁴² Furthermore, quality-of-life ratings among OHS patients appear to be lower than those with hypoventilatory respiratory disorders, such as obstructive lung disease.⁴³

The medical morbidity associated with a diagnosis of OHS can be quite varied as illustrated by Jennum and colleagues,⁴⁴ who evaluated 755 patients with a diagnosis of OHS from a Danish national patient registry and found that, in the 3 years before OHS diagnosis, these patients were more likely than age-matched and gender-matched controls to be diagnosed with many conditions, including cellulitis, carpal tunnel syndrome, diabetes, congestive heart failure, obstructive lung disease, and arthritis of the knee. It remains unclear if these conditions would be more prevalent than in an obese-matched cohort with uncomplicated OSA.

Cardiovascular morbidity is of particular concern in OHS. Kessler and colleagues² found a pulmonary hypertension prevalence of 58% among a cohort of 34 OHS patients compared with just 9% among a sample of similar OSA patients. Similarly, Berg and colleagues³ evaluated 20 OHS patients from a Canadian health registry and compared them with obese matched controls. OHS patients in their study were 9 times more likely to have a diagnosis of cor pulmonale and 9 times more likely to have a diagnosis of congestive heart failure.

SUMMARY

OHS prevalence is expected to increase, following global trends in obesity, and because significant morbidity is associated with this disease, timely diagnosis and appropriate therapy are imperative. The traditional criteria for OHS diagnosis include the presence of daytime alveolar hypoventilation (awake, sea-level, arterial P_{CO2}>45 mm Hg) among patients with BMI ≥ 30 kg/m² in the absence of other causes of hypoventilation, and

incorporating finger pulse oximetry and serum bicarbonate screening will likely aid in improving diagnosis. The major risk factors for OHS include obesity and OSA; therefore, a high index of suspicion is needed in these patients, particularly in the inpatient setting and before bariatric surgery.

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Box 1 Diagnostic features of OHS

Obesity

- BMI ≥ 30 kg/m²

Chronic hypoventilation

- Awake daytime hypercapnia (sea-level arterial P_{CO2} ≥ 45 mm Hg, P_{O2} < 70 mm Hg)
- Possible role of serum venous bicarbonate or calculated capillary blood gas bicarbonate greater than 27 mEq/L*

Sleep-disordered breathing[†]

- Obstructive sleep apnea (apnea-hypopnea index [AHI] ≥ 5 event/h)
- Nonobstructive sleep hypoventilation (AHI < 5 events/h, P_{CO2} increases by ≥ 7 mm Hg during sleep or oxygen saturation $\leq 88\%$ for at least 5 min without obstructive respiratory events)

Exclusion of other causes of hypoventilation

- Severe obstructive respiratory disease
- Severe interstitial lung disease
- Severe chest-wall disorders (eg, kyphoscoliosis)
- Severe hypothyroidism
- Neuromuscular disease
- Congenital hypoventilation syndromes

* Serum bicarbonate level assessment is not part of the definition but advocates suggest using this for screening.

[†] OSA is present in 90% of cases, with the remaining cases featuring nonobstructive sleep hypoventilation.

KEY POINTS

- Obesity hypoventilation syndrome (OHS) is defined as daytime alveolar hypoventilation (awake, sea-level, arterial $P_{CO_2} > 45$ mm Hg) among patients with body mass index ≥ 30 kg/m² in the absence of other causes of hypoventilation.
- Ninety percent of OHS patients have concomitant obstructive sleep apnea (OSA).
- Incorporating elevated serum bicarbonate levels and/or low finger pulse oximetry may augment OHS screening among obese OSA patients.
- The overall prevalence of OHS is estimated to be approximately 0.6% of the general adult population.
- OHS prevalence increases as the degree of obesity increases.

Fig. 1.

Estimated prevalence of OHS in the US adult population. The overall prevalence of OHS can be estimated using severe obesity (BMI >40 kg/m²) and OSA prevalence data. This estimate is likely conservative, because it does not take into account the roughly 10% of OHS patients who do not have OSA. OHS may be more prevalent in the United States because of the higher prevalence of severe obesity than other countries.

Fig. 2.

Prevalence of OHS in patients with OSA, sorted by BMI. In the UK study, the mean BMI was nearly 40 kg/m² and 38% of subjects had a BMI greater than 40 kg/m².¹⁹ Similarly, in the US study, the mean BMI was 43 kg/m² and 60% of subjects had a BMI greater than 40 kg/m².¹⁸ In contrast, the mean BMI in the French study was 34 kg/m² and only 15% of subjects had a BMI greater than 40 kg/m².²⁸ Italian data were provided by Onofrio Resta, personal communication.⁵

Table 1
Clinical features of patients with OHS

Clinical Features	Mean (Range)
Age (y)	52 (42–61)
Male (%)	60 (49–90)
BMI (kg/m ²)	44 (35–56)
Neck circumference (cm)	46.5 (45–47)
pH	7.38 (7.34–7.40)
Arterial P _{cO2} (mm Hg)	53 (47–61)
Arterial P _{O2} (mm Hg)	56 (46–74)
Serum bicarbonate (mEq/L)	32 (31–33)
Hemoglobin (g/dL)	15 (ND)
Apnea-hypopnea index (events/h)	66 (20–100)
Oxygen nadir during sleep (%)	65 (59–76)
Percentage time S _{pO2} <90%	50 (46–56)
FVC (% predicted)	68 (57–102)
FEV ₁ (% predicted)	64 (53–92)
FEV ₁ /FVC	0.77 (0.74–0.88)
Medical Research Council dyspnea class 3 or 4 (%)	69 (ND)
Epworth sleepiness scale score (out of 24)	14 (12–16)

Features are based on aggregated sample of 757 patients from 15 studies.

Abbreviations: FEV₁, forced expiratory volume in first second; FVC, forced vital capacity; ND, no data to calculate a range.

Data from Mokhlesi B. Obesity hypoventilation syndrome: a state-of-the-art review. *Respir Care* 2010;55(10): 1347–65.

Table 2
Physiologic differences between morbidly obese patients and those with OHS

	Normocapnic Morbid Obesity	Obesity Hypoventilation Syndrome
Waist:hip ratio	↑	↑↑
FEV ₁ /FVC	Normal	Normal/↓
Total lung capacity	Normal	Slight ↓
Functional residual capacity	↓	↓
Vital capacity	Normal/↓	↓↓
Expiratory reserve volume	↓	↓↓
Work of breathing	↑	↑↑
Hypercapnic/hypoxic ventilatory drive	Normal	↓
Inspiratory muscle strength	Normal	↓

Abbreviations: FEV₁, forced expiratory volume in first second; FVC, forced vital capacity.

Table 3
OHS prevalence and characteristics among patients with OSA^a

Author, Country	Year	Patients (No.)	Male (%)	Age (y)	BMI (kg/m ²)	AHI (Events/hr)	OHS (No.)	OHS (%)
Leech, ^{b,c} US ⁹	1987	111	68	47	0.71 ^d	58	41	37
Resta, ^{b,c} Italy ²⁴	2000	219	64	50	40	45	38	17
Verin, ^{b,c} France ²⁵	2001	218	92	55	34	51	21	10
Akashib ^b Japan ²⁷	2002	143	100	48	30	55	55	38
Nowbar, ^{b,e} US ⁴	2004	150	53	53	43	N/R	47	31
Laaban & Chailleux, ^b France ²⁸	2005	1141	83	56	34	55	126	11
Mokhlesi, ^{b,c} US ¹⁸	2007	522	56	48	44	59	124	24
Kawata, ^{b,c} Japan ²⁶	2007	1227	89	50	29	42	168	14
Banerjee, ^{b,f} Australia ²⁹	2007	74	54	43	59	62	23	31
Macavei, ^{b,c} UK ¹⁹	2013	344	64	52	39	25	71	21
Aggregate		4149					714	17

Data are mean or median values provided in each article. Aggregate prevalence calculated by dividing total patients diagnosed with OHS by total patients studied.

Abbreviations: AHI, apnea-hypopnea index; BMI, body mass index; N/R, not reported; OHS, Obesity hypoventilation syndrome; UK, United Kingdom; US, United States.

^aOSA defined variably as between AHI 5 up to AHI 15.

^bGender, Age, BMI, and AHI values are given as the mean of all patients (ie, OSA and OHS patients) and were calculated from the data provided by the authors in the article.

^cDid not exclude patients with obstructive deficits on spirometry in data presented here.

^dBMI not reported; value given as weight/height ratio reported in kg/cm.

^eStudy population comprised of patients hospitalized in medical non-ICU setting with BMI 35 kg/m².

^fEnrolled consecutive patients with BMI 50 and OSA (defined as AHI 5).