Health Leadership and Quality of Life. 2025; 4:612
doi: 10.56294/hl2025612

ORIGINAL

Obesity-Related Breathing Disorders: Diagnosis and Treatment of Hypoventilation Syndrome

Trastornos Respiratorios Relacionados con la Obesidad: Diagnóstico y tratamiento del síndrome de hipoventilación

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Cite as: Mishra P, Gupta S, Devmurari P, Raj N, Priya Natarajan N, Kumar A. Obesity-Related Breathing Disorders: Diagnosis and Treatment of Hypoventilation Syndrome. Health Leadership and Quality of Life. 2025; 4:612. https://doi.org/10.56294/hl2025612

Submitted: 02-06-2024 **Revised:** 26-10-2024 **Accepted:** 24-05-2025 **Published:** 25-05-2025

Editor: Neela Satheesh

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ABSTRACT

Introduction: obesity-related respiratory problems, such hypoventilation syndrome, are on the rise and have depressing property on one's health. The characteristic of hypoventilation syndrome are shallow breathing and low blood oxygen levels, which consequence in symptoms similar to fatigue, headache, and cognitive defeat.
Method: the analysis of this situation necessitates a mixture of clinical assessment, pulmonary function testing, and arterial blood gas capacity.Treatment options comprise weight defeat, positive airway pressure (PAP) treatment, and severe cases, tracheostomy or ventilator maintain.
Results: hypoventilation condition can enhance quality of life (QoL) and decrease the possibility of potentially fatal difficulty like heart failure and pulmonary hypertension (PH) with early diagnosis and treatment.
Conclusion: the research, we recognize the disease's diagnostic individuality and inspect the path physiology, morbidity, and mortality that are connected. The research covers the frequent treatment alternative obtainable nowadays as final point.

Keywords: Obesity, Breathing Disorders; Hypoventilation; Positive Airway Pressure Treatment.

RESUMEN

Introducción: los problemas respiratorios relacionados con la obesidad, como el síndrome de hipoventilación, van en aumento y tienen efectos depresivos sobre la salud. Las características del síndrome de hipoventilación son la respiración superficial y los bajos niveles de oxígeno en sangre, que tienen como consecuencia síntomas similares a la fatiga, la cefalea y la derrota cognitiva.
Método: El análisis de esta situación requiere una combinación de evaluación clínica, pruebas de la función pulmonar y gasometría arterial. Las opciones de tratamiento incluyen la reducción de peso, el tratamiento con presión positiva en las vías respiratorias (PAP) y, en los casos graves, la traqueotomía o el mantenimiento con ventilador.
Resultados: la condición de hipoventilación puede mejorar la calidad de vida (CdV) y disminuir la posibilidad de dificultades potencialmente fatales como insuficiencia cardíaca e hipertensión pulmonar (HP) con

diagnóstico y tratamiento precoz.
Conclusión: la investigación reconoce la individualidad diagnóstica de la enfermedad e inspecciona la fisiología de la patología, la morbilidad y la mortalidad que están conectadas. La investigación cubre la alternativa de tratamiento frecuente obtenible hoy en día como punto final.

Palabras clave: Obesidad; Trastornos Respiratorios; Hipoventilación; Tratamiento con Presión Positiva en la Vía Aérea.

INTRODUCTION

The obesity pandemic is presently distressing millions of people worldwide. Obesity is a main provider to breathing difficulties in addition to a lot of health danger it entails. Particularly severe respiratory harms like Hypoventilation Syndrome (HS) are regularly experiential in obese people. The over HS diagnosis and therapy.[1] When someone has HS, have less ventilation, which raises their body's levels of carbon dioxide. Numerous daytime tiredness, morning headaches, and breathing harms, while sleep, are a small number of indication that the confusion can create. Because of the buildup of fat in the upper body and belly, which can limit lung mobility and create breathing difficult, HS is additional common in obese people.[2] A thorough the patient's physical assessment, test data, and therapeutic history is necessary to analysis HS. To assess lung function, the doctor also conducts pulmonary function tests like spirometry. To assess the patient's breathing patterns during sleep, the sleep investigation can also be recommended.[3] The most popular therapy for HS is non-invasive positive pressure ventilation (NIPPV), which involves using a machine to provide air pressure to the lungs while sleep. The prevention of carbon dioxide buildup in the body and the maintenance of open airways are two benefits of the therapy. NIPPV helps patients with HS live better lives overall by reducing symptoms including excessive daytime sleepiness. Losing weight is another HS treatment that works. Weight loss can greatly lessen HS symptoms. Obesity is the primary cause of HS. Losing weight helps lessen lung pressure and enhance lung capacity, which can improve breathing and lower blood carbon dioxide levels.[4] Surgery can be required to treat HS in some circumstances. Gastric bypass and other bariatric procedures can help people lose weight and enhance lung function, which lessens the symptoms of HS. Surgery, however, is often viewed as a last choice when all other options have failed. Patients with HS can benefit from lifestyle changes in addition to the aforementioned therapies, such as abstaining from alcohol and sedatives, giving up smoking, and keeping a regular sleep pattern. These changes can aid in breathing improvement and lessen the intensity of HS symptoms.[5]

A serious respiratory condition known as HS is frequently observed in obese people. In addition to extreme daytime sleepiness, it can also result in morning headaches and respiratory problems while sleep. Crucial elements of the diagnosis of HS include the evaluation of the patient's medical history, physical examination, and laboratory testing. NIPPV, which includes utilizing a machine to give air pressure to the lungs during sleep, is the most widely used treatment for HS. In addition to improving lung function and reducing the intensity of symptoms, weight loss is a successful treatment for HS. A regular sleep schedule, refraining from alcohol and sedatives, and giving up smoking are among lifestyle changes that can be beneficial for HS patients. Right-sided cardiac failure and pulmonary hypertension are two major consequences of HS that can develop if it is not treated.[6] After checking out other causes of hypoventilation, obese individuals with daytime hypercapnia and sleep breathing difficulties develop obesity hypoventilation syndrome (OHS). Obstructive sleep apnea (OSA) is concurrent in the majority of individuals (approximately 90 %). Daytime hypersomnolence, snoring, exhaustion, and memory loss are a few symptoms. The aim of the diagnosis is to exclude any other potential reasons for alveolar hypoventilation and elevated blood pressure, as well as to gather proof of a lack of ventilation through polysomnography and hypercapnia. It raises mortality and is linked to metabolic and cardiovascular comorbidities such as high blood pressure, congestive heart failure, and insulin resistance. Though it is recommended that non-invasive positive airway pressure (PAP) treatment, its functions alongside non-invasive ventilation (NIV) and continuous positive airway pressure (CPAP). Both treatments seem to be equally beneficial in persons with OSA as their primary symptom.[7] OHS, or obesity-related body mass index (BMI) combined with hypercapnia during the day and sleep disordered breathing (SDB), is a condition that develops when all other possible causes of alveolar hypoventilation have been excluded with certainty. A delayed diagnosis has the potential to cause an enormous amount of cardio respiratory morbidity, including coronary disease, hypertension, and pulmonary heart failure. The primary factors contributing to the clinical state and poor quality of life include oxidative stress, sleep disruption, and obesity-related reconditioning. The diagnosis requires proper knowledge of its existence in the right clinical environment, together with a high degree of suspicion. Sleep research and arterial blood gases (ABGs) serve as the foundation for establishing the diagnosis with certainty. A comprehensive strategy for management is necessary, focusing on comorbidity

therapy, weight loss, lifestyle changes, and SDB control using CPAP, noninvasive ventilation, and pulmonary rehabilitation. The quality of life is enhanced and an optimistic prognosis is made possible by timely therapy.[8] The range of respiratory issues associated with obesity and their effects are generally underappreciated. To determine the main causes of hypoventilation, the features of obese individuals with euxinic obstructive sleeping apnea, obesity-related sleep hypoventilation (ORSH), and OHS. Using body plethysmography, blood gas analyses, transcutaneous capnometry, and measurements of the hypercapnic ventilatory response (HCVR), obese patients with OHS, ORSH, and OSA, were identified in the prospective, diagnostic investigation. To find the main causes of hypoventilation, a multivariable logistic regression analysis was used. Potential independent variables were identified as pathophysiological elements that are known to induce hypoventilation and varied considerably throughout the groups.[9] There have been recently described new classification criteria for OHS. Modifications to the criterion have not been observed in situations of OSA OHS prevalence. OHS prevalence has not previously been documented in India.[10]

Both obesity hypoventilation syndrome and obstructive sleep apnea are sleep-disordered breathing disorders (SBD) whose prevalence has increased globally along with that of obesity. These high-risk patients are more likely to experience higher perioperative mortality and morbidity due to comorbid diseases that are present, challenging intubation, and postoperative cardiorespiratory problems. A significant number of SDB patients do not acquire a diagnosis at the time of surgery as a result of a lack of knowledge among the perioperative team members. The perioperative outcome might be improved by identifying these dangerous patients and making preoperative adjustments.[11] A person with a BMI of more than 30 kg/m^2 can experience daytime alveolar hypoventilation that is unrelated to any other disease. The condition is known as OHS. The prevalence of the ailment is rising and with it the cost to healthcare systems around the world. The syndrome's well-known consequences include a right heart attack and pulmonary hypertension. In the example recounted here, a female patient with OHS who had considerable pulmonary hypertension when came to the hospital was properly treated and their condition improved. Also go through the treatment options for medical condition and its diagnosis.[12] OHS is a result of a complicated interaction between alterations in central ventilatory drive brought on by obesity, nap-disordered breathing, variations in respiratory mechanics, and neurohormonal changes. Given the related morbidity, diagnosis is crucial; nevertheless, the validity of relying on one measurement of partial pressure of arterial carbon dioxide ($PaCO_2$) in arterial blood gas has been interrogated. PAP is a proven treatment for OHS symptoms, sleep-disordered breathing, and improved gas exchange.[13] OHS is frequently associated with PH. There is, however, a dearth of information evaluating the pathogenic variables connected to PH. The goal is to evaluate potential risk factors for the etiology of PH without treatment OHS. A post hoc analysis of the Pickwick trials included bivariate research of the initial characteristics of individuals with and without PH. Theoretical pathogenic pathways were used to classify factors with a P value of 10 as probable risk factors across various adjusted models. The same research was performed on the two OHS phenotypes, one of which had significant simultaneous obstructive breathing while sleeping.[14] OSA and OHS can make clinical manifestation and therapy more difficult. The provided systematic approach to accurately diagnose OSA and OHS overlap patients and to optimize their management. Additionally, several advice and pitfalls are provided, including the need for routine ABG sampling in patients who are extremely obese of hypercapnia screening, thorough monitoring during the first three months after starting PAP treatment, and pressure titration targets that concentrate on both the correction of apnea/hypopnea and the restoration of oxygenation (hypoxemia/hypercapnia).[15]

In cases of elevated BMI (>30 kg/m^2) and persistent carbon dioxide retention, the diagnosis of OHS is made based on exclusion. OSA is the most common type of patient, and corpulmonale can occur. A dysfunctional respiratory system, alterations in ventilator control, and an increased load on the lungs are all parts of the pathophysiology. High serum bicarbonate levels found in obese patients should be checked as part of the OHS screening process. A low rest pulse oximetry value (94 %) should also be taken seriously.[16] Although it is crucial for risk prediction to understand the determinants of OHS, a disorder linked to increased morbidity and mortality, the current categorization is incomplete.[17] PAP treatment is recommended in longitudinal studies to treat OSA to reduce the risk of cardiovascular disease. The expected advantage, though, is not always present. To look at how PAP treatment affects naive endothelium cells after circulating exosomes from different people are released to the bloodstream to determine whether PAP therapy results in immediate changes in the function of endothelial cells, a preclinical marker of cardiovascular state.[18] Morbidity and death rates are higher for OHS patients. Exosomes play a part in the development and spread of tumors after discharged in the bloodstream. 24 OHS patients (13 men and 11 women) were assessed before starting CPAP therapy as well as three, twelve, and twenty-four months later. Naive NCI-H23 lung carcinoma cells were treated with plasma exosomes.[19] According to current recommendations, individuals with OHS should be voluntarily admitted to starting receiving home NIV while hospitalized. The idea was that an outpatient NIV arrangement would be more economical.[20]

Epidemics and clinical presentations

The average rate of OHS amongst patients with OSA, however, has been estimated to be between 10 % and 20 %, and it is higher in the subset of individuals who are extremely obese. Though the majority of OHS patients had previously been hospitalized, the official diagnosis of OHS is typically made in the 5th or 6th decade of life, following a discussion with a respiratory and medical expert. Most patients have the usual symptoms of OSA, which include severe daytime sleepiness, loud snoring, nocturnal choking episodes with documented apneas, and morning migraine. Unlike those with euxinic OSA, those with stable OHS commonly express their dyspnea complaints and can have corpulmonale symptoms. Numerous obese patients with an expanded neck circumference, a crowded oropharynx, a pronounced (P2) on the cardiac auscultation, and lower leg edema can be seen during a physical examination. Table 1 lists the clinical characteristics of 757 OHS patients that have been documented in the literature. Additionally, when treating patients with sleep disorders, physicians should rule out hypercapnia and monitor hypoxaemia using air in the room pulse oximetry while the patient is awake. Hypercapnia is obvious, chest imaging and lung function testing should be performed to rule out other reasons.

Table 1. Obesity Hypoventilation Syndrome Patients' Clinical Features

Variable	Mean (range)
Age, yr	52(42-61)
Bodymassindex, kg/m^2	44(35-56)
Men, %	60(49-90)
pH	7,38(7,34-7,40)
Pa_0, mmHg	56(46-74)
Neck circumference, cm	46,5(45-47)
Pa_{co}, mmHg	55(47-61)
Hemoglobin, g/dl	17
Serumbicarbonate, mEq/L	33(31-33)
Oxygen nadirduring sleep, %	67(59-76)
Apnea-hypopneaindex	67(20-100)
FVC,%pred	61(57-102)
Percenttime Sa_0lessthan90%,%	55(46-56)
FEV_1/FVC	79(74-88)
FEV_1,%pred	64(53-92)
Ep worth Sleepiness Scale, score	17(12-16)
MedicalResearchCouncildyspneaclass3and4,%	72

Pathophysiology

Morbid obesity causes hypoventilation through a complicated and poorly understood mechanism. The pathophysiology of OHS has been explained by several processes, including aberrant respiratory mechanics brought on by obesity, decreased brain responses to hypoxia and hypercapnia, sleep-disordered respiration, and neurohormonal abnormalities including eptin resistance (figure 1). The importance of sleep-related breathing difficulties in the pathophysiology of hypoventilation is well known, PAP treatment or tracheostomy usually cures the hypercapnia of OHS patients without affecting body mass CO2 production or the amount of empty space concurrently.

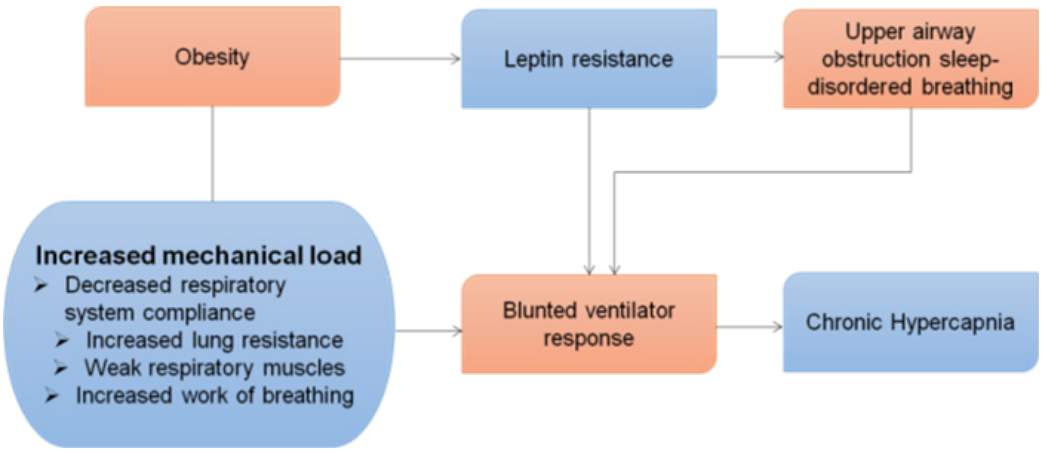

Figure 1. Chronic daytime hypercapnia brought on by obesity: its causes and mechanism

https://doi.org/10.56294/hl2025612

Fraudulence and death

Obesity and breathing problems are the defining pathogenic features in OHS, therefore it makes sense that these two conditions would account for the majority of the clinically recorded morbidity. In the five years prior to OHS being formally diagnosed, according to colleagues' research, OHS patients utilize healthcare services at a substantially higher rate than obese individuals who do not experience hypoventilation or the general population control group. This group was able to differentiate between the comorbidities associated with the obesity parse and those associated with hypoventilation by comparing the utilization of healthcare in cases versus obese and usual control of population patients.

Deaths caused by hypoventilation

Congestive heart failure was statistically substantially more likely to be detected in individuals with OHS compared to obese control participants Patients with OHS had greater rates of admittance to the intensive care unit and required invasive mechanical ventilation than those without hypoventilation but with a comparable degree of fat were also more likely to be hospitalized.

Obesity-related morbidity

Obesity is a feature of the metabolic disorder and is linked to numerous medical issues. Therefore, in addition to morbidities linked to hypoventilation, patients with OHS are more likely to experience morbidities spanning multiple organ systems. Particularly, the lot of probable to be identified with osteoarthritis, arterial hypertension, and hypothyroidism. There is a greater chance of hepatic dysfunction and hyperlipidemia, both of which are likely connected to obesity. According to other reports, patients with OHS had a greater incidence of pulmonary hypertension than eucapnic OSA patients. Given the numerous comorbidities, it is unsurprising that eucapnic people with OSA who match for ages, BMI, and compared to individuals with OHS, lung function patients have a worse quality of life (QOL).

Mortality

Prior research revealed that hospitalized individuals with OHS had a significant mortality rate; however 64 patients in recent prospective trials reported no in-hospital deaths. In contrast, untreated breathing problems increases the chance of mortality, as shown in retrospective research in which 7 of fifteen individuals who declined Noninvasive positive pressure ventilation (NPPV) medication perished over a 50-month monitoring period. In comparison to similar obese patients without hypoventilation, who had a 9 % mortality rate, 47 untreated individuals with OHS had a 23 % mortality rate, with the majority of deaths happening during the first three months of hospital discharge. The cumulative evidence suggests that early detection of OHS patients is crucial, and that treatment should start right away to prevent negative outcomes like return to a medical facility, mortality, as well as acute-on-chronic respiratory failure needing close observation in critical care.

Treatment

There are no defined guidelines for dealing with occupational safety and health. In reality, different perspectives on the underlying cause of the disease serve as the foundation for varied treatment methods. First off, there is evidence that upper airway blockage, which contributes significantly to the etiology of OHS, can be reversed with techniques like tracheostomy and CPAP in the nasal cavity and even invasive ventilation to assist breathing and reversal hypoventilation has been recommended because OHS is linked to failure of normal processes that prevent hypoventilation during sleep. Figure 2 shows the survival curves for people with OHS.

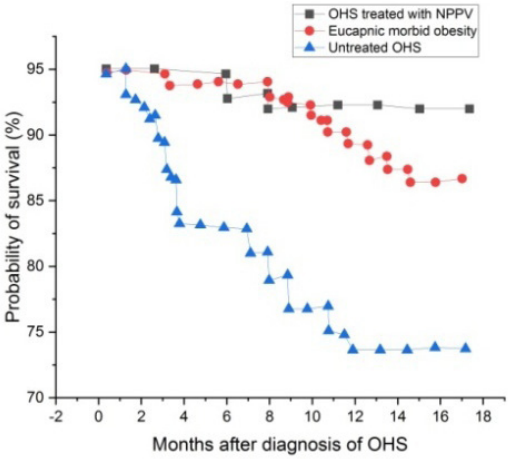

Figure 2. Survival curves for people with OHS

Getting rid of upper airway obstruction

It was found that in the majority of instances, this therapy was able to correct both awake hypoventilation and sleep-related respiratory failure. A mechanical appliance was also reported to reverse upper airway obstruction while the user was awake and improve respiratory failure. Following the development of nasal a continuous positive air this therapy was applied to OHS patients, causing the awake respiratory failure to remit. The effectiveness of CPAP nasal inflation alone as a therapy for OHS has been confirmed by later reports. Patients that responded well to CPAP treatment often needed pressures of 12–14 cm H_2O.

Air quality and OHS

Tracheostomy-based ventilation support has been utilized to treat obese patients with respiratory failure. Given the challenges associated with keeping a tracheostomy, particularly in patients with notably high fat in the neck area, this was certainly not an ideal procedure even though it was effective. Based on the success of this method in patients with other types of chest wall disease, NPPV utilizing a face or, later, nasal mask was frequently utilized in OHS.

Utilizing Oxygen

A kind of PAP treatment and supplementary nighttime oxygen are needed by almost half of OHS patients. In patients who adhere to PAP therapy, the requirement for nighttime and morning oxygen therapy considerably decreases. However, supplemental oxygen alone is insufficient and fails to treat hypoventilation.

Medicinal Respiratory Stimulation

Using pharmacologic medications to enhance breath would be an appealing approach given the likely role of decreased lung function in the etiology of OHS. On this strategy, there aren't many data, though. Progesterone, almitrine, or acetazolamide reports of initial promising outcomes have never led to continuing randomized controlled trials. Medroxyprogesterone can also make venous thromboembolism more likely.

Loss of Weight

Following up on the initial research by colleagues, several other studies have shown that weight loss improves sleep-disordered breathing, lowers open difficulty breathing, and improves lung function in OHS patients. A variety of surgical techniques can help patients lose weight quickly, but the majority of information on OHS comes from operations like gastric bypass and gastric banding. The large weight loss brought on by bariatric surgery can enhance sleep ventilation, which can eventually enhance daytime ventilation. OHS patients are more likely to expire following a gastric bypass operation, in part to the higher risk of perioperative breathing difficulties and the emergence of pulmonary embolism. In these patients, preoperative PAP therapy should be used until weight loss improves sleep breathing disorders sufficiently to allow therapy discontinuation. Therefore, to prevent postoperative respiratory failure, that patients with OHS should receive CPAP or bi-level PAP treatment both before surgery and right away following extubating. Additionally, there is no proof that therapy with PAP used in the immediate aftermath of surgery increases the risk of intestinal leakage or anastomotic disruption.

CONCLUSIONS

In conclusion, the obesity epidemic is expected to lead to a rise in OHS prevalence. A strong index of suspicion can result in the early diagnosis of the illness and the beginning of the proper therapy. To further understand the long-term treatment outcomes of individuals with OHS, additional research is required to better understand the therapy alternatives other than positive airway pressure. To avoid the severe negative effects of untreated OHS, doctors should promote positive airway pressure therapy adherence in the interim. Positive airway pressure is unable to produce the intended effects, weight-loss surgery or tracheostomy with or without medication using respiratory stimulants should be taken into consideration.

Limitations and Future Research

Patient adherence to therapy, particularly positive airway pressure, remains an issue that warrants further exploration. Future research should examine the efficacy of alternate therapeutic modalities, such as pharmaceutical therapies and innovative surgical techniques.

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FINANCING

The authors did not receive funding for the development of this research.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

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