Review Article Pol J Public Health, Vol. 134 (2024): 94-99

ALEKSANDRA WIKTORIA ŁOMŻA-ŁABA[1], BERNADETA MALISZEWSKA[1], DOMINIK ŁUCZYŃSKI[2], AGATA TOKARZEWSKA[3], ŁUKASZ ŁABA[4], STANISŁAW DZIURDA[5], MARCIN ŁASICA[6], MACIEJ MAŁYSZEK[1], BARTOSZ PAWŁOWSKI[7], KAROLINA HACZKUR-PAWŁOWSKA[7]

Oxygen therapy in the obesity hypoventilation syndrome

Abstract

Obesity hypoventilation syndrome (OHS) is a condition characterized by prevalence of obesity, sleep-disordered breathing, and a daytime hypoventilation caused by hypercapnia ($PaCO_2 \geq 45$ mmHg) with hypoxia ($PaO_2 < 70$ mm Hg). During global epidemic of obesity and the struggle with many related complications, the aim of this study is to focus on hypoventilation and respiratory alterations, caused by obesity hypoventilation syndrome. The comprehensive literature review was performed using the electronic databases: PubMed, ScienceDirect and Google Scholar. The search was limited to at least 2014. Keywords such as: "obesity hypoventilation syndrome", "obesity", "respiratory system", "oxygen therapy" and various combinations of the above were used. Considering many possible causes of alveolar hypoventilation and obesity related complications, the diagnosis of the OHS in the majority of patients is delayed. Patients burdened with sleep-related breathing disorders, including OHS, are most prone to develop life-threatening pulmonary hypertension or cardiovascular issues. The most proper treatment option for patients with OHS is positive airway pressure. Clinically documented mortality and morbidity in the co-occurrence of OHS and severe obesity were elevated. Hence, in addition to the recommended oxygen therapy in the OHS treatment, patients should be educated and supported by health professionals in their weight loss efforts.

Keywords: obesity hypoventilation syndrome, obesity, respiratory system, oxygen therapy.

DOI: 10.12923/2083-4829/2024-0020

INTRODUCTION

Obesity hypoventilation syndrome (OHS) is a chronic condition characterized by the combination of obesity, sleep-disordered breathing and a daytime hypercapnia ($PaCO_2 \geq 45$ mmHg) with hypoxia ($PaO_2 < 70$ mm Hg), caused by chronic alveolar hypoventilation (Figure 1.). The prevalence in the adult population is estimated circa 0.4% and increases with higher BMI levels. Many studies have described the prevalence of OHS in obese patients as being between 8% and 20%, among those who reported to sleep centers for diagnostics of sleep-disordered breathing [1]. Historically OHS was described for the first time by Bickelmann et al. in a case report with the term „Pickwickian syndrome". The name of the disease was inspired by the figure of Fat Boy Joe, created by Charles Dickens's in the „The Posthumous Papers of the Pickwick Club", who was constantly sleepy and hungry and often fell asleep during the day. The patient reported by Bickelmann et al. had daytime hypoventilation, polycythemia, chronic hypoxemia and pulmonary hypertension [2].

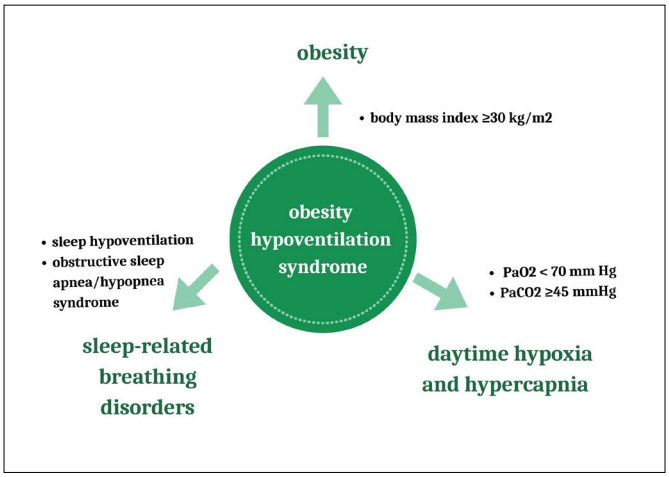

FIGURE 1. Diagnostic criteria for obesity hypoventilation syndrome.

The diagnosis of OHS centers upon exclusion of other causes of alveolar hypoventilation i.a. severe restrictive or obstructive pulmonary disease, hypothyroidism, kyphoscoliosis, neurological and muscular diseases, or other central

[1] Department of General Surgery, Provincial Specialist Hospital in Lublin, Poland
[2] Clinical Department of Cardiac Surgery, University Clinical Hospital No. 4 in Lublin
[3] Medical University of Lublin, Poland
[4] II Clinic of General, Gastroenterological and Digestive System Cancer Surgery, University Clinical Hospital No. 1 in Lublin, Poland
[5] Pediatric Department, Independent Public Health Care Complex in Wyszków, Poland
[6] Clinic of Orthopedics and Rehabilitation, University Hospital No. 4 in Lublin, Poland
[7] Neonatology Department, University Clinical Hospital No. 4 in Lublin, Poland

hypoventilation conditions. For excluding other causes of hypoventilation, the chest X-ray, testing of pulmonary function, assessment of respiratory muscle strength, electrocardiography, and testing of thyroid function should be performed. Moreover, patients suspected of being diagnosed with OHS should be screened using pulse oximetry and by determining serum levels of venous bicarbonate. Polysomnography is an examination required to diagnose OHS and to tailor individual therapy. During the diagnostic process many laboratory findings are supportive. First of all, the definitive test for alveolar hypoventilation, performed on room air, is an arterial blood gas. Furthermore, the elevated serum bicarbonate level due to metabolic compensation of respiratory acidosis is common in patients with chronic hypercapnia due to OHS. Many patients with OHS may also have reductions in maximal inspiratory and expiratory pressures caused by impaired respiratory muscles and abnormal respiratory mechanics. Although most patients with symptoms of OHS during their lifetime have prior hospitalizations, in the majority of patients the official diagnosis of OHS is delayed [3,4].

The pathogenesis of OHS is not fully understood. Several researchers proposed a few mechanisms, including decreased respiratory system compliance, increased lung resistance, weakness of respiratory muscles and increased work of breathing due to obesity, impaired central responses according to hypercapnia and hypoxia, upper airway obstruction, disordered breathing during sleep, neurological and hormonal abnormalities such as resistance of leptin. These pathophysiological changes lead to blunted ventilatory response and, as a result, chronic daytime hypercapnia [3,5]. Leptin is a protein synthesized in adipose tissue, which signals satiety and reduces appetite. Moreover, this protein plays a role in stimulation of the ventilatory response. In the state of obesity, the concentration of circulating leptin has been elevated, leading to a degree of leptin resistance. Increased level of leptin may impair the response to hypercapnia and hypoxia, indicating greater nighttime apnea-related hypercapnia and acidosis. Hence, leptin may be crucial in the development of OHS [6]. Main pathophysiological pathways include respiratory mechanics, respiratory drive, as well as, sleep- and obesity-related changes (Figure 2).

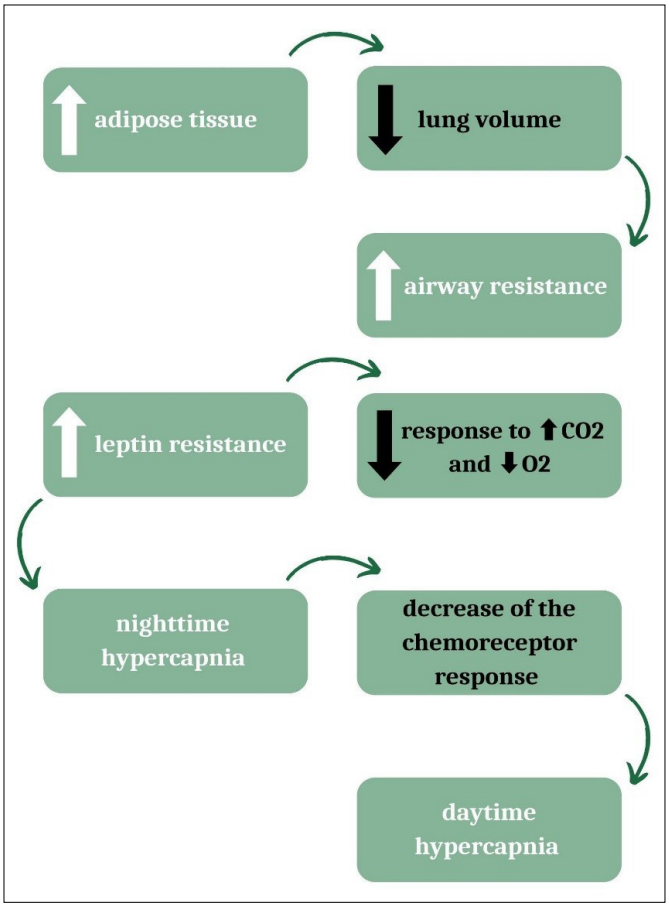

FIGURE 2. Pathophysiology of obesity hypoventilation syndrome.

The OHS syndrome most frequently exists with obstructive sleep apnea (OSA), defined by recurrent obstruction of the upper airway leading to apnea, hypopnea, oxygen desaturation, and sleep deprivation. This condition is also strongly linked to metabolic disorders, such as type 2 diabetes mellitus, insulin resistance, nonalcoholic fatty liver disease (NAFLD), hypothyroidism and osteoarthritis. These diseases are correlated with higher risk of cardiovascular events, hypertension, arrhythmia, coronary heart disease and stroke [7]. Berg et al. found that patients with OHS and obesity compared with only obese ones, were statistically much more likely to have diagnosis of congestive heart failure, angina pectoris and cor pulmonale. Moreover, patients with OHS were more often hospitalized, had higher rates of admission to the intensive care unit (ICU) and needed invasive mechanical ventilation [2].

In reference to pathogenic characteristics of OHS in combination with severe obesity and respiratory failure as a complication, the mortality and morbidity clinically documented in these two disorders were elevated. Several observational research have reported circa 24% all-cause mortality at 1.5-2 years in OHS patients without any treatment [1]. Collectively, in addition to several retrospective studies, the most common conclusion for improving the survival rate of OHS patients, would be early identification, diagnosis of patients and initiation of treatment without delay to avoid readmission to the hospital, acute respiratory failure requiring admission to ICU and death [3].

The comprehensive literature review was performed using the electronic databases: PubMed, Science Direct and Google Scholar. Keywords such as: "obesity hypoventilation syndrome", "obesity", "respiratory system", "oxygen therapy" and various combinations of the above were used. The search was limited by 2014 for all keywords. The articles were included by following criteria: English language, free access, proper theme of article, at least 2014 year of publication. The aim of this review paper is to summarize the current state of knowledge about obesity hypoventilation syndrome (OHS), obesity-related respiratory alternations and methods of treatment. Due to many research studies, the influence of obesity on health and numerous comorbidities are currently well-known. The importance of this review paper focuses on the impact of obesity leading to hypoventilation, changes in respiratory system and various complications, as well as on highlighting the oxygen treatment options of the OHS.

Current state of knowledge

Obesity related to the obesity hypoventilation syndrome

Obesity according to the WHO is defined as a Body Mass Index (BMI) above 30 kg/m^2. Another definition of obesity includes the abnormal distribution or excessive accumulation of body fat, which affects health [8]. Individuals are categorized into five different categories according to BMI: 18.5-24.9 kg/m^2: normal range, 25.0-29.9 kg/m^2: overweight, 30.0-34.9 kg/m^2:

class 1-obesity, 35.0-39.9 kg/m^2: class 2-obesity, equal or greater 40 kg/m^2: class 3-obesity. Grade 3 or grade 2 obesity plus significant obesity-related co-morbidities are referred to as morbid obesity [9]. Over the past few decades, obesity has become an increasing global public health issue. According to recent figures, the global prevalence of overweight and obesity is still rising with the number of people with excess body weight reaching >2 billion, ~30% of the world population [10]. Approximately 16% of adults aged 18 years and older worldwide were obese in 2022. The prevalence of obesity worldwide more than doubled between 1990 and 2022. Obesity is also associated with economic burden. The prevalence of obesity affects not only the Western hemisphere, but almost all countries in the world. It leads to steadily increasing healthcare costs [15]. Obesity can be complicated by other diseases, such as: type 2 diabetes mellitus (T2DM), cardiovascular diseases, stroke, dyslipidemia, hypertension, hepatic steatosis, gallbladder problems, breathing problems such as sleep apnea, osteoarthritis, multiple types of cancer (endometrial, breast, ovarian, liver, gallbladder, prostate, kidney and colon). These factors can lead to an increased risk of mortality [11]. There is a significant heterogeneity of phenotypes among people with obesity, which is directly related to the involvement of different molecules, genes and cells, in addition to environmental, social and economic factors. Therefore, different subtypes of obesity can be distinguished. Metabolically healthy obese (MHO) is the absence of metabolic disorders, including type 2 diabetes mellitus, hypertension and dyslipidemia. Individuals with MHO have a higher risk of developing metabolic syndrome compared to healthy normal-weight people. Definitions vary from study to study, mainly based on inflammatory markers and cut-off values. Associated with this subtype of obesity are decreased circulating levels of complement C3, TNF-α, hsCRP, IL-6, plasminogen activator inhibitor-1 and also increased adiponectin [8].

Metabolically abnormal obese (MAO) is described by 2 main factors, BMI and metabolic status, which includes having three or more points from the NCEP-ATP III, to define metabolic syndrome. This group includes a significant number of overweight and centrally obese individuals with metabolic syndrome, T2DM, cardiovascular or cerebrovascular disease, who may have diastolic or systolic hypertension and increased waist and hip circumference. This subtype differs significantly from the metabolically healthy obese subtype in terms of postprandial blood glucose, high-density lipoprotein cholesterol, triglycerides, insulin and adiponectin levels [8]. Increased uric acid and visceral adiposity are associated biomarkers with MAO subtype [12]. Sarcopenic obesity is described as a decrease in lean body mass and is related to prognostic factors associated with predicting factors such as increased age, low socio-economic status, smoking, decreased physical activity, atherosclerosis and pulmonary disease. These factors are connected to an accumulation of body fat and a decrease in skeletal muscle mass and muscle strength [13]. Patients with Metabolically obese normal weight (MONW) are characterized by hyperinsulinemia, insulin resistance, atherogenic lipid profile, increase abdominal and visceral adiposity, unfavorable adipokine profile, and also hypertension and hypertriglyceridemia, as well as higher levels of oxidative stress. The MONW is also known as metabolically obese healthy. Associated with this subtype of obesity are increases in body fat percent, uric acid and alanine transaminase, decrease in skeletal muscle percent, and body water percent, as well as increase in the production of triglycerides and glucose [14].

The main comorbidities of obesity are cardiovascular disease, heart failure, type 2 diabetes mellitus and non-alcoholic liver disease. Adipose tissue is a dynamic organ distributed and plays significant roles all over the body with a very high ability to increase obesity. The increased accumulation of adipose tissue, especially in the viscera, has been described as a key determinant of the relative risk of hypertension and cardiovascular disease [15]. Multiple paracrine and inflammatory processes in adipose tissue were identified. On the cellular level, macrophages enter the AT and release cytokine (such as TNF-alpha, IL-6) as well as adipocyte hypertrophy. Circulating cytokines affect insulin secretion in various tissues, causing an additional paracrine effect [17]. Adipokines such as leptin and adiponectin are secreted from AT and are strongly involved in appetite regulation and energy homeostasis. Leptin regulates food intake and reproductive function. It plays a role in fetal growth, pro-inflammatory immune responses, angiogenesis and lipolysis. Adiponectin has a number of anti-inflammatory effects and may alleviate insulin resistance and hepatic steatosis. Adiponectin levels increase as fat mass decreases. Obesity results in decreased plasma adiponectin levels and reduced expression of adiponectin receptors, which may increase hyperinsulinemia. The liver is a major organ in substrate metabolism and a destination for insulin action. The liver's fatty acid metabolism, changed in states of obesity, leads to the accumulation of triglycerides in hepatocytes and to non-alcoholic fatty liver disease (NAFLD). NAFLD is the largest contributor to chronic liver disease, which leads to cirrhosis and hepatocellular carcinoma (HCC) [15]. The main mechanism linking obesity and hepatic steatosis is insulin resistance, leading to increased lipolysis in adipose tissue and lipogenesis in the liver, causing a greater influx of free fatty acids into hepatocytes. Because of its close association with extrahepatic disease, NAFLD contributes significantly to the increased mortality observed in obesity [18]. NASH, cirrhosis and HCC represent advanced stages of chronic liver disease, causing further increases in mortality [19]. Type 2 diabetes is strongly associated with obesity and this clearly explains the high prevalence of type 2 diabetes in many countries. In contrast, type 2 diabetes is a major risk factor for CVD. Many high-risk patients with obesity are characterized by a clustering of metabolic and cardiovascular risk factors because obesity is often associated with hypertension and dyslipidemia [20]. Obesity additionally increases the overall risk for heart failure (HF). Moreover, obesity makes worse LV hypertrophy, the exercise-induced increase of pulmonary artery and wedge pressures and the resulting right ventricular dilation in patients with heart failure with preserved ejection fraction. Obesity is a strong risk factor for the HF and CVD. CVD and HF events occur in obese patients at a younger age. These individuals live with CVD for a greater proportion of their lives than normal weight individuals [16].

Obesity significantly alters the mechanical properties of the chest and lungs, especially in location of the fat in abdominal and mediastinum cavities. In the state of obesity, the pleural and abdominal pressures are massively increased, leading to disruptions of air flow. It can be measured in spirometry tests, as reduction in the expiratory reserve volume (ERV) and the functional residual capacity (FRC) [21]. Notable reductions in expiratory reserve volume may lead to abnormalities

in ventilation distribution, inducing ventilation perfusion incongruity and gas exchange deviation. Impaired ventilatory system in common with increased oxygen consumption and CO_2 production in the obese patients, predisposed to obesity hypoventilation syndrome [22]. Furthermore, these airflow alterations, such as reduction of lung volumes and peripheral airway diameter, may lead to both airway hyperresponsiveness and obstruction, classical features of asthma. The state of obesity has been shown to increase inflammation, by the role of leptin, which regulates T cell proliferation and activation with macrophages and monocytes activation [23]. Adipose tissue as an active tissue, produces pro-inflammatory cytokines, i.a. leptin and IL-6, which increased levels are observed due to airway inflammation in asthma. Obesity is significantly linked to pathogenesis of asthma and impacts asthma outcomes negatively, due to the state of chronic airway inflammatory [24].

Oxygen therapy in the obesity hypoventilation syndrome

Oxygen therapy is used as a common medical intervention in many conditions. Approximately 15-20% of patients receive oxygen therapy in hospitals. This rate is even higher in the pre-hospital setting. Different medical devices can be used to supply oxygen. Nasal prong is used to deliver oxygen at a rate up to 5L/min. Face masks are recommended for higher flows, i.e. 5-10L/min. Mask with reservoir bag provides flow rates up to 15L/min. Actual inspired oxygen fraction (F_IO_2) depends on many factors, such as patient's minute ventilation, mouth and nasal inspiratory airflows and the device's airflow. High flow oxygen therapy (HFOT) allows supplying flow up to 50L/min with adjustable level of F_IO_2. For most severely ill patients, invasive mechanical ventilation is used, for which F_IO_2 level can be precisely adjusted from 21% to 100% [25].

Continuous positive airway pressure (CPAP) is a form of ventilatory support which applies a constant level of positive end-expiratory pressure (PEEP) to a patient with spontaneous breathing. CPAP is typically administered using a nasal mask, as it is more efficient than an oronasal mask for long-term use [2]. PEEP increases a patient's FRC, opens alveoli, improves lung compliance. Work of breathing is decreased which improves oxygenation. Recommendations for using CPAP suggest starting at a level of 4cm H_2O and increasing by 1 or 2cm H_2O to a maximum of 20 cm H_2O. F_IO_2 can be set from 21% to 100%. If a patient's gas exchange and work of breathing has not improved, other forms of oxygen therapy should be considered.

Non-invasive ventilation (NIV), interchangeably called BiPAP (Bi-level Positive Airway Pressure) is another mode of ventilation. BiPAP devices alternate delivering inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP). The tidal volume is determined by the difference between these two pressures, with a larger tidal volume resulting in better alveolar ventilation. Minimal start settings are 8cm H_2O for IPAP and 4cm H_2O for EPAP. IPAP can be increased by the levels of 2 to maximum 20cm H_2O, while EPAP's maximum setting is 10cm H_2O. Increasing EPAP too much may decrease delivered tidal volume, therefore EPAP should be titrated carefully [26].

Adequacy of oxygen therapy should be monitored using arterial oxygen saturation (SaO_2) or arterial oxygen tension (PaO_2). Both require arterial blood sampling. Therefore, it is very common to use peripheral oxygen saturation (SpO_2) as a surrogate for SaO_2. Accuracy of SpO_2 is good enough to use both methods interchangeably. Although the use of oxygen therapy is very common, it should not be considered harmless. Recent studies show a small but significant increase in mortality associated with hyperoxemia, which can be caused by oxygen administration despite normal arterial oxygen saturation. PaO_2 measurement is most suitable for hyperoxemia detection, as hemoglobin saturation cannot exceed 100%, making SpO_2 and SaO_2 less useful parameters [25]. The target range for oxygen therapy depends on the presence of risk factors for oxygen-induced hypercapnia, such as asthma, chronic obstructive pulmonary disease or obesity-associated hypoventilation. For patients with those risk factors, target SpO_2 should be 88-92%. For other patients, oxygen therapy should be started when SpO_2 is below 92% and stopped when it is more than 96% [25,27].

Positive airway pressure (PAP) therapy – either CPAP or various modes of NIV – is the treatment of choice for patients with OHS. PAP therapy improves diurnal hypercapnia in a complex mechanism, which includes reduction of inspiratory muscular activity, decreasing the mechanical load, limiting obstructive events during sleep (apneas and hypopneas), decreasing the levels of serum leptin [28]. Studies showed much better improvement in pulmonary function, sleep quality and polysomnographic parameters after using CPAP or NIV, compared to a control group of patients without PAP therapy [29]. There were no significant differences in results between CPAP and NIV, however NIV resulted in slightly better pulmonary function improvement – bigger daytime $PaCO_2$ reduction and better spirometry parameters. NIV is also slightly more effective in improving cardiac function and structure, measured by echocardiography [30]. Treatment results, for both CPAP and NIV, are obviously dependent on adequate compliance. Using the PAP therapy for more than 4 hours per night is recommended for significant improvement to be seen, such as changes in daytime $PaCO_2$ [14]. Studies have not shown much difference in treatment compliance and failure between CPAP and NIV [1,29].

There is no clear superiority in either mode of PAP therapy in OHS treatment. Specific symptoms and their severity should be considered while choosing the best PAP mode for a patient. If severe obstructive sleep apnea (OSA - defined by apnea-hypopnea index >30 events/hour) is present, CPAP therapy is suggested as the first-line treatment [1,30]. Patients should be monitored closely for the first 2-3 months after initiation of therapy. If clinical response is inadequate, ventilation and oxygenation does not improve or the patient requires hospital admissions due to acute-on-chronic respiratory failure, PAP mode should be switched to NIV. Also, if a patient with OHS presents milder forms of OSA (<30 events/hour) or does not present OSA, NIV is considered as better treatment [1]. To assign CPAP therapy, patients must be clinically stable. Also, $PaCO_2$ should not be altered to more than 55 mmHg. If any of those are not met, NIV should be used [2].

Complications in patients with the obesity hypoventilation syndrome

The aforementioned clinical characteristics of OHS include sleep-disordered breathing and persistent daytime hypercapnia with $PaCO_2$>45 mmHg. These manifestations have a potential to induce several additional health complications, mainly associated with alteration in pulmonary hemodynamics [3]. Among these issues, patients burdened with sleep-related

breathing disorders, including OHS, are most prone to develop pulmonary hypertension. The most adequate tool for assessment of presence of pulmonary hypertension is measurement of pulmonary arterial pressure (PAP). Pulmonary arterial hypertension is diagnosed with PAP higher than 20 mmHg, pulmonary vascular resistance ≥3 in Wood units and pulmonary arterial wedge pressure ≤15 mmHg [31]. In case of OHS, onset of nocturnal hypoxemia and following elevation of PAP can be very severe, as the patient spends over 50% of sleep time with saturation values below 90%, with minimal saturation values lowering to 60% [1]. As nocturnal PAP increases in patients with sleep-related breathing disorders and hypoventilation, we can assume that analogical situation takes place in case of patients with OHS, however at the moment there are no studies supporting the hypothesis [32].

Since obesity is a civilization disease burdened with additional health complications by itself, it is understandable that patients with OHS are more prone to suffering from additional comorbidities and complications. One of major issues coexisting in this patient group is obstructive sleep apnea (OSA). It is estimated that up to 90% of patients with OHS suffer from OSA. The remaining 10%, even despite not meeting diagnostic criteria of OSA, are found to have hypoventilation worsening during rapid eye movement (REM) phase of sleep cycle [33]. Clinical manifestation of OSA includes recurrent in nature episodes of upper airway obstruction, resulting in desaturation and sleep fragmentation. On the contrary to OHS, there are no episodes of hypoventilation and hypercapnia present during the daytime [34]. Even despite the potential of these diseases to overlap, it is worth noting that they differ in terms of prevalence of additional comorbidities and mortality in obese patients. For instance, according to prospective observational study conducted by Almeneessier et at., there is a high chance of developing pulmonary hypertension in patients with OHS. Within the study 71.4% of women and 61.9% of men with OHS were also suffering from pulmonary hypertension [35]. A case series compiled by Ekici et al. gives basis for further investigations regarding correlation between OHS and prevalence of pulmonary embolism. All of the patients in the study showed signs of mosaic perfusion, enlargement of the main pulmonary artery and presence of segmental and subsegmental filling defects. Possible mechanisms of formation of pulmonary embolism in these patients may overlap with these found in chronic obstructive pulmonary disease, i.e. hypercoagulability being a result of hypoxia, sedentary lifestyle and systemic inflammation [36]. It is worth noting, however, that in case of OHS embolism-related changes are rather chronic in nature than acute. These results show a vast need for additional studies to be conducted regarding the matter in order to confirm these associations, especially given that obesity by itself makes for an important risk factor for pulmonary embolism, with a relative risk of 2.18 in comparison to non-obese control group [37]. Integrally with issues with the pulmonary system, patients with OHS are more likely to develop cardiovascular complications. According to a clinical review compiled by Zheng et al., patients with OHS are much more prone to have cardiovascular issues in comparison to patients with OSA alone. There were also higher rates of congestive heart failure and arrhythmia reported in patients with OHS, with prevalence of congestive heart failure varying from 8% to 60% across the studies, and rate of arrhythmia reaching up to 36% [38]. However, the prevalence of ischaemic heart disease (IHD) in this particular group is not fully elucidated, as results differ between the studies, showing similar or even lower rates of IHD in patients with OHS than with OSA. In studies showing higher rates of IHD the results may be potentially changed due to a group of patients with overlapping chronic obstructive pulmonary disease, which is a known cause for higher coronary disease rates [39,40]. Clinical importance of plurality of comorbidities in patients with OHS lays not only in burden of extended inpatient and outpatient care, but also in vastly increased risk of perioperative morbidity and complications. A study by Kaw et al. demonstrates statistically significant elevation of risk of postoperative respiratory failure (odds ratio of 10.9; 95% confidence interval 3.7-32.3) and risk of postoperative intensive care unit transfer (OR 10.9; 95% CI 3.7-32.3), along with noticeable higher probability of postoperative heart failure (OR 5.4; 95% CI 1.9-15.7; P=0.002), prolonged intubation (OR 3.1; 95% CI 0.6-15.3; P=0.2), longer intensive care unit and hospital stay lengths, in comparison to group of patients with OSA undergoing elective noncardiac surgeries. Within the study BMI has not shown any associations with postoperative outcomes [39]. Perioperative mortality rates are also a matter of great concern, since it affects from 2% to 8% of patients undergoing bariatric surgery [41]. The importance of obesity ventilation syndrome for clinical management of patients is best illustrated by the fact that it was taken into consideration in creation of obesity surgery mortality risk score (OS-MRS) – a tool allowing medical practitioners to assess risk of perioperative and postoperative mortality. One point is assigned based on each of five parameters: BMI>50, male gender, presence of hypertension, age ≥45 years, risk factors for pulmonary embolism (thromboembolism in the past, OHS, pulmonary hypertension, preoperative vena cava filter) [42]. Taking OHS into consideration in formulation of this scoring system emphasizes its significance in risk stratification in bariatric surgery, with potential to impact the qualification process in other operative interventions in the future [43].

CONCLUSION

The prevalence of obesity hypoventilation syndrome is bound to increase with the epidemic of the obesity. Due to many causes of alveolar hypoventilation, the diagnosis of OHS is prolonged and sometimes patients remain undiagnosed. This literature review emphasizes the significance of early diagnosis and oxygen treatment, by positive airway pressure (PAP) therapy (CPAP, or NIV) of the OHS patients. Early recognition of clinical symptoms, conducting the overnight polysomnography and excluding the other possible causes in common with proper oxygen treatment and weight loss, may be crucial to reduce the major morbidity and mortality of untreated OHS.

REFERENCES

1. Masa JF, Pépin JL, Borel JC, et al. Obesity hypoventilation syndrome. Eur Respir Rev. 2019;28(151):180097.
2. Athayde RAB, Oliveira Filho JRB, Lorenzi Filho G, et al. Obesity hypoventilation syndrome: a current review. J Bras Pneumol. 2018;44(6):510-8.
3. Mokhlesi B, Masa JF, Brozek JL, et al. Evaluation and management of obesity Hypoventilation Syndrome. An Official American Thoracic Society Clinical Practice Guideline. Am J Respir Crit Care Med. 2019;200(3):e6-e24.

4. Kaw R, Wong J, Mokhlesi B, et al. Obesity and Obesity Hypoventilation, Sleep Hypoventilation, and postoperative respiratory failure. Anesth Analg. 2021;132(5):1265-73.
5. Trang H, Samuels M, Ceccherini I, et al. Guidelines for diagnosis and management of congenital central hypoventilation syndrome. Orphanet J Rare Dis. 2020;15(1):252.
6. Crummy F, Piper AJ, Naughton MT. Obesity and the lung: 2. Obesity and sleep-disordered breathing. Thorax. 2008;63:738-46.
7. Brock JM, Billeter A, Müller-Stich BP, et al. Obesity and the Lung: What we know today. Respiration. 2021;99(10):856-66.
8. Mayoral LP, Andrade GM, Mayoral EP, et al. Obesity subtypes, related biomarkers & heterogeneity. Indian J Med Res. 2020;151(1):11-21.
9. Ashwell M, Mayhew L, Richardson J, et al. Waist-to-height ratio is more predictive of years of life lost than body mass index. PLoS One. 2014;9(9):e103483.
10. Caballero B. Humans against Obesity: Who will win? Adv Nutr. 2019;10(suppl_1):S4-S9.
11. Purnamasari D, Badarsono S, Moersadik N, et al. Identification, evaluation and treatment of overweight and obesity in adults: clinical practice guidelines of the obesity clinic, Wellness Cluster Cipto Mangunkusumo Hospital, Jakarta, Indonesia. J ASEAN Fed Endocr Soc. 2014;26(2):117.
12. Kjaer IG, Kolle E, Hansen BH, et al. Obesity prevalence in Norwegian adults assessed by body mass index, waist circumference and fat mass percentage. Clin Obes. 2015;5(4):211-8.
13. Tyrovolas S, Koyanagi A, Olaya B, et al. Factors associated with skeletal muscle mass, sarcopenia, and sarcopenic obesity in older adults: a multicontinent study. J Cachexia Sarcopenia Muscle. 2016;7(3):312-21.
14. Pluta W, Dudzińska W, Lubkowska A. Metabolic obesity in people with normal body weight (MONW) – Review of Diagnostic Criteria. Int J Environ Res Public Health. 2022;19(2):624.
15. Kloock A, Ziegler G, Dischinger U. Obesity and its comorbidities, current treatment options and future perspectives: Challenging bariatric surgery? Pharmacol Therapeut. 2023;251:108549.
16. Oikonomou EK, Antoniades C. The role of adipose tissue in cardiovascular health and disease. Nat Rev Cardiol. 2019;16:83-99.
17. Marques CG, Dos Santos Quaresma MVL, Nakamoto FP, et al. Does modern lifestyle favor neuroimmunometabolic changes? A Path to Obesity. Front Nutr. 2021;8:705545.
18. Berrington de Gonzalez A, Hartge P, Cerhan JR, et al. Body-mass index and mortality among 1.46 million white adults. N Engl J Med. 2010;363(23):2211-9.
19. Younossi Z, Tacke F, Arrese M, et al. Global perspectives on nonalcoholic fatty liver disease and nonalcoholic steatohepatitis. Hepatology. 2019;69(6):2672-82.
20. Piché ME, Tchernof A, Després JP. Obesity phenotypes, diabetes, and cardiovascular diseases. Circ Res. 2020;126(11):1477-500.
21. Dixon AE, Peters U. The effect of obesity on lung function. Expert Rev Respir Med. 2018;12(9):755-67.
22. Pépin JL, Timsit JF, Tamisier R, et al. Prevention and care of respiratory failure in obese patients. Lancet Respir Med. 2016;4(5):407-18.
23. Sutherland ER. Linking obesity and asthma. Ann N Y Acad Sci. 2014;1311:31-41.
24. Sharma V, Cowan DC. Obesity, inflammation, and severe asthma: an update. Curr Allergy Asthma Rep. 2021;21(12):46.
25. Allardet-Servent J, Sicard G, Metz V, et al. Benefits and risks of oxygen therapy during acute medical illness: Just a matter of dose! Rev Med Interne. 2019;40(10):670-6.
26. Popowicz P, Leonard K. Noninvasive ventilation and oxygenation strategies. Surg Clin North Am. 2022;102(1):149-57.
27. Gottlieb J. Oxygen therapy in intensive care. Dtsch Med Wochenschr. 2023;148(12):759-66.
28. Ramírez Molina VR, Masa JF, Gómez de Terreros Caro FJ,et al. Effectiveness of different treatments in obesity hypoventilation syndrome. Pulmonology. 2020;26(6):370-7.
29. Masa JF, Corral J, Alonso ML, et al. Efficacy of different treatment alternatives for obesity hypoventilation syndrome. Pickwick Study. Am J Respir Crit Care Med. 2015;192(1):86-95.
30. Corral J, Mogollon MV, Sánchez-Quiroga M, et al. Echocardiographic changes with non-invasive ventilation and CPAP in obesity hypoventilation syndrome. Thorax. 2018;73:361-8.
31. Adir Y, Humbert M, Chaouat A, et al. Sleep-related breathing disorders and pulmonary hypertension. Eur Respir J. 2021;57(1):2002258.
32. Simonneau G, Montani D, Celermajer DS, et al. Haemodynamic definitions and updated clinical classification of pulmonary hypertension. Eur Respir J. 2019;53(1):1801913.
33. Cooksey J, Mokhlesi B. Postoperative complications in obesity hypoventilation syndrome and hypercapnic OSA: CO2 Levels Matter! Chest. 2016;149(1):11-3.
34. Raveendran R, Wong J, Singh M, et al. Obesity hypoventilation syndrome, sleep apnea, overlap syndrome: perioperative management to prevent complications. Curr Opin Anaesthesiol. 2017;30(1):146-55.
35. Almeneessier AS, Nashwan SZ, Al-Shamiri MQ, et al. The prevalence of pulmonary hypertension in patients with obesity hypoventilation syndrome: a prospective observational study. J Thorac Dis. 2017;9(3):779-88.
36. Ekici A, Ekici M, İleri Ş, et al. Pulmonary embolism in obesity-hypoventilation syndrome. Clin Respir J. 2020;14(11):1099-104.
37. Hotoleanu C. Association between obesity and venous thromboembolism. Med Pharm Rep. 2020;93(2):162-8.
38. Zheng Y, Phillips CL, Sivam S, et al. Cardiovascular disease in obesity hypoventilation syndrome - A review of potential mechanisms and effects of therapy. Sleep Med Rev. 2021;60:101530.
39. Kaw R, Bhateja P, Paz Y Mar H, Hernandez AV, et al. Postoperative complications in patients with unrecognized obesity hypoventilation syndrome undergoing elective noncardiac Surgery. Chest. 2016;149(1):84-91.
40. McNicholas WT. Comorbid obstructive sleep apnoea and chronic obstructive pulmonary disease and the risk of cardiovascular disease. J Thorac Dis. 2018;10(Suppl 34):4253-61.
41. Efthimiou E, Court O, Sampalis J, et al. Validation of obesity surgery mortality risk score in patients undergoing gastric bypass in a Canadian center. Surg Obes Relat Dis. 2009;5(6):643-7.
42. DeMaria EJ, Murr M, Byrne TK, et al. Validation of the obesity surgery mortality risk score in a multicenter study proves it stratifies mortality risk in patients undergoing gastric bypass for morbid obesity. Ann Surg. 2007;246(4):578-82.
43. Kaw RK. Spectrum of postoperative complications in pulmonary hypertension and obesity hypoventilation syndrome. Curr Opin Anaesthesiol. 2017;30(1):140-5.

Corresponding author
Aleksandra Wiktoria Łomża-Łaba
Department of General Surgery, Provincial Specialist Hospital in Lublin
e-mail: aleksandra.lomza98@gmail.com