

Pneumologia

Non-invasive ventilation in super obese young patients: a case report

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Abstract

English:

This case highlights a rare and life-threatening manifestation of obesity hypoventilation syndrome (OHS), complicated by hypercapnic encephalopathy, pulmonary hypertension and hypertensive heart disease, in a 34-year-old male with morbid obesity. Initially diagnosed with cardiac pathology, but after failing to improve with diuretics and oxygen therapy alone, the patient presented with severe hypercapnic encephalopathy and pulmonary hypertension. Non-invasive ventilation (NIV) with bilevel positive airway pressure (BiPAP) was initiated and later switched to Average Volume Assured Pressure Support (AVAPS), resulting in significant clinical improvement. Despite persistently elevated carbon dioxide levels and respiratory acidosis, NIV therapy effectively enhanced gas exchange, improved consciousness and normalised blood gases. Multidisciplinary care, including dietary management and collaboration with cardiology and diabetes specialists, facilitated long-term recovery. This case highlights the critical role of early OHS diagnosis and the importance of tailored NIV strategies in managing severe respiratory failure, highlighting their potential to improve patient outcomes and reduce the need for intensive care.

Keywords

obesity hypoventilation syndrome • sleep apnoea • non-invasive ventilation

Ventilație noninvazivă la pacienții tineri cu obezitate severă: prezentare de caz

Rezumat

Romanian:

Acest caz evidențiază o manifestare rară și amenințătoare de viață a sindromului de obezitate hipoventilație (SOH), complicată de encefalopatie hipercapnică, hipertensiune pulmonară și boală cardiacă hipertensivă, la un pacient de 34 ani, cu obezitate morbidă. Inițial, pacientul a fost diagnosticat cu patologie cardiacă, însă, în absența unei ameliorări sub tratament cu diuretice și oxigenoterapie, a dezvoltat encefalopatie hipercapnică severă și hipertensiune pulmonară. S-a inițiat ventilația non-invazivă (VNI) cu presiune pozitivă în două niveluri (BiPAP), ulterior ajustată la suport ventilator cu presiune asigurată de volum mediu (AVAPS), ceea ce a determinat o îmbunătățire clinică semnificativă. Deși hipercapnia și acidoza respiratorie au persistat inițial, terapia VNI a optimizat schimburile gazoase, a ameliorat starea de conștiență și a normalizat gazele sangvine. Abordarea multidisciplinară, incluzând managementul nutrițional și colaborare cu specialiști în cardiologie și diabetologie, a fost esențială pentru recuperarea pe termen lung. Acest caz subliniază importanța diagnosticului precoce al SOH și rolul crucial al strategiilor personalizate de VNI în managementul insuficienței respiratorii severe, demonstrând impactul acestora asupra prognosticului pacienților și reducerea necesității îngrijirilor intensive.

Cuvinte-cheie

Sindrom de obezitate-hipoventilație • apnee în somn • ventilație non-invazivă

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Introduction

Obesity is a chronic, multifactorial pathology, often recurrent, difficult to treat, highly associated with significant mortality and morbidity, ranging from premature death to chronic cardiovascular, respiratory, metabolic or even malignant diseases, which can compromise the life expectancy of patients. Moreover, it is a significant contributing factor to the development of sleep apnoea syndrome (obstructive sleep apnoea [OSA]) and other sleep disorders through a combination of structural airway alteration, modified respiratory mechanics and impaired neuromuscular control. If left untreated, this condition can progress to chronic respiratory failure, pulmonary hypertension and cardiovascular complications. Obesity-related structural changes in the respiratory system, alterations in respiratory drive and breathing abnormalities during sleep play a crucial role in the pathophysiology of the mentioned conditions. These mechanisms can lead to upper airway obstruction, nocturnal hypoventilation and sleep fragmentation, ultimately increasing sleep-disordered breathing-associated morbidity (1).

This report describes a case of obesity-related sleep-disordered breathing complicated by hypercapnic encephalopathy, emphasising the role of non-invasive ventilation (NIV) in management and long-term outcomes. Obesity hypoventilation syndrome (OHS) is one of the six subtypes of sleep-hypoventilation disorders, being defined by presence of a chronic daytime alveolar hypoventilation leading to hypercapnia and hypoxia in obese patients, after ruling out other disorders that may cause alveolar hypoventilation; in addition, most of the patients have a type of sleep disordered breathing, often OSA (2, 3). OHS can be diagnosed with the following criteria: body mass index (BMI) $>30 \text{ kg/m}^2$, arterial carbon dioxide tension $>45 \text{ mmHg}$ (PaCO_2), sleep-disordered breathing, absence of other causes for hypoventilation – such as neuromuscular, mechanical or metabolic causes of hypoventilation. A classification system for OHS was proposed, based on BMI, daytime arterial oxygen pressure (PaO_2), daytime PaCO_2 and bicarbonate values, which categorises OHS in mild, moderate or severe (2).

Clinical problems, differential diagnosis

Globally, obesity rates have nearly tripled since 1975, primarily due to increasingly sedentary lifestyles, poor dietary habits and psychosocial stressors. In 2022, 43% of adults were overweight (4, 5) and over 1 billion individuals suffered from obesity (6). The WHO European Regional Obesity Report of 2022 indicates that 59% of adults in Europe are overweight or obese (7). In Romania, as of 2024, 66% of men and 56% of women are classified as obese. Higher prevalence is

observed among individuals with lower education, those in rural areas and the unemployed or retired (8). Projections estimate a 2.1% annual increase in adult obesity prevalence, suggesting that by 2035, 35% of Romanians could be affected (8).

The precise prevalence of OHS is unclear, but estimates suggest 0.4%–0.6% in the general U.S. population. OHS occurs in 8%–20% of obese patients referred for sleep-disordered breathing. Data indicate a prevalence of 0.15%–0.3% of OHS in the general adult population (9, 10). Prevalence rises with BMI: 8%–11% at 30–35 kg/m^2 , up to 31% at $\geq 40 \text{ kg/m}^2$ and 50% at BMI $>50 \text{ kg/m}^2$ (10). In Europe, prevalence among individuals with OSA and BMI $>40 \text{ kg/m}^2$ is 30% in the UK, 24% in France and under 20% in Italy (10). OSA and OHS remain underdiagnosed in Romania and across Europe, despite increasing prevalence. Recent data estimate that 9%–13% of adult males and 4%–6% of females in Europe suffer from moderate-to-severe OSA, yet fewer than 30% are diagnosed (8). In Romania, OSA prevalence is estimated to be over 10%, but national registries remain scarce. For OHS, epidemiological data are limited, but the condition is thought to affect approximately 0.3% of adults, with higher rates among the severely obese (10).

Several barriers contribute to underdiagnosis: limited access to diagnostic tools such as polysomnography, low public and even physician awareness and historically poor integration of sleep medicine into standard clinical pathways. Until 2023, the Romanian National Health Insurance Agency (CNAS) did not cover positive airway pressure (PAP) devices for sleep-related respiratory disorders, limiting access to therapy. Furthermore, OSA and OHS are often misattributed to general fatigue or comorbid cardiopulmonary diseases, delaying appropriate diagnosis. The lack of multidisciplinary approaches involving pulmonologists, cardiologists and sleep specialists also hinders timely recognition and management. Improved screening, funding and awareness are crucial to increase diagnosis and treatment rates in these high-risk populations.

Management of OHS focuses on weight loss and ventilatory support. While lifestyle changes are the first-line treatment step, bariatric surgery may be required for some patients (11). PAP therapy during sleep is essential. CPAP is preferred in patients with coexisting OSA (present in 90% of OHS cases), while bilevel positive airway pressure (BiPAP) is indicated in those with sleep-related hypoventilation or CPAP intolerance (12). Average volume assured pressure support (AVAPS), a newer NIV mode, adjusts inspiratory pressure to maintain a set tidal volume (13).

Delivered via a face mask, AVAPS optimises ventilation and synchrony. Long-term PAP therapy improves gas exchange, lowers PaCO_2 and BMI and enhances sleep quality and survival. While CPAP addresses upper airway obstruction, BiPAP and

AVAPS are superior in correcting alveolar hypoventilation. CPAP failure – defined by persistent hypercapnia, poor oxygenation or $\text{RDI} > 10/\text{h}$ – necessitates switching to BiPAP-S/T with or without AVAPS. NIV is the cornerstone of therapy in hypoventilation syndromes, including neuromuscular disease and severe chronic obstructive pulmonary disease (COPD). In OHS, nocturnal NIV improves lung mechanics, sleep and gas exchange, with AVAPS addition further reducing PaCO_2 and restoring normocapnia (14).

Results of clinical findings

We report the case of an obese 34-year-old male, without any known pre-existing pathology, who presented with progressive dyspnoea, peripheral oedema without any signs of acute respiratory failure prior to admission. The patient was initially presented for admission in the cardiology department, complaining of significant peripheral oedema, progressive effort dyspnoea, ultimately leading to resting dyspnoea. Clinically, the patient showed an impaired general condition, periorbital cyanosis, hyperaemic conjunctiva, morbid obesity ($\text{BMI} = 44 \text{ kg/m}^2$), oxygen saturation 85% in ambient air and a blood pressure of 180/100 mmHg. Laboratory tests revealed important polycythaemias, elevated liver enzymes and hyperglycaemia. CT angiography of the thorax ruled out a pulmonary embolism and showed no parenchymal lung disease. The cardiology investigations established the diagnoses of left ventricular hypertrophy, pulmonary hypertension, mitral regurgitation, tricuspid regurgitation, grade three arterial hypertension with very high additional risk, hypertensive cardiomyopathy and decompensated chronic pulmonary heart disease. Unfortunately, the thoracic CT and cardiac ultrasound images were not available to include in the documentation. Additionally, the patient was diagnosed with type 2 diabetes mellitus.

Based on clinical suspicion, further investigations were pursued for COPD and OSA. The treatment was started with diuretics, anticoagulants, antihypertensive and antidiabetic agents, along with oxygenotherapy to maintain SpO_2 at 94%. During the first 2 days, clinical improvement was noted with reduced dyspnoea, oedema and oxygen supplementation need. However, in the subsequent days, the patient experienced a deterioration of the level of consciousness, raising suspicion of a cerebrovascular accident (CVA). Cerebral CT ruled out CVA, haemorrhage, or hematoma, but revealed generalised cerebral oedema, suggestive of hypercapnic encephalopathy. Prior to admission to our department, arterial blood gas (ABG) analysis confirmed severe hypercapnic acidosis ($\text{pH} = 7.2$, $\text{pCO}_2 = 94.4 \text{ mmHg}$, $\text{pO}_2 = 69.4 \text{ mmHg}$, $\text{SpO}_2 = 92.6\%$; Table 1 in Supplementary Materials).

The patient was transferred to the pulmonology department, where continuous monitoring and NIV could be provided, as a dedicated unit for advanced medical care is available. Orotracheal intubation was avoided, and NIV in BiPAP mode – in spontaneous/timed (S/T) mode was initiated with an IPAP of 16 cmH_2O and an EPAP of 6 cmH_2O . Despite undergoing therapy, hypercapnia persisted ($\text{pH} = 7.26$, $\text{pCO}_2 = 109.6 \text{ mmHg}$, $\text{pO}_2 = 46.8 \text{ mmHg}$; Table 1 in Supplementary Materials). The previous therapeutic scheme was maintained, although with minimal correction of the patient's acidosis and the persistence of hypercapnia. The level of consciousness remained severely altered. Subsequently, BiPAP settings were adjusted to a set of 18 cmH_2O IPAP and 8 cmH_2O EPAP, resulting in minimal improvement ($\text{pH} = 7.27$, $\text{pCO}_2 = 93.8 \text{ mmHg}$, $\text{pO}_2 = 69 \text{ mmHg}$; Table 1 in Supplementary Materials). Due to persistent hypercapnia, AVAPS ventilation was initiated the following day with the following parameters: tidal volume of 600 mL, IPAP min/max = 16/30 cmH_2O , EPAP min/max = 6/12 cmH_2O , backup rate of 14 breaths/min, AVAPS rate of 2 $\text{cmH}_2\text{O}/\text{min}$, FiO_2 adjusted for SpO_2 88%–92% (90%–91%). ABG analysis 4 hr after AVAPS initiation demonstrated significant improvement in hypercapnia and acidosis, along with improvement in oxygen saturation. AVAPS was maintained for 6 days, throughout critically maintained status, showing continuous improvement in gas exchange and progressive clinical recovery. Forty-eight hours before discharge, the ventilation mode was switched to BiPAP S/T mode. The final ABG analysis revealed $\text{pH} = 7.38$, $\text{pCO}_2 = 56.2 \text{ mmHg}$, $\text{pO}_2 = 45.8 \text{ mmHg}$, $\text{SpO}_2 = 80.9\%$ (Table 1 in Supplementary Materials). Further nocturnal cardio-respiratory polygraphy confirmed moderate OSA: apnoea hypopnoea index (AHI) = 18 events/hr, oxygen desaturation index (ODI) = 18 events/hr, mean $\text{SpO}_2 = 91\%$ (Table 2 in Supplementary Materials) and pulmonary function tests revealed restrictive ventilatory dysfunction. Unfortunately, the official spirometry report was not available for inclusion in the documentation. Nevertheless, spirometry at the time indicated a restrictive ventilatory pattern, which is consistent with the patient's clinical profile and obesity-related respiratory impairment.

The patient was initiated on BiPAP therapy at home. The specific settings were as follows: IPAP 16 cmH_2O , EPAP 8 cmH_2O . This was recommended for the management of moderate OSA and to improve sleep quality by alleviating upper airway obstruction and enhancing ventilation during sleep. The BiPAP therapy at home was complemented by multidisciplinary recommendations from a cardiologist, diabetologist and nutrition specialist. Additionally, psychotherapeutic support was recommended to address underlying psychological factors contributing to poor adherence and overall well-being. Two months after the start of therapy, the patient was reassessed with a comprehensive

evaluation including BiPAP compliance and efficacy, ABG analysis and clinical assessment of his general health and functional status. The patient adhered to the prescribed BiPAP therapy, using the device for an average of 7 hr per night, while experiencing an important weight loss of 15 kg and showing a significant reduction in AHI, now measuring 4 events per hour, indicating a substantial reduction in the severity of sleep-disordered breathing. Furthermore, the PaCO_2 dropped to 41 mmHg, reaching normocapnia. Clinical signs of respiratory distress, including cyanosis and oedema, remitted. There was also significant improvement in normalisation of blood pressure, blood glucose levels and liver function tests, further supporting the effectiveness of the therapy. The patient was no longer in an acidotic state with ventilation leading to the resolution of metabolic acidosis. Also, the daily functioning status was reported to show a significant improvement, with increased ability to perform ordinary activities, workplace tasks and safe operation of a vehicle.

Even though the first nocturnal polygraphy showed an intermediate form of OSA, the examination was not repeated after an important weight loss occurred in the patient. Hence the mode of ventilatory support (Bip with similar RR and PS) was not switched to CPAP. It must be remembered, however, that the patient has not been re-evaluated at an adequately long period after weight loss for a new assessment of ventilatory requirements.

Case particularity

This case highlights a rare and severe presentation of OHS with associated hypercapnic encephalopathy, complicated by pulmonary hypertension and hypertensive heart disease. The patient, initially misdiagnosed with a primary cardiac pathology, ultimately demonstrated profound respiratory failure secondary to OHS and OSA. The key uniqueness of this case lies in the dramatic improvement achieved through NIV, particularly with the implementation of AVAPS, leading to significant resolution of hypercapnia, acidaemia and associated systemic complications. The multidisciplinary approach, including cardiology, diabetology and nutritional intervention, played a pivotal role in the patient's long-term recovery, demonstrating the crucial role of early recognition and comprehensive management in obesity-related respiratory failure. This case underscores the importance of considering OHS in obese patients presenting with dyspnoea and the effectiveness of individualised NIV strategies in reversing life-threatening respiratory dysfunction.

In our patient, AVAPS managed to avoid the transfer to the intensive care unit, although his overall condition, consciousness level and gas exchange alteration are consistent with those of a patient almost needing orotracheal intubation. By using AVAPS, we managed to avoid the risks of intensive care hospitalisation, such as ventilator-

associated pneumonia, nosocomial infection and prolonged hospital stay (3–4 days longer). By closely monitoring patient, using NIV techniques for acute respiratory failure and optimising pharmacological treatments, we have been able to manage critical cases in the respiratory department without overwhelming the ICU. This approach not only alleviates the burden on the ICU but also addresses the rising financial strain associated with ICU admissions. In Romania, the cost of a single ICU day is substantial, with estimates reaching up to several €100, significantly impacting healthcare budgets. Moreover, ICU bed blockage – due to increased patient admissions and limited resources – can result in delays for other critical patients, making efficient ward management even more important. Therefore, avoiding ICU transfers through well-organised respiratory care and resource optimisation not only reduces costs but also maximises the availability of ICU beds for more urgent cases.

Discussions

NIV is fundamental for managing both acute and chronic respiratory failure in OHS, improving gas exchange, reducing work of breathing and preventing complications. Two main modes exist: BiPAP in S/T mode and average volume-assured pressure support (AVAPS), which includes Average Volume Assured Pressure Support - Spontaneous mode (AVAPS-S), Average Volume Assured Pressure Support - Timed mode (AVAPS-T) and Average Volume Assured Pressure Support - Auto EPAP (AVAPS-AE) variants (15). BiPAP S/T delivers fixed inspiratory and expiratory pressures, requiring manual adjustments, whereas AVAPS automatically adjusts inspiratory pressure to maintain target tidal volumes despite variable effort or airway resistance (1). In acute decompensation, AVAPS rapidly normalises PaCO_2 and pH, while BiPAP S/T may enhance sleep continuity in the chronic phase, both demonstrating similar patient tolerance (16). AVAPS's adaptive support lowers awake pressures, reducing barotrauma risk and improving comfort (13, 14). Clinical studies report greater PaCO_2 reductions and improved Glasgow scores with AVAPS versus BiPAP S/T and early use in emergency settings lowers intubation and mortality rates (18). Long-term, Continuous Positive Airway Pressure (CPAP) remains first-line for OHS with OSA (18, 19), but AVAPS serves as an effective rescue therapy in cases refractory to CPAP or BiPAP, enhancing gas exchange, sleep quality and quality of life (20). Continuous monitoring of respiratory and sleep metrics guides optimal mode selection. Our patient's rapid and sustained normalisation of PaCO_2 – from 109.6 mmHg on BiPAP S/T to 41 mmHg after 2 months of home AVAPS – parallels findings from recent controlled studies. Patout et al. (15) demonstrated that AVAPS-AE

reduced daytime PaCO₂ by 24 ± 7 mmHg versus 14 ± 6 mmHg with ST mode after 48 hr ($P < 0.01$) in stable OHS patients. Xu et al.'s 2022 network meta-analysis also confirmed that AVAPS modes deliver superior hypercapnia correction versus CPAP or BiPAP S/T in acute decompensated OHS (21). Following the suboptimal response to BiPAP therapy, despite pressure adjustments (initially IPAP 16 cmH₂O/EPAP 6 cmH₂O, then increased to 18/8 cmH₂O), AVAPS was initiated in order to provide more stable and targeted ventilatory support. The chosen AVAPS settings included a target tidal volume of 600 mL, IPAP range 16–30 cmH₂O, EPAP range 6–12 cmH₂O, backup respiratory rate of 14 breaths/min and an AVAPS rate of 2 cmH₂O/min. Although exact pressures reached during AVAPS therapy were not continuously recorded, the significant improvement observed in gas exchange parameters 4 hr after initiation (pH = 7.34, pCO₂ = 68.2 mmHg, pO₂ = 82.6 mmHg, SpO₂ = 94.7%) strongly suggests that the system dynamically adjusted IPAP to higher values – likely in the range of 22–24 cmH₂O – which would have been difficult to implement manually using conventional BiPAP in S/T mode. The ability of AVAPS to gradually titrate inspiratory pressure while maintaining a consistent tidal volume likely played a key role in improving ventilatory efficiency and patient tolerance, ultimately contributing to clinical stabilisation and avoiding invasive ventilation.

The AHI declines from 18 events/hr to 4 events/hr following AVAPS and weight management aligns with randomised data showing AVAPS-AE achieves an AHI reduction of 71% versus 50% with ST mode (mean 22 ± 6 to 6 ± 3 events/hr; $P < 0.01$) over 2 months. There are reports of an increase in mean nocturnal SpO₂ by 5% with AVAPS compared to 3% with fixed-pressure BiPAP ($P = 0.03$), mirroring our observed SpO₂ stabilisation above 90% (22). These data underscore AVAPS's enhanced ability to maintain upper airway patency while ensuring adequate alveolar ventilation.

Current guidelines advocate first-line CPAP or BiPAP S/T for stable OHS, reserving AVAPS (or VAPS) for acute decompensation or inadequate response to fixed pressures. Also, the European Respiratory Society endorses VAPS modes for patients with persistent hypercapnia or significant sleep fragmentation (23).

Conclusion

This case highlights the interplay between OHS, OSA and cardiovascular comorbidities and demonstrates the critical role of NIV, particularly AVAPS, in the acute management of severe OHS complicated with hypercapnic encephalopathy. It also underscores the critical role of timely recognition and appropriate ventilatory support in managing OHS. Early identification and management with NIV, particularly AVAPS

during acute decompensation, are crucial in preventing further deterioration. The successful avoidance of orotracheal intubation underscores the efficacy of NIV in stabilising gas exchange, thus preventing the need for invasive mechanical ventilation and reducing the risks of prolonged ICU hospitalisation, such as ventilator-associated pneumonia and nosocomial infections. Transitioning to BiPAP S/T mode in the long term improves overall clinical outcomes and quality of life in patients with OHS. Moreover, the case demonstrates the importance of a tailored, multidisciplinary approach in the long-term management of OHS, integrating NIV therapy with weight loss strategies, metabolic control and cardiopulmonary optimisation. Early recognition and personalised interventions are crucial to improving outcomes and reducing the substantial morbidity and mortality associated with this condition. This report further supports the utility of AVAPS in achieving better ventilatory adaptation and enhanced patient comfort in the acute phase, while transitioning to BiPAP S/T for chronic respiratory support. Given the increasing global prevalence of severe obesity and its associated respiratory complications, early identification and optimal NIV strategies remain essential to improving patient outcomes and reducing healthcare burden.

Funding

None.

Conflicts of interest

The authors declare no conflict of interest.

Informed consent statement

Informed consent was obtained at admission.

Data availability statement

Data used to support the findings of this study are available from the corresponding author upon request.

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Supplementary Materials

Table nr. S1 Evolution of ABG

Evolution of ABG	1. At admission	2. First attempt of VNI	3. AVAPS initiation	4. At the end
pH	7, 202	7, 267	7,277	7, 388
pCO ₂	94, 4 mmHg	109,6 mmHg	93, 8 mmHg	56,2 mmHg
pO ₂	69, 4mmHg	46,8 mmHg	69,0 mmHg	45,8 mmHg

ABG: arterial blood gas

VNI: non-invasive ventilation

AVAPS: average volume assured pressure support.

Table nr. S2 Poligraphy report

AHI	ODI	SpO ₂	T90
18/h	18/h	91%	1,8 %

AHI: Apnea-Hypopnea Index

ODI: Oxygen Desaturation Index

SpO₂: Mean oxygen saturation

T90: *Time below 90% oxygen saturation*