

Case Report

Management of Referred Case of Obesity Hypoventilation Syndrome with Obstructive Sleep Apnea of Morbidly Obese Patient: A Case Report

Anil Shahi* , Bishaka Adhikari , Bir Bahadur Syangtan , Ulid Adhikari ,
Nischal Kafle 

Chitwan Medical College, Tribhuvan University, Kirtipur, Nepal

Abstract

Obesity hypoventilation syndrome (OHS) is a serious condition characterized by obesity ($\text{BMI} \geq 30 \text{ kg/m}^2$), daytime hypercapnia ($\text{PaCO}_2 \geq 45 \text{ mmHg}$), and sleep-disordered breathing, without other identifiable causes of reduced alveolar ventilation, such as lung or neuromuscular disorders. The World Health Organization (WHO) defines morbid obesity as a $\text{BMI} \geq 35 \text{ kg/m}^2$, which significantly increases the risk of obstructive sleep apnea (OSA) and poses challenges in airway management. This case report discusses the successful management of a morbidly obese patient ($\text{BMI} = 51.3 \text{ kg/m}^2$) suffering from hypoventilation and sleep apnea. Initially, the patient received continuous oxygen therapy via nasal prongs, but as symptoms worsened—marked by poor sleep quality and a significant drop in oxygen saturation at night—non-invasive ventilation (NIV) and high-flow nasal cannula (HFNC) were introduced. Proper positioning was also maintained to optimize breathing and prevent further deterioration. Despite the increased risk of prolonged ICU stays and extended mechanical ventilation in morbidly obese patients, this case demonstrates that a multimodal conservative approach can lead to successful outcomes within a short period. It highlights the effectiveness of non-surgical interventions in managing OHS and OSA while emphasizing the importance of early referral to specialized healthcare centers for timely and effective treatment, ultimately improving patient outcomes.

Keywords

Obesity Hypoventilation Syndrome, Obstructive Sleep Apnea, Morbid Obesity

1. Introduction

Obesity hypoventilation syndrome (OHS) is characterized by the obesity [body mass index ($\text{BMI} \geq 30 \text{ kg/m}^2$), daytime hypercapnia ($\text{PaCO}_2 \geq 45 \text{ mmHg}$), and sleep-disordered breathing, without other identifiable reasons for reduced alveolar ventilation, such as lung or neuromuscular disorders [1-5]. The World Health Organization (WHO) defines morbid obesity as a BMI of $\geq 35 \text{ kg/m}^2$. Patients with morbid

obesity frequently have concomitant obstructive sleep apnea syndrome and a significant risk of difficult airway [6]. His prevalence of OHS in the general population ranges between 0.15% and 0.4%, but it rises to 10-30% among individuals with sleep-related respiratory difficulties. Many individuals with OHS experience airway obstruction during sleep, which can deteriorate into a more serious condition known as ob-

*Corresponding author: shahianil211@gmail.com (Anil Shahi)

Received: 10 January 2025; **Accepted:** 5 February 2025; **Published:** 2 September 2025



Copyright: © The Author(s), 2025. Published by Science Publishing Group. This is an **Open Access** article, distributed under the terms of the Creative Commons Attribution 4.0 License (<http://creativecommons.org/licenses/by/4.0/>), which permits unrestricted use, distribution and reproduction in any medium, provided the original work is properly cited.

structive sleep apnea hypopnea syndrome (OSAHS). Several studies have indicated that hypercapnic conditions are present in 10-20% of OSAHS cases. [7]

Here, we present a case of obesity hypoventilation syndrome accompanied by obstructive sleep apnea and type 2 respiratory failure in a morbidly obese patient, which was referred to a tertiary care center for management and to prevent further complications associated with OHS.

2. Case Presentation

A 69-year-old active smoker, morbidly obese (BMI = 51.3 kg/m²) female with no prior comorbidities or history of PTB exposure, was referred to the emergency department of our hospital from another center, where she was treated for COPD, OSAS, and systemic hypertension. She presented with complaints of cough with whitish sputum, progressive dyspnea (MMRC-I to MMRC-III) associated with orthopnea and paroxysmal nocturnal dyspnea, and swelling around the neck and face, with a history of snoring during sleep at night. There was no history of fever, morning headache, chest pain, or palpitations.

On physical examination, the patient was conscious, alert, and well-oriented to time, place, and person, with bilateral pitting edema on the legs and bilateral wheeze and crackles in the chest. Her respiratory rate was 16 breaths/min, blood pressure was 140/80 mm Hg, heart rate was 66 beats/min, body temperature was 98.2 °F, oxygen saturation was 78% in room air, and general random blood sugar (GRBS) was 85 mg/dl. The patient's weight was 120 kg, and her height was 170 cm (body mass index 51.3 kg/m²). The patient was nebulized with normal saline (A; I; NS) in the emergency department and was subsequently admitted and examined.

After 1 hour in the emergency department, the patient was transferred to the MICU for observation and further management via trolley after counseling with the Department of Medicine, the patient, and the patient's family about the disease and its prognosis. In the MICU, the patient's oxygen saturation was maintained through nasal prongs at 2 liters/min, and blood investigations were sent. The blood report and venous blood gas analysis (VBG) results are presented in Table 1 and Table 2, respectively. Along with oxygen therapy, consultations from different departments (cardiology, otorhinolaryngology) were obtained.

The patient's echocardiography showed moderate to severe mitral regurgitation (MR), moderate tricuspid regurgitation (TR), and a dilated left atrium with a left ventricular ejection fraction of 60% as shown in Figures 1 and 2 below. The otorhinolaryngologist found no causes of upper airway narrowing. Based on the findings of the above investigations,

the patient was diagnosed with heart failure with preserved ejection fraction, systemic hypertension, obesity hypoventilation syndrome, obstructive sleep apnea syndrome, and type 2 respiratory failure.

Diuretics (tab. spironolactone-furosemide) were started for heart failure, along with a metered-dose inhaler (MDI) of formoterol-budesonide and nebulization for respiratory improvement. As the patient didn't sleep well at night and her oxygen saturation dropped by up to 10% while asleep, non-invasive ventilation/high-flow nasal cannula (NIV/HFNC) was initiated as needed, and her position was maintained. On the 2nd day of hospital admission, hypokalemia was noted, with a serum potassium level of 3.27 mmol/L (normal = 3.5-5.5 mmol/L), which decreased to 2.7 mmol/L on the 3rd day. Potassium chloride syrup and magnesium sulfate were immediately started. As a result of strict monitoring, the patient's hemodynamic status gradually improved, and she needed oxygen therapy only at night after the 5th day of hospital stay. Weight-lowering medication (tab. orlistat) and other medications (multivitamin capsule, tab. calcium carbonate, and vitamin D) were prescribed by the endocrinologist.

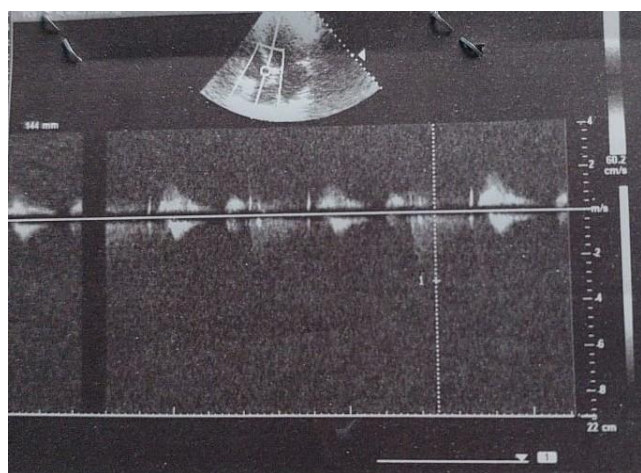
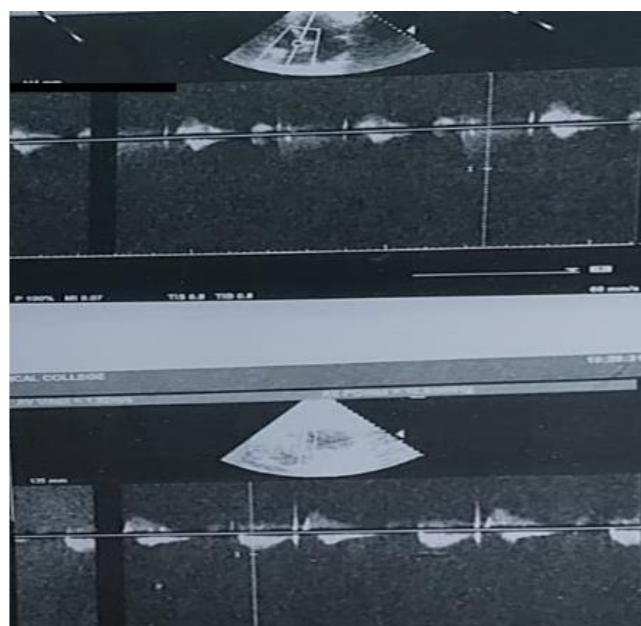
The patient was discharged with a few medications and advice once she became hemodynamically stable, with an oxygen requirement of up to 1 L/min and CPAP at night with pressure support of 20 cm of water after 6 days of hospitalization. She was advised to follow up after 2 weeks. At the time of discharge, the patient and her family were instructed on the use of BIPAP and advised to maintain oxygen saturation between 90-94% and use CPAP (20 cm of water) at night at home.

Table 1. Blood investigation report of patient.

Test	Result	Normal range
Hemoglobin	11.9 mg/dl	12-16 mg/dl
Urea	19.82 mg/dl	15-45 mg/dl
Sodium	142 mmol/L	135-145 mmol/L
Potassium	3.27 mmol/L	3.5-5.5 mmol/L
SGPT/ALT	99 U/L	<45 U/L
SGOT/AST	15 U/L	<40 U/L
Magnesium (serum)	1.77 mg/dl	1.8-2.4 mg/dl
NT-PROBNP	1717 pg/ml	<300 pg/ml
Creatinine	0.74 mg/dl	0.4-1.4 mg/dl
Troponin	0.39 ng/ml	0-0.04 ng/ml

Table 2. Blood gas analysis report of patient in different days.

Test	During the day of admission	3 rd day	4 th day	Normal range
pH	7.56	7.327	7.304	7.35-7.45
Pco ₂	41	69.8	77.8	35-35 mm hg
Po ₂	54	38.4	32.3	80-108 mm hg
Hco ₃ ⁻	37.8	30.6	30.8	22-26 mEq/L

**Figure 1.** Echocardiographic results of the patient. (mitral regurgitation (MR), moderate tricuspid regurgitation (TR)).**Figure 2.** Echocardiographic results of the patient. (mitral regurgitation (MR), moderate tricuspid regurgitation (TR), and a dilated left atrium with a left ventricular ejection fraction of 60%).

3. Discussion

Obesity hypoventilation syndrome is a diagnosis of exclusion and is considered only after systematically ruling out common causes of hypoventilation, such as airway, neuromuscular, and chest wall disorders [1, 8]. In morbid obesity, excessive weight, genetic factors, sleep-related breathing disorders, and leptin resistance are among the proposed mechanisms that may contribute to a decreased ventilatory response. In individuals with morbid obesity, obstructive sleep apnea (OSA) is a worsening factor for OHS. Several studies indicate that these patients suffer symptoms such as hypercapnia, excessive daytime sleepiness, shortness of breath, cyanotic extremities, muscle weakness, snoring, and edema, and experience a lower quality of life compared to obese individuals without hypercapnia. Fat deposits around the thoracic area and abdomen decrease thoracic compliance and mobility, leading to challenges in expelling carbon dioxide, a reduced central ventilatory drive, upper airway obstruction, and resistance to leptin, the protein involved in regulating appetite and energy use [9].

As visceral fat accumulates, it restricts diaphragm movement, leading to reduced chest wall movement. Additionally, lung volume decreases, limiting expansion, which results in hypoxia, particularly when the patient is lying down [10]. Assessment of patients suspected of having OHS should start with a physical examination and a review of clinical history to identify any comorbid conditions [9]. Untreated obese OSA patients are at higher risk for coronary artery disease, heart failure, and diabetes, with the likelihood and severity of these comorbidities increasing alongside BMI [9, 11]. Therefore, early detection and swift treatment of OHS are crucial to prevent such complications.

The first-line approach for OHS includes noninvasive positive airway pressure (PAP) therapy and weight loss. The most frequently utilized PAP methods are continuous positive airway pressure (CPAP) and noninvasive ventilation (NIV) [12]. To lessen the significant cardiovascular and metabolic burden of OHS, a comprehensive multimodal therapy that includes home NIV/CPAP combined with lifestyle changes and rehabilitation is required [12, 13]. In this case, a morbidly obese patient (BMI = 51.3 kg/m²) presented with sleep-disordered breathing and daytime hypercapnia (PaCO₂ > 69.8 mm Hg) in the absence of other

known causes of hypoventilation. The patient was referred to our hospital for sleep medicine. The patient was nebulized with normal saline in the emergency department and was subsequently transferred to the MICU for further management. In the MICU, she received oxygen support through positive airway pressure (PAP), and blood tests and other investigations were completed as detailed in the case presentation.

A prior study assessed echocardiographic findings in 47 patients with obesity hypoventilation syndrome (OHS) who did not have obstructive airway disease as determined by pulmonary function tests. Left ventricular function was evaluated in a minimum of 39 patients, while right ventricular function was assessed in 25 patients. Dysfunction in both the left and right ventricles was observed in some of the patients [14]. NT-proBNP serves as a convenient diagnostic tool for identifying congestive heart failure [15].

While morbid obesity is link Echocardiographic assessment of our patient revealed valvular heart diseases, including moderate to severe mitral regurgitation and moderate tricuspid regurgitation, alongside heart failure with preserved ejection fraction. The investigation also noted elevated NT-proBNP levels and hypokalemia. Additionally, the patient had difficulty in sleeping due to drops in oxygen saturation during the night. Her treatment regimen included a formoterol and budesonide inhaler for respiratory issues, diuretics such as spironolactone and furosemide for heart-related concerns, potassium chloride syrup and magnesium sulfate for hypokalemia, as well as positive airway pressure (PAP) therapy. we received consultations from various departments, including cardiology, endocrinology, and otolaryngology, regularly and monitored the patient closely by following instructions from consulted departments.

ed to extended ICU stays and longer durations of mechanical ventilation, with the impact on overall and ICU mortality still debated, various bariatric surgical options have been explored for patients with super obesity (BMI > 50 kg/m²) [16]. In contrast, our patient was successfully treated using a multimodal conservative approach and was discharged after a six-days hospital stay without any surgical intervention.

4. Conclusion

Obesity-related morbidities, including OHS, are frequently misdiagnosed, which can lead to serious problems if medical practitioners didn't pay concern about these conditions during management or if adequate facilities are not available in the healthcare institutions where patients are admitted. Therefore, medical practitioners should address this concern when assessing an obese patient with respiratory insufficiency, and well-equipped healthcare institutions are required for the diagnosis, observation, and treatment of these conditions. Otherwise, it is preferable to refer patients to a higher-level center with comprehensive facilities.

Abbreviations

BMI	Body Mass Index
OHS	Obesity Hypoventilation Syndrome
PaCO ₂	Partial Pressure of Carbon Dioxide in Arterial Blood
OSA	Obstructive Sleep Apnea
NIV	Non-Invasive Ventilation
HFNC	High-Flow Nasal Cannula
ICU	Intensive Care Unit
WHO	World Health Organization
OSAHS	Obstructive Sleep Apnea Hypopnea Syndrome
PTB	Pulmonary Tuberculosis
MMRC	Modified Medical Research Council Dyspnea Scale
MICU	Medical Intensive Care Unit
GRBS	General Random Blood Sugar
VBG	Venous Blood Gas Analysis
MR	Mitral Regurgitation
TR	Tricuspid Regurgitation
EF	Ejection Fraction
CPAP	Continuous Positive Airway Pressure
BIPAP	Bilevel Positive Airway Pressure
PAP	Positive Airway Pressure
SGPT/ALT	Serum Glutamate Pyruvate Transaminase/Alanine Aminotransferase
SGOT/AST	Serum Glutamic Oxaloacetic Transaminase/Aspartate Aminotransferase
NT-proBNP	N-Terminal Prohormone of Brain Natriuretic Peptide
pH	Potential of Hydrogen
Pco ₂	Partial Pressure of Carbon Dioxide
Po ₂	Partial Pressure of Oxygen
HCO ₃ ⁻	Bicarbonate
MDI	Metered-Dose Inhaler

Author Contributions

Anil Shahi: Conceptualization, Data curation, Formal Analysis, Writing - original draft

Bishaka Adhikari: Conceptualization, Data curation, Formal Analysis, Writing - original draft

Bir Bahadur Syangtan: Conceptualization, Resources

Ulid Adhikari: Visualization, Writing - review & editing

Nischal Kafle: Visualization, Writing - review & editing

Informed Consent

The patient provided written informed consent for publishing of this case report and accompanying photographs. A copy of the written consent is available for review by the Editor-in-Chief of this journal upon request.

Conflicts of Interest

There was no financial support from any organization for the submission of this case report.

References

- [1] Pıhtılı A, Bingöl Z, Kıyan E. The predictors of obesity hypoventilation syndrome in obstructive sleep apnea. *Balkan Med J* 2017; 34: 41-46.
- [2] Basoglu OK, Tasbakan MS. Comparison of clinical characteristics in patients with obesity hypoventilation syndrome and obese obstructive sleep apnea syndrome: A case-control study. *Clinical Respiratory Journal* 2014; 8: 167-174.
- [3] Mokhlesi B, Tulaimat A, Faibussowitsch I, Wang Y, Evans AT. Obesity hypoventilation syndrome: Prevalence and predictors in patients with obstructive sleep apnea. *Sleep and Breathing* 2007; 11: 117-124.
- [4] Park MH, Lee MJ, Kim JS, Park SH, Lee JH, Kim AJ. Veno-venous extracorporeal membrane oxygenation rescue for pulmonary hypertension and hypoxemic respiratory failure to obesity hypoventilation syndrome: a case report. *Ann Palliat Med* 2022; 11: 3341-3345.
- [5] Eldaabossi S, Alrashdan M, Aljanobi G et al. A rare association: Obesity hypoventilation syndrome with myasthenia gravis and systemic lupus erythematosus, case report. *Respir Med Case Rep* 2023; 44.
- [6] Feng H-S, Xie M-R, Meng Y, Pei H-S, Yu J-J. Successful use of remimazolam combined with remifentanyl for painless gastroscopy in a patient with morbid obesity: a case report. *Front Oncol* 2024; 14.
- [7] Takaryanto D, Erna A, Suryadinata H. Pulmonary Hypertension in Obesity Hypoventilation Syndrome: A Case Report. 2017.
- [8] Utpat K, Desai U, Joshi JM, Bharmal RN. Obesity Hypoventilation Syndrome: New Insights in Diagnosis and Management. *Indian J Sleep Med* 2020; 15: 9-14.
- [9] Nacif SR, Aguiar IC, Fernandes De Oliveira E et al. Obesity-hypoventilation syndrome: Early diagnosis and immediate intervention-a case report.
- [10] Liu C, Chen M-S, Yu H. Oncotarget 93168 www.impactjournals.com/oncotarget The relationship between obstructive sleep apnea and obesity hypoventilation syndrome: a systematic review and meta-analysis. 2017.
- [11] Heryana OM, Wijono H. Clinical condition of patients with Obesity Hypoventilation Syndrome (OHS): case report. *Jurnal Teknologi Laboratorium* 2023.
- [12] Saka DDS, Novida H. An Indonesian adult with obesity hypoventilation syndrome: a case report. *Annals of Medicine & Surgery* 2023; 85: 2169-2172.
- [13] Masa JF, P épin JL, Borel JC, Mokhlesi B, Murphy PB, Sánchez-Quiroga MÁ. Obesity hypoventilation syndrome. *European Respiratory Review* 2019; 28.
- [14] Terla V, Rajbhandari GL, Kurian D, Pesola GR. A case of right ventricular dysfunction with right ventricular failure secondary to obesity hypoventilation syndrome. *American Journal of Case Reports* 2019; 20: 1487-1491.
- [15] Ozturk TC, Unluer E, Denizbasi A, Guneyssel O, Onur O. Can NT-proBNP be used as a criterion for heart failure hospitalization in emergency room? 2011.
- [16] Tatusov M, Joseph JJ, Cuneo BM. A case report of malignant obesity hypoventilation syndrome: A weighty problem in our ICUs. *Respir Med Case Rep* 2017; 20: 38-41.