*CASE REPORT*

# Intensive Care Management of a Patient With Pickwickian Syndrome, Obesity, and Congestive Heart Failure

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## ABSTRACT

**Background:** Pickwickian Syndrome is a condition of alveolar hypoventilation characterized by hypercapnia due to decreased ventilatory drive and capacity caused by obesity. The prevalence of this syndrome aligns with the increasing obesity rate in various countries, especially developed nations, and is associated with obstructive sleep apnea (OSA). Individuals with OSA have a 20–30% risk of developing Pickwickian Syndrome.
**Case:** A 36-year-old male, weighing 160 kg and height 168 cm (body mass index (BMI): 56.69 kg/m²), presented with progressive dyspnea for one week. He had a history of hypertension and heart disease, managed with medication. On arrival at the emergency department (ED), he showed signs of hypoxemia with $SpO_2$ 80%, which improved to 97% after oxygen therapy with a non-rebreathable breathing mask at 10 L/min. Blood gas analysis revealed partially compensated respiratory acidosis. A Chest X-ray showed bilateral pulmonary edema and cardiomegaly. A diagnosis of Pickwickian Syndrome with congestive heart failure was established. The patient was managed in the intensive care unit (ICU) for 13 days with non-invasive ventilation (NIV) as ventilatory support.
**Discussion:** This case illustrates the complex interplay among morbid obesity, hypoventilation, and cardiac dysfunction. Obesity leads to increased airway resistance and impaired thoracic compliance, resulting in reduced effective ventilation and $CO_2$ retention. When combined with congestive heart failure, pulmonary edema, and further hypoxemia may ensue. The successful outcome in this case underlines the importance of early diagnosis, targeted respiratory support, and effective fluid management.
**Conclusion:** Intensive care of the patients with Pickwickian Syndrome and congestive heart failure requires a holistic multidisciplinary approach. Optimizing oxygenation, maintaining strict fluid balance, and administering appropriate pharmacologic therapy are crucial to preventing complications and improving prognosis.

**Keywords:** alveolar hypoventilation; congestive heart failure; non-invasive ventilation (NIV); obesity hypoventilation syndrome (OHS); pickwickian syndrome

## INTRODUCTION

Pickwickian Syndrome refers to the presence of alveolar hypoventilation characterized by hypercapnia ($PaCO_2$ > 45 mmHg) due to a blunted respiratory drive and reduced ventilatory capacity as a result of obesity (body mass index (BMI) > 30 kg/m²).[1] The prevalence of Pickwickian syndrome parallels that of obesity in general, particularly in developed countries, and is closely associated with obstructive sleep apnea (OSA). It is estimated that 20–30% of individuals with OSA also have Pickwickian syndrome, with the highest risk factors being female gender, black ethnicity, lower educational level, and age between 40 and 59 years.[2]

This syndrome is related to the retention of carbon dioxide caused by the suppression of respiratory function and compensatory mechanisms in obese patients, along with increased airway resistance due to significant anatomical obstruction commonly found in individuals with obesity.[3] This phenomenon can become a serious issue if respiratory failure occurs, potentially requiring the patient to be connected to mechanical ventilation.

Management of obese patients with Pickwickian syndrome focuses on resolving respiratory acidosis and correcting hypercapnia resulting from carbon dioxide retention.[3,4] Treatment varies from the use of continuous positive airway pressure (CPAP) through non-invasive ventilation (NIV) to endotracheal intubation and mechanical ventilation support.

## CASE

A 36-year-old male presented to the emergency department (ED) with the chief complaint of shortness of breath that had been progressively worsening for one week before admission. The dyspnea was exacerbated by physical activity and lying flat (orthopnea and paroxysmal nocturnal dyspnea), frequently awakening the patient at night. He reported greater comfort sleeping in a right lateral position using more than two pillows. The complaint was accompanied by swelling of both lower extremities and abdominal distension, but was not associated with fever, cough, rhinorrhea, chest pain, nausea, or vomiting. The patient denied any recent increase in physical activity and reported fluid intake of less than 1600 cc per day as recommended by his physician. He had a known history of hypertension and heart disease diagnosed eight months earlier, with a prior hospitalization in September 2024 due to heart failure. His discharge medications included furosemide, valsartan, and amlodipine. There was no history of stroke, kidney disease, myocardial infarction, diabetes mellitus, or dyslipidemia. He was an active smoker. There was no history of stroke, kidney disease, myocardial infarction, diabetes mellitus, or dyslipidemia. Other known causes of hypoventilation, including severe obstructive or restrictive parenchymal lung disease, kyphoscoliosis, neuromuscular disease, and congenital central hypoventilation syndrome, were clinically excluded based on physical examination findings. Severe hypothyroidism was ruled out through laboratory assessment of thyroid function, wasn't performed.

Vital signs upon arrival at the ED showed a blood pressure of 132/82 mmHg, heart rate of 98 beats per minute (bpm), respiratory rate of 24 breaths per minute, body temperature of 37.7°C, and oxygen saturation of 80% on room air, which improved to 97% after administration of oxygen via non-rebreathing mask (NRM) at 10 L/min.

The patient was alert and oriented, with a glasgow coma scale (GCS) of 15 (E4M6V5).

Physical examination revealed morbid obesity (weight 160 kg, height 168 cm, BMI 56.69 kg/m²). Thoracic assessment showed symmetrical chest movement without retractions or trauma; tactile fremitus was equal bilaterally, percussion was resonant, and auscultation revealed vesicular breath sounds with bibasilar rales. Abdominal examination showed distension with ascites, normal bowel sounds, a soft, non-tender abdomen without hepatosplenomegaly, but with shifting dullness. Other physical examinations were within normal limits. Jugular venous pressure was observed at a normal limit during initial physical examination.

Initial laboratory evaluation revealed elevated erythrocyte count (6.31 $\times 10^6/\mu L$), hemoglobin (16.1 g/dL), and hematocrit (55.3%), indicating polycythemia. Red cell indices showed normal MCV (87.6 fL) but decreased MCH (25.5 pg) and MCHC (29.1 g/dL), along with markedly increased RDW-SD (61.0 fL) and RDW-CV (19.9%). Nucleated red blood cells were present (NRBC% 0.3%; NRBC# 0.07 $\times 10^3/\mu L$). White blood cell count was within normal limits (6.8 $\times 10^3/\mu L$), but with neutrophilia (75.0%) and relative lymphopenia (10.6%; absolute 0.70 $\times 10^3/\mu L$). Monocyte percentage was mildly elevated (11.6%), while eosinophil and basophil counts were normal. Platelet count was normal (224 $\times 10^3/\mu L$) with unremarkable platelet indices.

Initial chest radiograph (Figure 1) revealed bilateral pulmonary infiltrates accompanied by increased and blurred vascular markings. There was evidence of cephalization, peribronchial cuffing, and a characteristic batwing appearance, along with visible air bronchograms. Additionally, a ground-glass opacity was observed occupying both hemithoraces, more pronounced on the right side, with signs of pleural effusion.

Arterial blood gas analysis in the ED revealed partially compensated respiratory acidosis, with pH 7.237, $pCO_2$ 79.9 mmHg, $pO_2$ 67 mmHg, $HCO_3^-$ 34.2 mmol/L, and oxygen saturation of 88%. Initial management included oxygen therapy via an NRM 10 L/min, followed by initiation of non-invasive ventilation (NIV) in the ICU. The patient also received 3 bolus doses (120 mg) of intravenous furosemide, continued with 40 mg of intravenous furosemide every 8 hours, with a target fluid balance of negative 500 to negative 1000 cc/day.

On the first day of ICU treatment, arterial blood gas results showed a pH of 7.241, $pCO_2$ increased to 95.4 mmHg, $pO_2$ 73.8 mmHg, $HCO_3^-$ 40.1 mmol/L, and oxygen saturation of 92.2%. NIV was maintained with settings of pressure support 12, PEEP 5, $FiO_2$ 70%, and exhaled tidal volumes ranging from 280–350 ml. During the ICU stay, the patient received a treatment regimen consisting of intravenous furosemide 20 mg every 12 hours, spironolactone 25 mg once, sacubitril/valsartan 100 mg twice daily, bisoprolol 1.25 mg once daily, and nifedipine 30 mg once daily for blood pressure and heart failure management. Antibiotic therapy with intravenous cefepime 1 gram every 8 hours. Additional supportive treatments included paracetamol 1 gram every 8 hours as needed for pain or fever, omeprazole 40 mg once daily for gastrointestinal protection, and N-

acetylcysteine 200 mg every 9 hours. Respiratory therapy included nebulization with Ipratropium bromide/albuterol every 1.5 hours.

On the second to third days, serial arterial blood gas evaluations demonstrated gradual improvement in chronic respiratory acidosis, with near-normal pH, improved oxygen saturation, and continued metabolic compensation as $HCO_3^-$ increased to 43.91 mmol/L. By the seventh day, arterial blood gas showed a pH of 7.38, $pCO_2$ 83.4 mmHg, and $HCO_3^-$ 50.13 mmol/L. Diuretic and antihypertensive therapies were continued, with an ongoing negative fluid balance strategy. (Table 1)

On the eighth day, the patient was transitioned from NIV back to NRM at 10 L/min. Diuretic and antihypertensive therapies were continued, with an ongoing negative fluid balance strategy. Clinical (oxygenation) and laboratory improvements (arterial blood gas pH) indicated a positive response to treatment, and the patient's further management focused on respiratory rehabilitation and long-term weight reduction.

During the ICU stay, the patient was planned to meet at least 80% of his nutritional requirements without increasing the burden on his respiratory system. The primary strategy was a low-carbohydrate, high-fat diet, designed to minimize carbon dioxide production and reduce ventilatory load. Using the Ireton-Jones equation for ventilator-dependent patients, his estimated energy requirement was 2432 kcal/day, with protein needs at 79.6 grams/day (1.3 g/kg ideal body weight), fats at 94.6 grams/day (35% of total energy), and carbohydrates limited to 315 grams/day. To initiate nutritional support, a hypocaloric regimen was implemented, starting at 900 kcal/day via oral intake through a nasogastric tube (NGT). The diet included a high-fat, low-carbohydrate formula, administered six times per day at 150 ml per serving—split into three main meals and three snacks. (Table 3)



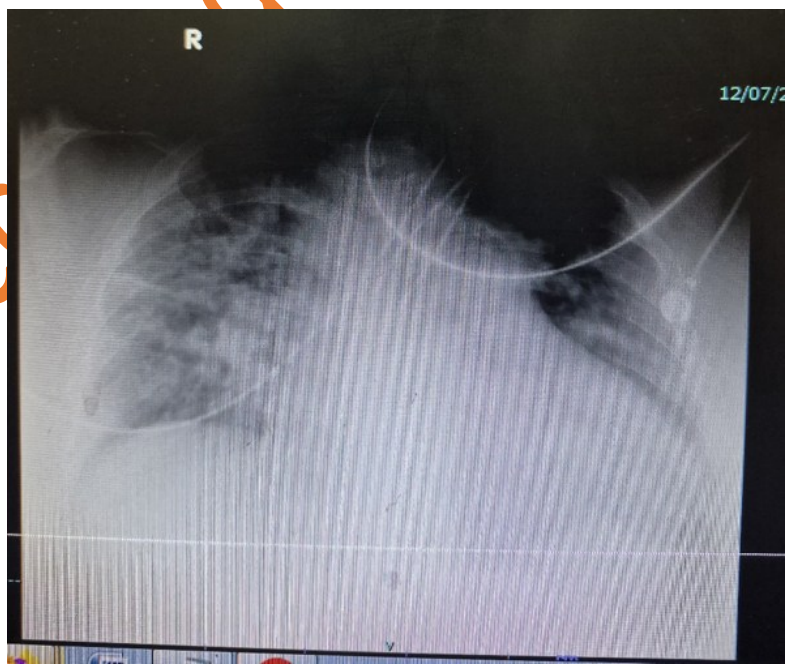


**Figure 1.** Initial chest radiograph

**Table 1**. Blood gas analysis result

| | D0 | D1 | D2 | D3 | D7 |
|---|---|---|---|---|---|
| pH | 7.237 | 7.241 | 7.29 | 7.293 | 7.38 |
| PaCO2 | 79.9 | 95.4 | 85.7 | 92.7 | 83.4 |
| PaO2 | 67 | 73.8 | 76.5 | 75.7 | 71 |
| HCO3- | 34.2 | 40.1 | 40.3 | 43.91 | 50.13 |
| SaO2 (%) | 88 | 92.2 | 95.3 | 94.7 | 92 |
| BE | 7 | 7.96 | 9.32 | 12.09 | 25 |

**Table 3. ……..**

| Day | Mode | IPAP (cmh 20) | EPAP (cmh20) | Fio2 | RR (Backup) | Notes |
|---|---|---|---|---|---|---|
| 1 | Bipap S/T | 18 | 8 | 0.6 | 16 | Severe hypercapnia acidosis correction initiated |
| 3 | Bipap S/T | 16 | 8 | 0.5 | 14 | More stable gas exchange |
| 5 | Bipap S/T | 15 | 7 | 0.45 | 14 | Better oxygenation, less desaturation at night |
| 7 | Bipap S/T | 14 | 6 | 0.4 | 12 | PaCO2 near baseline, O2 stable |
| 9 | Bipap S/T | 12 | 5 | 0.35 | 12 | Transition CPAP at night |
| 11 | CPAP | | 6 | 0.30 | - | Daytime room air trial |
| 12 | CPAP | | 5 | 0.25 | - | Ready to discharge |



## DISCUSSION

Pickwickian Syndrome, also known as obesity hypoventilation syndrome (OHS), refers to the presence of alveolar hypoventilation characterized by hypercapnia ($PaCO_2 > 45$ mmHg) due to a blunted respiratory drive and reduced ventilatory capacity caused by obesity (BMI > 30 kg/m²)[5]. The prevalence of Pickwickian Syndrome mirrors that of obesity in general, especially in developed countries, and is commonly associated with obstructive sleep apnea (OSA). It is estimated that 20–30% of individuals with OSA also have Pickwickian Syndrome, with the highest risk factors found in females, individuals of Black ethnicity, those with lower educational levels, and those aged 40–59[2]. In Asian populations, Pickwickian Syndrome can occur at lower BMIs compared to Western populations.[2,5]

Obesity hypoventilation syndrome (OHS) is associated with significant morbidity and mortality, with reported 5-year mortality rates ranging from 15–30%, largely due to cardiopulmonary complications such as pulmonary hypertension, right heart failure, and recurrent respiratory failure.[5] Management of hospitalized OHS patients with acute-on-chronic hypercapnic respiratory failure begins

with prompt identification of risk factors (e.g., severe obesity, symptoms of sleep-disordered breathing, comorbid cardiopulmonary disease) and confirmation of diagnosis through arterial blood gas analysis. Initial treatment targets include correcting hypoxemia and hypercapnia, often with non-invasive positive pressure ventilation (NIPPV), optimizing fluid balance, and addressing precipitating factors such as infection or heart failure exacerbation.[4] Once stabilized, evaluation for underlying pulmonary pathology with pulmonary function testing (PFT) is indicated; in OHS, PFTs often reveal a restrictive pattern with reduced total lung capacity but normal airflow rates, helping to differentiate from obstructive or mixed defects.[5,6] Bariatric surgery remains an evidence-based therapeutic option for selected patients, as it can result in substantial and sustained weight loss, improvement in gas exchange, and reduction in recurrence of respiratory failure; however, candidacy should be determined after multidisciplinary assessment, and in this case, bariatric surgery may be considered to prevent future decompensation.[5]

In general, Pickwickian Syndrome is caused by obesity, which suppresses respiratory function and central compensatory mechanisms for hypercapnia.[7] Obesity increases pressure on the upper airway, leading to suboptimal ventilation. In the thoracic cavity, obese individuals also experience increased resistance during the initiation of the inspiratory phase, resulting in reduced tidal volume, functional residual capacity, and forced expiratory volume. These mechanisms collectively contribute to reduced minute ventilation and fresh gas inspiration, leading to the retention and rebreathing of exhaled gases, and ultimately hypercapnia.[5,7]

Clinically, patients with Pickwickian Syndrome may maintain a relatively normal quality of life until decompensation occurs due to severe hypoventilation. A decrease in daily quality of life can manifest as daytime fatigue and other issues related to obesity (including metabolic factors). In chronic phases, pulmonary hypertension and right heart chamber hypertrophy may develop as a result of long-term compensation for hypoxemia and chronic hypoventilation.[1,8] If decompensation occurs due to elevated pulmonary artery pressure and/or accompanying congestive heart failure, respiratory failure may ensue in individuals with Pickwickian Syndrome.[4]

Diagnosis of Pickwickian Syndrome typically begins with arterial blood gas analysis, assessing $PaCO_2$ (partial pressure of carbon dioxide), $PaO_2$ (partial pressure of oxygen), pulmonary function (vital capacity, forced expiratory volume), and sleep studies in obese individuals (BMI > 30 kg/m²).[4,5] In Pickwickian Syndrome, hypoventilation is indicated by elevated $PaCO_2$ above the normal range during wakefulness ($PaCO_2$ > 45 mmHg), accompanied by hypoxemia ($PaO_2$ < 70 mmHg). The gold standard for diagnosing Pickwickian Syndrome is a sleep study that assesses oxygen saturation and the duration of sleep during which $SpO_2$ remains below 90%.[5]

Initial treatment for individuals with Pickwickian Syndrome involves the use of CPAP.[9] CPAP helps to keep the airway open, reducing excess pressure or resistance in the upper airway caused by obesity. The use of CPAP can reduce the retention and rebreathing of exhaled

gases. However, in individuals with acute respiratory distress and respiratory failure due to Pickwickian Syndrome, intubation and mechanical ventilation may be more appropriate to ensure airway protection in cases of severe shortness of breath.[3]

In this case, the patient presented with complaints of shortness of breath, which worsened when lying flat and improved when sitting upright or lying on one side. The patient also had swelling in the lower limbs and abdomen. The patient had a history of heart disease and hypertension, with regular follow-ups with a cardiologist, and was on routine diuretics and antihypertensive medication. According to the patient, their average daily systolic blood pressure ranged from 120 to 130 mmHg, well-controlled with a combination of one diuretic and two antihypertensive medications. The patient also had a large body habitus, weighing 160 kilograms with a height of 168 cm. The calculated body mass index was 56.59 $kg/m^2$, classified as morbid obesity.

Initial management in this patient focused on oxygenation. The patient presented with shortness of breath characterized by a respiratory rate above 20 breaths per minute and peripheral oxygen saturation below 94%. The respiratory rate was 24 breaths per minute, with an oxygen saturation of 80% on room air. Initial oxygen therapy was administered using an NRM with an oxygen flow rate of 10 liters per minute. After NRM use, the oxygen saturation improved to 97%.

Subsequent management focused on carbon dioxide clearance. Arterial blood gas analysis revealed elevated $PaCO_2$ along with decreased arterial pH and increased $HCO_3^-$, indicating partially compensated respiratory acidosis. A reduction in $PaO_2$ was also noted, indicating hypoxemia. The patient was connected to non-invasive ventilation (NIV) with a setting of Pressure Support 12, PEEP 5, $FiO_2$ 70%, and tidal volume ranging from 280–350 cc.

Non-invasive positive pressure ventilation (NIPPV) is contraindicated in patients with severe altered mental status, inability to protect the airway, excessive secretions, severe hemodynamic instability, facial trauma or deformity preventing mask seal, or recent upper airway or gastrointestinal surgery.[10] In this case, none of these contraindications were present. The effectiveness of early NIPPV is typically assessed within the first 1–2 hours of initiation.[11] Failure is suggested by persistent or worsening signs of respiratory distress (e.g., tachypnea > 35 breaths/min, use of accessory muscles), hypoxemia ($SpO_2$ < 90% or $PaO_2$ < 60 mmHg despite optimal settings), hypercapnia with acidosis (pH < 7.25), hemodynamic instability, or decreased level of consciousness.[10,11] If such parameters are met despite optimal support, transition to invasive mechanical ventilation is indicated.[11]

Physical examination revealed bilateral rales in the lower third of the lungs, and supporting examinations showed increased vascular markings, cephalization, and a batwing appearance suggestive of pulmonary edema and an increased cardio-thoracic ratio. Given the patient's history of heart disease and the use of diuretics and antihypertensives, the presence of cardiogenic pulmonary edema was concluded. In addition to NIV, the patient was given a diuretic regimen with furosemide and oral antihypertensives, including amlodipine and candesartan.

An elective echocardiographic examination showed global chamber dilatation and a reduced ejection fraction of 30%, consistent with dilated cardiomyopathy. The plan for this patient included maintaining adequate oxygen saturation and achieving a negative fluid balance.

Pulmonary hypertension is a recognized complication of OHS, often secondary to chronic hypoxemia and resultant pulmonary vasoconstriction.[5] In this patient, the possibility of pulmonary hypertension was considered; however, clinical examination did not reveal signs suggestive of right heart failure, such as elevated jugular venous pressure, significant peripheral edema, or a loud second heart sound. Definitive evaluation of pulmonary hypertension can be performed using transthoracic echocardiography to estimate pulmonary artery systolic pressure, with right heart catheterization considered the gold standard for diagnosis.[12] Additional supportive assessments include chest imaging to evaluate pulmonary vasculature and BNP/NT-proBNP levels as markers of right ventricular strain.[12]

Weight reduction is also an important focus in treating Pickwickian Syndrome. A calorie-controlled, low-carbohydrate diet may be introduced to minimize $CO_2$ production from metabolism, which is beneficial in hypercapnic states. In the ICU, this can be done by targeting a calorie goal based on ideal daily energy requirements. However, in obese individuals, the main principles of nutrition therapy are hypocaloric and high-protein feeding. A target of 65–70% of estimated energy expenditure is commonly recommended, roughly 11–14 kcal/kg actual body weight/day or 22–25 kcal/kg ideal body weight/day, depending on the patient's BMI.[13] Protein needs are elevated to 2.0–2.5 g/kg ideal body weight/day to support nitrogen balance and prevent muscle catabolism.[14,15] Enteral nutrition (EN) is the preferred route of feeding unless contraindicated, as it maintains gut integrity and reduces infection risk. Parenteral nutrition (PN) may be considered if EN is not feasible or fails to meet caloric targets.[16] Close monitoring of glucose, triglycerides, electrolytes, and fluid status is essential.

Ventilatory management in treating Pickwickian Syndrome focuses on correcting chronic alveolar hypoventilation, hypercapnia, and hypoxemia while reducing the patient's work of breathing. The first-line therapy is non-invasive ventilation (NIV), primarily using Bi-level Positive Airway Pressure (BiPAP), which provides inspiratory pressure support and expiratory pressure to improve ventilation and oxygenation[17]. Initial settings typically involve an inspiratory pressure (IPAP) of 10–20 $cmH_2O$ and expiratory pressure (EPAP) of 4–8 $cmH_2O$, adjusted according to arterial blood gas results and clinical response. NIV helps normalize $PaCO_2$, improve oxygen saturation and pH, relieve dyspnea, and enhance sleep quality, particularly in patients with concurrent OSA.

The main goal of NIV use is to resolve arterial pH by correcting hypercapnia, whether due to a primary airway problem or carbon dioxide retention.[9,18] Therefore, in this patient, routine and periodic blood gas analysis was conducted to monitor the resolution of respiratory acidosis following carbon dioxide retention. And only after the treatment goals were achieved, NIV was discontinued.

## CONCLUSION

The intensive care management of obese patients with Pickwickian Syndrome and congestive heart failure should target improved oxygenation, hemodynamic stability, and reduced morbidity through a coordinated multidisciplinary approach involving intensivists, pulmonologists, cardiologists, and nutritionists. Individualized ventilation strategies, strict fluid balance, and tailored pharmacotherapy must be complemented by continuous monitoring of arterial blood gases, oxygenation indices, and cardiac function. Beyond preventing fluid overload, clinicians should anticipate complications such as thromboembolism, pulmonary hypertension, and arrhythmias. Future research is needed to define optimal ventilatory protocols, long-term weight management strategies, and integrated care pathways to enhance both acute and long-term outcomes.

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