American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2024; 25: e945112
DOI: 10.12659/AJCR.945112

Received: 2024.05.10
Accepted: 2024.08.20
Available online: 2024.08.28
Published: 2024.10.02

Management of Obesity Hypoventilation Syndrome in Extreme Obesity: A Case Study

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Financial support: None declared
Conflict of interest: None declared

Patient: Male, 39-year-old
Final Diagnosis: Acute hypercapnic respiratory failure
Symptoms: Respiratory distress • altered consciousness • an inability to move independently
Clinical Procedure: —
Specialty: Critical Care Medicine

Objective: Rare coexistence of disease or pathology

Background: Obesity hypoventilation syndrome (OHS) is characterized by hypercapnia in obese patients, with acute hypercapnic respiratory failure often worsened by various conditions. Managing super-super obese patients presents complex challenges in critical care. Our case report details the successful treatment of acute respiratory failure in a patient with a body mass index (BMI) over 80 kg/m^2, highlighting the importance of comprehensive, multidisciplinary care in the Intensive Care Init (ICU).

Case Report: A 39-year-old man with a BMI of 81.1 kg/m^2 presented to our emergency department with respiratory distress, altered consciousness, and an inability to move independently. Arterial blood gas analysis revealed severe hypercapnia and hypoxemia, indicating decompensated OHS. Laboratory tests and computed tomography scans suggested his condition was exacerbated by pneumonia and congestive heart failure. The patient was managed in the ICU with endotracheal intubation, mechanical ventilation, and esophageal pressure monitoring. In addition to antibiotics, diuretics were used to manage fluid balance. His care included multidisciplinary support with nutritional management and active physiotherapy. After 15 days, he was weaned from the ventilator and discharged from the ICU on day 20, continuing rehabilitation until he was discharged home on day 60.

Conclusions: This case report describes the successful treatment of acute hypercapnic respiratory failure from decompensated OHS in a super-super obese patient. Addressing the underlying conditions and tailoring clinical practices to the patient's specific needs, especially regarding ventilatory support, fluid balance, and nutrition, were crucial. A collaborative multidisciplinary approach was essential for improving outcomes.

Keywords: Critical Illness • Hypoventilation • Obesity • Respiratory Insufficiency

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/945112

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Introduction

The obesity paradox refers to the curious observation that obesity increases the risk of associated diseases yet, paradoxically, confers a short-term survival advantage in acute medical conditions such as sepsis or acute respiratory distress syndrome [1]. However, this paradox does not extend to patients with extraordinary obesity.

Obesity hypoventilation syndrome (OHS) is defined by hypercapnia (typically partial pressure of arterial carbon dioxide ≥45 mmHg) in patients with obesity (body mass index (BMI) ≥30 kg/m^2), excluding other alternative causes for hypoventilation [2]. Acute hypercapnic respiratory failure in these patients can be exacerbated by various conditions, including neuromuscular or mechanical issues, as well as pneumonia and congestive heart failure.

Managing a patient with super-super obesity, defined as a BMI ≥60 kg/m^2 [3], presents multifaceted challenges [1]. Exceptionally few cases of patients with a BMI over 80 kg/m^2 and reluctance to treat have been reported in acute and critical care settings [4,5]. Complications of treating obese patients in the ICU include difficulties in mechanical ventilation, increased risk of infections, and challenges in mobilization and rehabilitation [1]. These complications necessitate a multidisciplinary approach to effectively manage and treat these patients.

Our case report details the successful comprehensive treatment of acute respiratory failure due to hypoventilation in a patient with a BMI exceeding 80 kg/m^2, possibly exacerbated by pneumonia and congestive heart failure. This report highlights the importance of a multidisciplinary team in the ICU, offering insights into the complexities of managing extremely obese patients in critical care settings. Through this report, we aim to enhance understanding and management strategies among clinicians dealing with such challenging cases.

Case Report

A 39-year-old Japanese man had been experiencing muscle weakness in his lower extremities over the past month. Despite his wife's recommendation to see a doctor, he initially declined. Until the day before his hospital visit, his wife had been assisting him with daily activities. However, on the day of emergency transport to our hospital, he presented with respiratory distress, altered consciousness, and an inability to move independently. He had been receiving psychiatric treatment for depression and panic disorder, taking paroxetine, nitrazepam, and bromazepam daily.

On arrival, his vital signs were as follows: Glasgow Coma Scale score of 8 (E1V2M5), with respiratory rate of 22 breaths/min, heart rate of 108 beats/min, oxygen saturation of 87% on supplemental oxygen at 15 L/min, and body temperature of 36.9°C. Blood pressure measurements were not possible as the cuff could not be applied. His height was 175 cm, and the patient's estimated weight was at least 250 kg (BMI 81.1 kg/m^2), as this was the upper limit of the measuring scale. A peripherally inserted central catheter was placed in his right upper arm, and an arterial line was inserted in his left brachial artery, showing his blood pressure of 159/101 mmHg. An arterial blood gas analysis revealed pH of 7.103, $PaCO_2$ of 125 mmHg, PaO_2 of 80 mmHg, and HCO_3^- of 37.3 mmol/L, indicating hypercapnic respiratory failure due to decompensated OHS. Blood tests were interpreted as indicating infectious diseases, abnormal liver function tests, and heart failure (**Table 1**). Chest computed tomography scan revealed infiltration shadows in the bilateral lung fields and cardiomegaly (**Figure 1**). No apparent abnormalities were identified in the abdominal and pelvic cavities.

Three hours after his arrival and admission to the emergency Intensive Care Unit (ICU), he appeared distressed. Based on his acute respiratory failure and distress from hypoventilation, non-invasive ventilation was not applied due to agitation and non-cooperation. Therefore, we opted for endotracheal intubation with the consent of the family. Mask ventilation was aided by a nasopharyngeal airway. After premedication with phytanyl and ketamine, endotracheal intubation was successfully performed using video laryngoscopy (**Figures 1B, 2A**). We utilized esophageal pressure monitoring and titrated positive end-expiratory pressure (PEEP) to maintain positive expiratory transpulmonary pressure. Initially, a PEEP of 18 to 20 cmH_2O was necessary during the first 10 days of mechanical ventilation, after which we gradually reduced PEEP levels to 10 cmH_2O, guided by transpulmonary pressure. As for his cardiovascular system, a poor acoustic window limited our ability to assess his cardiac function via transthoracic echocardiography. However, overall left ventricular wall motion was noted to be adequate. Additionally, a dilated inferior vena cava, measured at 32.4 mm via CT scan, indicated right heart failure likely due to pulmonary hypertension associated with severe obesity. Suspecting that his OHS was exacerbated by sepsis due to pneumonia, we initiated treatment with 4.5 g of piperacillin/tazobactam every 6 hours. Later, blood cultures showed no growth, while sputum cultures identified a polymicrobial strain. Piperacillin/tazobactam was discontinued on day 12. Key treatments also included diuretics to facilitate extra water removal and weight loss. Diuretics led to a decrease in total bilirubin to 3.89 mg/dL the next day, brain natriuretic peptide to 100.7 pg/mL the following day, and a prothrombin time-international normalized ratio of 1.15 a week later, indicating improvement in hepatic injury due to right heart failure. Additionally, unfractionated heparin was administered to prevent deep vein thrombosis. Enteral nutrition was initiated on the next day and gradually increased over the course of 7 days, reaching a total of 960

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Table 1. Laboratory data on admission.

Arterial blood gas (15 L/min of oxygen)		Biochemistry	
pH	7.103	Total protein	7.5 g/dL
$PaCO_2$	125 mmHg	Albumin	3.5 g/dL
PaO_2	80.0 mmHg	Total bilirubin	5.11 mg/dL
HCO_3^-	37.3 mmol/L	Direct bilirubin	2.81 mg/dL
Base excess	0.9 mmol/L	GGT	24 IU/L
Lactate	3.2 mmol/L	AST	77 IU/L
Complete blood count		ALT	51 IU/L
White blood cells	11,270/μL	LDH	475 IU/L
Neutrophil (%)	74.6	Creatinine kinase	1,403 IU/L
Lymphocyte (%)	15.6	BUN	18.7 mg/dL
Hemoglobin	17.0 g/dL	Creatinine	0.91 mg/dL
Hematocrit	60.4%	Sodium	141 mEq/L
Platelet count	22.5×10^4/μL	Potassium	5.6 mEq/L
Coagulation		Chloride	98 mEq/L
PT (%)	35	Glucose	141 mg/dL
PT-INR	1.70	CRP	4.69 mg/dL
APTT	39.5 s	Procalcitonin	0.508 ng/mL
Fibrinogen	261 mg/dL	BNP	562.4 pg/mL
D-dimer	26.3 μL/mL		

PT – prothrombin time; PT-INR – prothrombin time-international normalized ratio; APTT – activated partial thromboplastin time; GGT – gamma-glutamyl transpeptidase; AST – aspartate aminotransferase; ALT – alanine aminotransferase; LDH – lactate dehydrogenase; BUN – blood urea nitrogen; CRP – C-reactive protein; BNP – brain natriuretic peptide.

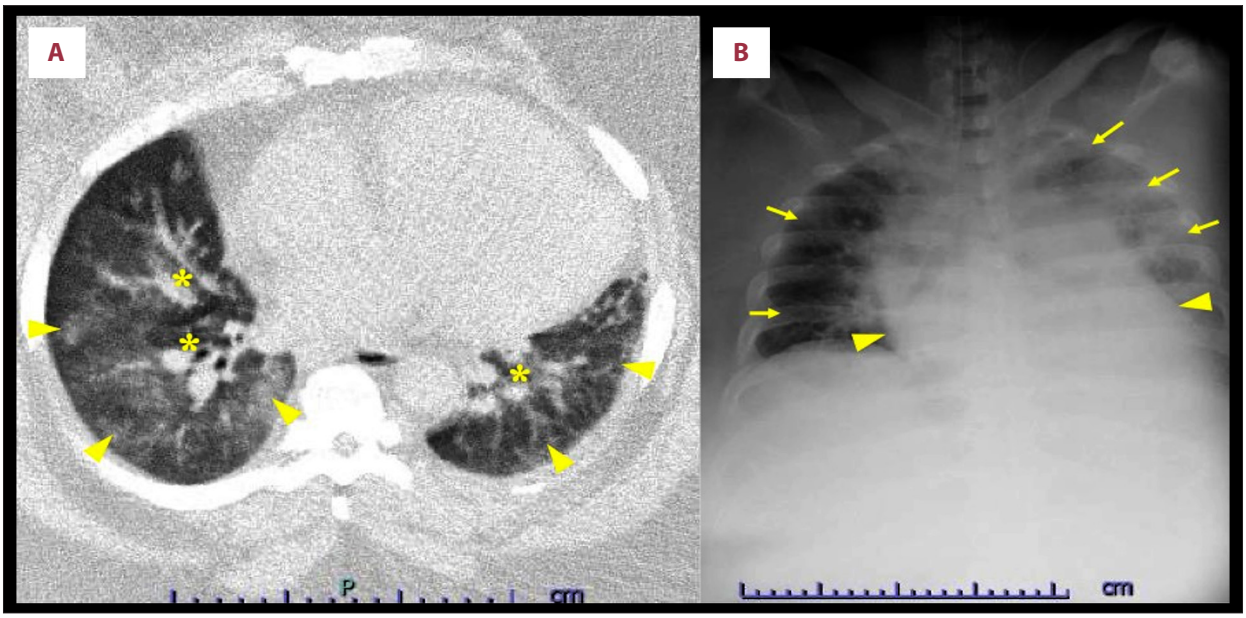

Figure 1. Chest computed tomography (**A**) and chest X-ray (**B**) upon admission. (**A**) Chest computed tomography demonstrating enhanced pulmonary vascular markings (asterisk) and infiltration shadows (triangle arrow) in the bilateral lung fields. (**B**) Chest X-ray taken immediately after endotracheal intubation, showing cardiomegaly (triangle arrow) and bilateral infiltrates (arrow).

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Figure 2. Patient's photos on ICU admission and on the18th day. (**A**) Patient with a body weight of at least 250 kg on ICU admission. (**B**) Encouraging the patient to get out of bed 3 days after extubation. ICU – Intensive Care Unit.

kcal/day with 48 g/day of protein. Psychiatrists intervened for delirium and insomnia, promoting daytime wakefulness, and adopting a semi-sitting position. Active physiotherapy was also implemented. **Figure 3** illustrates his clinical course, focusing on ventilator settings, fluid balance, and body weight.

On the 15th day, his body weight was 221.3 kg. The arterial blood gas analysis showed a pH of 7.385, PaO_2 of 72.7 mmHg, $PaCO_2$ of 56.9 mmHg, and HCO_3^- of 33.9 mmol/L, with F_IO_2 set at 0.5 in spontaneous mode. The patient was successfully weaned from the ventilator and extubated, followed by application of non-invasive positive pressure ventilation over the next 24 hours. Encouraging the patient to get out of bed (**Figure 2B**), he was discharged from the emergency ICU on hospital day 20. After leaving the ICU, continuous positive airway pressure was initiated during nighttime due to high suspicion of obstructive sleep apnea. This treatment was continued after discharge to home. Following further rehabilitation, the patient was discharged to home on day 60, weighing 189.4 kg.

Discussion

Acute respiratory failure in extremely obese patients presents significant challenges, including difficulties with mechanical ventilation, higher infection risks, and complexities in mobilization and rehabilitation [1]. Despite an emphasis on individualized, multidisciplinary care, optimal strategies for managing super-super obese patients are not well established. This case report addresses these gaps by showcasing a successful treatment approach, underscoring the importance of tailored clinical practices and a coordinated multidisciplinary team to improve patient outcomes.

The patient was presumed to have OHS, defined by a BMI of at least 30 kg/m^2 and elevated $PaCO_2$ (≥45 mmHg) during wakefulness [2], as evidenced by metabolic compensation in his blood gas analysis. Concurrently, obstructive sleep apnea was noted, but other potential causes of hypoventilation such as chronic obstructive pulmonary disease, restrictive lung diseases, or neuromuscular disorders were clinically excluded [6]. OHS, which is characterized by multi-organ dysfunction, can quickly progress to respiratory failure if intensified by conditions such as asthma, heart failure, or infections. In this case, despite limitations in echocardiography due to the patient's thick chest wall, congestive heart failure was diagnosed based on pulmonary congestion, dilated inferior vena cava, and high brain natriuretic peptide levels, with a potential complication from pneumonia. Thus, the primary treatments included the administration of antibiotics and diuretics.

In treating super-super obese patients, clinicians face profound challenges such as diagnostic and logistical issues, monitoring difficulties, vascular access problems, and pharmacokinetic changes [7]. Among them, complexities in airway and ventilatory management, as well as nutritional support are discussed.

Airway Management

We facilitated airway management by using a nasopharyngeal airway and video laryngoscopy for intubation under light sedation. Additionally, the efficiency of styletubation for tracheal intubation, which involves using a video-assisted intubating stylet, has been reported in super-super obese patients [8].

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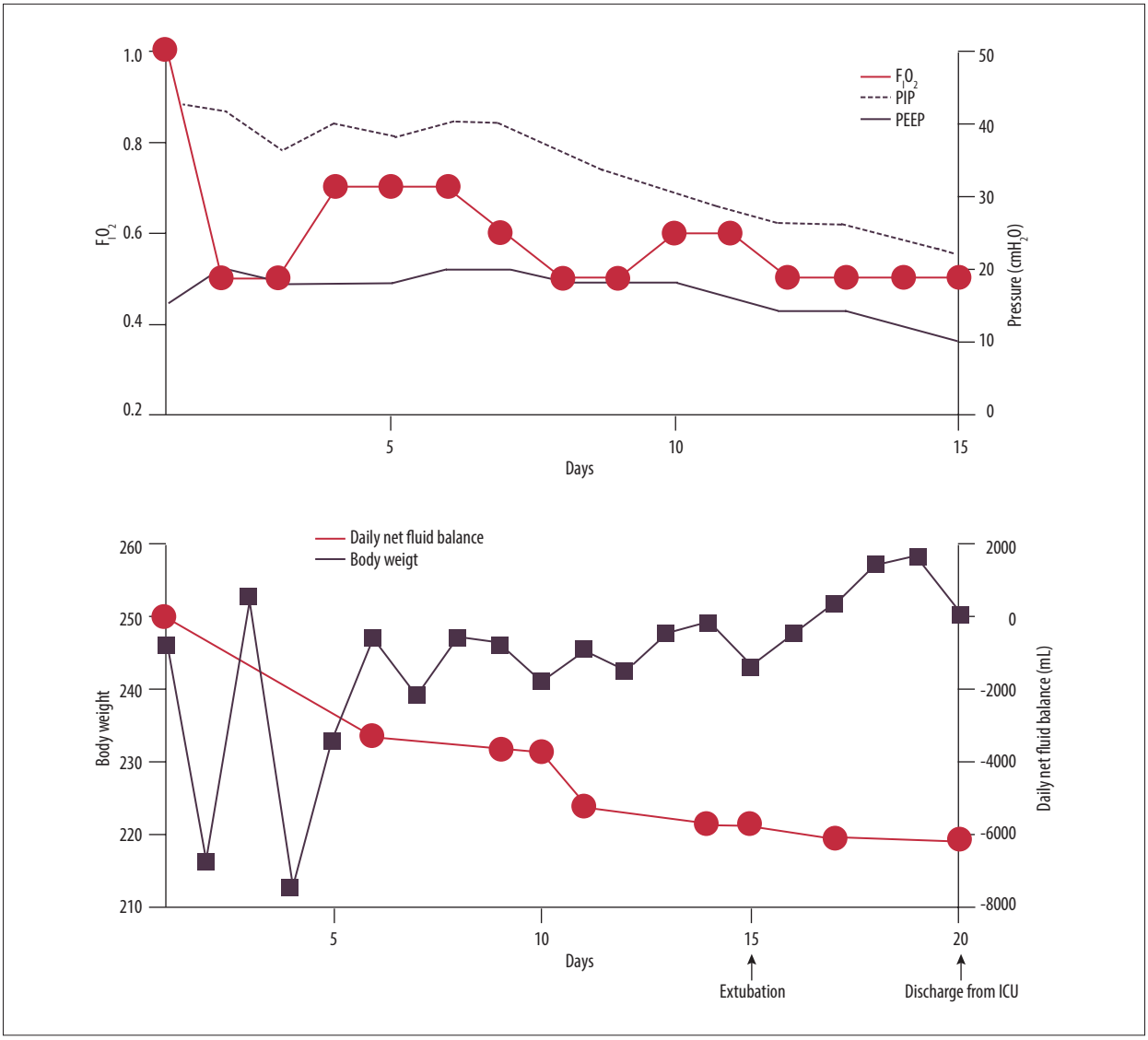

Figure 3. Patient's clinical course. (**A**) Ventilator settings. (**B**) Trajectories of daily net fluid balance and patient's body weight. PIP – peak inspiratory pressure; PEEP – positive end-expiratory pressure; ICU – Intensive Care Unit.

Ventilatory Support

Studies show benefits of using transpulmonary pressure monitoring in managing respiratory care in obese patients, potentially reducing ventilator-induced lung injuries [9]. The optimal PEEP settings for obese patients may differ significantly from standard protocols, suggesting the need for a tailored approach based on individual assessments, especially for those with super obesity. Regarding extubation, we used a PEEP of 10 cmH_2O just before extubation and followed up with non-invasive ventilation, potentially preventing reintubation [10].

Nutritional Management

As for nutritional management, relevant guidelines recommending 22-25 kcal/kg of ideal body weight per day (for BMI >50 kg/m^2) may not be applicable to those with super obesity [1]. According to these guidelines, the minimum requirement for our case was calculated to be 1482 kcal/day. However, only 960 kcal/day was actually administered during mechanical ventilation. Such underfeeding might be considered reasonable and acceptable for those with super-super obesity, based on a previous case report [11].

Furthermore, maintaining fluid balance was crucial, with diuretics aiding water removal and weight loss: literature supports careful fluid management in critically ill obese patients to

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prevent complications [1]. A multidisciplinary team, including nurses, physical therapists, nutritionists, and pharmacists, provided comprehensive care, addressing complex medical, psychological, and social needs. This case shows the effectiveness of a tailored, collaborative approach in managing acute respiratory failure due to decompensated OHS in an extremely obese patient, aligning with previous reports and highlighting the need for further research to refine management strategies [4,5,11].

Conclusions

This case report highlights the successful treatment of acute hypercapnic respiratory failure due to decompensated OHS in a patient with super-super obesity. In addition to addressing the underlying conditions contributing to decompensation, tailoring clinical practices to the specific physiological characteristics of these patients, particularly in terms of ventilatory support, fluid balance, and nutritional support, is crucial. A collaborative and multidisciplinary approach, such as nurses, pharmacists, physical therapists, and nutritionists, is essential for improving patient outcomes.

Department and Institution Where Work Was Done

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Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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