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Management of Acute Hypercapnic Respiratory Failure (AHRF) in Patients with Obesity Hypoventilation Syndrome (OHS) in the Intensive Care Unit (ICU)

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ABSTRACT

Introduction: Acute hypercapnic respiratory failure (AHRF) in patients with obesity hypoventilation syndrome (OHS) presents significant challenges in the intensive care unit (ICU), particularly when complicated by comorbidities such as community-acquired pneumonia and heart failure. Effective management requires a tailored approach addressing altered lung mechanics, infection control, and fluid balance. This case report highlights the multidisciplinary management of AHRF in a complex clinical scenario.

Case Description: A 35-year-old female with OHS presented with progressive dyspnea for one month, worsening over the last two days. Initial assessment revealed type II respiratory failure with a pCO2 of 89 mmHg. Management included intubation and mechanical ventilation, initially with pressure control-assist control (PC-AC) mode, gradually transitioned to pressure support ventilation (PSV). Empirical antibiotics were administered, later adjusted based on sputum culture results. Fluid management involved furosemide to address concurrent heart failure. Adequate positive end-expiratory pressure (PEEP) was crucial to optimize lung mechanics. Despite an initial failed extubation, the patient was successfully extubated on day 7 with high-flow nasal cannula support and subsequently transferred to a step-down unit with nasal cannula oxygen therapy.

Conclusion: This case underscores the importance of a multidisciplinary approach and dynamic therapy adjustments based on clinical response in managing AHRF in OHS patients with complex comorbidities. Early broad-spectrum antibiotics, careful fluid management, and gradual weaning from mechanical ventilation are critical for successful outcomes.

Keywords

Acute Hypercapnic Respiratory Failure, Obesity Hypoventilation Syndrome, Community-Acquired Pneumonia

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INTRODUCTION

Obesity remains a significant public health concern in Indonesia, with a reported prevalence of 20.53% in 2018 [1]. This condition increases the risk of chronic diseases, including cardiovascular and respiratory disorders [2]. Obesity Hypoventilation Syndrome (OHS), also known as Pickwickian syndrome, is characterised by obesity (BMI ≥ 30 kg/m²), diurnal hypercapnia (PaCO2 ≥ 45 mmHg), and sleep-related breathing disorders after excluding other causes such as pulmonary disease, chest wall deformities, hypothyroidism, or neuromuscular disorders [3]. The prevalence of OHS ranges from 0.15-0.31% in the general adult population and 19-31% among obese individuals, with rising global obesity trends suggesting an increasing burden [7,8]. Community-acquired pneumonia (CAP), defined as a parenchymal lung infection acquired outside healthcare settings, has seen its prevalence in Indonesia rise from 1.6% in 2013 to 2.0% in

2018 [1]. CAP is associated with high morbidity and mortality, particularly in the elderly populations [4]. Heart failure is a complex clinical syndrome resulting from structural or functional impairments in ventricular filling or ejection, further complicating respiratory management [6]. CAP can exacerbate lung function, increasing the risk of respiratory failure, while congestive heart failure disrupts optimal pulmonary circulation and worsens respiratory status [5]. Additionally, obesity increases cardiac and pulmonary workload and elevates the risk of obstructive sleep apnoea, all of which may contribute to acute hypercapnic respiratory failure (AHRF) in patients with OHS [3]. This case highlights the need for tailored interventions for such complex scenarios.

CASE DESCRIPTION

A 35-year-old female patient was diagnosed with Ny. H (medical record number 0002309xxx) presented to the emergency department with the chief complaint of dyspnoea. The patient, weighing 100 kg with a height of 160 cm (BMI 39 kg/m²), reported progressive dyspnea for two days prior to admission, accompanied by productive cough with yellowish sputum and fever. She reported relief in a semi-sitting position and had a history of sleeping with three pillows. Oedema in both legs was intermittent for two months, whereas dyspnoea on exertion persisted for six months. She also reported excessive daytime sleepiness and frequent nocturnal awakenings due to shortness of breath over the past two years. Her medical history included 9-day hospitalization 1.5 months prior to pneumonia and cardiac enlargement. The current medications included furosemide 40 mg daily, sildenafil 200 mg twice daily, and spironolactone 25 mg daily. Upon arrival, she was referred from the emergency department to the ICU due to declining consciousness and acute hypercapnic respiratory failure (AHRF), with initial arterial blood gas showing pH 7.316, pCO2 89.1 mmHg, pO2 173.7 mmHg, HCO3 46.1 mmol/L, BE 17.2, and SaO2 95.9%.

Table 1. Summary of Ventilation Parameters During ICU Stay Day

Day	Ventilation Mode	FiO_2 (%)	PEEP (cmH_2O)	PS (cmH_2O)	RR (bpm)	SpO_2 (%)	$EtCO_2$ (mmHg)	pCO_2 (mmHg)	pO_2 (mmHg)
1	PC-AC	80	7	–	18	95–100	45–51	89.1	173.7
2	PC-SIMV	85	8	20	24	91–98	38–51	66.2	89.1
3	PSV	60	6	20	16–22	96–100	41–48	61.7	130.4
4	PSV	60	6	18	16–22	100	36–45	63.5	119
5	PSV	50	5	12	18–22	100	37–48	47.6	159
6	PSV (pre-extubation)	45	5	5	22	97	45	52.3	99.2
7	HFNC	60	–	–	18–22	100	–	50.5	112
8	NRM	–	–	–	18–22	100	–	54.1	166
9	Nasal Cannula	–	–	–	18–22	95–96	–	–	–

Note: BP, Blood Pressure (systolic/diastolic); HR, Heart Rate; RR, Respiratory Rate; PS = Pressure Support; HFNC. High-Flow Nasal Cannula; NRM, Non-Rebreather Mask.

On ICU admission, the sedated and controlled patient exhibited vital signs, including systolic blood pressure of 145-200 mmHg and diastolic of 95-140 mmHg without vasopressors, heart rate of 98-114 beats per minute, and EtCO2 of 45-51 mmHg. Oxygen saturation was maintained at 95-100% on pressure control-assist control (PC-AC) ventilation (Pinsp 26, PEEP 7, rate 18/min, FiO2 80%), with a peak pressure of 26-27 cmH2O and tidal volume of 318-375 mL. Physical examination revealed regular heart sounds without murmurs and bilateral fine basal rales on lung auscultation with no wheezing. Initial laboratory results showed a hemoglobin of 9.0 g/dL, leukocyte count of 13,140/μL, and normal liver, renal, and electrolyte profiles. Arterial blood gas post-stabilisation indicated a pH of 7.423, pCO2 of 35.2 mmHg, and pO2 128.9 mmHg. Chest radiography revealed bilateral bronchopneumonia and cardiomegaly, while electrocardiogram showed sinus tachycardia. Prior imaging (1.5 months ago) included ultrasound showing minimal ascites and hepatomegaly, and echocardiography confirming pulmonary hypertension with dilated right atrium and ventricle, diastolic dysfunction, and ejection fraction of 76%.The management commenced with sedation using midazolam (3 mg/h) and rocuronium (30 mg/h), combined antibiotics (ceftazidime 2 g thrice daily and

levofloxacin 750 mg once daily), and furosemide 40 mg/24 h for fluid management. The head was elevated at 45-60 degrees, with omeprazole 40 mg twice daily and paracetamol 1 g every 6 h. Over nine days in the ICU, ventilation was adjusted from PC-AC to pressure support ventilation (PSV), with FiO2 reduced as tolerated. Sputum culture on day 6 identified Acinetobacter baumannii, which was resistant to ceftazidime and meropenem, but sensitive to amikacin and ampicillin-sulbactam, prompting antibiotic adjustment. The patient experienced failed extubation on day 6 due to breathing and desaturation, requiring reintubation, but was successfully extubated on day 7 with high-flow nasal cannula support. She was transferred to the step-down unit on day 9 with nasal cannula oxygen at 3 L/min, haemodynamics stabilised and dyspnoea resolved.

Table 2. Summary of Hemodynamic Parameters During ICU Stay Day

Day	Ventilation Mode	Blood Pressure (mmHg)	Heart Rate (bpm)
1	PC-AC	145–200 / 95–140	98–114
2	PC-SIMV	95–170 / 60–105	70–130
3	PSV	105–160 / 60–110	60–95
4	PSV	110–160 / 75–110	60–75
5	PSV	125–150 / 65–100	60–90
6	PSV (pre-extubation)	130–155 / 75–85	60–75
7	HFNC	125–175 / 75–110	60–80
8	NRM	122–172 / 71–109	98–108
9	Nasal Cannula	125–164 / 76–111	102

Note: BP, Blood Pressure (systolic/diastolic); HR, Heart Rate; bpm, beats per minute.

DISCUSSION

Obesity significantly predisposes patients to acute respiratory failure, with its severity escalating with increasing body mass index (BMI). In this case, the patient's BMI of 39 kg/m² classified her as having severe obesity, correlating with heightened airway resistance, reduced lung volume, and increased respiratory workload [6, 9]. These physiological changes, including decreased chest wall and lung compliance due to excess adipose tissue, lead to reduced functional residual capacity (FRC) and expiratory reserve volume (ERV), contributing to gas trapping and hypoxaemia [9]. The obesity as both a direct cause and risk factor for respiratory complications, a finding supported by its association with chronic microatelectasis and altered ventilatory mechanics [6].

Obesity hypoventilation syndrome (OHS) in this patient, evidenced by a BMI of 39 kg/m² and pCO2 of 89.1 mmHg, is compounded by obstructive sleep apnoea (OSA) and comorbidities, such as heart failure and community-acquired pneumonia (CAP) [6-9]. The multifactorial pathogenesis of OHS, including increased respiratory load, diminished central drive, and leptin resistance, exacerbates hypercapnia, with untreated cases linked to a 23% mortality rate within 18 months [7]. Acute hypercapnic respiratory failure (AHRF) likely results from chronic alveolar hypoventilation worsened by infection and cardiac dysfunction, necessitating invasive mechanical ventilation [10]. Initial management with the pressure control-assist control (PC-AC) mode aligned with recommendations for obese patients, targeting low tidal volumes and adequate PEEP to optimise compliance [1,2].

CAP, confirmed by radiographic infiltrates and clinical symptoms, contributed to the patient's respiratory decline, with early broad-spectrum antibiotics (ceftazidime and levofloxacin) adhering to the Infectious Disease Society of America (IDSA) guidelines [4]. However, subsequent Acinetobacter baumannii resistance highlights the need for culture-guided adjustments, underscoring the importance of dynamic antibiotic stewardship [4]. The role of corticosteroids remains debated, with some evidence suggesting reduced treatment failure, although not a mortality benefit [4].

Congestive heart failure (CHF), driven by pulmonary hypertension (PH) and chronic hypoxaemia, further complicates the case, with furosemide and vasodilators effectively managing fluid overload [6, 10]. The successful transition to high-flow nasal cannula (HFNC) post-extubation on day 7 reflects a tailored ventilatory strategy, although the initial extubation failure emphasises the need for meticulous readiness assessment [1,2]. Nutritional support initiated early with enteral feeding addressed catabolic stress, aligning

with ASPEN/SCCM guidelines for critically ill obese patients [3]. Overall, this case underscores the necessity of a multidisciplinary, adaptive approach to managing AHRF in OHS with complex comorbidities.

CONCLUSION

The management of acute hypercapnic respiratory failure (AHRF) in patients with obesity hypoventilation syndrome (OHS) necessitates a comprehensive strategy that incorporates noninvasive ventilation or invasive mechanical ventilation with low tidal volumes, moderate to high positive end-expiratory pressure (PEEP), and recruitment manoeuvres tailored to the patient's clinical status, with the primary goals of reducing respiratory muscle workload, correcting acidosis, alleviating hypercapnia, and improving hypoxia. Effective treatment of concomitant conditions such as community-acquired pneumonia and heart failure is critical to support patient recovery. Long-term weight loss achievable through structured dietary interventions or bariatric surgery is the most effective approach for mitigating OHS symptoms and facilitating successful weaning from mechanical ventilation, underscoring the need for a holistic, individualised treatment plan.

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None

CONSENT FOR PUBLICATION

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The authors declare no conflicts of interest in this case report.

AUTHORS' CONTRIBUTIONS

All authors made substantial contributions to the case report. DFN was responsible for patient management, data collection, and the initial drafting of the manuscript. All authors reviewed and approved the final version of the manuscript, ensuring its accuracy and integrity and being accountable for all aspects of the work.

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