

SUCCESSFUL EXTUBATION USING HFNC IN A MORBIDLY OBESE PATIENT WITH CHF AND PNEUMONIA: A CASE REPORT

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ABSTRACT

Background :Successful extubation strategies from mechanical ventilation in morbidly obese patients is challenging. Reduced functional residual capacity, decreased thoracic compliance, and increased intra-abdominal pressure predispose to atelectasis and impaired gas exchange. Elevated airway resistance raises work of breathing, while blunted ventilatory responses to hypercapnia and hypoxia worsen ventilatory load. Higher oxygen consumption and carbon dioxide production further increase demands. Comorbidities such as congestive heart failure (CHF) and pneumonia aggravate this condition by worsening pulmonary congestion, limiting cardiac reserve, and increasing ventilation-perfusion mismatch, thus heightening the risk of extubation failure in ICU patients.

Case : A 29-year-old female (BMI 57.7 kg/m²) with severe TR/MR-related CHF, AKI, hyperkalemia, and pneumonia underwent prolonged ventilation with gradual weaning. Echocardiography revealed RA/RV dilatation, severe TR, RV dysfunction (TAPSE 13 mm), pulmonary hypertension probability, and preserved LV function. A follow-up confirmed grade I diastolic dysfunction and mild pericardial effusion. This patient had persistent hypercapnia (pCO₂ 65–80 mmHg) from admission. During mechanical ventilation (Days 1–7), oxygenation (pO₂) was maintained with high FiO₂. Prior to weaning (Days 10–13), pCO₂ rose above 100 mmHg, indicating increased respiratory load. Serial chest X-rays showed gradual improvement of pulmonary infiltrates throughout ICU care. The serial pH also remains stable. After extubation with HFNC on Day 14, pCO₂ gradually decreased to 65–70 mmHg while pO₂ stabilized within 90–100 mmHg. Even after transition to NRM (Days 17–20), oxygenation remained adequate without reintubation.

Discussion : Although NIV is effective in reducing reintubation, HFNC was successfully used in this morbidly obese patient with CHF and pneumonia, maintaining stable oxygenation and controlled hypercapnia throughout the ICU stay

Conclusion : This case highlights that HFNC may be a feasible alternative to NIV. HFNC provided adequate gas exchange, improved comfort, and reduced the work of breathing, all of which supported gradual recovery without respiratory distress.

1. Introduction

Successful extubation remains a major challenge in the management of patients in the intensive care unit (ICU), particularly among those with complex comorbidities. Morbid obesity is recognized as a major risk factor for extubation failure due to impaired respiratory mechanics, increased work of breathing, and altered gas exchange. This condition becomes even more complicated when accompanied by advanced cardiac disease, such as congestive heart failure (CHF) with severe tricuspid regurgitation (TR) and severe mitral regurgitation (MR), as well as concomitant pneumonia.¹⁻³

Patients with morbid obesity undergo profound alterations in respiratory physiology. Reduced functional residual capacity (FRC) and decreased chest wall compliance, combined with increased intra-abdominal pressure, predispose them to atelectasis and impaired gas exchange. Airway resistance is increased, further aggravating the work of breathing and raising the risk of extubation failure. In addition, ventilatory responses to hypercapnia and hypoxemia are blunted, particularly in obesity hypoventilation syndrome. Metabolic demands are also elevated, with higher oxygen consumption and greater carbon dioxide production, thereby imposing an additional respiratory burden. These pathophysiological changes, compounded by comorbidities such as obstructive sleep apnea and cardiac dysfunction, significantly increase the likelihood of extubation failure.

The presence of congestive heart failure (CHF) and pneumonia further exacerbates the vulnerability of morbidly obese patients in the ICU. CHF leads to pulmonary congestion, reduced cardiac output, and decreased lung compliance, all of which worsen oxygenation and increase the risk of post-extubation fluid overload. Meanwhile, pneumonia adds further burden by causing ventilation-perfusion mismatch, inflammatory lung injury, and increased metabolic demands. The combination of CHF and pneumonia in morbidly obese patients creates a synergistic effect that significantly increases the risk of extubation failure, prolongs ICU stay, and complicates clinical decision-making.^{5,6,8}

Several epidemiological studies have identified obesity as an important risk factor for extubation failure. For example, patients with a body mass index (BMI) greater than 30 kg/m² have been shown to experience higher rates of extubation failure and longer ICU stays compared with non-obese patients. In the context of complex critical illness, obesity has also been associated with increased extubation failure rates (70.4% vs. 47.4%) and greater ventilatory burden, including higher mechanical power and ventilatory ratio. Pathophysiological factors such as reduced lung compliance, premature airway closure, and sleep-disordered breathing further contribute to this vulnerability.^{1,4,5}

In patients with morbid obesity complicated by heart failure (CHF) and pneumonia, strategies to improve extubation success include the use of high-flow nasal cannula (HFNC) or low-pressure support ventilation to better reflect spontaneous breathing effort, individualized optimization of positive end-expiratory pressure (PEEP) to maintain alveolar recruitment, and comparison with post-extubation non-invasive ventilation (NIV) to prevent recurrent respiratory failure. However, substantial variation in clinical practice remains regarding the optimal approach, given the complexity of these patients conditions and the limited available scientific evidence.⁵⁻⁸

This case reports aims to analyze strategies that have been reported to improve extubation success in ICU patients with morbid obesity, congestive heart failure (CHF) accompanied by severe tricuspid regurgitation (TR) and severe mitral regurgitation (MR), as well as pneumonia. This case

report is expected to contribute to more targeted clinical practice in managing these complex scenarios.

2. Case Report

A 29-year-old woman, Mrs. FDU, was admitted to the General Intensive Care Unit (GICU) with respiratory failure requiring mechanical ventilation. Her past medical history included morbid obesity (BMI 57.7 kg/m²), chronic congestive heart failure (CHF) secondary to severe tricuspid regurgitation (TR) and severe mitral regurgitation (MR), stage I acute kidney injury (AKI), hyperkalemia (K⁺ 5.8 mEq/L), and pneumonia. She had no history of hypertension, diabetes, asthma, allergies, or previous surgery.

The patient reported progressive shortness of breath over the past month, accompanied by productive cough and orthopnea requiring at least two pillows for sleep. Her daily activities had become severely limited, and she had been mostly bedridden for the past month. She also reported loud snoring for the past four years and excessive daytime sleepiness for the last two years.

An echocardiographic examination performed on June 22, 2025, at RSUD H. Abdul Manap, Jambi, revealed dilatation of the right atrium (RA) and right ventricle (RV), reduced RV systolic function (TAPSE 13 mm), severe TR, mild MR/PR, possible pulmonary hypertension, with preserved left ventricular (LV) systolic function (LVEF 69%). A repeat echocardiography on July 11, 2025, demonstrated normal LV systolic function (LVEF 64.7%), grade I diastolic dysfunction, persistent RA/RV dilatation, mild pericardial effusion (0.8 cm) without tamponade, and suspicion of atrial septal defect (ASD) with pulmonary hypertension.

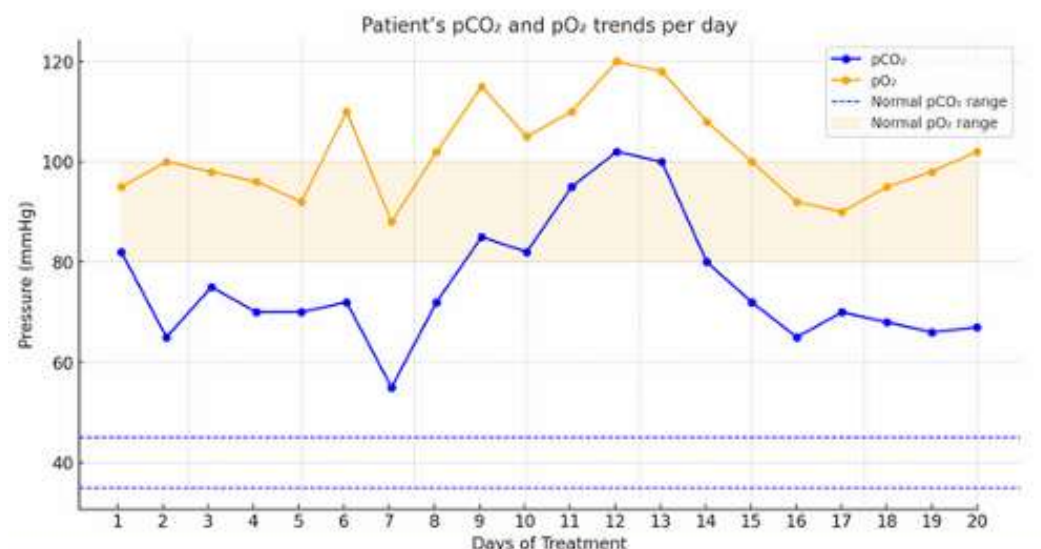
On admission, vital signs showed blood pressure of 170/89 mmHg, heart rate of 71 bpm, respiratory rate of 28 breaths/minute, and oxygen saturation of 75% while receiving oxygen via non-rebreather mask (NRM) at 15 L/minute. Physical examination revealed bilateral lower limb pitting edema with warm extremities. Laboratory investigations demonstrated hypercapnic respiratory acidosis (pH 7.281; pCO₂ 80.4 mmHg; PO₂ 73.2 mmHg; HCO₃⁻ 33.1 mmol/L), mild renal impairment (urea 54 mg/dL; creatinine 1.26 mg/dL), and elevated lactate (3.2 mmol/L). Chest radiography showed cardiomegaly with signs of pulmonary congestion.

Extubation was delayed until Day 14 because the patient initially demonstrated persistent severe hypercapnia, high oxygen requirements, active pulmonary infection, and ongoing volume overload related to CHF. Over time, gradual improvement in oxygenation, radiological findings, hemodynamic stability, consciousness level, and fluid status suggested recovery of both pulmonary and cardiovascular function. Daily fluid balance became progressively negative during the days preceding extubation (Day 11: -500 mL, Day 12: -850 mL, Day 13: -1200 mL), accompanied by improvement in peripheral edema and pulmonary congestion. These improvements provided a safer condition for extubation despite persistent chronic hypercapnia.

Echocardiographic evaluation was important in the extubation decision because cardiovascular dysfunction is a recognized cause of weaning and extubation failure. Although severe TR, RV dilatation, and pulmonary hypertension remained present, the patient demonstrated preserved LV systolic function, no evidence of tamponade physiology, stable hemodynamics without inotropic support, and improving volume status. These findings suggested sufficient cardiovascular reserve to tolerate the increased preload and afterload changes associated with spontaneous breathing after extubation.

Table 1. Summary of Patient Management

Hospital Day	Clinical Events
Day 1	Patient admitted to ICU with GCS E4M6V5, BP 125/45 mmHg, HR 98 bpm, RR 25 x/min, SpO ₂ 77% on HFNC flow 60 L/min FiO ₂ 95% (ROX 3.24). ABG showed severe hypoxemia (PO ₂ /FiO ₂ 58). Initial therapy: IV moxifloxacin 400 mg/24 h (Day 1), digoxin 0.25 mg/24 h, ramipril 2.5 mg/24 h, sildenafil 7.5 mg every 8 h, spironolactone 25 mg/24 h, simarc 2 mg/24 h via NGT, IV dobutamine 5 mcg/kg/min, IV furosemide, IV hydrocortisone 100 mg every 12 h.
Day 2	Decreased consciousness to GCS E3M6VT. Patient intubated and placed on ventilator PSIMV mode: rate 20, pressure support 26 cmH ₂ O, PEEP 8 cmH ₂ O, FiO ₂ 80%. SpO ₂ 98%, tidal volume 338–550 mL. Continued previous therapy and added IV dexmedetomidine.
Day 3	GCS E3M5VT, BP 155/77 mmHg, frequent PVC bigeminy observed. Dobutamine reduced to 3 mcg/kg/min.
Day 4	Stable condition, ventilator FiO ₂ reduced to 70%.
Day 5	Inotropic support discontinued; FiO ₂ increased back to 80% due to desaturation.
Day 6	GCS improved to E4M5T, hemodynamically stable, FiO ₂ maintained at 80%.
Day 7	FiO ₂ reduced to 60%. Based on sputum culture results, antibiotics changed to IV tigecycline 100 mg every 12 h (Day 1).
Day 8	FiO ₂ further reduced to 50%, respiratory condition improved.
Day 9	FiO ₂ increased again to 80% to maintain oxygen saturation.
Day 10	FiO ₂ reduced to 70%, stable condition.
Day 11	FiO ₂ 60%, condition remained stable.
Day 12	Minor ventilator adjustments, FiO ₂ maintained at 60%, hemodynamically stable.
Day 13	Weaning initiated to ASV mode 100%, PEEP 9, FiO ₂ 60%, SpO ₂ 94%. RR 22x/min, Tidal Volume 322–350 ml with RSBI 68 breaths/min/L. Additional therapy: IV NAC 5 g/24 h, sildenafil 20 mg every 8 h via NGT, hydrocortisone replaced with IV dexamethasone 10 mg every 8 h, nebulized Pulmicort 1 vial every 8 h.
Day 14	A spontaneous breathing trial (SBT) was performed before extubation using pressure support ventilation for 120 minutes. The patient tolerated the trial without tachypnea, desaturation, hemodynamic instability, or increased work of breathing. Respiratory muscle strength parameters such as MIP/NIF were not available, representing a limitation in the assessment of extubation readiness. Patient extubated and transitioned to HFNC flow 60 L/min FiO ₂ 80% (ROX 6.8), SpO ₂ 94%.
Day 15	HFNC flow 50 L/min FiO ₂ 70% (ROX 7.8), SpO ₂ 94%.
Day 16	HFNC flow 40 L/min FiO ₂ 60% (ROX 9.4), SpO ₂ 96%.
Day 17	HFNC discontinued and switched to NRM 12 L/min, SpO ₂ 95%.
Days 18–20	NRM 10 L/min, SpO ₂ 91–96%, hemodynamically stable. On Day 20, transfer to CVCU was planned if stability was maintained.



3. Discussion

Non-invasive ventilation (NIV) is effective in reducing early reintubation rates in obese patients after extubation, particularly within the first three days. Several included studies demonstrated that the alternating use of NIV with high-flow nasal cannula (HFNC) after extubation significantly reduced reintubation rates and ICU mortality in obese or overweight patients at high risk of extubation failure. However, other studies have also shown that HFNC alone may provide comparable effectiveness in preventing reintubation, particularly in patients with a body mass index (BMI) ≥ 40 kg/m², while also offering advantages in patient comfort and ease of use.

Oxygen therapy strategies—particularly HFNC and NIV—play an important role in reducing adverse post-extubation outcomes. In this patient, who had morbid obesity complicated by congestive heart failure (CHF) and pneumonia, the choice of HFNC as the primary post-extubation oxygen therapy strategy is consistent, especially given the high risk of extubation failure associated with these comorbidities. CHF further increases patient vulnerability through its contribution to pulmonary congestion and limited cardiac reserve, while pneumonia exacerbates hypoxemia and pulmonary inflammation, cumulatively impairing gas exchange. Despite this additional burden, HFNC was able to provide stable oxygenation and effective CO₂ elimination, underscoring its value in such complex clinical scenarios.

Although prophylactic NIV has been shown to reduce reintubation rates in obese patients, HFNC was selected as the initial post-extubation support in this case because the patient was fully conscious, cooperative, hemodynamically stable, and showed no signs of respiratory distress. HFNC was also considered more tolerable and facilitated secretion clearance, oral intake, and continuous communication. Furthermore, despite chronic hypercapnia, the patient maintained a stable pH, suggesting compensated chronic ventilatory impairment rather than acute ventilatory failure. NIV was reserved as a rescue strategy in case of worsening hypercapnia, increased work of breathing, or deterioration of gas exchange.

HFNC was selected in this case because it offers multiple physiological and clinical benefits beyond simple oxygen delivery. Physiologically, HFNC increases end-expiratory lung volume, reduces dead space, lowers respiratory rate, and decreases the work of breathing. Clinically, HFNC provides effective oxygenation in severe hypoxemia, improves patient comfort, allows oral intake, and reduces the risk of patient self-induced lung injury (P-SILI). These advantages are particularly important in morbidly obese patients with CHF and pneumonia, in whom reducing respiratory

burden and preventing further lung injury are crucial. NIV remains an important backup option, especially if worsening hypercapnia, hypoxemia, or signs of respiratory muscle fatigue develop.

Several key aspects are particularly relevant to this case:

1. Obesity-related comorbidities affecting extubation success – Factors such as CHF, reduced maximal inspiratory pressure (MIP), and increased respiratory load increase the risk of extubation failure.
2. Oxygen therapy strategies – HFNC may serve as the primary post-extubation oxygen therapy, with NIV as an adjunctive or rescue option when needed.
3. Impact of oxygen therapy on reintubation and ICU length of stay – HFNC and NIV can reduce reintubation rates in obese patients, although some studies indicate that HFNC may be associated with longer ICU stays.

In this case, the decision to extubate directly to HFNC was also supported by favorable clinical conditions, including negative fluid balance, full consciousness, reduced work of breathing (WOB), absence of dyspnea, and normal pH. HFNC was further justified by its physiological and clinical benefits, including increased end-expiratory lung volume, reduced respiratory workload, improved comfort, and decreased risk of patient self-induced lung injury. Interpretation of the patient's $p\text{CO}_2$ and $p\text{O}_2$ trends also supports the appropriateness of HFNC use: oxygenation remained stable after extubation, and chronic hypercapnia was well controlled without the need for reintubation. Nevertheless, close monitoring remains essential, and early transition to NIV should be considered if clinical deterioration occurs.

The successful use of HFNC in this patient was likely multifactorial. First, despite persistent chronic hypercapnia, the patient demonstrated several favorable conditions prior to extubation, including preserved consciousness, stable hemodynamics without inotropic support, improvement of pulmonary infiltrates, negative fluid balance, and absence of respiratory distress. These factors reduced the likelihood of immediate post-extubation respiratory failure.

Second, HFNC provided several physiological benefits that were particularly relevant in this morbidly obese patient. Obesity is associated with reduced functional residual capacity (FRC), increased airway closure, and elevated work of breathing. The high flow delivered by HFNC generates a low level of positive airway pressure, promotes alveolar recruitment, increases end-expiratory lung volume, and counteracts obesity-related atelectasis. In addition, continuous washout of nasopharyngeal dead space improves ventilatory efficiency and may facilitate carbon dioxide clearance.

Third, the patient's hypercapnia appeared to represent chronic compensated ventilatory impairment rather than acute respiratory failure. Although PCO_2 remained elevated throughout the ICU stay, arterial pH remained relatively stable, suggesting chronic adaptation. Following extubation, PCO_2 gradually decreased while oxygenation remained adequate, indicating that spontaneous ventilation supported by HFNC was sufficient to meet the patient's respiratory demands.

Cardiovascular optimization also likely contributed to extubation success. Improvement in fluid status and pulmonary congestion reduced the cardiopulmonary burden associated with spontaneous breathing. Although echocardiography demonstrated severe tricuspid regurgitation, right ventricular dilatation, and pulmonary hypertension, left ventricular systolic function remained preserved and no progressive hemodynamic instability was observed. These findings suggest that the patient possessed sufficient cardiovascular reserve to tolerate the transition from positive-pressure ventilation to spontaneous breathing.

Finally, HFNC may have improved patient comfort compared with NIV. Better tolerance, uninterrupted communication, easier secretion clearance, and avoidance of mask-related discomfort may have enhanced adherence to therapy during the critical post-extubation period. Collectively, these physiological and clinical factors likely explain why HFNC was successful in preventing reintubation in this high-risk patient.

4. Conclusion

NIV may reduce the risk of early reintubation in obese patients. However, in this case, HFNC was still used as the primary oxygen therapy strategy, as it was able to provide adequate oxygenation and maintain post-extubation stability in the ICU. The patient was successfully extubated using HFNC, subsequently transitioned to a non-rebreather mask (NRM), and did not require reintubation.

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