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Near-Fatal Asthma With Severe Hypercapnic Respiratory Failure Requiring Mechanical Ventilation In A Resource-Limited Setting: A Case Report

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Abstract

Near-fatal asthma (NFA) is a life-threatening manifestation of acute severe asthma characterized by hypercapnia, respiratory acidosis, altered consciousness, and the need for invasive mechanical ventilation. Early recognition and timely airway stabilization are essential to prevent respiratory arrest and mortality, particularly in resource-limited settings. A 41-year-old woman with a history of asthma was brought to the emergency department with decreased consciousness and severe respiratory distress. On arrival, her Glasgow Coma Scale score was 5, respiratory rate was 35 breaths per minute, and oxygen saturation was 46% on room air. Arterial blood gas analysis demonstrated life-threatening hypercapnic respiratory failure (pH 6.954; $PaCO_2$ 95.4 mmHg; P/F ratio 87). Due to worsening respiratory failure and decreased level of consciousness, the patient underwent immediate endotracheal intubation and invasive mechanical ventilation and was admitted to the intensive care unit. She received systemic corticosteroids, repeated bronchodilator therapy, aminophylline infusion, and broad-spectrum antibiotics. Lung-protective ventilatory strategies with prolonged expiratory time were applied. The patient improved rapidly and was successfully extubated on the second day of mechanical ventilation, with subsequent discharge in stable condition. Appropriate ventilatory strategies combined with guideline-based pharmacological therapy are essential to improve outcomes in patients with near-fatal asthma presenting with severe hypercapnic respiratory failure, even in resource-limited settings.

Keywords: Near-Fatal Asthma; Severe Asthma Exacerbation; Hypercapnic Respiratory Failure; Invasive Mechanical Ventilation.

INTRODUCTION

Asthma is a chronic and complex respiratory disorder characterized by persistent airway inflammation that leads to recurrent symptoms such as wheezing, shortness of breath, chest tightness, and coughing. This inflammatory process results in reversible airflow limitation and increased bronchial responsiveness to triggers such as allergens, environmental irritants, and respiratory infections (Reza et al., 2025).

Asthma exacerbations (AEs), especially severe episodes, remain a major global health concern because they contribute to increased morbidity, mortality, healthcare costs, and reduced quality of life among affected individuals. According to the Global Initiative for Asthma (GINA), approximately 8 to 13% of patients experience at least one exacerbation each year. Severe asthma exacerbations (SAEs) are associated with a higher risk of death, with asthma causing about 1,000 deaths worldwide every day. A history of previous exacerbations, particularly two or more in the past year, is also a strong predictor of future exacerbation risk (Jackson et al., 2025; Paredes et al., 2026; Global Initiative for Asthma, 2025).

Severe asthma exacerbations may progress to life-threatening conditions such as acute hypercapnic respiratory failure, altered consciousness, and the need for endotracheal intubation with mechanical ventilation. Near-fatal asthma represents an extreme clinical presentation marked by severe acidemia, carbon dioxide retention, and critical impairment of ventilation, requiring prompt recognition and aggressive management to prevent mortality. Early airway stabilization and appropriate intensive care support play an essential role in improving outcomes in these patients (D'amato et al., 2016). Here, we report a case of near-fatal asthma presenting with decreased

consciousness and severe hypercapnic respiratory failure requiring mechanical ventilation, highlighting the importance of rapid assessment and timely intervention in the emergency and intensive care settings.

RESULTS AND DISCUSSION

A 41-year-old woman was brought to the Emergency Department (ED) with decreased consciousness accompanied by shortness of breath and rapid breathing. The patient was unable to provide her medical history at presentation. According to her family, she had been experiencing dyspnea since the previous night, approximately 10 hours prior to hospital admission. The symptoms were not accompanied by fever, chest pain, nausea, or vomiting. Before coming to the hospital, the patient had taken over-the-counter medication obtained from a pharmacy, but there was no improvement in her condition. Her past medical history was notable for asthma.

Upon arrival, her initial Glasgow Coma Scale (GCS) score was 5. He was tachypneic with a respiratory rate of 40 breaths per minute. Other vital signs included blood pressure of 138/68 mmHg, heart rate of 60 beats per minute, temperature of 36.5°C, and oxygen saturation (SpO_2) of 46% on room air. Physical examination revealed reduced air entry bilaterally with widespread wheeze and minimal rhonchi on both lung fields. The extremities were noted to be cold on palpation. Auscultation revealed reduced air entry bilaterally with widespread wheeze.

Electrocardiography examination showed normal sinus rhythm (**Figure 1**). Laboratory evaluation (**Table 1**) revealed normal hematologic parameters, platelet count, renal function, and electrolyte levels. Laboratory evaluation revealed normal hematologic parameters, platelet count, renal function, and electrolyte levels. However, leukocytosis (26.6 $\times 10^3/\mu L$) with neutrophil predominance (70%) and hypoglycemia (62 mg/dL) were observed. Arterial blood gas analysis (**Table 2**) demonstrated life-threatening hypercapnic respiratory failure (Type II) with severe acidemia (pH 6.954) and marked hypercapnia (pCO_2 95.4 mmHg), accompanied by severe impairment of oxygenation (P/F ratio 87).

A diagnosis of sepsis and respiratory failure secondary to acute severe asthma was established. Due to increased work of breathing and the patient's decreased level of consciousness, endotracheal intubation was performed for airway protection, and the patient was immediately transferred to the Intensive Care Unit (ICU) for mechanical ventilation. The patient was intubated using a 7.0 mm endotracheal tube (ETT) inserted to a depth of 18 cm following premedication with midazolam 5 mg, propofol 100 mg, fentanyl 100 mcg, and rocuronium 30 mg. Chest XR postintubation is shown in **Figure 2a**. Initial mechanical ventilation was initiated in pressure-synchronized intermittent mandatory ventilation (P-SIMV) mode with an FiO_2 of 1.0, which was later gradually reduced to 0.5. The tidal volume was set at 0.35 L, with a respiratory rate of 18 breaths per minute and an inspiration-to-expiration (I:E) ratio of 1:4. Positive end-expiratory pressure (PEEP) was set at 5 cmH_2O.

During ICU management, the patient received comprehensive treatment directed at both severe asthma exacerbation and suspected sepsis, including bronchodilator therapy, systemic corticosteroids, broad-spectrum antibiotics, and intravenous fluid resuscitation. The administered treatments included an aminophylline infusion (250 mg every 8 hours), intravenous ceftriaxone 1 g every 12 hours, intravenous lansoprazole 30 mg every 12 hours, intravenous hydrocortisone 100 mg every 8 hours, oral azithromycin 500 mg once daily, and nebulized bronchodilator therapy with a combination of ipratropium bromide (0.5 mg) and salbutamol (2.5 mg) along with budesonide every 8 hours.

During the ICU stay, the patient was closely monitored for hemodynamic and respiratory parameters, including pulse rate, oxygen saturation, breath sounds, body temperature, and urine output. On the second day of mechanical ventilation, the patient's clinical condition improved, as evidenced by an oxygen saturation of 98% on minimal ventilatory support (FiO_2 0.21), a pulse rate of 86 beats per minute, and a blood pressure of 132/68 mmHg. Serial arterial blood gas analysis demonstrated significant improvement (**Table 2**), and follow-up chest radiographic evaluation also

showed resolution of previous airway inflammatory changes without new abnormalities (**Figure 2b**). The patient showed adequate spontaneous respiratory effort, and lung auscultation revealed normal breath sounds without wheezing or rhonchi. The patient was successfully extubated without complications. Following extubation, she remained clinically stable with no signs of respiratory distress and maintained satisfactory oxygen saturation levels. On the third hospital day, the patient was transferred to the general ward, and on the sixth day of hospitalization, she was discharged in stable condition with scheduled follow-up at the internal medicine outpatient clinic

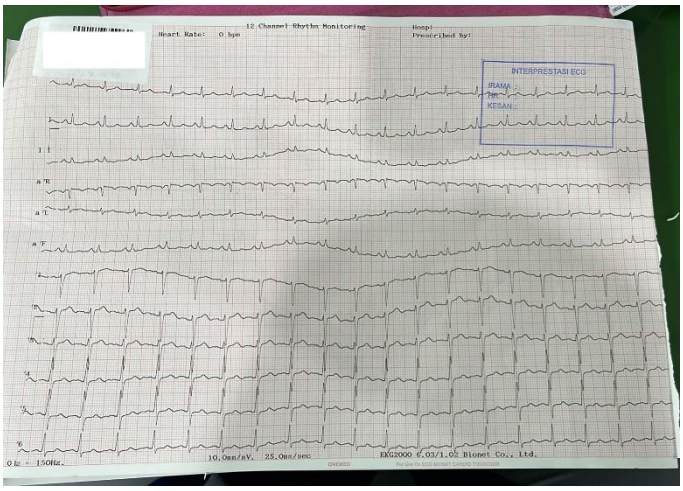

Figure 1. 12-Lead electrocardiogram on admission showed normal sinus rhythm

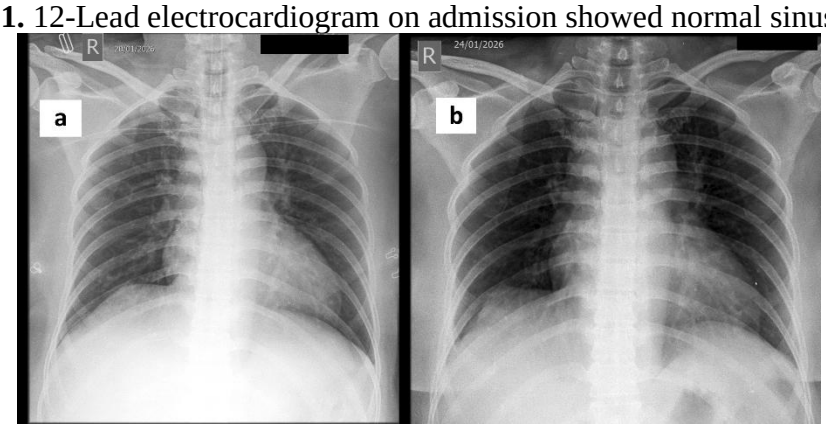

Figure 2. Serial chest radiographs: (a) Initial chest X-ray demonstrating findings suggestive of airway inflammation without evidence of cardiomegaly. An endotracheal tube (ETT) is visualized in appropriate position along the tracheal projection. (b) Follow-up chest X-ray demonstrating no significant pulmonary abnormalities.

Table 1. Laboratory findings on arrival at the emergency room (ER)

Parameters	**Results**
Hb	14.5 g/dL
Hematocryte	41.3 %
Erythrocyte	4.66 10^6/µL
Trombocyte	405 µL
Leukocyte	26.6 /mm^3
Lymphocytes	25 %
Neutrophil	70%
Monocytes	8.5%
Eosinophil	3%
Basophil	0%
Ureum	14.9 mg/dL

Creatinine	0.9 mg/dL
Blood glucose	62 mg/dL
Natrium	146.62 mmol/L
Kalium	4.3 mmol/L
Chlorida	111.46 mmol/L

Table 2. Serial Blood Gas Analysis

Parameter	**Initial et ER (20/1/2026)**	**Serial BGA (21/1/2026)**
Temperature	36.2°C	37.2°C
sO_2 (est)	84.69%	99.3%
pH	6.954	7.396
$PaCO_2$ (T)	95.4 mmHg	37.6 mmHg
pO_2 (T)	70 mmHg	170 mmHg
HCO_3^- Act	20.4 mmol/L	22.5 mmol/L
$CtCO_2$	23 mmol/L	24 mmol/L
BE (ecf)	-11.8 mmol/L	-2.3 mmol/L
pO_2 (A–a)	390 mmHg	211 mmHg
RI (T)	5.68	1.23
pO_2/FiO_2	87	283

Discussion

Despite advances in outpatient asthma management, severe asthma exacerbations remain a frequent cause of emergency presentation. Severe and life-threatening exacerbations are true medical emergencies requiring prompt evidence-based treatment (Gayen et al., 2024). All patients with asthma may experience exacerbations characterized by shortness of breath, coughing, and wheezing, along with reduced expiratory airflow that can be assessed through lung function testing. These episodes are commonly referred to as an asthma attack, acute asthma, asthmatic exacerbation, or status asthmaticus. The severity of exacerbations can range from mild to very severe. In extreme cases, they may progress to near-fatal asthma, which is associated with events such as cardiorespiratory arrest, hypercapnia (PaCO2 95.4 mmHg), acidemia (pH 6.954), the need for endotracheal intubation and mechanical ventilation, or admission to an intensive care unit (ICU). In some instances, the condition may ultimately result in fatal asthma (Mauad & Ferreira, 2025).

According to current international guidelines, including the Global Initiative for Asthma (GINA), BTS/SIGN, and ERS/ATS recommendations, the initial management of acute severe asthma should include targeted oxygen therapy to maintain oxygen saturation between 94% and 98%, repeated administration of high-dose inhaled short-acting β_2-agonists combined with anticholinergic agents, and early initiation of systemic corticosteroids. In patients with inadequate response to initial bronchodilator therapy, adjunctive treatments such as intravenous magnesium sulfate may be considered. For refractory or life-threatening exacerbations requiring intensive care support, additional therapies including intravenous aminophylline and intravenous β_2-agonists may be used selectively as rescue treatments, although their routine use remains controversial due to limited evidence of additional benefit and potential adverse effects. Early recognition of physiological deterioration and timely escalation of therapy, including mechanical ventilation when indicated, are essential to prevent progression to near-fatal asthma and improve clinical outcomes. Intubation and mechanical ventilation should be considered when there is respiratory arrest, deterioration of consciousness, respiratory muscle fatigue, or worsening acidosis and hypercapnia despite adequate treatment (Global Initiative for Asthma, 2025; Holguin et al., 2020; British Thoracic Society, 2019)

Endotracheal intubation and invasive mechanical ventilation should be considered in patients with acute severe asthma who develop respiratory arrest, worsening consciousness, respiratory muscle fatigue, or progressive respiratory acidosis with hypercapnia despite adequate treatment. Although only approximately 2% of patients with acute severe asthma require intubation, the proportion increases to 36–46% among those admitted to the intensive care unit. The decision to intubate is primarily clinical and based on signs of impending respiratory failure, including severe hypoxemia, altered sensorium, silent chest, and failure to respond to intensive therapy. Intubation should be performed using rapid sequence induction with careful hemodynamic monitoring, as complications such as bronchospasm, air trapping, and cardiovascular instability may occur during the procedure (Adan & Dirie, 2025; Stow et al., 2007; Nanchal et al., 2014).

Invasive mechanical ventilation is reserved for patients with life-threatening asthma who fail to respond to optimal medical therapy and primarily serves as supportive management while allowing time for bronchodilator therapy to take effect. The principal objective of ventilatory support in these patients is to minimize dynamic hyperinflation and air trapping, which may otherwise lead to barotrauma and hemodynamic instability. Both pressure-controlled and volume-controlled ventilation modes can be used; pressure-controlled ventilation allows better regulation of alveolar pressure but provides less precise control over tidal volume and minute ventilation, whereas volume-controlled ventilation ensures consistent tidal volume delivery at the expense of alveolar pressure control. Regardless of the selected mode, ventilator strategies should focus on reducing minute ventilation to allow adequate exhalation time and prolong the inspiratory-to-expiratory (I:E) ratio, thereby lowering alveolar pressure. Recommended approaches include the use of low tidal volumes (6–8 mL/kg ideal body weight), reduced respiratory rates (approximately 12–14 breaths per minute), prolonged expiratory time with an I:E ratio of approximately 1:4 to 1:5, and minimal external positive end-expiratory pressure due to the presence of intrinsic PEEP in obstructive airway disease. Prolongation of expiratory time helps reduce air trapping, auto-PEEP, end-expiratory lung volume, and dynamic hyperinflation. In selected cases, permissive hypercapnia may be tolerated to improve patient–ventilator synchrony and reduce ventilator-induced lung injury (Gayen et al., 2024; Talbot et al., 2024).

In the present case, the ventilator was initially set in pressure-synchronized intermittent mandatory ventilation (P-SIMV) mode with a tidal volume of 0.35 L, respiratory rate of 18 breaths per minute, PEEP of 5 cmH_2O, and an inspiratory-to-expiratory ratio of 1:4. Although conventional ventilatory strategies in acute severe asthma generally recommend lower respiratory rates (approximately 12–14 breaths per minute) to minimize dynamic hyperinflation and air trapping, a slightly higher respiratory rate was selected in this patient due to the presence of profound hypercapnic respiratory failure with severe acidemia ($PaCO_2$ 95.4 mmHg; pH 6.954) at presentation. In the setting of life-threatening hypercapnic acidemia, a modest increase in respiratory rate may be necessary to improve minute ventilation and facilitate carbon dioxide clearance, while maintaining prolonged expiratory time to avoid excessive intrinsic PEEP (Hickey et al., 2024). Importantly, the applied I:E ratio of 1:4 and lung-protective tidal volume helped minimize the risk of dynamic hyperinflation despite the higher respiratory rate. This individualized ventilatory adjustment contributed to rapid correction of hypercapnia and normalization of arterial blood gas parameters within 24 hours of mechanical ventilation.

In our patient, the presence of severe hypercapnic respiratory failure with marked acidemia and decreased level of consciousness represented clear indications for early endotracheal intubation and invasive mechanical ventilation. Prompt airway stabilization and initiation of lung-protective ventilatory support with prolonged expiratory time were essential to prevent further deterioration related to dynamic hyperinflation and respiratory arrest. Early administration of guideline-based pharmacological therapy, including systemic corticosteroids, bronchodilators, and adjunctive aminophylline, together with close physiological monitoring in the intensive care unit, contributed to rapid clinical improvement and successful extubation without complications. This case highlights the

importance of timely recognition of near-fatal asthma and early escalation to mechanical ventilation when indicated, particularly in resource-limited settings where delayed intervention may significantly worsen outcomes. However, this report has several limitations, including its single-case nature, which limits generalizability, the absence of long-term follow-up data, and limited access to advanced respiratory monitoring modalities.

CONCLUSION

Near-fatal asthma is a life-threatening condition that requires early recognition and prompt escalation of treatment, including invasive mechanical ventilation when indicated. In this case, timely endotracheal intubation and appropriate ventilatory support, combined with guideline-based pharmacological therapy, contributed to rapid clinical improvement and a favorable outcome. This report highlights that early airway stabilization should not be delayed in patients presenting with severe hypercapnic respiratory failure and decreased level of consciousness. Even in resource-limited settings, careful monitoring and timely intensive care management can result in successful recovery. Further studies are needed to better evaluate optimal ventilatory strategies and management approaches in patients with near-fatal asthma requiring mechanical ventilation.

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