

## Myasthenic crisis revealed by severe hypoxemic pneumonia and diagnosed after extubation failure: A case report

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### Abstract

**Background:** Myasthenic crisis is an acute worsening of bulbar and respiratory weakness in myasthenia gravis and may require mechanical ventilation. Initial presentation during severe pneumonia is uncommon and may represent a major diagnostic challenge in the intensive care unit.

**Case presentation:** An 18-year-old man with no relevant medical history was admitted for acute hypoxemic respiratory failure due to severe pneumonia with septic shock. After intubation and anti-infective therapy, an extubation attempt on ICU Day 3 failed because of delayed hypercapnic hypoventilation, ineffective cough, dysphagia, and diffuse weakness, with no pulmonary, cardiac, or metabolic explanation. A neuromuscular disorder was then suspected. Electromyography demonstrated a postsynaptic neuromuscular transmission defect consistent with myasthenia gravis, while cerebrospinal fluid examination was normal. Early tracheostomy was performed, followed by intravenous immunoglobulin at 0.4 g/kg/day for 5 days, with progressive respiratory recovery and subsequent decannulation.

**Conclusion:** Unexplained extubation failure in a young adult should prompt consideration of neuromuscular disease, particularly myasthenia gravis [3-5]. Early electrophysiological evaluation, avoidance of aggravating medications, rescue immunotherapy with intravenous immunoglobulin, and timely tracheostomy may facilitate ventilator weaning and functional recovery.

**Keywords:** Myasthenia Gravis; Myasthenic Crisis; Intensive Care; Extubation Failure; Pneumonia; Case Report

### 1. Introduction

Myasthenia gravis (MG) is an autoimmune disorder of the neuromuscular junction characterized by fluctuating weakness that variably affects ocular, bulbar, axial, and respiratory muscles. In most cases, the disease is associated with antibodies directed against acetylcholine receptors (AChR), whereas anti-MuSK, anti-LRP4, and seronegative forms account for the remaining presentations [1].

Myasthenic crisis is a severe acute complication defined by worsening bulbar or respiratory weakness requiring invasive or noninvasive ventilatory support [2,3]. It occurs during the course of disease in approximately 15% to 20% of patients and represents a life-threatening emergency [2,3].

Common precipitating factors include infection, especially respiratory infection, surgery, systemic stress, aggravating medications such as aminoglycosides, fluoroquinolones, and beta-blockers, as well as metabolic disturbances [2,3,6,10].

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When myasthenic crisis is the first manifestation of disease in the setting of severe infection, diagnosis may be delayed because neuromuscular symptoms are initially attributed to pneumonia, sepsis, or respiratory fatigue itself [3,6]. We report the case of previously unrecognized MG revealed by severe hypoxemic pneumonia and diagnosed after unexplained extubation failure in the ICU.

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## 2. Case Presentation

### 2.1. Patient information and initial assessment

An 18-year-old man with no significant past medical history presented to the emergency department with subacute dyspnea, fever, and productive cough that had progressed over two weeks in the context of general deterioration. On admission, respiratory rate was 32 breaths/min, peripheral oxygen saturation was 82% on room air, temperature was 39.2 C, blood pressure was 95/40 mmHg, and heart rate was 115 beats/min.

Initial clinical examination showed acute respiratory distress with tachypnea and intercostal and suprasternal retractions. The initial diagnostic impression was severe hypoxemic pneumonia, possibly occurring in an immunocompromised host, or pulmonary tuberculosis given the epidemiological context and constitutional symptoms. Frontal chest radiography showed bilateral lobar pneumonic infiltrates.

The patient was first hospitalized in an isolation area of the emergency department. He received intravenous ceftriaxone plus ciprofloxacin for 48 hours together with high-concentration oxygen therapy. A comprehensive laboratory workup, viral serology panel, and sputum testing for Mycobacterium tuberculosis were requested, with results pending at that time.

### 2.2. Clinical deterioration and ICU course

Clinical status then deteriorated abruptly, prompting transfer to the resuscitation bay. On examination, the patient had impaired consciousness with a Glasgow Coma Scale score of 6, equal and reactive pupils, severe respiratory distress with signs of exhaustion and respiratory pauses, and septic shock with blood pressure 72/30 mmHg, heart rate 145 beats/min, cold extremities, and markedly prolonged capillary refill time.

Fluid resuscitation with isotonic saline was started at 30 mL/kg over 3 hours, and norepinephrine was initiated at 0.5 microg/kg/min. Rapid-sequence intubation and sedation were performed for respiratory and neurological indications, and the patient was transferred to the intensive care unit.

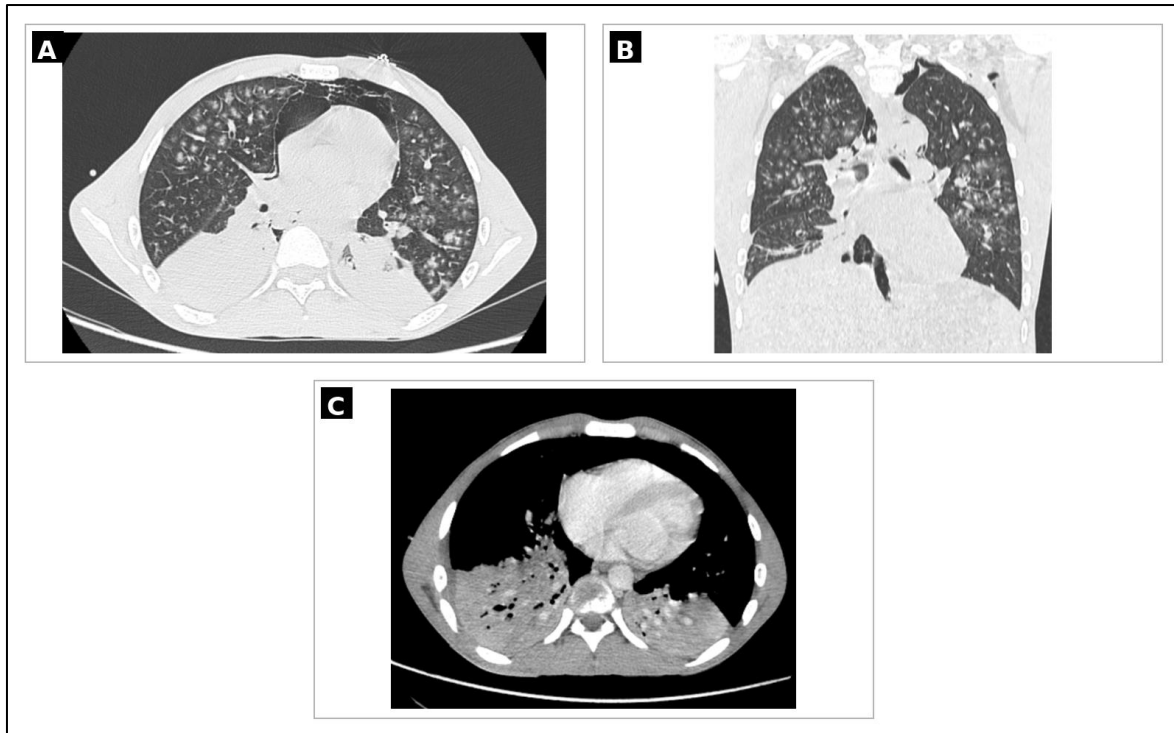
In the ICU, he underwent standard and then invasive monitoring with a right radial arterial catheter and an ultrasound-guided right internal jugular central venous catheter. Laboratory tests were repeated, including blood cultures and protected distal sampling. Antimicrobial therapy was escalated to piperacillin-tazobactam plus amikacin, and hydrocortisone hemisuccinate 50 mg every 6 hours was added as adjunctive therapy.

Initial arterial blood gas analysis showed severe hypoxemia with a PaO<sub>2</sub>/FiO<sub>2</sub> ratio of 92, major hypercapnia with a PaCO<sub>2</sub> of 85 mmHg, and hyperlactatemia of 12.4 mmol/L.

### 2.3. Imaging findings

Contrast-enhanced chest computed tomography showed the following findings (Figure 1A-C):

- Large pneumomediastinum extending into the deep soft tissues of the lower neck;
- Small left pneumothorax;
- Multiple bilateral pulmonary nodules and micronodules with centrilobular distribution associated with bilateral interlobular septal thickening;
- Bilateral lower-lobe parenchymal consolidations with air bronchograms and vascular opacification, consistent with aspiration-related infiltrates.



(A) Axial lung-window image showing diffuse bilateral centrilobular nodules/micronodules with posterior bibasal consolidations. (B) Coronal lung-window reconstruction confirming bilateral centrilobular nodular opacities and dependent lower-lobe consolidations. (C) Axial mediastinal-window image showing pneumomediastinum and a small left pneumothorax, with associated bilateral basal consolidations consistent with severe pneumonia and aspiration-related infiltrates.

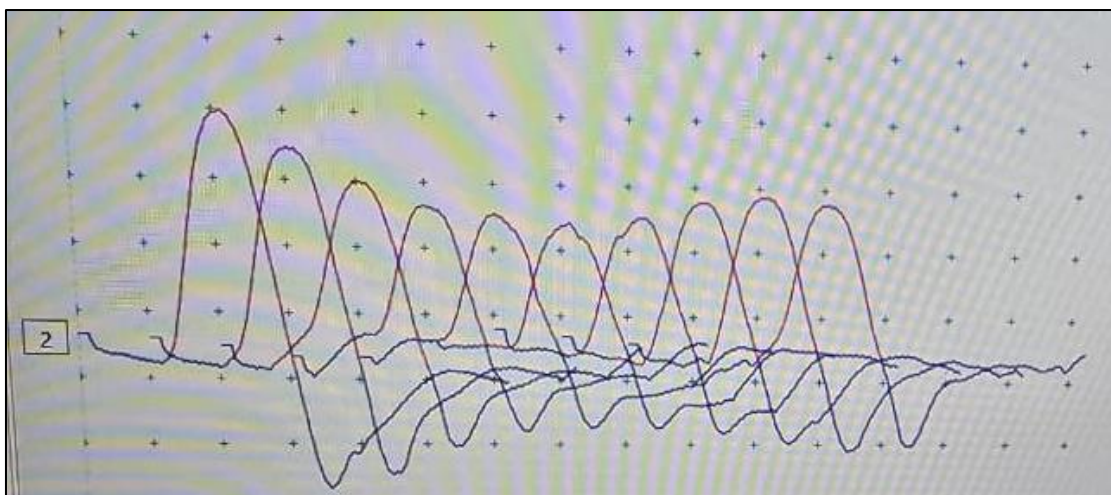
**Figure 1** Representative chest CT findings at admission

#### **2.4. Extubation failure, diagnostic workup, and treatment**

After 48 hours of mechanical ventilation and initial resuscitation, clinical, biological, and gas-exchange parameters improved. Sedation was discontinued, norepinephrine was weaned off.

On ICU Day 3, extubation was performed after complete awakening and a one-hour spontaneous breathing trial considered satisfactory. Two hours later, however, the patient developed respiratory failure with tachypnea, diaphoresis, poor thoracic expansion, ineffective cough, swallowing impairment, hypercapnia, and diffuse muscle weakness. Reintubation was required. No cardiac overload, electrolyte abnormality, or metabolic acidosis explained the extubation failure.

Early tracheostomy was performed. Given the strong suspicion of neuromuscular involvement, neurological investigations were undertaken. Electromyography showed postsynaptic block in the explored nerve-muscle pairs, with reduced motor amplitudes in both upper limbs (Figure 2). Cerebrospinal fluid examination was normal.



**Figure 2** Repetitive nerve stimulation electrophysiology showing a decremental response compatible with a postsynaptic neuromuscular transmission defect, supporting the diagnosis of myasthenia gravis

A diagnosis of inaugural myasthenic crisis revealing myasthenia gravis was made. Intravenous immunoglobulin was started at 0.4 g/kg/day for 5 days. The clinical course was favorable, with gradual recovery of respiratory strength, decannulation, initiation of pyridostigmine 60 mg three times daily, transfer to the neurology ward, and discharge home during the following week. Immunological testing subsequently showed positive anti-AChR antibodies (titer 1:20) and negative anti-MuSK antibodies.

## 2.5. Timeline

**Table 1** Timeline of the clinical course

Time point	Clinical events
Two weeks before admission	Progressive dyspnea, fever, productive cough, and general deterioration.
Emergency department admission	RR 32 breaths/min, SpO <sub>2</sub> 82% on room air, T 39.2 C, BP 95/40 mmHg, HR 115 beats/min; bilateral lobar pneumonia suspected; ceftriaxone plus ciprofloxacin and oxygen therapy started.
Clinical deterioration	Glasgow Coma Scale score 6, severe respiratory distress, septic shock (BP 72/30 mmHg, HR 145 beats/min); fluid resuscitation, norepinephrine, rapid-sequence intubation, and ICU transfer.
ICU day 0	PaO <sub>2</sub> /FiO <sub>2</sub> 92, PaCO <sub>2</sub> 85 mmHg, lactate 12.4 mmol/L; invasive monitoring; piperacillin-tazobactam plus amikacin; hydrocortisone; chest CT showing pneumomediastinum, small pneumothorax, diffuse nodules, and bilateral lower-lobe consolidations.
After 48 hours of ventilation	Clinical improvement; sedation stopped; norepinephrine discontinued; antibiotic de-escalation to ceftriaxone plus levofloxacin after positive rhinovirus PCR.
ICU day 3	Extubation after apparently successful spontaneous breathing trial.
Two hours after extubation	Delayed respiratory failure with ineffective cough, dysphagia, hypercapnia, and diffuse weakness; reintubation required.
Subsequent workup	Early tracheostomy; electromyography consistent with postsynaptic neuromuscular transmission disorder; cerebrospinal fluid normal.
Targeted treatment and follow-up	IVIg 0.4 g/kg/day for 5 days; anti-AChR positive, anti-MuSK negative; progressive respiratory recovery, decannulation, pyridostigmine initiation, neurology transfer, and discharge home.

### 3. Discussion

This case highlights several clinically important points. First, myasthenic crisis may be the inaugural presentation of MG, making diagnosis particularly difficult when initial manifestations are dominated by severe infectious respiratory failure [2,3,6]. In our patient, the absence of obvious ptosis and the impossibility of adequately assessing bulbar symptoms at presentation delayed consideration of an underlying neuromuscular disorder [3,6].

Second, respiratory infection is one of the most common triggers of myasthenic crisis [2,3,6]. In this patient, the combination of severe pneumonia, profound hypoxemia, and septic shock probably played a decisive role in unmasking MG. The respiratory pattern therefore involved both a hypoxemic parenchymal component and a hypercapnic neuromuscular component, which likely explains the marked fragility observed after extubation.

Third, unexplained extubation failure should be considered an early warning sign of myasthenic crisis. Extubation failure is common in ventilated patients with myasthenic crisis and has been associated with longer ICU stay; more recent multicenter data confirm the high frequency of difficult weaning and extubation failure, particularly in the presence of pneumonia and sepsis [4,5]. Conventional weaning criteria may be insufficient in neuromuscular disorders because they do not reliably capture delayed diaphragmatic fatigability or secondary bulbar worsening [3-5]. The sequence observed here, namely apparently successful spontaneous breathing trial followed by delayed hypercapnic hypoventilation, is highly illustrative.

Fourth, early electrophysiological assessment remains a key diagnostic tool in the ICU [1-3]. In our case, electromyography rapidly supported the diagnosis of a postsynaptic neuromuscular transmission disorder, while normal cerebrospinal fluid examination argued against Guillain-Barre syndrome, an important differential diagnosis in this setting [3].

Finally, this case underlines the importance of identifying aggravating medications and initiating targeted ICU management. Fluoroquinolones are recognized as drugs that may worsen MG or reveal its symptoms; international guidance recommends avoiding them or using them with caution in myasthenic patients, and the FDA has issued a reinforced warning regarding exacerbation of MG [2,10]. Although ciprofloxacin and then levofloxacin were probably not the sole cause of deterioration, they may have further increased diaphragmatic weakness in a patient already destabilized by severe infection.

Rescue therapy in myasthenic crisis relies mainly on intravenous immunoglobulin and plasma exchange [2,7-9]. Both strategies are considered valid, with the choice depending on clinical context, availability, contraindications, and hemodynamic stability [2]. Recent syntheses suggest that plasma exchange may provide faster improvement in some patients, but at the cost of a more invasive procedure, without a clear mortality difference [7,8]. In the present case, IVIG was a pragmatic and appropriate option in the context of recent septic shock, and the favorable response was consistent with published evidence [2,7-9]. Early tracheostomy also probably contributed to safer airway management and more progressive ventilator weaning [3-5].

From a practical standpoint, clinicians should actively consider MG in any young adult with unexplained extubation failure, especially when no cardiac, metabolic, or persistent parenchymal cause is identified. Electrophysiological testing should be requested promptly, aggravating factors should be corrected, and rescue immunotherapy as well as tracheostomy should be anticipated when clinically indicated [2-5].

#### 3.1. Selected Literature Relevant to the Present Case

**Table 2** Selected published data compared with the present case.

Study	Type / context	Main finding	Relevance to the present case
Wendell & Levine, 2011 [3]	Narrative review of myasthenic crisis	Defines myasthenic crisis, summarizes major triggers, and reviews ICU management principles.	Provides the conceptual framework for an inaugural ICU presentation.
Seneviratne et al., 2008 [4]	Retrospective series of ventilated patients with myasthenic crisis	Extubation failure is frequent and associated with longer ICU and hospital stay.	Mirrors the early reintubation observed in the present patient.

Neumann et al., 2024 [5]	Multicenter analysis of ventilator weaning in myasthenic crisis	Difficult ventilator weaning is common; pneumonia and sepsis are associated with failure.	Very close to the infectious and septic context of this case.
Pavlekovics et al., 2023 [7] / Raval et al., 2021 [8]	Comparative syntheses of IVIG versus plasma exchange	Both options are effective; plasma exchange may act faster but is more invasive.	Supports the choice of IVIG in a hemodynamically fragile patient.
Present case	Case report	Severe hypoxemic pneumonia revealed myasthenic crisis diagnosed after extubation failure, with a favorable outcome after early tracheostomy and IVIG.	Provides a practical ICU illustration of delayed neuromuscular diagnosis in severe infection.

#### 4. Conclusion

Severe pneumonia may reveal inaugural myasthenic crisis. Any unexplained extubation failure in a young adult should prompt consideration of myasthenia gravis, particularly when no cardiac, metabolic, or residual parenchymal cause is identified.

Rapid electrophysiological assessment, avoidance of aggravating medications, rescue immunotherapy with intravenous immunoglobulin, and early tracheostomy when indicated may allow effective ventilator weaning and satisfactory functional recovery.

#### Compliance with ethical standards

##### *Disclosure of conflict of interest*

The authors declare that they have no competing interests.

##### *Statement of informed consent*

Written informed consent for publication of the clinical details was obtained from the patient.

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##### *Author contributions*

All authors contributed to the conception of the manuscript, acquisition of clinical data, drafting or critical revision of the text, and approval of the final version.

##### *Data availability*

All relevant clinical data are included in this article. Additional de-identified information may be made available by the corresponding author on reasonable request, subject to patient confidentiality.

##### *Declaration of generative AI and AI-assisted technologies in the manuscript preparation process*

During the preparation of this work the author(s) used Chatgpt in order to adjust the text and correct grammar. After using this tool/service, the author(s) reviewed and edited the content as needed and take(s) full responsibility for the content of the published article.

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