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Case Report

Unmasking Acute Myasthenic Crisis Presenting as Respiratory Failure in an Elderly Male

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ABSTRACT

Background: Myasthenic crisis is a life-threatening neurological emergency characterized by respiratory failure due to neuromuscular weakness. Diagnosis may be delayed in elderly patients with overlapping cardiopulmonary comorbidities.

Case Summary: A 73-year-old male with hypertension, diabetes mellitus, and recently diagnosed bronchial asthma presented with progressive dyspnea. Despite relatively stable vital signs, clinical examination revealed bulbar dysfunction and neck flexor weakness. Arterial blood gas analysis demonstrated hypercapnic respiratory failure. Electrophysiological studies and serology confirmed previously undiagnosed myasthenia gravis presenting as myasthenic crisis. The patient required invasive mechanical ventilation and was treated with intravenous immunoglobulin, acetyl-cholinesterase inhibitors, and corticosteroids, resulting in full clinical recovery.

Conclusion: This case highlights the importance of early recognition of myasthenic crisis in elderly patients presenting with respiratory failure and emphasizes the value of careful neurological examination in the emergency setting.

KEYWORDS

• Myasthenic crisis • Respiratory failure • Emergency medicine • Emergency department • Neurocritical care

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INTRODUCTION

Myasthenia gravis (MG) is an autoimmune disorder affecting the neuromuscular junction, leading to fluctuating skeletal muscle weakness. Myasthenic crisis represents a severe exacerbation characterized by respiratory failure requiring ventilatory support.

Approximately 15–20% of patients with MG experience a crisis during their disease course. In older adults, diagnosis may be challenging due to atypical presentation and coexistence of cardiopulmonary disease. This case report describes a previously undiagnosed myasthenic crisis presenting primarily as respiratory failure.[1–3]

Case Presentation

A 73-year-old male presented to the Emergency Department (ED) with progressive breathlessness over two weeks. The dyspnea was insidious in onset and gradually worsening. There was no history of chest pain, orthopnoea, paroxysmal nocturnal dyspnea, recent infections, or medication changes.

His past medical history included hypertension and type 2 diabetes mellitus for over 20 years, both on regular treatment. Two months prior, he had been diagnosed with bronchial asthma and initiated on inhaled corticosteroids and bronchodilators. There was no prior history of neuromuscular disease, dysphagia, or similar symptoms. Family and psychosocial history were non-contributory.

Clinical Findings

On presentation, vital signs were as follows: heart rate 88 beats/min, blood pressure 138/84 mmHg, respiratory rate 22 breaths/min, oxygen saturation 90% on room air, and temperature 101°F. The patient was alert and oriented (Glasgow Coma Scale 15/15).

Neurological examination revealed nasal speech, mild bilateral ptosis, and weakness of neck flexor muscles, with preserved limb strength. Respiratory examination demonstrated paradoxical abdominal movements and markedly reduced bilateral air entry without wheeze or crackles. Cardiovascular and abdominal examinations were unremarkable.

Diagnostic Assessment

Laboratory investigations, including complete blood count, serum electrolytes, renal function, and liver function tests, were within normal limits. Arterial blood gas analysis demonstrated respiratory acidosis (pH 7.29, $PaCO_2$ 60 mmHg, PaO_2 68 mmHg).

Chest radiography and computed tomography of the thorax showed no evidence of consolidation, pneumothorax, or pulmonary embolism. Electrocardiography and echocardiography excluded acute cardiac pathology.

Given the presence of bulbar symptoms and hypercapnic respiratory failure disproportionate to pulmonary findings, a neuromuscular etiology was suspected. Repetitive nerve stimulation revealed a decrement in response at 3 Hertz. Serum anti–acetylcholine receptor antibodies were elevated, confirming the diagnosis of myasthenia gravis presenting as myasthenic crisis. The Tensilon test was not performed due to respiratory instability.

Therapeutic Intervention

The patient was electively intubated and mechanically ventilated for impending respiratory failure. Immuno-modulatory therapy with intravenous immunoglobulin (0.4 g/kg/day for five days) was initiated. Pyridostigmine was started cautiously via nasogastric tube following stabilization, and corticosteroids were introduced subsequently. Supportive management included chest physiotherapy, deep vein thrombosis prophylaxis, glycaemic control, and close monitoring for infection.

Follow-up and Outcome

The patient showed gradual neurological and respiratory improvement. He was successfully weaned from mechanical ventilation and extubated on day 12 of hospitalization. At discharge, he was ambulatory, breathing comfortably on room air, and tolerating oral medications. He was discharged on oral pyridostigmine and prednisolone with planned neurology follow-up. No complications were noted at discharge.

DISCUSSION

Myasthenic crisis (MC) represents the most severe, potentially life-threatening complication of myasthenia gravis (MG), defined by acute neuromuscular respiratory weakness necessitating ventilatory support.

MC occurs in approximately **15–20% of patients** with MG, often within the first 2–3 years of disease onset, but can also be the **initial presentation** in a minority of cases, as seen in our patient.[3,8]

Pathophysiology and Clinical Spectrum

MG is an autoimmune disorder characterized by autoantibodies (typically anti-acetylcholine receptor [AChR], less commonly MuSK or other targets) that impair neuromuscular transmission at the postsynaptic membrane, resulting in fatigable skeletal muscle weakness.[1,14] Bulbar, ocular, and proximal limb muscles are preferentially affected, and **respiratory muscle involvement** including the diaphragm and accessory muscles can precipitate a crisis state. MC may result not only from diaphragmatic failure but also from **bulbar dysfunction**, leading to ineffective airway protection and secretion clearance, compounding respiratory compromise.[2, 7]

Clinical recognition requires high suspicion because respiratory failure in MG patients may be obscured by atypical features or absent overt pulmonary disease, particularly in elderly individuals with co-morbid conditions. A case series highlights that respiratory dysfunction in MG can be multifactorial and that careful neurologic assessment is essential to differentiate MC from non-myasthenic causes of hypoxia or hypercapnia.[3,15]

Triggers and Risk Factors

Common precipitants include **respiratory infections, systemic stress, surgery, medication changes**, and specific drugs known to worsen neuromuscular transmission (e.g., fluoroquinolones, aminoglycosides, and beta-blockers). However, in up to **30–40 % of MC episodes no clear trigger is identified**, underscoring the unpredictable nature of this complication. Older age alone appears to be a risk factor for acute respiratory failure in MG, with patients ≥80 years showing higher rates of ventilatory failure compared with younger cohorts.[7,10]

In our patient, although respiratory symptoms were initially attributed to presumed asthma exacerbation, the **absence of primary pulmonary pathology**, presence of bulbar signs (ptosis, nasal speech), and development of hypercapnia directed attention to a neuromuscular etiology, consistent with prior reports emphasizing comprehensive neurologic evaluation in unexplained respiratory failure.

Diagnostic Considerations

Electrophysiological testing (e.g., repetitive nerve stimulation demonstrating decrement) and serologic assays for AChR antibodies remain diagnostic cornerstones in MC, particularly when bulbar or respiratory signs dominate the clinical picture. This is critical given that **MC can present without classic limb weakness or ocular symptoms**, making early diagnosis challenging.[7,14]

Management Principles

The cornerstone of MC management is **prompt airway protection and ventilatory support.**[2,3] Historical analyses showed that early intubation and mechanical ventilation substantially improve survival compared with delayed intervention, with small airway reflexes and bulbar dysfunction often improving only with adequate respiratory support.[3,4]

Immunomodulatory rescue therapy with plasma exchange (PLEX) or intravenous immunoglobulin (IVIG) is widely used to hasten recovery.[6,7,10] While older literature and retrospective data suggest equivalent clinical endpoints between rescue and standard therapy over the long term, there is **a trend toward faster ventilator weaning and shorter ICU stay with PLEX compared with IVIG** in some cohorts.[5,6] Both modalities are considered appropriate first-line interventions; choice often depends on resource availability, contraindications, and patient factors. In some refractory cases, **complement inhibitors such as ravulizumab or eculizumab**—targeting C5—have shown promise in Case Report for facilitating recovery when conventional immunotherapies fail, though controlled data are limited.[3,4,8]

Corticosteroids and acetyl-cholinesterase inhibitors are typically part of chronic MG management but are used cautiously during MC; anticholinesterases may be withheld acutely due to concerns about increased secretions and bronchospasm.[12] Corticosteroids are often introduced once respiratory status stabilizes.

Prognosis and Outcomes

Advances in critical care, including invasive and non-invasive ventilation strategies, have

markedly reduced MC mortality over the past decades. Modern series report favourable outcomes with appropriate and timely intervention, although prolonged ICU stays and complications remain significant, particularly in elderly and comorbid populations.

CONCLUSION

This case reinforces several key principles:

1. **High index of suspicion** for MC is warranted in patients with respiratory failure disproportionate to pulmonary findings, especially when accompanied by subtle bulbar signs.
2. **Early neurologic evaluation and electrophysiological testing** can expedite diagnosis in atypical presentations.
3. **Immediate respiratory support and immunomodulation** are essential to reduce morbidity and mortality.

Given the observed risk of crisis in older MG patients and the potential for atypical presentation, clinicians should maintain diagnostic vigilance even in the absence of classical MG symptoms.

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