CASE REPORT

The Role of CRRT in Optimizing the Management of Septic Shock in Patients with Myasthenia Gravis Improves Outcomes

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ABSTRACT

Background: Septic shock is a life-threatening condition arising from an uncontrolled host response to infection, leading to organ dysfunction and high mortality. Patients with myasthenia gravis are more susceptible to respiratory infections, particularly pneumonia, which can aggravate their clinical condition and increase the risk of severe complications.
Case: We report a case of a 47-year-old woman with a known history of myasthenia gravis who developed septic shock secondary to pneumonia, accompanied by acute kidney injury (AKI). The patient presented with respiratory distress and hemodynamic instability requiring mechanical ventilation and vasopressor support. She was managed with a comprehensive approach including fluid resuscitation, broad-spectrum antibiotics, ventilatory support, and continuous renal replacement therapy (CRRT). Immunomodulatory therapies such as plasmapheresis or intravenous immunoglobulin (IVIG) were not administered.
Discussion: During the course of treatment, the patient showed gradual clinical improvement, as indicated by stabilization of hemodynamic status, correction of metabolic and electrolyte imbalances, recovery of renal function, and successful weaning from mechanical ventilation. The improvement observed may be related to adequate control of the underlying infection and optimization of organ support, including the use of CRRT, which may help maintain metabolic stability and contribute to the removal of inflammatory mediators. This case also suggests that in certain clinical settings, management focused on the underlying cause of deterioration may be sufficient without immediate use of immunomodulatory therapy.
Conclusion: Comprehensive management of septic shock, including infection control, hemodynamic stabilization, and organ support with CRRT, may lead to favorable clinical outcomes in patients with myasthenia gravis. Further studies are needed to better define the role of CRRT as an adjunctive therapy in this context.

Keywords: CRRT; myasthenia gravis; plasmapheresis; pneumonia; septic shock

INTRODUCTION

Septic shock occurs when the body's immune response to infection becomes dysregulated, leading to tissue damage and organ dysfunction. In pneumonia, pathogens such as Streptococcus pneumoniae or Pseudomonas aeruginosa can trigger an excessive inflammatory response.[1]

Patients with myasthenia gravis have a higher risk of infection, particularly pneumonia, due to respiratory muscle weakness and long-term use of immunosuppressive therapy. Pneumonia in myasthenia gravis patients may precipitate symptom exacerbation and increase the risk of serious complications such as shock.[2]

Septic shock is a medical emergency characterized by organ dysfunction resulting from an uncontrolled host response to infection and is associated with high mortality. The combination of myasthenia gravis, pneumonia, and septic shock requires prompt and appropriate management to prevent further complications and improve patient outcomes.[2]

CASE

A 47-year-old female patient presented to the emergency department with a complaint of shortness of breath accompanied by sputum that was difficult to expectorate for one month prior to admission. The dyspnea progressively worsened, and one day prior to admission, the patient was brought to the emergency department of Bhakti Asih Hospital, where nebulization therapy and oxygen were administered. The symptoms improved, but the patient still felt weak. Numbness or tingling was denied. Nausea and vomiting were also denied. The patient had a history of myasthenia gravis diagnosed six months earlier and had been routinely taking pyridostigmine (Mestinon) since October 2024.

Upon arrival at the emergency department of Fatmawati Hospital, the patient was in a soporous state with blood pressure 70/52 mmHg, heart rate 105 beats/min, respiratory rate 34 breaths/min, and oxygen saturation 90%. The patient was intubated and started on norepinephrine infusion at 0.05 µg/kgBW/min.

Physical examination upon intensive care unit (ICU) admission revealed a soporous, critically ill patient who had already been intubated and placed on mechanical ventilation in the emergency department. Vital signs were blood pressure 113/90 mmHg, heart rate 78 beats/min, respiratory rate 14 breaths/min, and oxygen saturation 99% on mechanical ventilation (SIMV mode, respiratory rate 14 breaths/min, tidal volume 360 mL, PEEP 5 cmH_2O, FiO_2 50%). Urine output was anuric for 9 hours, with a fluid balance of +548 mL over 6 hours. General physical examination was within normal limits, and neurological status was also within normal limits.

Laboratory results indicated infection, with leukocyte count 2,400/µL and differential count 1/0/93/4/3, D-dimer 608, CRP 19.91, ionized calcium (Ca++) 1.29, and lactate 5.6. The patient also developed acute kidney injury (AKI) with urea/creatinine levels of 31.2/0.5. Chest radiography showed infiltrates in the perihilar and bilateral lower lung fields suggestive of pneumonia, with increased findings compared to previous imaging, as well as suspected left pleural effusion with decreased impression. The patient was diagnosed with septic shock due to pneumonia, lymphopenia, myasthenia gravis with a history of dysphagia, and AKI.

On the first day of ICU admission, the patient received intravenous antibiotics consisting of ceftriaxone 2 g twice daily and levofloxacin 1 g once daily, which was subsequently replaced with amikacin 750 mg once daily. Antifungal therapy with fluconazole 400 mg twice daily was also initiated. Fluid correction was provided using 250 mL of 5% albumin infusion, and continuous renal replacement therapy (CRRT) was planned.

A central venous catheter (CVC) was inserted into the left jugular vein, and a double-lumen catheter (CDL) was placed in the right jugular vein for vascular access. CRRT was indicated in this patient due to anuria for 9 hours and septic shock accompanied by elevated blood lactate levels.

On the second day of ICU care, the patient developed dyspnea due to obstruction of the endotracheal tube (ETT), and reintubation was performed. Physical examination revealed blood pressure of 136/88 mmHg, heart rate of 124 beats/min, respiratory rate of 25 breaths/min, and SpO_2 of 99% on mechanical ventilation. Laboratory results showed leukocyte count 15,000/µL, lactate 3.8 mmol/L, sodium 142 mmol/L, and potassium 2.9 mmol/L (under correction). A tracheostomy was planned for day four.

On the third day of ICU care, the patient's condition was stable. Physical examination showed blood pressure 116/73 mmHg, heart rate 107 beats/min, respiratory rate 18 breaths/min, and SpO_2 99%. Laboratory results showed potassium 2.8 mmol/L, and potassium correction was administered with KCl 50 mEq. Urine output remained low, therefore furosemide infusion was increased to 5 mg/hour. Preparation for urgent tracheostomy was initiated.

On the fourth day of ICU care, the patient underwent tracheostomy. Hemodynamic status remained stable, and the patient was maintained on mechanical ventilation in SIMV mode with routine suctioning. Post-tracheostomy sputum culture was obtained, and GeneXpert results were negative (not detected). Vital signs were blood pressure 130/78 mmHg, heart rate 106 beats/min, respiratory rate 12 breaths/min, and SpO_2 99%.

On the fifth day of ICU care, the patient showed clinical improvement, and gradual ventilator weaning was initiated. Oxygen saturation was 100% with oxygen supplementation at 3 L/min. Vital signs were blood pressure 117/74 mmHg, heart rate 74 beats/min, respiratory rate 18 breaths/min, and SpO_2 100%.

On the eighth day of ICU care, the patient was prepared for transfer to the high care unit (HCU). Urine output was stable, minimal gastric residual volume was noted from nasogastric tube feeding, and no results fever was observed, although leukocytosis persisted. Laboratory showed leukocyte count 15,300/µL and lactate 2.7 mmol/L. The patient continued antibiotic therapy and pulmonary physiotherapy before being transferred to the HCU.

Table 1. Lab finding in ICU

Day	Hb	Ht	Leu	Tro	Eri	GDS	Ca	Na	K	Cl	AL	Ur	Cr	eGFR	D-dimer	NLR	LA
1	12.1	37.7	24	303	4.15	153	3.1	142	4.3	101	5.6	31.2	0.51	115.68	608	26.5	841
2	10.2	31	14.7	183			1.22									12.7	1026
3	11.4	34.8	15	216		87	8	142	2.9	96	3.8					13.6	972
4	11.7	37.1	19.6	251							2.9				2692	16.1	1076
5	11.4	37	16.4	263		111		143	3.6	97	2.8	83.6	1.02			6.6	
8	10.7	34.7	13.6	389				132	3.2	83	2.1						1636
9	10.9	35.2	15.3	426		88		132	3.6	84	2.7					10.1	1284

DISCUSSION

Pneumonia complicated by septic shock is associated with significant morbidity and mortality. Approximately 10.9% of patients present with septic shock at hospital admission, a finding consistent with previous studies on community-acquired pneumonia (CAP), where the incidence of septic shock ranges from 7.4% to 10%.[3] One important risk factor for sepsis in CAP is the presence of underlying comorbidities. Previous studies have demonstrated increased 30-day mortality in septic patients with CAP who have chronic kidney disease or neurologic disorders, including myasthenia gravis.[2]

The pathophysiology of septic shock secondary to pneumonia begins when pathogens infect the lungs and trigger a dysregulated systemic inflammatory response. Excessive immune activation leads to the release of pro-inflammatory cytokines such as TNF-α, IL-1, and IL-6, resulting in vasodilation, increased vascular permeability, and impaired tissue perfusion. Reduced oxygen delivery to tissues promotes anaerobic metabolism and lactate production, contributing to metabolic derangements and organ dysfunction. Severe pneumonia may also cause hypoxia and mitochondrial dysfunction, further worsening metabolic instability and leading to multiorgan failure.[4,5,6]

In patients with myasthenia gravis, systemic infection and septic shock can worsen neuromuscular weakness through indirect mechanisms such as hypoxia, metabolic acidosis, electrolyte imbalance, and systemic inflammation, rather than solely through autoimmune activity. This mechanism is important in understanding why stabilization of septic shock may lead to improvement in neuromuscular function without specific immunomodulatory therapy.

Sepsis-induced acute kidney injury (AKI) is common in critically ill patients, accounting for approximately 40–50% of AKI cases in the ICU. Exposure of renal tubular cells to inflammatory mediators and hypoxic conditions during sepsis results in adaptive metabolic downregulation and cell-cycle arrest, which protect cells from further injury but reduce glomerular filtration and lead to clinical AKI.[7] In this patient, anuria and elevated lactate levels supported the diagnosis of septic shock with organ dysfunction based on the sequential organ failure assessment (SOFA) criteria.[4]

Management of septic shock due to pneumonia requires early and comprehensive intervention. Broad-spectrum empirical antibiotics should be administered promptly after microbiological sampling. Fluid resuscitation and vasopressor therapy, particularly norepinephrine, are recommended to maintain a mean arterial pressure ≥65 mmHg. Mechanical ventilation with a lung-protective strategy is indicated in patients with respiratory failure.[5,6] In severe CAP requiring ICU admission, ATS-IDSA guidelines recommend a combination of a beta-lactam antibiotic with a macrolide or fluoroquinolone.[8]

In patients with myasthenia gravis complicated by infection and septic shock, therapies such as plasmapheresis or intravenous immunoglobulin (IVIG) are often considered, particularly in myasthenic crisis. However, evidence suggests that immunomodulatory therapy may not always be necessary when the primary precipitating factor is severe infection. A national observational study in Japan published in *Clinical Infectious Diseases* (2015) found no significant difference in 28-day mortality between septic pneumonia patients treated with IVIG and those who were not.[9] Similarly, recent reviews emphasize individualized decision-making regarding IVIG or plasmapheresis based on patient condition and clinical context.[8]

In this case, improvement in neuromuscular function occurred following stabilization of septic shock through infection control, hemodynamic support, ventilatory management, and organ support, without the use of immunomodulatory therapy. This finding highlights that addressing the underlying precipitating factor — septic shock due to pneumonia — can lead to clinical improvement in myasthenia gravis symptoms.

Continuous renal replacement therapy (CRRT) was initiated due to anuria and hemodynamic instability. CRRT is commonly used in septic patients with AKI to maintain fluid, electrolyte, and acid-base balance while providing hemodynamic stability.[10–12] Compared with intermittent hemodialysis, CRRT offers gradual solute and fluid removal, which is better tolerated in unstable patients.[13] Additionally, hemofiltration may contribute to the removal of inflammatory mediators in sepsis.[14,15]

Although CRRT does not directly treat myasthenia gravis pathophysiology, stabilization of metabolic and hemodynamic status may support neuromuscular recovery. In this patient, improvement in clinical condition, including respiratory function and overall stability, was achieved after septic shock management and CRRT support, even in the absence of immunomodulatory therapy.

These findings emphasize the importance of identifying and treating precipitating factors in myasthenia gravis patients with critical illness. Adequate management of septic shock can result in significant clinical improvement in myasthenia gravis, allowing recovery without the need for plasmapheresis or IVIG.

CONCLUSION

This case highlights that early, comprehensive management of septic shock in patients with myasthenia gravis, focusing on infection control, hemodynamic stabilization, and organ support, can lead to favorable clinical outcomes. The use of CRRT played an important role in managing AKI and maintaining metabolic and electrolyte balance, while potentially contributing to the modulation of the inflammatory response.

Notably, clinical improvement was achieved without immunomodulatory therapies such as plasmapheresis or IVIG, suggesting that in myasthenia gravis patients where infection is the primary precipitating factor, optimal sepsis management may be sufficient to support neuromuscular recovery. Further studies are needed to clarify the role of CRRT as an adjunctive therapy in such complex clinical settings.

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