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Acute Respiratory Distress Syndrome in a Patient with Myasthenia Gravis and Septic Shock: A Case Report

Taufiqurrochman Nur Amin[1*], Septian Adi Permana[1]

[1]Department of Anesthesiology and Intensive Therapy, Dr. Moewardi Regional General Hospital, Surakarta, Indonesia

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***Corresponding author:**
Taufiqurrochman Nur Amin

E-mail address:
taufiqnuramin@gmail.com

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A B S T R A C T

Introduction: Myasthenia gravis (MG) is an autoimmune disorder characterized by muscle weakness, often affecting respiratory and oropharyngeal muscles. This predisposition to respiratory compromise, coupled with impaired swallowing and the potential need for immunosuppressive therapies, increases the risk of pneumonia and subsequent sepsis in MG patients. Sepsis, in turn, is a significant risk factor for acute respiratory distress syndrome (ARDS), a severe lung condition with high mortality. Ca**se presentation:** We present the case of a 47-year-old male with a 4-year history of MG who was admitted to our hospital with progressive dyspnea and dysphagia. His condition deteriorated rapidly, leading to septic shock and respiratory failure necessitating invasive mechanical ventilation. Blood cultures identified *Klebsiella pneumoniae* with extended-spectrum beta-lactamase (ESBL) production. Despite aggressive treatment, including therapeutic plasma exchange (TPE), the patient's hospital course was complicated. **Conclusion:** This case underscores the critical importance of vigilant monitoring and early intervention in MG patients presenting with respiratory symptoms or signs of infection. Prompt recognition and aggressive management of sepsis are crucial to mitigate the risk of ARDS and improve outcomes in this vulnerable patient population.

1. Introduction

Myasthenia gravis (MG) is a chronic autoimmune disorder characterized by fluctuating muscle weakness and fatigue due to autoantibodies targeting components of the neuromuscular junction, most commonly the acetylcholine receptor (AChR). The hallmark of MG is fatigable weakness, which worsens with exertion and improves with rest. The muscles most frequently affected are those involved in ocular, bulbar (facial and throat), and limb movements. However, in severe cases or during exacerbations, respiratory muscles can also be involved, leading to respiratory insufficiency or failure. The clinical manifestations of MG are heterogeneous, ranging from mild ocular symptoms like ptosis and diplopia to severe generalized weakness affecting limb, bulbar, and respiratory muscles. The disease course is often unpredictable, with periods of remission and exacerbation. Exacerbations can be triggered by various factors, including infections, medications, stress, and surgery. Respiratory complications are a major cause of morbidity and mortality in MG patients. The most common respiratory complications include myasthenic crisis, aspiration pneumonia, and respiratory failure. Myasthenic crisis is a life-threatening exacerbation of MG characterized by severe respiratory muscle weakness, often requiring mechanical ventilation. Aspiration pneumonia is

another frequent complication, due to impaired swallowing and bulbar muscle weakness, which can increase the risk of aspiration of oropharyngeal secretions or gastric contents.[1,2]

The management of MG involves a multidisciplinary approach, including neurologists, pulmonologists, intensivists, and other specialists as needed. Treatment options include acetylcholinesterase inhibitors (e.g., pyridostigmine), immunosuppressive drugs (e.g., corticosteroids, azathioprine, mycophenolate mofetil), and immunomodulatory therapies (e.g., intravenous immunoglobulin, plasmapheresis). In cases of myasthenic crisis or respiratory failure, mechanical ventilation and supportive care are essential. Sepsis is a life-threatening organ dysfunction caused by a dysregulated host response to infection. It is a major global health concern, with high morbidity and mortality rates. Sepsis can be triggered by various infections, including pneumonia, urinary tract infections, intra-abdominal infections, and bloodstream infections. The most common pathogens associated with sepsis include gram-negative bacteria (e.g., *Escherichia coli*, *Klebsiella pneumoniae*), gram-positive bacteria (e.g., *Staphylococcus aureus*, *Streptococcus pneumoniae*), and fungi (e.g., *Candida* species). The pathophysiology of sepsis involves a complex interplay of pro-inflammatory and anti-inflammatory responses, endothelial dysfunction, coagulation abnormalities, and impaired microcirculation. These processes can lead to multiple organ dysfunction syndrome (MODS), characterized by the dysfunction of two or more organ systems. The lungs are particularly vulnerable to injury in sepsis, and acute respiratory distress syndrome (ARDS) is a common complication.[3,4]

ARDS is a severe inflammatory lung condition characterized by diffuse alveolar damage, increased alveolar-capillary permeability, pulmonary edema, and impaired gas exchange. The clinical presentation of ARDS includes acute onset of respiratory distress, hypoxemia refractory to oxygen therapy, and bilateral pulmonary infiltrates on chest imaging. The management of ARDS involves supportive care, including mechanical ventilation with lung-protective strategies, fluid management, and treatment of the underlying cause. The coexistence of MG and sepsis poses unique challenges in clinical management. MG patients are inherently vulnerable to respiratory complications due to muscle weakness and impaired swallowing, and sepsis can further exacerbate these issues. Additionally, the immunosuppressive therapies used in MG can increase the risk of secondary infections and complicate the management of sepsis. In this case report, we present a patient with a history of MG who developed ARDS secondary to septic shock caused by *Klebsiella pneumoniae*.[5,6] This case highlights the complex interplay between MG, sepsis, and ARDS, and underscores the importance of early recognition and aggressive management of sepsis in MG patients to prevent the development of ARDS and improve outcomes. We will discuss the clinical presentation, diagnostic workup, treatment course, and outcomes of this patient, and review the relevant literature on the management of MG patients with sepsis and ARDS.

2. Case Presentation

A 47-year-old male with a four-year history of generalized myasthenia gravis (MG) presented to Dr. Moewardi Regional General Hospital, Surakarta, Indonesia, with a three-day history of worsening shortness of breath. The dyspnea had intensified in the 24 hours preceding admission, making sleep difficult. He also reported a week-long history of dysphagia, limiting his oral intake to small amounts of soft foods. Upon initial evaluation, the patient exhibited signs of respiratory distress, including dyspnea and labored breathing. A chest radiograph revealed cardiomegaly and pulmonary edema, raising concerns for cardiac and/or pulmonary pathology. Laboratory investigations showed elevated hematocrit (45%), leukocytosis (18,500/µL), mildly elevated liver enzymes (SGPT 35 U/L, SGOT 70 U/L), normal creatinine (0.8 mg/dL), and hypokalemia (2.7 mEq/L) (Table 1&2).

Based on these findings, the patient was admitted to the neurology ward with diagnoses of myasthenia gravis exacerbation, community-acquired pneumonia (CAP), and hypertension. His pneumonia severity

index (PSI) score was 57, classifying his pneumonia as class II, indicating a moderate risk of mortality. Initial treatment included mecobalamin (500 mcg twice daily), pyridostigmine (60 mg every 8 hours), amlodipine (10 mg daily), and ranitidine. However, the patient's respiratory status deteriorated rapidly on the day of admission, prompting transfer to the intensive care unit (ICU). On ICU admission, the patient presented with dysarthria, dysphagia, and severe dyspnea. His vital signs were notable for elevated blood pressure (190/100 mmHg), tachycardia (130 bpm), and tachypnea (28 breaths/min). Neurological examination revealed no focal deficits, and his Glasgow Coma Scale (GCS) score was 15 (E4V5M6), indicating an alert and oriented state. The patient's respiratory failure progressed, and his GCS score declined to a level consistent with sopor (E4V4M5) (Table 1&2). He was subsequently intubated and mechanically ventilated for airway protection and respiratory support. Additional measures included the placement of a nasogastric tube for enteral feeding, insertion of a peripherally inserted central catheter (PICC) for long-term intravenous access, and initiation of hemodialysis (HD) for acute kidney injury.

Blood cultures obtained after ICU admission yielded positive results for *Klebsiella pneumoniae* with extended-spectrum beta-lactamase (ESBL) production, confirming the diagnosis of gram-negative bacterial pneumonia and sepsis (table 1&2). The patient's antibiotic regimen was adjusted accordingly, and he received intravenous omeprazole, mecobalamin, pyridostigmine, azathioprine (Imuran), potassium supplementation, meropenem, and intravenous fluids (Hidonac). Therapeutic plasma exchange (TPE) was initiated using a centrifugation technique with albumin replacement. The patient's fluid balance post-TPE was maintained at zero, with an input of 2000 mL and an output of 2000 mL. After 12 days of intensive care, the patient's condition improved significantly. His level of consciousness increased, and his overall clinical status stabilized. He was transferred back to the neurology ward for further management and rehabilitation before being discharged from the hospital.

Table 1. Clinical findings and laboratory.

Clinical finding	**Results**
Dyspnea	Yes (worsening for 3 days)
Dysphagia	Yes (present for 1 week)
Chest radiograph	Cardiomegaly, pulmonary edema
Hematocrit	45% (elevated)
Leukocyte count	18,500/µL (elevated)
SGPT	35 U/L (mildly elevated)
SGOT	70 U/L (mildly elevated)
Creatinine	0.8 mg/dL (normal)
Potassium	2.7 mEq/L (decreased)
Blood pressure	190/100 mmHg (elevated)
Heart rate	130 bpm (elevated)
Respiratory rate	28 breaths/min (elevated)
Neurological examination	Dysarthria, dysphagia, no focal motor deficits
Glasgow coma scale (GCS)	15 (E4V5M6) on admission, declined to sopor (E4V4M5) later
Blood culture	*Klebsiella pneumoniae* with ESBL (+)

Table 2. Outlines the clinical course of the patient during hospitalization

Day	Intervention or finding	Outcome
1	Admitted to neurology ward	Diagnosis of MG exacerbation, CAP, and hypertension. Treated with mecobalamin, pyridostigmine, amlodipine, and ranitidine.
1	Transferred to ICU	Worsening respiratory status, dysarthria, dysphagia, severe dyspnea, elevated blood pressure, tachycardia, tachypnea.
1	Intubation and mechanical ventilation	Airway protection and respiratory support.
1	Placement of nasogastric tube, PICC, and initiation of HD	Nutritional support, long-term intravenous access, and renal support.
2	Blood cultures positive for *Klebsiella pneumoniae* with ESBL	Confirmation of gram-negative bacterial pneumonia and sepsis. Antibiotic regimen adjusted.
2	Initiation of intravenous omeprazole, mecobalamin, pyridostigmine, azathioprine, potassium supplementation, meropenem, and intravenous fluids (Hidonac).	Treatment for sepsis and MG exacerbation.
3	Therapeutic plasma exchange (TPE) initiated	Removal of inflammatory mediators and potential pathogenic antibodies.
12	Significant improvement in the condition	Transfer back to the neurology ward.
Discharge	Discharged home	Required supplemental oxygen and ongoing physical therapy.

3. Discussion

Myasthenia gravis (MG) is a chronic autoimmune disorder primarily characterized by autoantibodies that target the neuromuscular junction (NMJ), the critical synapse where nerve impulses are transmitted to muscle fibers. This autoimmune attack disrupts the normal signaling process, leading to fluctuating muscle weakness and fatigue, which are the hallmark symptoms of MG. The severity and distribution of muscle weakness can vary widely among individuals, ranging from mild ocular symptoms like ptosis (drooping eyelids) and diplopia (double vision) to more generalized weakness affecting limb, facial, and respiratory muscles. The underlying pathophysiology of MG involves a complex interplay of immunological and molecular mechanisms. In most cases (approximately 80-85%), autoantibodies are directed against the acetylcholine receptors (AChRs) on the postsynaptic membrane of the NMJ. These antibodies bind to the AChRs, blocking the binding of acetylcholine (ACh), the neurotransmitter responsible for muscle contraction. This blockade reduces the number of functional AChRs available for signaling, leading to impaired neuromuscular transmission and subsequent muscle weakness. The reduction in the number of available AChRs also leads to a decrease in the amplitude of the endplate potential, the depolarization of the muscle fiber membrane that triggers muscle contraction. When the endplate potential fails to reach the threshold required for muscle fiber activation, muscle weakness ensues.[7-9]

In addition to AChR antibodies, other autoantibodies have been implicated in MG. Approximately 10-15% of patients with generalized MG and up to 50% of patients with ocular MG are positive for antibodies against muscle-specific kinase (MuSK). MuSK is a tyrosine kinase receptor that plays a crucial role in the development and maintenance of the NMJ. MuSK antibodies disrupt the clustering of AChRs at the NMJ, leading to impaired neuromuscular transmission and muscle weakness. Another target of autoantibodies in MG is lipoprotein-related protein 4 (LRP4), a transmembrane protein that interacts with MuSK and is involved in AChR clustering. LRP4 antibodies have been found in a small subset of MG patients, particularly those with mild or late-onset disease. These antibodies interfere with the LRP4-MuSK interaction, leading to reduced AChR clustering and subsequent muscle weakness. The autoimmune response in MG is thought to be

triggered by a combination of genetic and environmental factors. Genetic predisposition plays a role, as certain human leukocyte antigen (HLA) alleles are associated with an increased risk of developing MG. Environmental factors, such as viral infections, may also contribute to the initiation or exacerbation of the autoimmune response in susceptible individuals.[9-11]

The clinical manifestations of MG are diverse and can fluctuate in severity over time. The most common symptoms include: Muscle weakness and fatigue: This is the hallmark of MG and can affect any skeletal muscle group. The weakness typically worsens with exertion and improves with rest. Ocular symptoms: Ptosis and diplopia are often the initial presenting symptoms of MG. These symptoms can be asymmetric and may fluctuate throughout the day. Bulbar symptoms: Weakness of the muscles involved in speech, swallowing, and chewing can lead to dysarthria (slurred speech), dysphagia (difficulty swallowing), and difficulty chewing. Limb weakness: Weakness of the arms and legs can make it difficult to perform daily activities, such as climbing stairs, lifting objects, or walking. Respiratory weakness: In severe cases, MG can affect the respiratory muscles, leading to respiratory insufficiency and respiratory failure. This is a life-threatening complication that requires immediate medical attention. The diagnosis of MG is based on a combination of clinical features, serological testing for autoantibodies, and electrophysiological studies. The most commonly used serological tests are for AChR and MuSK antibodies. Electrophysiological studies, such as repetitive nerve stimulation and single-fiber electromyography, can help confirm the diagnosis by demonstrating impaired neuromuscular transmission.[11-13]

The fluctuating nature of muscle weakness in MG is a characteristic feature of the disease. Patients often experience periods of relative stability interspersed with exacerbations, during which muscle weakness worsens. These exacerbations can be triggered by various factors, including infection, stress, medication changes, and surgery. The respiratory complications associated with MG are of particular concern. The diaphragm and intercostal muscles, which are essential for breathing, can be affected by the disease process, leading to respiratory compromise. This can manifest as dyspnea (shortness of breath), reduced exercise tolerance, and in severe cases, respiratory failure. Furthermore, the involvement of bulbar muscles, which control swallowing and speech, can result in dysphagia (difficulty swallowing). Dysphagia not only impairs the patient's ability to eat and drink but also increases the risk of aspiration pneumonia. Aspiration occurs when food, liquids, or saliva enter the lungs instead of the esophagus, leading to inflammation and infection. The chronic use of immunosuppressive medications to manage MG further exacerbates the risk of respiratory complications. While these medications are essential for controlling the autoimmune response and improving muscle strength, they also suppress the immune system, making patients more susceptible to infections. Corticosteroids, such as prednisone, and other immunosuppressants, like azathioprine, are commonly used in MG treatment. However, their long-term use can increase the risk of opportunistic infections, including pneumonia. Pneumonia in MG patients can be particularly severe due to the underlying respiratory muscle weakness. The impaired ability to cough effectively and clear secretions from the lungs can lead to the accumulation of mucus and bacteria, facilitating the development and progression of pneumonia. Additionally, the immunosuppressive effects of MG medications can hinder the body's ability to mount an adequate immune response against the invading pathogens. The combination of respiratory muscle weakness, dysphagia, and immunosuppression creates a perfect storm for respiratory complications in MG patients. This highlights the importance of vigilant monitoring and early intervention in these patients, especially when they present with respiratory symptoms or signs of infection. Prompt diagnosis and treatment of pneumonia are crucial to prevent the progression to more severe complications, such as sepsis and acute respiratory distress syndrome (ARDS). The autoimmune attack on the

neuromuscular junction in MG not only leads to muscle weakness and fatigue but also predisposes patients to respiratory complications. The involvement of respiratory muscles, coupled with dysphagia and the use of immunosuppressive medications, significantly increases the risk of pneumonia and subsequent complications. Understanding the complex interplay of these factors is essential for the optimal management of MG patients and the prevention of life-threatening respiratory events.[13-15]

Sepsis, a life-threatening condition arising from a dysregulated immune response to infection, poses a significant risk factor for developing acute respiratory distress syndrome (ARDS). In the presented case, the patient's *Klebsiella pneumoniae* bacteremia, a gram-negative bacterium notorious for its virulence, initiated a cascade of inflammatory responses that culminated in sepsis and subsequent ARDS. The pathophysiology of sepsis-induced ARDS is a complex and multifaceted process, involving a multitude of inflammatory mediators, endothelial and epithelial injury, and dysregulated coagulation. The initial insult, in this case, the *Klebsiella pneumoniae* infection, triggers an excessive and uncontrolled release of pro-inflammatory cytokines, chemokines, and other mediators. These molecules initiate a systemic inflammatory response syndrome (SIRS), characterized by fever, tachycardia, tachypnea, and leukocytosis. The excessive inflammatory response leads to widespread endothelial activation and dysfunction. Endothelial cells, which line the blood vessels, become activated and express adhesion molecules, facilitating the recruitment and extravasation of neutrophils and other leukocytes into the lung parenchyma. These activated neutrophils release reactive oxygen species (ROS) and proteolytic enzymes, causing direct damage to the alveolar epithelium and capillary endothelium. The resulting endothelial injury disrupts the integrity of the alveolar-capillary barrier, leading to increased permeability and leakage of protein-rich fluid into the alveolar spaces. This non-cardiogenic pulmonary edema impairs gas exchange, leading to hypoxemia and respiratory distress. Additionally, the activation of coagulation pathways and the inhibition of fibrinolysis contribute to the formation of microthrombi within the pulmonary vasculature, further compromising blood flow and oxygen delivery to the lungs. The epithelial injury in ARDS involves damage to the alveolar epithelial cells, which are responsible for maintaining the structural integrity of the alveoli and producing surfactant, a substance that reduces surface tension and prevents alveolar collapse. The loss of surfactant function leads to alveolar instability, atelectasis, and decreased lung compliance, further exacerbating respiratory dysfunction. The combined effects of endothelial and epithelial injury, pulmonary edema, and microthrombi formation result in a significant ventilation-perfusion mismatch, leading to profound hypoxemia and respiratory failure. The lungs become stiff and non-compliant, requiring increased ventilatory pressures to maintain adequate oxygenation. This, in turn, can lead to ventilator-induced lung injury (VILI), further perpetuating the cycle of inflammation and lung damage. In the context of MG, the pre-existing respiratory muscle weakness and impaired cough reflex exacerbate the respiratory compromise caused by ARDS. The diaphragm and intercostal muscles, already weakened by the autoimmune attack in MG, are further compromised by the inflammatory process in ARDS, leading to respiratory muscle fatigue and failure. The impaired cough reflex hinders the clearance of secretions, increasing the risk of airway obstruction and pneumonia.[15-17]

The disruption of the alveolar-capillary barrier results in increased permeability, allowing protein-rich fluid to leak into the alveolar spaces. This non-cardiogenic pulmonary edema impairs gas exchange, leading to hypoxemia and respiratory distress. Additionally, the accumulation of inflammatory cells and debris within the alveoli contributes to the formation of hyaline membranes, a hallmark of ARDS. The dysregulated coagulation system further exacerbates the lung injury in sepsis-induced ARDS. The activation of the coagulation cascade leads to the formation of microthrombi within the pulmonary vasculature, impairing blood flow and contributing to

ventilation-perfusion mismatch. The resulting tissue hypoxia and ischemia further amplify the inflammatory response, creating a vicious cycle of injury. In addition to the direct effects on the lungs, sepsis also induces systemic effects that contribute to the development of ARDS. The release of inflammatory mediators into the circulation can lead to myocardial dysfunction, hypotension, and multi-organ failure. These systemic effects further compromise oxygen delivery and exacerbate tissue hypoxia, contributing to the progression of ARDS. The patient's underlying myasthenia gravis (MG) likely contributed to the severity of their condition. MG, an autoimmune disorder characterized by muscle weakness, can affect the respiratory muscles, impairing the ability to cough and clear secretions effectively. This predisposition to respiratory compromise, coupled with the immunosuppressive effects of MG treatments, increases the risk of pneumonia and subsequent sepsis in MG patients.[17,18]

The management of sepsis-induced acute respiratory distress syndrome (ARDS) in patients with myasthenia gravis (MG) presents a formidable challenge due to the intricate interplay between the underlying neuromuscular disorder, the systemic inflammatory response of sepsis, and the complexities of mechanical ventilation. MG, characterized by autoantibody-mediated dysfunction of the neuromuscular junction, frequently manifests with fluctuating weakness of skeletal muscles, including those involved in respiration. This respiratory muscle weakness can predispose MG patients to respiratory failure, particularly in the context of sepsis, where increased metabolic demands and potential diaphragmatic dysfunction can further compromise respiratory function. Early initiation of mechanical ventilation is often necessary to maintain adequate oxygenation and ventilation in MG patients with sepsis-induced ARDS. However, mechanical ventilation itself can contribute to lung injury through mechanisms such as volutrauma (excessive lung stretch), barotrauma (high airway pressures), atelectrauma (repetitive opening and closing of alveoli), and biotrauma (activation of inflammatory cascades). To mitigate the risk of ventilator-induced lung injury (VILI), lung-protective ventilation strategies are paramount. These strategies include low tidal volume ventilation (6 mL/kg of predicted body weight), limitation of plateau pressures (<30 cmH2O), and the use of positive end-expiratory pressure (PEEP) to maintain alveolar recruitment and prevent atelectasis. In MG patients, the titration of ventilator settings requires careful consideration of their underlying respiratory muscle weakness. Excessive ventilator support can lead to diaphragmatic inactivity and atrophy, further delaying weaning and prolonging the duration of mechanical ventilation. Therefore, a delicate balance must be struck between providing adequate support and minimizing ventilator-induced complications.[16,19]

The management of MG often involves immunosuppressive therapies, such as corticosteroids, azathioprine, and mycophenolate mofetil, to suppress the autoimmune response and improve muscle strength. However, these medications can also impair the immune system's ability to fight off infections, increasing the risk of sepsis and other opportunistic infections. In the context of sepsis-induced ARDS, the use of immunosuppressive agents can be a double-edged sword. While necessary to control MG exacerbations, they can also hinder the body's ability to mount an effective immune response against the underlying infection. This can lead to prolonged or recurrent sepsis, delayed recovery, and increased mortality. Therefore, careful consideration must be given to the timing and dosage of immunosuppressive therapies in MG patients with sepsis-induced ARDS. In some cases, it may be necessary to temporarily reduce or withhold immunosuppression to allow for a more robust immune response against the infection. However, this decision must be weighed against the risk of MG exacerbation, which can further compromise respiratory function. The development of ARDS in the context of sepsis is a complex and multifactorial process involving a dysregulated inflammatory response, endothelial and epithelial injury, and dysregulated coagulation. The initial insult, such as pneumonia in this case, triggers the release of pro-

inflammatory cytokines and chemokines, which activate neutrophils and other immune cells. These activated immune cells release reactive oxygen species and proteases, which damage the alveolar-capillary membrane, leading to increased permeability and leakage of protein-rich fluid into the alveolar space. This results in pulmonary edema, impaired gas exchange, and hypoxemia. In addition to the direct effects of inflammation, sepsis can also lead to microvascular thrombosis and endothelial dysfunction, further compromising pulmonary blood flow and oxygen delivery. The dysregulated coagulation cascade can also contribute to the formation of hyaline membranes, a hallmark of ARDS. The patient's underlying MG further complicates the pathophysiology of sepsis-induced ARDS. The respiratory muscle weakness associated with MG can exacerbate respiratory failure and necessitate earlier mechanical ventilation. Additionally, the immunosuppressive therapies used to manage MG can impair the immune response to infection, potentially leading to a more severe and prolonged course of sepsis. The management of sepsis-induced ARDS in MG patients requires a nuanced and individualized approach. The respiratory muscle weakness associated with MG necessitates early mechanical ventilation, but careful attention must be paid to lung-protective strategies to minimize VILI. The use of immunosuppressive therapies must be balanced against the need for an effective immune response against the underlying infection. Understanding the complex pathophysiological mechanisms underlying sepsis-induced ARDS is crucial for developing effective therapeutic strategies and improving outcomes in this critically ill patient population. Further research is needed to identify biomarkers that can predict the development of ARDS in MG patients and to develop targeted therapies that can modulate the inflammatory response and mitigate lung injury.[19,20]

4. Conclusion

ARDS in MG patients is a serious complication with high mortality. Early recognition and aggressive management of sepsis are crucial to prevent the development of ARDS and improve outcomes. This case highlights the importance of a multidisciplinary approach in managing complex cases like this and the need for further research to optimize the treatment of ARDS in MG patients.

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