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THERAPEUTIC PLASMA EXCHANGE IN SEPTIC SHOCK

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ABSTRACT

Background : Therapeutic plasma exchange (TPE) is an emerging adjunctive therapy in septic shock, a condition marked by dysregulated immune response and high mortality. By removing circulating inflammatory mediators, endotoxins, and other pathogenic substances, TPE aims to restore immune balance and improve hemodynamic stability.

Case : A 52-year-old female patient presenting with shock sepsis secondary to pneumonia, She was admitted in a critical condition, already intubated and receiving IV fluids, and subsequently underwent three cycles of TPE each with a total replacement volume of 2700 cc consisting of diluted albumin 25%, Gelafusal, and normal saline. She developed left pleural effusion, hypoalbuminemia, and anemia, and was supported with comprehensive antimicrobial therapy including cefepime, amikacin, and antifungal micafungin, as well as corticosteroids and pyridostigmine via NGT. Invasive lines including a CVC, femoral DLC, and radial arterial line were placed. Her condition gradually improved with passive mobilization, nutritional optimization, regular suctioning, and eye care. After 18 days of ICU treatment, she was transferred to a lower-dependency unit with stable hemodynamics, improving lab parameters, and internal medicine follow-up.

Discussion : The presented case underscores the promising role of TPE an adjunctive treatment in septic shock, by demonstrating clinical improvement following targeted immunomodulation and removal of inflammatory mediators. In this 52-year-old female patient, TPE contributed to hemodynamic stabilization and reduction of systemic inflammation over three treatment cycles, amidst a complex ICU course involving pneumonia, pleural effusion, and multiple invasive interventions.

Conclusion : This case highlights the critical role of TPE in managing septic shock secondary to pneumonia and cytokine storm in a 52-year-old female patient.

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1. Introduction

Sepsis, as defined by the Third International Consensus, is a life-threatening condition characterized by organ dysfunction resulting from a dysregulated host response to infection. When circulatory failure persists despite adequate fluid resuscitation and is accompanied by elevated serum lactate levels, the condition progresses to septic shock. Despite advances in critical care, septic shock continues to carry a high mortality rate, reaching up to 60% in severe cases.[1,2]

The pathophysiology of sepsis involves a complex and interrelated cascade of systemic inflammation, endothelial injury, capillary leakage, and profound disturbances in coagulation. Excessive cytokine release, widespread endothelial activation, and recruitment of inflammatory cells contribute to microvascular dysfunction. Endothelial damage promotes platelet aggregation and consumption of coagulation factors, frequently culminating in disseminated intravascular coagulation and progressive multi-organ failure.[1,2]

Several therapeutic strategies have attempted to target individual components of this dysregulated host response. However, apart from the early administration of appropriate antimicrobial therapy, most interventions have failed to consistently demonstrate survival benefit. One previously approved targeted therapy, recombinant activated protein C, was ultimately withdrawn after subsequent studies failed to confirm clinical efficacy. These outcomes underscore the complexity of sepsis and the difficulty of modulating isolated pathways within a multifactorial syndrome.[1,2]

Therapeutic plasma exchange (TPE) has emerged as a potential adjunctive treatment because of its ability to simultaneously influence multiple pathogenic mechanisms. TPE enables the removal of circulating inflammatory mediators, cytokines, and pro-thrombotic factors while concurrently replenishing protective plasma components through replacement with donor plasma. In sepsis, dysregulation of coagulation is partly mediated by an imbalance between von Willebrand factor (vWF) and ADAMTS13, a protease responsible for cleaving ultra-large vWF multimers. Reduced ADAMTS13 activity and elevated vWF levels have been associated with increased disease severity and mortality. By removing excessive pro-thrombotic mediators and restoring deficient anticoagulant and fibrinolytic factors such as protein C, antithrombin III, and ADAMTS13, TPE may help rebalance coagulation and mitigate microvascular thrombosis.[3,4]

Over the past decades, numerous case reports and case series have suggested that adjunctive TPE may improve hemodynamic stability, reduce vasopressor requirements, attenuate inflammatory burden, and decrease short-term mortality in patients with sepsis-induced organ dysfunction. Nevertheless, prospective randomized controlled trials remain limited, and their findings have been inconclusive. Multiple meta-analyses have attempted to clarify the clinical benefit of TPE in sepsis; however, current evidence remains insufficient to recommend its routine use. Accordingly, the 2023 American Society for Apheresis (ASFA) guidelines classify TPE for sepsis-induced organ dysfunction as a Category III, Grade 2A recommendation, indicating that its optimal role has not yet been established and should be considered on an individual basis.[5,6]

Given the ongoing uncertainty and the biological plausibility supporting its mechanism of action, further evaluation of TPE in septic shock is warranted. This study aims to comprehensively review and analyze the available literature to reassess the impact of adjunctive therapeutic plasma exchange on short-term mortality and clinical outcomes in critically ill adult patients with septic shock and multiple organ dysfunction.[1,3]

This case report aims to describe the clinical course and outcome of a patient with septic shock treated with adjunctive therapeutic plasma exchange, highlighting its potential role in improving hemodynamic stability and organ dysfunction.

2. Case Report

A 52-year-old woman was admitted to the intensive care unit after referral from Gorontalo Hospital, with a primary complaint of generalized weakness accompanied by shortness of breath. Prior to transfer, she had been treated in the ICU for 12 days and was referred in an intubated state with ongoing peripheral intravenous fluid therapy. During treatment at the referring hospital, the patient developed sepsis secondary to pneumonia with progressive respiratory failure and septic shock requiring fluid resuscitation and vasopressor support. Before initiation of norepinephrine, the patient experienced hypotension with blood pressure recorded at 82/48 mmHg, mean arterial pressure of 59 mmHg, tachycardia up to 124 beats per minute, and elevated serum lactate of 2.64 mmol/L, indicating tissue hypoperfusion. Vasopressor therapy with norepinephrine was subsequently initiated due to persistent hypotension despite fluid administration. Her past medical history revealed a similar episode two years earlier, during which she required mechanical ventilation but was successfully weaned.

Upon admission, she was in a severe general condition. Her body weight was 65 kg and height 155 cm. The airway was secured with a tracheal cannula connected to mechanical ventilation in VC-SIMV mode with FiO2 90%, tidal volume 360 mL, respiratory rate 25 breaths per minute, inspiratory time 0.8 seconds, and PEEP 5 cmH2O. Oxygen saturation was 99%. Hemodynamically, the patient remained dependent on norepinephrine infusion at 0.15 mcg/kg/min to maintain adequate perfusion, with blood pressure 128/74 mmHg, mean arterial pressure 84 mmHg, and heart rate 112 beats per minute. Despite apparently stable blood pressure at ICU admission, the patient fulfilled septic shock criteria because adequate MAP could only be maintained with continuous vasopressor support following fluid resuscitation. Additional findings supporting septic shock included progressive leukocytosis, thrombocytopenia, markedly elevated inflammatory biomarkers, and worsening metabolic stress during ICU treatment.

Neurologically, she was fully conscious with a Glasgow Coma Scale score of E4M6Vx, not under sedation, with a Behavioral Pain Scale score of 4 and a negative CAM-ICU assessment. A urinary catheter was in place, with urine output of 0.48 mL/kg/hour and a calculated creatinine clearance of 120 mL/min. Fluid balance was positive at +4489 mL. A nasogastric tube was inserted with no gastric residual volume noted. Peripheral perfusion was adequate, with warm extremities, capillary refill time under two seconds, and body temperature of 36.7°C.

On April 2, 2025, laboratory examination revealed a leukocyte count of 15,900/μL and a platelet count of 131,000/μL. Hemoglobin and hematocrit levels were 11.7 g/dL and 35.7%, respectively. The lymphocyte and monocyte counts were 3.7% and 3.0%. Random blood glucose (GDS) was 140 mg/dL. Renal function tests showed a urea level of 49 mg/dL and creatinine of 0.4 mg/dL, with an estimated creatinine clearance (CrCl/eGFR) of 120 mL/min. Serum electrolytes demonstrated sodium 134 mEq/L, potassium 4.4 mEq/L, and chloride 94 mEq/L. Liver enzymes showed SGOT 20 U/L and SGPT 63 U/L.

On April 3, 2025, the leukocyte count was 14,200/μL and platelets decreased to 82,000/μL. Hemoglobin and hematocrit were 12.4 g/dL and 37.7%. Lymphocytes and monocytes were 2.6% and 2.7%. Coagulation parameters showed PT 13.4 / 14.2 seconds, INR 0.96 / 1.06, and APTT 27.1 / 32.1

seconds. Inflammatory markers were elevated, with CRP 48.00 mg/L, procalcitonin (PCT) 19.4 ng/mL, and D-dimer 2.9.

On April 4, 2025, leukocytes increased to 16,000/μL and platelets further decreased to 69,000/μL. Hemoglobin was 12.1 g/dL and hematocrit 36.3%. Lymphocytes and monocytes were 3.7% and 3.0%. Random blood glucose was 126 mg/dL. Urea was 53 mg/dL, creatinine 0.4 mg/dL, and CrCl 120 mL/min. Electrolytes showed sodium 135 mEq/L, potassium 4.2 mEq/L, and chloride 94 mEq/L. Liver enzymes were SGOT 21 U/L and SGPT 73 U/L. Coagulation results showed PT 13.1 / 13.8 seconds, INR 0.95 / 1.02, and APTT 25.9 / 31.9 seconds.

On April 5, 2025, there was marked leukocytosis with leukocytes 26,300/μL and thrombocytopenia with platelets 47,000/μL. Hemoglobin and hematocrit increased to 13.4 g/dL and 41.8%. Band neutrophils were 86.4%, with lymphocytes 7.6% and monocytes 6.8%. Random blood glucose was 157 mg/dL. Urea rose to 68 mg/dL and creatinine to 0.6 mg/dL, with CrCl 105 mL/min. Sodium was 142 mEq/L, potassium 4.7 mEq/L, and chloride 104 mEq/L. SGOT/SGPT were 12/38 U/L. Albumin level was 3.52 g/dL. Total/direct bilirubin was 0.47 / 0.21 mg/dL. Coagulation tests showed PT 20.8 / 14.0 seconds, INR 1.50 / 1.04, and APTT 38.8 / 31.9 seconds. CRP decreased to 24.00 mg/L, while PCT markedly increased to 71.5 ng/mL.

On April 6, 2025, leukocytes remained elevated at 26,350/μL and platelets increased to 77,000/μL. Hemoglobin dropped to 10.5 g/dL and hematocrit to 33.2%. Lymphocytes and monocytes were 8.0% and 3.9%. Random blood glucose was 168 mg/dL. Urea increased further to 84 mg/dL, creatinine was 0.6 mg/dL, and CrCl 105 mL/min. Sodium was 143 mEq/L and potassium 4.7 mEq/L. SGOT/SGPT were 5 / 21 U/L. Albumin was 3.42 g/dL and total bilirubin 0.54 mg/dL. Coagulation parameters showed PT 90.6 / 14.0 seconds, INR 8.65 / 1.04, and APTT 140.0 / 31.9 seconds.

On April 7, 2025, leukocytes were 26,700/μL and platelets 80,000/μL. Hemoglobin was 10.1 g/dL and hematocrit 31.3%. Lymphocytes were 5.6%. Random blood glucose decreased to 120 mg/dL. Urea was 73 mg/dL, creatinine 0.4 mg/dL, and CrCl 120 mL/min. Sodium 136 mEq/L, potassium 4.3 mEq/L, and chloride 102 mEq/L were recorded. SGOT/SGPT were 6 / 11 U/L. Albumin remained 3.42 g/dL, and total/direct bilirubin was 0.54 / 0.28 mg/dL.

On April 8, 2025, leukocytes decreased to 21,800/μL and platelets increased to 101,000/μL. Hemoglobin and hematocrit declined to 9.6 g/dL and 29.1%. Lymphocytes and monocytes were 5.3% and 3.8%. Random blood glucose was 86 mg/dL. Urea was 71 mg/dL, creatinine 0.4 mg/dL, and CrCl 120 mL/min. Sodium 137 mEq/L, potassium 4.4 mEq/L, and chloride 106 mEq/L were noted. SGOT/SGPT were 7 / 12 U/L. Coagulation values were recorded as PT 1.31, INR 1.32, and APTT 0.9.

On April 9, 2025, leukocytes further decreased to 18,300/μL and platelets increased to 138,000/μL. Hemoglobin was 9.8 g/dL and hematocrit 27.3%. Lymphocytes and monocytes were 5.2% and 4.3%. Random blood glucose was 77 mg/dL. Urea decreased to 54 mg/dL, creatinine remained 0.4 mg/dL, and CrCl was 120 mL/min. Sodium increased to 153 mEq/L, potassium was 4.3 mEq/L, and chloride 100 mEq/L. SGOT/SGPT were 10 / 21 U/L. Albumin was 3.37 g/dL, and CRP decreased to 4.8 mg/L. Microbiological culture examination identified Pseudomonas aeruginosa as the causative pathogen.

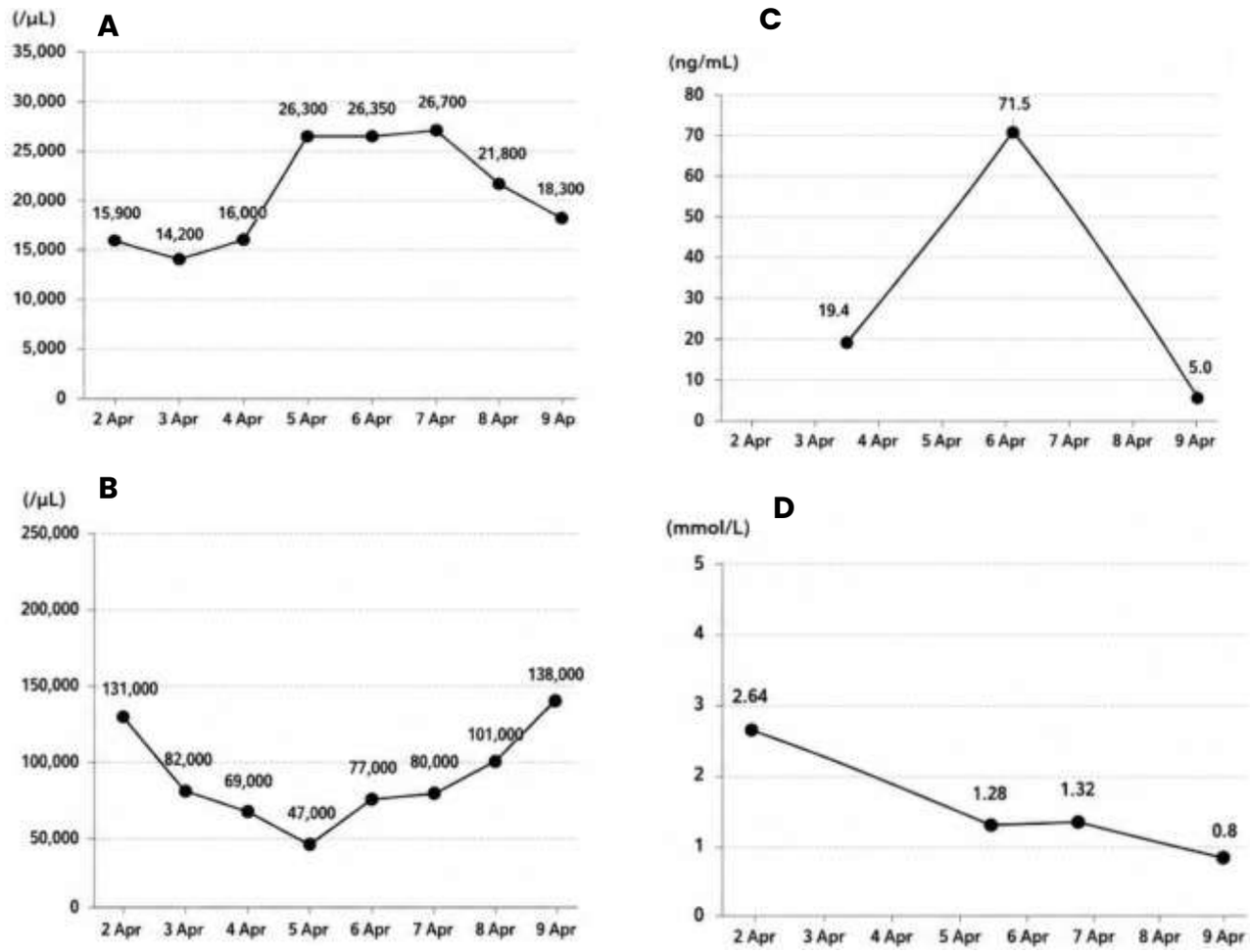

Figure 1. Trend of the patient's laboratory results, A : Leukocyte, B: Thrombocyte, C: Procalcitonin, D: Lactate.

The patient was diagnosed with respiratory failure requiring mechanical ventilation support, myasthenia gravis, suspected hospital-acquired pneumonia, left pleural effusion, anemia with hemoglobin level of 11.9 g/dL, and hypoalbuminemia.

The patient received meropenem 1 g intravenously every 8 hours as the primary broad-spectrum antibiotic regimen, combined with amikacin for additional Gram-negative coverage during the early critical phase. Antifungal therapy with micafungin 100 mg intravenously once daily was administered due to prolonged ICU stay, broad-spectrum antibiotic exposure, and high risk for invasive fungal infection. Previous empiric antibiotics including ceftriaxone, ceftazidime, and cefepime were discontinued to minimize redundant antimicrobial coverage and reduce the risk of antimicrobial resistance and drug-related toxicity.

Additional medications included paracetamol 1 g intravenously every 8 hours as needed for pain and fever control, lansoprazole 30 mg intravenously once daily for stress ulcer prophylaxis, and supportive intravenous fluid therapy consisting of Ringer's lactate and balanced fluids. Enteral medications administered via nasogastric tube included N-acetylcysteine 200 mg every 8 hours, vitamin C 250 mg once daily, vitamin B complex, zinc supplementation, folic acid, pyridostigmine 60 mg every 6 hours for myasthenia gravis management, and prednisone 20 mg every 8 hours. Nutritional support was optimized through enteral feeding with caloric targets adjusted according to body weight and clinical condition, with adequate protein intake and controlled daily fluid administration. Supportive intensive care measures included ventilator bundle implementation, head-of-bed elevation, routine airway suctioning, passive mobilization, glycemic monitoring, catheter care, eye protection, and regular laboratory evaluation including inflammatory markers,

coagulation profile, renal and liver function tests, arterial blood gas analysis, and serial procalcitonin monitoring. Bronchoscopy was planned for airway evaluation and mucus plug management.

Considering the severe septic shock condition with uncontrolled inflammatory response, TPE was initiated as adjunctive therapy for septic shock management. The primary indication for TPE was severe sepsis-associated hyperinflammation accompanied by vasopressor requirement, elevated lactate levels, progressive thrombocytopenia, and markedly elevated procalcitonin levels approaching 100 ng/mL. Although the patient had underlying myasthenia gravis, TPE in this case was primarily directed toward management of septic shock and cytokine-mediated inflammatory dysregulation.

Therapeutic plasma exchange was performed in three cycles, each with a total replacement volume of 2700 mL consisting of 1000 mL of 5% albumin prepared by diluting 25% albumin with normal saline, 500 mL of Gelafusal, and 1200 mL of normal saline.

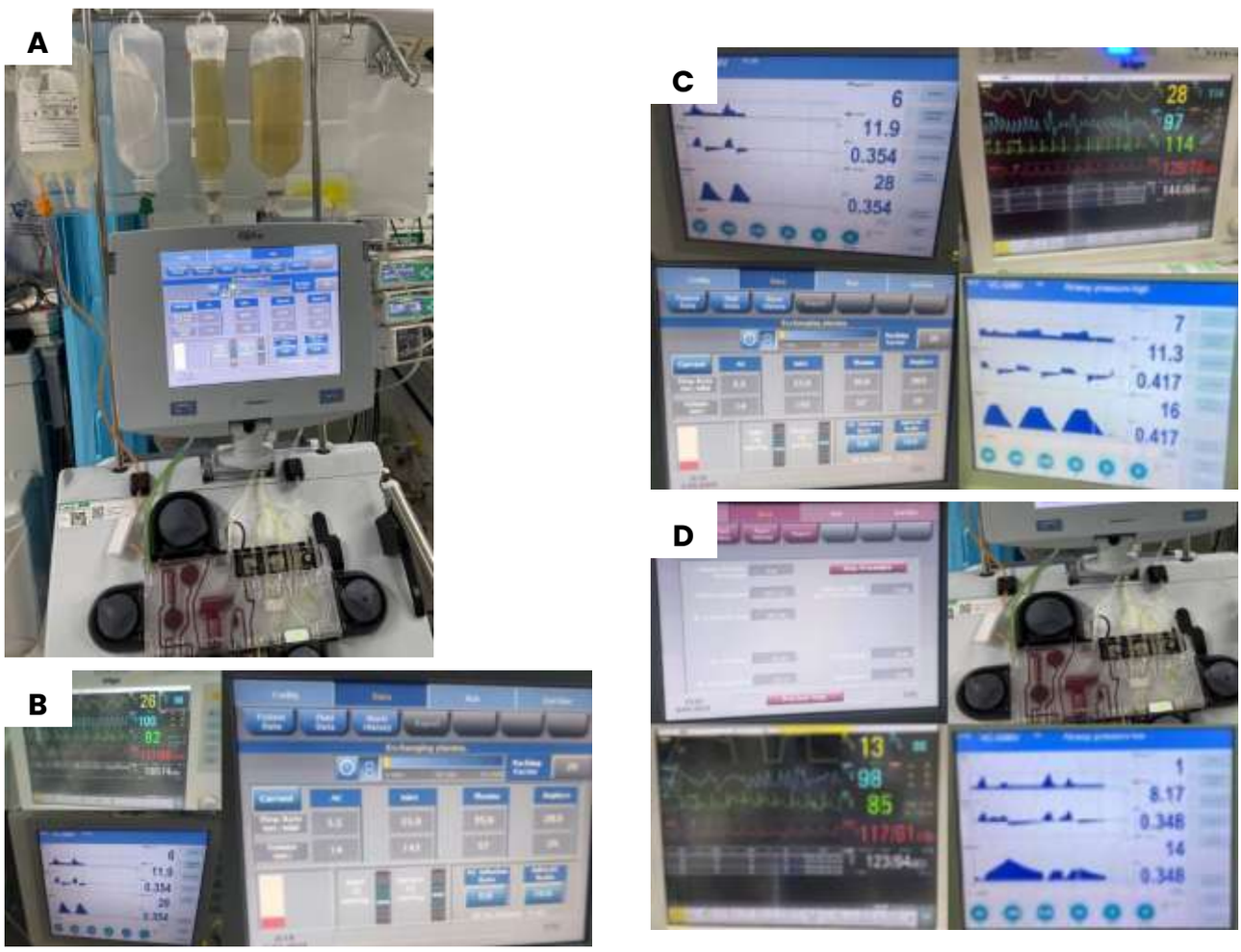

Figure 2. A: Therapeutic plasma exchange, B: Cycle 1, C: Cycle 2, D: Cycle 3

Following three cycles of TPE the patient demonstrated progressive clinical and laboratory improvement. Procalcitonin levels gradually decreased, accompanied by recovery of platelet counts from severe thrombocytopenia. Hemodynamic status also improved significantly, allowing discontinuation of norepinephrine support. In parallel, respiratory function improved with better ventilator tolerance, enabling initiation of the ventilator weaning process. These findings suggested a favorable response to adjunctive TPE therapy in controlling the hyperinflammatory state associated with septic shock.

After 18 days of intensive care management, the patient demonstrated significant clinical improvement. She achieved stable hemodynamics without vasopressor support, laboratory parameters showed improvement, platelet counts increased, and signs of sepsis were resolving. She was subsequently transferred to a lower-dependency unit for continued internal medicine, pulmonary, and rehabilitation follow-up.

Tabel 1. Clinical Timeline of Septic Shock Management and Therapeutic Plasma Exchange

Hospital Day / Date	Clinical Condition & Diagnosis	Hemodynamic / Laboratory Findings	Intervention	Clinical Outcome
Prior to referral (ICU day 1–12 at referring hospital)	Severe respiratory failure secondary to myasthenic crisis with suspected pneumonia	Failed ventilator weaning, progressive dyspnea and generalized weakness	Mechanical ventilation, broad-spectrum antibiotics	Persistent ventilator dependence
ICU admission	Septic shock secondary to pneumonia in patient with myasthenia gravis	Hypotension requiring norepinephrine 0.15 mcg/kg/min, elevated lactate 2.64 mmol/L, leukocytosis, respiratory failure	Vasopressor support, ventilator support, meropenem, micafungin, supportive ICU management	Hemodynamic stabilization partially achieved
April 2–3, 2025	Persistent septic shock with hyperinflammatory state	Leukocytes 15,900→14,200/µL, platelets 131,000→82,000/µL, CRP 48 mg/L, PCT 19.4 ng/mL	Broad-spectrum antimicrobial therapy continued	Persistent inflammation and thrombocytopenia
April 4, 2025	Progressive sepsis-associated inflammatory response	Platelets decreased to 69,000/µL, persistent leukocytosis, increasing organ dysfunction markers	Central venous catheter and arterial line insertion	Ongoing critical condition
April 5, 2025 (before TPE)	Peak hyperinflammatory septic shock	Leukocytes 26,300/µL, platelets 47,000/µL, PCT increased markedly approaching 100 ng/mL, vasopressor requirement persisted	Decision to initiate therapeutic plasma exchange (TPE) as adjunctive therapy for septic shock	Severe inflammatory dysregulation documented
April 5, 2025 – TPE Cycle 1	Septic shock with cytokine storm-like inflammatory response	Persistent thrombocytopenia and elevated inflammatory markers	First TPE cycle performed with total replacement volume 2700 mL	Early hemodynamic improvement
April 6, 2025 – TPE Cycle 2	Ongoing septic shock with improving perfusion	Platelets increased to 77,000/µL, lactate and vasopressor requirement improved	Second TPE cycle performed	Norepinephrine requirement decreased
April 7, 2025 – TPE Cycle 3	Improving septic condition	Platelets increased to 80,000/µL, inflammatory markers gradually decreased	Third TPE cycle performed	Successful vasopressor weaning and improved ventilator tolerance
April 8–9, 2025	Recovery phase	Leukocytes decreased to 21,800→18,300/µL, platelets improved to 101,000→138,000/µL, CRP decreased to 4.8 mg/L	Continued supportive care and antimicrobial therapy	Clinical improvement, successful ventilator weaning process
End of ICU stay	Resolving septic shock	Stable hemodynamics without vasopressors, improving laboratory profile	Transfer to lower-dependency unit	Favorable clinical outcome

Tabel 2. APACHE II Score Assessment

Organ System	Patient Data	SOFA Score
Neurologic (Brain)	GCS E4M6Vt (approximately GCS 10T–11T, intubated without sedation)	**2**
Respiratory	On mechanical ventilation (VC-SIMV) with FiO2 90%, estimated PaO2/FiO2 ratio <150	**3**
Cardiovascular	Required norepinephrine 0.15 mcg/kgBW/min to maintain MAP 84 mmHg	**2**
Liver	Total bilirubin 0.47–0.54 mg/dL	**0**
Renal	Creatinine 0.4–0.6 mg/dL with urine output 0.48–0.58 mL/kg/hour	**1**
Hematologic	Lowest platelet count 47,000/µL	**4**
Total score		**12**

Tabel 3. APACHE II Score Assessment

Parameter	Patient Data	APACHE II Point
Temperature	36.2–36.7°C	**0**
Mean Arterial Pressure (MAP)	Initial septic shock with MAP <70 mmHg before norepinephrine support	**2**
Heart Rate	112 beats/minute	**2**
Respiratory Rate	25 breaths/minute	**1**
Oxygenation	Mechanical ventilation with FiO2 90%, estimated severe oxygenation impairment	**3**
Arterial pH / HCO3	7.498–7.506	**0**
Sodium	134–143 mEq/L	**0**
Potassium	4.2–4.7 mEq/L	**0**
Serum Creatinine	0.4–0.6 mg/dL	**0**
Hematocrit	27.3–41.8%	**0**
Leukocyte Count	Peak 26,700/µL	**2**
Glasgow Coma Scale	GCS E4M6Vt	**5**
Age Score	52 years old	**2**
Total score		**18**

3. Discussion

Sepsis is defined as life-threatening organ dysfunction caused by a dysregulated host response to infection, while septic shock represents a subset of sepsis characterized by profound circulatory and metabolic abnormalities requiring vasopressor support to maintain adequate perfusion. According to the latest Surviving Sepsis Campaign guidelines, septic shock is identified by persistent hypotension requiring vasopressors despite adequate fluid resuscitation, frequently accompanied by elevated lactate levels and increased mortality risk.[1,2] In the present case, the patient fulfilled septic shock criteria due to persistent hypotension with an initial blood pressure of 82/48 mmHg, mean arterial pressure of 59 mmHg, elevated lactate level of 2.64 mmol/L, and continuous norepinephrine requirement despite fluid administration. The patient additionally demonstrated progressive leukocytosis, severe thrombocytopenia, markedly elevated procalcitonin levels, and coagulation abnormalities, indicating severe systemic inflammatory dysregulation secondary to pneumonia-associated sepsis.[1,3]

Pneumonia remains one of the most frequent causes of sepsis in critically ill patients, particularly among individuals requiring prolonged mechanical ventilation. Ventilator-associated pneumonia contributes significantly to ICU morbidity due to impaired airway clearance, bacterial colonization, microaspiration, and prolonged ventilator exposure.[4,5] In this case, the patient had undergone mechanical ventilation for approximately 12 days prior to referral, representing a major risk factor for nosocomial pulmonary infection. Microbiological culture identified Pseudomonas aeruginosa as the causative pathogen, consistent with hospital-acquired pneumonia in critically ill patients. The presence of respiratory failure, mucus plugging requiring bronchoscopy planning, and left pleural effusion further supported severe pulmonary infection as the primary septic source.

Myasthenia gravis (MG) is an autoimmune neuromuscular disorder caused by antibodies directed against acetylcholine receptors at the neuromuscular junction, resulting in fluctuating skeletal muscle weakness and possible respiratory muscle failure. Infection is a well-recognized

precipitating factor for myasthenic crisis.[6,7] In this patient, underlying MG likely contributed to prolonged ventilator dependence and respiratory deterioration during severe pneumonia and septic shock. TPE is an established therapy for myasthenic crisis because it rapidly removes circulating pathogenic autoantibodies.[6] However, in the present case, the primary indication for TPE was directed toward management of severe septic shock with hyperinflammatory features rather than MG alone. TPE was initiated during the peak inflammatory phase characterized by vasopressor dependence, progressive thrombocytopenia, elevated lactate levels, worsening leukocytosis, and markedly elevated procalcitonin approaching 100 ng/mL.

The rationale for TPE in septic shock is based on its immunomodulatory properties. Sepsis involves excessive activation of inflammatory pathways, endothelial dysfunction, complement activation, coagulation disturbances, and microcirculatory impairment.[8,9] TPE may theoretically attenuate this dysregulated response by removing circulating inflammatory mediators, endotoxins, activated complement components, damage-associated molecular patterns (DAMPs), and procoagulant factors. Simultaneously, plasma replacement may replenish protective anticoagulant and endothelial regulatory proteins, including protein C,antithrombin, and ADAMTS13.8-10 These mechanisms may contribute to improved vascular tone, reduced capillary leakage, correction of coagulopathy, and improved tissue perfusion.

In this case, TPE was performed in three consecutive cycles with replacement fluids consisting of albumin, Gelafusal, and normal saline. Following TPE initiation, progressive clinical and laboratory improvement was observed. Platelet counts increased from a nadir of 47,000/μL to 138,000/μL, inflammatory markers gradually decreased, and vasopressor support was successfully discontinued. Procalcitonin levels also demonstrated improvement following completion of TPE cycles. Parallel improvement in respiratory status enabled ventilator weaning initiation, suggesting recovery from both systemic inflammatory dysregulation and respiratory compromise. These findings are consistent with previous reports suggesting that TPE may contribute to hemodynamic stabilization and improvement of inflammatory parameters in selected patients with severe septic shock.[8-11]

The patient additionally demonstrated severe sepsis-associated coagulopathy, characterized by marked prolongation of PT, INR, and APTT during the peak inflammatory phase. Sepsis-associated coagulopathy results from complex interactions between inflammatory cytokines, endothelial injury, platelet activation, and dysregulated thrombin generation, potentially progressing to disseminated intravascular coagulation.[3,12] TPE may theoretically improve this condition through removal of activated coagulation mediators and restoration of physiologic plasma anticoagulant components. In this patient, coagulation abnormalities gradually improved following combined intensive care management and TPE therapy, paralleling improvement in thrombocytopenia and inflammatory biomarkers.

Current sepsis management guidelines emphasize early antimicrobial administration, adequate source control, fluid resuscitation, vasopressor therapy, organ support, and individualized adjunctive therapies.[1,2] Antimicrobial therapy in this case was rationalized following microbiological evaluation. Meropenem was maintained as the primary antipseudomonal agent due to confirmed Pseudomonas aeruginosa infection, while overlapping cephalosporin therapy including ceftriaxone, ceftazidime, and cefepime was discontinued to minimize redundant antimicrobial spectrum, antimicrobial resistance risk, and drug-related toxicity. Amikacin was utilized during the early critical phase for additional Gram-negative coverage, while micafungin was administered because of prolonged ICU stay, prior broad-spectrum antibiotic exposure, and elevated risk for invasive fungal infection. Comprehensive supportive ICU management, including

ventilator bundle implementation, nutritional optimization, glycemic monitoring, airway management, and prevention of ICU-related complications, likely contributed substantially to the patient's recovery.

This case provides clinical insight into the potential dual benefit of TPE in a patient with concomitant myasthenia gravis and septic shock. In addition to its established role in MG crisis, TPE may have contributed to modulation of sepsis-associated hyperinflammation and coagulopathy. Nevertheless, the precise therapeutic contribution of TPE cannot be definitively concluded because the patient simultaneously received broad-spectrum antimicrobial therapy, vasopressor support, mechanical ventilation, nutritional optimization, and comprehensive intensive care management. Therefore, improvement observed in this case likely reflects a multifactorial response to combined therapy rather than TPE alone. Larger prospective studies and randomized controlled trials remain necessary to clarify patient selection, optimal timing, and clinical efficacy of TPE in septic shock management.[8-11]

4. Conclusion

This case highlights the potential role of TPE as adjunctive therapy in septic shock secondary to Pseudomonas aeruginosa pneumonia in a patient with myasthenia gravis. Following three TPE cycles combined with comprehensive intensive care management, the patient demonstrated improvement in inflammatory markers, platelet count, hemodynamic stability, and respiratory function, allowing vasopressor discontinuation and ventilator weaning. Although the exact effect of TPE cannot be definitively determined due to concurrent standard therapy, TPE may offer additional immunomodulatory benefit in selected patients with severe hyperinflammatory septic shock.

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