CASE REPORT

Therapeutic Plasma Exchange (PE) and Non-Invasive Ventilation in *Guillain-Barré Syndrome* (GBS): A Case Report

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ABSTRACT

Background: *Guillain-Barré Syndrome* (GBS) is a progressive acute inflammatory polyradiculopathy caused by immune system dysregulation that carries a high risk of triggering respiratory failure. In its management, plasma exchange (PE) and intravenous immunoglobulin (IVIG) are essential primary therapeutic modalities to eliminate pathological autoantibodies and inhibit disease progression. Although impending respiratory failure in GBS patients has been widely reported and is generally managed with invasive mechanical ventilation, literature discussing the successful use of an alternative approach utilizing non-invasive ventilation (NIV) combined with PE therapy remains highly limited.

Case: We report a case of a 34-year-old male diagnosed with flaccid tetraparesis accompanied by dyspnea, suspected to be GBS. The clinical features, cerebrospinal fluid (CSF) analysis, and electroneuromyography are indicative of GBS. During treatment in the intensive care unit (ICU), the patient received intravenous methylprednisolone therapy, PE, and respiratory support via NIV. The patient underwent intensive care for 13 days. Post-PE, the patient's clinical condition showed significant improvement, allowing for transfer to the general ward.

Discussion: Managing respiratory weakness in GBS demands a delicate balance in ventilatory strategy: weighing the hazards of early invasive intubation against the high risk of aspiration or sudden failure when utilizing non-invasive support. Since the therapeutic benefits of PE are gradual, patients face a precarious window. Consequently, rigorous risk stratification and precisely tailored airway interventions are imperative to safely bridge the patient to recovery.

Conclusion: In this case, the combination of PE therapy and appropriate airway management gradually improved clinical outcomes. A collaborative approach through a multidisciplinary team is essential in planning and executing optimal treatment strategies.

Keywords: critical care; *guillain-barré syndrome*; non-invasive ventilation; plasma exchange; respiratory failure

INTRODUCTION

Guillain-Barré Syndrome (GBS) is an immune-mediated disorder of the peripheral nervous system causing demyelination and secondary axonal damage, which triggers acute polyradiculopathy. Clinically, GBS is characterized by ascending and symmetrical extremity weakness. Often, 1–3 weeks prior to symptom onset, patients have a history of infection, particularly respiratory or gastrointestinal infections.[1] This history of infection likely triggers a mechanism of molecular mimicry, where structural similarities exist between pathogen surface antigens and gangliosides in the peripheral nerves. This provokes a cross-reactive immune response, initiating at the nerve roots, which subsequently causes segmental demyelination. GBS is progressive and can lead to respiratory muscle paralysis and severe respiratory failure. The diagnosis of GBS is based on a combination of clinical manifestations supported by ancillary investigations. Most diagnostic criteria for GBS require a combination of patient history, neurological examination, cerebrospinal fluid (CSF) analysis (specifically cytoalbuminologic dissociation), and electrodiagnostic findings.[2]

GBS is the most common cause of acute flaccid paralysis, with a global incidence of approximately 1–2 per 100,000 people annually. Other studies report similar data, with incidence rates ranging from 0.6–1.9 per 100,000 population, evenly distributed across various countries.[3] Although the syndrome can affect individuals of any age, the incidence increases with age, peaking between 50 and 70 years. Additionally, males have an approximately 1.5 times greater risk of developing GBS compared to females.[4]

In the clinical course of this patient, initial laboratory results revealed leukocytosis suggestive of an underlying infection, prompting early antibiotic therapy. This finding is clinically significant, as approximately two-thirds of GBS cases are triggered by a preceding respiratory or gastrointestinal infection. The systemic inflammatory response, indicated by the elevated leukocyte count, likely contributed to the acute autoimmune phase of the demyelination process observed in this case.

The patient initially presented with dyspnea, accompanied by facial and extremity weakness. Following the administration of targeted therapies such as therapeutic plasma exchange (PE) and NIV, alongside supportive antibiotic treatment, the patient demonstrated progressive clinical improvement. In progressive cases, the average time from initial symptom onset to respiratory muscle paralysis is approximately 12 days. Given its progressive nature and potential fatality due to respiratory failure, rapid, precise, and comprehensive management is required to prevent clinical deterioration. To date, the primary therapeutic modalities for GBS include plasma exchange (PE), also known as plasmapheresis, and intravenous immunoglobulin (IVIG).[5]

Some literature suggests that combining high-dose corticosteroids with PE may inhibit GBS progression and prevent respiratory and bulbar muscle involvement; however, some cases still progress to respiratory failure requiring ventilatory support. While invasive mechanical ventilation is the standard of care for GBS patients developing acute respiratory failure, its use is associated with complications such as ventilator-associated pneumonia (VAP) and

prolonged intensive care unit (ICU) stays. Conversely, the use of NIV in GBS remains controversial and underreported, primarily due to concerns regarding bulbar muscle weakness, impaired airway clearance, and the high risk of aspiration. Currently, there is a lack of established clinical criteria defining the ideal patient profile or the optimal timing for NIV trial in GBS. Furthermore, it remains unclear whether early initiation of PE rapidly halts the progression of bulbar palsy, thereby creating a therapeutic window where NIV can be safely and effectively applied without transitioning to IMV.[6,7] While there are numerous reports regarding the use of invasive mechanical ventilation in GBS patients, case reports specifically discussing the successful use of non-invasive ventilation (NIV) combined with PE remain limited.[8] Therefore, this case report will discuss the management of a GBS patient treated with a combination of PE and NIV.

CASE

A 34-year-old male was referred with a chief complaint of facial paresthesia, accompanied by dysphagia and frequent choking, indicating bulbar involvement. The history of the present illness began with ascending lower limb weakness that progressed to involve all four extremities (tetraparesis) and the left facial nerve. During admission at the previous hospital, the patient's neurological deficits worsened, accompanied by progressive dyspnea, prompting referral to RSUP Dr. Sardjito, Yogyakarta, for further management.

Ten days prior to admission, the patient had a history of acute respiratory tract infection manifesting as cough and cold, which resolved spontaneously without specific treatment. The patient had a history of controlled bronchial asthma, with the last exacerbation occurring approximately 15 years ago. He reported a smoking history of one pack per day for the past 10 years. This represents a concern in the respiratory management of the patient. (Figure 1)

On initial physical examination, the patient appeared to be in respiratory distress with a compos mentis level of consciousness. Vital signs showed a blood pressure of 126/78 mmHg, tachycardia (98 bpm), tachypnea (30 breaths/min), temperature of 36.8°C, and oxygen saturation (SaO2) of 96–98% on 15 liters/minute oxygen via a non-rebreathing mask (NRM). Initial neurological examination revealed flaccid tetraparesis with a motor strength of 4 in all four extremities. Physiological reflexes were normal (+2) in both upper extremities but diminished (+1) in both lower extremities. Other than facial nerve paresis, no additional cranial nerve deficits were found.

The blood gas analysis showed pH 7.425, pO2 50, pCO2 51.5, HCO3 28.1, BE 4, FiO2 0.70, sO2 94, P/F ratio of 100, and a lactate level of 2.73, indicated mixed-type respiratory failure. Complete blood count showed secondary polycythemia (suspected due to hypoxia) and leukocytosis with a left shift. The chest X-ray revealed no abnormalities except for cardiomegaly.

The CSF analysis revealed a cell count of 1 cell/µL, 13% polymorphonuclear (PMN) cells, and 87% mononuclear (MN) cells, with a total protein of 0.057 and glucose of 112. Based on the electroneuromyography examination, the motor conduction study showed prolonged distal latency with normal amplitude and nerve conduction velocity (NCV) in the right and left median nerves as well as the right and left tibial

nerves. Meanwhile, the sensory nerve conduction study revealed prolonged distal latency in the right and left median nerves with normal amplitude and NCV. Based on these findings, combined with supportive clinical features consistent with GBS. (Figure 2)

During 13 days of care in the ICU, the patient received pharmacological therapy including: intravenous methylprednisolone 125 mg every 8 hours, intravenous omeprazole 40 mg every 24 hours, intravenous mecobalamine 500 mcg every 12 hours, intravenous ceftriaxone 1 g every 12 hours, and oral N-acetylcysteine 300 mg every 12 hours. A dialysis catheter was inserted to establish access for PE. Nutrition was provided as a high-calorie, high-protein (HCHP) liquid diet of 250 cc via nasogastric tube (NGT), along with maintenance fluid of 0.9% NaCl at 30 cc/hour.

In this patient, muscle weakness was not highly progressive, and the autonomic system remained intact, as evidenced by controlled blood pressure and heart rate, along with preserved cough and swallowing reflexes. Therefore, the use of NIV could be considered to avert early intubation. Nevertheless, as previously stated, GBS patients receiving NIV require rigorous monitoring and strict risk assessment for mechanical ventilation. This ensures that prompt intubation can be performed in the event of progressive clinical and dysautonomic deterioration, preventing further decline into a more critical condition.

The patient's clinical condition showed gradual improvement during treatment. On day 9, respiratory distress and work of breathing improved significantly, allowing for the weaning of ventilatory support from NIV with mode PS 5, positive end-expiratory pressure (PEEP) 5, FiO2 70-80%, SpO2 95-98%, VTe 5-8 cc/kg BB, respiratory rate 14-22 x/mnt to NRM. On day 13, neurological status improved significantly with the return of motor strength to grade 5 (normal) in all extremities. The patient's GBS disability score showed significant improvement, decreasing from grade 5 at the time of admission to grade 2 by day 13 post-PE therapy, thereby facilitating their transfer from the ICU to a regular ward for further recovery.

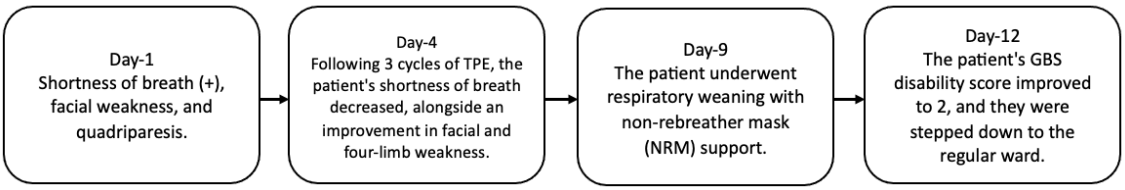

Figure 1. Patient's clinical timeline

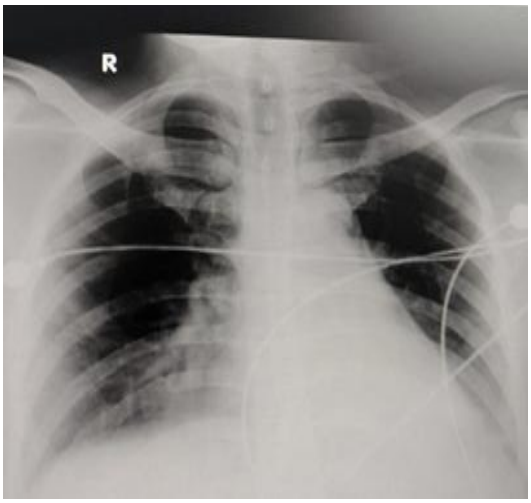

Figure 2. Posteroanterior projection thoracic X-ray examination results

DISCUSSION

Guillain-Barré Syndrome (GBS) is an acute inflammatory polyradiculopathy generally triggered by an antecedent infection. In this case, the patient was a 34-year-old male presenting with ascending tetraparesis following a respiratory tract infection 10 days prior. This finding is consistent with the literature stating that the incidence of GBS is higher in males and is frequently preceded by respiratory or gastrointestinal infections that trigger a mechanism of molecular mimicry. The diagnosis in this patient was established based on classical clinical manifestations, including symmetrical progressive weakness, hyporeflexia, and autonomic dysfunction, supported by ancillary investigations to exclude differential diagnoses.[1,9]

Approximately 20–30% of GBS patients develop severe respiratory failure requiring mechanical ventilation.[10] This critical complication primarily stems from the rapid progression of ascending neuromuscular paralysis, which eventually compromises the diaphragmatic and intercostal muscles. Furthermore, concurrent bulbar muscle weakness impairs the swallowing mechanism and cough reflex, leading to an inability to clear airway secretions effectively and increasing the risk of aspiration.[7] In the present case, the patient exhibited these high-risk clinical features, presenting with progressive respiratory distress and bulbar involvement. Such a condition requires an immediate and tailored respiratory assessment, accompanied by the urgent initiation of immunotherapy to halt further neurological decline.

The Modified Erasmus GBS Respiratory Insufficiency Score (mEGRIS) is frequently utilized to predict the risk of mechanical ventilation, with rapid disease progression and bulbar involvement identified as major risk factors.[11,12] Generally, the routine use of NIV is not recommended in the management of GBS. This is primarily due to two interrelated clinical challenges. First, GBS often presents with bulbar dysfunction, which compromises the airway and significantly increases the risk of aspiration. Second, neuromuscular weakness leads to an inability to effectively clear respiratory secretions. Consequently, these factors make standard NIV application hazardous; however, NIV may still be considered in highly selected cases under strict

monitoring.[6,7,13] However, NIV could still be considered in this patient due to the relatively non-progressive muscle weakness and an intact autonomic system, indicated by controlled blood pressure and heart rate, along with well-preserved cough and swallowing reflexes, thereby minimizing the risk of aspiration.

The decision to utilize NIV in pressure support (PS) mode with a PEEP of 10 cmH2O proved effective in maintaining oxygenation and preventing endotracheal intubation. The patient was successfully weaned from NIV to NRM on the 10th day of treatment without complications such as aspiration or respiratory arrest.[14,15] This success highlights that NIV may be considered in selected GBS patients under close monitoring in the ICU.

Intubation with mechanical ventilation is initiated in the presence of overt clinical signs of respiratory distress, significant bulbar muscle involvement (such as dysphagia, absent gag reflex, and impaired airway clearance), or severe, life-threatening dysautonomia. Conversely, the routine use of NIV is not recommended and remains a subject of considerable debate. Nevertheless, an NIV trial may be considered in a highly selected subset of patients under strict monitoring in an intensive or high-care unit (ICU/HCU), specifically those without bulbar muscle weakness who are hemodynamically stable, fully cooperative, and maintain a normal level of consciousness (compos mentis).

The primary management of GBS aims to eliminate pathogenic autoantibodies and limit nerve damage. In this case, the patient underwent therapeutic PE for three cycles, with an average volume of 1,800–1,900 cc per cycle. Each cycle of therapeutic PE resulted in progressive clinical improvement for the patient. Following the first therapeutic PE cycle, there was an improvement in the patient's respiratory status and muscle strength, accompanied by a decrease in the work of breathing. After the second cycle, the patient showed increased motor strength. Following the third cycle, respiratory function further improved, allowing for the initiation of weaning by alternating between NIV and a high-flow nasal cannula (HFNC).

In this patient, the Hughes Disability Scale score was 3, despite the absence of autonomic dysfunction, this constituted a strong indication for the immediate initiation of therapeutic PE. Furthermore, the patient's symptom onset was within 14 days, thereby fulfilling the optimal criteria for commencing therapeutic PE.[16] Therapeutic PE has been proven to accelerate motor recovery if initiated within 4 weeks of symptom onset, with effectiveness comparable to IVIG.[17] The selection of therapeutic PE in this patient was based on resource availability and cost-effectiveness. In addition to therapeutic PE, the patient also received intravenous corticosteroids (methylprednisolone). While current scientific evidence has yet to support the efficacy of corticosteroid monotherapy in GBS patients, clinical observation in this case reveals a contrasting success profile when integrated with therapeutic PE. The administration of corticosteroids as an adjunctive therapy demonstrated favorable clinical outcomes alongside a robust safety profile. This outcome underscores the necessity of differentiating between the limitations of standalone corticosteroid use and its potential as a combination therapy in managing symptomatic GBS.[18,19]

The clinical course of GBS encompasses progressive, plateau, and recovery phases. This patient exhibited significant improvement post-therapeutic PE and intensive supportive care, characterized by the restoration of motor strength to normal (grade 5) on day 13 and improvement in respiratory function. This is consistent with data indicating that 60–80% of GBS patients achieve independent ambulation within 6 months.[3] Although the risk of long-term sequelae such as fatigue or persistent paresthesia remains, early intervention with therapeutic PE and appropriate airway management—including closely monitored NIV—plays a crucial role in preventing mortality and accelerating patient recovery.

CONCLUSION

This case report underscores the successful comprehensive management of severe GBS complicated by impending respiratory failure and bulbar involvement. In this instance, the combination of early-initiated PE and NIV was highly effective in ameliorating neurological deficits and averting invasive intubation. This success was facilitated by the mildly progressive nature of the muscle weakness and the preservation of adequate swallow and cough reflexes, thereby mitigating the need for intubation. Ultimately, successful clinical outcomes are highly dependent on swift diagnosis, precise risk stratification for respiratory failure, close observation of muscle weakness progression, rigorous ICU monitoring, and timely immunotherapeutic intervention to minimize morbidity and mortality. However, further studies are still required to determine the success of NIV compared to invasive ventilation, alongside rigorous clinical assessment, as inaccurate evaluation may increase the risk of poor patient outcomes.

REFERENCE

1. Guo J, Pang X. Guillain-Barré syndrome. Chinese Journal of Neurology. 2023 Aug 1;56(8):924–31. doi:10.3760/cma.j.cn113694-20230309-00171
2. Busl KM, Fried H, Muehlschlegel S, Wartenberg KE, Rajajee V, Alexander SA, et al. Guidelines for Neuroprognostication in Adults with Guillain–Barré Syndrome. Neurocrit Care. 2023;38(3). doi:10.1007/s12028-023-01707-3
3. van Doorn PA, Van den Bergh PYK, Hadden RDM, Avau B, Vankrunkelsven P, Attarian S, et al. European Academy of Neurology/Peripheral Nerve Society Guideline on diagnosis and treatment of Guillain–Barré syndrome. European Journal of Neurology. 2023. doi:10.1111/ene.16073
4. Manfroi B, Dalari LA. Síndrome de Guillain-Barré / Guillain-Barré Syndrome. Brazilian Journal of Health Review. 2022;5(1). doi:10.34119/bjhrv5n1-229
5. Leonhard SE, Mandarakas MR, Gondim FDAA, Bateman K, Ferreira MLB, Cornblath DR, et al. Evidence based guidelines. Diagnosis and management of guillain-barré syndrome in ten steps. Medicina (B Aires). 2021;81(5).
6. Dang HH, Nguyen KV, Le ST, Vu TT, Le VTY, Nguyen TB, et al. Prognostic factors of mechanical ventilation in Guillain–Barré syndrome among adults and children in Vietnam. Clin Neurophysiol Pract. 2025;10. doi:10.1016/j.cnp.2025.02.009
7. Melone MA, Heming N, Meng P, Mompoint D, Aboab J, Clair B, et al. Early mechanical ventilation in patients with Guillain-Barré syndrome at high risk of respiratory

failure: a randomized trial. Ann Intensive Care. 2020;10(1). doi:10.1186/s13613-020-00742-z
8. Jufan AY, Sudadi, Sunantara IGNPM. MANAJEMEN PADA PASIEN SINDROMA GUILLAIN-BARRÉ DI ICU RSUP DR. SARDJITO. Jurnal Komplikasi Anestesi. 2023;9(2). doi:10.22146/jka.v9i2.8347
9. Rabello F de APCJ, Pupe CCB, Jurno ME, Monteiro Júnior RS, Carneiro LSR de SF, Nascimento OJM. Diagnostic accuracy and mechanical ventilation risk in Guillain-Barré syndrome. Revista Neurociências. 2022;30. doi:10.34024/rnc.2022.v30.13973
10. Rodríguez-Méndez AA, Briseño-Ramírez J, Rivas-Ruvalcaba FJ, Solis-Estrada J, Alcázar-García LB, Díaz-Ramírez K, et al. Clinical predictors for mechanical ventilation assistance in Guillain-Barré syndrome. Frontiers in Neurology . 2024;15. doi:10.3389/fneur.2024.1385945
11. Van den Bergh PYK, van Doorn PA, Hadden RDM, Avau B, Vankrunkelsven P, Allen JA, et al. European Academy of Neurology/Peripheral Nerve Society guideline on diagnosis and treatment of chronic inflammatory demyelinating polyradiculoneuropathy: Report of a joint Task Force—Second revision. Journal of the Peripheral Nervous System. 2021;26(3). doi:10.1111/jns.12455
12. Luijten LWG, Doets AY, Arends S, Dimachkie MM, Gorson KC, Islam B, et al. Modified Erasmus GBS Respiratory Insufficiency Score: A simplified clinical tool to predict the risk of mechanical ventilation in Guillain-Barré syndrome. J Neurol Neurosurg Psychiatry. 2023;94(4). doi:10.1136/jnnp-2022-329937
13. Watson V, Ghishan S, Avalishvili T, Zeleke S, Tigabe S, White A, et al. Exploring Extubation Readiness in Guillain-Barré Syndrome: A Case Report and Literature Review. J Investig Med High Impact Case Rep. 2025;13. doi:10.1177/23247096251331859
14. Lund K, Grime C, Spinty S. Neuromuscular disease and respiratory failure. ERS Monograph. 2021;2021(92). doi:10.1183/2312508X.10014620
15. Galassi G, Marchioni A. Acute neuromuscular syndromes with respiratory failure during COVID-19 pandemic: Where we stand and challenges ahead. Journal of Clinical Neuroscience. 2022. doi:10.1016/j.jocn.2022.03.048
16. Hughes RAC, Cornblath DR, Jacobs BC, van Doorn PA. Guillain-Barré Syndrome Disability Scale. Journal of the Peripheral Nervous System. 2025. doi:10.1111/jns.70064
17. Verboon C, Doets AY, Galassi G, Davidson A, Waheed W, Péréon Y, et al. Current treatment practice of Guillain-Barré syndrome. Neurology. 2019;93(1). doi:10.1212/WNL.0000000000007719
18. Shang P, Feng J, Wu W, Zhang HL. Intensive Care and Treatment of Severe Guillain–Barré Syndrome. Frontiers in Pharmacology. 2021. doi:10.3389/fphar.2021.608130
19. Shahrizaila N, Lehmann HC, Kuwabara S. Guillain-Barré syndrome. The Lancet. Elsevier B.V.; 2021. p. 1214–28. doi:10.1016/S0140-6736(21)00517-1 PubMed PMID: 33647239