# Autonomic Dysfunction Progression in a Guillain-Barré Syndrome Patient with Suspected Sepsis: A Case Report

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**Abstract**

Guillain-Barré Syndrome (GBS) is an autoimmune disease affecting the peripheral nervous system, potentially leading to progressive muscle weakness, respiratory failure, and autonomic dysfunction. Severe autonomic dysfunction may result in arrhythmias, blood pressure fluctuations, and vital organ dysfunction, often worsened by sepsis. This report adopted a case report approach of a patient with GBS and severe autonomic dysfunction accompanied by suspected sepsis. Data were thoroughly collected through direct observation, comprehensive review of medical records, and detailed analysis of laboratory, electrocardiogram (ECG), and radiological findings. Analysis was conducted using both clinical and theoretical frameworks. This case report aims to describe autonomic symptom changes in GBS with suspected sepsis and to analyze their clinical implications for nursing care in critically ill patients A 51-year-old male patient presented with progressive extremity weakness, respiratory failure, and required mechanical ventilation. During intensive care, the patient developed second-degree AV block, sinus tachycardia, hypotension, persistent fever, and leukocytosis. These manifestations indicate severe autonomic dysfunction affecting both sympathetic and parasympathetic control on patient. Interventions included mechanical ventilation, intravenous immunoglobulin (IVIg), vasopressors, broad-spectrum antibiotics, and haemodynamic monitoring. The patient's autonomic symptom changes reflected dysfunction of both sympathetic and parasympathetic nervous systems, as indicated by cardiac rhythm disturbances and blood pressure instability during intensive care. AV block indicated sinoatrial node involvement, while sinus tachycardia suggested sympathetic dominance during sepsis. Haemodynamic instability, observed through fluctuating heart rate and hypotension requiring vasopressor support, illustrated the complex interaction between GBS and sepsis. This case highlights that severe autonomic dysfunction in GBS may lead to AV block and sinus tachycardia, aggravated by sepsis. This case suggests that nurses caring for patients with GBS should maintain a high level of vigilance for autonomic instability including arrhythmias and blood pressure changes, especially in the presence of sepsis. Intensive management and continuous monitoring are critical to stabilize autonomic function and improve clinical outcomes in critically ill patients.

**Keywords:** Autonomic Dysfunction, Guillain-Barré Syndrome, Haemodynamics, Intensive Care, Non-Invasive Monitoring.

## Introduction

Guillain-Barré Syndrome (GBS) is a rare autoimmune condition characterised by sudden, progressive paralysis, usually starting with distal limb weakness and progressing to other parts of the body. GBS occurs when the body's immune system mistakenly attacks the peripheral nervous system, impairing motor and sensory function. GBS can affect nerves that play a role in muscle movement as well as nerves that transmit sensations, such as pain, temperature, and touch, which can lead to muscle weakness, loss of sensation in the legs and arms, and impaired swallowing or breathing (WHO, 2023). Overall, GBS is a potentially life-threatening neurological disorder that can cause significant functional impairment and require intensive medical care. Understanding the burden of this condition is therefore essential, which can be further illustrated through epidemiological data across different populations.

Epidemiologically, GBS incidence rates vary geographically and increase with age. In South Korea, the incidence increased from 1.28 to 1.82 cases per 100,000 population between 2010 and 2016, while in China it was recorded at 0.698 cases per 100,000 person-years, with the highest incidence in the adult population (Kim et al., 2021; Zheng et al., 2022). In Indonesia, the prevalence of GBS has not been documented nationally but as of 2010-2014 at Cipto Mangunkusumo Hospital (RSCM) Jakarta, an average of 7.6 cases per year was recorded (Pranata, 2020). In general, the incidence of GBS tends to increase with age, with an increase in risk of approximately 20% for every 10-year increase in age (Mirian et al., 2021).

GBS is primarily characterized by an immune-mediated attack on peripheral nerves, resulting in typical triggering factors and progressive neuromuscular manifestations. The primary mechanism of GBS is an autoimmune reaction attacking the myelin or axolema of peripheral nerves that is often preceded by gastrointestinal infection, respiratory infection, vaccination, or other non-infectious conditions. Among infectious agents, Campylobacter jejuni is known to be the most common trigger, especially in one third of GBS cases (Finsterer, 2022). However, in the majority of patients, the exact cause of GBS remains unclear. Early symptoms often reported include fever, weakness in the extremities, and rapidly progressing paresthesias. The clinical hallmark of GBS is muscle weakness that is symmetrical and progressive from the lower to the upper extremities, accompanied by a decrease or loss of tendon reflexes (Florian et al., 2021). Other frequent manifestations include impaired walking ability, imbalance, pain, and serious complications such as respiratory distress (Ghazavi et al., 2020).

A common serious complication in GBS patients is autonomic dysfunction, a disorder of the autonomic nervous system that regulates vital body functions. Autonomic dysfunction occurs in approximately 41-60% of cases and in some studies has even been reported in up to 95% of patients (Graham et al., 2023). Clinical manifestations include blood pressure fluctuations, bradycardia or tachycardia, body temperature disturbances, bladder dysfunction, and gastrointestinal motility disorders. In severe cases, AV block may occur which requires intervention such as pacemaker insertion (Abbas & Sardar, 2021). Autonomic dysfunction is also a predictor of the need for mechanical ventilation, intensive care, and delayed functional recovery (Bazán-Rodríguez et al., 2023).

On the other hand, sepsis is a systemic infectious complication that can worsen the condition of GBS patients. Sepsis can develop in GBS patients due to various factors, including prolonged hospitalisation, use of mechanical ventilators, and catheter-related infections (Nagayama et al., 2023). In rare cases, GBS may present after septic shock, as reported in a patient with amitriptyline overdose and Klebsiella pneumoniae infection (B. Zhang et al., 2022).

In this case, a 51-year-old male patient with GBS developed respiratory failure and severe autonomic dysfunction. During intensive care, the patient showed fluctuations in blood pressure. Follow-up examination showed leucocytosis and the patient had grade II AV block and suspected Sinus Node Dysfunction indicating suspected sepsis and autonomic nerve disorders. The patient was mechanically ventilated and treated with antibiotics and intravenous immunoglobulin

(IVIg).

The clinical management of GBS complicated by autonomic dysfunction and suspected sepsis requires a rapid and coordinated approach, especially in terms of haemodynamic monitoring and appropriate nursing management. Non-invasive monitoring of vital parameters such as blood pressure, cardiac frequency, respiration and oxygen saturation is crucial to detect early changes and evaluate therapeutic response (Hamzaoui & Boissier, 2023). Techniques such as perfusion index measurement, pulse pressure, and continuous cardiac frequency monitoring allow for more precise therapeutic decision making.

Management of the condition also includes pharmacological support such as IVIg to suppress the autoimmune response, vasopressors to maintain perfusion, and broad- spectrum antibiotics to manage the bacterial infection underlying sepsis. The combination of these interventions is important in managing the multisystem dysfunction that occurs. From a nursing perspective, care is centered on continuous cardiac and haemodynamic monitoring, early recognition of autonomic instability such as arrhythmias and blood pressure fluctuations, and timely reporting of clinical deterioration to the multidisciplinary team. Nurses also play a critical role in preventing sepsis-related complications through infection surveillance, temperature control, and supportive care during prolonged mechanical ventilation. The complexity and rapid progression of autonomic dysfunction in GBS require prompt and coordinated multidisciplinary responses, with nursing vigilance being essential to adapting care to dynamic clinical changes. The urgency of managing such cases lies in multidisciplinary involvement and speed in responding to the patient's clinical dynamics. Unfortunately, there is limited understanding of the complex interactions between GBS, autonomic dysfunction and sepsis, especially in the context of critical care nursing. A systematic approach through actual case studies can be an important basis for strengthening evidence-based clinical practice.

Severe autonomic dysfunction in GBS presents significant challenges for nursing management in critical care settings, particularly when complicated by suspected sepsis, yet practical nursing-focused evidence remains limited. This paper aims to explore factors contributing to altered autonomic symptoms in patients with GBS accompanied by suspected sepsis and to clinically analyze the emergence of sinus node dysfunction, including grade II AV block and sinus tachycardia, in the context of severe autonomic involvement. Through this case report, we seek to highlight clinical observations that may inform nursing surveillance, monitoring, and supportive care strategies for critically ill patients with GBS and systemic infectious complications.

## Methods

This paper uses a case report approach that aims to explore in depth the clinical and nursing dynamics of a patient with GBS who developed acute respiratory failure and showed symptoms of severe autonomic dysfunction with suspected sepsis. This case occurred in a male patient who was admitted to the Intensive Care Unit of a hospital in West Java in January 2025. Clinical data were collected over a 10-day ICU stay. This study covers the stages during treatment in the ICU, the course of the disease, and follow-up therapy carried out during the intensive care period. Nursing assessments were conducted systematically as part of routine intensive care practice and focus on neurological status, cardiovascular stability, respiratory function, and signs of infection. Autonomic and haemodynamic parameters monitored included heart rate, cardiac rhythm (via continuous ECG monitoring), blood pressure, body temperature, oxygen saturation, and vasopressor requirements. Changes in these parameters were documented daily through nursing notes, vital sign charts, and medical progress records.

Data collection was carried out through several methods, namely direct observation of the patient's condition, interviews with the family, nursing assessment, and secondary data in the form of supporting examination results such as laboratory, ECG, radiology, and blood gas analysis. Clinical data were also obtained from medical progress notes

and nursing interventions recorded in the patient's medical record. The collected data were arranged chronologically to build a complete clinical narrative.

The patient's autonomic symptoms, their association with systemic infection, and response to interventions were analysed using a descriptive and interpretative case analysis. The case review was conducted by comparing the patient's clinical findings to current theories and scientific literature on the pathophysiology and management of GBS, autonomic dysfunction, and sepsis. In addition, the nurse's role in haemodynamic monitoring and supportive therapy was analysed as part of a holistic nursing care approach.

This case report has received informed consent from the patient's family and in the writing process the researcher upholds ethical principles, especially related to patient autonomy and the principle of beneficence. The patient's identity was preserved through anonymisation and all data used solely for scientific purposes and the development of clinical knowledge.

## Case Description

A 51-year-old male patient, Mr. A was admitted to the GICU of one of the designated referral hospital in West Java with a diagnosis of Guillain-Barré Syndrome (GBS). The patient presented with a chief complaint of weakness in all four extremities. The complaint had been experienced since six days before admission (SMRS) and began with high fever. On the third day after the onset of fever, the patient began to experience sudden weakness in both lower limbs accompanied by the inability to lift the limbs. Initially the patient was still able to crawl when getting off the vehicle but then the weakness showed significant progressivity and spread to both upper arms. The weakness that the patient felt caused the patient to be unable to walk accompanied by paresthesias felt in all extremities, especially at the tips of the fingers. There was no history of systemic diseases such as hypertension or diabetes mellitus but the patient was known to have a history of active smoking since a young age.

Nursing interventions are tailored to the patient's progressive motor weakness. Routine neurological assessments are performed to monitor muscle strength, symmetry, and functional changes. Due to severe limb weakness and immobility, nurses provide total assistance with activities of daily living, schedule repositioning to prevent pressure sores, and use careful positioning to optimize respiratory mechanics. Airway protection and secretion management are prioritized through suctioning and ventilator monitoring. These interventions aim to maintain patient safety and prevent secondary complications associated with severe neuromuscular weakness. Nursing interventions are also primarily directed at maintaining haemodynamic stability and patient safety during periods of autonomic fluctuations. This includes continuous ECG monitoring to identify arrhythmias, frequent evaluation of mean arterial pressure to support perfusion, strict temperature monitoring, and close observation of fluid balance and urine output.

The patient received a diagnosis of GBS and developed acute respiratory failure so that a mechanical ventilator (P-SIMV mode) was installed through a 7.5 mm endotracheal tube at a depth of 21 cm. Based on medical records, in early January 2025 the patient had received IVIg therapy for three cycles but had not improved. During treatment in the GICU, on 20 January 2025 the patient showed signs of haemodynamic instability, with the lowest systolic blood pressure reaching 78 mmHg, diastolic blood pressure 51 mmHg, and the lowest Mean Arterial Pressure (MAP) value at 61 mmHg. Vital sign monitoring showed haemodynamic instability with fluctuations in blood pressure, high fever, and tachycardia. The following table summarises the vital examination results during the observation period.

**Table 1. Monitoring the patient's Vital Signs on 20th January 2025 Monitoring**

| Monitoring Time | 15.00 | 16.00 | 17.00 | 18.00 | 19.00 | 20.00 | 21.00 |
|---|---|---|---|---|---|---|---|
| NIBP | 105/60 | 78/52 | 79/59 | 80/51 | 116/70 | 98/67 | 130/61 |
| MAP | 75 | 61 | 66 | 61 | 85 | 77 | 84 |
| HR | 126 | 125 | 128 | 125 | 123 | 138 | 124 |
| RR | 29 | 28 | 24 | 23 | 16 | 24 | 21 |
| Temperature | 38,8 | 38,5 | 38,4 | 38,2 | 38,2 | 38,2 | 38,3 |
| SpO2 | 98 | 98 | 100 | 100 | 99 | 100 | 100 |

**Figure 1: Patient Haemodynamic Monitoring Chart**

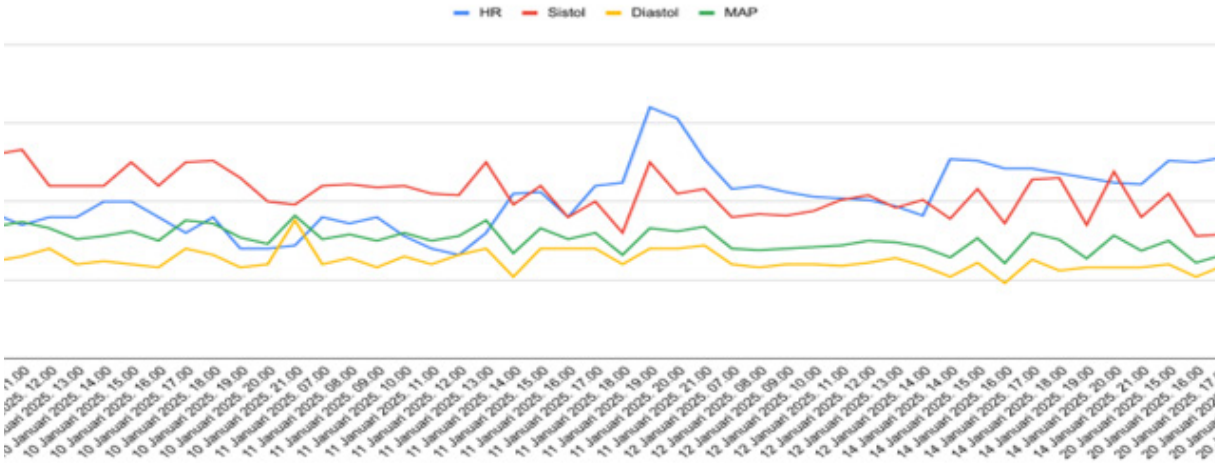



**Figure 2: Temperature, RR and Oxygen Saturation Monitoring Chart of The Patient**

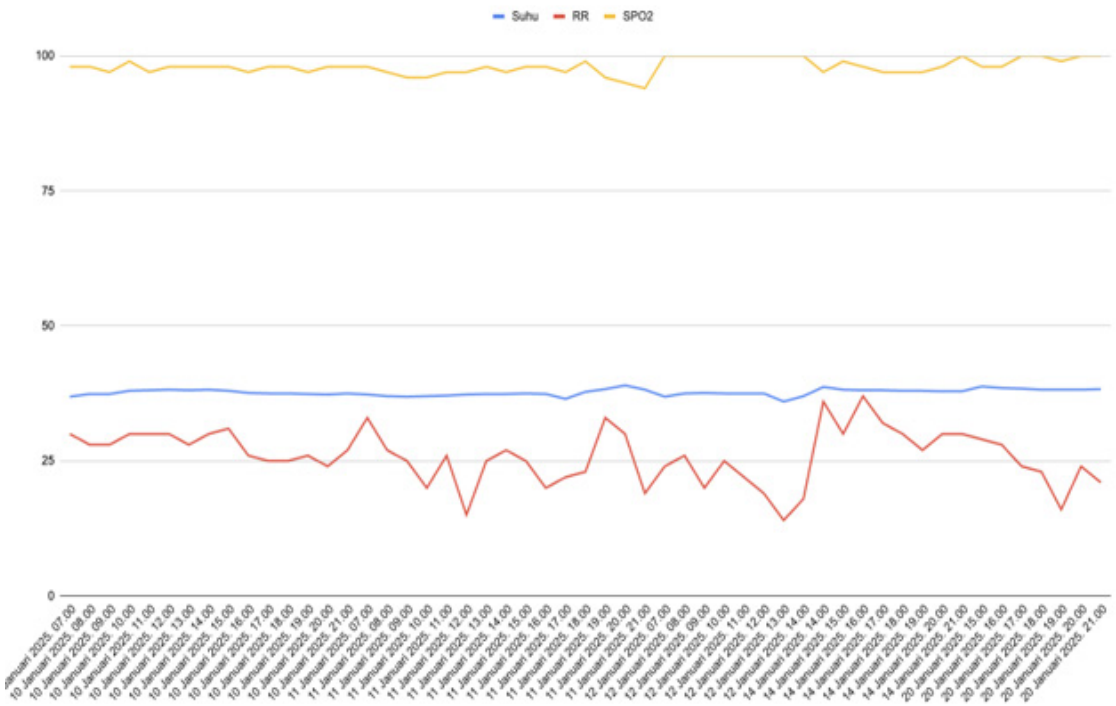
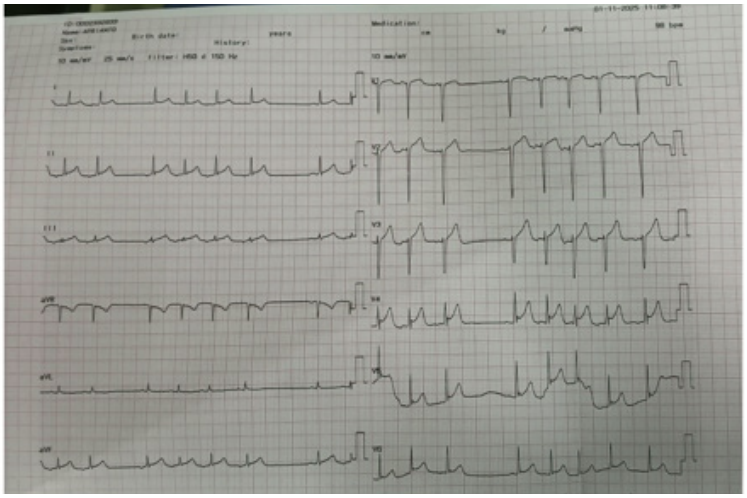


**Figure 3: ECG results on 11 January 2025**



Interpretation of the patient's ECG results showed that the patient had Grade II AV Block and suspected Sinus Node Dysfunction. This could be a clinical manifestation of severe autonomic dysfunction often encountered in the acute phase of GBS and could potentially compromise the patient's haemodynamics.

Laboratory examination revealed an active inflammatory process, impaired perfusion, and decreased nutritional status. The patient had haemodynamic and metabolic disturbances, including anaemia (low haemoglobin and haematocrit), leucocytosis suggestive of an infectious or inflammatory process, and some disturbances in acid-base status (high pCO2, HCO3, tCO2 and BE-b). The following table presents the patient's laboratory results:

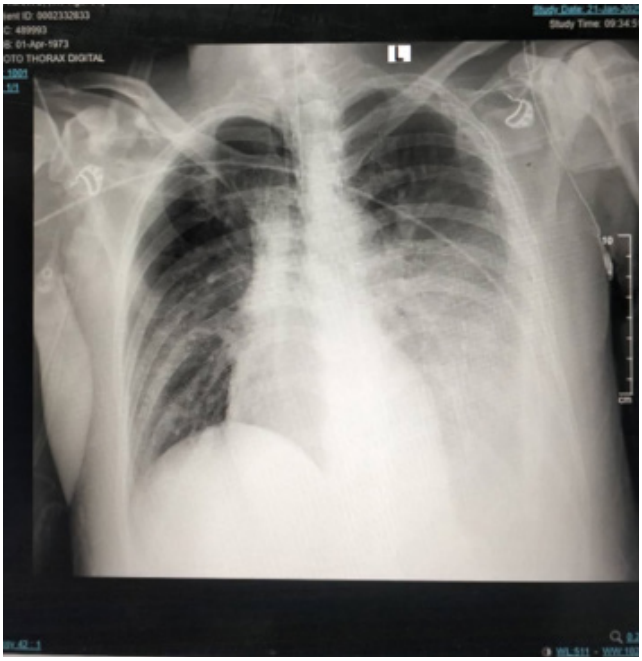
**Table 2. Results of the patient's laboratory examination on 20th January 2025**

| Pemeriksaan | Hasil | Nilai Normal | Keterangan |
|---|---|---|---|
| **Haematology 10 Parameters** | | | |
| **Haemoglobin** | **7.7 g/dl** | **12,3-15,3 g/dl** | **Low** |
| **Hematocrit** | **23%** | **36,0-45.0%** | **low** |
| **Leukocytes** | **37.000/mm3** | **4.400-11.300/mm3** | **High** |
| **Erythrocyites** | **2,5 juta/uL** | **4,5-5,1 juta/uL** | **Low** |
| **Trombocytes** | **357 ribu/uL** | **150-450 ribu/uL** | **Normal** |
| **Index Eritrocyites** | | | |
| **MCV** | **92 fL** | **80-96 fL** | **Normal** |
| **MCH** | **30,8 pg** | **27,5-33,2 pg** | **Normal** |
| **MCHC** | **33,5%** | **33,4-35,5%** | **Normal** |
| **RDW-CV** | **14,4%** | **11,5-14,5%** | **Normal** |
| **RDW-5D** | **47,6 fL** | **36,4-46,3 fL** | **High** |
| **Albumin** | **1,82 gr/dl** | **3,5-5,0 gr/dl** | **Low** |
| **Blood Gas Analysis** | | | |
| **pH** | **7,418** | **7.35 – 7.45** | **Normal** |
| **pCO2** | **49,2 mmHg** | **35.0 – 45.0 mmHg** | **High** |
| **pO2** | **142 mmHg** | **80 – 105 mmHg** | **High** |
| **Acid Base Status** | | | |
| **HCO3** | **32,1 mmol/L** | **22 – 26 mmol/L** | **High** |

| | | | |
|---|---|---|---|
| **tCO2** | **33,6 mmol/L** | **23.05 – 27.35 mmol/L** | **High** |
| **Standard BE-b** | **7,3 mmol/L** | **(-2) – (+2)** | **High** |
| **O2 Saturation** | **99,3 %** | **95 – 100 %** | **Normal** |

Radiological examination of the chest X-ray on 21th January 2025 showed bilateral pneumonia and left pleural effusion. In addition, scrotal ultrasound showed bilateral thickening leading to the diagnosis of scrotal oedema, with no structural abnormalities of the testes.

**Figure 4: Radiological Results, Thorax X-ray on 21 January 2025**



The clinical and nursing interventions provided were collaborative and intensive. The patient was mechanically ventilated with P-SIMV mode, $FiO_2$ to 90%, tidal volume 480 mL, and PEEP 8 $cmH_2O$. Broad-spectrum antibiotics-ampicillin, vancomycin, and levofloxacin-were administered intravenously to manage systemic infection. Haemodynamic support was provided through the administration of norepinephrine (0.8 mcg/kg/BB/min) and dobutamine (5 mcg/kgBB/min). For temperature control and sedation, the patient received paracetamol (4x500 mg) and dexmedetomidine infusion (0.2 mcg/kg/hour). Respiratory therapy support was performed through the administration of combivent, pulmicort, and acetylcysteine by inhalation. The patient was also given periodic closed airway suction, monitoring of vital parameters, monitoring of fluid balance, and continuous monitoring of haemodynamic status. Family education was also provided according to holistic nursing principles in the intensive care unit.

This case highlights how sepsis may exacerbate autonomic dysregulation in GBS, increasing the risk of sudden cardiovascular deterioration. From a critical care nursing perspective, this case is unique in demonstrating the pivotal role of continuous nursing assessment and haemodynamic monitoring in the early identification of autonomic instability

## Discussion

This case demonstrates a severe and fluctuating pattern of autonomic dysfunction in GBS complicated by suspected sepsis, marked by alternating parasympathetic and sympathetic dominance. The development of grade II AV block followed by persistent sinus tachycardia reflects profound autonomic instability, which poses a high risk for sudden cardiovascular deterioration in critically ill patients.

GBS is an autoimmune disorder that attacks the peripheral nervous system and can involve the autonomic nervous system. Autonomic dysfunction results from demyelinisation or axonal damage to sympathetic and parasympathetic nerve fibres resulting in an imbalance of autonomic regulation. Manifestations include fluctuations in blood pressure, tachycardia, bradycardia, and atrioventricular block (AV block), with the prevalence of autonomic dysfunction occurring in approximately 41-60% of cases and in some studies even reported up to 95% of patients (Graham et al., 2023; Singh et al., 2020). In GBS patients, arrhythmias such as ventricular tachycardia, AV block, and asystole can occur due to impaired cardiac autonomic regulation (Davarashvili & Balkin, 2018; Fryman et al., 2020).

In this case, the patient has received three cycles of IVIg therapy in early January 2025 but has not shown any improvement in the patient's clinical condition. IVIg is a therapy in the management of GBS, especially in patients who have lost the ability to walk independently (Walgaard et al., 2018). IVIg is known to promote the expansion of regulatory T cells and increase the secretion of anti-inflammatory cytokines in GBS patients (G. Zhang et al., 2019). IVIg is known to accelerate the recovery process but the effectiveness of the therapy may vary depending on the GBS subtype of the patient (Kalita et al., 2022). In more severe cases, plasma exchange (PE) is an alternative treatment option and a number of comparative studies have been conducted to evaluate the relative effectiveness of the two treatment modalities (Zaki et al., 2023).

On 11 January 2025, the patient presented with significant cardiac and vascular dysfunction consistent with the clinical picture of autonomic dysfunction. Vital signs (VTS) observation data clearly illustrated haemodynamic instability. Heart rate (HR) fluctuations were extreme, recorded from a bradycardia of 66x/min at 12.00pm, to a high tachycardia of 160x/min at 19.00pm and 153x/min at 20.00pm. These marked HR variations, especially the bradycardia episodes, support the suspicion of sinus dysfunction indicating impairment in the ability of the sinoatrial node to properly generate or regulate cardiac impulses. Furthermore, the ECG results indicated a grade II AV block and this condition may indicate a possible disturbance in the electrical conduction system between the atria and ventricles such that not all electrical impulses from the atria are successfully transmitted to the ventricles to trigger effective contractions. AV block is a disturbance in the transmission of electrical impulses from the atria to the ventricles which is categorised into several degrees of severity and this disturbance can appear physiologically due to increased vagal tone activity, or pathologically due to degenerative processes in the cardiac conduction system (Aizawa & Kawamura, 2020). In second-degree AV block, the clinical manifestations can vary in terms of symptoms and severity, requiring careful electrocardiographic (ECG) evaluation to determine the appropriate management strategy (Lim et al., 2018). This combination of sinus dysfunction and second-degree AV block then comprehensively explains the extreme HR fluctuations that can be observed in patients.

In this patient, autonomic dysfunction progressed to grade II AV block indicating involvement of the cardiac conduction system, specifically the sinoatrial and atrioventricular nodes. SA node dysfunction may result from extreme parasympathetic dominance or direct damage to the autonomic nerve fibres that regulate heart rhythm. Extreme parasympathetic activity can potentially result in complete heart block, although the incidence is relatively low in the early stages of the disease (Kuruppuarachchi et al., 2018). In GBS severe autonomic dysfunction can intermittently block the transmission of impulses from the atria to the ventricles, resulting in a Mobitz type I or II pattern which if not recognised may risk progression to complete block or asystole (Abbas & Sardar, 2021). Interestingly, in less than 24 hours after AV block detection, patients show a change towards sinus tachycardia. This change did not reflect an improvement in heart rhythm but showed a sharp shift from parasympathetic dominance to sympathetic dominance which is characteristic of fluctuating autonomic dysfunction in GBS. This mechanism is closely related to central or peripheral disorders of the autonomic nervous system which is no longer able to

maintain homeostasis, causing a surge in sympathetic activity resulting in tachycardia, vasoconstriction and labile blood pressure.

The patient's non-invasive blood pressure (NIBP) showed substantial instability. There were multiple episodes of hypotension with the lowest values being 80/60 mmHg at 6pm and 98/52 mmHg at 2pm. Mean arterial pressure (MAP) was also frequently below the optimal perfusion threshold (66-67 mmHg) potentially leading to organ hypoperfusion. Haemodynamic instability in patients characterised by fluctuations in HR and blood pressure are classic manifestations of autonomic dysfunction in GBS patients (James et al., 2024). Autonomic dysfunction in GBS can directly affect the cardiac conduction system as well as peripheral vascular tone regulation, potentially leading to arrhythmias and significant haemodynamic instability, and in severe cases can be life-threatening (Abbas & Sardar, 2021; Davarashvili & Balkin, 2018; Fryman et al., 2020).

Sinus tachycardia may be influenced by systemic clinical conditions such as hypotension, hypoxia, fever and systemic inflammatory activation, as was the case in this patient who presented with a high body temperature >39°C, severe leucocytosis (37,000/mm³) and radiological signs of bilateral pneumonia. Sinus tachycardia in this context reflects a compensatory response to inadequate tissue perfusion and is also part of the early hyperdynamic phase in sepsis. Cardiovascular failure associated with sepsis is characterised by ventricular dilatation, decreased contractile function, and impaired overall cardiac function, with pathophysiological mechanisms involving complex interactions between systemic factors and structural and functional changes in cardiomyocytes (L. Martin et al., 2019). The progression to sepsis in this patient can be explained by a combination of possible infection as confirmed by the bilateral pneumonia on radiology, as well as possible organ dysfunction reflected by hypoalbuminemia, anaemia, and blood gas fluctuations. Sepsis is known to exacerbate autonomic dysfunction through the release of inflammatory cytokines, impaired microcirculatory perfusion and baroreflex disruption.

Sepsis is known to exacerbate autonomic dysfunction through various mechanisms. The release of inflammatory cytokines during sepsis triggers autonomic nervous system dysfunction that can lead to severe systemic inflammation and even death (Kiryachkov et al., 2020). In addition, impaired microcirculation perfusion occurs due to endothelial dysfunction, which is a hallmark of sepsis. Neutrophils and neutrophil extracellular traps (NETs) contribute to this by degrading endothelial glycocalyx and increasing permeability leading to tissue hypoperfusion and organ failure (H. Zhang et al., 2023). Baroreflex dysfunction is also part of a broader autonomic nervous system dysfunction in sepsis that affects cardiac output, vascular tone and other vital functions (Badke et al., 2018). These mechanisms are interrelated, involving endothelial and microvascular dysfunction, immune and autonomic dysregulation, and reprogramming of cellular metabolism (Pool et al., 2018). The interaction between GBS and sepsis creates a pathological loop that aggravates each other, GBS triggers autonomic dysfunction then autonomic dysfunction worsens compensation to infection and sepsis amplifies autonomic imbalance. In this context, AV block and sinus tachycardia are not two separate entities, but rather part of a fluctuating and dynamic spectrum of symptoms of severe dysautonomia.

The phenomenon of rapid change from bradycardia or heart block to tachycardia reflects the instability of autonomic tone and suggests that the patient's autonomic nervous system is in a state of complete dysregulation. The literature suggests that patients with extreme autonomic fluctuations have a higher risk of requiring mechanical ventilation, experience prolonged ICU stay and have worse functional outcomes (Bazán-Rodríguez et al., 2023). The interventions provided, including mechanical ventilation, vasopressors (norepinephrine), inotropes (dobutamine), broad-spectrum antibiotics and close haemodynamic monitoring, are an important part of patient stabilisation. Autonomic stabilisation in GBS complicated by sepsis requires not only medical management, but also active involvement of nurses in vital parameter monitoring, ECG interpretation, early detection of autonomic

dysfunction symptoms, and effective multidisciplinary communication.

Management in this patient involved collaborative and intensive clinical and nursing interventions. Intensive respiratory support was provided through mechanical ventilation with P-SIMV mode, $FiO_2$ up to 90%, tidal volume 480 mL, and PEEP 8 $cmH_2O$. The use of mechanical ventilation is crucial given the potential for progressive respiratory muscle weakness in GBS, which can lead to respiratory failure. Severe neuromuscular weakness at hospital admission, including lower limb strength $\leq 2$ and cranial nerve involvement, is associated with increased need for mechanical ventilation in paediatric patients with GBS (Randhawa et al., 2022). Optimisation of mechanical ventilation management, including appropriate timing of intubation, early implementation of tracheostomy, and prevention of complications, plays an important role in improving the long-term outcomes of GBS patients (Shang et al., 2020). Meanwhile, additional respiratory therapy with inhalation (combivent, pulmicort, acetylcysteine) and closed airway suction is performed with the aim of maintaining airway patency and mobilising secretions.

Haemodynamic support is a crucial component in the management of autonomic instability. The patient received dobutamine (5 mcg/kg/BB/min) as an inotropic agent to increase myocardial contractility and cardiac output. Administration of norepinephrine (Vascon) was started on 14 January 2025 with an initial dose of 0.1 mcg/kg/BB/min and then increased gradually until it reached 0.8 mcg/kg/BB/min and the dose was maintained. Based on the TTV data on 14 January 2025, the need for vasopressor intervention was indeed indicated by the presence of hypotensive episodes (89/52 mmHg at 14.00 and 86/48 mmHg at 16.00) and low MAP (64.33 mmHg at 14.00 and 60.67 mmHg at 16.00). The increase in norepinephrine dose reflects an attempt to achieve an adequate blood pressure target. Administration of norepinephrine as a vasopressor directly addresses hypotension by increasing systemic vascular resistance. Norepinephrine is the first-line vasopressor agent in management to achieve adequate blood pressure (Hamzaoui et al., 2023; Jentzer & Hollenberg, 2021; Russell, 2019). Its main mechanism of action is to increase systemic vascular resistance, making it effective in correcting hypotensive conditions (Jentzer & Hollenberg, 2021). Early detection of the need for vasopressors and gradual adjustment of norepinephrine dose is recommended to optimise resuscitation outcomes (Hamzaoui et al., 2023). Nonetheless, the TTV data on 20 January 2025 still showed fluctuations in blood pressure and episodes of hypotension (e.g. 78/52 mmHg at 16.00 and 80/51 mmHg at 18.00), as well as MAP sometimes below 65 mmHg (e.g. 61 mmHg at 16.00 and 18.00), suggesting that the patient's haemodynamic instability was still persistent despite intensive vasopressor and inotropic support. This may indicate the severity of the autonomic dysfunction experienced by the patient and the challenges in achieving consistent haemodynamic stability.

In an attempt to manage the systemic infection, the patient received antibiotics, the regimen of which was adjusted according to clinical progress and laboratory results. On 10 January 2025, Levofloxacin and Meropenem were administered. However, on 14 January 2025, Meropenem was discontinued and replaced with Amikacin. Subsequently, on 20 January 2025, the regimen switched to Levofloxacin, Vancomycin, and Ampicillin. Analysis of the patient's leucocyte trends showed a direct correlation with the inflammatory response and effectiveness of this antibiotic therapy. Leukocytes from 13,260 on 10 January 2025 jumped significantly to 20,690 on 14 January 2025 indicating an active infection. Despite showing a downward trend post-antibiotic change on 16 January 2025 to 8,730, leukocytes again increased sharply, reaching 37,000 on 20 January 2025. This drastic increase in leucocytes may indicate an uncontrolled infection resulting in an aggressive modification of the antibiotic regimen. Antibiotic regimen changes are a frequent occurrence in patients with complicated Gram-negative bacterial infections, with up to 72% of patients undergoing therapy adjustments (Yamaki et al., 2024). The frequency of these therapy modifications can occur 0 to 6 times per patient, generally triggered by initial culture

results, non-achievement of clinical response, or the occurrence of decompensation(Yamaki et al., 2023; Yamaki & Dillon, 2022). In these cases the fluctuations emphasise the dynamic nature of the patient's infectious condition and the challenges in achieving effective infection control. The high rate of regimen changes reflects the complexity in early detection of resistant pathogens as well as the need for development of antibiotics with optimised efficacy (Yamaki et al., 2024).

For temperature control and sedation, the patient received paracetamol (4x500 mg) and dexmedetomidine infusion (0.2 mcg/kg/hour). Paracetamol was intended to control persistent fever that may be related to autonomic thermoregulatory dysregulation or infection, while dexmedetomidine facilitated sedation necessary for the management of mechanically ventilated patients. Paracetamol is a non-opioid synthetic analgesic and antipyretic agent that acts through a central mechanism and its antipyretic effect is obtained through inhibition of the cyclooxygenase-3 (COX-3) enzyme and decreased prostaglandin synthesis in the central nervous system, which in turn modulates body temperature regulation in the hypothalamic thermoregulatory centre (Chiumello et al., 2017). Dexmedetomidine produces sedation effects while maintaining the level of consciousness in patients admitted to the ICU, the use of this agent is associated with shorter extubation times increased number of days free from coma or delirium, decreased incidence of delirium-related agitation, prevention of delirium, and lower mortality rates in certain patient groups compared to other sedative agents (Shehabi et al., 2019).

Nurses play a central role in the management of GBS patients with severe autonomic dysfunction in the ICU. The nurse's responsibilities include close haemodynamic monitoring to detect and respond to frequent blood pressure fluctuations and arrhythmias, optimal airway management and mechanical ventilation to prevent respiratory failure, managing various autonomic complications such as gastrointestinal and bladder dysfunction, thermoregulatory instability, and renal salt wasting (RSW) all of which require careful monitoring of fluid balance, and organ function, prevent common ICU complications, and play a role in providing education, psychosocial support for patients and families, collaborating with multidisciplinary teams to ensure comprehensive care planning and implementation, so as to optimise physiological stability and clinical outcomes of complex patients (Martin, 2024; Nguyen et al., 2023). Overall, the interventions are multidisciplinary and responsive to the patient's clinical dynamics. In this case, the patient's progress showed that despite intensive support, the patient still faced serious challenges in controlling infection and achieving adequate haemodynamic stability, reflected by significant leucocyte fluctuations and ongoing vasopressor requirements.

From a critical care nursing perspective, this case highlights the essential role of continuous haemodynamic and cardiac rhythm monitoring in detecting early signs of autonomic deterioration. Frequent nursing assessments enabled timely recognition of heart rate variability, hypotensive episodes, and rhythm disturbances, supporting rapid escalation to medical intervention. Nursing vigilance was central to maintaining patient safety by anticipating sudden cardiovascular changes, optimizing ventilatory support, and coordinating multidisciplinary responses in a highly unstable clinical situation.

It can be concluded that the changes in autonomic symptoms in the GBS patient in this case were triggered by a combination of immunological interference to the autonomic nervous system and exacerbated by systemic inflammation due to sepsis. The worsening culminated in the appearance of grade II AV block as a representation of sinoatrial node dysfunction which then shifted to sinus tachycardia as an expression of sympathetic dominance. This dynamic emphasises the importance of continuous monitoring, rapid interventional response and holistic coordination of intensive care.

## Conclusions

This case highlights the complexity of severe autonomic dysfunction in a GBS patient exacerbated by a systemic infectious process with suspected sepsis. The clinical manifestations of grade II AV block,

bradycardia that shifted to sinus tachycardia, as well as blood pressure fluctuations suggested a sharp imbalance of autonomic regulation due to sympathetic-parasympathetic damage and uncontrolled inflammatory response. These findings reinforce the understanding that autonomic dysfunction in GBS not only reflects neuro- cardiovascular symptoms but also signals a serious risk to haemodynamic stability, especially when accompanied by severe infection. The dynamics of AV block and sinus tachycardia are not two separate entities, but rather part of a spectrum of fluctuating and potentially life-threatening autonomic responses.

From a critical care nursing perspective, this case provides an important contribution by illustrating how continuous haemodynamic and cardiac rhythm monitoring, frequent vital sign assessment, and early recognition of autonomic instability support timely clinical decision-making and patient safety. Nursing-led surveillance enabled rapid identification of dynamic autonomic changes, facilitated prompt escalation of care, and supported interdisciplinary interventions aimed at stabilising cardiovascular function in a highly unstable clinical context.

Although this report is limited by its single-case design and cannot be generalised, it offers clinically relevant insights into the nursing management of fluctuating autonomic dysfunction in critically ill GBS patients. The findings highlight the need for heightened nursing vigilance, structured autonomic monitoring, and proactive communication in critical care settings. Future studies involving larger cohorts are needed to further define nursing assessment frameworks and intervention strategies that optimise outcomes in GBS patients with autonomic dysfunction complicated by sepsis.

## References

Abbas, Z., & Sardar, Z. (2021). Use of pacemaker in GBS dysautonomia. *BMJ Case Reports* CP, 14(10), e242464. https://doi.org/10.1136/BCR-2021-242464

Aizawa, Y., & Kawamura, A. (2020). Exercise-induced Atrioventricular Block. *Internal Medicine*, 60(6), 827. https://doi.org/10.2169/INTERNALMEDICINE.6150-20

Badke, C. M., Marsillio, L. E., Weese-Mayer, D. E., & Sanchez-Pinto, L. N. (2018). Autonomic nervous system dysfunction in pediatric sepsis. *Frontiers in Pediatrics*, 6. https://doi.org/10.3389/FPED.2018.00280,

Bazán-Rodríguez, L., Ruiz-Avalos, J. A., de Saráchaga, A. J., Martinez-Jimenez, E., López-Hernández, J. C., & Vargas-Cañas, S. (2023). Autonomic dysfunctionand related outcomes in Guillain-Barre syndrome. *Autonomic Neuroscience: Basic and Clinical,* 245. https://doi.org/10.1016/j.autneu.2022.103070

Chiumello, D., Gotti, M., & Vergani, G. (2017). Paracetamol in fever in critically ill patients—an update. *Journal of Critical Care,* 38, 245–252. https://doi.org/10.1016/J.JCRC.2016.10.021

Davarashvili, I., & Balkin, J. (2018). Ventricular Tachycardia in the Guillain-Barre Syndrome. Cardiac complications in Guillain-Barre Syndrome, Review of the Literature. *Journal of Coronary Heart Diseases*, 2(2), 1–4. https://www.hilarispublisher.com/abstract/ventricular-tachycardia-in-the-guillainbarre-syndrome-cardiac-complications-in-guillainbarre-syndrome-review-of-the-lite-26625.html

Finsterer, J. (2022). Triggers of Guillain–Barré Syndrome: Campylobacter jejuni Predominates. *International Journal of Molecular Sciences* 2022, Vol. 23, Page 14222, 23(22), 14222. https://doi.org/10.3390/IJMS232214222

Florian, I. A., Lupan, I., Sur, L., Samasca, G., & Timiș, T. L. (2021). To be, or not to be… Guillain-Barré Syndrome. *Autoimmunity Reviews*, 20(12). https://doi.org/10.1016/J.AUTREV.2021.102983,

Fryman, E., Saleem, S., & Singh, A. (2020). Guillain-Barré Syndrome Induced Autonomic dysfunction Resulting in Cardiac Arrest. *Cureus*, 12(12). https://doi.org/10.7759/CUREUS.12149

Ghazavi, A., Abbasi, E., & Mohammadi, S. (2020). Evaluation of demographic, clinical and paraclinical features of children with Guillain-Barré syndrome. *Journal of Preventive Epidemiology*, 5(2), e23–e23. https://doi.org/10.34172/JPE.2020.23

Graham, B., David, O., Matthew, E., Syed, S. U. H., Laura, C., Ryan, K., Helen, D., Aisling, C., & Timothy, L. (2023). Screening for autonomic dysfunctioncomplications in guillain-barré syndrome (GBS): a multicentre retrospective cohort study. *Journal of Neurology, Neurosurgery & Psychiatry*, 94(Suppl 1), A27–A27. https://doi.org/10.1136/JNNP-2023-ABN.81

Hamzaoui, O., & Boissier, F. (2023). Hemodynamic monitoring in cardiogenic shock. *Journal of Intensive Medicine,* 3(2), 104–113. https://doi.org/10.1016/j.jointm.2022.10.003

Hamzaoui, O., Goury, A., & Teboul, J. L. (2023). The Eight Unanswered and Answered Questions about the Use of Vasopressors in Septic Shock. *Journal of Clinical Medicine* 2023, Vol. 12, Page 4589, 12(14), 4589. https://doi.org/10.3390/JCM12144589

James, N., Reddy, S., Maheshwari, U., Elamurugan, N., Kumar, N., Kumar, A. N., Tejaswini, S. D., Ranganathan, L. N., & S, B. (2024). Incidence of Cardiovascular Instability in Patients With Guillain-Barré Syndrome: A Retrospective Study. *Cureus*, 16(1). https://doi.org/10.7759/CUREUS.52778

Jentzer, J. C., & Hollenberg, S. M. (2021). Vasopressor and Inotrope Therapy in Cardiac Critical Care. *Journal of Intensive Care Medicine,* 36(8), 843–856. https://doi.org/10.1177/0885066620917630/ASSET/38283277-5C8F-42BA-AA9F-A1B81DAB014A/ASSETS/IMAGES/LARGE/10.1177_0885066620917630-FIG3.JPG

Kalita, J., Misra, U. K., Chaudhary, S. K., Das, M., Mishra, A., Ranjan, A., & Kumar, M. (2022). Outcome of Guillain–Barré syndrome following intravenous immunoglobulin compared to natural course. *European Journal of Neurology*, 29(10), 3071–3080. https://doi.org/10.1111/ENE.15500,

Kim, A. Y., Lee, H., Lee, Y. M., & Kang, H. Y. (2021). Epidemiological features and economic burden of guillain-barré syndrome in south korea: A nationwide population-based study. *Journal of Clinical Neurology* (Korea), 17(2), 257–264. https://doi.org/10.3988/JCN.2021.17.2.257,

Kiryachkov, Y. Y., Bosenko, S. A., Muslimov, B. G., & Petrova, M. V. (2020). Dysfunction of the autonomic nervous system and its role in the pathogenesis of septic critical illness (review). *Sovremennye Tehnologii v Medicine*, 12(4), 106–118. https://doi.org/10.17691/STM2020.12.4.12,

Kuruppuarachchi, A. N., Sundaresan, K. T., & Thivakaran, T. (2018). Rare presentation of Guillain-Barre syndrome with complete heart block and neuropathic chest pain mimicking acute coronary syndrome: a case report. *Ceylon Medical Journal*, 63(3), 156. https://doi.org/10.4038/CMJ.V63I3.8713

Lim, Y., Singh, D., & Poh, K. K. (2018). High-grade atrioventricular block. *Singapore Medical Journal,* 59(7), 346–350. https://doi.org/10.11622/SMEDJ.2018086

Martin, L., Derwall, M., Al Zoubi, S., Zechendorf, E., Reuter, D. A., Thiemermann, C., & Schuerholz, T. (2019). The Septic Heart: Current Understanding of Molecular Mechanisms and Clinical Implications. *Chest,* 155(2), 427–437. https://doi.org/10.1016/j.chest.2018.08.1037

Martin, P. (n.d.). 8 *Guillain-Barre Syndrome Nursing Care Plans - Nurseslabs*. 2024. Retrieved June 18, 2025, from https://nurseslabs.com/guillain-barre-syndrome-nursing-care-plans/#google_vignette

Mirian, A., Nicolle, M. W., & Budhram, A. (2021). Guillain–Barré syndrome. *CMAJ,* 193(11), E378. https://doi.org/10.1503/CMAJ.202710,

Nagayama, T., Ohara, J., Sano, C., & Ohta,

R. (2023). Staphylococcus epidermidis Bacteremia in an Older Patient With Guillain-Barré Syndrome With Fever of Unknown Origin: A Case Report. *Cureus,* 15(9). https://doi.org/10.7759/CUREUS.45940

Nguyen, T. P., Taylor, R. S., & Boyle, A. G. R. (2023). Guillain-Barre Syndrome (Nursing). StatPearls. https://www.ncbi.nlm.nih.gov/books/NBK568815/

Pool, R., Gomez, H., & Kellum, J. A. (2018). Mechanisms of Organ Dysfunction in Sepsis. *Critical Care Clinics*, 34(1), 63–80. https://doi.org/10.1016/j.ccc.2017.08.003

Pranata, J. (n.d.). *Guillaine Barre Syndrome GBS.* 2020. Retrieved June 18, 2025, from https://rsud.nunukankab.go.id/detailpost/guillaine-barre-syndrome-gbs

Randhawa, M. S., Iyer, R., Bansal, A., Mukund, B., Angurana, S. K., Nallasamy, K., Jayashree, M., Singhi, S. C., Singhi, P., Baranwal, A. K., & Sankhyan, N. (2022). Clinical Features Associated with Need for Mechanical Ventilation in Children with Guillain-Barré Syndrome: Retrospective Cohort from India. *Pediatric Critical Care Medicine*, 23(5), 378–382. https://doi.org/10.1097/PCC.0000000000002930

Russell, J. A. (2019). Vasopressor therapy in critically ill patients with shock. *Intensive Care Medicine*, 45(11), 1503–1517. https://doi.org/10.1007/S00134-019-05801-Z/TABLES/2

Shang, P., Zhu, M., Baker, M., Feng, J., Zhou, C., & Zhang, H. L. (2020). Mechanical ventilation in Guillain–Barré syndrome. *Expert Review of Clinical Immunology*, 16(11), 1053–1064. https://doi.org/10.1080/1744666X.2021.1840355;SUBPAGE:STRING:ACCESS

Shehabi, Y., Howe, B. D., Bellomo, R., Arabi, Y. M., Bailey, M., Bass, F. E., Bin Kadiman, S., McArthur, C. J., Murray, L., Reade, M. C., Seppelt, I. M., Takala, J., Wise, M. P., & Webb, S. A. (2019). Early Sedation with Dexmedetomidine in Critically Ill Patients. *New England Journal of Medicine*, 380(26), 2506–2517. https://doi.org/10.1056/NEJMOA1904710/SUPPL_FILE/NEJMOA1904710_DATA-SHARING.PDF

Singh, J., Raja, V., Irfan, M., Hashmat, O., Syed, M., & Shahbaz, N. N. (2020). Frequency of Autonomic Dysfunction in Patients of Guillain Barre Syndrome in a Tertiary Care Hospital. *Cureus*, 12(12). https://doi.org/10.7759/CUREUS.12101

Walgaard, C., Jacobs, B. C., Lingsma, H. F., Steyerberg, E. W., Cornblath, D. R., van Doorn, P. A., de Wit, M. C. Y., van den Berg, B., Doets, A. Y., Leonhard, S. E., Verboon, J. C., van Woerkom, M., Tio-Gillen, A. P., van Rijs, W., Huizinga, H., Badrising, U. A., Bienfait, H. M. E., Blom, R. J., van Boheemen, C. J. M., … Hintzen, R. Q. (2018). Second IVIg course in Guillain-Barré syndrome patients with poor prognosis (SID-GBS trial): Protocol for a double-blind randomized, placebo-controlled clinical trial. *Journal of the Peripheral Nervous System*, 23(4), 210–215. https://doi.org/10.1111/JNS.12286,

WHO. (n.d.). **Guillain–Barré syndrome.** 2023. Retrieved June 18, 2025, from https://www.who.int/news-room/fact-sheets/detail/guillain-barr%C3%A9-syndrome

Yamaki, J., & Dillon, R. J. (2022). 653. Characterizing Antibacterial Regimen Modification, Patient Characteristics, and Outcomes for Patients with Complicated Gram-negative Bacterial Infections. *Open Forum Infectious Diseases*, 9(Supplement_2). https://doi.org/10.1093/OFID/OFAC492.705

Yamaki, J., Mikhail, M., Beuttler, R., Robinson, P., Yücel, E., & Watanabe, A. H. (2024). Characterizing Antibiotic Regimen Modification Behavior, Patient Characteristics, and Outcomes for Patients with Gram-Negative Bacterial Infections, A Retrospective Single-Center Study. *Antibiotics* 2024, Vol. 13, Page 302, 13(4), 302. https://doi.org/10.3390/ANTIBIOTICS13040302

Yamaki, J., Yucel, E., & Watanabe, A. H. (2023). 2282. Characterizing

Antibacterial Regimen Modification, Patient Characteristics, and Outcomes for Patients with Complicated Gram-Negative Bacterial Infections. *Open Forum Infectious Diseases,* 10(Supplement_2). https://doi.org/10.1093/OFID/OFAD500.1904

Zaki, H. A., Iftikhar, H., Najam, M., Masood, M., Al-Marri, N. D. R., Elgassim, M. A. M., Fayed, M., & Shaban, E. E. (2023). Plasma exchange (PE) versus intravenous immunoglobulin (IVIG) for the treatment of Guillain-Barré syndrome (GBS) in patients with severe symptoms: A systematic review and meta-analysis. *ENeurologicalSci*, 31. https://doi.org/10.1016/j.ensci.2023.100468

Zhang, B., Duan, L., Ma, L., Cai, Q., Wu, H., Chang, L., Li, W., & Lin, Z. (2022). Rapidly progressive Guillain–Barré syndrome following amitriptyline overdose and severe Klebsiella pneumoniae infection: A case report and literature review. *Frontiers in Medicine*, 9, 991182. https://doi.org/10.3389/FMED.2022.991182

Zhang, G., Wang, Q., Song, Y., Cheng, P., Xu, R., Feng, X., & Li, X. (2019). Intravenous immunoglobulin promotes the proliferation of CD4+CD25+ Foxp3+ regulatory T cells and the cytokines secretion in patients with Guillain-Barré syndrome in vitro. *Journal of Neuroimmunology*, 336. https://doi.org/10.1016/j.jneuroim.2019.577042

Zhang, H., Wang, Y., Qu, M., Li, W., Wu, D., Cata, J. P., & Miao, C. (2023). Neutrophil, neutrophil extracellular traps and endothelial cell dysfunction in sepsis. *Clinical and Translational Medicine*, 13(1). https://doi.org/10.1002/CTM2.1170

Zheng, P., Tian, D. C., Xiu, Y., Wang, Y., & Shi, F. D. (2022). Incidence of Guillain-Barré syndrome (GBS) in China: A national population-based study. *The Lancet Regional Health - Western Pacific*, 18. https://doi.org/10.1016/j.lanwpc.2021.100302