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Management af a Patient with Guillain-Barré Syndrome Miller Fisher Type and Hospital-Acquired Pneumonia in the Intensive Care Unit : a Case Report

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ABSTRACT

Introduction: Guillain-Barré Syndrome (GBS) is an acute, flaccid polyneuropathy that occurs following an infection and is mediated by an autoimmune process. Patients with GBS frequently experience respiratory complications that necessitate mechanical ventilation. Neurological impairments associated with GBS, such as decreased airway patency, ineffective cough, and difficulty swallowing, increase the risk of pulmonary infections like hospital-acquired pneumonia (HAP).

Case Report: We present the case of a 59-year-old woman who experienced respiratory failure due to GBS and HAP and required treatment in the Intensive Care Unit (ICU). The patient was managed with Therapeutic Plasma Exchange (TPE), which resulted in clinical improvement. It took 18 days for the patient to be weaned off mechanical ventilation.

Conclusion: A detailed review of the management strategies for GBS and HAP is essential to enhance future treatment approaches and ensure they align with current literature.

Guillain Barre Syndrome, Hospital Acquired Pneumonia

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INTRODUCTION

Guillain–Barré syndrome (GBS) is an autoimmune disorder that affects the peripheral nervous system, characterized by acute demyelinating polyneuropathy and acute flaccid paralysis mediated by an autoimmune response. The global incidence of GBS is reported to be 1.1-4 cases per 100,000 individuals annually.[1,2] Guillain–Barré Syndrome (GBS) encompasses four primary subtypes: motor-sensory GBS, pure motor GBS, Miller Fisher Syndrome, and a variant involving other presentations. GBS is frequently triggered by infections, including those caused by Campylobacter jejuni, cytomegalovirus, herpes simplex virus, and upper respiratory tract infections. The pathological process involves complement activation, which leads to the destruction of myelin in the peripheral nervous system. Patients with GBS may experience acute respiratory complications due to progressive weakness of the respiratory muscles, with approximately 30% of patients requiring mechanical ventilation.[2] Approximately two-thirds of GBS cases are preceded by symptoms of respiratory or gastrointestinal infections.

Hospital-acquired pneumonia (HAP), also known as nosocomial pneumonia, is defined as pneumonia that occurs 48 hours or more after hospital admission and was not present at the time of admission. HAP is a common infection in intensive care units (ICUs). However, the mortality rate associated with HAP is estimated to be between 5% and 13%.[3] The 2013 Basic Health Research (Riskesdas) data indicate that the prevalence

of pneumonia in Indonesia is 0.63%. The mortality rate for pneumonia among hospitalized patients ranges from 5% to 15%, and it increases to between 20% and 50% for patients receiving care in Intensive Care Units (ICUs).[4] Wereport a case of Guillain–Barré Syndrome (GBS) complicated by hospital-acquired pneumonia (HAP) that required mechanical ventilation. The patient was treated in the Intensive Care Unit (ICU) for 18 days.

CASE REPORT

A 59-year-old woman presented with primary complaints of gradual and worsening weakness and paresthesia in all four limbs over the past 10 days. The weakness initially affected both legs, particularly at the feet, and then progressed to both arms, accompanied by numbness and tingling, The patient also reported difficulty gripping objects with her hands.

Additionally, the patient experienced balance disturbances while walking for the past 8 days, which worsened, particularly when opening her eyes, and she began to have difficulty moving her eyes. She exhibited unsteady gait when standing or walking, which did not worsen with position changes and improved when closing her eyes. The patient needed support while walking, requiring assistance from family members for daily activities. The patient also presented with left-sided facial droop and slurred speech for the past 7 days. There were no complaints of bowel or urinary disturbances, neck pain radiating, or altered consciousness. Headache, vomiting, and seizures were denied. There was no unilateral numbness or weakness.

The patient experienced fever and persistent cough for the past 5 days, accompanied by hoarseness and difficulty swallowing. Two weeks prior, she had a history of productive cough and fever and had been on medication with no improvement. The patient was initially admitted to RS Tasikmalaya with a preliminary diagnosis of suspected stroke and pneumonia for 3 days but showed no improvement and was subsequently referred to RSHS. After reassessment, the diagnosis pointed towards Guillain–Barré Syndrome, leading to referral to RSHS. Prior treatments included Meropenem 3x1 gram, Mecobalamin 3x500 mg, Citicoline 2x500 mg, Lansoprazole 2x30 mg IV, Aspilet 1x80 mg, and NAC 3x200 mg.

The patient had no prior history of similar symptoms. There were no reports of prolonged fever, night sweats, weight loss, or contact with tuberculosis patients. No history of lumps elsewhere or neck trauma was noted. The patient did not smoke. Symptoms such as transient vision loss, perioral numbness, vertigo, and tinnitus were denied. She had completed her COVID-19 vaccination up to the third dose. There was no history of non-communicable diseases such as stroke, hypertension, diabetes mellitus, gout, high cholesterol, heart disease, kidney disease, or trauma.

On physical examination, the patient was conscious and oriented. Before intubation, her vital signs included a blood pressure of 130/88 mmHg, a pulse rate of 122 beats per minute, a respiratory rate of 28 breaths per minute, an oxygen saturation of 96% on a non-rebreathing mask at 15 liters per minute, and a temperature of 38.3°C. Nutritional status: weight 60 kg, height 160 cm. No anemia was observed in the conjunctiva, and the sclera was not icteric. Jugular venous pressure was 5 + 2 cm H2O, with no enlarged lymph nodes, symmetrical carotid pulses, and no carotid bruit. Thoracic shape and movement were symmetrical. Cardiac borders: left border at ICS V 2 cm lateral to LMCS, right border at LSD; heart sounds S1-II were regular with no murmur or gallop. Lung examination revealed normal percussion notes, vesicular breath sounds on both sides, with ronchi in the lower right and left lung fields. Abdominal examination showed a flat, supple abdomen with no palpable liver or spleen, normal bowel sounds, and no tenderness. Extremities were warm, without pitting edema or cyanosis, and capillary refill time was < 2 seconds.

Neurological examination showed no meningeal signs such as neck stiffness, Lasegue/Kernig, or Brudzinski signs. Cranial nerve examination: pupils were round and isocoric (3 mm), light reflexes were present both direct and indirect, eye movements showed paresis of CN III and CN VI, CN VII had left peripheral paresis, while CN IX and X were within normal limits, and CN XII was symmetrical. Motor examination showed normal tone with no atrophy or fasciculations, but tetraparesis with muscle strength of 2/2/2/2 in all segments. Toe flexion was positive, saddle hypesthesia was negative, and anal reflex was positive.

Abdominal reflexes were positive in all segments. Sensory examination revealed gloves and stocking hypesthesia. Autonomic function was within normal limits. Higher functions were intact. No physiological reflexes (e.g., BTR, KPR, APR) or pathological reflexes (e.g., Babinski, Chaddock, Hoffman, Tromner, and clonus) were noted. No regression reflexes (e.g., Palmomental) were observed.

In the Emergency Department at RSHS, the clinical pathology results revealed the following: Hemoglobin was 11.2 g/dL, hematocrit was 35.0%, and leukocyte count was 18.80 x 10^3/µL. The platelet count was 342,000/µL. The differential count showed basophils at 0%, eosinophils at 4%, lymphocytes at 17%, monocytes at 9%, band neutrophils at 3%, and segmented neutrophils at 67%. The neutrophil-to-lymphocyte ratio was 4.12. Albumin levels were 4.20 g/dL, and random glucose was 99 mg/dL. Urea was elevated at 27.9 mg/dL, while creatinine was 0.39 mg/dL. Electrolytes included sodium (Na) at 134 mEq/L, potassium (K) at 3.8 mEq/L, chloride (Cl) at 107 mEq/L, calcium at 5.13 mEq/L, and magnesium at 2.5 mmol/L. Blood gas analysis showed a pH of 7.426, pCO2 of 30.9 mmHg, pO2 of 116.8 mmHg, HCO3 of 20.5 mEq/L, base excess (BE) of -2.1, and SaO2 of 98.6%. Additional diagnostic imaging included a chest X-ray, which was suggestive of bronchopneumonia, and an electrocardiogram (ECG) report is also attached.

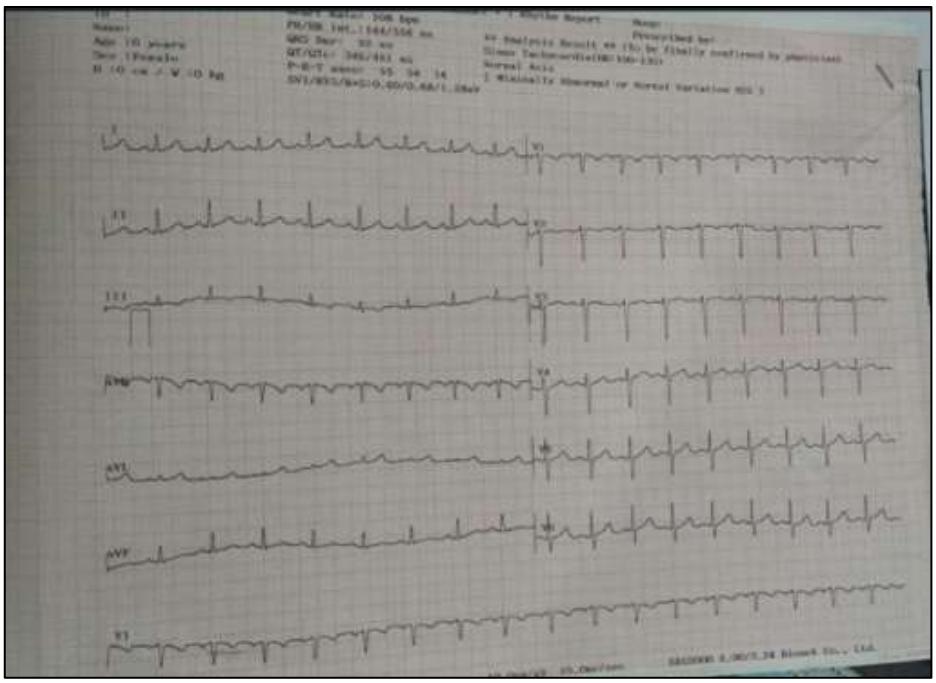

Fig.1 ECG

In the Emergency Department, the patient experienced increased respiratory effort while remaining conscious, with a blood pressure of 180/100 mmHg, a pulse rate of 132 beats per minute, a respiratory rate of 40 breaths per minute, and an oxygen saturation of 94% on a non-rebreathing mask at 15 liters per minute. The patient also had a fever of 38.2°C. Due to these findings, the patient was consulted for intubation and was subsequently admitted to the ICU. The patient was assessed as having respiratory failure due to Guillain–Barré Syndrome (GBS), with an Erasmus Guillain-Barre Respiratory Insufficiency Score (EGRIS) of 6, along with hospital-acquired pneumonia. Diagnostic tests showed leukocytosis at 18.80 x 10^3/µL, physical examination revealed ronchi in both lungs, and chest X-ray demonstrated opacity in the lower lobes of both lungs. Intubation and mechanical ventilation were performed, and the patient was then managed in the ICU. Prior to this, at previous hospital, the patient had been treated with Citicoline 2 x 500 mg, Mecobalamin 3 x 500 mg, Lansoprazole 2 x 30 mg IV, Meropenem 3 x 1 g IV, and Aspilet 1 x 80 mg.

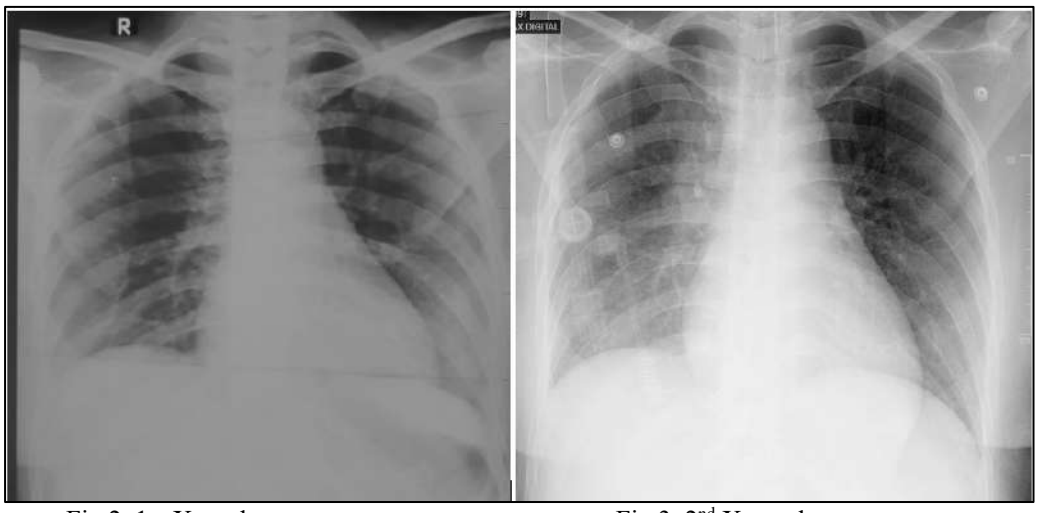

Fig 2. 1st Xray thorax

Fig 3. 2nd X-ray thorax

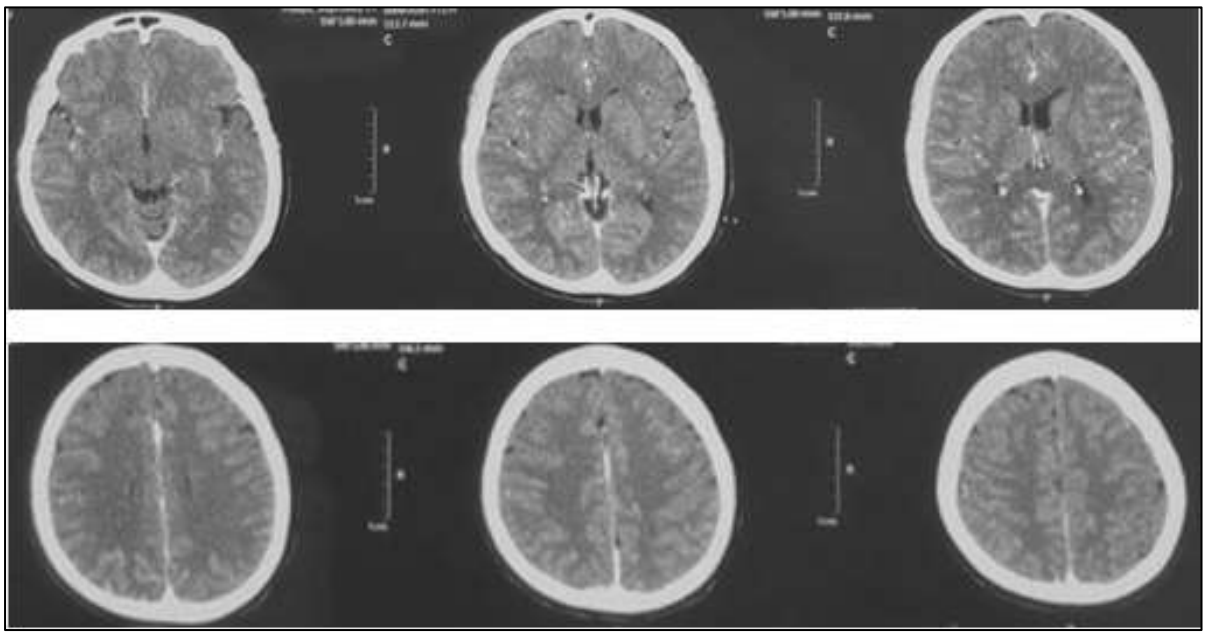

Fig. 4 Contrast Head CT scan shows no evidence of acute infarction or Transient Ischemic Attack (TIA). No signs of space-occupying lesions (SOL) or intracranial hemorrhage were observed.

The patient was diagnosed with respiratory failure secondary to Guillain–Barré Syndrome (GBS), specifically the Miller Fisher variant, complicated by hospital-acquired pneumonia. The management plan included ICU care, bed rest, and specific treatments comprising Mecobalamin 3x500 mg IV, Ceftriaxone 2x1 gram IV, Levofloxacin 1x750 mg IV, and plasma exchange (plasmapheresis) sessions 1 through 6. A nerve conduction study (NCS) was planned for 14 days after onset, and a lumbar puncture was scheduled for day 14. The plasmapheresis regimen was as follows: Regimen I with a total of 3000 ml, including 1000 ml of plasma, 1000 ml of colloid, and 1000 ml of crystalloid; Regimen II with a total of 3500 ml, including 1000 ml of plasma, 1500 ml of colloid, and 1000 ml of crystalloid. Regimen I-II was performed consecutively, while Regimens II-VI were administered non-consecutively.

On the initial days of ICU care, the patient received continuous sedation with midazolam at a dose of 5 mg/hour IV and antibiotics including Ceftriaxone 2x1 g IV and Levofloxacin 1x750 mg IV. Plasma exchange therapy was planned for managing GBS. ICU care continued, and chest X-rays indicated improvement in pneumonia. Plasma exchange was initiated on the 4th day of ICU care.

By the 7th day of ICU care, there was no significant improvement in respiratory parameters (weaning from mechanical ventilation remained challenging). Chest X-ray on that day showed increased opacity in the lower

right lung and an increase in secretions, which was assessed as Ventilator-Associated Pneumonia (VAP). Consequently, antibiotic therapy was escalated with Meropenem 3x1 g and Vancomycin 2x1 g (bacterial cultures performed on the first day of ICU admission showed no pathogens; repeat cultures were done). A tracheostomy was performed on the 13th day of ICU care to facilitate patient management and improve weaning from mechanical ventilation. By the 16th day of ICU care, the patient was still unable to be weaned from mechanical ventilation, but successfully weaned off on the 18th day.

DISCUSSION

Guillain–Barré Syndrome (GBS), the most common cause of acute paralytic neuropathy, is an inflammatory polyneuropathy characterized by acute onset, rapid progression, symmetrical muscle weakness, and hypo-or areflexia.[1-6]

In this case, a 59-year-old woman was referred to the Emergency Department at RSHS from another hospital, presenting with complaints of progressive weakness and tingling in all four limbs, which had gradually worsened over the past 10 days. The weakness initially affected both legs, particularly at the feet, and then extended to both arms, accompanied by numbness and difficulty gripping objects. Additionally, the patient experienced balance disturbances while walking for the past 8 days, which worsened, especially when opening her eyes, and there was increasing difficulty in eye movements. The patient exhibited an unstable gait when standing or walking, which did not worsen with position changes and improved when closing her eyes. She required assistance to walk, necessitating family support for daily activities. Furthermore, the patient presented with left-sided facial droop and slurred speech for the past 7 days.

Neurological examination revealed paresis of cranial nerves III and VI on both sides, with left-sided peripheral facial paresis (CN VII) and tetraparesis with motor strength rated as 2/2/2/2 in all segments. Sensory examination showed gloves and stocking-type hypesthesia. Based on the clinical history of limb weakness and physical examination findings, the patient is suspected to have the classic sensorimotor type of Guillain–Barré Syndrome (GBS), which is among the least commonly observed types, occurring in 5-25% of cases.

The diagnosis of Guillain–Barré Syndrome (GBS) is established based on the clinical history and physical examination, supported by confirmatory diagnostic tests such as cerebrospinal fluid analysis and electrodiagnostic studies.[5] Laboratory tests, including complete blood count, blood glucose levels, electrolytes, kidney function tests, and liver enzymes, are also performed on suspected GBS patients to rule out differential diagnoses. These results can help exclude other causes of acute flaccid paralysis, such as infections, metabolic disorders, or electrolyte imbalances.[5] In this case, the patient was diagnosed with Guillain–Barré Syndrome (GBS) due to the presentation of limb weakness starting in the legs, progressing to the upper limbs, and accompanied by difficulty swallowing, ocular movement abnormalities, left-sided facial paresis, and respiratory distress. The patient also had a history of a previous respiratory infection. Intubation and admission to the ICU were necessary due to signs of respiratory failure and an Erasmus Guillain–Barré Respiratory Insufficiency Score (EGRIS) of 6, which indicated severe respiratory compromise.

Nearly 50% of Guillain–Barré Syndrome (GBS) patients require ICU care, primarily due to respiratory issues (17-30%) and autonomic nerve involvement (20%). Clinical factors associated with an increased risk for the need for mechanical ventilation include rapid and progressive symptoms, simultaneous weakness of both upper and lower limbs, cephaloplegia, facial weakness, and bulbar involvement.[7] GBS patients should be admitted to the ICU if one of the following three criteria is met: (a) rapid progression of respiratory muscle weakness, (b) development of respiratory distress, (c) severe dysautonomia or dysphagia, or (d) an Erasmus Guillain–Barré Respiratory Insufficiency Score (EGRIS) greater than 4.[8]

Mechanical ventilation is a crucial decision in the management of Guillain–Barré Syndrome (GBS). It is important to carefully determine the appropriate timing for tracheostomy, the correct dosage of antibiotics, and the prediction of the duration of mechanical ventilation.[9] If mechanical ventilation is expected to exceed 3 weeks in the ICU, early tracheostomy should be considered. Early tracheostomy can offer several potential benefits for GBS patients, including increased comfort, earlier initiation of enteral nutrition, improved oral

hygiene, easier oral communication, and enhanced mobility. Conversely, delaying tracheostomy for more than 2 weeks may increase the risk of Ventilator-Associated Pneumonia (VAP), damage to the laryngeal nerve/mucosa/vocal cords, and formation of fistulas.[1]

In this patient, tracheostomy was performed on the 13th day, and 5 days after the procedure, the patient was successfully weaned from the ventilator and transferred to the High Care Unit (HCU). For GBS patients, tracheostomy is often necessary due to the extended duration of mechanical ventilation. Early tracheostomy typically facilitates quicker weaning from the ventilator and can shorten the overall ICU stay.

As of now, the standard treatment for Guillain–Barré Syndrome (GBS) includes Therapeutic Plasma Exchange (TPE) and Intravenous Immunoglobulin (IVIG). Recent studies indicate that both IVIG and TPE offer similar benefits, including curative effects, reduced likelihood of relapse, decreased time on mechanical ventilation, and comparable risk of complications. However, IVIG is generally preferred due to its ease of administration. IVIG is especially prioritized for managing patients with severe GBS symptoms.[10-12]

In this case, the patient received Therapeutic Plasma Exchange (TPE) with a total of 6 sessions. The typical regimen involves performing 5-6 exchanges over a period of 2 weeks, with an exchange volume of 50 ml/kg body weight. However, there is still some debate regarding the optimal number of sessions for effectiveness. The volume of therapy is usually 1 to 1.5 times the total plasma volume, estimated as follows: plasma volume (in liters) = 0.07 × body weight (kg) × (1 - hematocrit). TPE is most beneficial when administered within the first 4 weeks after the onset of symptoms, with the greatest effects observed if started within the first 2 weeks. Potential side effects of TPE include infections and sepsis.[13-16]

Pneumonia is one of the most common complications encountered in Guillain–Barré Syndrome (GBS). Hospital-acquired pneumonia (HAP) is defined as an infection of the pulmonary parenchyma caused by pathogens acquired in the hospital, occurring more than 48 hours after admission.[17-20]

The diagnosis of hospital-acquired pneumonia (HAP) was confirmed by initial chest X-rays upon arrival at the emergency department, which suggested pneumonia in both the right and left lungs. The patient had been admitted to a previous hospital for 3 days, and a comparison of the initial X-ray with the one taken upon admission to RSHS showed worsening opacities. According to the latest guidelines from the American Thoracic Society and the Infectious Diseases Society of America (ATS/IDSA), the choice of empirical antibiotic therapy for HAP should be based on the timing of onset and risk factors for multidrug-resistant (MDR) pathogens. Additionally, local patterns of microbial types and resistance should be considered. Recommended antibiotics in these guidelines target specific pathogens. Empirical therapy should be adjusted or discontinued based on microbiological results to prevent hospital-acquired resistance. After 72 hours, antibiotics should be tailored according to microbiological findings. If β-lactam antibiotics were initially prescribed and the microorganisms are found to be sensitive to β-lactam, the therapy should continue. However, if resistance is noted, an alternative β-lactam antibiotic may be used. If MRSA (Methicillin-resistant Staphylococcus aureus) is not detected in cultures, MRSA-targeted antibiotics should be discontinued. Fluoroquinolones and aminoglycosides may be stopped after 3-5 days of treatment, based on the clinical response and microbiological results.[10]

In this patient, following the administration of empirical antibiotics, the next step is to de-escalate the antibiotic therapy based on culture results. Cultures were obtained upon the patient's arrival at the hospital, in accordance with the pneumonia management guidelines.

CONCLUSION

Guillain-Barré Syndrome (GBS) is an acute, flaccid polyneuropathy that typically follows an infection and is mediated by an autoimmune response. Patients with GBS frequently experience acute respiratory disturbances that require mechanical ventilation. Neurological impairments in GBS, such as reduced airway patency, ineffective cough, and difficulty swallowing, also increase the risk of pulmonary infections, which can exacerbate the clinical condition of GBS patients. Hospital-acquired pneumonia (HAP) is defined as pneumonia that occurs 48 hours or more after hospital admission and was not present at the time of admission.

The severity of community-acquired pneumonia is directly related to mortality, and appropriate antibiotic therapy can reduce the duration of hospitalization and lower mortality rates. Empirical antibiotic therapy for HAP should be guided by the suspicion of resistant bacteria, pending culture results. Management of respiratory failure due to GBS and HAP in the Intensive Care Unit (ICU) can include Therapeutic Plasma Exchange (TPE), which has been shown to improve outcomes according to existing literature.

DECLARATIONS

Ethics approval and consent to participate. Permission for this study was obtained from the Ethics Committee of Padjadjaran University / Hasan Sadikin General Hospital Bandung.

CONSENT FOR PUBLICATION

The Authors agree to publication in Journal of Society Medicine.

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COMPETING INTERESTS

The authors declare that there is no conflict of interest in this report.

AUTHORS' CONTRIBUTIONS

All authors significantly contribute to the work reported execution, acquisition of data, analysis, and interpretation, or in all these areas. Contribute to drafting, revising, or critically reviewing the article. Approved the final version to be published, agreed on the journal to be submitted, and agreed to be accountable for all aspects of the work.

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