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Management of Patients with Guillain Barre Syndrome Accompanied by Ventilator Associated Pneumonia

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ABSTRACT

Introduction: Guillain-Barré syndrome (GBS) is one of the most common autoimmune diseases affecting the peripheral nervous system in the world. This disease has manifestations of weakness, progressive muscle areflexia and can cause weakness in the respiratory muscles. This causes the patient to need mechanical ventilation assistance. Pneumonia is one of the most common complications of GBS. Ventilator-Associated Pneumonia (VAP) is one of the common nosocomial infections in 10-20% of patients on mechanical ventilators in the Intensive Care Unit (ICU). The crude mortality rate in patients who develop VAP ranges from 30-70%. In addition, VAP can significantly increase the duration of hospitalisation, as well as the cost of care. Increasing bacterial resistance in today's world makes it difficult to treat VAP with empirical antibiotic therapy.

Case: A 44-year-old man came to the hospital with complaints of weakness in four limbs. The patient had previously been treated at another hospital with a diagnosis of GBS, because the patient's Erasmus GBS Respiratory Insufficiency Score (EGRIS) assessment was 5 and required further treatment in the ICU, the patient was referred to the main hospital. The complaint of being unable to move all four limbs was felt since 5 days before admission. The patient was admitted to the previous hospital for two days and received Mecobalamin, Gabapentin, and Ceftriaxone therapy.

Conclusion: GBS is a post-infectious autoimmune disease that results in nerve cell destruction. Severe muscle weakness can lead to respiratory failure resulting in the need for mechanical ventilation therapy. Nosocomial pneumonia is a common complication in patients in critical condition and the leading cause of death from nosocomial infections, especially ventilator-associated pneumonia in intubated patients.

Keywords

Guillain barre syndrome, Ventilator associated pneumonia, Intensive Care Unit

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INTRODUCTION

Guillain-Barré syndrome (GBS) is one of the most common autoimmune diseases affecting the peripheral nervous system in the world.[1] Worldwide, the incidence of GBS is 1.[3-4] per 100,000 cases each year.[1,2] GBS disease is more common in the young adult and elderly (>50 years) age ranges. About 75% of the GBS population is male.[2] However, for the Indonesian region there is currently no specific registered data regarding the incidence of GBS. The disease is characterised by acute neuromuscular paralysis ending in respiratory failure and approximately 25% of patients require mechanical ventilation and ICU care.[1-3]

Ventilator associated pneumonia (VAP) is one of the nosocomial infections that occur in 10-20% of patients on mechanical ventilators in the ICU.[4] The mortality rate associated with VAP is difficult to define, but some reports suggest a crude mortality rate of up to one-third or even up to half of cases.[4,5] In this case

report, a patient with GBS who developed respiratory failure requiring treatment in the ICU will be presented. During the ICU stay, the patient developed complications such as pneumonia that it took up to 18 days for the patient to be discharged from the ICU.

CASE REPORT

A 44-year-old man came to the hospital with complaints of weakness in four limbs. The patient had previously been treated at another hospital with a diagnosis of GBS, because the patient's Erasmus GBS Respiratory Insufficiency Score (EGRIS) assessment was 5 and required further treatment in the ICU, the patient was referred to the main hospital. The complaint of being unable to move all four limbs was felt since 5 days before admission. The patient was admitted to the previous hospital for two days and received Mecobalamin, Gabapentin, and Ceftriaxone therapy. The underlying complaint was limb weakness since 10 days before and difficulty swallowing. Three weeks prior to treatment the patient had a history of chickenpox infection. When the patient arrived at the hospital, the patient was fully conscious but there was an increase in respiratory effort where the respiratory rate was 42x/min and Levofloxacin 1x750 mg iv, the patient was planned to receive Intravenous Immunoglobulin (IVIG) for the management of GBS. ICU treatment continued and chest X-ray monitoring showed improvement in pneumonia. IVIG administration was started on day 2 of ICU care.

On day 6 of ICU stay, the patient had completed 5 cycles of IVIG therapy, but had not made significant improvement in respiration parameters (weaning from mechanical ventilation was still difficult) and due to a chest x-ray on that day showing new bilateral opacities, was assessed as VAP. Antibiotics was administered with Meropenem 3x1 g and Vancomycin 3x2 g (bacterial culture performed on the first day of ICU oxygen saturation was 94% with the use of 15 L/m non-rebreather mask oxygen.

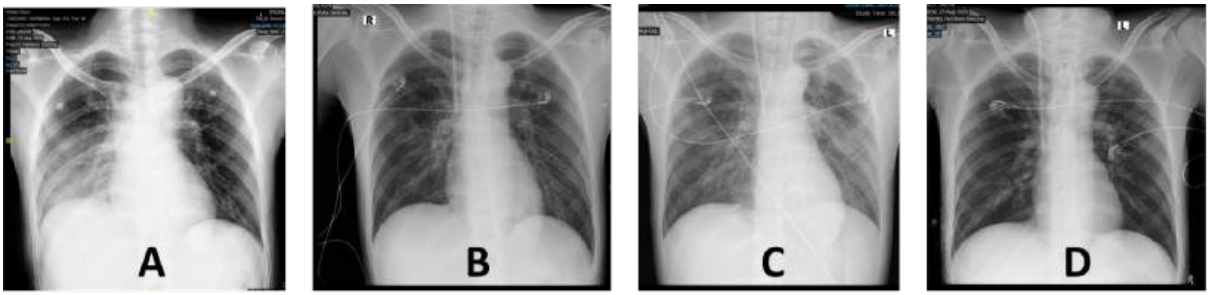

Figure 1. Thoracic X-ray of the patient during treatment (A) day 1 on admission to the emergency room showing right pneumonia (B) day 2 in ICU showing improvement (C) day 8 in ICU showing bilateral pneumonia (D) day 1 showing improvement.

The patient was assessed as respiratory failure et causa GBS accompanied by hospital-associated pneumonia (supporting examination showed leucocytosis at 17,630/uL and opacity in the lower lobe of the right lung). The patient was intubated and mechanically ventilated and continued to be treated in the ICU.The initial day of treatment in the ICU, sedation was given with continuous midazolam at a dose of 5 mg / hour iv and antibiotics with Ceftazidime 3x2 gr iv and Levofloxacin 1x750 mg iv, the patient was planned to receive Intravenous Immunoglobulin (IVIG) for the management of GBS. ICU treatment continued and chest X-ray monitoring showed improvement in pneumonia. IVIG administration was started on day 2 of ICU care.

On day 6 of ICU stay, the patient had completed 5 cycles of IVIG therapy, but had not made significant improvement in respiration parameters (weaning from mechanical ventilation was still difficult) and due to a chest x-ray on that day showing new bilateral opacities, was assessed as VAP. Antibiotics was administered with Meropenem 3x1 g and Vancomycin 3x2 g (bacterial culture performed on the first day of ICU admission did not show the presence of germs, re-culture was performed). Finally, percutaneous dilational tracheostomy (PDT) was performed on the 8th day of treatment to facilitate and fasten the weaning process. On the 10th day of treatment, the culture results showed the presence of Candida albicans culture in sputum, the patient was given anti-fungal therapy with Fluconazole. On the 15th day of treatment, the patient still could not be

extubated, the results of re-culture of germs returned there were other germs, namely Acinobacter baumanii, finally replaced antibiotics with ampicillian sulbactam according to the results of sensitive antibiotics. The patient was successfully discharged from mechanical ventilation on the 18th day of ICU care.

DISCUSSION

Guillain-Barré syndrome (GBS) is a type of peripheral neuropathy and is the cause of acute phakic paralysis characterised by symmetrical limb weakness, hyporeflexes or areflexes and elevated cerebrospinal fluid protein in the absence of a cellular reaction (pleocytosis) that can become progressively worse within 4 weeks.[1] GBS is an inflammatory disease of the peripheral nerves and is the most common cause of acute flaccid paralysis, with an annual global incidence of approximately 1-2 per 100,000 people each year.[1,2] GBS occurs more frequently in men than women and the incidence of GBS increases with age, although all age groups can be affected. GBS is usually precipitated by upper respiratory tract infections and gastrointestinal infections that occur 2 weeks before the onset of weakness.[6,7] About 25% of GBS patients develop respiratory distress due to respiratory muscle weakness and 20% require mechanical ventilation.[1,2,6]

The typical symptoms of Guillain-Barré syndrome (GBS) are bilateral, symmetrical, progressive, rapid motor weakness in combination with areflexia or hyporeflexia.[8-10] The weakness is classically described as ascending and usually starts in the distal lower extremities, but may start more proximally in the legs or arms.[11-13] The diagnosis of GBS is based on the patient's history and neurological, electrophysiological and cerebrospinal fluid examinations.[2,5] Specific management of GBS includes TPE and intravenous immunoglobulin (IVIG). If the patient becomes unable to walk unaided or if there is a significant reduction in pulmonary vital capacity or signs of severe oropharyngeal weakness, TPE or IVIG should be performed immediately. This usually occurs on the fifth to tenth day after the first appearance of symptoms but may be as early as one day or as late as 3 weeks from the initial onset.

In this case, the patient received IVIG therapy on the second day of ICU admission, which was 12 days from the onset of limb weakness. Intravenous immunoglobulin is derived from a plasma pool of 10,000 to 100,000 donor blood and plasma. Intravenous immunoglobulin may contain more than 10,000,000 specific antibodies. Natural antibodies and anti-idiotypic autoantibodies are prominent, but other immunological molecules, such as cytokines, soluble receptors, and soluble CD4, CD8, and HLA molecules may also be contained in some IVIGs. In controlled trials, there are two treatment options that are currently considered the standard of care in GBS, namely intravenous immunoglobulin (IVIG) and TPE. IVIG is given at 2 grams/kilogram divided over 5 days.[10,11] TPE and IVIG have been shown to be equally effective. Effects appear if either treatment is given within 4 weeks, but stronger effects may be present if the treatment is given within two weeks.[8-11]

Pneumonia is one of the most common complications of GBS. Nosocomial pneumonia is an infection of the lung parenchyma caused by pathogens in the hospital and occurs after 48 hours of hospitalisation. VAP is one of the nosocomial pneumonia in the ICU that occurs in patients who are on mechanical ventilators for more than 48 hours.4,5 In a study at Arifin Achmad Hospital in Riau, the incidence of VAP reached 18.58% in 2013-2014.[12,13] Another study conducted at RSCM, the mortality rate of patients with VAP reached 57.2% in 2003-2012. A recent study reported the mortality rate due to VAP was 9-13% in surgical patients, as well as patients with moderate general illness at the time of admission.[14,15]

Most nosocomial pneumonia is caused by more than one pathogen. The microorganisms found differ based on ICU germ type patterns, duration of ICU stay as well as regular hospitalisation and specific diagnostic methods. Pathogens causing VAP are often MDR aerobic pathogens, including P. aeruginosa, Acinetobacter species, carbapenemase-containing Klebsiella pneumoniae, and MRSA. Other potentially MDR pathogens include S. maltophilia and Burkholderia cepacia. On the other hand, S. pneumoniae, H. influenzae, methicillin-sensitive S. aureus (MSSA), and antibiotic-sensitive Enterobacteriaceae are not MDR.[16-18]

The diagnosis of VAP is established by several criteria, namely: an increase in the inspiratory fraction of oxygen or positive end-expiratory pressure; and fever or hypothermia or leukocytosis or leukopenia; and new antibiotics started and positive airway culture or other criteria, but not including radiological criteria.[19,20]. The scoring method using the Clinical Pulmonary Infection Score (CPIS) is widely used because it has a high correlation rate with quantitative culture with samples from bronchoalveolar lavage (BAL) If the score is greater than/equal to 6, the patient is identified as VAP.

Table 1. Clinical Pulmonary Infection Score (CPIS)

Criteria	0	1	2
Tracheal secretions	None	Not purulent	Profuse and purulent
Infiltrates on thoracic X-ray	None	Diffuse	Localised
Temperature	≥36.5 and ≤38.4	≥38.5 or ≤38.9	≥39 or ≤36
Leukocytes	≥4,000 and ≤11,000	<4,000 or >11,000	<4,000 or >11,000 + immature neutrophils >50% or >500
PaO2/FiO2	>240 or ARDS		≤240, without ARDS
Microbiology	Negative		Positive

In this case, the patient initially experienced HAP but experienced improvement, on day 6 the patient had a fever, increased sputum secretions accompanied by a decrease in P / F ratio with a leukocyte examination of 15,000 / mm3 with a CPIS score of 6. The patient was given empirical antibiotics Vancomycin and Meropenem.The principle of antibiotic therapy in cases of VAP is to achieve the best therapeutic effectiveness, as well as prevent excessive antibiotic use. The latest guidelines from the American Thoracic Society and Infectious Diseases Society of America (ATS/IDSA) in the management of VAP recommend the selection of empirical antibiotic therapy should be based on the time of occurrence and risk factors for MDR pathogens (Table 2). In addition, germ type patterns and local resistance patterns also need to be considered.[16 ,20]

Table 2. Risk factors of MDR pathogens causing nosocomial pneumonia

No.	Risk Factors
1	Antibiotic therapy in the last 90 days
2	Treatment period greater than/equal to 5 days
3	Multiple antibiotic resistance in the community or in one of the hospital units
4	There are risk factors for pneumonia related to health facilities: - Hospitalised for more than/equal to 2 days in the last 90 days - Living in a nursing home - Home infusion therapy (including antibiotics) - Chronic dialysis in the last 30 days - Home wound care - Family member has an MDR pathogen
5	Immunosuppressive disease and/or therapy

In this case, the patient had risk factors such as receiving antibiotics in the last 90 days with a treatment period of more than/equal to 5 days, there were risk factors for pneumonia related to health facilities, namely hospitalisation >2 days. Empirical therapy should be stopped immediately if the microbiological examination results are out to prevent germ resistance in the hospital. If gram-negative bacteria are present, it is recommended to stick to one antibiotic as the use of combination antibiotics does not improve outcomes.[20]

After 72 hours, antibiotics should be adjusted according to the results of microbiological examination. In this case, on the 10th day of treatment, culture results were obtained for Candida albicans, so the patient was given fluconazole therapy 1 x 200 mg iv. On the 15th day of treatment, antibiotics were given according to culture, namely Ampicillin Sulbactam 3 x 1.5 grams intravenously. Antibiotic therapy took 3 days to achieve clinical improvement. Treatment failure was assessed on days 3 to 5 of antibiotic administration. Definitions of therapeutic failure include: (1) Improvement in PaO2/FiO2, or need for intubation due to pneumonia; (2)

Persistent fever and hypothermia with purulent airway secretions; (3) Infiltrates on chest X-ray increased by more than/equal to 50%; (4) Sepsis shock or multiple organ dysfunction syndrome occurs.

In this case, the VAP was well treated with antibiotics and antifungals according to the culture results. Percutaneous dilatational tracheostomy (PDT) on day 8 also contributed to ease the patient off the ventilator. Early tracheostomy placement in mechanically ventilated critically ill patients is associated with lower mortality and length of stay in several metaanalyses. Tracheostomy can reduce airway space loss and resistance, work of breathing, sedation drug requirements, and oropharyngeal and laryngeal lesions. It also facilitates care and drainage of secretions, improves oral nutrition, provides patient comfort, and improves communication.[21,22] need for mechanical ventilation therapy. Nosocomial pneumonia is a common complication in patients in critical condition and the leading cause of death from nosocomial infections, especially ventilator-associated pneumonia in intubated patients.

CONCLUSION

GBS is a post-infectious autoimmune disease that results in nerve cell destruction. Severe muscle weakness can lead to respiratory failure resulting in the need for mechanical ventilation therapy. Nosocomial pneumonia is a common complication in patients in critical condition and the leading cause of death from nosocomial infections, especially ventilator-associated pneumonia in intubated patients. The primary therapy should be performed as early as possible to achieve an effective outcome. In case of complications of ventilator-associated pneumonia, the causative germ should be considered based on the local ICU germ pattern, duration of hospitalisation, and previous antibiotic therapy. VAP caused by MDR microorganisms accounts for the highest morbidity and mortality rates. Early tracheostomy placement can also help reduce the length of stay on mechanical ventilation and improve patient outcomes.

DECLARATIONS

The research has received approval from the Faculty of Medicine, Padjadjaran University and Dr Hasan Sadikin Central General Hospital of Health Research and Ethics Committee. Participants was informed about subject of this case.

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The Authors agree to publication in Journal of Society Medicine.

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

AUTHORS' CONTRIBUTIONS

All authors are responsible for conceptualization, manuscript preparation, manuscript editing, and manuscript assurance.

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