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CROSS-REACTIVITY OF GUILLAIN-BARRE SYNDROME AND THROMBOTIC THROMBOCYTOPENIC PURPURA WITH COMPLICATIONS RESPIRATORY FAILURE: CASE REPORT

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ABSTRACT
Guillain-Barre Syndrome (GBS) and Thrombotic Thrombocytopenic Purpura (TTP) are two medical conditions that are rare but can cause serious complications if they appear simultaneously. Both conditions involve autoimmune mechanisms and can result in organ failure, including respiratory failure. Although there are several case reports of each condition, research exploring the interaction between GBS and TTP in the context of respiratory failure is limited. Purpose: The aim of this study is to describe a case of cross-reactivity of Guillian-Barre Syndrome and Thrombotic Thrombocytopenic Purpura with complications respiratory failure. Methods: This research uses a case study design involving an adult patient who was treated at a hospital in Bandung. Data was collected through a comprehensive nursing assessment. Patients provided informed consent before data were collected. Data analysis was carried out descriptively to describe clinical findings and patient management results. Results: The patient is a young woman 27 year old admitted to hospital with a diagnosis of GBS. Eleven days before entering the hospital the patient experience dascending paralysis, fever, nausea and vomiting.TTP cross-reactivity and peripheral nervous system inflammation in GBS.Complications of respiratory failure requiring invasive mechanical ventilation (IMV) in the ICU. Laboratory and radiological examinations supported the diagnosis, and medical intervention was performed according to protocol for both conditions. This shows the importance of close monitoring and prompt treatment to prevent further complications. These findings emphasize the need for a multidisciplinary approach in managing patients with complex medical conditions. Conclusion: An in-depth understanding of the interaction between GBS and TTP is essential for effective clinical management, especially in preventing and managing complications of respiratory failure.

Keywords: guillain-barre syndrome; respiratory failure; thrombotic thrombocytopenic purpura

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INTRODUCTION

Guillain–Barre syndrome (GBS), a peripheral nervous system disease caused by autoimmune with symptoms of progressive muscle weakness. GBS is triggered by viral or bacterial infection and has the characteristics of acute symmetric ascending flaccid paralysis with or without sensory system involvement (Majumder & Basu, 2019). The annual global incidence of GBS is around 1 to 2 cases per 100,000 people each year, tends to occur more frequently in men than women, with the highest incidence at around 30-50 years of age, and the risk of occurrence increases with age (Mishra et al., 2017). Guillain–Barre syndromecharacterized by

symmetrical weakness, especially proximal versus distal, with hyporeflexia or areflexia without sensory or sphincter involvement involving cranial nerves and respiratory muscles (Ottaviani et al., 2020). GBS is often triggered by infectious or non-infectious processes, although the exact mechanism remains uncertain. A number of studies show that Campylobacter jejuni bacterial infection is often associated with GBS cases. Infection by this bacteria can cause symptoms in the digestive system or respiratory system (Jodjana & Adja, 2022).

GBS andThrombotic Thrombocytopenic Purpura(TTP) are both associated with bacterial and viral infections, and antibodies are formed against them which can result in cross-reactivity due to molecular mimicry (Hussain et al., 2022). The prevalence of TTP is around 10 cases/million people with 1 new case/million people every year(Mariotte et al., 2016).TTP is caused by disruption of the activity of the ADAMTS13 enzyme involved inblood clotting process. Deficiency of the ADAMTS13 enzyme causes the blood clotting process to become very active. The number of blood clots that occur causes the platelet count to decrease (thrombocytopenia) (George & Nester, 2014). Platelets are thought to provide a connection between the blood and the nervous system in nervous system diseases (Leonhard et al., 2019). This facilitates cross-reactivity between platelets in TTP and peripheral nervous system inflammation in GBS (Hagen & Ousman, 2021).

Complications that occur in patients with Guillain-Barre Syndrome (GBS) are respiratory failure which requires mechanical ventilation assistance. This can cause complications in the form of pneumonia, sepsis, as well as Acute Respiratory Distress Syndrome (ARDS), arrhythmias and cardiac arrest. Patients who require mechanical ventilation have a higher mortality rate (8.5%) compared to those who do not require mechanical ventilation (5%-6.5%) (Setiari & Sudjud, 2018).The need for mechanical ventilation in GBS patients is a significant concern, and it has been emphasized that respiratory failure is the most serious short-term complication of GBS (Rashid et al., 2017).

GBS and TTP are two conditions that involve both the immune and hematological systems, with potential cross-reactivity between the two (Hussain et al., 2022). GBS is an autoimmune disorder that attacks the peripheral nervous system, causing muscle weakness and paralysis, while TTP is a hematological condition characterized by the formation of micro-thrombi in small blood vessels, resulting in thrombocytopenia and hemolytic anemia (Mariotte et al., 2016). Theoretically, the two may interact due to underlying autoimmune mechanisms; Abnormal immune response in GBS can trigger endothelial damage, which is a major triggering factor in the pathogenesis of TTP (Nicolotti et al., 2021). Previous study have indicated a link between autoimmune and hematological conditions, where a hyperactive or dysfunctional immune system can lead to a variety of overlapping clinical manifestations (Hindilerden et al., 2020). Some study also suggest that certain infections or environmental factors can trigger autoimmune cross-reactions, worsening both conditions simultaneously (Nicolotti et al., 2021).

This study is of high significance as understanding the interaction between GBS and TTP in the context of respiratory failure is crucial for the management of patients with complex conditions. Respiratory failure as a complication of cross-reactivity between GBS and TTP can increase patient morbidity and mortality, so in-depth knowledge of the mechanisms and management of this condition is needed (Wang et al., 2020). This study provides direct benefits to nursing practice, especially in identifying and effectively responding to complications that arise in patients with such a dual diagnosis. The case study method was

chosen because it allows in-depth exploration of clinical details and patient management dynamics, providing rich and specific insights that may be missed in studies with other methods. The aim of this study is to describe a case of cross-reactivity of GBS and TTP with complications of respiratory failure.

METHOD

This research used a case study design to explore complex clinical conditions in a patient at a hospital in Bandung. The case study stages included data collection, data analysis, and interpretation of results in a specific clinical context. The study was conducted on one patient who experienced cross-reactivity between GBS and TTP with complications of respiratory failure. Data collection was carried out through a comprehensive nursing assessment, including history taking, physical examination, and review of laboratory and radiology results. The patient provided informed consent before data collection began, ensuring that they understood the purpose of the study and agreed to participate. Data analysis was carried out descriptively, describing clinical findings and assessment results in detail to understand the patient's condition and the effects of the intervention provided..

RESULTS

A women with 27 year old referral patient was admitted to the hospital on April 7 2024 for plasmapheresis. Eleven days before admission, the patient experienced tingling and weakness in both legs and found it difficult to move. The results of the physical examination showed bilateral motor weakness with motor strength 0/5, which developed rapidly and increased in all four extremities the following day. Complaints of Bell's palsy and slurred speech four days before entering the hospital. Complaints of pain in the neck radiating to the shoulders, chest pain and low back pain. Complaints of dizziness, ringing in the ears, double vision (diplopia). The patient experienced fever, nausea and vomiting ± 7 days before entering the hospital. There is a history of enlargement of the thyroid gland since ± 9 years ago and was treated in hospital, but for ± 5 years he has not taken medication and has been controlling his thyroid disease. There was no history of trauma, defecation (liquid, black, blood), and changes in behavior. The patient experienced decreased consciousness accompanied by shortness of breath and finally underwent intubation and mechanical ventilation (IMV).

The patient was admitted to the emergency room on April 7 2024 at 22.30 in a state of mechanical ventilation (IMV). From the results of the history and physical examination, the patient was diagnosed with GBS and action was planned plasmapheresis. Results of the physical examination of the Coma patient's consciousness, GCS (E1M1Vett). BP 122/58 mmHg, pulse 109 x/minute, SB 36 BB: 60 kg, Ventilator mode: P-SIMV, PS: 15, I:E Ratio 12, PEEP: 5, IPL;17, FiO2: 50%, RR 12x/minute, Tv: 480, MV: 8.6 L, Uk ETT: 6.5 mm, ETT Depth: 19 cm Lip line, Suction+, SPO2: 98%, Pain (CPOT/1), Catheter Inserted, Urine; 755 cc/9 hours, nasogastric tube (NGT),Intravenous Fluid Drops(IVFD), and bedside monitors. The patient underwent supporting examinations including laboratory examinations and chest x-rays. The patient was then admitted to the Intensive Care Room on April 8 2024 at 00.51 in the Intensive Care Room for intensive care and the plan to insert a Double Lumen Catheter (CDL) for plasmapheresis. Plasmapheresis therapy is planned 5 times. The patient received intravenous fluids and drugs therapy: Ringer's Lactate 1,500 cc/24 hours and mecobalamin 500 mg/8 hours/iv.

The laboratory examination of the patient revealed significant abnormalities. The hemoglobin level was 8.6 g/dl, which is below the normal range of 12.3 - 15.3 g/dl, indicating anemia. The hematocrit level was also low at 28.1%, compared to the normal range of 36.0 - 45.0%, further

supporting the presence of anemia. Leukocyte count was elevated at 17.50 x 10^3/ul, above the normal range of 4.4 - 11.3 x 10^3/ul, suggesting an inflammatory or infectious process. The erythrocyte count was 3.1 million/ul, which is below the normal range of 4.5 - 5.1 million/ul, consistent with anemia. The thrombocyte count was markedly low at 37 thousand/ul, significantly below the normal range of 150 - 450 thousand/ul, indicating thrombocytopenia. Renal function tests showed a ureum level of 107.5 mg/dl, far above the normal range of 15 - 40 mg/dl, indicating possible renal impairment. The albumin level was 2.14 g/dl, which is below the normal range of 3.5 - 5.2 g/dl, indicating hypoalbuminemia. Coagulation tests revealed a prolonged prothrombin time (PT) of 18.9 seconds (normal: 12 - 16 seconds) and an international normalized ratio (INR) of 1.32 (normal: 0.8 - 1.2), indicating a potential bleeding risk. The activated partial thromboplastin time (APTT) was also prolonged at 48.00 seconds (normal: 28.4 - 44.6 seconds).

The D-Dimer quantitative result was significantly elevated at 3.83 ug/ml, above the normal value of < 0.55 ug/ml, suggesting a high level of fibrin degradation products, which is indicative of thrombotic activity. Arterial blood gas analysis revealed a pH of 7.298 (normal: 7.35 - 7.45), indicating acidosis. The partial pressure of carbon dioxide (pCO2) was elevated at 62.9 mmHg (normal: 35.0 - 45.0 mmHg), indicating respiratory acidosis. The partial pressure of oxygen (pO2) was 117.8 mmHg (normal: 80 - 105 mmHg), possibly due to supplemental oxygen. The bicarbonate (HCO3) level was 31.1 mmol/L (normal: 22 - 26 mmol/L), and total CO2 (tCO2) was 33.0 mmol/L (normal: 23.05 – 27.35 mmol/L), both indicating a compensatory metabolic alkalosis. The standard base excess (BE-b) was 4.4 mmol/L (normal: -2 to +2), indicating metabolic compensation. Oxygen saturation (O2 sat) was 96.9%, within the normal range of 95 - 100%. The initial chest X-ray on April 8, 2024, showed right-sided bronchopneumonia. A follow-up chest X-ray on April 13, 2024, indicated an improvement in the right-sided bronchopneumonia; however, a new finding of left-sided pleural effusion was noted. There was no evidence of cardiomegaly. Another chest X-ray on April 14, 2024, continued to show improvement in the right-sided bronchopneumonia with no signs of cardiomegaly.

DISCUSSION

The patient in this case was admitted to the hospital with complaints of tingling and weakness in both legs and difficulty in moving. The results of the physical examination showed bilateral motor weakness with motor strength 0/5, which progressed rapidly and increased in all four extremities (tetraplegia) the following day. From the results of other studies, involvement of the sensory and cranial nervous systems was found, namely facial palsy, slurred speech, and double vision (diplopia) which occurred before entering the hospital. There were also reports of complaints of pain in the neck radiating to the shoulders, chest pain and low back pain. Based on clinical manifestations, history and physical examination, it suggests GBS. The standard criteria for a diagnosis of GBS according to the National Institute of Neurological and Communicative Diseases (NINCDS) include progressive, relatively symmetrical muscle weakness with or without sensory involvement and hyporeflexia (Galassi & Marchioni, 2020).

The findings of this research indicate that cross-reactivity between GBS and TTP can lead to serious complications such as respiratory failure. This highlights the importance of accurate diagnosis and timely medical intervention. Close monitoring is essential to detect changes in the patient's condition early, while rapid management with invasive mechanical ventilation (IMV) in the ICU is crucial in preventing mortality (Galassi & Marchioni, 2020). The study also underscores the necessity of a multidisciplinary approach in managing patients with complex conditions, where collaboration among various medical specialists is required to

provide comprehensive care (Yadav et al., 2022). Consequently, this research offers valuable insights for clinical practice in handling patients with intricate medical conditions and reinforces the importance of integrating diagnosis and evidence-based therapy (Sukumar et al., 2021).

GBS is an acute monophasic demyelinating neuropathy characterized by progressive motor weakness of the affected limbs.accompaniedwith areflexia. This condition is initially preceded by infection, viruses and bacteriawhich attacks the peripheral nerves. From the results of the study, the patient experienced fever, nausea and vomiting ± 7 days before entering Hospitalbronchopneumoniaright. Patients with GBS have a history of respiratory and gastrointestinal infections,up to 70% of patients have reportedwithin 1 to 6 weeks before disease onset (Wu et al., 2022). TTP is caused by disruption of the activity of the ADAMTS13 enzyme involved inblood clotting process. Deficiency of the ADAMTS13 enzyme causes the blood clotting process to become very active (Albiol et al., 2020). ADAMTS13 deficiency results in unfractionated von Willebrand multimers (VWF), a protein involved in blood clotting at the site of injury, with additional triggers resulting in increased platelet aggregation. The number of blood clots that occur causes the platelet count to decrease (thrombocytopenia) (George & Nester, 2014).

From the results of the patient's laboratory examination, the platelet value was 37 103/ul.Among various cellular components and blood proteins, platelets are thought to provide a link between the blood and the nervous system in nervous system diseases (Sukumar et al., 2021). Evidenced by the invasion and deposition of platelets in peripheral nervous system inflammation (Orian et al., 2021). The concept that platelets are involved in neuroinflammation is supported by the high degree of vascularization of the nervous system, which facilitates cross-reactivity between platelets in TTP and peripheral nervous system inflammation in GBS (Hindilerden et al., 2020).

The interaction between platelets and the peripheral nervous system in GBS involves crosstalk between platelets,leukocytes, and neutrophilsactivated by blood circulation. In inflammatory conditions, platelets also release extracellular vesicles (EVs) which act as complementary pro-inflammatory mediators, which are able to infiltrate tissues and capture and activate neutrophils (Nicolotti et al., 2021). The functional capacity of platelets to act as inflammatory mediators and potentially modulate neuroinflammation is supported by the fact that platelets are the first cells to reach sites of injury or inflammation (Melone et al., 2020). Neuroinflammation, which contributes to the pathophysiology of various neurological diseases, consists of a series of accumulating damages involving cellular and molecular components, including the generation of reactive oxygen species, the release of cytokines and chemokines, the unfolding and aggregation of neuronal proteins, and neuronal cell death (Longhini et al., 2019).

Extremity weakness (98%) is the most common manifestation, followed by dyspnea (47%) and sensory involvement (39%) which often occurs in demyelinating conditions (Jodjana & Adja, 2022). Muscle weakness in cases of GBS causes type 2 respiratory failure and is a medical emergency that requires immediate treatment (Setiari & Sudjud, 2018). In addition, cross-reactivationplatelets contribute to platelet aggregation, recruitment of circulating platelets, and increased coagulation leading to arterial thrombosis (Gangula et al., 2017). Complications of GBS are dysautonomy or severe impairment of autonomic function.Clinical predictors of dysautonomy are quadriplegia, neck or bulbar flexor weakness, and use of invasive mechanical ventilation (IMV).This dysautonomy will later trigger arrhythmias,

cardiac arrest and respiratory failure (Walgaard et al., 2017). Respiratory failure is a critical and potentially life-threatening complication in patients with GBS, occurring in 20-45% of cases (Wen et al., 2021).Respiratory system dysfunction in GBS is caused by involvement of the phrenic nerve and loss of motor innervation to the intercostal muscles, abdomen and accessory respiratory muscles (Galassi & Marchioni, 2020). Weakness of the diaphragm and oropharynx exacerbates upper airway stenosis and increases respiratory load (Melone et al., 2020).

The patient's clinical improvement experienced significant changes after receiving intensive treatment. The patient's consciousness becomes compos mentis with GCS (E4M6Vett) and using a ventilator: PSV, PS: 10, I:E, Ratio 1:2, PEEP: 5, IPL;17, FiO2: 50%. However, the weaning process cannot yet be carried out. This is influenced autonomy in GBS which exacerbates respiratory distress through decreasing airway vagal tone, ventilatory response to hypoxia and hypercapnia, and limiting the effects of anticholinergic drugs (Scheidl et al., 2020). Muscle weakness, axonal degeneration, and non-excitable nerves (i.e. sciatic, median, ulnar, peroneal nerves) are predictive signs for prolonged ventilation (Dalakas, 2020).

CONCLUSION

From the cases presented in this report, it can be concluded that GBS and Thrombotic Thrombocytopenic Purpura (TTP) are two medical conditions that can occur simultaneously in a patient. GBS is characterized by symptoms of muscle weakness that develop rapidly and can cause complications such as respiratory problems, while TTP is caused by impaired enzyme activity which results in a decrease in the number of platelets in the blood. Cross-reactivity between these two conditions can worsen the patient's condition and requires appropriate medical treatment. Complications of respiratory failure in this case indicated a serious escalation of the condition and required intensive care in the ICU with the installation of invasive mechanical ventilation (IMV). This is important to ensure adequate oxygen supply to the patient's lungs and body, as well as assisting in maintaining optimal respiratory function. With proper treatment and appropriate therapy, it is hoped that patients can recover from GBS and TTP conditions complicated by respiratory failure.

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