Comprehensive Management of Guillain-Barré Syndrome with Associated Cardiac Complications: A Case Report

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ABSTRACT:

Guillain-Barré syndrome (GBS) is an immune-mediated disorder characterized by sudden muscle weakness and paralysis. We present a case of a 44-year-old female with the areflexic acute motor and sensory axonal neuropathy (AMSAN) variant of GBS, accompanied by severe tricuspid regurgitation. The patient experienced persistent lower limb pain, generalized weakness, and exhibited physical findings such as exophthalmos, pallor, and pitting edema. Diagnostic evaluations revealed cardiac abnormalities, uric acid crystals in urine, elevated liver enzymes, and increased cerebrospinal fluid protein levels. The patient was managed using a multidisciplinary approach, including vitamin supplementation, intravenous immunoglobulin administration, dialysis physiotherapy, and hypertension management. This case emphasizes the importance of a comprehensive management strategy for GBS patients with associated cardiac complications. Further research is warranted to enhance our understanding of the relationship between GBS variants and cardiac abnormalities, enabling improved clinical management.

INTRODUCTION:

The most prevalent cause of abrupt muscle weakness and paralysis is Guillain-Barré syndrome (GBS). It was found more than a century ago. Over the past 100 years, significant progress has been made in understanding the immune-related causes of the disease, recognising the various ways it might manifest, improving diagnosing techniques, forecasting outcomes, and testing treatments in clinical trials. Because of the potential harm that this uncommon ailment could inflict if left untreated, it is essential for all clinicians to be knowledgeable about it.[1]GBS and its variations are immune-mediated neuropathies that frequently develop following an infection. The Zika virus, respiratory diseases, and Campylobacter jejuni have all been related to GBS. Nerves may unintentionally be attacked by the immunological response that is triggered to fight these illnesses. Additionally, surgeries, drugs, and, in a very small number of cases, flu shots have all been linked to GBS. However, the chance of getting GBS after contracting the flu is higher than it is after getting the flu shot[3][4][5].A neurological condition called Guillain-Barré syndrome (GBS) is characterised by paralysis and muscle weakness. According to research, up to 70% of GBS patients report having had prior infections, suggesting a connection between infections and the development of the condition. Molecular mimicry is one theory put forth, whereby the immune response to an infection incorrectly targets peripheral nerve components, resulting in harm. An illustration of molecular mimicry is seen in infections brought on by the

widespread bacterial disease Campylobacter jejuni.a typical pathogenic microorganism. Campylobacter jejuni's lipooligosaccharide and the gangliosides present in peripheral nerve membranes have comparable structural features. The axonal form of GBS, specifically acute motor axonal neuropathy, has been mimicked in clinical symptoms in rabbits passively immunised with these ganglioside-like lipooligosaccharides [6][7].It has been discovered that ganglioside antibodies target different peripheral nerve organelles.The nodes of Ranvier, the neuromuscular junction, and paranodal myelin can all be accessed by antibodies against GD1a [8]. Peripheral neurons or the neuromuscular junction have been discovered to bind to antibodies against GM1 and GQ1B [9].The various clinical manifestations seen in GBS may be influenced by the different targets that these antibodies have. For instance, anti-GQ1B antibodies are linked to Miller-Fisher syndrome, a form of GBS. Similar to this, anti-GM1 antibodies in GBS may be linked to the axonal motor neuropathy type, regardless of whether they cause the condition or are merely an epiphenomenon. Additionally, the pathophysiology of the most prevalent variety of GBS in the United States, acute inflammatory demyelinating polyneuropathy (AIDP), is still poorly understood.IVIG and plasma exchange are two treatments for Guillain-Barré syndrome (GBS) that are regarded as standard of care.The immune system is modulated by IVIG, which is administered over 5 days at a dose of 2 grams/kilogram, however the precise mechanism is still unclear [10]. Over the course of five sessions, plasma exchange removes pathogenic antibodies, humoral mediators, and complement proteins. Both therapies have demonstrated comparable efficacy. Early treatment beginning within two weeks can have a bigger impact. Contrary to placebo, IVIG, or plasma exchange, corticosteroids have not been shown to be superior to those treatments [11].Up to 85% of GBS patients successfully recover, and the majority of them are able to walk independently; nevertheless, 20% of patients may still have some residual morbidity. Additional research on therapy combinations has not yielded appreciable gains. Results from a trial employing two cycles of IVIG are anticipated soon [12], and ongoing studies are looking into the use of complement inhibitors in refractory GBS.Critical disease neuropathy/myopathy, tick paralysis, acute intermittent porphyria, HIV infection, spinal cord disorders, toxic neuropathies, and some infections are additional diagnosis for GBS. To identify GBS from its imitators, clinical assessment, history, and auxiliary tests, including electromyography and cerebrospinal fluid studies, are crucial [13].

CASE PRESENTATION:

A 44-year-old female presented to the emergency department with a chief complaint of pain in her lower left limb persisting for the past 12 days. The pain was described as non-radiating and extended from the thigh to the foot. The patient also reported generalized weakness. Her past medical history included hypertension.Upon examination, the patient was conscious, coherent, and had a pulse rate of 64 beats per minute. Her blood pressure was 110/60 mmHg, and her random blood sugar level was 178 mg/dL. Physical examination revealed the presence of exophthalmos (protrusion of the eyeballs), pallor, and pitting edema.Further investigations were conducted to evaluate the patient's condition. Echocardiography revealed mild mitral regurgitation, severe tricuspid regurgitation, and mild pulmonary arterial hypertension. An

ultrasound scan of the abdomen showed no abnormalities. Complete urine examination revealed the presence of uric acid crystals. The urine protein values were found to be 26%, with a urine creatinine level of 44 mg%. The protein to creatinine ratio was calculated to be 0.5. Liver function tests indicated an increase in aspartate transaminase (AST) levels, with a value of 74 U/L. CT scans of the abdomen and chest did not reveal any acute findings. To further assess the patient, a lumbar puncture was performed, which indicated increased cerebrospinal fluid protein values.Based on the comprehensive evaluation, the patient was diagnosed with the areflexic acute motor and sensory axonal neuropathy (AMSAN) variant of Guillain-Barré syndrome (GBS) in the context of severe tricuspid regurgitation.The management approach for this patient involved a multidisciplinary approach. Vitamin supplements were administered to support the patient's nutritional status. Intravenous immunoglobulin (IVIG) was given to address the underlying autoimmune response associated with GBS. Dialysis physiotherapy sessions were initiated to aid in the patient's rehabilitation and improve motor function. Additionally, the patient's hypertension was managed with the administration of intravenous furosemide.

DISCUSSION:

Guillain-Barré syndrome (GBS) is an immune-mediated disorder characterized by sudden muscle weakness and paralysis. It can occur following various infections, including Campylobacter jejuni, respiratory illnesses, and Zika virus, as well as after certain medications, surgeries, and rarely, flu vaccinations [1][2][3][4][5]. The immune response triggered during these infections or immunological triggers can mistakenly attack the peripheral nerves, leading to nerve damage and the development of GBS [6].In our case, the patient presented with the areflexic acute motor and sensory axonal neuropathy (AMSAN) variant of GBS. This variant is characterized by severe motor and sensory deficits, and it is associated with a poor prognosis compared to other GBS subtypes. The patient experienced persistent lower limb pain, generalized weakness, and exhibited physical findings such as exophthalmos, pallor, and pitting edema. These clinical features are consistent with the typical presentation of GBS and highlight the importance of recognizing the diverse clinical manifestations of this syndrome.Cardiac involvement in GBS is a rare but recognized complication. In this case, the patient exhibited cardiac abnormalities, including severe tricuspid regurgitation, which further highlights the need for comprehensive management in GBS patients. Although the exact mechanism underlying the cardiac complications in GBS is not fully understood, it is postulated that the immune-mediated inflammation targeting peripheral nerves may also affect cardiac autonomic nerves, leading to cardiac dysfunction.The management of GBS involves a multidisciplinary approach aimed at addressing the underlying autoimmune response, providing supportive care, and promoting recovery. In our case, the patient received intravenous immunoglobulin (IVIG) as the standard treatment for GBS. IVIG acts by modulating the immune system, although the exact mechanism is not fully understood [10]. It has been shown to be effective in reducing the severity and duration of symptoms in GBS patients. Additionally, vitamin supplementation was administered to support the patient's nutritional status, and dialysis physiotherapy sessions were initiated to aid in rehabilitation and improve motor function. Hypertension was managed with intravenous furosemide. The prognosis of GBS varies among patients, with the majority recovering well and achieving independent ambulation. However, approximately 20% of patients may experience

residual morbidity, such as motor deficits or sensory abnormalities. Early initiation of treatment, within two weeks of symptom onset, is associated with better outcomes. Ongoing trials are investigating novel therapeutic approaches, including complement inhibitors, for refractory GBS cases [11].

Conclusion:

This case emphasizes the importance of a comprehensive management strategy for GBS patients, particularly those with associated cardiac complications. GBS is a rare condition that requires prompt recognition, diagnosis, and appropriate treatment. Further research is needed to enhance our understanding of the relationship between GBS variants and cardiac abnormalities, leading to improved clinical management and outcomes.

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