Chettinad Health City Medical Journal
Volume 14, Issue 1 - 2025, Pg. No. 103-105
Peer Reviewed & Open Access Journal

Case Study

The Clinical Odyssey: Exploring the Diagnosis and Management of Acute Motor Axonal Neuropathy (AMAN) in Guillain-Barré Syndrome: A Case Study

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DOI: https://doi.org/10.24321/2278.2044.202514

I N F O

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How to cite this article:
Radhanpura Y, Gadhiya H, Tirgar P. The Clinical Odyssey: Exploring the Diagnosis and Management of Acute Motor Axonal Neuropathy (AMAN) in Guillain-Barré Syndrome: A Case Study. Chettinad Health City Med J. 2025;14(1):103-105.

Date of Submission: 2023-10-17
Date of Acceptance: 2024-01-29

A B S T R A C T

The complex degenerative neurological condition known as Guillain-Barre syndrome (GBS) can be acute in origin. It is an acquired syndrome that manifests as gradual, symmetrical tingling and weakening both proximally and distally. There is often sensory loss and reduced or absent nerve stretch responses. In spinal nerve roots, depolarisation is a disease, even though the cause is yet unclear. Early detection and prompt referral are crucial in critical cases since some individuals may experience respiratory problems and cardiovascular instability. A case of a 21-year-old female presenting with Acute Motor Axonal Neuropathy (AMAN), a subtype of Guillain-Barre Syndrome (GBS) with seizures has been presented here. The importance of a correct diagnosis by the chiropractor and the subsequent management is reviewed.

Keywords: Acute Motor Axonal Neuropathy (AMAN), Guillain-Barre Syndrome (GBS), Cerebrospinal Fluid (CSF), Intravenous Immunoglobulin (IVIG)

Introduction

The most common reason for sudden languid paralysis is Guillain-Barré syndrome (GBS), an inflammatory illness affecting the peripheral nerves, with an annual 1–2 occurrences per 100,000 person-years worldwide. Although GBS can affect people of any age, it affects men more commonly than women and the frequency rises with age.[1] Patients with GBS frequently have sensory complaints and weakness that radiates from their legs into their hands and cranial muscles, despite the disease's changing clinical presentation and several distinct clinical variants. The diagnosis of GBS is made using the patient's medical history in conjunction with neural, neuroanatomical, and cerebrospinal fluid (CSF) studies.[2,3] It's important to rule out any conditions with a similar clinical presentation to GBS.[3] Acute motor axonal neuropathy (AMAN), acute inflammatory demyelinating polyradiculoneuropathy (AIDP), and acute motor sensory axonal neuropathy (AMSAN) are the subtypes of GBS, may be distinguished using electrophysiological investigations, which show Peripheral Nervous System (PNS) dysfunction.[4] Rapid disease development can happen, and most GBS patients attain their maximal level of impairment in less than two weeks. Approximately 20% of GBS patients experience respiratory failure and need mechanical ventilation. The

autonomic nervous system can have a role in cardiac arrhythmias and blood pressure instability. Even with the finest medical treatment available, the death rate for people with GBS is estimated to be 3–10%. This is because the autonomic nerve system is involved.[5,6]

Case Report

A 21-year-old female from Rajkot, Gujarat diagnosed with GBS Acute Motor Axonal Neuropathy (AMAN) variety was admitted to the hospital. The symptoms included weakness of all four limbs, facial twitching, and convulsion with deviation of the right side of the mouth. The patient had a history of severe episodes of psychiatric attacks in childhood. She was on a mixed diet and had no significant family or personal history. She has no social history of using alcohol or smoking.

Cerebrospinal fluid (CSF) indicated a high protein level: 600 mg/100 mL (15–60 mg/100 mL) with < 5/uL of CSF WBC during spinal tap (lumbar puncture) test, thereby confirming GBS.

During hospitalisation; the patient was treated with intravenous immunoglobulin (IVIG) 0.4 g/kg bodyweight per day for five consecutive days, ceftriaxone 2 g once a day, levetiracetam 500 mg twice a day, KCL, and folic acid. For a speedy recovery, the patient was constantly under observation.

Discussion

The immune system of the body assaults a portion of the peripheral nerve system in GBS. The illness may also affect the nerves that control muscle contraction in addition to those that transmit pain, temperature, and sensations of touch. This may lead to breathing or swallowing issues, weakening of the muscles and/ or numbness in the hands, legs, or arms.

It is an uncommon disorder that may affect anyone of any age, although it tends to afflict adults and men more frequently.[7]

Most people recover without experiencing long-term and severe neurological problems. Weakness and tingling are generally the initial symptoms of GBS. Usually beginning in the legs, these symptoms might progress to the hands and face leading to paralysis of the arms, legs, or facial muscles in certain persons. The chest muscles in around one-third of persons are impaired, which makes breathing difficult. In extreme instances of GBS, speaking and swallowing abilities may be impacted. Since these conditions are thought to be life-threatening, those who are affected should get intensive treatment.[8]

Even those with the most severe GBS recover completely, while some people continue to feel weak. Even under ideal circumstances, a tiny percentage of GBS patients pass away from its consequences, which might include cardiac arrest, blood infection, lung clots, or paralysis of the breathing muscles.[8]

The diagnosis is made primarily on symptoms and neurological examination results, particularly the loss or diminution of deep-tendon reflexes. Although therapy should not be delayed, lumbar punctures and electromyography (EMG) can be performed to provide supportive data. GBS cannot be diagnosed without additional testing, such as blood work to determine the underlying cause, and treatment should not be postponed until this time. Respiratory problems should be constantly examined in anybody suspected of having GBS.[9]

GBS has the potential to be fatal. It is appropriate to admit GBS patients to the hospital so they may be carefully watched. Monitoring of respiration, pulse, and blood pressure are all part of supportive care. When someone's capacity to breathe is compromised, they are typically placed on a ventilator. The risk of consequences, which might include an irregular heartbeat, blood clots, infections, and elevated or lowered blood pressure, should be kept an eye on in all GBS patients. While there is no known therapy for GBS, there are ways to lessen its effects and alleviate its symptoms. Due to the autoimmune nature of the illness, immunotherapy is frequently used to treat the acute stage, such as plasma exchange to eliminate antibodies from the blood or intravenous immunoglobulin. When started seven to fourteen days after symptoms start, it is frequently effective. Patients may need rehabilitation therapy if muscle weakness continues after the acute stage of the disease in order to strengthen their muscles and regain mobility.[10]

Conclusion

In this case patient has a neurological condition called Guillain-Barre syndrome typically causes symmetrical muscular paralysis. Many patients may experience minor or severe involvement, and in a tiny number of cases, this may result in death. Patients with polyradiculoneuralgia symptoms may visit a chiropractic practice, where they may first be misdiagnosed as having radicular pain with a spinal origin. Potential severe cases must be quickly identified and referred in order to initiate the appropriate investigations (for instance, electrodiagnostic testing and spinal taps) and receive the proper care. The need for differential diagnosis cannot be overstated.

Conflict of Interest: None

Source of Funding: None

Authors' Contribution: YR collected the case details and prepared the article. He also gathered and typed the

ISSN: 2278-2044
DOI: https://doi.org/10.24321/2278.2044.202514

case-related information. HG handled the formatting and arrangement. PT guided the process, conducted the final check, and provided assistance whenever needed.

Declaration of Generative AI and AI-Assisted Technologies in the Writing Process: None

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ISSN: 2278-2044
DOI: https://doi.org/10.24321/2278.2044.202514