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Unmasking Guillain-Barré Syndrome: A Case Report and Clinical Insights

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ABSTRACT:
Guillain-Barré Syndrome (GBS) is an acute, immune-mediated polyneuropathy that often presents with rapidly progressive weakness, sensory abnormalities, and autonomic dysfunction. Though the condition is rare, it is a leading cause of acute flaccid paralysis worldwide. Although it is simple to diagnose classical Guillain-Barré syndrome, certain forms of the condition may go unnoticed due to the limitations of the current diagnostic criteria. Immunotherapy helps the majority of Guillain-Barré syndrome patients, however, there is a significant percentage of people who remain disabled and may even pass away. This case report discusses the presentation, diagnosis, and management of a patient with GBS, shedding light on the complexities of early recognition and treatment.

KEYWORDS: Barré syndrome, acute flaccid paralysis, neurology, miller fisher syndrome, electrophysiology, campylobacter jejuni, intravenous immunoglobulin, children, neurology, polyneuropathy

INTRODUCTION:

The most frequent cause of acute flaccid paralysis in the world is Guillain-Barré syndrome. Before increasing motor weakness, most patients come with an antecedent disease, typically an upper respiratory tract infection. Numerous microbes have been linked to Guillain-Barré syndrome, including Campylobacter jejuni, Zika virus, and, as of 2020, coronavirus 2 which causes severe acute respiratory syndrome. Strong evidence points to an autoantibody-mediated immune response in C jejuni-related Guillain-Barré syndrome, which is set off by molecular mimicry between the microbe and structural elements of peripheral nerves. [1,2]

Guillain-Barré syndrome usually manifests as tingling, increasing weakening, and discomfort. Forms frustes and variations may make identification more difficult. Miller-Fisher syndrome has several variations, the most well-known of which is the sensory ataxic variety, which also affects the brain stem and oculomotor nerves. Many pathogenic causes cause damage to the demyelinating, axonal, or mixed demyelinating-axonal regions. Unidentified antigens in the demyelinating form are deduced by complement activation, myelin breakdown, and cleaning by macrophages. Gangliosides (GM1, GD1a, and GQ1b) in the axonal and Miller Fisher variants are immunoglobulin-targeted and share antigenic epitopes with some viral and bacterial antigens.[3]

CASE REPORT:

A 9-year-old female patient weighing 23 kgs was admitted to the pediatric department with chief complaints of weakness in the bilateral lower limbs, tingling sensations, and difficulty walking. The parents reported that the patient had experienced frequent falls while walking over the past three days. Two weeks before admission, the patient had an upper respiratory illness characterized by a dry cough and low-grade fever, which was treated at a local clinic with full recovery. This prior illness is notable, as infections are common precursors to GBS, where the immune system may erroneously target the body's nerves in response to the infection. Within 24 hours of the onset of symptoms, the patient developed bilateral lower limb weakness. Over the next few days, her condition worsened, with the weakness gradually extending to the upper limbs. The patient also experienced paresthesia (abnormal sensations) in the lower limbs, leading to difficulty in walking and performing fine motor tasks.

Electrophysiological studies revealed significant demyelination and nerve damage, as evidenced by nerve conduction studies (NCS) and needle electromyography (EMG). Cerebrospinal fluid (CSF) analysis showed elevated protein levels (85 mg/dl) without pleocytosis, consistent with GBS. Other laboratory investigations were within normal limits, including complete blood count, renal and liver function tests, serum electrolytes, and inflammatory markers. Serological tests for preceding infections, such as Campylobacter jejuni, Epstein-Barr virus, and cytomegalovirus, were negative.

A diagnosis of Guillain-Barré syndrome (GBS) was made based on clinical presentation, electrophysiological studies, and CSF analysis. The patient was immediately started on intravenous immunoglobulin (IVIG) therapy, which is a standard treatment for GBS. [4,5] IVIG works by providing the patient with antibodies that can help modulate the immune response and hasten recovery. The patient received a course of IVIG at a dosage of 400 mg/kg over 14 days, administered in two sessions. Frequent neurological assessments to monitor the progression of weakness and respiratory function. Strict bed rest with frequent repositioning to prevent complications such as pressure sores and deep vein thrombosis. Adequate hydration and nutritional support to maintain optimal health. Physiotherapy was initiated early in the course of the illness to maintain joint mobility, prevent contractures, and promote functional recovery. The rehabilitation program was tailored to the patient's condition, with a focus on gradual improvement in muscle strength and coordination. However, some challenges were faced during the treatment process. The patient experienced some mild side effects from the IVIG therapy, including headache & fatigue.

Over the following weeks, the patient's muscle strength improved gradually, with motor function returning in a proximal-to-distal manner. After three months, she made significant progress, almost recovering with only slight weakness in the distal lower-limb muscles. Follow-up neurology consultations were planned to ensure long-term monitoring of her condition.

DISCUSSION:

Guillain-Barré Syndrome (GBS) is an acute, immune-mediated polyneuropathy often triggered by an infection that presents with rapid-onset muscle weakness and can progress to paralysis. [1] Though it can affect individuals of any age, GBS is relatively rare in children, making each pediatric case noteworthy for its clinical insights and management challenges.[6] Our case of a 9-year-old female represents the

importance of early recognition and management in younger patients. It also highlights the need for awareness among healthcare professionals regarding the variable presentation of GBS in children. Her complaint to the doctor was weakness in her bilateral lower limbs in the last 24 hours and worsening of condition over the next few days; she felt paresthesia in her lower limbs, The Weakness extended gradually to her upper limbs therefore she experienced difficulty in walking and performing fine motor tasks.[7] Some investigations were done to rule out the disease such as hematological profile, electrophysiological studies, cerebrospinal fluid analysis, and serological tests which indicated Guillain Barre's syndrome. According to the lab reports, the treatment plan was prescribed and initiated to treat the syndrome.

According to the previous studies, GBS frequently affects boys more than girls. Different ratio of male to female ratios has been reported for GBS. [8,9] Young children are more prone to infections contributing to the pathogenesis of GBS. Another explanation is higher susceptibility to demyelination in younger children.[10] The proportion of children with atypical clinical presentation of GBS was high with a rate of 24.2%.[11]

CONCLUSION:

Comparatively, paediatric GBS cases tend to have a better prognosis than adult cases.[7] However, response to the treatment varies with individualized care plans. Our case aligns with existing literature, demonstrating a typical clinical course and positive response to standard treatment protocols.[6] Some studies in this field have reported a higher rate of GBS in children younger than 5yr old, while others reported a higher rate in children aged 5-10 yr. Almost all the studies have reported low occurrence of GBS in children older than 10 yr old.[12-15] This case explains the importance of timely diagnosis, appropriate immunotherapy, and comprehensive supportive care in paediatric patients with GBS. Further research into paediatric-specific aspects of GBS could enhance understanding and treatment outcomes for the population.

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