Case Report / *Olgu Sunumu*

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Acute Motor Axonal Neuropathy Variant of Guillain-Barré Syndrome Following SARS-CoV-2 Infection in a 3-Year-Old: A Case Report

3 Yaşındaki Bir Çocukta SARS-CoV-2 Enfeksiyonu Sonrası Gelişen Akut Motor Aksonal Nöropati Varyantı ile Guillain-Barré Sendromu: Bir Olgu Sunumu

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Abstract

Guillain-Barré syndrome (GBS) is the most common cause of acute flaccid paralysis in children. Among its subtypes, acute motor axonal neuropathy (AMAN) is less frequent but often more severe. Recent studies suggest a possible association between severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infection and GBS, including axonal variants, in pediatric patients. We report a previously healthy three-year-old girl who developed the AMAN subtype of GBS following a recent SARS-CoV-2 infection. She initially presented with fever and upper respiratory tract symptoms. Within days, she developed progressive ascending weakness, neck flexor muscle weakness, and areflexia. Spinal magnetic resonance imaging revealed enhancement of the anterior nerve roots in the cauda equina. Cerebrospinal fluid analysis demonstrated albuminocytologic dissociation. Elektromyelography (EMG) was consistent with AMAN, and anti-GT1a immunoglobulin G antibodies were positive. The patient received intravenous immunoglobulin therapy, followed by therapeutic plasma exchange due to minimal initial improvement. Because of the severity and slow clinical response, repeated courses of immunotherapy were administered. Gradual motor improvement was observed over several weeks, and the patient eventually regained independent ambulation. This case highlights the potential of SARS-CoV-2 to act as

Öz

Guillain-Barré sendromu (GBS), çocuklarda akut flask paralizinin en yaygın nedenidir. Alt tipleri arasında akut motor aksonal nöropati (AMAN) daha nadir görülmekle birlikte genellikle daha ağır seyreder. Son çalışmalar, pediyatrik hastalarda şiddetli akut solunum yolu sendromu koronavirüs-2 (SARS-CoV-2) enfeksiyonu ile aksonal varyantlar da dahil olmak üzere GBS arasında olası bir ilişki olduğunu düşündürmektedir. Daha önce sağlıklı olan ve yakın zamanda geçirilen SARS-CoV-2 enfeksiyonunun ardından GBS'nin AMAN alt tipini geliştiren üç yaşında bir kız çocuğunu bildirmekteyiz. Hastamız başlangıçta ateş ve üst solunum yolu semptomlarıyla başvurdu. Günler içinde ilerleyici asendan güçsüzlük, boyun flexor kas güçsüzlüğü ve arefleksi gelişti. Spinal manyetik rezonans görüntüleme, kauda ekuinadaki ön sinir köklerinde kontrast tutulumu görüldü. Beyin omurilik sıvısı analizi, albüminositolojik dissosiyasyon mevcuttu. Elektromyografi (EMG) AMAN ile uyumluydu ve anti-GT1a immünoglobulin G antikorları pozitifti. Hastaya intravenöz immünoglobulin tedavisi uygulandı ve ardından başlangıçta gözlenen minimal iyileşmenin devam etmemesi nedeniyle terapötik plazma değişimi yapıldı. Hastalığın şiddeti ve yavaş klinik yanıt nedeniyle tekrarlayan immünoterapi kürleri uygulandı. Birkaç hafta içinde kademeli motor iyileşme gözlendi ve hasta sonunda bağımsız hareket kabiliyetini geri

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a trigger for severe pediatric GBS, particularly the axonal AMAN variant. Prompt diagnosis and early, intensive immunotherapy are crucial for favorable outcomes.

Keywords: Guillain-barré syndrome, AMAN, COVID-19, plasmapheresis, IVIG

kazandı. Bu vaka, SARS-CoV-2'nin özellikle aksonal AMAN varyantı olmak üzere şiddetli pediyatrik GBS için tetikleyici rol oynama potansiyelini vurgulamaktadır. Erken tanı ve yoğun immünoterapi, olumlu sonuçlar için kritik öneme sahiptir.

Anahtar Kelimeler: Guillain-barré sendromu, AMAN, COVID-19, plazmaferez, IVIG

Introduction

Guillain-Barré syndrome (GBS) is an acute, immune-mediated polyradiculoneuropathy, most frequently precipitated by infections, and constitutes the most common cause of acute flaccid paralysis in the pediatric population. Acute motor axonal neuropathy (AMAN) subtype is a less frequent but more severe variant, characterized by axonal degeneration and poor prognosis. Since the emergence of the coronavirus disease-2019 (COVID-19) pandemic, GBS has been increasingly reported in association with severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infection, including in pediatric populations.

Case Report

A three-year-old girl developed AMAN type GBS shortly after a SARS-CoV-2 infection. She initially presented with fever and upper respiratory symptoms. Two days later, she developed progressive limb weakness, ascending flaccid paralysis, and neck muscle involvement. She was unable to sit, walk, or hold up her head. Her parents were second-degree relatives, and she had a history of recurrent bronchiolitis. On examination, deep tendon reflexes were absent, and muscle strength was significantly reduced (1/5 in the upper extremities, flaccid in the lower limbs). SARS-CoV-2 was detected on respiratory polymerase chain reaction (PCR) panel. Cerebrospinal fluid analysis showed normal glucose (54 mg/dL), normal protein (195 mg/L) and no pleocytosis. Cerebrospinal fluid (CSF) culture was negative. Magnetic resonance ımaging revealed enhancement and thickening of the anterior cauda equina fibers (Figure 1). The anti-ganglioside antibody panel was positive for GT1a IgG, supporting an AMAN variant of GBS. Electromyography (EMG) confirmed acute motor axonal polyneuropathy with spontaneous denervation affecting motor fibers.

The patient was initially treated with IVIG (0.4 g/kg/day for 5 days) and underwent five sessions of plasmapheresis. Minimal improvement was observed after the initial course. Due to persistent weakness and absent reflexes, a second course of IVIG was administered, and plasmapheresis was extended to a total of 10 sessions. Following the complete treatment course, the patient gradually regained strength: she was able to swallow, lift her head, and raise her arms. Muscle strength improved to 3/5 bilaterally. At discharge, she required physical therapy. One week later, she could sit unaided and move her arms with ease. By one month, she was walking with support

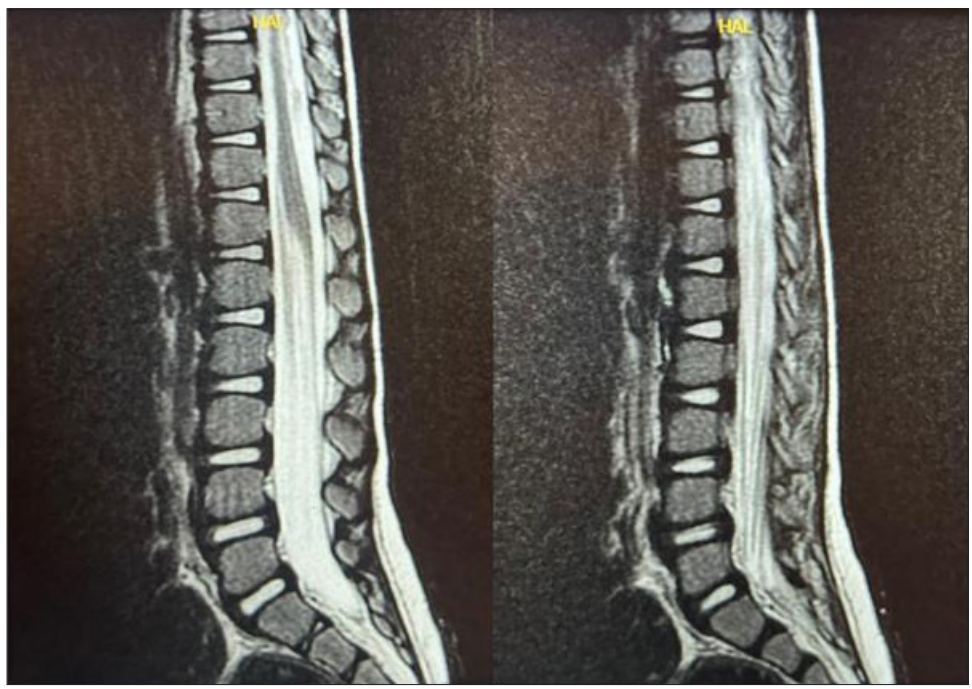

Figure 1. Magnetic resonance imaging demonstrated thickening of the anterior cauda equina nerve roots.

and able to form three-word sentences. At her six-month examination, her gait was normal; however, she was unable to jump. She could climb stairs only with support. Deep tendon reflexes, including the patellar reflex, were normoactive.

Discussion

GBS in children most commonly presents as the demyelinating subtype (AIDP), but during the COVID-19 pandemic, although most reported pediatric GBS cases associated with SARS-CoV-2 infection were demyelinating forms, AMAN-type GBS is rarer and often more severe (1). Children with AMAN seem to have higher short-term morbidity, more severe clinical course, and slower recovery than those with AIDP (2). There are only three reported pediatric cases of COVID-19-associated GBS with the AMAN variant in the literature, and our case represents the fourth and the youngest one. The first pediatric case was a 15-year-old male with SARS-CoV-2 infection and electrophysiological findings consistent with AMAN, highlighting SARS-CoV-2 neurotropism beyond the respiratory tract (3). The second case was an 11-year-old boy presenting with progressive ascending weakness and positive SARS-CoV-2 PCR; electrodiagnostic studies confirmed acute generalized axonal motor neuropathy (4). The third reported case was a six-year-old male, who presented with severe ascending paralysis, respiratory failure requiring ventilation, elevated CSF protein, and AMAN confirmed by nerve conduction studies. This patient received plasma exchange, corticosteroids, and IVIG, showing partial recovery at discharge (5). The presented case is notable for the AMAN subtype confirmed by EMG and anti-GT1a IgG positivity, following a documented SARS-CoV-2 infection. AMAN involves direct axonal damage to motor fibers, leading to a more prolonged and refractory clinical course (1,2). This was evident in this patient, who showed limited response to initial IVIG and plasmapheresis, requiring extended immunomodulatory treatment.

The temporal association with SARS-CoV-2, positive PCR, and absence of alternative etiologies support a COVID-19-associated AMAN diagnosis. While most GBS cases in children are of the demyelinating type, clinicians should be alert to more severe axonal subtypes like AMAN, especially during or after SARS-CoV-2 infection. Anti-ganglioside antibodies, neuroimaging, and EMG are critical for early identification and classification of GBS subtypes.

The presented case highlights the importance of considering SARS-CoV-2 as a potential trigger for GBS, including its axonal forms. Although IVIG and plasmapheresis are established therapies, prolonged or repeated courses may be necessary in severe presentations. Pediatric clinicians should include SARS-CoV-2 testing in the diagnostic workup of children presenting with acute flaccid paralysis or GBS-like symptoms during and after the pandemic period.

In conclusion, this case, the youngest reporting pediatric AMAN-type GBS associated with COVID-19, highlights the association between SARS-CoV-2 and the severe AMAN subtype of GBS in children. Early diagnosis and appropriate treatment, including extended immunomodulatory therapies, are essential for managing these rare, severe cases.

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