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Atypical Guillain–Barré Syndrome with Bulbar Palsy, Autonomic Dysfunction, and Respiratory Failure Requiring Prolonged Mechanical Ventilation in an Adolescent Male: A Case Report

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Abstract

Background

Guillain–Barré syndrome (GBS) is an acute immune-mediated polyradiculoneuropathy characterized by rapidly progressive weakness, areflexia, and variable sensory and autonomic involvement. Atypical presentations with focal neurological deficits, bulbar palsy, and respiratory failure may mimic central nervous system disorders, leading to diagnostic challenges.

Case Presentation

A 13-year-old male presented with worsening headache, dysphagia, and acute-onset left upper limb weakness following a fall. Initial evaluation raised suspicion for meningoencephalitis, tubercular meningitis, or intracranial pathology. The child subsequently developed respiratory failure requiring intubation and mechanical ventilation. MRI brain demonstrated diffuse meningeal enhancement, while cerebrospinal fluid examination was unremarkable. Despite intensive treatment, progressive bulbar weakness and respiratory compromise persisted. Nerve conduction velocity studies eventually revealed findings consistent with atypical Guillain–Barré syndrome. The patient received intravenous immunoglobulin (IVIG) therapy, prolonged ventilatory support, and tracheostomy. During hospitalization, he developed pneumothorax, subcutaneous emphysema, autonomic dysfunction, and ventilator-associated Pseudomonas infection. Gradual neurological recovery was observed following treatment, and the patient was discharged in stable condition with a tracheostomy tube and rehabilitation plan.

Conclusion

This case highlights an unusual presentation of Guillain–Barré syndrome with focal weakness, bulbar involvement, autonomic dysfunction, and respiratory failure. Early recognition and timely

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immunomodulatory therapy are crucial for improving outcomes in atypical paediatric GBS.

Keywords: Guillain–Barré syndrome, atypical GBS, bulbar palsy, respiratory failure, autonomic dysfunction, paediatric neurology, IVIG.

Introduction

Guillain–Barré syndrome (GBS) is the most common cause of acute flaccid paralysis in children. It is an autoimmune disorder affecting peripheral nerves and nerve roots, often triggered by preceding infections. The clinical spectrum ranges from mild weakness to severe paralysis with respiratory failure requiring mechanical ventilation. Atypical presentations may mimic central nervous system disorders, resulting in delayed diagnosis. Bulbar weakness, autonomic instability, and respiratory involvement are associated with severe disease and increased morbidity. We report a challenging case of atypical GBS presenting initially as suspected meningoencephalitis with unilateral weakness and subsequent respiratory failure.

Case Presentation

A 13-year-old male, third-born child of a non-consanguineous marriage, presented with intermittent headache for two years, with worsening intensity over the preceding few days. He developed difficulty swallowing for two days and left upper limb weakness following a fall while playing.

On admission, the child was conscious and oriented with a Glasgow Coma Scale score of 13/15 (E4V3M6). Oxygen saturation was 94% on oxygen delivered through nasal prongs. Shortly after admission, he developed sudden respiratory distress and desaturation, necessitating endotracheal intubation and mechanical ventilation.

Clinical Examination

General examination revealed:

- Heart rate: 144/min
- Blood pressure: 142/106 mmHg
- Respiratory rate: 26/min
- Oxygen saturation: 77% on room air, improving to 94% on oxygen
- Afebrile

Neurological examination showed

- Left upper limb hypotonia
- Muscle power 1/5 in left upper limb

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- Power >3/5 in right upper limb and bilateral lower limbs
- Areflexia
- Bilateral extensor plantar responses
- Bulbar dysfunction with dysphagia
- Unequal pupils (pharmacologically dilated)

No signs of meningeal irritation were present.

Investigations

Initial laboratory investigations demonstrated normocytic normochromic anemia, hypoalbuminemia, and hypocalcemia.

MRI Brain revealed:

- Diffuse meningeal enhancement suggestive of meningitis/meningoencephalitis
- Bilateral mastoid effusion/mastoiditis
- Chronic sinusitis involving maxillary, sphenoid, and ethmoidal sinuses
- No acute intracranial pathology

Cerebrospinal fluid analysis, culture, and CBNAAT were within normal limits.

MRI Whole Spine was normal and showed no evidence of spinal cord pathology.

Nerve Conduction Velocity (NCV) studies demonstrated reduced motor amplitudes in upper limb nerves, consistent with atypical Guillain–Barré syndrome.

Microbiological investigations during hospitalization revealed:

- Tracheal aspirate positive for carbapenemase-producing Pseudomonas aeruginosa
- Central blood culture positive for Candida albicans
- Peripheral blood cultures negative

Differential Diagnosis

The initial differential diagnoses included:

1. Meningoencephalitis
2. Tubercular meningitis
3. Intracranial space-occupying lesion
4. Stroke
5. Autoimmune demyelinating disorders

Following negative CSF studies and characteristic NCV findings, a diagnosis of atypical Guillain–Barré syndrome with bulbar palsy and autonomic dysfunction was established.

Treatment and Hospital Course

Empirical treatment included:

- Intravenous meropenem
- Vancomycin

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- Doxycycline
- Supportive intensive care management

Persistent hypertension was managed with amlodipine.

The patient developed several complications during hospitalization:

Respiratory Complications

- Acute respiratory failure requiring prolonged ventilation
- Subcutaneous emphysema involving neck and abdominal wall
- Right-sided pneumothorax requiring intercostal drainage
- Ventilator-associated Pseudomonas infection

Neurological Complications

- Progressive bulbar weakness
- Autonomic dysfunction
- Persistent respiratory muscle weakness

After confirmation of atypical GBS, intravenous immunoglobulin (IVIG) was administered over five days.

Due to prolonged ventilatory dependence, tracheostomy was performed on day 13 of hospitalization.

Subsequently, gradual neurological improvement was observed with recovery of limb strength, truncal muscle power, swallowing function,and respiratory status. Ventilator support was gradually weaned to CPAP and later to tracheostomy-based spontaneous breathing trials.

At discharge, the child was afebrile, hemodynamically stable, maintaining oxygen saturation on room air via tracheostomy, and tolerating oral feeds.

Discussion

Guillain–Barré syndrome typically presents with ascending symmetrical weakness and areflexia. However, atypical variants can manifest with asymmetrical weakness, cranial nerve involvement, and respiratory failure, creating significant diagnostic uncertainty.

In this patient, unilateral upper limb weakness and MRI findings suggestive of meningeal inflammation initially favoured a central nervous system etiology. Normal cerebrospinal fluid findings and progressive peripheral neurological deficits prompted further evaluation. Nerve conduction studies proved crucial in establishing the diagnosis.

Bulbar palsy and autonomic dysfunction are recognized markers of severe GBS and are associated with prolonged hospitalization and mechanical ventilation. The occurrence of pneumothorax, ventilator-associated pneumonia, candidemia, and prolonged tracheostomy further complicated management.

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Prompt initiation of IVIG resulted in significant neurological recovery. This case underscores the importance of considering GBS in children presenting with unexplained respiratory failure and progressive neurological deficits, even when initial investigations suggest alternative diagnoses.

Conclusion

Atypical Guillain–Barré syndrome may present with focal weakness, bulbar dysfunction, autonomic instability, and respiratory failure, closely mimicking central nervous system disorders. Early electrophysiological evaluation and timely immunotherapy are essential for accurate diagnosis and favorable outcomes. Multidisciplinary intensive care management plays a pivotal role in the recovery of severe pediatric cases.

Learning Points

1. Atypical GBS may present with unilateral weakness and mimic meningoencephalitis or stroke.
2. Normal cerebrospinal fluid findings do not exclude Guillain–Barré syndrome.
3. Nerve conduction studies are critical for diagnosis in atypical presentations.
4. Bulbar palsy and autonomic dysfunction indicate severe disease requiring intensive monitoring.
5. Early IVIG administration can significantly improve neurological recovery.

Patient Consent

Written informed consent for publication obtained from the patient's parent or legal guardian. Personal identifiers have been omitted to ensure confidentiality.

Data Availability Statement

All data generated or analysed during this study are included within this published article. Additional patient-level data are not publicly available to protect patient privacy and confidentiality but may be made available by the corresponding author upon reasonable request and with appropriate institutional approval.

Conflict of Interest

The authors declare no conflict of interest.

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