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ACUTE DISSEMINATED ENCEPHALOMYELITIS WITH OPTIC NEURITIS AND LONGITUDINALLY EXTENSIVE MYELITIS IN A TODDLER: A CASE SUGGESTIVE OF MOG ANTIBODY-ASSOCIATED DISEASE

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Abstract:
Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is an inflammatory demyelinating disorder of the central nervous system increasingly recognized in the pediatric population. We report a case of a 2-year-old male child who presented with acute febrile illness, seizures, and altered sensorium. Neurological examination revealed focal deficits, and neuroimaging demonstrated multifocal involvement of the brain, optic nerves, and spinal cord. Cerebrospinal fluid analysis showed lymphocytic pleocytosis with elevated protein levels. The clinical and radiological findings were suggestive of MOG antibody-associated disease. The patient was treated with high-dose intravenous corticosteroids and plasma exchange, resulting in partial clinical improvement. This case highlights the importance of early diagnosis and prompt immunotherapy in pediatric demyelinating disorders.
Keywords: MOGAD, ADEM, optic neuritis, transverse myelitis, pediatric demyelination

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INTRODUCTION:

Myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD) is an immune-mediated inflammatory disorder affecting the central nervous system. It is commonly observed in children and often presents as acute disseminated encephalomyelitis (ADEM), optic neuritis, or transverse myelitis. The disease is characterized by antibodies directed against myelin oligodendrocyte glycoprotein, leading to demyelination. Early recognition is essential as the condition is highly responsive to immunotherapy but may have a relapsing course.

MATERIALS AND METHODS:

This is a descriptive case report of a pediatric patient admitted with acute neurological symptoms. Clinical data, laboratory findings, cerebrospinal fluid analysis, and radiological investigations were collected and analyzed. Ethical standards were maintained, and patient identity was kept confidential.

RESULTS AND DISCUSSION:

Case Presentation

A 2-year-old male child was admitted with complaints of fever for four days, vomiting, decreased activity, and a history of generalized seizure. On examination, the child was febrile and drowsy but arousable. Neurological examination revealed left lateral gaze restriction and decreased movements in the right upper limb. Meningeal signs were present.

Investigations

Magnetic resonance imaging of the brain revealed bilateral symmetric hyperintense lesions involving the thalami, basal ganglia, midbrain, pons, and medulla with associated edema. Bilateral optic nerve enhancement was observed, suggestive of optic neuritis. Spinal imaging demonstrated long-segment involvement of the spinal cord consistent with longitudinally extensive transverse myelitis.

Cerebrospinal fluid analysis showed elevated protein levels, lymphocytic pleocytosis, and normal glucose. Microbiological studies were negative. Electroencephalography showed diffuse slowing without epileptiform discharges.

Management

The patient was initially treated with intravenous fluids, empirical antibiotics, and antiepileptic drugs. Based on the suspicion of an inflammatory demyelinating disorder, high-dose intravenous methylprednisolone was administered. Due to incomplete response, plasma exchange therapy was initiated. Supportive care including ventilatory support was provided during the hospital stay.

DISCUSSION:

MOGAD is increasingly recognized as a distinct entity among demyelinating disorders. In children, it frequently presents with ADEM-like features accompanied by encephalopathy and multifocal neurological deficits. The presence of bilateral optic neuritis and longitudinally extensive transverse myelitis strongly supports this diagnosis. MRI findings typically include bilateral, poorly demarcated lesions involving both white and deep gray matter structures. CSF findings usually demonstrate lymphocytic predominance and elevated protein levels. Early treatment with corticosteroids is associated with favorable outcomes, although some patients may require escalation therapy such as plasma exchange or intravenous immunoglobulin.

CONCLUSION:

MOG antibody-associated disease should be considered in pediatric patients presenting with acute encephalopathy and multifocal central nervous system involvement. Early diagnosis through neuroimaging and serological testing is essential for prompt initiation of immunotherapy, which can significantly improve clinical outcomes.

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