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Original Research Article

Complete Oculomotor Nerve Palsy: A Rare Presentation of Monofocal Clinically Isolated Syndrome in a Child

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Abstract

Objective: Assess the rare presentation of a clinically isolated syndrome of cranial nerve III (CN III) palsy in a pediatric patient.

Methods: We present the case of a12-year-old boy with acute onset double vision with left eye pain, left-sided ptosis, and left ophthalmoplegia. The patient had no other systemic or neurological symptoms.

Results: An extensive work up including serum and CSF studies for autoimmune, infective, or malignant etiologies was performed. MRI Brain showed mild enlargement and enhancement of the cisternal segment of the left CN III. The remainder of the work up was negative for any secondary etiologies. The patient was treated with high dose methyl prednisone 250 mg IV every 6 hours for 5 days followed by 2 weeks of oral prednisone. There was complete resolution of symptoms at the 3-week follow-up clinical visit.

Conclusion: In rare instances, clinically isolated syndrome can present as cranial nerve III palsy in a pediatric patient.

Keywords: Oculomotor Nerve Palsy, Pediatrics.

Introduction

Isolated cases of cranial nerve palsy III (CN III) in children are rare with an estimated incidence of 7.6 per 100,000 children.[1] Etiologies of CN III Palsy in children include congenital, trauma, tumor, vascular, meningitis, idiopathic causes, with the most common being congenital. Congenital cases can present with a history of birth trauma or forceps delivery. Trauma cases may involve blunt trauma to the skull with subsequent orbital fracture.[2] Isolated CNIII palsy has been reported in children with COVID infection recently. It has also been reported as a manifestation of anti-GQ1b antibody, pituitary apoplexy and varicella zoster encephalitis.[3-4] However, CN III palsy as an isolated finding presenting as a clinically isolated syndrome has not been reported previously

Case Presentation: A 12-year-old male with no significant past medical history presents with a chief complaint of double vision with left eye pain, left-sided ptosis, and left ophthalmoplegia. The symptoms had been present for approximately five days without relief. He has no previous history of vision problems, neurological symptoms or family history of neurological disorders. Physical exam revealed a well-developed 12-year-old male in no acute distress. The cardiopulmonary exam was normal. Cranial nerve exam demonstrated anisocoria with approximately a 2-millimeter difference and nonreactive appearing pupil on initial assessments but improving reactivity of his left pupil was noted on subsequent evaluations. There was persistent ptosis and impaired ocular movements of the left eye suspicious for CN III/IV involvement [Fig.1B 1-2 and Fig.1C 1-6]. No other abnormal cranial nerve involvement was noted, and the remainder of his neurological exam was normal.

Contrasted CT imaging of the head and orbits were normal with no evidence of any intracranial pathology. MRI of the brain and orbits with and without contrast initially demonstrated subtle increased enhancement within the left extraocular muscles without parenchymal involvement.

Upon further review between neurology and radiology, there was mild enlargement noted and enhancement of the cisternal segment of the left CN III [Fig.A]. Lumbar Puncture was also performed which did not indicate an infectious etiology with non-elevated protein and a normal white count. Additional labs found negative CSF oligoclonal bands, normal IgG concentrations, and normal IgG index. Myelin basic protein was also within normal ranges. Acetyl choline receptor antibodies (AChR) and MUSK antibodies for Myasthenia Gravis and Aquaporin-4 (AQP4) antibodies were negative.

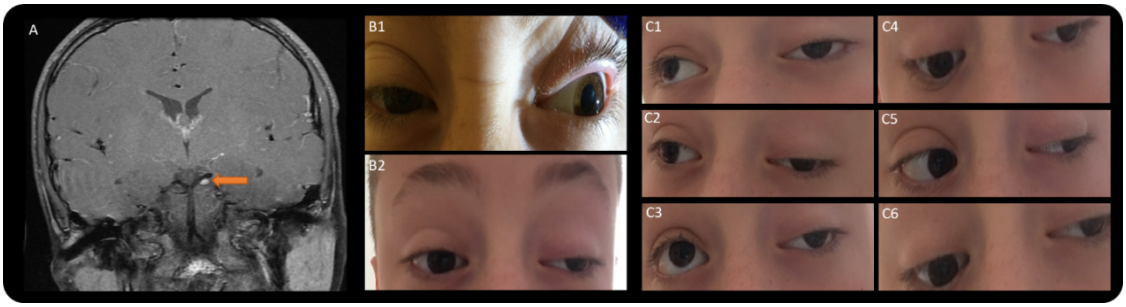

Figure 1:

Figure Legend A: Coronal post contrast T1 weighted image demonstrates marked enlargement and enhancement of the cisternal segment of left oculomotor nerve as it passes between the left posterior cerebral artery and left superior cerebellar artery.

B1 & B2: Left eye anisocoria with complete ptosis.

C1 to C6: Left eye adduction, elevation, and depression deficits

Discussion

We present an interesting and rare case of CNIII palsy appearing as a clinically isolated syndrome. Our patient demonstrated pupil sparing exam with primarily motor impairment with "down and out" pupil as well as ptosis. Differentiating pupil involving versus pupil sparing lead to identifying etiology broadly as compressive versus micro ischemic (affecting superficial vs deep structures of nerve respectively) and is an important first step. The more superficial autonomic pupillary motor fibers traveling along the nerve are affected in compressive lesions resulting in nonreactive pupils. Micro ischemic changes will affect vasa vasorum and subsequently deeper structures in CN III resulting in impaired somatic function leading to oculomotor palsy with retained superficial autonomic fibers and therefore intact pupillary reactivity.[5] Etiology to an acquired CN III palsy would include space-occupying lesion along the course of CN III, local inflammation, trauma, aneurysm, ophthalmoplegic migraine, or atypical ocular myasthenia. Certain syndromes such as Benedikt, Weber, Nothnagel and Claude can present with CN III palsy but are all associated with further neurological changes including tremor, hemiplegia, cerebellar ataxia which were not present in this case.[6] As an isolated symptom of MS or even as an initial event, CN III palsy is quite rare; however,it has been reported with a somewhat similar presentation of eye pain and CN III palsy.[7] In a retrospective study of multiple sclerosis patients, 1.6% exhibited an isolated cranial nerve palsy. Those with CN III involvement at initial diagnosis totaled only 0.4%. [8] The rare concurrency of CN III palsy with multiple sclerosis makes this an unlikely cause for the patient. With the relative sparing of his left pupil and evidence on MRI of enhancement in the very proximal aspect of CN III, there is higher suspicion for a clinically isolated syndrome in the setting of CN III inflammation.

The most common infectious and inflammatory conditions affecting the cranial nerves are viral neuronitis. Infection and inflammation often occur within the cavernous sinus. Inflammatory material can be deposited within the cerebellopontine angle (CPA), perimesencephalic and basilar cisterns encasing the cisternal segments of cranial nerve III, leading to neural ischemia.[9]

Conclusion

In rare instances, clinically isolated syndrome can present as cranial nerve III palsy in a pediatric patient. Early diagnosis and treatment can lead to complete resolution of the symptoms.

Conflict of Interest Disclosures: The authors declare no competing interests.

Ethics Approval: Our institution does not require ethical approval for reporting individual cases or case series.

Informed Consent: Written informed consent was obtained from the patient and legal guardian for the anonymized information to be published in this article.

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