

Case report

Acute mydriasis and ophthalmoplegia in a 10-year-old girl

Comment [WU 1]: Please kindly provide purposeful title

UNDER PEER REVIEW

Abstract:

This case reports a 10-year-old girl who presented with sudden-onset diplopia and bilateral mydriasis following a recent upper respiratory infection. Ocular examination showed poor light response and mild abduction deficits. Initial low-resolution MRI was normal, but a high-resolution MRI revealed bilateral enhancement of the abducens and oculomotor nerves. Viral-induced cranial nerve palsies were suspected. The use of systemic steroids was considered but concerns about side effects in children led to a conservative approach. Patient achieved full recovery four months later. This case highlights the importance of high-resolution imaging in diagnosing cranial nerve palsies in children and raises questions about the role of corticosteroids in managing viral-induced neuropathies. Further research is needed to determine optimal treatment strategies for such cases.

Keywords: Paediatrics, Paediatric ophthalmology, Neuro-ophthalmology

Comment [WU2]: Please add relevant keywords. These entites must focus or emphasise on topic of discussion of this report.

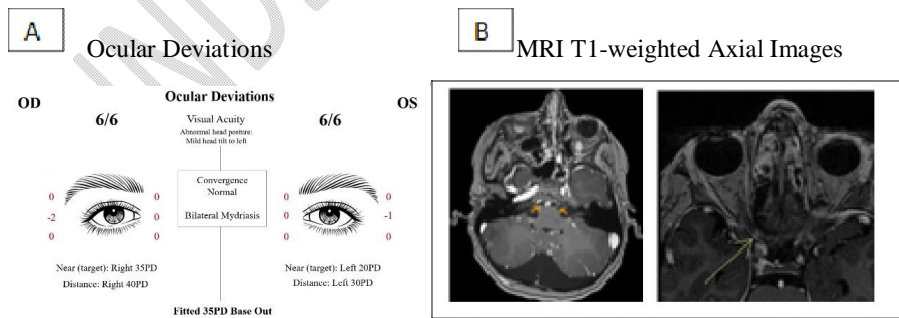
Introduction

Mydriasis is due to stimulation of the iris dilator muscle or compromise of the parasympathetic tone of the iris sphincter muscle, or both(Rathnasiri et al., 2024).Bilateral mydriasis typically arises when sympathetic innervation exceeds parasympathetic innervation(Kang et al., 2024).This is frequently associated with systemic disorders such as Fisher syndrome or botulism, specific pharmaceutical agents, midbrain lesions (specifically in the pretectal area and third nerve nuclei), bilateral tonic pupils, iris ischaemia due to vasculitis or atherosclerosis, or a condition known as autoimmune autonomic ganglionopathy(Xu et al., 2021).Ophthalmoplegia refers to the condition of the paralysis of eye muscles, which can be caused by various factors such as nerve damage, systemic diseases, or genetic mutations.Ophthalmoplegia is most commonly caused by damage to the ocular motor nerves (Dietz, 2018; Das, 2020). This study is a case of Acute mydriasis and ophthalmoplegia in a girl.

Comment [WU3]: Please kindly elaborate in two or three sentences. Textbook content are highly appreciated.

Comment [WU4]: What kind of ophthalmoplegia,

Case Presentation:



Comment [WU5]: Enhancement suggestive of viral neuritis

Figure A. The patient presented with bilateral esotropia and bilateral mydriasis. Patient fitted with Fresnel prisms. Figure B, MRI T1 weighted axial images demonstrating bilaterally enhancing abducens nerves (left) and right oculomotor enhancing nerve (right).

A 10-year-old Caucasian girl presented to an eye center with sudden onset diplopia and bilateral mydriasis. One week earlier, while on holiday, she became unwell with lethargy, headaches, fever and progressive cough. She was diagnosed with upper respiratory tract infection and was managed conservatively with gradual resolution of her systemic symptoms. She had no previous ocular history and denied any trauma.

On examination, best-corrected Snellen visual acuity was 6/6 either eye. There was bilateral symmetrical mydriasis with poor response to light and accommodation (Figure A). There was no ptosis. On extra-ocular muscle testing the child exhibited -2 abduction deficit in the right and -1 abduction deficit in the left. Anterior segments appeared healthy and dilated funduscopy was unremarkable. There were no other neurological findings on examination.

Comment [WU6]: Please comment on Diplopia. Provide Hess Chart of the patient

Blood investigations including infectious screen, autoimmune screen, aquaporin-4 and myelin oligodendrocyte glycoprotein and anti-gangliosides antibodies were all negative. Low-resolution magnetic resonance imaging (MRI) of the brain and orbits with contrast enhancement did not reveal any abnormality. Lumbar puncture demonstrated normal cerebrospinal fluid (CSF) studies, including infections screen and oligoclonal bands, and the opening pressure was 21cmH₂O.

Diagnosis:

Suspected Viral Induced Cranial Nerve Palsies

Next Step:

Repeat high-resolution MRI of brain with contrast

Discussion:

Acquired third, fourth, and sixth cranial nerve palsies are rare in children compared with those in adults and often signal serious pathologies. Neoplasia and trauma are the leading causes in this age group for all three palsies. Prompt diagnostic work-up is crucial due to the potential for long-term loss of function and life-threatening conditions.

Comment [WU7]: Provide reference

The initial diagnostic work-up of the patient did not confirm a diagnosis. Even though an infectious aetiology was suspected due to the temporal association with the preceding upper respiratory tract infection, it was necessary to exclude the possibility of neoplasia. The primary low-resolution MRI was reported as normal with no space occupying lesion or raised intracranial pressure but was considered inadequate in visualising cranial nerve pathways. Therefore, a multiplanar high resolution 3T MRI with contrast enhancement was requested which demonstrated bilateral abducens and oculomotor nerve enhancement (Figure B).

Palsies of abducens, oculomotor or trochlear nerve may arise from disease processes which affect them at any point along their anatomical pathways. Each nerve travels along a highly stereotypical path beginning at the individual cranial nerve nuclei in the brain and terminating at the neuromuscular junction, where they innervate their respective extraocular

muscle(s). Three-dimensional MRI steady-state free precession sequences and modified fully refocused steady-state sequences such as constructive interference in steady state (CISS) should be requested to allow detailed visualization of the cranial nerve pathways in patients with unexplained cranial nerve palsies.

Comment [WU8]: Add reference please.

A lumbar puncture was performed to rule out intracranial hypertension, which is rare in paediatric patients, as well as to assess for inflammatory, infectious, or infiltrative processes through CSF studies. Viral serology for common viruses was also undertaken and was negative. Due to the COVID-19 pandemic and recent travel history, COVID infection was suspected. The child had a negative rapid test on holiday but could not undergo a polymerase chain reaction (PCR). A PCR was performed 12 days after the onset of her systemic symptoms and was also negative. She did have though high levels of COVID antibodies with no previous history of vaccination and last confirmed COVID infection almost a year ago.

Comment [WU9]: Please mention

The treatment for viral-related oculomotor and abducens nerve palsy remains unclear. The palsy is often self-limiting, usually within months, and therefore a more conservative approach may be warranted¹. For symptomatic relief Fresnel prisms were fitted and the patient was asymptomatic in primary position.

There was a debate among the patient's physicians whether systemic steroids should be prescribed. The role of steroids in children with viral-induced cranial nerve palsy is not clear. Steroids have been used successfully in pediatric populations to treat oculomotor and abducens nerve palsies². Steroids have been shown to hasten recovery in cranial nerve neuropathies caused specifically by COVID-19^{3,4}. However, children are more susceptible to steroid side effects than adults⁵. Given the self-limiting nature of the palsies, the risk of significant adverse effects may not outweigh the benefits of corticosteroid administration. Nevertheless, more research is required to determine suitability of corticosteroids as a treatment.

Patient Outcome:

The repeat high-resolution MRI imaging helped localize the inflammation along the pathways of the oculomotor and abducens cranial nerves. The most likely diagnosis was viral related cranial nerve palsy, as most other aetiologies were excluded and there was a close temporal association with the preceding upper respiratory tract infection. COVID-19 was suspected but could not be proven. The patient attained full recovery four months from the onset of symptoms and remained symptom-free two years later.

References:

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