

Vision loss in an immunocompetent child post varicella infection: A case report

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Abstract

Chickenpox may lead to several neurological complications. Optic neuritis is one of the complications which has rarely been described, especially in immunocompetent individuals. We report a case of an 11-year-old immunocompetent girl who presented with sudden onset bilateral vision loss three weeks after varicella eruption. Ophthalmic examination revealed bilateral optic disc edema. Diagnosis of bilateral optic neuritis secondary to varicella was established based upon the preceding medical history, supported with clinical and radiological findings.

Introduction

Optic neuritis is a condition characterized by inflammation of the optic nerve, leading to acute visual loss. It commonly occurs bilaterally in the pediatric population and can be attributed to viral infection or demyelinating disease. Optic neuritis is a rare consequence of varicella infection.¹ This case report is to highlight that optic neuritis can occur as one of the complications after chickenpox infection in an immunocompetent child.

Case Report

An 11-year-old girl presented with sudden onset bilateral reduced vision associated with painful eye movements for a week with rapid progression. No prior treatment was

sought for her ocular symptoms. She had varicella eruption three weeks prior and was treated in a primary healthcare center without acyclovir. She was pre-morbidly healthy and had completed immunization up to her age. There was no redness, eye discharge, floaters, or flashes of lights. Otherwise, she gave no other significant ocular history or neurological symptoms.

Her bilateral visual acuity was counting finger. Relative afferent pupillary defect was positive on right eye. The optic nerve function tests revealed failed Ishihara color vision. Anterior segments were normal. Bilateral optic discs were diffusely swollen and hyperemic (**Figure 1A & 1B**). Systemic and neurological examination was normal.

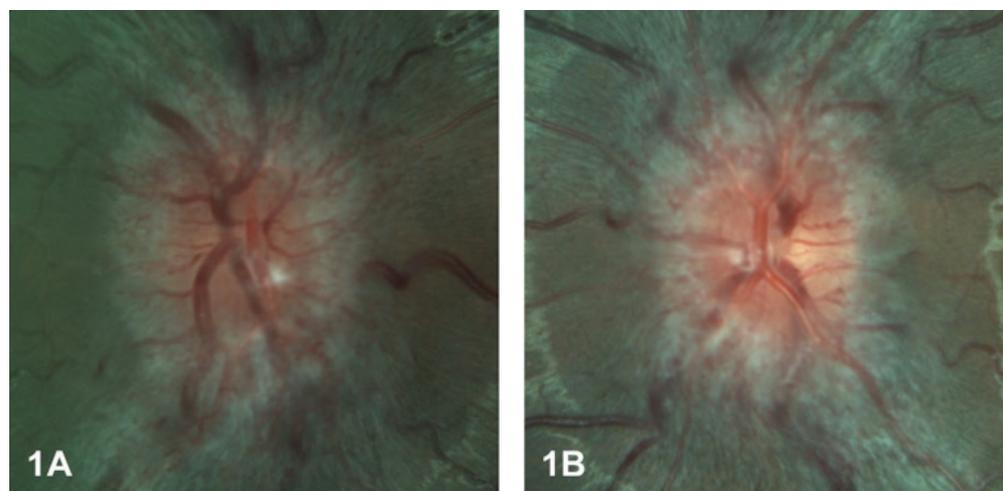


Figure 1A & 1B: Right eye (1A) and left eye (1B) showed hyperemic and diffusely swollen optic discs.

Magnetic resonance imaging (MRI) of orbits showed enhancement of both optic nerves (**Figure 2**). There was no evidence of demyelination or transverse myelitis on brain and spine MRI. Lumbar puncture revealed normal opening pressure and cerebrospinal fluid findings. Infective screens (Venereal Disease Research Laboratory test, toxoplasmosis, rubella, cytomegalovirus, herpes simplex virus, human immunodeficiency virus), and screens for antinuclear antibodies and anti-aquaporin-4 antibodies were negative. Diagnosis of bilateral optic neuritis secondary to varicella infection was made based on history of preceding chickenpox, clinical, and radiological findings.



Figure 2: MRI of orbits showed contrast enhancement of bilateral optic nerve, consistent with optic neuritis.

She was treated with intravenous methylprednisolone (30 mg/kg per day with maximum 1 g daily) for five days, followed by nine days of oral prednisolone (2 mg/kg daily). Her bilateral visual acuity had markedly improved to 6/6 with normal color vision after two weeks of treatment. Subsequent review showed resolution of optic discs swelling (**Figure 3A & 3B**). One month after initial presentation, her bilateral visual acuity was 6/6 with normal optic nerve function tests. Patient had defaulted subsequent follow-up after the last visit.

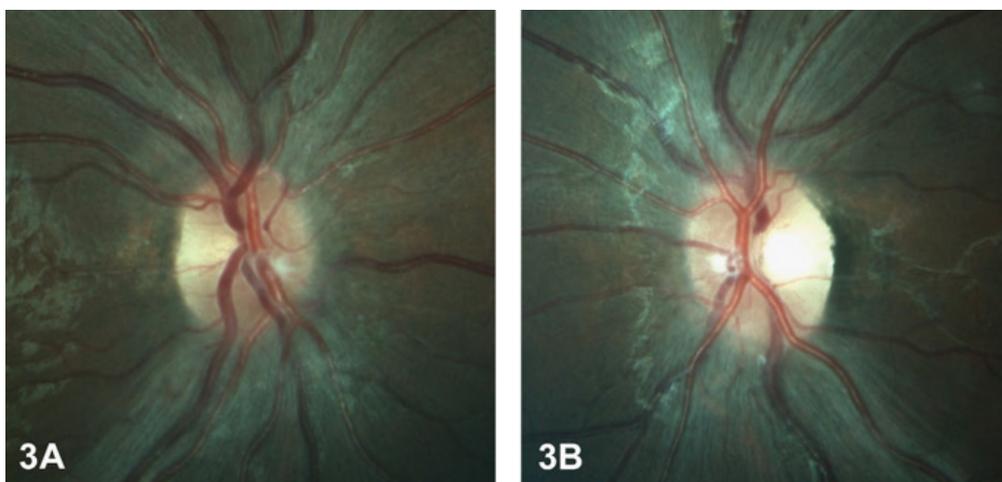


Figure 3A & 3B: Resolution of right eye (3A) and left eye (3B) optic disc swelling after treatment.

Discussion

Optic neuritis in the pediatric population is uncommon.² The presentation is usually bilateral and more severe compared to that in adults. However, the visual prognosis is better in children.^{1,3,4}

Optic neuritis usually presents post-infection or post-immunization in pediatrics. However, if it is associated with other neurologic features, childhood multiple sclerosis and acute disseminated encephalomyelitis should be considered in the differential diagnosis.^{1,5}

Varicella is a well-known contagious disease caused by the varicella-zoster virus. It may lead to neurological complications, including encephalitis, transverse myelitis, Guillain-Barré syndrome, facial nerve palsy, and cerebral ataxia, but optic neuritis has rarely been described. Ocular manifestations may occur at any time during the course of the disease or post-varicella infection.^{1,5} The course of post-varicella optic neuritis is reported to be significantly more severe and often includes acute retinal necrosis in immunocompromised individuals.²

The pathogenesis of varicella infection leading to optic nerve involvement is not well understood. It has been postulated to occur via direct nerve invasion by the virus or an autoimmune-mediated disease process. In our case, there was a three-week interval from the onset of varicella eruption to the development of symptoms. The delayed onset may suggest a secondary autoimmune process. The postulated mechanisms for this process include molecular mimicry between viral and neural antigens and incorporation of virally coded antigens into neural tissue.^{2,3,5}

Acute demyelinating optic neuritis and transverse myelitis were safely excluded in this case by MRI study of the central nervous system. Lumbar puncture is not routinely performed in all cases of optic

neuritis. It should be considered only in atypical cases with bilateral presentation and both neurological and infective symptoms.⁶ Confirmatory serologic testing for varicella is indicated only when there is an unclear medical history.⁴

Studies have shown evidence of irreversible injury and functional decline after resolution of optic neuritis in children. These include reduced color vision, visual field defects, thinner optic nerve fiber layers, and optic atrophy.^{7,8}

The use of corticosteroids in treating optic neuritis in the pediatric population is controversial. Based on the Optic Neuritis Treatment Trial (ONTT), the visual benefit from treating acute optic neuritis in adults with intravenous corticosteroid is only limited to an accelerated rate of recovery with no change of final visual outcome. It also reduces the rate of recurrences and risk of development of a second demyelinating event.^{7,9} However, there is still no standardized guideline for intravenous corticosteroid treatment among pediatric patients. In our case, we opted for a short course of systemic corticosteroid in view of the disease severity (bilateral involvement and poor initial visual acuity), with the aim to halt the progression of optic neuritis.

Conclusion

Pediatric patients who experience sudden vision impairment with recent preceding varicella infection should be urgently evaluated to rule out optic neuritis. Timely treatment is of utmost importance for halting the progression of optic neuritis and preventing detrimental sequelae.

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How does this paper make a difference to general practice?

1. Varicella-zoster infection is a common contagious disease in our population.
2. General practitioners should aware that optic neuritis, although rare, can be a complication of chickenpox infection in an immunocompetent patient.
3. Early diagnosis of optic neuritis and intervention are important for halting its progression and preventing sequelae.

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