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SHORT COMMUNICATION

Presumed infective meningoencephalitis complicated by bilateral optic neuritis

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PURPOSE. *To report a case of presumed infective meningoencephalitis complicated by bilateral optic neuritis.*
METHODS. *Interventional case report.*
RESULTS. *A 7-year-old Pakistani girl presented with fever and multiple right-sided focal seizures. Despite empirical treatment with antibiotic, anti-viral and anti-tuberculous therapy for presumed infective meningoencephalitis, she further deteriorated, developing altered consciousness, hemiplegia and severe, bilateral optic neuritis. No infectious agent could be identified. Following the addition of high dose systemic corticosteroid therapy, her optic nerve function in both eyes began to recover. At 3-month follow-up, the hemiplegia had completely resolved, and visual acuity was 6/6 bilaterally with normal pupillary responses.*
CONCLUSIONS. *This case demonstrates that meningoencephalitis of presumed infective origin may be complicated by acute, severe, bilateral optic neuritis, the latter often mediating profound visual loss. In the setting of rapidly deteriorating vision, aggressive corticosteroid treatment, with antimicrobial cover, albeit unproven, remains the mainstay of treatment. (Eur J Ophthalmol 2007; 17: 864-6)*

KEY WORDS. *Encephalitis, Meningitis, Optic neuritis*

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INTRODUCTION

Optic neuritis in children is often memorable because of its dramatic presentation. Compared with adults, it is more often bilateral (60–70%), although frequently asymmetric, with gross optic disc swelling, often resulting in profound visual loss (6/60 or worse in 84% in one series) (1, 2). At presentation, additional signs of optic neuropathy are typically found, including a relative afferent pupillary defect, loss of color vision and a large central scotoma. Although optic disc swelling occurs in the majority of cases (64–87%), previous case series have highlighted that retrobulbar neuritis predominates in adolescents (1).
Correlating well with the established seasonal predominance from spring to autumn, pediatric optic neuritis is often associated with a preceding febrile illness, reported in 66% of patients by Morales et al within 2 weeks of visual symptoms (1). Viruses, including adenovirus, varicella, measles and mumps, as well as post-vaccination (specifically measles, mumps, rubella, and tetanus), are thought to trigger an autoimmune-mediated demyelinating process (3).
The main differential diagnoses, including orbital cellulitis can usually be excluded on history and examination. Neuroimaging is required to exclude other causes of optic nerve dysfunction, such as optic nerve and optic nerve sheath tumors; it is indicated also in children with associated neurologic impairment, such as seizure activity, whether isolated or as part of acute disseminated encephalomyelitis (3, 4).

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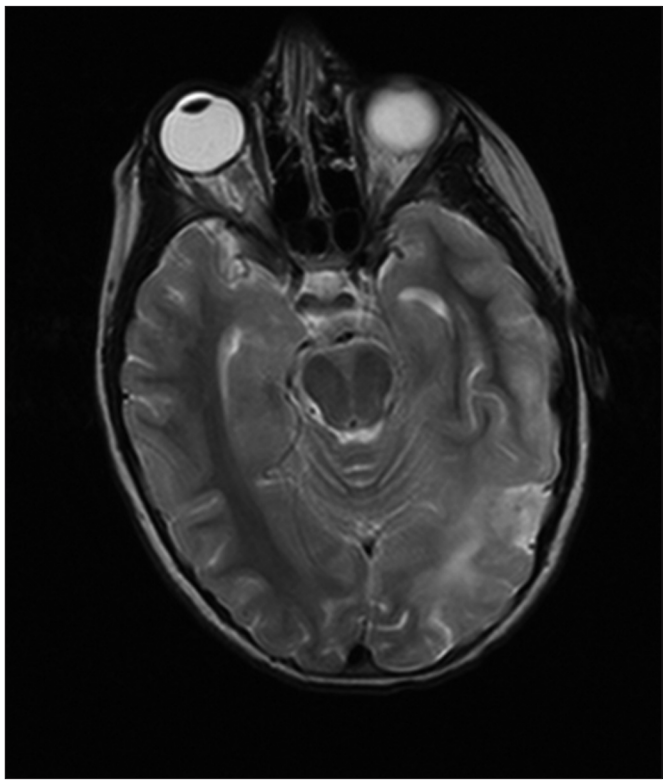

Fig. 1 - *Axial T2-weighted, nonenhanced magnetic resonance image showing left cortical lesion with white matter involvement of the parietal and occipital lobes.*

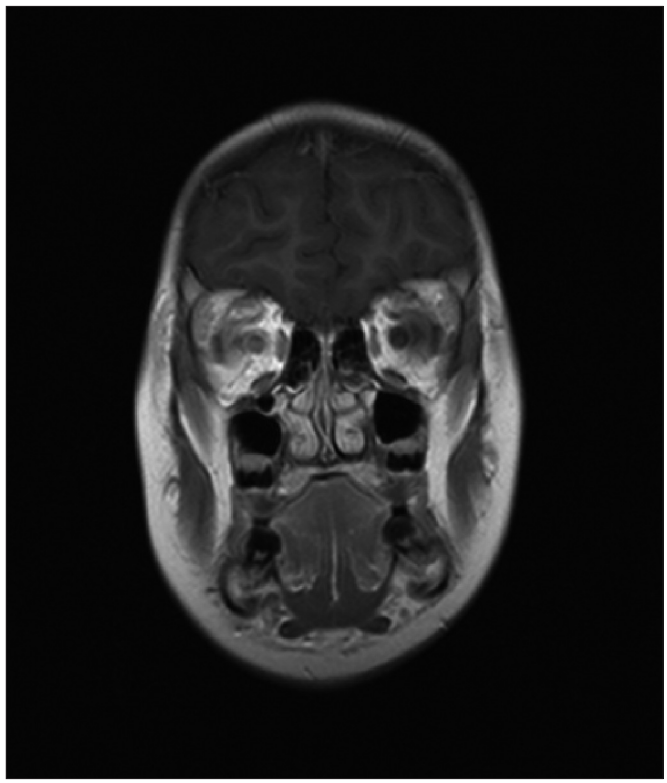

Fig. 2 - *Coronal T1-weighted, gadolinium-enhanced magnetic resonance image demonstrating swelling and enhanced signal in both optic nerves, worse on the right.*

Early, rapid recovery of visual function in pediatric optic neuritis is typically seen, especially in the very young, possibly reflecting their greater remyelinating capacity. Overall, 77% of patients, either treated or untreated, recover 20/20 vision or better (5). Children aged 6 years and under appeared in one cohort to have an improved visual outcome (92–100% >20/40 vs 50% aged >6), though this was not statistically significant (2). In the same series, the difference in visual outcome between bilateral and unilateral disease (78% and 72% respectively) was not significant; however, Morales et al demonstrated in a 15-patient series that unilateral presentation was associated with recovery >20/40 in 100% of patients, compared with 50% of those affected bilaterally (1).

Treatment with corticosteroids remains a contentious issue given the generally good spontaneous visual recovery. Two cohorts found no correlation between final visual outcome and the use of corticosteroids (1, 2). However, intravenous methylprednisolone is often used to speed up recovery, especially with severe neurological involvement or profound bilateral visual loss.

Case report

A 7-year-old girl, resident in Pakistan for the preceding 4 months, presented with a 6-week history of fever and multiple right-sided focal seizures. Despite treatment for presumed infective meningoencephalitis, including antibiotic, anti-viral and anti-tuberculous therapy, she further deteriorated, developing altered consciousness and a right hemiplegia. Bar positive IgM serology for typhoid (IgG negative), no infectious agent was identifiable on blood and cerebrospinal fluid (CSF) culture or CSF polymerase chain reaction. Initial magnetic resonance imaging (MRI) confirmed the left cortical lesion with white matter involvement of the parietal, temporal, and occipital lobes (Fig. 1). However, subsequent ophthalmologic review revealed no perception of light, a dense relative afferent pupillary defect, and gross papillitis in the right eye. Initial left visual acuity was 6/6 with full color vision, though early optic disc swelling was apparent. In light of these findings, the original MRI scans were reviewed which revealed swelling and enhanced signal in both intraorbital

optic nerves, worse on the right (Fig. 2). Within 24 hours, vision in the left eye deteriorated to 3/60, with no color perception and worsening papillitis. Under the coordinated care of the neurology, infectious disease, and ophthalmology teams, her intravenous hydrocortisone therapy was changed to pulsed methylprednisolone, followed by maintenance oral prednisolone. Less than 24 hours on this more aggressive corticosteroid regimen, her left optic nerve function stabilized and the vision in both eyes began to recover.

DISCUSSION

In the case presented, meningoencephalitis was accompanied by bilateral optic neuritis. Although IgM serology for typhoid was detected it was considered highly unlikely to be causative of the presenting disorder. The devastating sequential loss of vision in both eyes prompted aggressive corticosteroid therapy. At 3 months' follow-up, her vision had recovered to 6/6 in each eye with normal pupillary responses, despite subtle bilateral optic disc pallor and marked atrophy of the left occipital lobe. In addition, her hemiplegia had completely resolved.

Competing and financial interests: None.

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