

CASE REPORT

OPTIC NEURITIS CAUSED BY INTRAOCULAR ANGIOSTRONGYLIASIS

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Abstract. A 21-year-old Thai man first presented with eosinophilic meningitis. One week later he developed visual impairment of the left eye with a visual acuity of 20/600. He had a Marcus Gunn pupil, constricted visual field, prolongation of visual evoked potential and the presence of inflammatory cells in the vitreous cavity of the affected eye. On funduscopic examination there was disc swelling with hyperemia of the left eye. These ocular findings are compatible with optic neuritis. The causative agent, *Angiostrongylus cantonensis*, was identified in the vitreous cavity. To our knowledge this is the first case of optic neuritis caused by intraocular angiostrongyliasis. Ten days after administration of corticosteroid his severe headache was resolved, and by 4 weeks the disk swelling of the left eye subsided. Eight months after treatment the visual acuity of the left eye had not improved due to permanent damage to the retinal pigment epithelium caused by the intraocular parasite.

INTRODUCTION

Eosinophilic meningitis is commonly found in Thailand. *Angiostrongylus cantonensis* is the most common causative agent. Most patients present with acute to subacute severe headache without neurological signs. Some patients present with cranial nerve abnormalities such as Bell's palsy along with eosinophilic meningitis (Chotmongkol *et al*, 2000). Here, we report additional ocular findings in a case previously included in our case series (Sawanyawisuth *et al*, 2007). Optic neuritis related to eosinophilic meningitis caused by *A. cantonensis* was previously reported in 2006 (Wang *et al*, 2006). In that case, the optic neuritis was related to meningitis with no intraocular parasite identification. This is the first case of optic neuritis

that we are aware of that was directly related to intraocular angiostrongyliasis.

CASE REPORT

A 21-year-old man had been suffering from progressive headache for two weeks. He was afebrile but had neck stiffness, left facial palsy, lower motor neuron type, and left lateral rectus palsy. He had eaten raw snails two months previously, then developed a headache. A complete blood count demonstrated eosinophilia. Lumbar puncture was performed with an opening pressure of 450 mmH₂O. The leukocyte count in the cerebrospinal fluid (CSF) was 210 cells/mm³, with 20% eosinophils. No bacteria or fungi were detected on CSF culture. The patient was treated with repeat lumbar punctures to relieve the severe headache. However, he still experienced headache. One week after treatment, he developed gradual onset of blurred vision in his left eye. The vi-

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sual acuities of the right and left eyes were 20/30 and 20/600, respectively. The intraocular pressure in both eyes was 15 mmHg. The pupil diameters were 3 mm in both eyes with a positive relative afferent pupillary defect (RAPD) on the left side. A constricted visual field on his left eye was detected by Goldmann perimetry. The visual evoked potential (VEP) showed prolonged latency for his left eye. Inflammatory cells were seen in the vitreous cavity at 1+, but no cells were seen in the anterior chamber. Funduscopic examination showed disc swelling with hyperemia of the left eye with retinal pigment epithelial alteration at the mid-peripheral retina and fovea. A larva of *A. cantonensis* was found in the vitreous space of the left eye. The parasite was a slightly moving translucent nematode with some pigment. His serum was positive for *A. cantonensis* by immunoblot method (Maleewong *et al*, 2001). An argon green laser was then directly applied to the parasite. Intravenous methylprednisolone (250 mg) was administered every 6 hours for three days followed by a high dose oral prednisolone. The headaches and optic disc swelling of the left eye resolved completely by ten days and four weeks of the treatment, respectively. At eight months follow-up, the patient's visual acuity of the right and left eyes were 20/20 and 20/400, respectively. Inflammation in vitreous cavity resolved, but the retinal pigment epithelial alteration persisted.

DISCUSSION

Deterioration of vision is a presumptive symptom of ocular involvement of ocular angiostrongyliasis that may or may not be associated with meningitis (Sawanyawisuth *et al*, 2007). *A. cantonensis*'s larvae usually infect the subarachnoid space or brain parenchyma. Only a few worms migrate to pulmonary arteries (Sonakul, 1978) or move randomly to other tissues such as cranial nerves or orbits. Hence, ocular symptoms develop after eosinophilic meningitis in cases with ocular involvement and coexisting meningitis.

A. cantonensis can also cause necrotizing retinitis in the eye (Liu *et al*, 2006).

The presence of a relative afferent pupillary defect (RAPD or Marcus Gunn pupil), disc edema, vision loss, and prolongation of VEP confirm the diagnosis of optic neuritis of the left eye. Although unilateral optic neuritis can be caused by other disorders such as multiple sclerosis or toxic substances, there was a previously report of an unidentified parasite (Potasman *et al*, 1998).

The detection of the *A. cantonensis* worm and a positive serologic test for *A. cantonensis* confirmed the cause of optic neuritis in this patient. Therefore, intraocular angiostrongyliasis is a possible cause of unilateral optic neuritis. Recently, Wang *et al* (2006) reported a case of eosinophilic meningitis caused by *A. cantonensis* with unilateral optic neuritis without the identification of in intraocular parasite. The increased intracranial pressure was postulated to explain the unilateral optic neuritis. In our case, we believe the worm directly caused optic nerve inflammation along with high intracranial pressure from eosinophilic meningitis.

Some conditions, such as HIV infection, may be associated with optic neuritis. However, acute HIV infection has been reported to be accompanied by bilateral optic neuritis (Larsen *et al*, 1998). Optic neuritis may present with other neurological disorders such as cranial neuropathy, long tract signs, and/or gait ataxia in advanced HIV infection (Fadil *et al*, 2007).

Although a three-day-course of methylprednisolone results in recovery of visual function in acute demyelinating optic neuritis from multiple sclerosis, there is no clear evidence with angiostrongyliasis. Visual impairment was initially caused by optic neuritis and retinal inflammation. The optic disc swelling completely

subsided by four weeks after initial treatment. This may indicate resolution of inflammatory process in the optic nerve. However, visual acuity did not improve due to permanent damage to or persistent retinal pigment epithelial alteration. We recommend corticosteroid therapy in cases of intraocular angiostrongyliasis with optic neuritis, as well as other inflammatory lesions, such as retinitis (Liu *et al*, 2006) and eosinophilic meningitis. The therapeutic aim of corticosteroid in these conditions is to eradicate the acute inflammatory process. Anthelmintics are still controversial in the treatment of intraocular angiostrongyliasis.

This case brings out another facet to the spectrum of causes of unilateral optic neuritis, a parasitic agent.

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