methylprednisolone 1000 mg daily, followed by oral prednisolone 30 mg daily. During this treatment, diabetes mellitus was controlled with insulin. His visual acuity became 0.9 in the right eye and 1.2 in the left eye. MRI of the head again detected no abnormalities 2 months after the initial visit.

Three months after the initial visit, the patient returned to our hospital with sudden loss of vision. His visual acuity was no light perception in both eyes. He also showed blepharoptosis and complete ophthalmoplegia on the right side. Emergency CT scan of the head disclosed an ill-defined, soft-tissue-density mass lesion extending from the upper ethmoid sinus to the right orbital apex and the suprasellar region (Fig. 1). He underwent biopsy of the lesion by right frontal craniotomy. A part of the thickened, whitish meningeal tissue extending from the tuberculum sella to the right anterior clinoid process was resected for pathological examination. During the resection, pus-like fluid spurted out and red mucosa-like tissue was observed deeply underneath. Pathological examination revealed infection by a fungus of the genus *Aspergillus*, with granulation tissue (Fig. 2). Culture confirmed *Aspergillus fumigatus*. Intravenous amphotericin B was started, making it responsive to pulse corticosteroid therapy, but the patient died of hydrocephalus and pulmonary embolism 5 months after the initial visit.

**Comments**

In this patient, craniotomy revealed granulation tissue with *Aspergillus* infection as the cause of the ill-defined mass extending from the suprasellar region to the right orbital apex. As the most plausible route of infection, mucosal infection by *Aspergillus* in the ethmoid sinus would extend intracranially to the suprasellar region and then to the orbital apex, causing bilateral optic neuropathy at the initial stage and then complete ophthalmoplegia at the final stage. The patient was taking oral prednisolone for years and had diabetes mellitus, indicating that he was in an immunocompromised state. He was therefore prone to develop an infection such as aspergillosis. In the initial phase, repeat CT scan or MRI disclosed no abnormalities and optic neuropathy was responsive to pulse corticosteroid therapy, making it difficult to reach the diagnosis of aspergillosis.

Central nervous system aspergillosis either extends from the contiguous paranasal sinuses or spreads hematogenously from a focus of active pulmonary infection. Aspergillosis has been reported to cause orbital apex syndrome, as in this patient. Aspergillosis in the central nervous system is difficult to diagnose and has a poor prognosis. Repeat CT scan or MRI is mandatory to establish the correct diagnosis.

**Key Words:** aspergillosis, ethmoid sinus, optic neuropathy, orbital apex syndrome

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**References**


**Bilateral Isolated Acute Optic Neuritis in a Child After Acute Rubella Infection**

Optic neuritis is an acute demyelinating disorder of the optic nerve. It may occur in patients with multiple sclerosis (MS) or as a monosymptomatic event, which is referred to as isolated optic neuritis.

We report a pediatric case of anterior optic neuritis after acute rubella infection in the absence of encephalomyelitis.

**Case Report**

A 6-year-old girl presented with bilateral decreased vision. She had a history of fever, rash, and bilateral posterior auricular lymphadenopathy of 28 days, which led to the diagnosis by a local pediatrician of rubella infection. Visual impairment had begun 10 days after complete recovery from the infection, with marked loss of vision for 4 days. The parents were consanguineous. The patient had been vaccinated against rubella, and did not have any previous ophthalmologic complaints. On initial examination, visual acuity was 20/800 in the right eye and 20/400 in the left eye. Light pupillary reactions were weak bilaterally. The optic discs were clearly swollen, edematous, and hyperemic, and retinal vessels were tortuous (Fig. 1). Results of the other neurological and systemic physical examinations were unremarkable.

The laboratory examinations revealed a normal complete blood count and blood chemistry. Serum rubella IgM
titer was 3.4 (normal range <0.8); IgG was 53.4 (normal range <10) IU/ml. Cerebrospinal fluid (CSF) IgM and IgG titers were 1.81IU/ml (normal range <0.8) and 28.73IU/ml, respectively; CSF pressure was 7mmHg. Oligoclonal bands were not detected. The results of computed tomography and magnetic resonance imaging of the brain and both eyes were normal.

Anterior optic neuritis was diagnosed, associated with acute rubella infection in the absence of encephalomyelitis. The treatment of the patient involved high-dose methylprednisolone (30mg/kg per day for 3 days, 20mg/kg per day for 3 days, and 10mg/kg per day for 2 days), followed by oral prednisone 1mg/kg per day, which was tapered off within 1 month. By the end of the first week, bilateral visual acuity had improved to 20/200. Optic disc swelling had improved bilaterally. Serum rubella IgG titers in the first and second weeks of treatment were 102 and 213IU/ml. Rubella IgM titer was 2.2IU/ml (0.8 > NIU/ml) 2 weeks after the corticosteroid therapy. By the end of the first month, visual acuity was 20/20 in both eyes and the swelling of the optic discs was completely resolved (Fig. 2). The light pupillary reactions were almost normal.

**Comments**

Rubella infection is known as an innocent disease of childhood with very few and rare complications. Neurological involvement is also rare, with encephalitis being the most common. Although there are some reports of bilateral anterior optic neuritis after measles/rubella vaccination, only one case of optic neuritis without encephalomyelitis after acute rubella infection has been reported. The patient in that report was an 11-year-old boy, who developed severe visual loss bilaterally 1 week after the onset of rash. Only a topical corticotrophin gel was used, and a complete recovery was achieved within 17 days. Rubella-specific IgM was present in all sera up to day 197. In our case, the onset of the ophthalmologic symptoms was later than in the case described above.

The mechanism of corticosteroid therapy for the treatment of optic neuritis is unclear; however, the good response of our patient is suggestive of an autoimmune mechanism. Severe and bilateral loss of vision in our patient was the reason for initial intravenous therapy. The fast recovery of our patient could be attributed to the intravenous therapy; however, since most postviral optic neuritis cases recover spontaneously, it may not be necessary to use steroids in all such cases unless they are associated with severe bilateral visual loss. On the other hand, the possibility of permanent loss of vision cannot be totally excluded in such a patient until the pathophysiology is clear.

Despite rare neurological complications, mostly in the form of encephalitis, after childhood rubella infections, it should be kept in mind that isolated optic neuritis presenting with acute loss of vision can also occur, and in severe cases corticosteroids may be helpful to shorten the time of recovery.

**Key Words:** optic neuritis, rubella

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