Optic neuritis is a disease characterized with subacute monocular visual loss in days or weeks. It occurs mostly in young patients with a mean age of 32 years, and more often in females. About 65% of the patients show normal appearance of the optic disc; and that’s what is called retrobulbar type. Patients show dyschromatopsia, and a relative afferent pupillary defect is always noted unless in bilateral cases. The visual field defects are usually general depression, altitudinal field loss or central scotoma. In typical cases, the visual acuity always improves within one month. The visual prognosis is always good, and 95% of patients recover to a level of 20/40 or better. In such condition, a scan including the course of the optic nerve is not necessary, as suggested by the Optic Neuritis Treatment Trial (ONTT). The ONTT later reported that the result of MRI obtained in this setting could have prognostic significance with regard to recurrent episodes or progression to multiple sclerosis. However, the incidence of multiple sclerosis is low in Asian people, so a scan in optic neuritis may not be so practical in Asian people.

CASE REPORT

A 39-year-old female came to our outpatient department with the chief complaint of recurrent episodes of blurred vision of her left eye with a central black shadow. She was treated elsewhere with oral prednisolone 2 months before this visit, and the vision of the left eye improved then. At visit, the visual acuity of the left eye was 6/5, and the left 6/10. The anterior segment was quiet bilaterally. The fundus examination was also normal except for mild pigmentary change of macula, OS. The disc showed clear margin, pinkish color and no elevation, OU. The first impression was a sequela of central serous chorioretinopathy.

Two weeks later, the patient came back with further decrease in the vision of her left eye. The visual acuity was 6/5 in her right eye still, but the vision of the left eye went down to “counting fingers” only. Painful sensation on eyeball movement was also noted around the left eye. There was no proptosis bilaterally. The oculomotor examination remained nothing particular. There was a marked RAPD sign in her left eye. The color vision of the right eye was...
normal, and totally absent in the left eye. The visual field was normal in the right eye, and there was a central scotoma with general depression of the left eye (Fig. 1). The visual evoked potential was delayed in the left eye.

Under the impression of retrobulbar optic neuritis, OS, the patient was treated with methylprednisolone 1g/day intravenously. The vision recovered well and rapidly. Three days later, the visual acuity improved to 6/8.6, and the color vision improved to 12/21 on Ishihara color testing plate. So she was discharged with oral prednisolone 60 mg/day. However, because of intolerance to high-dose oral prednisolone, she developed dyspnea and lower leg edema. Rapid reduction of the dosage to 20 mg/day 5 days later was decided to alleviate the systemic symptoms.

One week later, she noted exacerbation of the left eye vision again. The visual acuity was 1/60 then, and the color vision was 1/21 of the left eye, while the visual function of the right eye remained normal. We increased the dosage of prednisolone to 50 mg/day, but 2 weeks later, the visual acuity only improved to 3/60, and the color vision did not show any improvement. During that time, the ocular examination remained unremarkable,

Fig. 1. The visual field of the patient on first admission (Octopus G1 program).

Fig. 2. MRI scan of this patient showed a large tumor mass in the planum sphenoida and tuberculum sella region. (A) axial view post-contrast T1; (B) coronal view T2.
and she was admitted again for further survey.

On second admission, the visual acuity of the left eye was light perception only, and 6/6 in her right eye. The visual evoked potential was not recordable from her left eye. Brain MRI was done, and a 3.2.5 x 2.5 cm tumor mass was found in the planum sphenoida and tuberculum sella region, with compression of bilateral optic nerves (Fig. 2). So, she was sent to the neurosurgical department for management. A left craniotomy with removal of the tumor mass with duraplasty and reconstruction of the skull base was done smoothly. The pathological report was meningioma, meningotheliomatous type.

After removal of the tumor, her left eye vision had no light perception. Gradually, the left optic disc became pale (Fig. 3). Two months postoperatively, the vision of her left eye recovered to “hand motion” at 20 cm.

**DISCUSSION**

Optic neuritis is diagnosed based on clinical symptoms and signs, so it is not rare to misdiagnose a disease as an optic neuritis, especially the retrobulbar type. In this case, there were several clues that were misleading. First of all, there was no proptosis in appearance, no headache from history, and the visual loss was relatively quick compared with the “traditional image” of a brain tumor. These did not lead us to an impression of a brain tumor. Also, the patient was a female in the fourth decade and showed pain on ocular movement, and there were no obvious visual field defects in the other eye. These led us to the impression of a retrobulbar optic neuritis.

Compressive optic neuropathy caused by a suprasellar tumor is not rare at all. Generally speaking, there are signs such as proptosis, disc swelling, headache, progressively deterioration of visual symptoms, or other neurological signs. These were not obviously found in this particular patient, who finally was proved to be a case of compressive optic neuropathy caused by a suprasellar meningioma.

Another interesting point in this case was the rapid and excellent response to intravenous methylprednisolone, which is also seen in optic neuritis cases. However, this condition can happen in some infiltrative disorders like leukemia, but not so often in a solid-mass lesion like meningioma. Prednisolone might reduce the edema surrounding a tumor, and alleviate the compressive effect. Usually, it takes a long time for a brain tumor to cause visual disturbance, and the victim usually perceives persistent and progressive visual dysfunction instead of sudden visual loss or fluctuation of visual functions, but these were what this patient showed. In this case, it seemed
that the tumor as well as the perifocal edema extending from the meningioma produced the compressive effect on the optic nerve, the latter can be alleviated by steroid effect, however, the former only by surgical removal.

It seems that a neuroimaging study in every case is the best tool to avoid misdiagnosis. However, the ONTT concluded that neuroimaging is of limited practicality and is not cost-effective in the diagnosis of optic neuritis. In the rare cases of a compressive lesion masquerading as optic neuritis, the patients’ atypical courses will alert the clinician to the need for neuroimaging studies. That was exactly what happened in this case, and a delay in diagnosis and treatment seemed unavoidable. To our regret, the left eye vision once returned to 6/6 but ended with “hand motion” only. It might have been better if the diagnosis and management had not been delayed.

In an analysis of blindness from intracranial tumors in 60 cases, Zhou et al. found 61.7% of blinding tumors located in the sellar region in 60 cases. Most of them were pituitary adenoma (22 out of the 60), and only 4 were meningioma. The ocular findings showed papilledema in 17 cases (28.3%), and optic atrophy in 40 cases (66.7%). Monocular blindness was noted in 37 patients; 10 showed decreased vision in the other eye also. Bilateral blindness was noted in 23 patients. It may be due to the conservative personality and tradition in Chinese people that a delay of seeking medical help usually presents. So, there is such a high ratio of blindness, as well as high percentage of optic atrophy. Additionally, of the 60 patients, 5 had been diagnosed previously as optic neuritis, retrobulbar optic neuritis, or optic nerve ischemic lesions.

Symon and Rosenstein reported that in 101 patients with suprasellar meningiomas, loss of vision was noted as the first symptom in 74.2%. The Ophthalmologists may be the first physicians they consulted. If we could find the tumor earlier, the patients might not go completely blind. Rosenstein and Symon reported that 41.8% of patients who complained of monocular visual loss also showed some vision reduction of the other eye. This may be an important clue to the ophthalmologists for the need of a neuroimaging study.

The experience of Jallu et al. in Saudi Arabia is more fascinating. They reported 70 patients with suprasellar meningioma, and in 80% of them, the tumors were larger than 5 cm in diameter. Sixty patients showed deterioration of vision as the primary symptom. Thirty-four of them also showed headache before visit. The correlation of vision loss and intracranial tumor is close, especially in suprasellar tumors. In this report, 31 patients showed no light perception in one or both eyes, and bilateral optic atrophy was noted in 45 patients. This also might be due to a delay in seeking medical help. It’s a tragedy that so many people went blind because of a disease that could have been treated earlier.

In conclusion, diagnosis of optic neuritis is based on clinical features. Usually, an MRI scan is done to predict the possibility of multiple sclerosis. In an Asian country like Taiwan, the population of multiple sclerosis is scanty, so a MRI is not routinely done for every patient. However, any patients that showing atypical courses should be scanned, and a brain tumor causing compressive optic neuropathy should be ruled out.

REFERENCES