



Case Report

Unilateral Abducens Nerve Palsy in Increased Intracranial Pressure Caused by Brain Neoplasm

Zei-Lun Huang¹, Chen-Nen Chang², Min-Muh Sheu¹, Rong-Kung Tsai^{1*}

¹Department of Ophthalmology, Buddhist Tzu Chi General Hospital and Tzu Chi University, Hualien, Taiwan

²First Division of Neurosurgery, Department of Surgery, Chang Gung Memorial Hospital, Taoyuan, Taiwan

Article info

Article history:

Received: May 3, 2007

Revised: June 13, 2007

Accepted: August 7, 2007

Keywords:

Abducens nerve palsy

Esotropia

Increased intracranial pressure
(IICP)

Meningioma

Abstract

A 45-year-old woman with a 2-year history of chronic headaches and intermittent transient obscured vision presented with acute onset of diplopia for 2 weeks. Ophthalmologic examination showed esotropia due to right abducens nerve palsy and bilateral chronic papilledema. Brain magnetic resonance imaging demonstrated a meningioma in the left parasagittal frontal region causing a right shift of the anterior midline and compression of the lateral ventricle. The tumor was completely removed and her diplopia and esotropia disappeared. At the 2-month follow-up, papilledema had subsided. Acute onset of esotropia resulting from right abducens nerve palsy may be an ominous sign of brain tumor. (*Tzu Chi Med J* 2007;19(4):253–256)

*Corresponding author. Department of Ophthalmology, Buddhist Tzu Chi General Hospital, 707, Section 3, Chung-Yang Road, Hualien, Taiwan.

E-mail address: rktsai@tzuchi.com.tw

1. Introduction

The most common causes of isolated abducens nerve palsy in adults are diabetes mellitus, vascular disease and post-viral infections. The prognosis in these conditions is usually good. In adults, the differential diagnoses of isolated abducens nerve palsies include non-localizing processes and localizing mass lesions. The former include vasculopathy, myasthenia gravis, thyroid eye disease, restrictive myopathy, and other refractory or strabismus problems (1,2). The latter include pontine syndromes (3–6), middle fossa disorders and cavernous sinus lesions (e.g., neoplasms, inflammatory lesions, infection, vascular lesions) (2–7). We present this case of acute onset of abducens palsy caused by a brain neoplasm and focus on how to prevent misdiagnosis.

2. Case report

A 45-year-old woman presented to our ophthalmologic clinic in December 2003 with acute onset of binocular horizontal double vision while driving 2 weeks previously. She had developed recurrent but transient obscured vision in the past 2 years. She had headaches that were more severe at night without localization. She covered one eye alternatively to eliminate double vision. She did not present with meningismus, nausea and vomiting, vertigo, nystagmus or tinnitus. She denied having diabetes mellitus and hypertension.

Ocular examination showed esotropia in the primary position. The extraocular movement showed 90% restriction of abduction in the right eye. The Hess chart screen revealed weakness in abduction

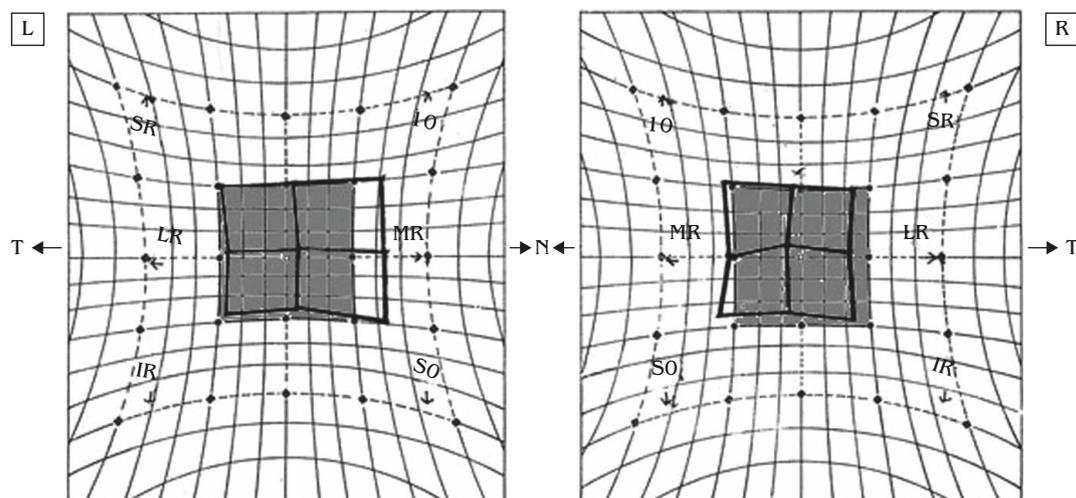


Fig. 1 — The Hess chart screen reveals weakness of the right lateral rectus muscle. Bilateral central gray squares show orthotropia. In the right eye, the lateral rectus (LR) muscle shows some limited abduction. In the left eye, the medial rectus (MR) muscle shows compromised over-action.

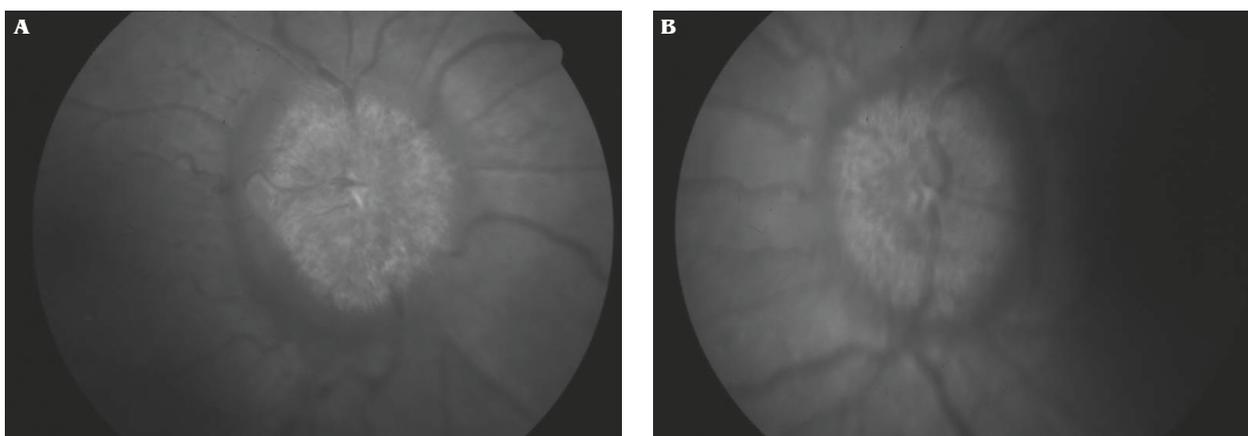


Fig. 2 — Disc pictures (OU) show bilateral chronic papilledema without hemorrhage or exudates.

of the right eye (Fig. 1). Her visual acuity was 20/25 OD and 20/200 OS (previous amblyopia) and color vision was 16/16 on Ishihara plates OU. Her pupil sizes were equal without relative afferent pupillary defect. The confrontation test showed no obvious visual defect. Intraocular pressure was normal. Slit-lamp examination was normal OU. Fundoscopy showed bilateral papilledema without hemorrhage or exudates (Fig. 2). The impression was increased intracranial pressure (IICP) with right abducens nerve paresis. Brain magnetic resonance imaging disclosed an intense enhanced mass with perifocal edema, causing a mid-line shift and compression of the frontal horn of the lateral ventricle, favoring a diagnosis of meningioma (Fig. 3).

The patient was referred to a neurosurgeon for further treatment. The well-encapsulated tumor was

removed completely. Pathological study showed whorl-like spindle cells and psammoma bodies, indicating a Grade I meningioma (meningothelial meningioma) (Fig. 4). One day after surgery, the patient noted that her double vision had disappeared and eye movement had normalized. Follow-up disc photos 2 months postoperatively showed that the papilledema had subsided (Fig. 5).

3. Discussion

Patients with vasculopathic abducens nerve palsy are identified at a mean age of 65 years and complete recovery is the general rule (8). In a population-based study, the peak incidence of abducens nerve palsy was in the seventh decade of life. In one study

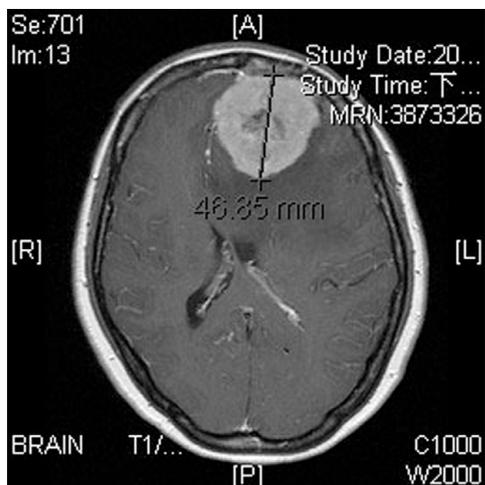


Fig. 3 — Magnetic resonance imaging documents an intense enhanced 52×40×40 mm tumor in the left parasagittal frontal region with marked perifocal edema. The mass effect is causing a midline shift and compression of the lateral ventricle.

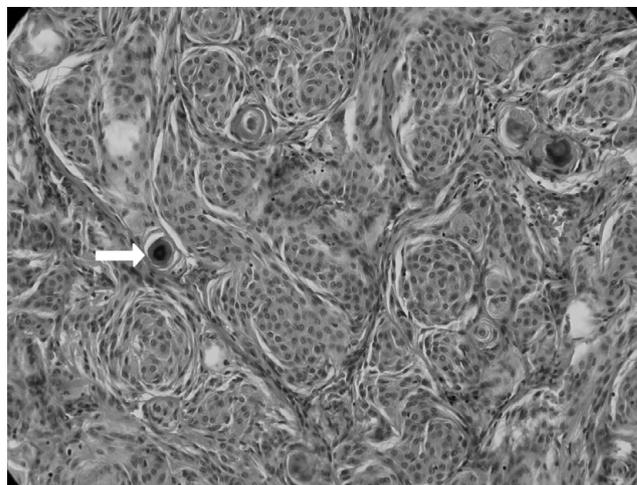


Fig. 4 — Hematoxylin and eosin staining shows whorl-like spindle cells and psammoma bodies (400×), characteristic of Grade I meningioma (meningothelial meningioma).

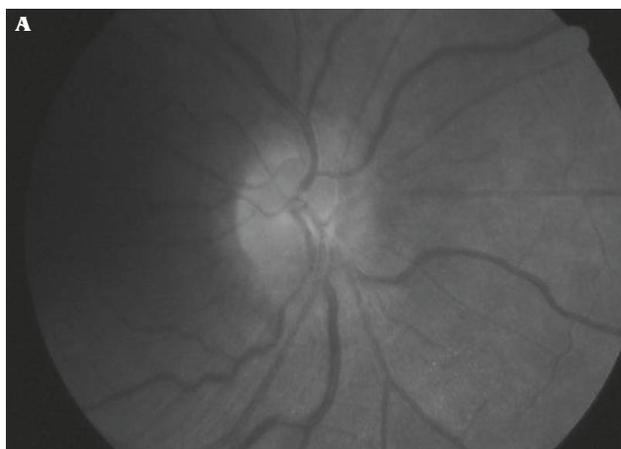


Fig. 5 — Disc swelling (OU) has subsided 2 months after surgery.

of 137 cases, the causes were undetermined (26%), hypertension alone (19%), coexistent hypertension and diabetes (12%), trauma (12%), multiple sclerosis (7%), neoplasm (5%), and aneurysm (2%) (9). In a retrospective case series, the most common cause of abducens nerve palsy in 45 young adults (mean age, 39 years; age range, 20–50 years) was a central nervous system mass lesion (33%), followed by multiple sclerosis (24%), idiopathic (13%), viral infection (9%), and idiopathic intracranial hypertension (7%) (10). The most common causes of acute abducens palsy are different in young, middle-aged and elderly patients.

Acute comitant esotropia with abducens nerve palsy has been described in intracranial tumors caused by acute obstructive hydrocephalus (11–13). Robertson

et al reported 133 children with acute abducens paresis and found intracranial neoplasms in one third of cases (14). Chen et al also reported eight patients whose acute esotropia was the presenting sign of intracranial neoplasm (15). Acute esotropia may be an initial presentation of brain tumor and careful evaluation for the possibility of intracranial tumor is important, especially in young and middle-aged patients. Our patient was 45 years old and had no history of hypertension or diabetes mellitus. She did not belong to an age group in which vasculopathy would be suspected first. Our patient was middle-aged. Neuroimaging should be done immediately when neurological symptoms and signs are found in young or middle-aged patients with acute abducens paresis. If a middle-aged Chinese person has sixth nerve

palsy without papilledema, nasopharyngeal carcinoma or another tumor interfering with the abducens nerve should be considered.

The abducens nerve begins as a nucleus in the pons, enters the subarachnoid space and passes through the cavernous sinus and superior orbital fissure to the lateral rectus muscle (16). Some patients with pontine hemorrhage could present with isolated abducens palsy, which could be misdiagnosed as a lesion of diabetic or vascular origin (3–6). The location of the lesion affects the symptoms and signs. For example, IICP in our case caused non-localized palsy, possibly due to compression or stretching of the right abducens nerve in the subarachnoid space against the sharp edge of bone. Why is the abducens nerve the most frequently affected cranial nerve in cranial infections and high cranial pressure? In infections, the nerve is probably involved due to its medial location in the cavernous sinus (17,18); its long intracranial course probably makes it sensitive to elevations in intracranial pressure (19,20).

Compared with unilateral involvement, bilateral abducens nerve palsies more commonly occur with neurogenic insults such as tumors, demyelination, hemorrhage, meningitis, and IICP. Kocak et al reported a case of isolated bilateral abducens nerve palsy with metastatic neoplasm involving the skull base and bilateral cavernous sinus (21). Chang presented a case of isolated left abducens nerve palsy with carcinomatous meningitis confirmed by cytology of the cerebral spinal fluid (1).

In our patient, right abducens palsy with a presentation of IICP resulted from the mass effect of a parasagittal meningioma. Her transient obscured vision associated with headache was a symptom of papilledema and IICP. Transient obscured vision is a sudden onset of blurred vision for several seconds after a change in posture from sitting or standing, followed by quick recovery. Understanding this meaning of the symptom could lead to early diagnosis.

Most cases of abducens palsy caused by elevated intracranial pressure rapidly resolve as pressure is relieved. In our patient, immediate abducens palsy relief was noted 1 day postoperatively, and papilledema subsided 2 months later. In one study, fully developed papilledema was relieved 6–8 weeks post-craniotomy (22). However, there may be sequelae of optic atrophy if the papilledema lasts too long.

The most common causes of isolated abducens nerve palsy in adults are diabetes mellitus and vascular disease, especially in elderly patients (9,23). This case alerted us to differentiate isolated abducens palsy caused by a non-localizing process from that caused by a localizing mass. We emphasize the importance of careful evaluation of acute onset of esotropia and diplopia in young and middle-aged patients.

References

1. Chang AK. Diplopia in a patient with carcinomatous meningitis. *J Emerg Med* 2002;23:351–4.
2. Eggenberger E. A brutal headache and double vision. *Surv Ophthalmol* 2000;45:147–53.
3. Donaldson D, Rosenberg NL. Infarction of abducens nerve fascicle as cause of isolated sixth nerve palsy related to hypertension. *Neurology* 1988;38:1654–60.
4. Fujioka T, Segawa F, Ogawa K, et al. Ischemic and hemorrhagic brain stem lesions mimicking diabetic ophthalmoplegia. *Clin Neurol Neurosurg* 1995;97:167–71.
5. Fukutake T, Hjrayment K. Isolated abducens nerve palsy from pontine infarction in a diabetic patient. *Neurology* 1992;42:2226–30.
6. Johnson LN, Hepker RS. Isolated abducens nerve paresis from intrapontine, fascicular abducens nerve injury. *Am J Ophthalmol* 1989;108:459–61.
7. Ada M, Kaytaz A, Tuskan K, et al. Isolated sphenoid sinusitis presenting with unilateral VIth nerve palsy. *Int J Ped Otorhinolaryngol* 2004;68:507–10.
8. Sanders SK, Kawasaki A, Purvin VA, et al. Long-term prognosis in patients with vasculopathic sixth nerve palsy. *Am J Ophthalmol* 2002;134:81–4.
9. Patel SV, Mutyala S, Leske DA, et al. Incidence, associations, and evaluation of sixth nerve palsy using a population-based method. *Ophthalmology* 2004;111:369–75.
10. Peters GB, Bakri SJ, Krohel GB. Cause and prognosis of nontraumatic sixth nerve palsies in young adults. *Ophthalmology* 2002;109:1925–8.
11. Astle WF, Miller SJ. Acute comitant esotropia: a sign of intracranial disease. *Can J Ophthalmol* 1994;29:151–4.
12. Hoyt CS, Good WV. Acute onset concomitant esotropia: when is it a sign of serious neurological disease? *Br J Ophthalmol* 1995;79:498–501.
13. Hoyt WF, Daroff RB. Supranuclear disorders of ocular control systems in man. In: Bach-y-Rita P, ed. *The Control of Eye Movements*. New York: New York Academic Press, 1971.
14. Robertson DM, Hines DJ, Rucker CW. Acquired 6th nerve paresis in children. *Arch Ophthalmol* 1970;83:574–9.
15. Chen HY, Wu DL, Tsai RK. Acute esotropia may be a presenting sign of intracranial neoplasm. *Kaohsiung J Med Sci* 1998;14:710–6.
16. Porter JD, Baker RS. Anatomy and embryology of the ocular motor system. In: Miller NR, Newman NJ, eds. *Clinical Neuro-Ophthalmology*, 5th edition. Baltimore: Williams & Wilkins, 1998: Chapter 25.
17. Deans JA, Welch AR. Acute isolated sphenoid sinusitis: a disease with complications. *J Laryngol Otol* 1991;105:1072–4.
18. Sethi DS. Isolated sphenoid lesions: diagnosis and management. *Otolaryngol Head Neck Surg* 1999;120:730–6.
19. Erans RW. Complications of lumbar puncture. *Neurol Clin* 1998;16:83–105.
20. Niederm LU, Trinka E, Bauer G. Abducens palsy after lumbar puncture. *Clin Neurol Neurosurg* 2002;104:61–3.
21. Kocak Z, Celik Y, Uzal MC, et al. Isolated bilateral sixth nerve palsy secondary to metastatic carcinoma: a case report with a review of the literature. *Clin Neurol Neurosurg* 2003;106:51–5.
22. Miller NR. Papilledema. In: Miller NR, Newman NJ, eds. *Clinical Neuro-Ophthalmology*, 5th edition. Baltimore: Williams & Wilkins, 1998: Chapter 10.
23. Shrader EC, Schlezinger NS. Neuro-ophthalmologic evaluation of abducens nerve paralysis. *Arch Ophthalmol* 1960;63:108–15.