

Sixth nerve palsy of para-infectious origin

Dirk Deleu, MD, PhD, Menandros Lagopoulos, MD, PhD.

ABSTRACT

Acute abducens palsy of para-infectious origin is uncommon in adulthood. We present the case history of a 28-year-old woman suffering from acute onset diplopia due to abducens palsy. Magnetic resonance imaging revealed signs suggestive of frank ipsilateral maxillary sinusitis. The lack of anatomical relationship between the maxillary sinus and the abducens nerve led us to conclude that the abducens palsy was of para-infectious origin.

Keywords: Para-infectious abducens palsy.

Neurosciences 2001; Vol. 6 (2): 122-124

Isolated abducens nerve palsy is the commonest type of extraocular palsy and can result from any lesion along the long intracranial course of the nerve.^{1,2} Lesions of the nerve need to be ruled out at the skull base, in the subarachnoid space, the tip of the petrous temporal bone, cavernous sinus, superior orbital fissure or orbital cavity. We report a case of isolated abducens nerve palsy in a young woman with ipsilateral maxillary sinusitis in the absence of vascular risk factors or any lesion along the course of the abducens nerve. Isolated abducens nerve palsy of para-infectious origin has not been reported in relation with maxillary sinusitis.

Case reports. A 28-year-old Indian woman presented with a 10 day history of acute onset horizontal diplopia which was accompanied for the last two days with vague right temporal headache and retrobulbar pain. There was no history of fever, immunization, head trauma, hypertension, diabetes or any other systemic disorder.

Neurological examination on admission showed an alert patient with normal language ability and cognitive functions. Neuro-ophthalmologic examination revealed normal visual acuity, field and

fundi. Pupils were equal, and normally reactive to light and near stimuli. No ptosis or proptosis was observed. There was mild right esotropia in primary position. The extraocular eye movements were full except for complete right abducens nerve palsy. Forced duction test was normal. Attempted eye movements to the right were not painful. No nystagmus or internuclear ophthalmoplegia was observed. Other cranial nerves were functioning normal and the rest of the neurological examination was also normal. She was afebrile and here blood pressure was 130/80 mmHg. Percussion over the right maxillary sinus was slightly tender. Cardiopulmonary examination and the remainder of the general examination were normal. All serum laboratory values (glucose, glycosylated hemoglobin, electrolytes, renal and liver function) were within the normal range. The following hematological laboratory values were found: white cell-count, 6,600/mm³ with differential count of 82% neutrophils, 2% eosinophils, 15% lymphocytes and 1% monocytes), erythrocyte count 3.84 x 10⁶/mm³, hemoglobin 10.4 g/dl, hematocrit 28.4% and erythrocyte sedimentation rate 35 mm/h.

In view of her origin and lack of any specific signs

From the Department of Clinical Pharmacology and Neurology Clinic (Deleu), Department of Clinical Anatomy (Lagopoulos), College of Medicine, Sultan Qaboos University, Muscat, Sultanate of Oman.

Received 17th June 2000. Accepted for publication in final form 30th July 2000.

Address correspondence and reprint request to: Dr Dirk Deleu, Department of Clinical Pharmacology, College of Medicine, Sultan Qaboos University, PO Box 35, Al Khod, Muscat 123, Sultanate of Oman. Tel. 00 968 515 106. Fax. 00 968 513 419. E-mail: deleu@omantel.net.om

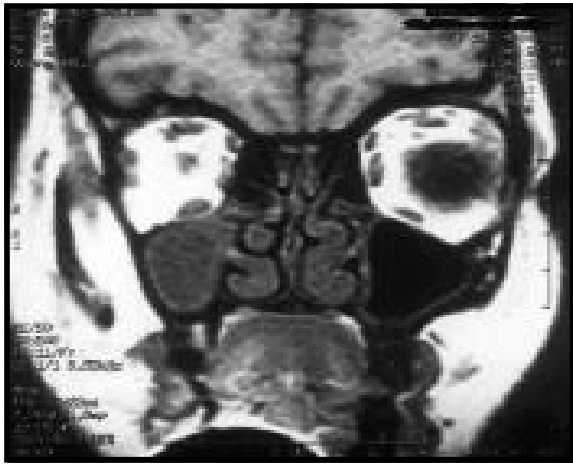


Figure 1 - (a) T1 weighted coronal MRI image showing overt signs of right sided maxillary sinusitis. (b) T1 weighted horizontal MRI image showing overt signs of right sided maxillary sinusitis.

a tentative diagnosis of tuberculous meningitis was considered. Plain skull x-ray (not shown) revealed opacity over the right maxillary sinus. Magnetic resonance imaging of the head revealed frank inflammatory changes in the right maxillary and ethmoidal sinuses (Figure 1). No abnormalities were detected within the brain, skull base, subarachnoidal space, sphenoidal sinuses, cavernous sinus, postorbital, superior orbital fissure or intraorbital. Furthermore, orbital walls did not show any sign of destruction or erosion and the extraocular muscles within both orbits were normal. There were no signs of middle ear infection or petrositis on magnetic resonance imaging (MRI). Cerebrospinal fluid analysis was normal and cultures were negative. Other investigations including electromyography, tensilon test and viral serology (including Epstein-Barr virus, mycoplasma pneumonia, cytomegalovirus) were non contributory. Three weeks after the onset of her symptoms nasal antrostomy was performed and pus was drained from the right maxillary sinus. Although no pathogens were isolated from cultures she was treated with broad-spectrum antibiotics for two weeks. Following the drainage periorbital pain gradually disappeared and over a period of two weeks there was substantial improvement of her diplopia with complete recovery over two months.

Discussion. In our patient, diplopia was due to isolated abducens nerve palsy, since extraocular muscles including the lateral rectus muscle and other intraorbital structures were intact on MRI. Furthermore forced ductions were normal and tensilon test failed to improve diplopia. In addition,

MRI did not show evidence of inflammation along its intracranial course, in particular, at the middle ear, the petrous tip of the temporal bone or sphenoidal sinus. Therefore, it is very likely that we were dealing with an abducens nerve palsy of para-infectious origin. Alternatively, however less likely, the infectious process in the maxillary sinus could have triggered inflammation resulting in compression of the vasa vasorum supplying the abducens nerve.

Transient isolated abducens nerve palsy developing one to three weeks after immunization, viral (varicella, mononucleosis infectiosa, cytomegalovirus) and bacterial infections (*Mycoplasma pneumonia*) or even following non specific (febrile) infections has been reported, particularly in childhood.²⁻⁷ However, this phenomenon is extremely rare in adults. In the age category between 15-50 years, one third of patients with isolated non-traumatic abducens palsies were classified as vasculopathic (diabetes, arteroclerosis, or hypertension). Inflammatory disorders (e.g. multiple sclerosis, meningoencephalitis, accompanying systemic viral infections and post-immunization) accounted for one fifth of cases, while tumors were encountered in 11-16% of cases.⁸⁻¹⁰ Across these studies, in one fourth of patients the etiology of the abducens nerve palsy remained unclear, particularly in the group between 20 and 40 years of age. This large percentage of undefined etiology might however partly be explained by the fact that these series were published before the MRI era. More recently, Miller and co-workers reviewed the records of 407 patients with acquired isolated non-traumatic abducens nerve palsy.¹¹ Using more advanced neuroimaging techniques (high-resolution

computerized tomography scan and MRI) vasculopathies accounted for 60% of causes of abducens nerve palsy while the percentage of undetermined causes was only 16%. It is very likely that the latter group included para-infectious forms. In none of these studies, however, isolated abducens nerve palsy of para-infectious origin was reported in relation with maxillary sinusitis. Large series indicated that the spontaneous recovery rate in acute non-traumatic isolated abducens nerve palsy of vascular and undetermined origin is generally favorable^{8,12}: more than 75% of patients recovered, one third by 8 weeks and 74% by 6 months. In view of this para-infectious origin, we believe that drainage was perhaps not really necessary.

References

1. Kanski JJ. Neuro-ophthalmology. In: Kanski JJ, ed. *Clinical Ophthalmology: a systematic approach*. 3rd ed. Oxford: Butterworth-Heinemann; 1997. p. 476-478.
2. Smith CH. Nuclear and infranuclear ocular motility disorders. In: Miller NR, Newman NJ, eds. *Walsh and Hoyt's Clinical Neuro-ophthalmology*. 5th ed. Baltimore: Williams and Wilkins; 1998. p. 1189-1281.
3. Straussberg R, Cohen AH, Amir J, Varsano I. Benign abducens palsy associated with EBV infection. *J Pediatr Ophthalmol Strabismus* 1993; 30: 60.
4. Christen HJ, Aksu F, Petersen CE. Isolated abducens nerve paralysis in infectious mononucleosis. *Monatsschr Kinderheilkd* 1983; 131: 532-4.
5. Wang CH, Chou ML, Huang CH. Benign isolated abducens palsy in mycoplasma pneumoniae infection. *Pediatr Neurol* 1998; 18: 71-2.
6. Cohen HA, Nussinovitch M, Ashkenazi A, Staussberg R, Kaushansky A. Benign abducens nerve palsies of childhood. *Pediatr Neurol* 1993; 9: 394-5.
7. Lee MS, Galetta SL, Volpe NJ, Liu GT. Sixth nerve palsies in children. *Pediatr Neurol* 1999; 20: 49-52.
8. Rush JA, Younge BR. Paralysis of cranial nerves III, IV, and VI. Cause and prognosis on 1,000 cases. *Arch Ophthalmol* 1981; 99: 76-9.
9. Moster ML, Savinao PJ, Sergott RC, Bosley TM, Schatz NJ. Isolated sixth-nerve palsy in younger adults. *Arch Ophthalmol* 1984; 102: 1328-30.
10. Berlitz P. Isolated and combined pareses of cranial nerves III, IV and VI. A retrospective study of 412 patients. *J Neurol Sci* 1991; 103: 10-5.
11. Miller RW, Lee AG, Schiffman JS, Prager TC, Gazza R, Jenkins PF et al. A practice pathway for the initial diagnostic evaluation of isolated sixth nerve palsies. *Med Decis Making* 1999; 19: 42-8.
12. King AJ, Stacey E, Stephenson G, Trimble RB. Spontaneous recovery rates for unilateral sixth nerve palsies. *Eye* 1995; 9: 476-8.