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Case Reports

Bilateral sixth-nerve palsy associated with dural arteriovenous malformation

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Abstract

A fifty-two year old postmenopausal woman was admitted to the hospital with complaints of diplopia, headache, humming over the head, and pain over the left eye. Neurological examination showed right abducens nerve palsy. In a few days, she also developed left abducens nerve palsy and chemosis, exophthalmos, and proptosis of both eyes. There was pulsation over the left eye. Intraocular pressure was found to be elevated bilaterally. Selective carotid angiograms showed the presence of bilateral dural arteriovenous malformations (AVM) supplied by the external carotid arteries. Two months after the embolisation of the AVMs, eye movements improved. Repeat angiograms showed the absence of flow into the previously embolised AVMs.

Key words : Bilateral abducens nerve palsy ; bilateral dural arteriovenous malformation ; diabetes mellitus ; trauma ; embolisation.

Introduction

Bilateral isolated sixth-nerve palsy is a much less common clinical entity comparing to unilateral involvement. Even though small vessel ischemic infarction in hypertensive and diabetic patients is the most likely cause of unilateral palsy, other diseases like neoplasms, demyelinating disease, trauma, subarachnoid haemorrhage, and meningeal infarction must also be considered when bilateral involvement occurs (Keane, 1976 ; Loster *et al.*, 1984 ; Biousse *et al.*, 1998). Bilateral abducens nerve palsy associated with bilateral dural arteriovenous malformation (AVM) is a rare event. We here describe such a patient.

Case Report

A fifty-two-year old woman was admitted to the hospital with complaints of diplopia, headache, humming over the head, pain over the left eye for a few days. She has been diabetic for 13 years and her blood glucose could not be regulated despite insulin treatment. She has been menopausal for

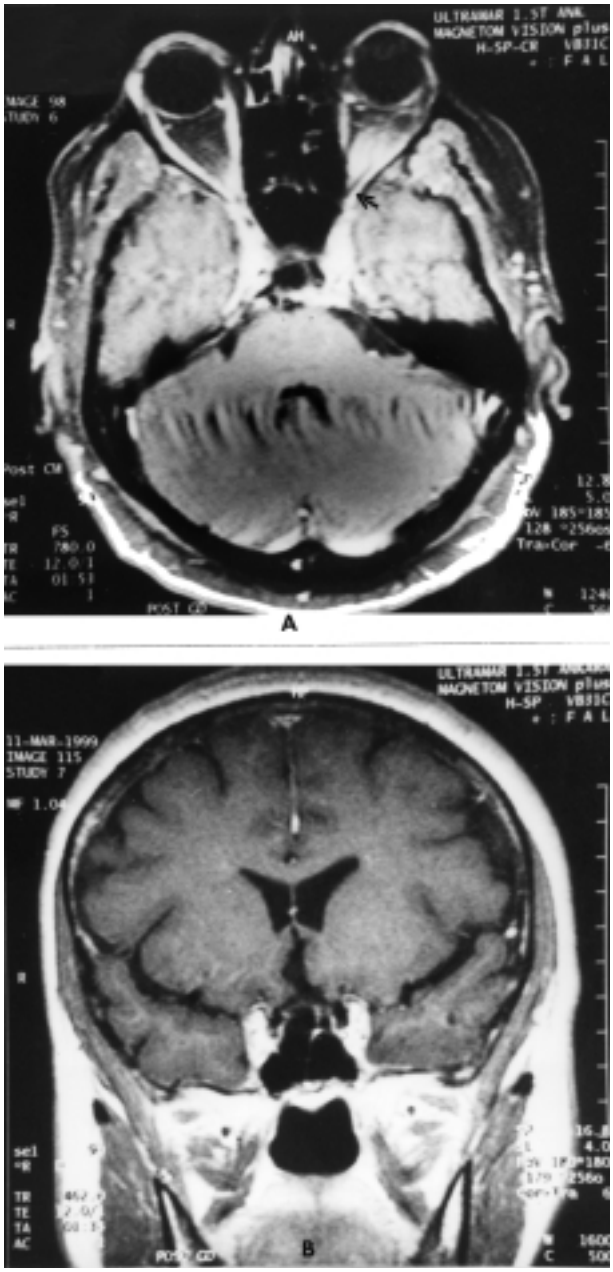

FIG. 1. — Magnetic resonance imaging. (A) T2-weighted axial image showing dilated left superior ophthalmic vein (arrow). (B) T2-weighted coronal image. No cerebral lesions suggestive of sinus thrombosis are present.

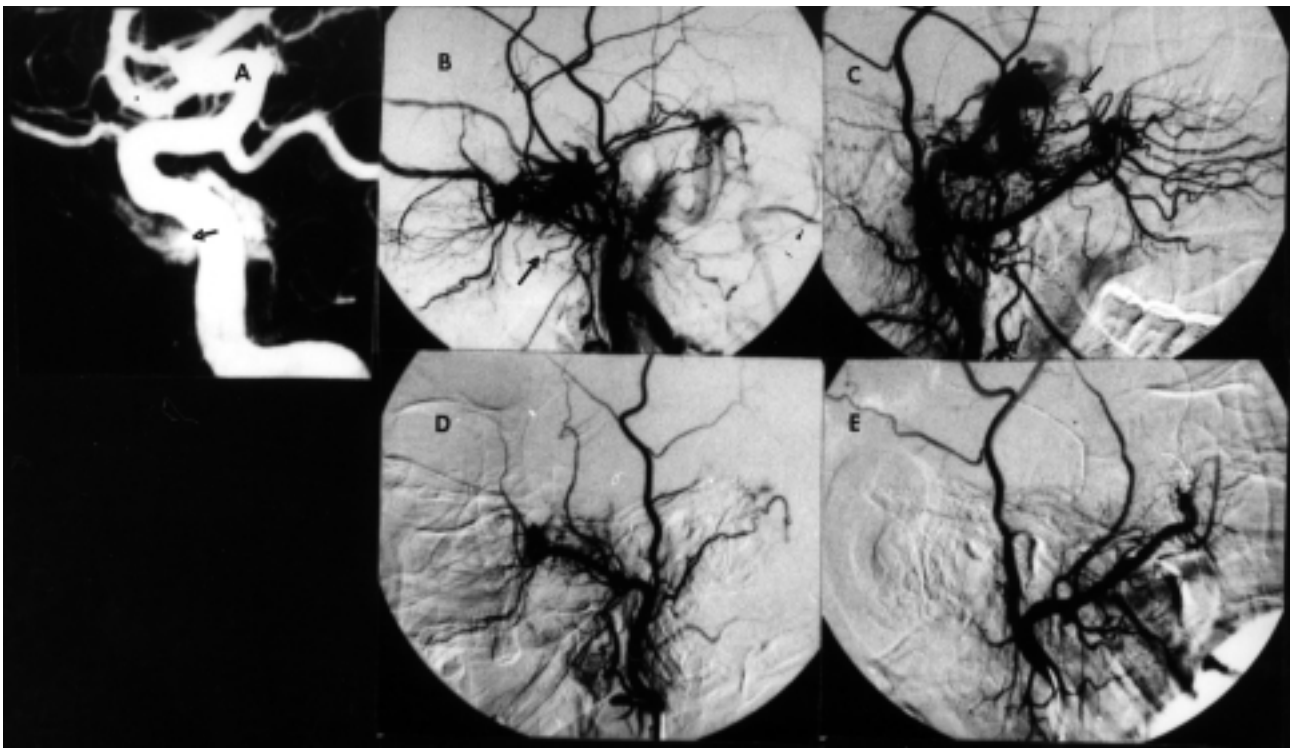

FIG. 2. — Carotid angiograms : Left internal carotid angiogram showing the meningeal branches of carotid artery (arrow) in the left cavernous sinus (A). Left (B) and right (C) dural AVM (arrows) supplied by both external carotid arteries. The left (D) and right (E) dural AVMs can not be visualised after the embolisation.

six years. She described mild head trauma (she hit the left side of the head to the wall during a fight three months ago). Neurological examination was normal except for limited abduction of the right eye. During the next few days, the pain increased and she also developed abduction paresis of the left eye. Smooth pursuit confirmed bilateral abducens nerve palsy. Conjunctival vessels were dilated and tortuous. Both eyes were exophthalmic and proptotic. There was also pulsation over the left eye. Visual acuity and visual field studies were normal. Cranial magnetic resonance imaging (MRI) showed a dilated superior ophthalmic vein on the left side (Fig. 1A). There was no cerebral lesion suggestive of sinus thrombosis (Fig. 1B). Intraocular pressure was elevated bilaterally (30 mmHg on the left, 24mmHg on the right). The left internal carotid angiogram showed the meningeal branches going to the left cavernous sinus (Fig. 2A). Selective carotid angiograms showed bilateral dural AVMs supplied by both external carotid arteries (Fig. 2B,C). The arterial branches of the external carotid arteries that supply the AVMs were embolised bilaterally and obliteration of the flow was confirmed (Fig. 2D,E). There was no flow from the left internal carotid artery to the cavernous sinus either. The embolisation was performed using a mixture of 0.5ml N-butyl-Cyanoacrylate (Histoacryl) and 2ml lipiodol solutions injected through a cerebral microcatheter. Abduction of both eyes became almost normal and exophthalmos regressed to some extent in two months. Repeat selective angiograms showed the absence of flow into the previously embolised AVMs. However, the arterialised conjunctival vessels somewhat persisted. Blood glucose level could never be regulated satisfactorily.

Discussion

Diabetes mellitus and trauma have been reported to cause bilateral abducens nerve palsy (Shrader and Schlezinger, 1960 ; Keane, 1976). In the report by Shrader and Schlezinger (1960) that consists of 104 patients with isolated sixth-nerve paralysis, 15% were diabetic and 2.9% of those patients had antecedent trauma. Only one diabetic patient in that series had bilateral sixth-nerve paralysis. Keane (1976) reported 11 cases of bilateral abducens nerve paralysis caused by trauma. Vascular diseases were listed as etiological factors in both series but no case of dural AVM was reported (Shrader and Schlezinger, 1960 ; Keane, 1976). Considering the presence of proptosis, chemosis, glaucoma, and improvement of the extraocular muscle paresis after embolisation, we suggest the dural AVMs are the etiological factor in this case. Diabetes mellitus and trauma could be contributing factors.

Carotid-cavernous sinus fistulas were previously reported to be associated with ophthalmoplegia (Leonard *et al.*, 1984). The authors suggested

different etiologies for the ophthalmoplegia in a series of 15 patients with carotid-cavernous sinus fistula. Similar to our case, 4 patients had isolated abduction failure and normal size extraocular muscles and most were related to dural fistulas draining to posterior portion of cavernous sinus. However, generalised ophthalmoplegia with enlarged extraocular muscles was usually associated with a direct fistula located more anteriorly. The interesting point of our case is bilateral abduction failure due to bilateral dural AVMs. As far as the central nervous system is concerned, the case could be accepted as a bilateral isolated abducens nerve palsy but ocular involvement was also present. Anatomically, the abducens nerve is located much closer to the internal carotid artery in the cavernous sinus comparing to the oculomotor, ophthalmic, and trochlear nerves which are located at the lateral wall of the sinus (Miller, 1998). The anatomical proximity between abducens nerve and the internal carotid artery might be a factor for the isolated sixth-nerve involvement. Also, a dural fistula related to dorsomeningeal branches might interfere with the blood supply of the VI nerve (Leonard *et al.*, 1984). Depending on its size and hemodynamics, a dural AVM may produce variable symptoms and signs and could affect both sides with more severe involvement on one side (Biousse *et al.*, 1998). In our case, symptoms and signs were more pronounced on the left side. After the embolisation, the ocular motility disturbance disappeared ; however, dilation of the conjunctival vessels persisted which we suggest is a sequela.

Conclusively, bilateral dural AVMs can be an etiological factor for bilateral sixth-nerve paralysis, which can be treated with embolisation.

REFERENCES

Biousse V., Mendicino M. E., Simon D., Newman N. J. The ophthalmology and intracranial vascular abnormalities. *Am. J. Ophthalmol*., 1998, **125** : 527-44.

Keane J. R. Bilateral sixth nerve palsy, analysis of 125 cases. *Arch. Neurol*., 1976, **33** : 681-3.

Leonard T. J. K., Moseley I. F., Sanders M. D. Opthalmoplegia in carotid cavernous sinus fistula. *Br. J. Opthalmol.*, 1984, **68** :128-134.

Loster M. L., Savino P. J., Sergott R. C., Bosley T. M., Schatz N. J. Isolated sixth-nerve palsies in younger adults. *Arch. Ophthalmol*., 1984, **102** : 1328-30.

Miller N. R. Carotid cavernous fistula. In : *Walsh and Hoyt's Clinical Neuro-opthalmology*. 5th ed. Miller N. R., Newman N. J. (eds.). Baltimore : Williams and Wilkins. chapter 17, 1998, pp. 553-5.

Shrader E. C., Schlezinger N. S. Neuroopthalmologic evaluation of abducens nerve paralysis. *Arch. Ophthalmol*., 1960, **63** : 84-91.

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