Ptosis in an elderly man

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An 84-year-old man presented with three-week history of painless ptosis of the right eye. About three months ago he had an attack of iritis which was attended to by an ophthalmologist and treated successfully. Otherwise there were no other associated symptoms, either prior to or after the onset of ptosis. About nine years ago he was found to have hypertension during the evaluation of a transient ischaemic attack. At presentation he was taking sotalol, nifedipine and warfarin, which he has been taking for the last nine years. Until the onset of ptosis he was still playing golf regularly.

Physical examination revealed an elderly gentleman, in good general health, who had walked independently into the clinic. He had complete ptosis of his right eye (figure 1) with complete palsy of cranial nerves III, IV and VI on the right side. His right pupil was dilated and not reacting to light, either directly or consensually. There were no other neurological signs and rest of the examination was unremarkable. The nasal passage was clear and a right antral washout performed under local anaesthesia was also normal for cytology and culture. Chest X-ray, full blood count, clotting profile and routine biochemistry, including random glucose, was normal. A computed tomography (CT) scan was unhelpful in arriving at a diagnosis. Subsequently a magnetic resonance imaging (MRI) scan (figure 2) was done which showed the lesion.

During the process of investigations he was admitted with general deterioration and died before any treatment could be offered to him.

Figure 1 Photograph showing complete ptosis of right eye

Figure 2 MRI scan showing the tumour mass

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Question

What is the most likely cause of the ptosis and ophthalmoplegia in this patient?

Answer

Possible causes of ptosis with ophthalmoplegia are listed in the box. In this particular case bleeding due to over-anticoagulation was another possibility.

Because of involvement of cranial nerves III, IV and VI, a tumour involvement was considered to be the most likely cause, hence MRI of the orbit and base of the skull was done (figure 2) which showed a large mass occupying the posterior ethmoid and sphenoid sinus with destruction of clivus and invasion into the right orbit. *Post-mortem* examination showed that he had suffered a full thickness myocardial infarction. A pink lobulated tumour was also found, filling the ethmoid sinus and extending around the pituitary fossa with no evidence of distant metastasis. Histological examination showed it to be of anaplastic variety.

Discussion

Malignant neoplasms of the paranasal sinus are rare, accounting for only 0.2% to 0.8% of all neoplasms with an incidence of less than 1.0 per 100 000 per year.¹ Squamous cell carcinoma is the most common variety and the maxillary sinus is most frequently involved.^{1,2} Carcinoma of the ethmoidal sinus is rare, accounting for only 13% to 19% of all paranasal neoplasms. Facial pain, facial swelling, nasal obstruction, and epistaxis is the most common presenting feature.² Up to 59% of patients with a paranasal tumour have some sort of orbital involvement, with up to 34% having orbital symptoms and signs at the time of initial hospitalisation.²

Cranial nerve involvement and ophthalmoplegia is a recognised feature of paranasal tumour, however, presentation of paranasal tumour in this fashion is rare. The only other similar reported case in the English literature is of a 79-year-old Chinese woman who had non-Hodgkin's lymphoma of the ethmoid sinus.³ In her case ophthalmoplegia was preceded by a four-week history of pain on the right side of her face. In another case, a papillary carcinoma of the sphenoid sinus in association with an abscess resulted in cavernous sinus syndrome.⁴ There are two other cases in the Japanese literature, one of which was a paranasal malignant lymphoma causing bilateral ophthalmoplegia⁵ and the other an adenoid cystic carcinoma of the sphenoid, resulting in cavernous sinus syndrome.⁶ Our patient was unusual in that ophthalmoplegia with complete ptosis was the only symptom on presentation and remained so during his brief period of survival.

Ptosis (drooping of the upper eyelid) may be the result of paralysis of the levator palpebrae superioris, which is a mixture of striated muscle innervated by the oculomotor nerve and smooth muscle innervated by sympathetic nerves. Hence, myasthenic or myopathic weakness of the levator palpebral superioris or a defect of sympathetic or oculomotor nerve innervation results in ptosis. In some circumstances lesions affecting the eyelid, eg, oedema,

tumours, involutional (senile-degenerative process) can also result in ptosis.

Broadly speaking, neurological lesions cause unilateral ptosis and muscular disorders cause bilateral ptosis but there are many exceptions to the rule. Involvement of sympathetic innervation usually causes partial ptosis whereas a third nerve lesion usually causes complete ptosis, although a partial third nerve lesion may result in partial unilateral ptosis.

Interruption of sympathetic fibres (Horner's syndrome) may be due to a lesion in the hemisphere, brainstem, T1 root, or involvement of the sympathetic chain in the neck. Similarly the oculomotor nerve may be damaged at any point from its origin in the mid-brain to its insertion in the ocular muscles. Because of its close proximity to other cranial nerves in the cavernous sinus, a lesion at this site usually also involves other cranial nerves. Rush and Younge⁷ evaluated 1000 cases of cranial nerve palsy and found that, in the majority of cases of third nerve paralysis, the cause remains unknown. Where the cause was known, the most common was head trauma, neoplasm, vascular disorders (including diabetes mellitus, hypertension and atherosclerosis) and aneurysm, in that order (box). Neoplasm was found to be the most frequent cause of multiple cranial nerve paralysis, the most frequent pattern being involvement of cranial nerves III, IV and VI.⁷

In evaluating a patient with ptosis it is necessary to differentiate between a sympathetic lesion, oculomotor nerve damage or other cause. Although certain pupillary and other clinical signs are helpful in evaluation, arriving at a correct diagnosis may not always be easy. A diagnosis of 'ocular myasthenia' may be extremely difficult, even for experienced clinicians. There are no pathognomonic features on physical examination and diagnostic tests, including administration of anticholinesterase agents, repetitive nerve stimulation, and measurement of antibodies to acetylcholine receptor, may be inconclusive or misleading.⁸ Certain intracranial mass lesions may present with features suggestive of myasthenia gravis: alternatively, myasthenia gravis may mask the signs of coexisting intracranial mass.⁸

Neither the onset, completeness, presence or absence of pain or spontaneous recovery

Causes of ptosis
<ul style="list-style-type: none">● head trauma● neoplasm● vascular disorders● aneurysm● myasthenia gravis● ocular myopathies● Miller-Fisher syndrome● cranial arteritis● post-infectious neuropathy● demyelination● toxins● infections● dystrophia myotonica

seems to be a 'reliable' indicator of the aetiology or site of the lesion. It is often said that the so-called 'surgical lesions' are characterised by pupillary involvement and 'medical causes' of third nerve palsy do not involve the pupil. However, in about 20% of cases of diabetic third nerve palsy, the pupil is involved⁹ and these cases cannot be differentiated clinically from those caused by an aneurysm. Also, in third nerve palsy due to an intracavernous aneurysm, the pupil is often unaffected and may often be small from simultaneous involvement of the sympathetic innervation in the paracarotid plexus. Similarly, up to 42% of patients with diabetes mellitus presenting with ptosis and ophthalmoplegia have other conditions to explain their symptoms.⁹ A diagnosis of third nerve paralysis

of diabetic origin has been made in a patient who, in fact, had a rhabdomyosarcoma of the orbit.⁹ Up to 77% of patients with cranial nerve paralysis due to vascular causes recover, regardless of the nerve affected.⁷

In the light of this we feel that all cases presenting with ptosis and ophthalmoplegia should have routine CT or MRI scans.

Final diagnosis

Anaplastic carcinoma of ethmoidal sinus causing ophthalmoplegia and ptosis.

Keywords: ptosis, ophthalmoplegia, anaplastic carcinoma of ethmoid sinus

- 1 Robin PE, Powell JD, Stansbie JM. Carcinoma of the nasal cavity and paranasal sinuses. Incidence and presentation of different histological types. *Clin Otolaryngol* 1979; 4: 431–56.
- 2 Johnson LN, Krohel BG, Yeon EB, Parnes SM. Sinus tumours invading the orbit. *Ophthalmology* 1984; 91: 209–17.
- 3 Koh CS, Tan CT, Alhady SF. Cavernous sinus syndrome. A manifestation of non-Hodgkin lymphoma. *Med J Aust* 1989; 2: 451–2.
- 4 Colak A, Benly K, Donmez T, Onol B. Papillary carcinoma of sphenoid sinus associated with sphenoid abscess presenting as cavernous sinus syndrome. A case report. *J Clin Neurol* 1990; 10: 18–20.

- 5 Shibata M, Shimoda M, Sato O. A case of bilateral ophthalmoplegia caused by malignant lymphoma extending into the skull base (Japanese) *No Shinkei* 1992; 20: 717–21.
- 6 Yamamoto T, Imai T. Adenoid cystic carcinoma presenting as cavernous sinus syndrome (Japanese) *No to Shinkei* 1989; 41: 519–25.
- 7 Rush JA, Younge BR. Paralysis of cranial nerves III, IV and VI: cause and prognosis in 1,000 cases. *Arch Ophthalmol* 1981; 99: 76–9.
- 8 Moorthy G, Behrens MM, Drachman DB, et al. Ocular pseudomyasthenia or ocular myasthenia plus: a warning to clinicians. *Neurology* 1989; 39: 1150–4.
- 9 Zorrilla E, Kozak GP. Ophthalmoplegia in diabetes mellitus. *Ann Intern Med* 1967; 67: 968–76.

Hypernatraemia

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A 71-year-old woman was admitted with severe dehydration and an eight-day history of having 'taken to her bed'. In spite of free access to water, her liquid intake had been very poor and she denied extensive thirst at any point. On examination she was hypovolaemic with a supine blood pressure of 150/90 mmHg and sitting 90/60 mmHg. Her pulse was 100 and regular with a respiratory rate of 17 and she was orientated in person only. Her only medications on admission were metoprolol 50 mg bid and simvastatin.

Biochemical investigations showed the following: sodium 186 mmol/l, urea 16.2 mmol/l, creatinine 168 mmol/l, calcium (corrected) 2.52 mmol/l, glucose 4.3 mmol/l, plasma osmolality 414 mosm/kg and urine osmolality 851 mosm. Chest X-ray, electrocardiogram, liver and thyroid function tests, mid-stream urine, computed tomography (CT) of the head, clotting screens, B12, folate and VDRL, were all normal.

After appropriate restoration of fluid volume, full biochemical and clinical improvement occurred. She gave a history of fear of drinking lasting six years, since choking on a rushed gin and tonic while on holiday. She had no dysphagia or odynophagia and had normal awareness of thirst. She had recently been stressed by her grandson leaving for university. She denied ever feeling particularly thirsty in the days leading to her admission. She refused further investigations. On follow-up at clinic one month later she had gained weight, was drinking well, and had normal plasma electrolytes. She had been started on antidepressant treatment by her general practitioner.

Questions

- 1 By how much was she volume-depleted on presentation?
- 2 What are the main causes of hypernatraemia?
- 3 What is a possible underlying cause in this case?

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