Ptosis as the early manifestation of pituitary tumour

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Abstract

Three patients who developed unilateral ptosis followed by partial third nerve palsy were found to have a pituitary tumour. The visual field defects were minimal and asymptomatic. Two patients had a chromophobe adenoma and one patient had a prolactinoma. The importance of recognising a pituitary tumour as the cause of acquired unilateral ptosis is emphasised.

Loss of vision and visual field are the predominant ocular symptoms of pituitary tumour. In 1000 cases of pituitary tumours reported by Hollenhorst and Younge, 613 patients had visual symptoms; 421 of them had loss of vision as their presenting complaint. Pituitary adenomas cause disorders of eye movements even less commonly than they produce loss of vision and visual field, and those disorders usually occur late in the course of the disease.

We now report three cases which presented with the main complaint of unilateral ptosis due to pituitary tumour.

Case reports

CASE 1

A 30-year-old woman was admitted to the neurology section of the Veterans General Hospital on 10 April 1985 with the main complaint of ptosis of the right eye for 10 days. The ptosis had begun suddenly on 1 April. The course of the ptosis fluctuated, being better in the morning and worse with fatigue, and then improving. Because the Tensilon (edrophonium) test was negative she was sent for a neuro-ophthalmological consultation on 13 April 1985. Examination showed her visual acuity was OD 20/30—9·0 DS/-2·50 DC×180; OS 20/30—9·0 DS/-2·50 DC×180. She could read 15 out of 15 Ishihara colour plates with each eye. The pupils measured 3 mm in the right eye and 4 mm in the left eye, and reached normally to light. No afferent pupillary defect could be detected. The palpebral fissure was 6·5 mm on the right and 10 mm on the left (Fig 1). There was 25-prism dioptre exophoria on near fixation and 25 prism dioptre exotropia on far fixation. Mild limitation of the right medial rectus muscle was noted. Both fundi were normal. Goldmann visual fields showed bitemporal hemianopia on 12e target (Fig 2). A pituitary tumour with partial third nerve palsy was diagnosed. A routine x-ray of the skull was normal, but a CT scan of the brain showed an enlarged pituitary fossa with marginal enhancement, which was compatible with pituitary adenoma.

The patient underwent trans-sphenoid hypophysectomy on 7 May 1985. The visual fields became full and the oculomotor palsy disappeared one week after the operation (Fig 3). The tumour was a chromophobe adenoma, diffuse type.
CASE 2
A 62-year-old man presented on 12 April 1985 with the main complaint of ptosis of the right eye for a few days. An ophthalmic examination showed visual acuity with the best correction was OD 20/25 and OS 20/20. He could read 15 out of 15 of Ishihara colour plates with each eye. The palpebral fissure was OD 7 mm and OS 10 mm (Fig 4). On both sides the pupil size was 3 mm, with normal light reactions, and the extraocular movements were normal. The edrophonium test was negative. The Goldmann visual fields showed bitemporal upper sector defects on I2e target (Fig 5). A pituitary tumour was diagnosed. A partial right third nerve palsy then developed progressively while the patient was waiting for a CT scan. The scan of the brain showed a lobulated sellar mass with suprasellar extension, more on the right side. The patient underwent trans-sphenoid hypophysectomy. The tumour was a chromophobe adenoma. The oculomotor palsy and visual field defects were found on postoperative follow-up to be cured.

CASE 3
A 61-year-old man came on 23 February 1988 with the main complaint of sudden onset of ptosis of the right eye on 9 February. Senile cataract in both eyes had been diagnosed several months previously. His vision with best correction was 20/70 in each eye. He could read 15 out of 15 of Ishihara colour plates with each eye. The palpebral fissure was OD 3-5 mm and OS 8 mm (Fig 6). The pupil size was OD 4 mm and OS 3 mm, with normal direct and indirect light reactions. No relative afferent pupillary defect could be detected. There was limitation of the medial rectus of the right eye. The lenses were moderately opaque. The fundi were normal. Goldmann visual fields with small target examination showed temporal upper sector depression on the right and a normal result on the left (Fig 7). A CT scan of the brain revealed a sellar mass with right suprasellar extension. A hormone study revealed prolactin 987 µg/l (normal <22). The patient underwent craniotomy. Histologically the tumour was found to be a prolactinoma. Postoperative follow-up showed the oculomotor palsy was improving. In July 1988 the patient suffered from headache and bitemporal hemianopia, when a CT scan of the brain showed a recurrent tumour. But this time no oculomotor abnormalities were found.

Discussion
Pituitary tumours may produce a variety of symptoms, depending on their direction of...
growth. As they increase in size the first structure they compress is the chiasm. An ocular nerve palsy resulting from encroachment on the cavernous sinus is less common but has been reported in 1–6% of patients with pituitary tumours.

Although oculomotor palsy may occur in isolation, they generally develop as the end stage of a pituitary tumour, and are combined with loss of vision or visual field.

With lateral extension of the tumour there is involvement of the oculomotor, trochlear, and abducens cranial nerves because of their proximate location in the cavernous sinus. The oculomotor nerve is most frequently involved. Occasionally the first and second divisions of the trigeminal nerve may be affected.

Robert et al. noted that in cases of ocular palsy occurring with pituitary tumours, of all the muscles supplied by the third nerve the levator palpebrae superioris was the most commonly affected, as shown by partial or complete ptosis.

The oculomotor nerves arise from a group of nuclei at the level of the superior colliculus. Crossed and mainly uncrossed fibres course through the red nucleus and the inner side of the substantia nigra to merge with the sella turcica in the outer wall of the cavernous sinus and, through the superior orbital fissure, to supply the medial, superior, and inferior rectus muscles and the inferior oblique and levator palpebrae muscles.

The topographic arrangement of the fibres within the oculomotor nerve in the cavernous sinus is unclear. Our patients developed ptosis in the early stages of oculomotor palsy, which suggested an effect on the fibres going to the levator palpebrae muscle located superficially in the medial portion of the nerve within the cavernous sinus.

Several mechanisms have been proposed as causing oculomotor palsy. The tumour may transmit pressure to the wall of the cavernous sinus. Or it may break through the wall of the cavernous sinus and directly compress the nerve or its blood supply. Because the trans-sphenoidal approach was used, it was impossible to tell if the cavernous sinus was compressed or invaded.

Most of the tumours causing ocular palsy are chromophobe adenomas. This tumour is almost never diagnosed before it has grown large enough to compress the anterior visual pathways, causing field defects.

Prolactinoma is not normally considered as a cause of third nerve palsy. Most patients with prolactinoma do not have neurological or visual symptoms or signs, because the tumour is still relatively small when it is discovered. A review of the literature revealed one case of prolactinoma presenting with intermittent isolated third nerve palsy and one presenting with fifth and sixth cranial nerve palsy. Our third patient presented with partial third nerve palsy and a mild field defect in one eye, which is also unusual.

All three of our patients sought medical advice for ptosis. The cases are a reminder that patients with acquired ptosis need detailed neuro-ophthalmological examination.
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