

Short report

Transient visual obscurations, without papilloedema

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SUMMARY Two cases of frontal, space occupying tumour without papilloedema are reported. Both presented with frequent, stereotyped attacks of visual disturbance with orbital headache, neck pain and unsteadiness of gait. Intermittent occipital lobe ischaemia, related to compression of the posterior cerebral artery against the tentorium by distorted, herniating brain, seems a probable explanation.

Transient visual obscurations, usually lasting seconds, are a well known accompaniment of raised intracranial pressure and established papilloedema. Ethelberg and Jensen¹ suggested that these episodes reflect intermittent occipital lobe ischaemia as a result of posterior cerebral artery compression against the tentorial margin occurring as a distant effect of a space occupying lesion. Walsh and Hoyt,² however, noting the occurrence of uniocular attacks in many cases felt that ischaemia resulting from transient pressure changes at the nerve head, as proposed by Cogan,³ could not be excluded. Transient visual disturbance as a presenting feature of intracranial space occupying lesions without papilloedema or direct involvement of visual pathways appears to be very rare.

Case reports

Case 1 (NHQS B18631)

A 54-year-old man was admitted to the National Hospital, Queen Square in May 1981 under the care of Dr JN Blau with a five month history of episodes of transient loss of vision accompanied by headache. Each episode began with stiffness in the neck followed by pain radiating over the head to the forehead and temples. Within seconds this

would be followed by an ache and total loss of vision in both eyes. Pain and visual loss would persist for five to 30 minutes (average 20 minutes). Over a few seconds the headache would resolve and vision return. During the attacks he felt unsteady on his feet. He had no other symptoms with the attacks and between episodes he was symptom-free. The episodes had been occurring five to eight times daily with no days free of attacks. He had not previously suffered from headaches and there was no family history of migraine. Prior to presentation at this hospital, a diagnosis of migraine had been entertained and he had taken aspirin, diazepam, propranolol and clonidine without benefit. One month before admission, assessment at another hospital had shown normal visual fields and optic discs. Examination on admission revealed normal fundi. The visual acuity on the right was 6/9 and on the left 6/12 corrected with a pin hole. There was a homonymous right upper quadrantanopia showing mild incongruity. Examination including sense of smell was otherwise normal. His attacks continued. Seven days after admission he complained of poor vision in his left eye. Repeated examination showed diminished sense of smell on the left. Acuity on the right was unchanged, but on the left had fallen to counting fingers. The quadrantanopia persisted, but he now had a left central scotoma in addition. The left disc now appeared pale and the right showed mild papilloedema. There was mild weakness of right hip flexion. On writing he demonstrated mild dysgraphic errors. A CT scan demonstrated a cystic tumour in the left frontal region extending back to the anterior temporal region. The left lateral ventricle was compressed, with midline shift to the right. He was commenced on steroids and during the next three days prior to operation, no further attacks of visual loss occurred. At operation Mr D Thomas found a cystic gemistocytic astrocytoma and performed a left frontal lobectomy. The patient made a good recovery and in the three weeks prior to discharge, he had no further episodes of visual loss. Visual acuity in the left eye had not improved, but there was some diminution in the quadrantanopia.

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*Transient visual obscurations, without papilloedema***Case 2 (United Oxford Hospitals 596211)**

A 38-year-old man was admitted to the Churchill Hospital in May 1974 under the care of Dr JMK Spalding. Six months prior to admission he was woken from sleep by pain in the left eye. Vision was not disturbed. The pain persisted for 36 hours and gradually subsided over three days. He then began to suffer repeated episodes of a tight sensation between his shoulder blades which radiated as pain up the back of his neck and around the sides of his head to a position across both eyes with an associated tender patch in the left temporal area. During most of these attacks, vision in the right half field was disturbed by "a shimmering appearance". The attacks were accompanied by a sensation of weakness in the legs which on several occasions caused him to trip. They lasted approximately 15 minutes and occurred up to six times daily with a maximum of two days freedom between attacks. There were no symptoms between attacks. Neurological examination was normal. After admission he had a further attack during which his visual fields were full to confrontation, but there was undoubted neck stiffness and ataxia of gait. An isotope brain scan showed an area of increased uptake in the left frontal region. Carotid angiography revealed a large left frontal mass with marked midline shift to the right and an early filling vein. He was commenced on dexamethasone and in the eight days leading up to surgery, the frequency of his attacks was considerably reduced. At operation Mr CBT Adams removed a left frontal convexity meningioma. Since operation he has had occasional major fits, but no further episodes of pain or visual disturbance.

Discussion

Both cases demonstrated recurrent transient visual disturbance either as the presenting feature or as one of the presenting symptoms of a frontal tumour in the absence of papilloedema. No relation to exertion, change in posture or change in temperature was apparent. Although it is said that headaches of migrainous nature may occasionally occur in association with progressive intracranial lesions, we think the nature and frequency of attacks in our two cases clearly distinguish them from classical migraine. Fisher⁴ reported a patient of 62 years who had episodes of blindness of similar duration occurring up to three times daily for two weeks which were attributed to late onset migraine. The patient vaguely recalled a similar episode one year earlier and an episode of darkening of vision seven years before the presenting attack. We have been unable to find any other reports of cases of recurrent visual obscuration of such frequency and length of history in the absence of papilloedema. The interpretation of events in case 1 is complicated by the development of the classical Foster Kennedy syndrome. Furthermore although the homonymous field defect was in keeping with the CT scan evidence of involvement of the anterior portion of the left temporal lobe, left optic tract involvement cannot be excluded. How-

ever, against this is the fact that the visual fields were recorded as normal one month prior to admission. No evidence of a persistent left temporal field defect was found to suggest involvement of the chiasm itself. Intermittent chiasmal compression by a cystic craniopharyngioma was reported by Frisen *et al*⁵ but the frequency and duration of attacks and lack of accompanying symptoms were quite unlike our cases. Furthermore, Ethelberg and Jensen¹ who found 140 cases with visual obscurations in reviewing 500 cases of intracranial space occupying lesion, noted the absence of visual obscurations in all of their cases of pituitary adenoma and suprasellar meningioma. In our case 1 the long history of visual obscurations prior to admission appears quite distinct from the rapid, progressive left visual failure following admission and we think it unlikely that the initial symptoms represent a direct effect of the tumour.

In case 2 the visual symptom consisted of a homonymous hemianopic shimmering disturbance contralateral to the side of the tumour and the side of the original frontal and orbital headache. Six of Ethelberg and Jensen's¹ cases described hemianopic obscuration. It is difficult to account for this in terms of anterior visual pathway disturbance as opposed to occipital lobe ischaemia. It is interesting to note that Knox and Cogan⁶ found occipital lobe thrombosis to be accompanied by ipsilateral orbito-frontal headache. Neck rigidity was a relatively common sign in Gassel's⁷ review of false localising signs in a large series of cases of supratentorial meningioma. Although he found only one case of homonymous hemianopia occurring as a false localising sign, occipital lobe infarction is well recognised as a terminal event in the coning brain.⁸ The occurrence of neck pain and stiffness, unsteadiness of gait and orbito-frontal headache at the onset of attacks of visual disturbance in our patients, appears to us to fit well with Ethelberg and Jensen's¹ suggestion of uncocal herniation and compression of the brainstem and posterior cerebral arteries against the margin of the tentorial hiatus. The duration of attacks in our cases was unusually long for visual obscurations which rarely lasted for more than 30 seconds in Ethelberg and Jensen's¹ series. They found only two patients with attacks of comparable duration and only one of their cases presented with visual disturbance as the initial symptom. During their attacks both our patients retained normal consciousness which would be against a generalised disturbance of cerebral blood flow. The response to steroids and surgery would seem to exclude occipital emboli as the explanation. Although it may seem unlikely that our patients had tentorial herniation for several months in view of their lack of signs and symptoms

between attacks, we propose that for most of this time there was inadequate distortion of normal structures to produce symptoms but that periodic increases in intracranial pressure were sufficient to disturb the state of equilibrium. Jefferson⁹ commented on the intimate relationship between the posterior cerebral arteries and the sharp edge of the tentorium and expressed surprise that clinical sequelae were not a more common result. He also noted that the tentorial pressure cone can exist in the presence of only minimal papilloedema. Lindenberg¹⁰ pointed out that the calcarine area is supplied by a separate branch of the posterior cerebral artery and that this may be compressed in isolation from the other branches. The occurrence of episodes of apparently spontaneous increase in intracranial pressure in patients with a variety of pathological processes was described by Lundberg.¹¹ He found a close correlation between such plateau waves, which may occur in the absence of papilloedema,¹² and several transient neurological symptoms. Increased cerebral blood volume has been recorded during plateau waves¹³ and it seems inevitable that such periodic increases in cerebral blood volume would tend to increase the amount of distortion in patients with intracranial space occupying lesions if cerebral tissue were not free to expand equally in all directions. The occurrence of associated symptoms would then depend not only on the amount, but also on the direction of displacement, normal variation in the size of the tentorial hiatus relative to its contents and factors such as systemic blood pressure and the amount of any local collateral circulation. Differences in these factors might account for the apparent rarity of the combination of symptoms and signs in the present cases.

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