Short report

Afferent pupillary defect in pineal region tumour

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SUMMARY A patient is described in whom an afferent pupillary defect was an early sign of a tumour in the pineal region. It is suggested that this was due to involvement of the pupillary afferent fibres between the optic tract and pretectal nucleus contralateral to the affected pupil.

Tumours in the posterior third ventricle region frequently result in pupillary abnormalities due to involvement of the central pupillary pathways in the rostral midbrain.¹⁻³ The pupils may be unequal, dilated and commonly show light-near dissociation on clinical testing. Pupillographic studies⁴ have indicated that the near reaction may not be normal and show some impairment of reflex amplitude. Patients with a mid-brain lesion have been reported to show a greater amplitude of the direct than the consensual light reflex (alternating contraction anisocoria) and this has been taken as indicating a lesion in the region of the posterior commissure⁵ although recent studies suggest that alternating contraction anisocoria may be a physiological phenomenon.⁶ One patient with a third ventricle tumour has been described⁷ as showing irregular, eccentric pupils (corectopia) with abnormal light reactions and this finding has been reported in other cases with mid-brain pathology.⁸

A patient is described in whom an afferent pupillary defect was an early sign of a posterior third ventricle tumour. A possible explanation for this observation is given.

Method

The pupillary reactions were studied on a Whittaker Series 1800 Binocular infra red television pupillometer after 30 minutes dark adaptation. 100 ms white light stimuli focused to a 2 mm beam were delivered every 8 seconds at an angle of 17° temporal to the visual axis with the patient fixating a distant target. The visual threshold was first determined with this apparatus and the stimulus intensity employed for pupillography was 7 log units above threshold.

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Case history

The patient KS a male aged 19 years (National Hospital No. B14550) had a previous history of convulsive seizures between the ages of 4 and 6½ years but was well until February, 1977 when over two days he noticed tingling and heaviness of the left face, arm and leg with double vision. Two weeks after the onset neurological assessment revealed no abnormality on examination and skull radiographs and isotope brain scan were negative. After three months he had made a complete symptomatic recovery and remained well for two years when he again developed heaviness and numbness in the left arm and leg which spread to the left face and the left side of the tongue. He was admitted to another hospital where neurological assessment showed normal visual acuity, fields and optic disc appearances. The left pupil was 2 mm larger than the right, and the light reactions were said to be normal. There was mild left limb ataxia and subjective diminution of light touch appreciation over the left half of the body. Other sensory modalities were normal. Investigations showed negative WR, and normal EEG, CT scan and CSF. VERs showed no definite abnormality. A provisional diagnosis of multiple sclerosis was made and he was discharged to follow-up, remaining asymptomatic until September 1980 when he noticed, on waking, left sided numbness. Touching the left side produced tingling. These symptoms progressed over four days and then remained static. He noticed transient double vision with horizontal image separation and a mild non specific frontal headache. He was admitted to the National Hospital in October 1980 aged 22 years. Examination showed normal visual acuity, fields and disc appearances. The left pupil at 5 mm was 1 mm larger than the right in room light and there was a left relative afferent pupillary defect. There was subjective decrease to pin prick and light touch sensation in the left trigeminal area with normal corneal reflexes. The motor system was normal and sensory testing showed left hemisensory impairment to pin prick and light touch. There was mild left limb ataxia.

Investigations showed normal CSF with no oligoclonal bands. Visual evoked responses were normal including full field and half field responses to standard check size. Skull radiographs were normal and CT scan showed a high

density lesion in the region of the right thalamus and pineal region (fig 1). Carotid and vertebral angiography were normal. Pupillometry showed no significant anisocoria in darkness, but on left eye stimulation there was impairment of the direct response in the left eye and the consensual response in the right eye. Right eye stimulation showed normal reflex amplitudes (fig 2). The near reaction was normal. It was thought that the most likely diagnosis was a thalamic glioma.

Discussion

The afferent pupillary defect is predominantly a feature of lesions in the ipsilateral retina or optic nerve.⁹ An afferent pupillary defect without other evidence of visual pathway involvement has not been described previously in lesions of the pineal region. In the reported case no impairment of visual acuity, colour vision, visual field or optic disc change was present and the visual evoked response to pattern reversal was normal. An optic nerve lesion was therefore unlikely and there are no reports of afferent pupillary defects ascribed to optic nerve disease occurring in patients without some additional clinical evidence of optic nerve pathology or abnormality of the VER.

Afferent pupillary defects have been described in lesions of the optic tract¹⁰ and explained on the basis of the asymmetrical distribution of receptors in the nasal and temporal retina. The nasal retina has the more dense population of photoreceptors¹¹ and axons of the retinal ganglion cells of the nasal retina decussate in the optic chiasm joining the fibres from the temporal retina of the other eye. Thus an optic tract lesion will affect more pupillary afferent fibres from the contralateral eye which will have the temporal visual field defect. A relative afferent pupillary

Fig 1 Enhanced computed tomographic scan showing a high density lesion in the right pineal region.

Fig 2 Pupillograph showing light reflexes elicited by the same stimulus intensity in the left (A) and right (B) eyes. The light reflexes from each eye are indicated (L = left, R = right). The amplitude of the direct and consensual light reflexes is greater on right than left eye stimulation.

defect may be seen in this eye. Asymmetrical decussation of fibres arising from nasal and temporal retina has been reported to occur in the optic chiasm (ratio crossed:uncrossed, 53:47).¹² If such an imbalance occurs in the anterior optic tract containing both the visual and pupillary fibres, this imbalance will also be present after the pupillary afferents leave the optic tract and reach the pretectal nucleus by way of the superior brachium.

It is suggested that in this case the lesion affected the pupillary afferent fibres between the optic tract and the pretectal nucleus giving rise to an afferent pupillary defect without the homonymous hemianopia of optic tract involvement. The tomographic scan indicated the lesion to be in the appropriate area on the side contralateral to the affected pupil. The anisocoria suggests additional central or efferent pupillary pathway involvement but could not alone explain the presence of an afferent pupillary defect which when it occurs due to anisocoria is seen in the eye with the smaller pupil.

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