Ocular flutter in suspected multiple sclerosis: a presenting paroxysmal manifestation

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Summary: A patient with suspected multiple sclerosis is described who presented with attacks of blurring of vision and ocular flutter. This has not previously been reported as an isolated paroxysmal manifestation of brain stem demyelination. As with other paroxysmal disturbances ocular flutter may present as the first sign of the disease.

Introduction

Paroxysmal neurological disturbances are known to occur in the early stages of multiple sclerosis (MS), often as the initial manifestation (Twomey & Espir, 1980). The most frequently cited examples are tonic seizures, paroxysmal dysarthria and ataxia, paroxysmal paraesthesia, trigeminal neuralgia, and paroxysmal akinesia (Espir *et al.*, 1966; Matthews, 1975). They are distinguished by their sudden onset, brief repetitive nature and localization to the brain stem. Ocular flutter exhibits the same characteristics as these accepted paroxysmal manifestations. The attacks are recurrent, of high frequency, short duration and of brain stem origin (Goldberg & Jampel, 1963). Our criteria for defining ocular flutter, which is rapid oscillatory eye movements causing oscillopsia, conforms to that of Cogan (1954) and is distinct from other oscillatory phenomena such as ocular motor dysmetria, opsoclonus, fixation nystagmus and periodic alternating nystagmus (Goldberg & Jampel, 1963).

The occurrence of ocular flutter in established multiple sclerosis is documented (Gresty *et al.*, 1980) though rare, and has not been described as a presenting symptom nor as an isolated sign in this condition. This paper suggests that ocular flutter is another paroxysmal event which may present as the initial manifestation of MS.

Case history

A previously healthy female, aged 23, developed short-lived episodes of visual blurring caused by recurrent attacks of horizontal ocular flutter. Each oscillatory saccade lasted for a maximum of 2 s but clustered into short repetitive bursts causing great distress to the patient. The frequency of these attacks increased over 72 h until they were occurring every few minutes. They were particularly numerous at the end of the day, with as many as 40 attacks occurring within an hour.

On the second day of her illness she became acutely ataxic for several minutes without any visual symptoms. She described incoordination affecting all her limbs with an unsteady gait.

Physical examination was normal. Individual and conjugate eye movements were full and there was no gaze-evoked nystagmus. Every few minutes however, a series of involuntary bursts of pendular, horizontal oscillations of the eyes were seen, associated with subjective blurring of vision. Between these attacks her visual acuity was 6/6 bilaterally and the pupillary responses were normal. There were no additional abnormal neurological signs.

The attacks were controlled in 48 h of commencing treatment with carbamazepine (600 mg/d) and the frequency was reduced to one or two attacks an hour. When carbamazepine was withdrawn they reverted to their former frequency after 2 d. Re-introduction of carbamazepine with daily injections of ACTH rapidly diminished the number of attacks, which ceased within three weeks. Treatment was then stopped and there has been no recurrence of her ocular flutter.

Investigations

Standard haematological and biochemical investigations were normal and sequential serum and cerebro-

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Accepted: 21 June 1984

spinal fluid viral titres were negative. Electroencephalogram and computed tomographic brain scan were normal. Visual evoked potentials, from flash and pattern reversal monocular stimuli, were of normal amplitude and latency. There was no abnormality detected in colour vision (Ishihara plates and the 100 Hue Test). Bilateral arcuate scotomata were consistently documented on tangent screen perimetry, similar to those previously reported in MS (Patterson & Heron, 1980).

Brain stem auditory evoked potentials, using bipolar click stimuli monaurally, showed well developed responses in the left ear. In the right ear however, delayed latencies and a reduction in amplitude were seen in the intermediate wave components compatible with a localized abnormality in brain stem function.

Cerebrospinal fluid pressure was normal, there were 7 lymphocytes per mm³ and the protein content was 15 mg/dl. IgG synthesis was increased at 18.9 mg/day (normal laboratory range < 10 mg/day) and IgG concentration raised at 6.9 mg/dl (normal < 5 mg/dl). The IgG/albumin ratio was 1:3; these findings are consistent with demyelination (Tourtellotte & Booc, 1978).

Discussion

Paroxysmal neurological disturbances may be associated with diseases affecting the brain stem other than multiple sclerosis, such as encephalitic, vascular, neoplastic and inflammatory lesions. These were not considered relevant in our patient in whom a diagnosis was made of suspected or early probable/latent MS (McDonald & Halliday, 1977).

The classification of early probable or latent multiple sclerosis, rather than that of suspected MS can be

accepted if the arcuate scotomata reported are taken as evidence of subclinical involvement of the optic nerves (Patterson & Heron, 1980). The firm diagnosis of multiple sclerosis can only be established when there is evidence of demyelination occurring at other sites in the central nervous system. This is recognized in paroxysmal events when they are the presenting feature of multiple sclerosis; reported remissions, before other manifestations of MS develop, range from less than one year to 21 years (Twomey & Espir, 1980). The character of the brain stem paroxysmal attacks in a young woman, favourable response to carbamazepine (Espir & Millac, 1970), and the isolated short episode of ataxia are evidence that the underlying pathology is demyelinating in this case. The abnormal brain stem evoked responses (Chiappa *et al.*, 1980), and the increased cerebrospinal fluid immunoglobulin are also significantly supportive to the diagnosis of demyelination in our patient.

Paroxysmal events in multiple sclerosis are relevant to our understanding of the pathophysiology of developing and potentially reversible conduction defects in demyelination. Intermittent conduction block (Espir *et al.*, 1966), or transversely spreading ephaptic activation of axons (Osterman & Westerberge, 1975), in partially demyelinated lesions within the brain stem are mechanisms invoked to explain these phenomena. Both ocular flutter and transient ataxia may result from a lesion in the region of the cerebellar peduncles (Goldberg & Jampel, 1963; Osterman & Westerberge, 1975). Cross-excitation between adjacent tracts within a developing brain stem plaque is acceptable as an explanation of the symptoms and signs seen in our patient and the noted delay in brain stem transmission.

We believe that the episodes of ocular flutter in our patient represent a further example of paroxysmal events occurring as the presenting manifestation of latent multiple sclerosis.

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