This one minute test, which does not require instruments and was performed in isolation by non-medical staff, detected 74% of cases of iritis out of 426 patients with miscellaneous eye complaints. It should enable doctors to detect and refer patients with iritis earlier and so prevent potentially blinding complications such as cataract and glaucoma.

Finger to nose convergence test produces synkinetic triple response; convergence of eyes confirms that this has occurred. If iritis is present pain is exacerbated, at reproducible distance.

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1 Hitchings RA. The symptom of ocular pain. *Trans Ophthalmol Soc UK* 1980;100:257-9.

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Idiopathic third cranial nerve palsy associated with herpes simplex virus infection

Occasionally a healthy individual develops a third cranial nerve palsy and the cause remains obscure despite evaluation for cerebral aneurysm, an intracranial mass, and diabetes mellitus. We describe here two such patients in whom there was strong circumstantial evidence of an infection with herpes simplex virus type 1.

Case 1

A 51 year old Japanese housewife had a 10 year history of episodic right sided headache followed on each occasion by an eruption of vesicles on the right upper face. Episodes occurred two or three times a year and led to mild scarring on the face. She was referred with a right sided third cranial nerve palsy, which developed a few days after such a headache; the headache was typical except for the absence of skin lesions. Examination showed nothing abnormal except limitation of adduction, elevation, and depression of the right eye and pupillary asymmetry, with the right pupil larger and with impaired right pupillary responses. A glucose tolerance test, computed tomography of the head, cerebral angiography, and spinal fluid examination yielded normal results. Serum neutralising antibody titre to herpes simplex virus type 1 was 1/32, and IgG antibody to herpes simplex virus type 1 was detectable in the spinal fluid by enzyme linked immunosorbent assay (ELISA). The eye movement disorder gradually improved, but one and a half months after onset of the third nerve palsy vesicles developed by the right ear. Virus isolated from these vesicles was shown to be herpes simplex virus type 1 by restriction enzyme analysis.

Case 2

A 40 year old white man developed left sided retro-orbital pain followed in a few days by diplopia. Examination showed limitation of adduction, elevation, and depression of the left eye and a dilated left pupil. He had a 30 year history of herpes labialis occurring once or twice a year, but no skin lesions developed at the time of the third nerve palsy. Again, a glucose tolerance test, computed tomography of the head, cerebral angiography, and spinal fluid examination yielded normal results. The serum antiherpes simplex virus type 1 neutralising antibody titre rose significantly from 1/1280 two and a half weeks after the onset of the nerve palsy to 1/10 240 six and a half weeks after onset. Spinal fluid antibody was undetectable at six and a half weeks but present twelve and a half weeks after onset (titre 1/10). The eye movement disorder gradually improved over the next four months.

Comment

Herpes simplex virus type 1 causes recurrent epithelial eruptions as a result of reactivation of a latent infection in a sensory ganglion. Less well understood is the pathogenesis of central nervous system infections. Encephalitis caused by herpes simplex virus type 1 is usually focal in the temporal lobes and in two thirds of the victims results not from a primary infection but from reinfection or reactivation of a latent infection.¹ Herpes simplex virus type 1 deoxyribonucleic acid has been shown in the brain stem during latency,² and there has been speculation about the virus in immune mediated central nervous system diseases.

Varicella zoster virus occasionally causes cranial mononeuropathies or segmental motor paresis, but similar observations have not been made for herpes simplex virus type 1 except for some reports attempting to implicate herpes simplex virus type 1 in Bell's palsy.³ In experimental animals a focal herpes simplex virus type 1 encephalitis has been established in a cranial nerve motor nucleus (the 12th nerve), and latency in these motor neurones has been reactivated experimentally.^{4,5} It is conceivable that a similar type of infection may occur in man and that reactivation may lead to cranial mononeuropathies without other brain stem symptoms or signs. Alternatively, reactivated virus in ganglia may atypically spread to the central nervous system, as has been shown in mice.⁵ The present clinical cases lend support to these speculations and encourage further efforts to implicate infection with herpes simplex virus type 1 in cranial mononeuropathies.

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Correction

Very low birthweight survivors: illness and rehospitalisation in the first fifteen months of life

We regret that the authors' names were printed in the wrong order in this short report (5 September, p 579). Dr H Skeoch from the Social, Paediatric, and Obstetric Research Unit, University of Glasgow, should have appeared as the first author.