The treatment is to discontinue the consumption of the adulterated mustard oil and give supportive therapy with diuretics, vitamins, anti-histaminics and anti-prostaglandin agents. The inadvertent use of systemic steroids in this instance probably contributed to the CSR-like lesions. Systemic steroids are not routinely given to control the acute phase of epidemic dropsy. This report highlights the potential problems of using oral steroids in these patients. It is also essential to screen other non-symptomatic members (who consumed the oil) for glaucoma and to continue IOP monitoring over a period of 2 months for the exposed population.

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Sir,

One-and-a-half syndrome: a different type

In 1967, Fisher[1] first described two patients with clinical disorder of extraocular movements characterised by a conjugate gaze paresis on attempted gaze to the lesion side and impaired adduction of the ipsilateral eye (an internuclear ophthalmoplegia) on attempted gaze to the contralateral side (one-and-a-half syndrome). Since then, similar cases have been reported in the literature and have confirmed the specificity of this syndrome.[2–6] We describe here a different type of one-and-a-half syndrome in a 61-year-old man in whom the preserved eye movement was adduction.

Case report

A 61-year-old man was admitted to the hospital because of gradual progression of marked weakness and hypoesthaesia of the right side. The patient was well until 2 days earlier, when he experienced the onset of weakness and hypoesthaesia of the right side that made it impossible for him to walk. Cranial computed tomography (CT) findings were normal. Two days after the scan he became right hemiplegic, and he complained of diplopia. There was a 6-year history of hypertension, which was managed with enalapril maleate (10 mg/day).

On physical examination, blood pressure was 180/120 mmHg, heart rate was 84/min and his body temperature was 36.5 °C. General physical examination was normal. On neurological examination the patient was fully oriented and his speech was fluent. There was 3 mm of ptosis of the left eyelid. Fundus examination found no papilloedema bilaterally. His pupils were anisocoric (right: 2.5 mm, left: 4 mm) and pupillary reflex was absent on the left side. Decreased corneal reflex on the right side was established. Horizontal gaze to the right showed a total conjugate gaze paresis. On left gaze there was paresis of abduction of the left eye, although the right eye did adduct with gaze-evoked horizontal jerk nystagmus (Fig. 1). Vertical eye movements and vestibulo-ocular responses were intact in right eye and his other cranial nerve functions were normal. Visual fields demonstrated a right homonymous hemianopia.

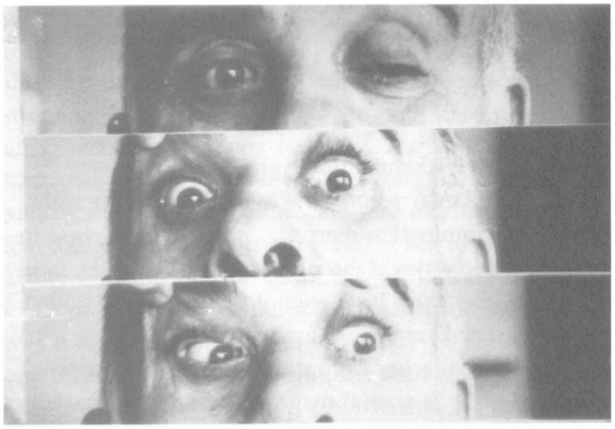
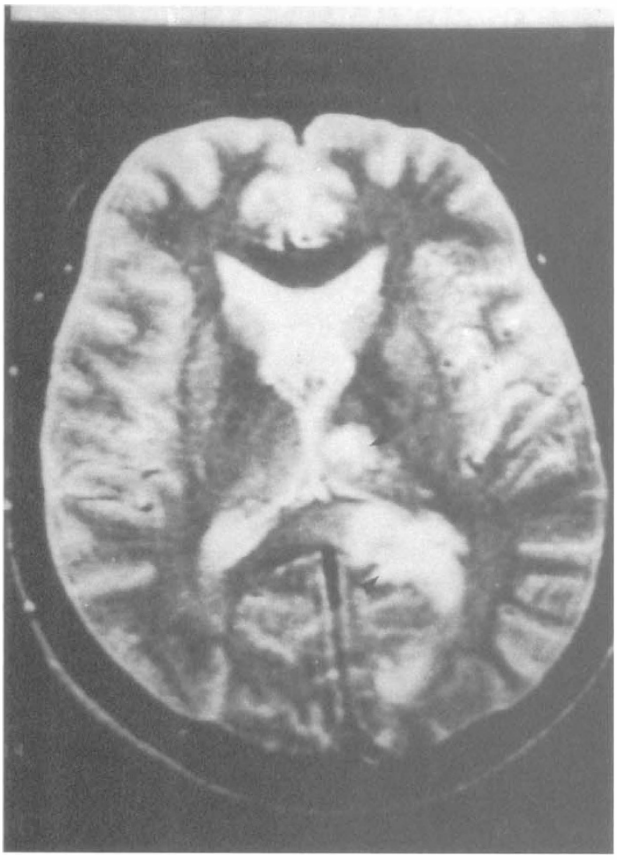
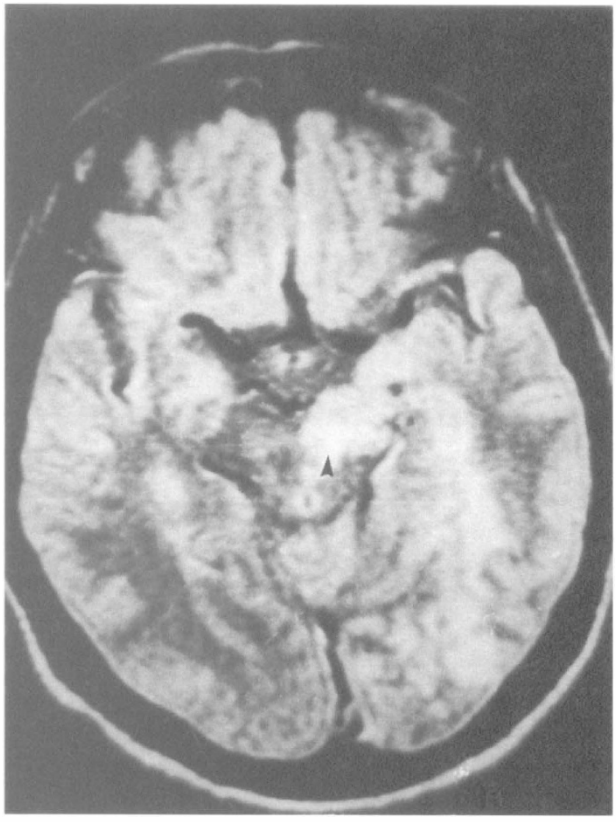


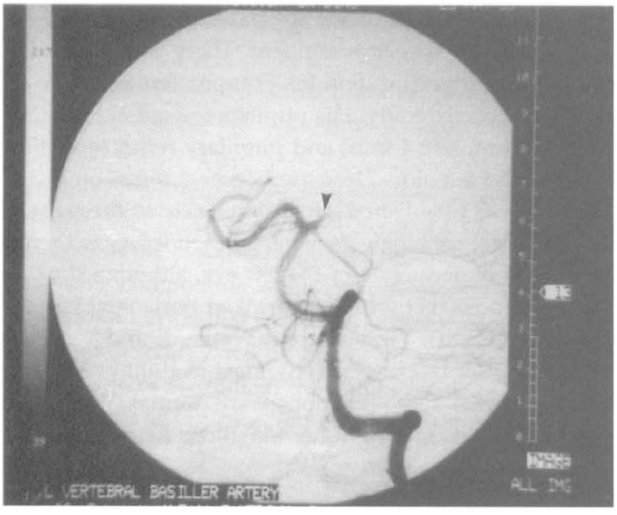
Fig. 1. *Top: Note marked ptosis of the left eye in the primary position of gaze. Centre: Horizontal conjugate gaze palsy looking right. Bottom: On left gaze there was paresis of abduction of the left eye, although the right eye did adduct.*



(a)



(b)



(c)

Fig. 2. *(a) Axial T2-weighted (TR/TE: 2200/90) and (b) proton density (TR/TE: 2200/35) MR images showing a large hyperintense lesion from ventral pons to thalamus, and parieto-occipital white matter on the left (arrowheads). (c) Digital subtraction angiography showing proximal left posterior cerebral artery occlusion (arrowhead).*

On motor power examination right hemiplegia was present. The plantar response was extensor at the right. On sensory examination there was a marked decrease in all sensory modalities at the right side including the face.

Routine laboratory tests were normal. Carotid and vertebro-basilar Doppler sonograms and a transoesophageal echocardiogram did not show any embolic sources. A cranial magnetic resonance imaging (MRI) scan revealed a large hyperintense lesion from ventral pons to thalamus, and parieto-occipital white matter on the left (Fig. 2a,b). A digital subtraction angiography (DSA) scan showed proximal left posterior cerebral artery occlusion (Fig. 2c). The patient was started on antiplatelet therapy. Within weeks the patient improved minimally.

Comment

This is perhaps the second reported case of conjugate gaze paresis in which the preserved eye movement was adduction (a different type of one-and-a-half syndrome, as opposed to the abduction described in the original report). To our knowledge, only one similar case has been reported in the English literature (in 1994 by Carter and Rauch[7]), but they attributed the conjugate gaze

paresis to mucormycosis infection of the sphenoid sinus that involved the cavernous sinus and resulted in occlusion of the intracavernous internal carotid artery. In contrast, we attributed the conjugate gaze paresis to posterior cerebral artery occlusion that was confirmed angiographically.

We consider that the mechanism of ocular movement disorder in our case may be explained as follows. The conjugate gaze paralysis in the contralateral direction is attributable to the left fronto-pontine pathway lesion at the midbrain level on the left, but the right fronto-pontine pathway from the right frontal eye field to the left pontine paramedian reticular formation is intact, and therefore conjugate horizontal gaze to the left is possible.[8] Total ophthalmoplegia, dilated pupil and ptosis of the eyelid in the left eye are attributable to an ipsilateral third and sixth nerve palsies due to midbrain and ventral pontine lesions. Thus, only adduction of the contralateral eye was preserved. The additional findings, which include hemiplegia and hemihypoesthaesia of contralateral side, are attributable to involvement of the left corticospinal and right spinothalamic tracts in the ipsilateral brain stem and thalamus.[9] Also, because of the left thalamic involvement, all sensory modalities markedly decreased on the right side of the body. The sensory symptoms in the patient's face and decreased corneal reflex suggested that the trigeminothalamic tract was becoming involved.

All these signs in our patient may be explained by posterior cerebral artery occlusion. The paired posterior cerebral arteries supply the occipital cerebral cortex, medial temporal lobes, thalamus and rostral midbrain. Also, extension of the infarction area to the pons in the patient's MRI scans additionally reflects occlusion of the pontine branches of the basilary artery.[9] Since these branches are quite small-calibre arteries, they were not visualised at DSA.

We report this case to emphasise an unusual case of the one-and-a-half syndrome in a patient in whom the only preserved eye movement was adduction.

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Sir,

Late clouding of an acrylic intraocular lens following routine phacoemulsification

Acrylic intraocular lenses (IOLs) are becoming increasingly popular in phacoemulsification surgery. There are different acrylic IOLs and variations in the side-chain components of the acrylate/methacrylate polymer backbone give the lenses different physical and biological properties.[1,2] The advantages of acrylic IOLs include a lower inflammatory response,[3] lower risk of pigmented precipitates[4] and a lower rate of posterior capsule opacification[5] compared with either standard polymethylmethacrylate or silicone lenses. The acrylic lenses are usually foldable, allowing their insertion through a smaller incision.

There have been few complications reported from the use of acrylic IOLs. These include scratches on the lens optic,[6] stress fractures[7] and transient marks[1] during folding, and post-operative glistenings.[8] We report a case of clouding/fogging of a foldable acrylic IOL (SC60B-OUV, Medical Devices Research, FL) made from poly-2-hydroxyethyl methacrylate polymer. This case is unique in that the fogging became apparent only 7 months after surgery and seems permanent.

Case report

A 73-year-old Caucasian woman underwent uneventful phacoemulsification with lens implant in her right eye. Her post-operative recovery was uneventful and vision improved from 6/24 pre-operatively to 6/12 with correction on a Snellen chart. Her past ocular history includes bilateral panretinal photocoagulation for proliferative diabetic retinopathy, mild to moderate dry macular degeneration of both eyes and known left optic atrophy which was long-standing. The left eye also has tractional retinal detachment involving the macular area and hence has poor vision. One year following surgery she complained of worsening vision but her Snellen acuity remained stable. Clinically her IOL was noted to be cloudy centrally, making it appear as though there was a nuclear cataract in the IOL (Fig. 1). The anterior and posterior chambers were quiet with no inflammatory cells or blood. Four months prior to this the visual axis was recorded as clear. A short course of prednisolone 0.5% drops was prescribed but made no difference to the cloudy appearance of the IOL. By the following month, the cloudiness increased and retinoscopy became more