Sir,

Reply to Abnormalities on the multifocal electroretinogram may precede clinical signs of hydroxychloroquine retinotoxicity

The article 'Abnormalities on the multifocal electroretinogram may precede clinical signs of hydroxychloroquine retinotoxicity' by Teoh *et al*[1] raises several questions.

1. The authors state that the patient had a normal 24–2 Humphrey VF 6 years after commencing hydroxychloroquine. It is recognized that VF testing that does not emphasize the macula will often miss the early defects of HC toxicity.[2] Thus, a paracentral scotoma may well have existed in 1999 had a 10–2 VF programme been used rather than a 24–2. That is, readers should not take the case presentation of Teoh to suggest that toxicity was not evident 6 years after commencing HC. Rather, the point to drive home is that the test ordered (24–2 VF) was the wrong test.
2. The authors' calculation of the patient's HC dosage is suspicious. They state that she received an approximate daily dose of 3.91 mg/kg. My calculation differs. We are told she received 3039 g of HC over 11 years. Thus, she received on average 3039/11 = 276.3 g HC yearly. Assuming that she took a daily dose of HC, the average daily dose would have been 276.3 g/365 days = 757 mg/day. Her height is listed at 1.50 m or 59 inches, for which top normal body weight is 119 lbs or 54.1 kg.[3] Thus, her daily HC dose, on average, was 757 mg/day/54.1 kg = 13.99 mg/kg/day, massively above the recommended upper limit of 6.5 mg/kg/day.

The main public health issue with regard to HC use is not the need for a more sensitive test to detect retinal toxicity, such as a multifocal ERG. Rather, it is greater awareness of proper dosing guidelines for the drug based on top normal body weight, which is in turn a function of height. The 10–2 VF is an appropriate and widely available screening test of adequate sensitivity to serve the purpose. Simple tables of appropriate dosages of HC have been published and can be kept in clinical lanes where patients taking are screened.

References

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Sir,

Choroidal folds secondary to parasellar meningioma

Choroidal folds can develop in association with any intra- or extraocular process that induces sufficient compressive stress within the choroid, Bruch's membrane, and retina to force these tissues to buckle. In other words, any deviation from a spherical shape (that represents the least surface area shape) results in increased surface area that is compensated for by folding of tissues. Choroidal folds can be secondary to hypotony, posterior scleritis, ocular/orbital mass, macular degeneration, scleral buckle, hyperopia, and papilledema. In some cases, no clear aetiology can be determined (idiopathic). Herein, we describe a case of parasellar meningioma, with extension into orbital apex and optic nerve, in a patient presenting with ipsilateral choroidal folds.

Case report

A 51-year-old white male, without significant past medical or ocular history, complained of decreased visual acuity left eye of 3 months duration. Visual acuity was 20/20 and 20/30 in right and left eyes, respectively. Pupils were equally reactive to light, without an afferent pupillary defect. Anterior segment examination was unremarkable and intraocular

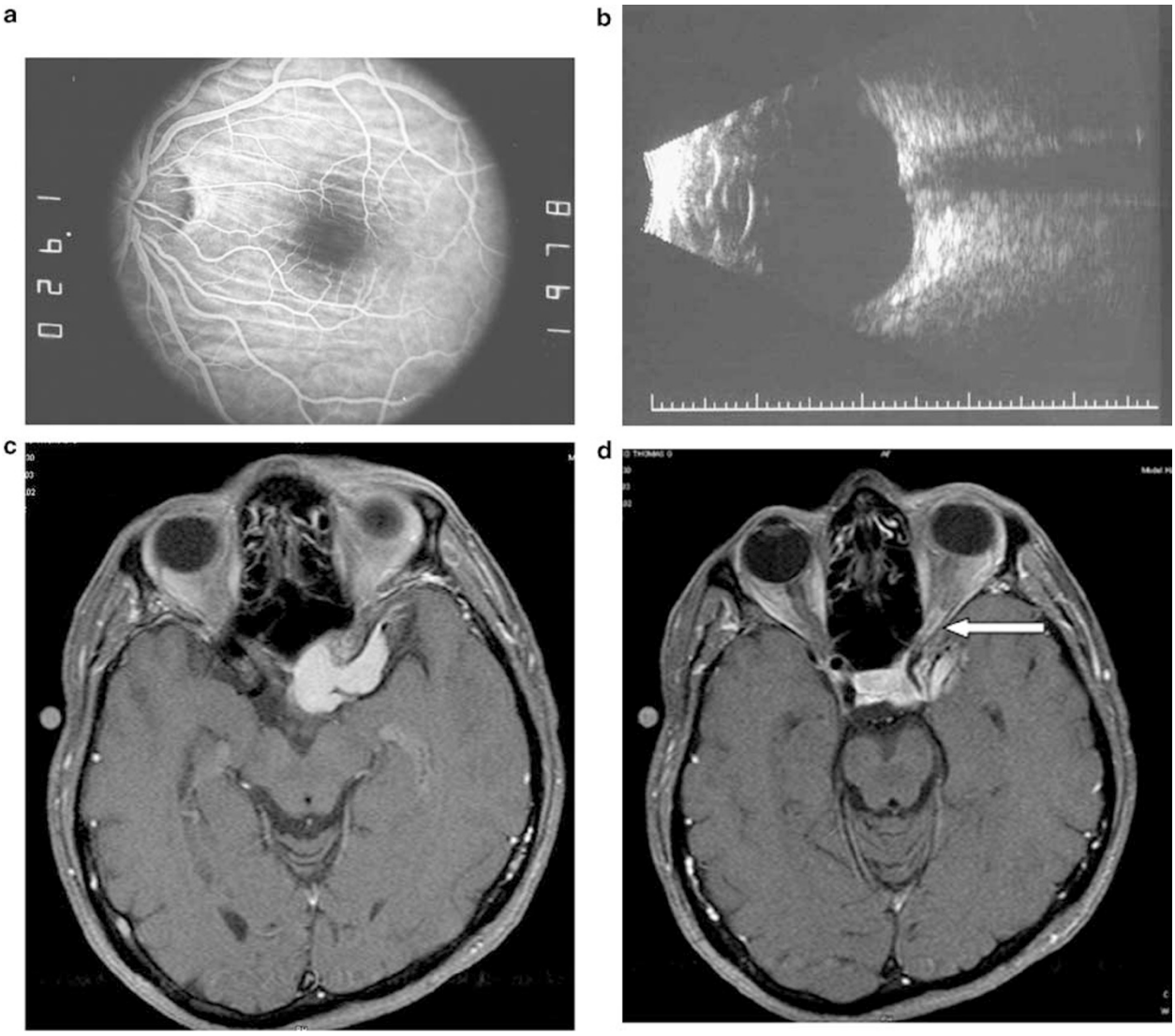

Figure 1 (a) Fluorescein angiography of the left eye showing choroidal folds with characteristic alternating hypofluorescent and hyperfluorescent lines at the level of the RPE and choroid. (b) B-scan ultrasound of the left eye illustrating flattening of the posterior pole. There is no evidence of orbital mass or scleritis. (c) T_1-weighted, contrast enhanced, axial MR imaging of the head and orbit showing a large, parasellar meningioma with extension into the left orbital apex. (d) Note thickening and enhancement of the left optic nerve sheath (arrow).

pressures were 18 mmHg both eyes. Dilated fundus examination revealed choroidal folds in the left eye, confirmed by fluorescein angiography (Figure 1a). No disc oedema or atrophy was present. Orbital ultrasound did not reveal an orbital mass or any signs of scleritis, but did show flattening of the posterior pole (Figure 1b). The patient cancelled follow-up appointments at 3 and 6 months and presented 2 years later complaining of slow, progressive worsening of vision in the left eye. Visual acuity was now 20/70 in the left eye. Afferent pupillary defect, reduced colour vision, and mild disc oedema with temporal pallor were observed. Humphrey visual fields confirmed central and peripheral defects in the left eye. Magnetic resonance (MR) imaging of the brain and orbits revealed a large parasellar meningioma, with extension into the left orbital apex along with thickening and enhancement of the left optic nerve sheath (Figure 1c and d). The patient underwent complete excision of the tumor. Intraoperatively, the tumor was found to encase the proximal optic nerve, with displacement superiorly and medially. On follow-up examination 1 year later, his vision in the left eye improved to 20/40 but the choroidal folds were still present, with no gross change.

Comment

We present an unusual case of parasellar meningioma presenting as ipsilateral choroidal folds, which initially did not have any disc oedema. Jacobson was the first to suggest that raised intracranial pressure (ICP) could be an independent cause of choroidal folds.[1] Griebel and Kosmorsky[2] showed elevated lumbar puncture pressures in five of six patients with idiopathic choroidal folds. Sharma *et al*[3] described a patient with unilateral choroidal folds followed 3 months later by papilledema. Hence, choroidal folds may be a sign of 'localized' elevated ICP within the subarachnoid space surrounding the optic nerve. Although disc oedema may not necessarily precede choroidal folds in all patients.

The presence of choroidal folds in our patient is most likely secondarily due to the parasellar meningioma with extension into ipsilateral orbital apex and proximal optic nerve, causing disc oedema. The disc oedema may have initially been transient. Cerebrospinal fluid could be forced through the subarachnoid space of the optic canal, into the subarachnoid space surrounding the optic nerve. Owing to the tumor encasement of the subarachnoid space at the level of the optic canal, the subarachnoid space would effectively become a one-way valve, trapping the fluid in the subarachnoid space of the optic nerve and elevating the pressure within this compartment, while not necessarily increasing the cerebrospinal fluid pressure of the cranium. The distention of the subdural space at the neuro-ocular junction would then cause the dura/arachnoid to buckle the posterior sclera and thereby induce a change in the normally spherical shape of the posterior sclera.

We believe the concurrence of the meningioma and choroidal folds in our case is not a coincidence for above-mentioned reasons. The fact that the choroidal folds have not resolved one year after surgery is not surprising, as it has been shown that most choroidal folds remain even after treating the underlying condition.[4,5] Moreover, it is extremely unlikely that choroidal folds and ipsilateral parasellar meningioma would occur coincidentally. The reported incidence of intracranial meningioma, silent or symptomatic, in men is between 1 and 5 per 100 000.[6,7] The incidence of 'idiopathic' choroidal folds is probably even less. The likelihood of both of these entities to occur independently would be very low.

In the past, the standard evaluation of choroidal folds has included complete ophthalmic examination, intraocular pressure, fluorescein angiography, and orbital ultrasound to demonstrate flattening of the posterior pole, thickening of choroid/scleral complex, and an enlarged optic nerve subarachnoid space.[8] We believe that choroidal folds could represent a sign of 'localized' elevated ICP secondary to intracranial pathology that could be localized to a single optic nerve. We recommend MR imaging to better assess the optic nerve and parasellar regions. This would not result in a significant increase in MR requests since it only needs to be performed if no other etiology (ie hypotony, posterior scleritis, ocular/orbital mass, macular degeneration, scleral buckle, hyperopia, and papilledema) is found to explain the choroidal folds. Essentially, it is only for those 'idiopathic' cases, which are rare.' A lumbar puncture should also be considered if imaging is unrevealing to exclude the possibility of raised ICP.

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Sir,

Toric posterior chamber (in-the-bag) intraocular lens implantation to correct postpenetrating keratoplasty astigmatism

Postkeratoplasty astigmatism is a cause of significant visual impairment and can be difficult to treat satisfactorily. Treatment options include compression sutures, astigmatic keratotomy, targeted suture removal, hard contact lens wear, excimer laser photoablation, and toric intraocular lenses. This is the first United Kingdom report of the use of a toric posterior chamber intraocular lens used to correct this type of astigmatism.

Case report

A 51-year-old lady underwent phacoemulsification and toric posterior chamber intraocular lens implantation in order to correct 11.50 Dioptres (D) of postpenetrating keratoplasty (PKP) astigmatism. She had undergone a right PKP 5 years previously for corneal scarring and contact lens intolerance. After the removal of her corneal sutures, she required a prescription of –5.00/ $+15.00 \times 175$; a contact lens trial failed. Paired arcuate keratotomies and compression sutures (subsequently removed) resulted in a refraction of $-4.50/+11.50 \times 170$. She had an early nucleosclerotic cataract and she was listed for a right phacoemulsification and toric intraocular lens implantation under local anaesthetic.

Preoperative refraction revealed OD: $-4.50/+11.50 \times 170$ (OS: $-0.75/+4.0 \times 70$). Her preoperative keratometry showed: 38.31 D at 80° and 50.55 D at 170° (IOLMaster, Carl Zeiss) equating to 12.44 D at 170°. Corneal topography (Keratron, Optikon 2000) showed a similar degree of regular astigmatism (Figure 1). After preoperative refraction and biometry, a custom-made toric intraocular lens of spherical power 13.00 D and cylindrical power 15.00 D was ordered from HumanOptics (Mannheim, Germany) with a refractive aim of plano ($+0.13$ Spherical equivalent). The required IOL power was calculated by the manufacturer, using the above preoperative measurements.

Cataract surgery was performed under topical and intracameral anaesthetic (amethocaine 0.5% and

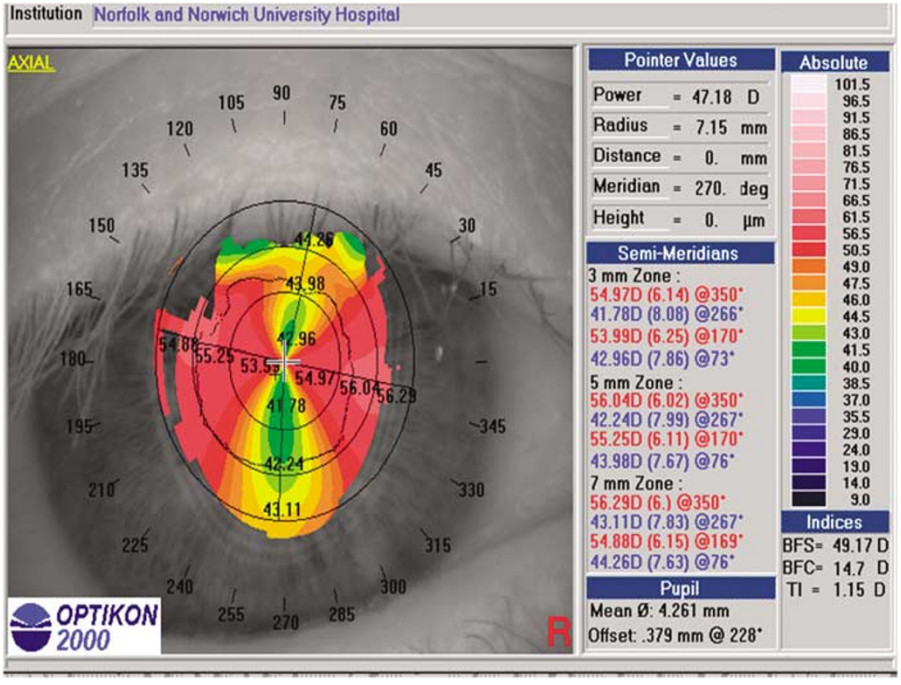

Figure 1 Corneal topography of the patient's right eye following removal of the corneal sutures, revealing significant against-the-rule astigmatism.