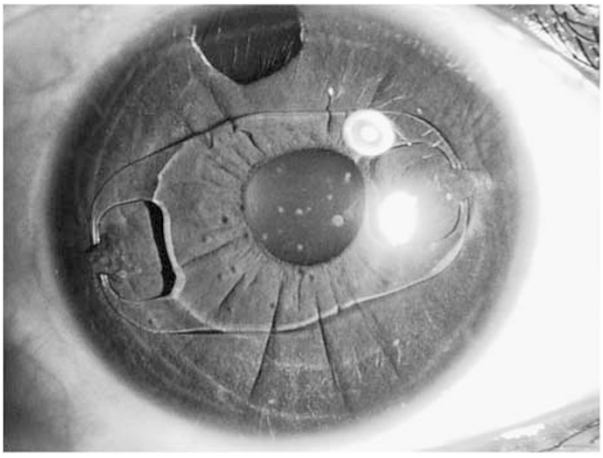

Figure 2 Slit lamp view at last follow-up showing a quiet eye and complete resolution of the fibrin coagulum.

further to counting fingers at 2 m. There were fine KPs on the endothelium. The coagulum appeared retracted but was covering both sides of the lens (Figure 1b). She was continued on the same treatment and the membrane progressively retracted. The intraocular pressure remained normal. The steroid drops were tapered. She was last seen 5 weeks after surgery, when she had an unaided visual acuity of 20/40 in the right eye and the best corrected visual acuity of 20/30 with correction. The eye was quiet and the fibrin coagulum had completely retracted. There were a few pigments on the intraocular lens (Figure 2).

Comment

Since iris supported model of intraocular lens is fixated directly to the iris tissue, causing pressure or shear forces when the eye is moving, chronic inflammation is a concern. Studies using iris angiography have shown no leakage of the iris vessels.[3,4] Studies using laser flare cell meter revealed diverging results. Fechner *et al*[4] showed no elevated flare levels in 109 eyes with at least 12 months of follow-up. Perez Santonja *et al*[5] found elevated flare levels compared to normal population in 30 eyes at 12, 18, and 24 months after surgery. In all of the studies, clinically relevant inflammation could only be detected in individual cases. Careful monitoring of inflammation following surgery is necessary.

We present this case to highlight that severe inflammation can follow iris fixated phakic intraocular lens for myopia. The interesting feature of this case was that the severe inflammation with fibrin and a coagulum, which became focal was found on the intraocular lens with iris tissue absolutely spared suggesting that the inflammation was sterile. We decided not to explant the lens and continue on intensive steroid therapy. In case, if similar cases are encountered, the patient can be monitored closely and treated with steroids thus delaying surgical intervention.

References

1 Pom M, Payette Y. Initial results of endothelial cell counts after Artisan lens for phakic eyes: an evaluation of the United States food and drug administration Ophthec study. *Ophthalmology* 2004; **111**: 309–317.
2 Kohen T, Mirashahi A, Buhren J, Kasper T, Baumeister M In: Hardten DR, Lindstorm RL, Davis EA (eds). *Phakic Intraocular Lens, Principles and Practice* 2004 pp 81–96.
3 Menezo JL, Avino JA, Cisneros A, Rodriguez-Salvador V, Martinez-Costa R. Iris claw phakic intraocular lens for high myopia. *J Refract Surg* 1997; **13**: 545–555.
4 Fechner PU, Strobel J, Wichmann W. Correction of myopia by implantation of a concave worst iris claw lens into phakic eye. *J Refract Corneal Surg* 1991; **7**: 286–298.
5 Perez Santonja JJ, Iradier MT, Benitez del Castillo JM, Serrano JM. Zatoma Chronic sub-clinical inflammation in phakic eyes with intraocular lens to correct myopia. *J Catract Refract Surg* 1996; **23**: 183–187.

MS Sridhar[1], AB Majji[2] and PK Vaddavalli[1]

[1]Cornea & Anterior Segment Service, LV Prasad Eye Institute, Hyderabad, India

[2]Kanuri Santhamma Vitreo-Retinal Center, LV Prasad Eye Institute, Hyderabad, India

Correspondence: MS Sridhar, LV Prasad Eye Institute, L.V.Prasad Marg, Banjara Hills, Hyderabad 500 034, India
Tel: +91 40 306 12520;
Fax: +91 40 235 48271.
E-mail: mss@lvpei.org

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Sir,

Unilateral papilloedema with transient visual obscurations

Unilateral optic disc swelling is most frequently caused by a local optic nerve or intraocular pathology. We present a case of unilateral disc swelling secondary to

raised intracranial pressure and associated with bilateral visual obscurations.

Case report

A 49-year-old female presented with a 1-month history of generalised headaches and transient visual obscurations. The episodes of visual loss were bilateral, lasted a few seconds and occurred several times a day. She was otherwise well. Past ophthalmic history included left amblyopia with a definite history of occlusion treatment in childhood. She was a nonsmoker and had controlled essential hypertension.

Ocular examination revealed corrected visual acuity of 6/5 right and 6/24 left. Anterior segment examination, intraocular pressures, and colour vision were normal. There was no afferent pupillary defect. The right optic disc was swollen and hyperaemic with superficial haemorrhages (Figure 1a). The left optic disc appeared normal (Figure 1b). Ocular movements were full and there was no proptosis.

Magnetic resonance imaging (MRI) demonstrated a large left-sided medial sphenoid wing mass involving the lateral wall of the cavernous sinus and partially encasing the cavernous portion of the left internal carotid artery. No lesions were identified close to the right optic nerve, orbit, or orbital apex. There was evidence of midline shift suggesting raised intracranial pressure (Figure 2).

The patient underwent a craniotomy and excision of the tumour. At surgery the tumour was found to be adherent to the optic and occulomotor nerves. It was dissected from these structures but a residual layer was densely adherent to the lateral wall of the cavernous sinus. A small amount of residual tumour was cauterised. Histologically, the tumour was confirmed as a meningioma with a slightly raised mitotic count, it was therefore classified as atypical meningioma WHO Grade 2.

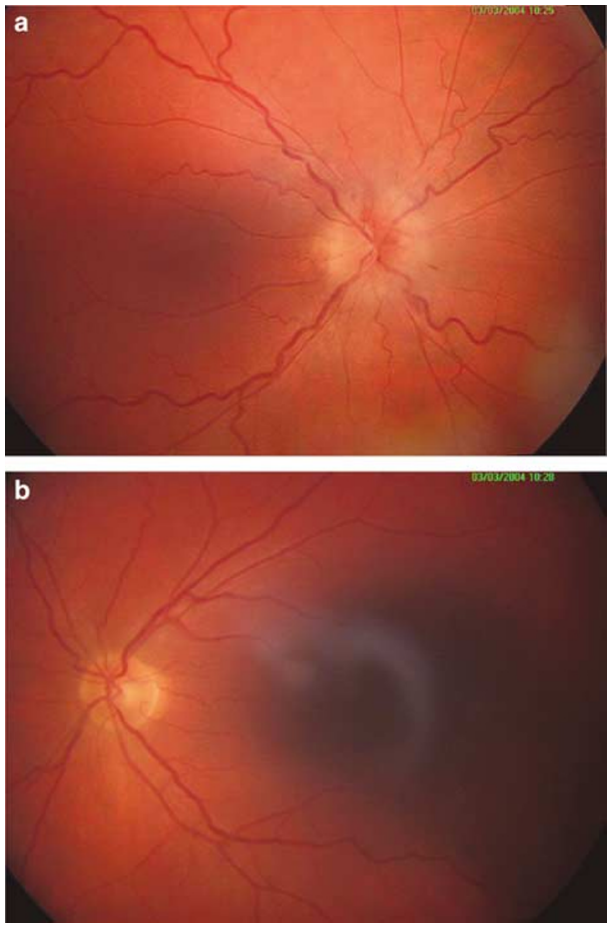

Figure 1 Fundus photograph of right (a) and left (b) optic discs.

Comment

The term papilloedema refers specifically to optic disc swelling in the presence of raised intracranial pressure. Truly unilateral papilloedema is rare and poses a diagnostic dilemma. An intact optic nerve sheath is apparently necessary for the development of disc oedema in the presence of raised intracranial pressure.[1] Acquired and congenital anomalies in the optic nerve sheath which prevent transmission of increased cerebrospinal fluid pressure to the optic nerve, have been hypothesised as a reason for unilateral papilloedema. Other postulated mechanisms include abnormalities in the venous sinuses and a difference in the lamina cribrosa between the two discs resulting in decreased transmission of the raised intracranial to the optic nerve in the scleral canal.[2]

In the case presented, the potential mechanism for the unilateral papilloedema is the presence of a tumour adherent to the intracranial optic nerve preventing transmission of the increased pressure down the perioptic space to the optic nerve head. High resolution CT or MRI imaging, not performed in this case, may demonstrate asymmetrical optic nerve sheath swelling, but this is not universal. Huna-Baron *et al*[2] and To and Warren[3] were unable to demonstrate a difference in size of the optic nerve sheaths using orbital CT or MRI in patients with unilateral papilloedema. This would support the conclusion that development of a single swollen disc in the presence of increased intracranial pressure does not result from gross difference in the optic nerve sheath between the two sides. The possibility of minute differences in the size of the optic nerve sheaths,

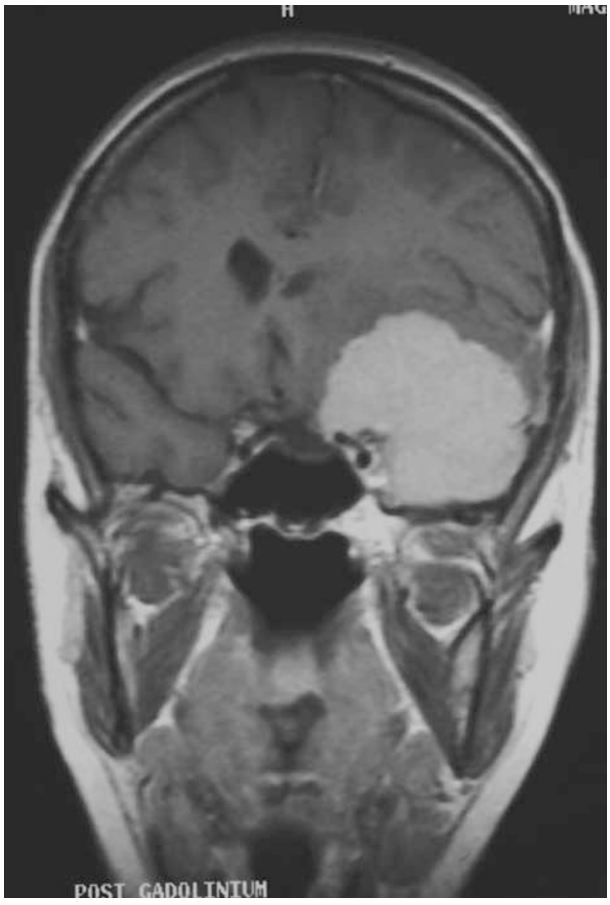

Figure 2 Gadolinium enhanced coronal MRI scan demonstrating large intracranial mass.

beyond the sensitivity of CT or MRI cannot be eliminated.

Transient visual obscurations, usually lasting seconds, are a well-known accompaniment of raised intracranial pressure. They may occur with or without the presence of optic disc swelling. The pathogenesis remains something of an enigma. Hayreh[4] explained the phenomenon as being due to ischaemia of the optic nerve head and downward herniation of the parahippocampal gyrus in the tentorial notch. Another theory is of intermittent occipital lobe ischaemia related to compression of the posterior cerebral artery usually from uncal herniation.[5] Sadun *et al*[6] suggested that transient ishaemia of the optic nerve head consequent to increased tissue pressure could explain the presence of unilateral visual obscurations occurring with or without raised intracranial pressure. This theory would, however, not explain the presence of bilateral visual obscurations in the presence of unilateral papilloedema.

This case serves to remind us that unilateral papilloedema, though rare, does occur and that transient visual obscurations can occur without or prior to development of optic disc swelling.

References

1 Hayreh SS. The sheath of the optic nerve. *Ophthalmologica* 1984; **189**: 54–63.
2 Huna-Baron R, Landau K, Rosenberg M, Warren FA, Kupersmith MJ. Unilateral swollen disc due to increased intracranial pressure. *Neurology* 2001; **56**: 1588–1590.
3 To KW, Warren FA. Unilateral papilloedema in pseudotumor cerebri. *Arch Ophthalmology* 1990; **108**: 644–645.
4 Hayreh SS. Optic disc edema in raised intracranial pressure. VI. Associated visual disturbances and their pathogenesis. *Archives of Ophthalmology* 1977; **95**(9): 1566–1579.
5 Hilton-Jones D, Ponsford JR, Graham N. Transient visual obscurations without papilloedema. *Journal of Neurology, Neurosurgery and Psychiatry* 1982; **45**(9): 832–834.
6 Sadun AS, Currie JN, Lessell S. Transient visual obscurations with elevated optic discs. *Annals of Neurology* 1984; **16**(4): 489–494.

A Cole and NDL George

Department of Ophthalmology, Ninewells Hospital and Medical School, Dundee DD1 9SY, UK

Correspondence: A Cole,
Tel: +44 1382 660111;
Fax: +44 1382 632893.
E-mail: abosede_s@yahoo.co.uk

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Sir,

Visual loss precluding acute onset of Budd–Chiari syndrome

Budd–Chiari Syndrome (BCS) occurring as a result of occlusion of the major hepatic veins manifests either acutely or chronically with a triad of abdominal pain, hepatomegaly and ascites. The natural course is progressive with a mortality rate of 75%.[1] Most patients require anticoagulation and urgent decompression or shunting to avoid progressive liver damage. We are unaware of any previous reports of retinal vein occlusions precluding this life threatening complication.

Case report

A 39-year-old lady of Nigerian ancestry presented to the eye casualty department of a London hospital with a 1-week history of painless visual loss in her left eye (LE).