Case report

A 50-year-old Caucasian woman presented with an acute decrease in vision in her right eye that deteriorated over approximately 48 h. Visual acuity was counting fingers from 1 foot on the right and 20/80 on the left. There were nuclear cataracts in both eyes. The right fundus demonstrated retinal oedema, cherry-red spot and 'kickboxing' of the retinal arterial flow. The left fundus appeared normal. Since the symptoms had been present for a little less than 48 h the patient was treated with ocular massage, sub-lingual isosorbide dinitrate, intravenous acetazolamide 500 mg, parenteral glycerol 50% 1 mg/kg, anterior chamber paracentesis, retrobulbar tolazoline and intravenous methylprednisolone 500 mg followed by streptokinase 750 000 units, but vision remained unchanged. Concomitantly, she was treated with coumadin 2.5 mg/day.

Two years earlier the patient had been diagnosed as having primary antiphospholipid syndrome. She suffered from systemic hypertension, chronic renal failure, deep vein thrombosis, ulcus cruris, thrombocytopenia, seizure disorder and had a history of 11 consecutive spontaneous abortions followed by 2 normal deliveries. On admission, the coagulation profile showed a prolonged partial thromboplastin time (52 sec), IgG anticardiolipin antibodies and positive lupus anticoagulant. Serum creatinine was 4.1 mg% and urea 122 mg%, indicating impaired renal function. Plasma cholesterol was 230 mg% and triglycerides were 186 mg%. Other serial laboratory tests were negative for antinuclear and anti-DNA antibodies, VDRL and HbS antigen. Serum protein electrophoresis was normal. Echo Doppler sonography of the carotids and echocardiography were normal. However, magnetic resonance imaging of the brain demonstrated multiple cortical infarcts around the tentorium and in the left thalamus, and cortical atrophy. When CRAO occurred the patient was under treatment with nifedipine 20 mg b.i.d., benazepril HCl 2.5 mg 1.q.d., pentoxifylline 400 mg t.i.d., allopurinol 100 mg 1.q.d., frusemide 40 mg 1.q.d. and slow-Fe 160 mg 1.q.d. Currently the patient also takes dipyridamole 75 mg t.i.d. and lovastatin 20 mg 1.q.d., and her blood pressure is well controlled.

Comment

The ophthalmic findings described in primary antiphospholipid syndrome have been attributed to microemboli, initiated by circulatory immune complexes. In our patient they may have occurred in a larger vessel, the central retinal artery, without any other funduscopic findings. Demonstration of multiple brain infarcts by magnetic resonance imaging suggests that these are also a result of multiple emboli or thrombi; and these findings in undiagnosed patients should raise the suspicion of other possible systemic disorders such as systemic lupus erythematosus (as part of secondary antiphospholipid syndrome), Sneddon's syndrome and Susac's syndrome.[4] All these clinically different entities may eventually be reclassified due to different underlying defects.

Although streptokinase may disintegrate the thromboemboli, the time interval between the occlusion and treatment of the widespread coagulation disorder was probably the cause for the irreversible visual loss.

Several factors, including systemic hypertension and hyperlipidaemia, may have caused CRAO in this patient; however, the underlying disorder for these findings is primary antiphospholipid syndrome. We suggest that primary antiphospholipid syndrome should be considered as a rare cause for retinal artery and vein occlusions, especially in young patients and patients with history of thrombotic events. A complete coagulation profile should be obtained for these patients to rule out other coagulation disorders, including secondary antiphospholipid syndrome, protein C and protein S deficiency,[5] since these entities share a common final pathway causing thromboembolic events.

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Sir,

Posterior dislocation of Staar plate haptic silicone lenses following Nd:YAG capsulotomy

We present three cases of delayed spontaneous posterior dislocation of Staar plate haptic silicone lenses following Nd:YAG capsulotomy. All patients underwent elective phacoemulsification with continuous circular

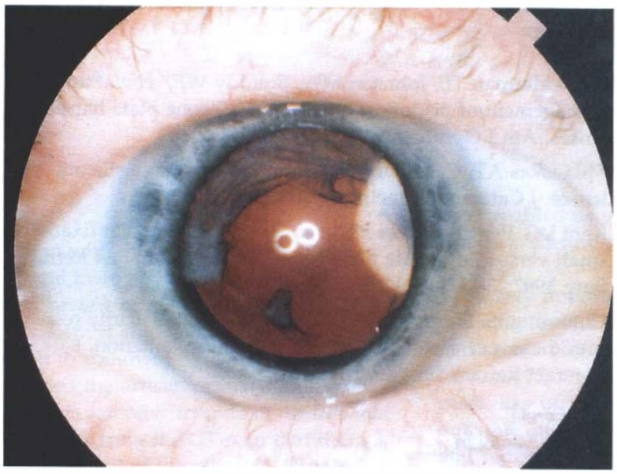

Fig. 1. *Case 1 following dislocation of the intraocular lens.*

capsulorrhexis and uncomplicated in-the-bag placement of a 12 mm diameter Staar AA-4203V silicone plate haptic lens.

Case reports

Case 1. A 64-year-old man developed progressive posterior capsular opacification. Nd:YAG capsulotomy was performed 3 months after surgery. Two months later he experienced a sudden drop in vision and examination revealed that the intraocular lens had dislocated posteriorly into the vitreous. The size of the posterior capsulotomy was noted to be much smaller than the size of the intraocular lens. The patient underwent an anterior vitrectomy with insertion of a secondary posterior chamber intraocular lens. The dislocated silicone lens remained in the vitreous cavity and became increasingly mobile resulting in intermittent blurring of vision. Pars plana vitrectomy with removal of the silicone lens was therefore performed. His final corrected visual acuity was 6/9.

Case 2. A 75-year-old woman achieved a visual acuity of 6/9 with correction following cataract surgery. Progressive posterior capsular fibrosis led to clouding of the vision and a Nd:YAG capsulotomy was performed 21 months after surgery. Five months after this procedure the patient experienced a sudden drop in vision on wakening and examination revealed that the intraocular lens had dislocated posteriorly. A pars plana vitrectomy with intraocular lens exchange was performed and the final visual acuity was 6/9 with astigmatic correction.

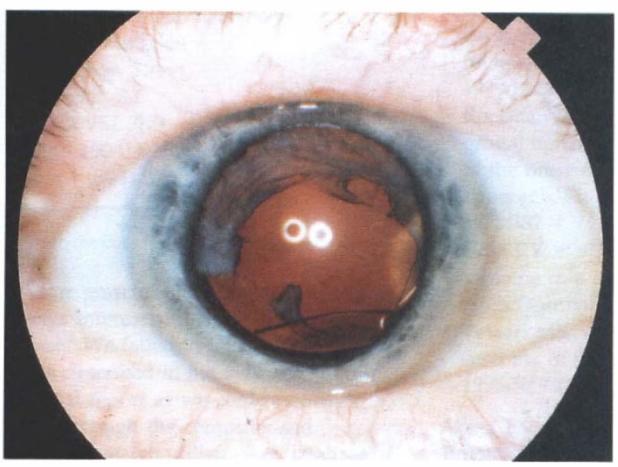

Fig. 2. *Case 1 following dislocation of the intraocular lens and immediately after a vertical saccade. The edge of the Staar lens is visible inferiorly.*

Case 3. A 78-year-old woman had a visual acuity of 6/12 with correction post-operatively due to posterior capsular opacification. A Nd:YAG capsulotomy was performed 7 weeks after surgery. Ten days later the patient complained of sudden loss of vision and reported a highly mobile 'rectangular shadow with holes in it'. Posterior dislocation of the silicone plate haptic lens into the vitreous had occurred and the patient underwent pars plana vitrectomy and lens exchange. The post-operative corrected visual acuity was 6/9.

Comment

The cataract surgery was performed by three different surgeons using the same surgical techniques. In all cases surgery and the subsequent Nd:YAG capsulotomy was uncomplicated but in these cases spontaneous delayed posterior dislocation of the 12 mm Staar plate haptic lens occurred. Case 1 had a circular capsulotomy and cases 2 and 3 had cruciate capsulotomies. The mean time of Nd:YAG capsulotomy following cataract surgery was 8.5 months (range 1.5–21 months) and the mean time to dislocation was 7.3 months (range 0.3–5 months).

Schneiderman *et al.*[1] reported a series of 11 consecutive eyes that suffered delayed posterior dislocation of the silicone plate haptic lens. In 8 of these eyes this occurred after Nd:YAG capsulotomy. The average time to dislocation was 1.8 months after Nd:YAG capsulotomy (range 0–6.5 months). In one case the posterior dislocation was seen at the time of Nd:YAG capsulotomy. They advocate pars plana vitrectomy with intraocular lens repositioning or exchange and report excellent post-operative visual acuity with minimal complications with this technique. One patient suffered a retinal detachment but did well following further surgery.

It is postulated that shrinkage of the anterior capsule puts tension on the flexible plate haptic implant causing the optic to move posteriorly and exert pressure on the posterior capsule. Release of this tension through defects in the capsule following Nd:YAG capsulotomy leads to dislocation into the vitreous. Anterior capsule phimosis may have a purse-string effect on the capsular bag making dislocation more likely.

Unlike loop haptics, plate haptics do not become fixed to the capsule by capsular fibrosis.[2] Instead they are held in place by pockets formed when the anterior capsule fibroses onto the posterior capsule. Large-hole Staar haptics have recently been developed in the hope that larger holes will allow proliferation of lens epithelial cells through them and thus increase the security of capsular bag fixation. Studies in rabbit eyes have not, however, demonstrated a significant difference in the amount of

force required to dislocate a large- and a small-hole silicone plate haptic lens from the capsular bag 2 months after implantation.[3]

Posterior dislocation of silicone plate haptics can occur in a delayed fashion without Nd:YAG capsulotomy. In these cases some other weakness in the capsule following complications during surgery can often be identified. A review of surgical complications in 536 cases of plate haptic silicone lens implantation demonstrated posterior dislocation in 2 cases.[4] In both cases a posterior capsular tear occurred during surgery.

The silicone Staar plate haptic lens is convenient to insert at the time of surgery and has the advantage of only requiring a 3 mm section, thus minimising post-operative astigmatism. There is, however, a small risk of delayed posterior dislocation of silicone plate haptic lenses after Nd:YAG posterior capsulotomy. Risk factors have not been identified and the time interval between cataract surgery and capsulotomy in our cases was variable. It is not possible to recommend a safe period. Surgeons should therefore inform their patients of the small risk of complications requiring further surgery when signing consent for Nd:YAG capsulotomy.

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