Delayed ciliochoroidal detachment following intraocular lens implantation

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Abstract
Three patients who had extracapsular cataract extractions with intraocular lens implants developed delayed ciliochoroidal detachments, which responded to systemic steroid therapy. This rare complication may have been due to ciliary sulcus fixation of the implant. The possible mechanism and treatment are discussed.

Delayed ciliochoroidal detachment following cataract surgery is a rarely described complication which may occur some months or years after surgery. The cause is unclear, though trauma, with possible reopening of the section, has been suggested.1

We describe three cases which developed approximately nine months after ciliary sulcus fixation of two different types of intraocular lens implants.

Case reports

CASE 1
A 63-year-old woman who was generally well underwent an uncomplicated right extracapsular cataract extraction with intraocular lens implantation for a posterior subcapsular cataract. A 21-dioptre posterior chamber Surgidev BUV 20–20 intraocular lens was inserted. There were no immediate postoperative complications. Three weeks after surgery her best corrected vision was 6/6.

Eight months later she presented as a casualty with a one-week history of pain in the right eye. On examination the vision was 6/12+1 with correction. Mild conjunctival injection was present with flare and 2+ cells in the anterior chamber. The intraocular pressure was 1 mm Hg. Gonioscopy revealed an open angle with no evidence of separation of the ciliary body from the scleral spur. On dilatation the intraocular lens was noted to be well centred, though both haptics were in the ciliary sulcus. A small choroidal detachment was present round the entire retina, with some posterior extension infero-temporally. The left eye was normal, with an intraocular pressure of 11 mm Hg.

The patient was admitted to hospital. Oral prednisolone 45 mg daily and topical dexamethasone 0·1% four times a day, cyclopentolate 1% four times a day, and phenylephrine 10% once daily to the right eye were prescribed. Two days later the choroidal detachment had increased, and the anterior chamber was shallow. The eye was padded, and acetazolamide 250 mg four times a day prescribed for three days. The topical steroid was changed to prednisolone acetate 1% two-hourly for the duration of her admission. Four days later the anterior chamber was deeper, though there was no discernible change in the choroidal detachment. The prednisolone was increased to 60 mg daily. On this dosage the patient became restless so it was reduced to its former level after four days, and acetazolamide 250 mg four times a day was reinstituted the following day. Two days later the choroidal detachment appeared the same, the anterior chamber was formed, and visual acuity had fallen to 6/24. Fluorescein angiography showed no evidence of macular oedema. Five days later the anterior chamber was of normal depth, and the choroidal effusion had started to resolve. She was discharged on the same medication.

On review in the outpatient department 10 days after discharge from hospital her visual acuity was normal and the choroidal detachment had resolved completely. Her medications were reduced. Four weeks later all medication had been withdrawn and her visual acuity was 6/6.

Three months after discharge she attended as a casualty again with a sore eye. On examination the visual acuity was 6/7·5, and flare and 1+ cells were present in the anterior chamber. A small inferior choroidal detachment was seen on funduscopy. This resolved on treatment with topical dexamethasone 0·1% and cyclopentolate 1%. There have been no further recurrences.

CASE 2
A 65-year-old woman, who was fit and well, underwent an uneventful left extracapsular cataract extraction with insertion of a 21-dioptre posterior chamber Rayner Pearce tripod intraocular lens into the ciliary sulcus. Two years before she had had an uncomplicated right cataract extraction with implantation of a similar lens into the capsular bag.

On review six months later her best corrected visual acuity in the left eye was 6/18. Dry macular degeneration was noted. No intraocular inflammation was present, and intraocular pressure was normal. The patient was discharged.

Two months later she presented with a two-week history of photophobia and blurred vision in the left eye. The visual acuity was reduced to 6/24. Moderate flare and 2+ cells were present in the anterior chamber. The intraocular pressure was less than 1 mm Hg, with shallowing of the anterior chamber. On gonioscopy the iris root was displaced forwards by the feet of the implant in the ciliary sulcus, but there was no evidence of a cyclodialysis cleft. On funduscopy a ciliary
body and 360° annular choroidal detachment were present. The right eye was quiet, with no fundal abnormality apart from early dry macular degeneration.

The patient was admitted to hospital, and oral prednisolone 80 mg daily together with topical dexamethasone 0·1% four times a day and cyclopentolate 1% three times a day to the left eye were prescribed. Five days later the anterior chamber was quiet, and the annular choroidal detachment had almost completely resolved.

The dose of prednisolone was reduced and the patient allowed home. One month later the anterior chamber was still quiet, and there was no sign of ciliochoroidal detachment. The intraocular pressure was 14 mm Hg and her visual acuity had returned to 6/18. All medication was stopped, and on review one month later there was no sign of recurrence.

Six months later the left anterior uveitis recurred. On this occasion the intraocular pressure was elevated at 30 mm Hg, but there was no sign of ciliochoroidal detachment. The condition responded to a three-week course of topical steroid and cycloplegic drops.

CASE 3

A 74-year-old hypertensive woman underwent an uneventful left extracapsular cataract extraction with insertion of a 20-dioptre posterior chamber Rayner Pearce tripod intraocular lens. Postoperatively it was obvious that fixation was in the ciliary sulcus. The postoperative course was uneventful. Three months after surgery her corrected visual acuity was 6/6, no inflammation was present, and the intraocular pressure and fundus were normal.

Nine months later she presented with a six-week history of mild photophobia and blurred vision in her left eye. Examination of the left eye revealed a best corrected visual acuity of 3/60, mild anterior uveitis, a shallow anterior chamber, an intraocular pressure of less than 1 mm Hg, ciliary body detachment, and a 360° annular choroidal detachment. The choroidal detachment extended posteriorly, inferiorly, and temporally to involve the macula. There was no evidence of retinal detachment. Gonioscopy showed a similar appearance to the iris root to that noted in the second case. There was no evidence of a cyclodalysis cleft. The other eye was entirely normal.

The patient was admitted to hospital. Oral prednisolone 40 mg daily, topical dexamethasone 0·1% four times a day, and atropine 1% twice a day to the left eye were prescribed. After five days the anterior chamber was quiet but the ciliochoroidal detachment remained the same. Consequently the oral prednisolone was increased to 60 mg daily. Over the following three weeks the choroidal detachment gradually resolved, the ciliary body became reattached, the anterior chamber deepened, and the intraocular pressure returned to normal. The patient was discharged on reducing doses of systemic steroids, and four weeks later all medications were stopped. There have been no recurrences, but owing to secondary macular changes the best corrected vision in the left eye remains 6/24.

Discussion

The cause of delayed ciliochoroidal detachment following extracapsular cataract extraction with intraocular lens implantation is unclear. Trauma with reopening of the wound section has been suggested as a possible cause in some cases.1 This could result in ocular hypotony and a shallow anterior chamber, with ciliary body and choroidal detachment. In the three cases described above the sections appeared well healed, with no sign of disruption or leakage. The choroidal detachment preceded the flat anterior chamber in the first case, which would be unlikely with wound leakage. Furthermore the choroidal detachments resolved on high-dose steroids, which would be expected to delay rather than accelerate healing of a leaking wound.

A cyclodalysis cleft may cause hypotony and choroidal effusion. Hypotony due to a cyclodalysis cleft has been described as late as 14 months after intracapsular cataract surgery with a medallion clip intraocular lens;1 it has also been described following rotation of an intraocular lens haptic through a peripheral iridectomy into a cyclodalysis cleft in an eye which had undergone a combined trabeculectomy, cataract extraction, and posterior chamber lens implantation.1 Hypotony in an eye is one of the main causes of a choroidal effusion.1 However, in the three cases described there were no signs of a cyclodalysis cleft on gonioscopy, and no peripheral iridectomy was present.

In all three cases the intraocular lens haptics were placed in the ciliary sulcus, and the choroidal effusions resolved on high-dose steroids. Signs of anterior chamber inflammation were also present. Ciliary sulcus fixation in intraocular lens implantation causes a greater change in the blood-aqueous barrier than does capsular bag fixation.2 In aqueous fluorophotometry the amount of fluorescein appearing in the anterior chamber is significantly greater after cataract extraction with ciliary sulcus lens fixation than with capsular bag fixation.3

Disruption of the blood-aqueous barrier could be produced by mechanical contact of a lens haptic with the surfaces of the iris and ciliary body in ciliary sulcus fixation. In capsular bag fixation the intervening capsule may protect these surfaces from contact. This disruption could result in increased permeability of uveal vessels and transudation of fluid from them into the supraciliary and suprachoroidal spaces. This could cause detachment of the ciliary body and choroid, with consequent decreased production of aqueous and hypotony as described by Chandler and Maumee.4 The hypotony would produce an increased pressure gradient between the choroidal vessels and suprachoroidal space and thus promote further transudation of fluid into the suprachoroidal space, increasing the size of the effusion.

Steroids have been shown to reduce pathologically raised capillary permeability in a variety of human ocular inflammatory conditions.5 High-dose oral steroids in these cases may have reversed the disruption of the blood-aqueous barrier and reduced the permeability of the uveal vessels, thus permitting resolution of the ciliochoroidal detachment.
The intraocular lens implant material may have caused inflammation and alteration of the blood-aqueous barrier in the three cases described above. Nylon and polypropylene as used in the haptics of older types of intraocular lenses can activate complement and thus cause intraocular inflammation. Both the Surgitex BUV 20–20 and the Rayner Pearce tripod are made entirely of polymethylmethacrylate (PMMA), which is more inert. However, it has been shown that it can also activate complement. The PMMA lenses used in the above cases may have produced a persistent low-grade activation of complement which may have caused the alteration in the blood-aqueous barrier and intraocular inflammation noted in all three. Steroid treatment, both topical and systemic, would have inhibited the activation of complement.

The delayed occurrence of ocular inflammation and ciliochoroidal detachment in all three cases described above is of interest. The routine topical postoperative steroids could have suppressed an early occurrence, but all the patients had been off medication for at least several months. Two possibilities may account for the delayed response. The intraocular lens haptics might have migrated from their original site and eroded the ciliary body. Ciliary body and iris erosion by PMMA lens haptics and even obliteration of the major arterial circle of the iris have been demonstrated at necropsy. Such erosion would take several months and may be commoner in ciliary sulcus than capsular bag fixation.

The other possibility is that the PMMA in the lens haptics has undergone degradation in the eye, producing a compound capable of initiating inflammation perhaps via the activation of complement. This might take several months to occur and would explain the delay before the inflammation and ciliochoroidal detachment occurred. However, PMMA does not seem to undergo biodegradation. Scanning electron micrographs of intraocular lenses that have been implanted in an eye for many years show little or no changes in the surface of the PMMA. Indeed, when a PMMA Boberg-Ans lens which had been implanted in a patient for 23 years was compared with an unused lens of the same age by scanning electron microscopy, there was no evidence of biodegradation. It is unlikely that the lenses in the three cases above could have been degraded.

To avoid this complication we would suggest that, where circumstances permit, intraocular lens implantation in the capsular bag is preferable to the ciliary sulcus. Treatment of this condition consists of high-dose oral and topical steroids. If these are ineffective, adding acetazolamide to the medication should be considered. In the first case acetazolamide appeared to aid restoration of anterior chamber depth and may have contributed to the resolution of the ciliochoroidal detachment. Finally, if the condition fails to resolve on both topical and systemic medication, removal of the implant may be necessary.

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