

CLINICAL COMMUNICATION

Refractory cystoid macular oedema due to intraocular lens haptic perforating the iris

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Cystoid macular oedema is a well-known complication of cataract surgery associated with intraocular lens decentration or dislocation. A 55-year-old man, who had undergone a phacoemulsification and intraocular lens implantation surgery two months previously was referred because of reduced vision in the right eye. Ocular examination revealed that one of the haptics had perforated the iris at 6 o'clock. There was cystoid macular oedema of the right eye. A topical non-steroid anti-inflammatory drug, followed by intravitreal injections did not produce a significant regression. Finally, the haptic was repositioned surgically and the macular oedema dramatically resolved. Correct placement of the intraocular lens might avoid post-operative complications including cystoid macular oedema.

Key words: cystoid macular oedema, intraocular lens, iris perforation, macula, retina

Cystoid macular oedema (CME) can be a serious consequence of cataract surgery. It can be subclinical but sometimes results in transient or even permanent visual loss. It is more likely to occur when cataract surgery is complicated by intraocular lens (IOL) decentration or dislocation.^{1–3} We report a case of persistent CME, where an IOL haptic perforated the peripheral iris.

CASE REPORT

A 55-year-old man who had undergone a phacoemulsification and IOL implantation two months previously was referred because of reduced vision in the right eye first noted four weeks after surgery. The early post-operative phase had been complicated by moderate irritation and con-

junctival congestion and these were still present at the time the patient attended our clinic. On admission, visual acuities were 6/30 (Snellen) on the right and 6/7.5 on the left eye. Intraocular pressure measurements were within normal limits.

Ocular examination revealed a foldable hydrophilic acrylic single piece IOL implanted in the posterior chamber. The IOL had been positioned in the sulcus not in the capsular bag; however, one of the haptics had perforated the iris at 6 o'clock during surgery and the tip of the haptic became visible on slitlamp biomicroscopy (Figure 1A). The cornea was clear and the anterior chamber was quiet. Fundus examination revealed increased central macular thickness, while other structures appeared normal. Fundus fluorescein

angiography and optical coherence tomography (OCT; Heidelberg Engineering, Germany) confirmed the diagnosis of CME (Figure 1B). Foveal macular thickness on OCT was 526 microns. There was no vitreoretinal traction or epiretinal membrane. Foveal macular thickness in the fellow eye was 268 microns.

Topical ketorolac drops qid were prescribed. No improvement in either visual acuity or macular thickness was observed during the following four months. Interventions with intravitreal injections of triamcinolone acetonide and bevacizumab were attempted but did not produce a significant regression of the macular oedema (Figure 1C). Foveal macular thickness was 441 microns. After eight months, the lack of improvement led to discussion with the

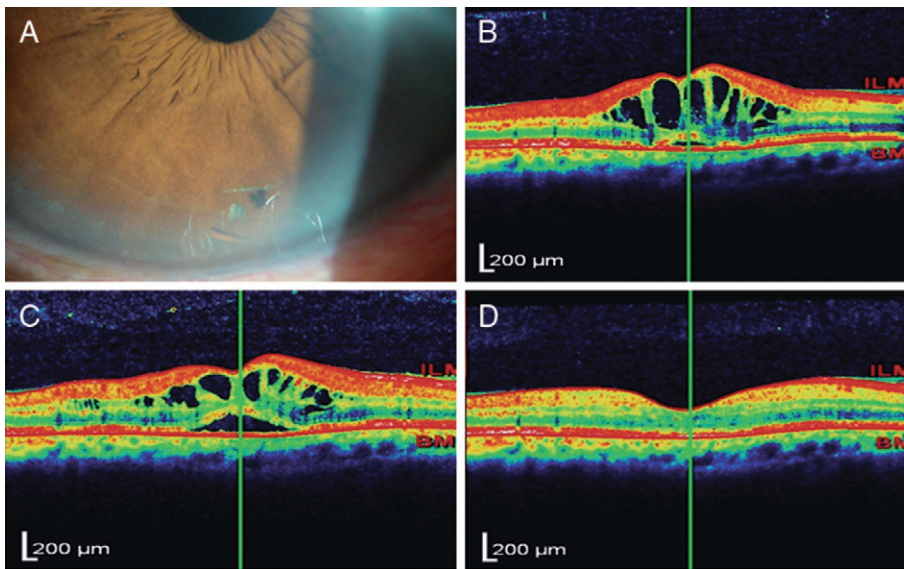


Figure 1A. Intraocular lens haptic perforating iris at 6 o'clock

Figure 1B. Optical coherence tomography image showing cystoid macular oedema before medical treatment

Figure 1C. Cystoid macular oedema persists despite medical treatment before repositioning the intraocular lens

Figure 1D. Dramatic resolution of cystoid macular oedema following haptic repositioning

patient about repositioning the IOL, which was subsequently achieved. The procedure was uncomplicated with the usual post-operative anti-inflammatory drugs and antibiotics. The macular oedema reduced quickly and the visual acuity improved 6/9 at approximately four weeks (Figure 1D).

DISCUSSION

There are several reports demonstrating the resolution of macular oedema following IOL repositioning.^{4,5} In the present case, the iris had been perforated by the lens haptic and we propose that inflammatory mediators released by this process led to the macular oedema, which did not respond to medical treatment. Following the repositioning, OCT imaging of the macula revealed a dramatic improvement.

Several studies have suggested that medical intervention like topical non-

steroidal anti-inflammatory drugs, intravitreal steroid or anti-vascular endothelial growth factor injections for the initial treatment of chronic pseudophakic CME,⁶⁻⁸ however, efforts to treat this patient's macular oedema with intravitreal triamcinolone and bevacizumab were unsuccessful. Although triamcinolone has been shown to reduce macular thickness and improve visual acuity in post-operative CME, its efficacy might be limited by the underlying cause of the CME.⁷ In addition, little is known about the use of intravitreal bevacizumab in post-operative CME. It is likely that uveal irritation caused by the lens haptic created a significant persistent and chronic stimulus for CME, which was too great for a drug therapy.

We believe that this is the first time a case of refractory CME due to iris perforation by the IOL haptic with a rapid resolution has been reported following surgical correction. Although the incidence of clinical

CME following modern cataract surgery is 0.1 to 2.0 per cent, correct IOL placement is desirable to avoid a lot of complications, including CME.⁹

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