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Pseudophakic macular edema and oral acetazolamide: An optical coherence tomography measurable, dose-related response

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PURPOSE. *Although uncommon after phacoemulsification surgery, cystoid macular edema (CME) is an important cause of postoperative reduction of vision after cataract surgery. To demonstrate an optical coherence tomography (OCT) measurable, dose-related response to orally administered acetazolamide in a patient presenting with pseudophakic CME.*

METHODS. *A 76-year-old woman with reduced vision in right eye due to cataract had uneventful phacoemulsification. Surgery was complicated by early onset endophthalmitis that was treated with intravitreal antibiotics with good visual recovery. At 6 months follow-up, she presented with further reduction of vision (0.5 LogMAR) due to CME and a central foveal thickness (CFT) of 587 µm.*

RESULTS. *Acetazolamide treatment was started in combination with topical ketorolac. At a daily dose of 500 mg, CFT and visual acuity were 262 µm and 0.34 LogMAR, respectively. Dose reduction of acetazolamide to 250 mg/day was associated with worsening of the CFT to 335 µm. CFT was subsequently measured at 230 µm on increasing the acetazolamide dose to 500 mg/day and measured 353 µm when acetazolamide dose was halved. CFT was 478 µm when acetazolamide was ceased.*

CONCLUSIONS. *In this report, the authors have shown a dose-related response of pseudophakic CME to oral acetazolamide. This would suggest that the clinical response to oral acetazolamide may be titrated to the extent of CME and monitored by OCT. (Eur J Ophthalmol 2008; 18: 1011-3)*

KEY WORDS. *Acetazolamide, Cataract surgery, Cystoid macular edema, Optical coherence tomography, Pseudophakic*

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INTRODUCTION

Advances in cataract surgical techniques and increasing awareness of the importance of minimizing vitreous loss have resulted in a marked reduction in the incidence of cystoid macular edema (CME) compared to the era of intracapsular cataract surgery (1, 2). Cystoid macular edema is now considered an infrequent complication and the literature suggests that the incidence of pseudophakic CME following uncomplicated phacoemulsification of the lens and implantation of an in-the-bag intraocular lens is 0.6–6% (2). Several studies have considered the effect of pharmacologic and surgical treatments for pseudophakic CME. Topical nonsteroidal anti-inflammatory drugs (NSAIDS), intravitreal steroids, and oral acetazolamide have been shown to be effective treatments. Here, we demonstrate an optical coherence tomography (OCT) measurable, dose-related response to orally administered acetazolamide in a patient presenting with CME after complicated cataract surgery.

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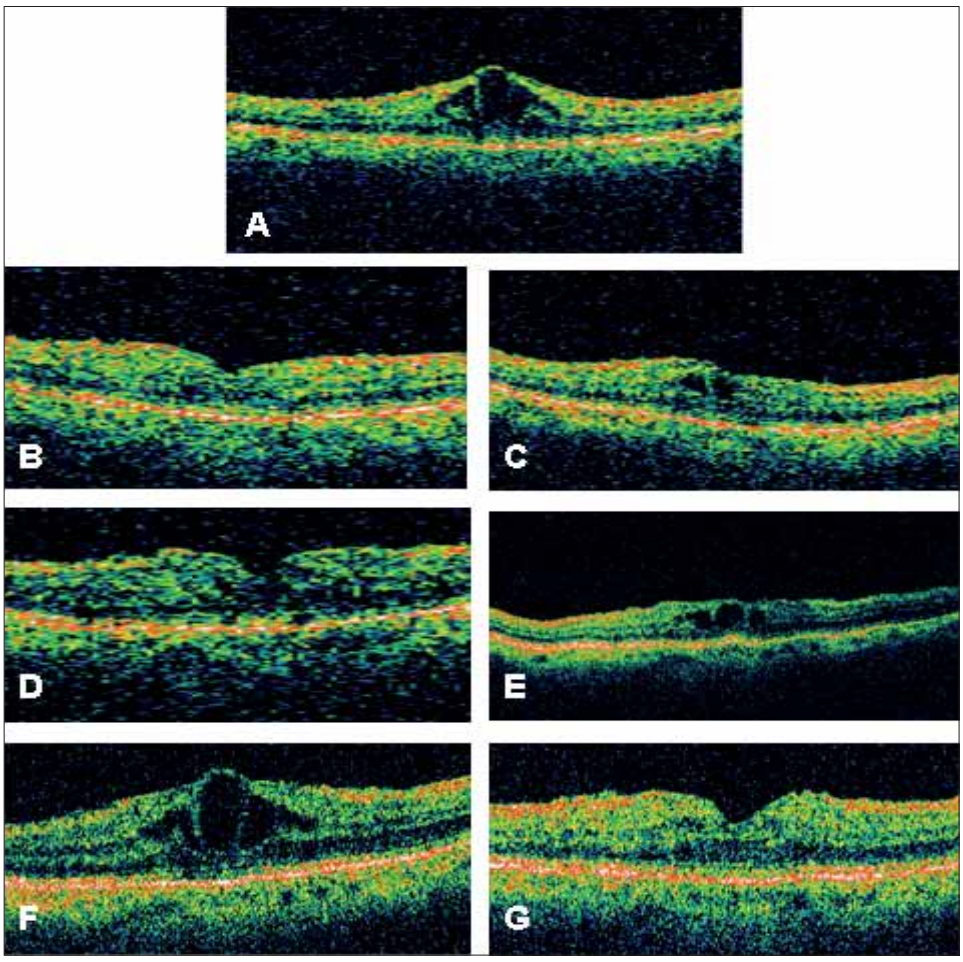

Fig. 1 - *Optical coherence tomography images showing variation in central foveal thickness (CFT) with acetazolamide administration. Presentation 6 months after surgery (no treatment), CFT 587 µm* **(A)***; ketorolac 0.5% qds and oral acetazolamide SR 250 mg bd, CFT 262 µm* **(B)***; ketorolac 0.5% qds and oral acetazolamide SR 250 mg od, CFT 305 µm* **(C)***; ketorolac 0.5% qds and oral acetazolamide SR 250 mg bd, CFT 230 µm* **(D)***; ketorolac 0.5% qds and oral acetazolamide SR 250 mg od, CFT 345 µm* **(E)***; ketorolac 0.5% qds only, CFT 478 µm* **(F)***; orbital floor triamcinolone acetonide 40 mg, CFT 210 µm* **(G)**.

Case report

A 76-year-old woman presented to our service with reduced vision in the right eye. She had no relevant past ocular history and no significant medical history. Right visual acuity (RVA) was 0.1 LogMAR due to lens opacity. Retinal examination was unremarkable.

The patient underwent uneventful right phacoemulsification cataract extraction with posterior chamber in-the-bag lens implant. She then presented 1 day postoperatively with ocular pain, reduced vision, hypopyon, and vitritis. A diagnosis of acute postoperative endophthalmitis was made and she was promptly managed with vitreous tap and intravitreal vancomycin 2 mg/0.1 mL and amikacin 0.4 mg/0.1 mL as well as 25 µg intracameral tissue plasminogen activator. Vitreous cultures yielded no growth after 2 weeks.

The patient made an excellent recovery with complete resolution of ocular pain and improved visual function to 0.4 LogMAR. At the 6-month follow-up visit, she presented with a further reduction in her visual acuity of approximately 2 months to 0.5 logMAR secondary to CME. Findings on OCT demonstrated intraretinal CME and no subretinal fluid. Central foveal thickness (CFT) measured 587±15 µm in the right eye (Fig. 1A).

We commenced treatment with oral acetazolamide 250 mg SR bd and topical ketorolac eyedrops 0.5% qds to the right eye. There was an improvement after 3 weeks with an increase of visual acuity to 0.3 LogMAR and a decrease in the CFT to 262±16 µm (Fig. 1B). Figure 2 shows LogMAR acuity and CFT in response to changes in systemic treatment. Topical ketorolac qds was maintained all through the acetazolamide course. Alternative treatment options were considered given the presence of CME when stopping acetazolamide. Orbital floor triamcinolone injection was thus administered and resulted in a dramatic improvement of RVA to 0.2 LogMAR with a corresponding reduction in CFT to 210±15 µm. The steroid effect, however, was maintained for 6 weeks only (Fig. 1F).

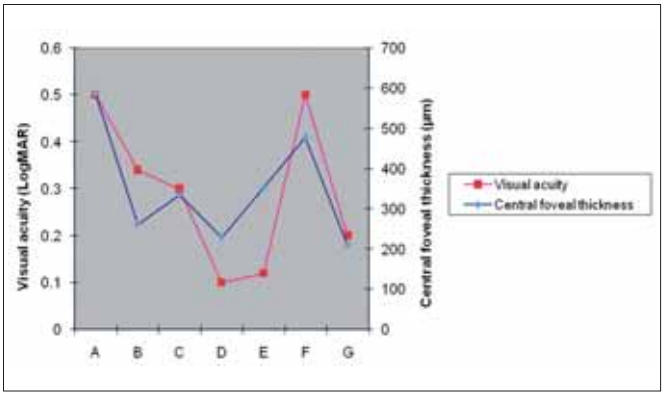

Fig. 2 - *Visual acuity and central foveal thickness changes with cystoid macular edema administered treatment. Presentation 6 months after surgery (no treatment)* **(A)***; ketorolac 0.5% qds and oral acetazolamide SR 250 mg bd* **(B)***; ketorolac 0.5% qds and oral acetazolamide SR 250 mg od* **(C)***; ketorolac 0.5% qds and oral acetazolamide SR 250 mg bd* **(D)***; ketorolac 0.5% qds and oral acetazolamide SR 250 mg od* **(E)***; ketorolac 0.5% qds only* **(F)***; orbital floor triamcinolone acetonide 40 mg* **(G)**.

DISCUSSION

The case presented demonstrates a dose-related response to oral acetazolamide with corresponding visual acuity changes consistent with the degree of severity of CME. Although the peak prevalence of acute CME occurs approximately 6 weeks after cataract surgery, this case is atypical because surgery was complicated by acute postoperative endophthalmitis. It is therefore possible that low grade CME had been present but was masked by the coexisting intraocular infection.

CME following cataract surgery results from the maldistribution of intravascular fluid within the macula. Leakage of intravascular contents from the dilated perifoveal capillaries initially causes thickening of the macula, which may progress to cystoid expansions within the outer plexiform (Henle's) layer and inner nuclear layer of the retina (2). Most investigators agree that inflammation is the major etiologic factor with prostaglandin release being implicated as the possible mediator of inflammation (3). In a patient with endophthalmitis postcataract surgery, an alternative explanation would be the compromised retinal pigment epithelium function from toxin release. However, the documented improvement in visual acuity and CFT with acetazolamide would suggest that retinal edema in this case is secondary to cataract surgery rather than being uveitic in origin (4). This is also supported by the absence of intraocular inflammation throughout the postoperative course from about 2 months postoperatively, and the response to acetazolamide without the addition of local or systemic corticosteroid therapy.

In patients with NSAID-resistant CME, small studies have demonstrated the effectiveness of acetazolamide with partial or complete resolution of macular edema and improvement of visual function (2, 5). Acetazolamide is thought to improve the pumping function of the retinal pigment epithelium, thereby reducing intraretinal fluid and improving vision (6). Acetazolamide may also have an effect on retinal capillaries (2). However, side effects such as paraesthesiae, electrolyte disturbance, and psychological disturbances limit the therapeutic application of this treatment. In this report, we have shown a dose-related response of pseudophakic CME to oral acetazolamide. These results suggest that the dose of oral acetazolamide may be titrated to the extent of CME and monitored by timely OCT examinations.

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