Case Report

Selective Laser Trabeculoplasty Complicated by Cystoid Macular Edema: Report of Two Cases

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Abstract

Purpose: Selective laser trabeculoplasty, a relatively novel treatment for open angle glaucoma, is frequently associated with mild post-operative intraocular inflammation.

Methods: We report two uncommon cases of cystoid macular edema within a few weeks of routine selective laser trabeculoplasty.

Results: Visual acuities and macular thicknesses of the two cases returned to baseline after medical treatment, but in one case, the cystoid macular edema persisted for months.

Conclusion: Cystoid macular edema after selective laser trabeculoplasty is fortunately a rare complication, but it might be more common in patients with predisposing factors, and it can be resistant to treatment. (***Eye Science 2012; 27:193-197***)

Keywords: selective laser trabeculoplasty; cystoid macular edema; glaucoma

Introduction

Selective laser trabeculoplasty (SLT) is a relatively new treatment for open angle glaucoma. It uses a Q-switched 532-nm, frequency-doubled Nd:YAG laser that emits a short pulse (3 nanoseconds) to photolyze pigmented cells in the trabecular meshwork selectively[1]. It is believed to induce much less collateral thermal and mechanical damage than previous argon laser trabeculoplasty (ALT) approaches[1]. In an early study[2], anterior chamber inflammation was demonstrated in 83% of post-SLT eyes (180 degrees of treatment), but all resolved within 5 days. Despite the high frequency of uveitis, the only report of cystoid macular edema (CME) after SLT was in a patient with CME from complicated cataract surgery who subsequently developed steroid-response intraocular pressure (IOP) elevation that was treated with 180-degree SLT[3]. Recurrent CME was cured within three weeks with medical treatment. Although this case was relatively mild and cannot be definitively attributed to the laser treatment, we report two cases of post-SLT CME, one of which was highly resistant to treatment for several months.

Case reports

The relevant clinical findings of case 1 and case 2 are summarized in Table 1.

Case 1

A 79-year old white woman with primary open angle glaucoma (POAG) experienced progressive losses on the Humphrey visual field despite maximal medical treatment. She had had an untreated superotemporal branch retinal vein occlusion (BRVO) in the left eye without macular edema since 2005, and in 2009 had undergone uneventful phacoemulsification with intraocular lens implant in the left eye. SLT was performed 360 degrees in both eyes, one week apart. A month later, best corrected visual acuity (BCVA) of the left eye dropped to 20/50. The slit lamp examination was significant for the lack of cells in the anterior chamber. Dilated fundus examination revealed a few microaneurysms and telangiectasias near the fovea, which were essential-

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Table 1 Summary of the two cases with cystoid macular edema after selective laser trabeculoplasty

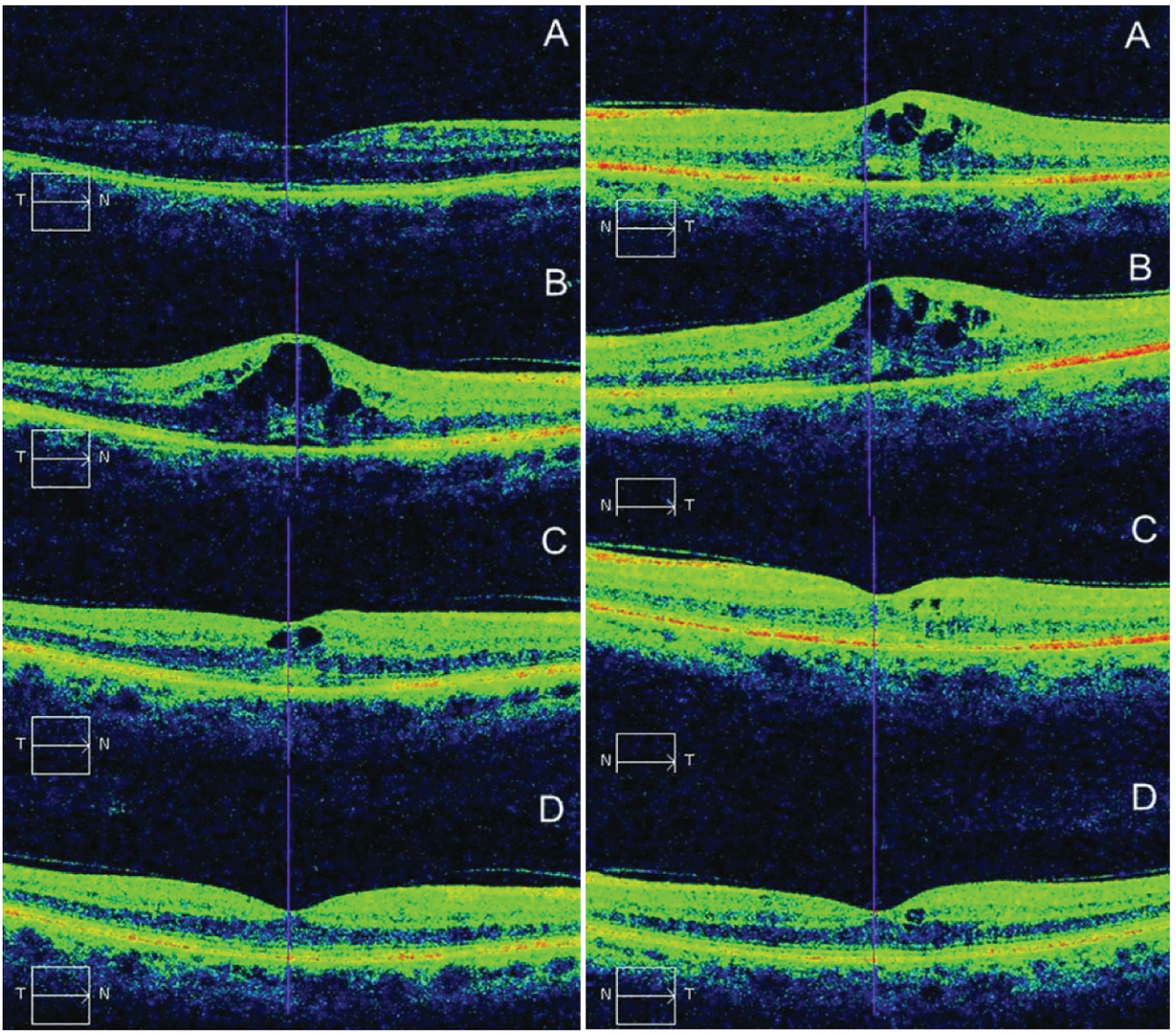
		Case 1	Case 2	
Age (year)		79	68	
Sex		Female	Female	
Possible predisposing risk factor for CME		BRVO OS in 8/2005 Pseudophakia OS since 3/2009	Diabetes mellitus Type II since 2005 Pseudophakia OU since 8/2009	
Medication treatment before SLT		0.5% Timolol/2% Dorzolamide BID 0.1% Brimonidine BID 0.004% Travoprost QN	0.5% Timolol/2% Dorzolamide BID	
Eye		OS	OD	OS
SLT laser parameters		0.9 mJ, 93 applications	1.1 mJ, 108 applications	1.1 mJ, 105 applications
CST by OCT (microns)	After SLT(maximum thickness)	421	537	514
	After SLT(last visit)	230	299	305
BCVA	Baseline	20/25	20/20	20/20
	After SLT(first post-op visit)	20/50	20/150	20/150
	After SLT(last visit)	20/25	20/20	20/20
IOP (mmHg)	Pre-SLT	16	15	15
	Post-SLT (last visit)	12 (no change in meds)	16 (meds discontinued)	16 (meds discontinued)

CME: Cystoid macular edema; SLT: Selective laser trabeculoplasty; BRVO: Branch retinal vein occlusion; CST: Central subfield thickness; OCT: Optical coherence tomography; BCVA: Best-corrected visual acuity; IOP: Intraocular pressure; OD: Right eye; OS: left eye; BID: Twice a day; QID: Four times a day; QN: Every night

ly unchanged since the onset of BRVO. However, there was CME (central subfield thickness, CST, 421 microns), as measured by Cirrus optical coherence tomography (OCT, Carl Zeiss Meditec, Dublin, CA). The phakic right eye´s macula was normal. The patient was started on ketorolac 0.5% twice daily in the left eye. A month later, her CST and BCVA had returned to baseline.

Case 2

A 68-year old diabetic white woman with POAG on maximal tolerated medical therapy was found to have progressive nerve fiber layer loss on the Cirrus OCT. In 2009, she underwent uneventful phacoemulsification with intraocular lens implantation in both eyes. The patient elected to undergo SLT in both eyes. Preoperatively she had scattered microaneurysms in the periphery but no pathology in the maculae. SLT was performed in the left eye and then in the right eye one week later. A week after SLT was performed in the second eye, she presented with pain, tearing, photophobia, and decreased vision in both eyes. Examination revealed irregular corneal epithelium, mild corneal edema, 1-2+ cells in the anterior chamber, and decreased foveal reflex in both eyes. OCT revealed cystoid macular edema in the left eye (CST 416 microns), but was unremarkable in the right eye. Prednisolone acetate 1% and preservative-free lubricant eye drops were initiated four times a day (QID) in both eyes, and ketorolac 0.5% QID was initiated in the left eye. Glaucoma drops were discontinued because of superficial keratitis. Another week later, her uncorrected visual acuity was 20/125 in the right eye and 20/70 in the left eye. Anterior chamber cells had disappeared in both eyes. However, OCT revealed cystoid edema in both maculae (CST 537 microns in the right eye, 514 microns in the left eye). The patient was instructed to use prednisolone acetate 1% and ketorolac 0.5% QID in both eyes. Three weeks later, CME had significantly improved in the right eye but cystoid spaces were clearly evident in the left eye (CST 315 microns in the right eye, 364 microns in the left eye). The patient was followed up every other week with slow incremental improvements noted at each visit. Approximately 12 weeks after the SLT procedures, the macular edema finally resolved in the right eye, but small cystoid spaces were still discernible on OCT in the left eye. Mild punctate epithelial erosions persisted in both eyes. At this point, both prednisolone acetate and ketorolac were



1–1 1–2

Figure 1 The OCT changes of macula in both eyes of Case 2

1–1. The right eye of Case 2

A: 1 week after SLT, normal macula appearance (CST = 295 microns)

B: 1 month after SLT, CME with localized neuroepithelial layer detachment (CST = 570 microns)

C: 6 weeks after SLT, CME improved with neuroepithelial layer reattached (CST = 389 microns)

D: 8 weeks after SLT, CME resolved completely (CST = 304 microns)

1–2. The left eye of Case 2

A: 2 weeks after SLT, CME with local neuroepithelial layer detachment (CST = 416 microns)

B: 1 month after SLT, CME worsened (CST = 514 microns)

C: 7 weeks after SLT, CME improved with neuroepithelial layer reattached (CST = 324 microns)

D: 18 weeks after SLT, mild CME remained (CST = 305 microns)

discontinued to allow the cornea to recover, leaving only lubricant eye drops QID in both eyes. Four more weeks later, almost four months after the original procedure, BCVA had finally returned to baseline despite very mild residual CME on OCT in the left eye. Corneal epithelial pathology had completely resolved. No other pathological findings, except decreased foveal reflex, were evident in the maculae. Some illustrative macular OCT images are shown in Figure 1–1 (right eye) and Figure 1–2 (left eye).

Discussion

SLT is thought to have equivalent efficacy compared to ALT with less trabecular meshwork damage[1]. However, the lower energy imparted during treatment can still induce significant intraocular inflammation, including one case associated with postoperative choroidal effusion[4]. The larger spot size of SLT may also cause a larger area of damage, affecting not just the pigmented cells in the trabecular meshwork but also surrounding pigmented tissues[2].

The mechanism of SLT remains controversial, but one theory holds that the laser stimulates macrophage activity in the trabecular meshwork to remodel the extracellular matrix, resulting in increased aqueous outflow. Crossover effects in the other eye (10% lower IOP in the contralateral eye) support this theory[1]. SLT has been shown to cause release of chemotactic and vasoactive agents, including various cytokines.5 These cytokines and the activation of the immune system may play a role in the induction of intraocular inflammation, which can lead to CME.

The association between SLT and CME may not be obvious, as illustrated by our first case in which identifying the cause of the cystoid edema is complicated by pre-existing BRVO and the use of prostaglandin analogs. Similar to the other case reported in the literature[3], our patient may be pre-disposed to the development of macular edema even with relatively mild intraocular inflammation. However, our second case, in which the patient did not develop CME from cataract surgeries two and a half years earlier, the only other risk factor besides the SLT treatment was relatively mild nonproliferative diabetic retinopathy, which did not appear to affect the macula on examination. Unfortunately, we were not able to obtain fluorescein angiography on the patient, which could have provided additional information about the macular microvasculature. Curiously, despite resolution of anterior chamber inflammation, the CME persisted for several months. In our experience, our treatment protocol has a low incidence of CME. The occurrence in both eyes in our second patient suggests that genetic or other patient–specific factors may have pre–disposed her to CME.

Treatment of CME usually targets intraocular inflammation. However, in the setting of glaucoma patients necessitating SLT, there is the theoretical risk that suppressing intraocular immune activation may negate the IOP-lowering benefits of SLT. Nonsteroidal anti-inflammatory drugs (NSAIDs) may also damage the corneal epithelium, which is inevitably affected by the contact lens used during laser treatment as well as by glaucoma medications. An additional concern is steroid-induced IOP elevation. While our first patient responded quickly to an NSAID, the second patient was treated for several months. Fortunately, despite frequent follow-up, we did not detect higher IOP in this patient. If IOP had occurred, carbonic anhydrate inhibitors such as acetazolamide and dorzolamide, which may suppress CME with the dual benefit of lowering IOP, would likely have been good alternatives.

In summary, selective laser trabeculoplasty is effective in lowering IOP, but it may rarely be associated with cystoid macular edema, particularly in patients who harbor pre-disposing factors, such as otherwise non-significant retinal vascular disease.

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