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CASE REPORT

Cystoid macular edema following selective laser trabeculoplasty in a patient with ocular hypertension

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Abstract
Purpose Presentation of a case with ocular hypertension who developed cystoid macular edema (CME) following selective laser trabeculoplasty (SLT).
Case report A 62-year-old male patient who had undergone phacoemulsification surgery for traumatic cataract 9 years ago was on ocular hypertension treatment for the last 5 years. Due to uncontrolled intraocular pressure and side effect of medications, a 360° SLT was performed. The patient returned with decrased visual acuity 1 week after the procedure. There was CME in the treated eye. It healed completely after topical prednisolone acetate QID and nepafenac QID for 1 week, and no recurrence was observed during follow-ups.
Conclusion Cystoid macular edema may occur following SLT treatment in patients with previous traumatic cataract surgery which responds to topical treatment.

Keywords Selective laser trabeculoplasty · Cystoid macular edema · Ocular hypertension · Cataract surgery

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Background

Selective laser trabeculoplasty (SLT) has become an established treatment to decrease intraocular pressure (IOP) in ocular hypertension (OHT) and glaucoma patients. By targeting pigmented trabecular meshwork cells, it causes selective photolysis and acts mostly on cellular level with minimal or no structural damage [1–3].

Most complications of SLT are usually mild, transient and self-limiting, presumably related to anterior chamber inflammation [4]. Despite high incidence of anterior chamber inflammation, cystoid macular edema (CME) following SLT is a very rare occurrence reported in only 4 cases. Possible predisposing risk factors for CME were reported as previous complicated cataract surgery, diabetes mellitus and branch retinal vein occlusion [5–7].

Here, we report a case of CME following SLT treatment in a patient who had previous traumatic cataract extraction after closed globe injury.

Subject and observations

A 62-year-old male patient applied to the outpatient clinic for stinging and redness in the right eye. He had no systemic diseases like diabetes mellitus or systemic hypertension. He was on brimonidine 0.15% (Alphagan-P®) BID, dorzolamide hydrochloride 2% and

timolol maleate 0.5% fixed combination (Dorzotim®) BID treatment for ocular hypertension for 5 years. He had a blunt trauma caused by a piece of wood and was operated for traumatic cataract.

On examination, spherical equivalent was − 1.00 OD and + 0.375 OS, and corrected distance visual acuity (CDVA) was 6/6 in both eyes. IOP was 26 and 18 mmHg, respectively. Gonioscopy revealed grade 4 open angle in Schaffer's grading with increased pigmentation and angle recession at inferior nasal quadrant (Fig. 1). Conjunctival hyperemia and corneal diffuse punctate staining were observed in the right eye. There was corneal lamellar scarring at 9 o'clock. The eye was pseudophakic and posterior capsule was intact. Posterior segment examination revealed normal status. Optical coherence tomography (Retinascan Advanced RS-3000, NIDEK) revealed normal retinal nerve fiber analysis. Visual field testing was also normal.

Due to uncontrolled IOP and side effect of anti-glaucomatous medications, SLT was applied to right eye. The patient returned with decreased vision and increased redness 1 week after SLT treatment. Visual acuity was decreased to 6/19. Conjunctival injection was more than pre-SLT level and + 2 cells were detected in the anterior chamber. Diagnosis of CME was confirmed with fundus fluorescein angiography (FFA) and OCT imaging (Fig. 2a, b). Prednisolone acetate 1% (Pred forte®) QID and nepafenac 0.1% (Nevanac®) QID were initiated. Visual acuity was increased to 6/6 at 1 week, and no recurrence was observed at OCT scans during the treatment course (Fig. 2c).

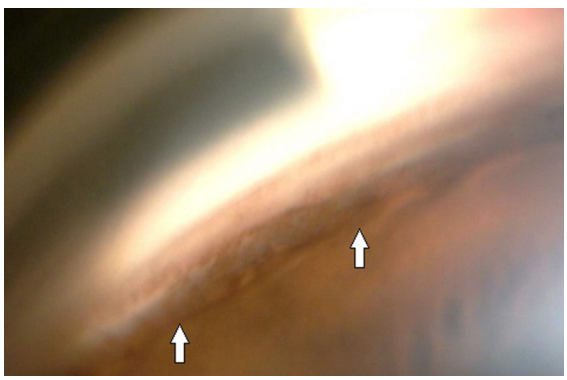

Fig. 1 Grade 4 open angle in Shaffer's grading with increased pigmentation and widening of ciliary body band (angle recession) (between arrows) at inferior nasal quadrant

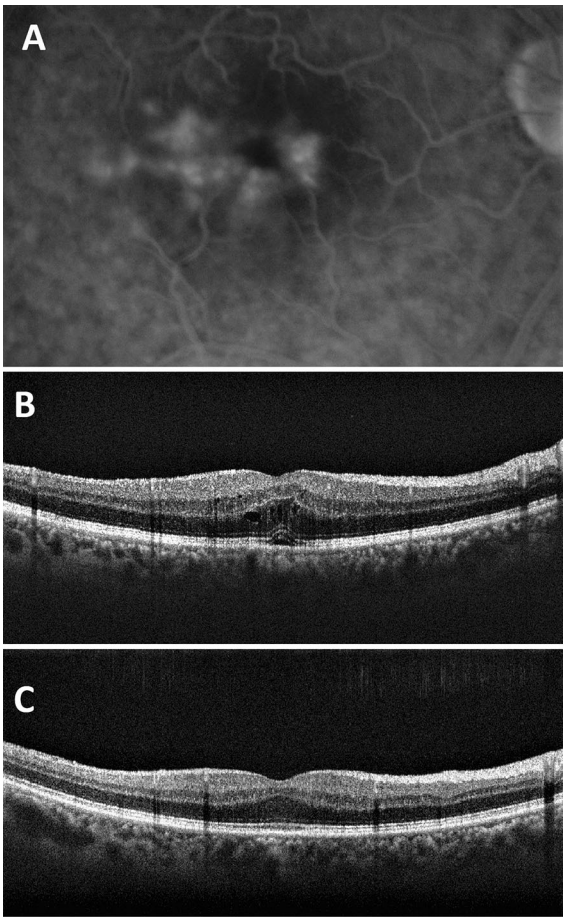

Fig. 2 **a** FFA: Coalescence of leaking points forming flower-petal pattern of cystoid macular edema. **b** OCT: Cystic areas of low reflectivity in outer nuclear, outer plexiform and inner nuclear layers with minimal subretinal fluid. **c** OCT: No cystic space and subretinal fluid was observed 1 week after topical treatment

Conclusions

The mechanism of SLT is not so clear, but one theory holds that it stimulates macrophage activity in trabecular meshwork which remodels extracellular matrix and increases aqueous outflow. Crossover effect, that is 10% lowering of IOP in contralateral eye, supports this theory [8]. Release of chemotactic and vasoactive agents like cytokines have been reported after SLT application [9]. These cytokines and activation of immune system may play a role in intraocular inflammation which may lead to previously reported complications like choroidal effusion, subretinal fluid accumulation and CME [5–7, 10, 11].

Wechsler et al. reported a case of CME after SLT in a patient who received a complicated cataract surgery with ruptured posterior capsule. Postoperative CME was treated with topical dexamethasone which led to increased IOP. SLT was performed, and CME recurred in the same eye [5]. SLT might have caused increased access of inflammatory mediators to the posterior segment due to ruptured posterior capsule. Patients with CME after complicated cataract surgery may also be at risk of CME recurrence. Wu et al. reported two cases of CME complicating SLT. The first case had preexisting branch retinal vein occlusion and used prostaglandin analog. The patient had no history of macular edema until SLT was applied, even after phacoemulsification surgery which was done 3 years before SLT. The second case had CME in both eyes, and the only risk factor was mild non-proliferative diabetic retinopathy without diabetic maculopathy. The patient had uncomplicated cataract surgery about 3 years before SLT treatment. Ha et al. [6] reported a case of CME exacerbated by SLT for steroid-induced ocular hypertension in a diabetic patient with moderate non-proliferative diabetic retinopathy [7]. Bilateral subretinal fluid (SRF) accumulation was reported for the first time by Philis and Bourke in a patient with open-angle glaucoma 24 h after bilateral inferior 180-degree SLT application. They found no specific predisposition in this patient and postulated that anterior segment inflammation secondary to SLT resulted in posterior segment fluid accumulation due to inflammatory upregulation [11].

It was not very clear whether OHT was a consequence of blunt ocular injury or cataract surgery or neither of them. There was approximately only 1 h angle recession, and it is known that the greater the number of clock hours of angle recession, the more the likelihood of developing elevated IOP. Although traumatic cataract extraction was done 9 years ago, there were no intra- or postoperative complications and OHT was diagnosed 4 years after. Therefore, increased IOP may not be a complication of previous cataract surgery.

The case had OHT, history of traumatic cataract extraction and no CME history. Possible zonular dehiscence may facilitate access of inflammatory mediators from anterior to posterior segment, and this might have led to increased vascular permeability and CME. The patient whom Wechsler et al. reported had CME history. Only case without previous CME had mild non-proliferative diabetic retinopathy as predisposing factor. Whether previous CME is present or not, patients with retinal vascular disease and history of complicated and traumatic cataract surgery seem to be under risk of CME following SLT.

The case reported by Philis and Bourke developed SRF without developing CME [11]. Inflammatory mediators should cause intraretinal fluid before subretinal fluid accumulation if passage of inflammatory mediators from anterior segment to posterior segment is the only causative factor. Therefore, additional mechanisms may be responsible for SRF and CME occurrence after SLT treatment.

CME after SLT may be observed in OHT patients with a history of ocular trauma and cataract extraction which resolves completely after topical treatment. Pretreatment with non-steroidal anti-inflammatory agents before SLT application may be considered in patients who had predisposing factors for CME development.

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