Argon-laser iridoplasty in the management of uveitis-induced acute angle-closure glaucoma

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INTRODUCTION

Glaucoma can be a serious complication of uveitis and its management is a challenge (1, 2). The angle can be closed (completely or partially) or open, each due to different mechanisms, and glaucoma often results from more than one of these. The angle closure can arise from 1) pupillary block secondary to posterior synechiae or cataract, 2) peripheral anterior synechiae (PAS), and 3) forward rotation of the ciliary body. We present a case of unilateral acute angle-closure glaucoma occurring within 15 days of initiating therapy with 1% prednisolone acetate for recurrent uveitis.

METHODS AND RESULTS

On October 30, 2007, a 46-year-old Caucasian man experienced sudden visual loss (right eye [OD]) and acute ocular pain and was put on a regimen of hourly 1% prednisolone acetate, scopolamine, and intravenous methylprednisolone by his general practitioner. The patient had a history of ipsilateral recurrent uveitis secondary to ankylosing spondylitis, treated each time with 1% prednisolone acetate three times daily, the last episode being 2 weeks previously. He was referred by his ophthalmologist with an intraocular pressure (IOP) of 65 mmHg and severe ocular pain in his right eye. In our hospital, the visual acuity (VA) was hand movement, IOP 62 mmHg, and pachymetry 568 µm (OD). Anterior segment examination showed an injected eye with corneal edema, markedly shallow peripheral anterior chamber (AC), and cataract (Fig. 1). Neither fundus examination nor gonioscopy was possible. The ophthalmic status of the left eye was within normal limits.

The diagnosis of acute angle closure secondary to pupillary block from related posterior synechiae (seclusio pupillae) was made. Thirty minutes after treatment with systemic analgesia and acetazolamide (500 mg intravenously), Cosopt (Merck, timolol + dorzolamide), 1% prednisolone acetate, NaCl 5%, scopolamine, and acetazolamide, the cornea cleared just enough to perform peripheral laser iridotomy (LPI) at the 12 and 1 o’clock position. The angle opened slightly at 6 and to a lesser extent at 12 o’clock. However, there was persistent marked iridocorneal contact and IOP was 58 mmHg (Fig. 2).
Argon laser iridoplasty (ALPI) was then attempted starting at 6 o’clock in the mid-peripheral iris, moving progressively to the periphery as the AC widened (39 spots, 0.5 sec, 500 µm, with an energy level lowered from 260 mW to 160 mW) (Fig. 3). The IOP dropped to 48 mmHg immediately after ALPI and to 21 mmHg after 1 hour on 1% prednisolone acetate Cosopt, Alphagan (Allergan, brimonidine 0.1%), phenylephrine, and scopolamine. Three hours later, IOP had decreased to 3 mmHg (Fig. 4). The next day, VA was 0.15, IOP 13 mmHg, and after another 24 hours, 0.3 and 9 mmHg, respectively, on prednisolone 1%, scopolamine, and phenylephrine. The AC was deep but showed a marked inflammatory reaction; gonioscopy showed Shaffer grade 3. Three months later, VA was 0.4 and IOP remained stable at 21 mmHg without any ocular hypotensive treatment.
CONCLUSIONS

To our knowledge, there have been no previous reports on the effect of ALPI on uveitic glaucoma. A PubMed search for keywords “acute angle-closure glaucoma”, “uveitis” and “laser iridoplasty” yielded no relevant publications. The management of acute angle closure usually relies on systemic hypotensive treatment followed by LPI (3). LPI, however, only eliminates pupillary block and lowers IOP if more than ¼ of the angle circumference is open. In uveitic angle closure, there is a high risk for recurrence, which warrants regular follow-up with gonioscopy.

In this patient, there was still marked convexity of the iris with marked iridocorneal touch from the periphery to the midperiphery and 360 degrees seclusio pupillae after two patent iridotomies and 24 hours of unsuccessful mydriatic treatment. At least partial relief of the pupillary block after LPI was evident by some deepening of the anterior chamber in the midperiphery, especially at the 6 o’clock position, 180º away from the iridotomies (Fig. 2). This allowed ALPI to be started there; it was then possible to continue it gradually more peripherally as the iris was progressively moving away from the cornea just peripheral to the LPI impacts. The relief of peripheral appositional closure or rather early peripheral synechiae was the result of ALPI although its effects were potentiated by prior LPI and use of mydriatic agents. The cornea cleared as a continuum as the IOP decreased.

Argon laser peripheral iridoplasty has recently been proposed as an efficient first-line intervention in cases of acute angle closure glaucoma (4). In this case, it not only allowed rapid reduction of IOP but also prevented permanent marked peripheral anterior synechiae. Although emergency surgery was prevented, which is particularly important in active uveitis cases, ALPI enhanced the inflammatory reaction; therefore, the concomitant use of systemic immunosuppression or thromboplastin activators could be argued.

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