Recurrent Angle Closure Glaucoma Following A Patent 75-Micron Laser Iridotomy: A Case Report

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SUMMARY

Laser iridotomy is becoming increasingly popular as an effective alternative therapy to surgical iridectomy; however, we report a case of recurrent angle closure glaucoma following laser treatment that produced a patent iridotomy. Possible mechanisms for the failure and its implications are discussed.

INTRODUCTION

Since Meyer-Schwickerath first reported the use of photocoagulation to produce iridotomies in 1956, a search for the ideal instrument and technique, with the fewest complications, has been underway. Xenon, Ruby, and Argon have been used at various energy levels, exposure times, and spot sizes to produce iridotomies. The recent literature suggests a trend toward smaller spots, lower energy, and multiple shorter exposures with the Argon laser to produce successful results with a minimum of complications. Using these techniques, we have performed twelve iridotomies, obtaining a patent opening in one sitting in all patients. Proof of patency was determined in each case by gonioscopic deepening of the angle, anterior lens capsule visibility through the iridotomy, and a clear red reflex through the iridotomy. We have had no corneal burns, no lens opacities, and no retinal burns. One of our patients, however, returned in the early post-treatment period with a recurrent acute angle closure attack, but with a patent 75-micron iridotomy.

CASE REPORT

A 59-year-old white female was seen in the University of Arkansas for Medical Sciences (UAMS) Eye Clinic on November 12, 1981, with a one-week history of headache, nausea, vomiting, and blurred vision in the left eye. Her past medical and ocular history were unremarkable.

Examination revealed a visual acuity of 20/20 OD, 20/50 OS, with a manifest refraction of +1.75 + 1.00 X 180 OU. The pupillary reactions were 3 mm and brisk OS and 4 mm and sluggish OS, without afferent defect. Slit-lamp examination of the anterior segment was normal in the right eye, but there was 2+ injection, 1+ corneal edema, and 1+ anterior chamber flare in the left eye. Tension by applanation (Tapp) was 16 mm Hg OD and 54 mm Hg OS. Gonioscopic examination revealed a closed angle 360° OS, and slit to Grade I angles OD. The diagnosis of acute angle closure glaucoma was made in the OS, and treatment with topical 0.5% timolol maleate, 4% pilocarpine, oral glycerol, and oral acetazolamide was instituted. In addition, intermittent corneal pressure with a four mirror Zeiss gonioscopic lens was used. After one hour, repeat intraocular pressure (IOP) was 10 mm Hg. The patient was sent home on 4% pilocarpine OU q.i.d., prednisolone acetate OS q.i.d., and acetazolamide 250 mg q.i.d. On 24-hour follow-up, the visual acuity was 20/20 OU and Tapp 15/11. Diamox was stopped, but the pilocarpine and prednisolone acetate were continued and laser iridotomy was scheduled.

On December 17, 1981, laser iridotomy was performed in the left eye without difficulty using Mandelkon’s technique: After topical proparacaine, the patient was seated at the Coherent Argon laser 900, and the Abraham anterior segment lens was placed on the eye. A ring of six burns was placed at the junction of the outer 1/3 with the inner 2/3 of the iris at 10:00 o’clock, using 100 micron spots, 600 mw, and 0.02 seconds. Penetration and enlargement to a 75-micron iridotomy were performed without difficulty using 324 50 micron spots at 800 to 1200 mw and 0.02 seconds. Successful iridotomy was determined by gonioscopic deepening of the chamber and angle, the anterior lens capsule was visible through the iridotomy, and a good red reflex through the iridotomy was obtained on retroillumination. The patient’s IOP was 14 mm Hg one hour after
treatment, and she was sent home on no medications and asked to return in one week for follow-up and for a laser iridotomy on the right eye.

Five days later, she returned to the Eye Clinic complaining of an eight-hour history of pain and blurred vision in her previously treated left eye. Examination revealed a visual acuity of 20/60 and a mid-fixed pupil. There was 1+ to 2+ epithelial edema but a good red reflex, and the anterior lens capsule could be seen through a patent iridotomy (Figure). Intraocular pressure was 16 mm Hg OD, 54 mm Hg OS. Her angle in the left eye was closed on gonioscopic examination. The diagnosis of angle closure glaucoma was made again, and it was treated in the same manner as the first attack with good response. After one hour the IOP was 17 mm Hg. The patient was discharged on pilocarpine 4% OU q.i.d., timolol maleate 5% OS b.i.d., acetazolamide 250 mg q.i.d., and prednisolone acetate OS q.i.d., and asked to return the next day. On December 23, 1981, the left anterior segment was again quiet with a visual acuity of 20/20 and an IOP of 12 mm Hg. The Argon laser was used to enlarge the iridotomy to approximately 125 micron using 207 spots of 50 micron diameter, 0.02 seconds, and 1000 to 1300 mW. Post-laser gonioscopy again revealed an open angle without synechia. Her postoperative course has been without additional angle closure attacks, and on February 3, 1982, the patient underwent a successful laser iridotomy in the right eye.

**DISCUSSION**

A review of the literature revealed only one reported case of angle closure glaucoma following laser iridotomy with one case of angle closure following unsuccessful laser iridotomy. Pollack has had no angle closure attacks in over 1200 iridotomy patients (Personal Communication, March 13, 1982). There are a number of possible reasons why laser iridotomy might fail. They include plateau iris, non-patent opening, pigment avalanche occluding a patent opening, posterior synechiae surrounding the iridotomy, and a patent iridotomy too small for adequate flow of aqueous.

Plateau iris may be associated with angle closure but without significant pupillary block. The abnormality involves an anterior insertion of the iris on the ciliary body, causing trabecular occlusion during pupillary dilation with subsequent angle closure glaucoma. Distinguishing characteristics include a flat iris plane, running directly toward Schwalbe’s line; and a central chamber deeper than that seen with the usual narrow-angled, hyperopic eye. Plateau iris should be suspected in any case of angle closure glaucoma following patent iridectomy. Because of our patient’s shallow central anterior chamber and deepening of the chamber and angle post-iridotomy, we felt that our case was not consistent with a plateau iris diagnosis.

We used three criteria to determine patency of our iridotomies after treatment: red reflex seen through the iridotomy on retroillumination; gonioscopic deepening of the anterior chamber and angle, and, most importantly, direct visualization of the anterior lens capsule through the iridotomy. All of our patients have met these criteria at the end of one treatment with the Argon laser.

The pigment avalanche, with partial or complete occlusion of the iridotomy, usually occurs within the first one or two weeks and is rarely seen after six weeks post-laser. Occclusion can occur from “settling” of dispersed pigment into a small iridotomy, and/or proliferation of posterior pigment epithelium with bridging of the iridotomy. This situation may require additional low levels of laser energy to re-establish an adequate opening. Because of the slit-lamp appearance of our iridotomy during the angle closure attack, we felt that pigmentary and fibrous occlusion of the iridotomy had not occurred. Admittedly, this can be a very difficult determination since some of the fine posterior iridotomy membranes are nearly transparent.

The hazard of posterior synechia around the iridotomy site is a reported complication, and could definitely play a role in the failure of the iridotomy to function. Because we simply enlarged the primary iridotomy after angle closure without placing a second iridotomy, we felt that posterior synechiae were not causing an occlusion. It is possible that the second treatment could have lysed unseen posterior synechiae, but the iridotomy was enlarged only 25 to 50 micron. Synechiae wide enough to occlude the opening and precipitate angle closure would surely extend wider than just the treated area. Mandelkorn reported one case of angle closure due to swelling of the surrounding iris stroma following formation of a 50-micron iridotomy. Our case could have been due to this mechanism, though no significant stromal swelling was appreciated, and the iridotomy was placed in the outer third of the iris avoiding anterior capsule contact and iridotomy “aperture block.”

It has been postulated that an iridotomy need only be large enough to allow the passage of H2O molecules to the anterior chamber. However, one might argue that a patient with narrow angles, in the prone position or after mydriasis, could exceed the flow capabilities of such a tiny aperture and proceed to an angle closure attack. What, then, is the critical minimal diameter necessary for an iridotomy to ensure adequate posterior to anterior aqueous flow under all conditions? Pollack feels that the larger iridotomies are...
less likely to fill in with pigment. He routinely produces two iridotomies in his narrow angle patients. (Personal communication, March 13, 1982) Quigley states that iridotomies of 250 micron should be adequate, but if only 50 to 100 micron holes are produced, it may be advisable to place a second iridotomy to improve chances of a functional result. Wheeler theorized that an aperture of 15 micron was sufficient to permit full flow of aqueous humor through the human iris, but he stated that ultimately the size may be limited by the tendency to close as the pupil dilates. He produced 200 micron iridotomy in rabbits and felt that these were much larger than required for pressure equalization on both sides of the iris. Our patient had a 75-micron iridotomy and was in between the above 15-micron and 250-micron diameters. It is our feeling that this 75-micron diameter was too small to allow adequate flow and thus eventually led to a recurrent attack of acute angle closure glaucoma.

CONCLUSION

It is presently unknown how large a laser iridotomy must be in order to ensure adequate flow to prevent an acute angle closure attack. Other possible mechanisms for angle closure, including plateau iris, non-patent iridotomy, early pigment proliferation, and posterior synechiae, are discussed. None of these were felt to be responsible for our patient’s angle closure. We conclude that there is a minimal diameter iridotomy required to maintain anterior-posterior pressure equalization. A 75-micron diameter opening is apparently not satisfactory for certain eyes, since we had a failure with this size opening. It is recommended that at least 150 to 200 micron openings be made with the laser until further research elucidates the critical minimal size.

REFERENCES