Latanoprost for the prevention of ocular hypertension after neodymium:yttrium-aluminum-garnet laser posterior capsulotomy

C. F. Chung,1 MRCS (Ed), Jimmy S. M. Lai,1 FRCOphth, Dennis S. C. Lam,2 FRCOphth
1 Department of Ophthalmology, United Christian Hospital, Hong Kong, China.
2 Department of Ophthalmology & Visual Sciences, The Chinese University of Hong Kong, Hong Kong Eye Hospital, Hong Kong, China.

Correspondence and reprint requests:
Jimmy S. M. Lai, Department of Ophthalmology, United Christian Hospital, Hip Wo Street Kwan Tong, Kowloon, Hong Kong, China.

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Abstract

Aim: To evaluate the efficacy of latanoprost for the prevention of ocular hypertension after neodymium:yttrium-aluminum-garnet laser posterior capsulotomy.
Methods: Fifty six eyes of 56 consecutive patients receiving neodymium:yttrium-aluminum-garnet laser capsulotomy for posterior capsule opacification were randomized to group 1, who received 1 drop of latanoprost 0.005%, or to group 2, who received 1 drop of normal saline, 1 hour before the laser treatment. Intraocular pressures were taken before instillation of the eye drop, and at 1 hour and 2 hours after the laser procedure. Student’s t-test was used to analyze the differences in intraocular pressures between the 2 groups.
Results: There were no statistically significant changes in the mean intraocular pressures of the 2 groups at 1 and 2 hours after neodymium:yttrium-aluminum-garnet laser capsulotomy compared with the baseline. There was no statistically significant difference in the mean intraocular pressures between the 2 groups at the same time intervals.
Conclusions: A single application of latanoprost given 1 hour before neodymium:yttrium-aluminum-garnet laser posterior capsulotomy did not produce a statistically significant intraocular pressure lowering effect during the first 2 hours after the laser procedure when compared with a control group.

Key words: Intraocular pressure, Laser surgery

Introduction
Posterior capsule opacification (PCO) frequently occurs after cataract extraction and posterior chamber intraocular lens (PCIOL) implantation and can cause deterioration of visual acuity. Neodymium:yttrium-aluminum-garnet (Nd:YAG) laser posterior capsulotomy is the standard treatment for PCO to restore vision.1 It has been reported that the intraocular pressure (IOP) may rise significantly within 3 hours after Nd:YAG capsulotomy.2-5 Apraclonidine,
pilocarpine, and timolol have been studied for the prevention of such IOP spikes.6-10 The prophylactic effect of apraclonidine for IOP spikes after laser procedures may become insignificant for patients already receiving long-term apraclonidine treatment.3 Besides, apraclonidine is associated with adverse effects including mild ocular inflammation, corneal edema, eyelid retraction, blepharospasm, and irregular heart rate.7

Latanoprost is an effective IOP lowering agent for primary open angle glaucoma and ocular hypertension.11-18 Latanoprost is a prostaglandin (PG) analogue — PG F2α.17 The main mechanism of action is to increase aqueous outflow through the uveoscleral route.18 The onset of action occurs approximately 4 hours after application, with a peak effect at 8 to 12 hours.11,19 Latanoprost has minimal systemic side effects and its ocular side effects are relatively mild. These include local irritation, dry eye and foreign body sensation, iris hyperpigmentation, conjunctival hyperemia, iritis, lacrimation, itching, and cystoid macular edema (CME).14-16,20-22 Due to the minimal adverse effect profile, latanoprost is theoretically a safe pharmacological agent for prophylactic use against post-laser IOP spikes.

A double blind randomized controlled trial was conducted to investigate the IOP-lowering effect and safety of prophylactic latanoprost for the prevention of ocular hypertension after Nd:YAG laser posterior capsulotomy.

Materials and methods

Consecutive patients with PCO after uncomplicated cataract extraction and PCIOL implantation requiring Nd:YAG capsulotomy were enrolled. Patients with single eye, pregnancy, history of ocular hypertension, glaucoma, uveitis, long-term steroid use, or intraoperative complications of vitreous loss were excluded. If the intraocular lens was cracked during Nd:YAG capsulotomy, these patients would also be excluded.

The study was approved by the ethics committee of the United Christian Hospital. Informed consent was obtained from all patients. Prior to laser capsulotomy, the visual acuity and the IOP were measured. The anterior segment and the fundi were examined. Patients were randomly assigned to either group 1, to receive latanoprost, or group 2 as a control. Patients in group 1 received 1 drop of latanoprost 0.005% and patients in group 2 received 1 drop of normal saline 1 hour before laser treatment, with 15 minutes leeway. Patients who received the medications longer than 1 hour and 15 minutes before laser treatment were excluded. The surgeons were blinded to the assigned treatment.

The eye was anesthetized with 1 drop of topical benoxinate 0.4%. No mydriatic was given for pupil dilatation. The opacified posterior capsule was perforated using Nd:YAG laser (Alcon 3000LE, Irvine, USA). The total amount of laser energy used was recorded. IOP readings before instillation of the eye drop and at 1 and 2 hours after laser treatment were taken by investigators blinded to the assigned treatment.

The patients were followed up 1 week after the laser capsulotomy to check the visual acuity. If fundal examination revealed clinical CME, fluorescein angiography was arranged to confirm the finding.

The changes in IOP readings were analyzed using Student’s t-test. Pearson’s correlation was employed for analysis of correlation between laser energy and mean changes in IOPs.

Results

Fifty six eyes of 56 consecutive Chinese patients were recruited. Patients were randomized to receive latanoprost (29 patients) or saline (27 patients). Table 1 shows the patients’ demographic data, the mean YAG laser energies, and the mean IOPs of the 2 groups at different time intervals. The mean IOPs between the 2 groups before and at 1 and 2 hours after Nd:YAG capsulotomy did not show any statistically significant differences.

One hour after capsulotomy, the mean change in IOPs from baseline was -0.6 and +1.0 mm Hg for groups 1 and 2, respectively. The difference in the IOP changes was not statistically significant. After 2 hours, the mean change in IOPs for the latanoprost and control groups was +0.5 and +0.3 mm Hg, respectively, — a non-significant difference (Figure 1).

Four patients had a rise in IOP of >5 mm Hg after laser capsulotomy. One patient (3.4%) was receiving latanoprost and 3 (11.1%) were in the control group. One of the 3 patients in the control group had an increase in IOP to 27 mm Hg 2 hours after the capsulotomy. This patient was treated with systemic acetazolamide.

The mean laser energies used for the treatment and control groups were 14.4 ± 11.9 mJ and 19.3 ± 17.5 mJ, respectively. There was no significant difference in the laser energy used for the 2 groups.

Pearson’s correlation was used to look for any correlation between the laser energy and the mean changes in IOP for

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Group 1 (latanoprost)</th>
<th>Group 2 (control)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of eyes</td>
<td>29</td>
<td>27</td>
<td></td>
</tr>
<tr>
<td>Mean age + SD (years)</td>
<td>70.6 ± 8.7</td>
<td>71.0 ± 9.2</td>
<td></td>
</tr>
<tr>
<td>Men:women</td>
<td>10:19</td>
<td>11:16</td>
<td></td>
</tr>
<tr>
<td>Mean laser energy (mJ)</td>
<td>14.4 ± 11.9</td>
<td>19.3 ± 17.5</td>
<td>0.22</td>
</tr>
<tr>
<td>Intraocular pressure (mm Hg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre-laser</td>
<td>11.7 ± 3.6</td>
<td>11.8 ± 2.5</td>
<td>0.95</td>
</tr>
<tr>
<td>Post-laser 1 hour</td>
<td>11.2 ± 4.5</td>
<td>12.8 ± 4.2</td>
<td>0.17</td>
</tr>
<tr>
<td>Post-laser 2 hours</td>
<td>11.5 ± 5.2</td>
<td>13.6 ± 4.3</td>
<td>0.14</td>
</tr>
</tbody>
</table>
patients in both the treatment and control groups 1 and 2 hours after capsulotomy. No correlation was found as the p values for the correlation coefficients were greater than 0.05.

One patient (male, 68 years) in the control group developed CME, confirmed by fluorescein angiography 1 week after capsulotomy. This patient had diabetic retinopathy treated with panretinal photocoagulation more than 1 year before the capsulotomy. This was the same patient who had an IOP spike requiring systemic acetazolamide. This patient’s visual acuity was 0.1 before and after laser capsulotomy.

**Discussion**

IOP may rise significantly within 3 hours after Nd:YAG posterior capsulotomy. Ocular hypertension may be due to obstruction of the trabecular meshwork by lens capsule particulate debris and inflammatory cells or due to shockwave damage to the trabecular endothelial cells. Since latanoprost increases aqueous outflow through the uveoscleral route, it may prevent the IOP spike after Nd:YAG capsulotomy. In this study, latanoprost was given 1 hour before laser surgery so that its onset of action might coincide with the post-laser IOP spike. However, latanoprost did not show any significant IOP lowering effect compared with the control group. Single application of latanoprost has also been found to be ineffective for the prevention of ocular hypertension after phacoemulsification and uveoscleral lens implantation. The explanation may be similar for both situations since the uveoscleral outflow enhancement by prostaglandins requires remodeling of the ciliary muscle and this effect may only occur with long-term administration of latanoprost.

This study did not identify any contributing factor for the post-laser IOP spike. The 4 patients (1 in the latanoprost group and 3 in the control group) who had post-laser IOP spikes of 5 mm Hg greater than the pre-laser level did not require significantly higher Nd:YAG laser energy. The laser energy used for the patient in the latanoprost group was 20 mJ, while the mean energy for this group was 14.4 ± 11.9 mJ, and the energy level for the 3 patients in the control group ranged from 18 to 30 mJ, while the mean energy for this group was 19.3 ± 17.5 mJ. Analysis using Pearson’s correlation failed to show a positive correlation between the laser energy and the mean changes in IOP in both groups 1 and 2 hours after capsulotomy.

Nd:YAG posterior capsulotomy has been shown to cause CME in 3% of patients. Latanoprost has also been reported to be related to CME. In this study, none of the patients in the latanoprost-treated group developed CME. The only patient who developed CME was in the control group. This patient also had diabetic retinopathy, which could be the cause of the CME. Moreover, since the incidence of laser capsulotomy- and latanoprost-induced CME is rare and the sample size in this study was small, no conclusion as to the cause of the CME may be reached.

**Conclusion**

A single drop of latanoprost given 1 hour before Nd:YAG posterior capsulotomy had no significant IOP lowering effect in the immediate post-laser period.

**References**


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