Risk factors for persistent inflammation post-laser peripheral iridotomy.

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Abstract

Purpose: To investigate risk factors associated with persistent inflammation after Laser Peripheral Iridotomy (LPI).

Methods: We performed a single-center, retrospective, case-control study of patients with primary angle closure suspicion, primary angle closure glaucoma, or chronic angle closure glaucoma treated with Laser Peripheral Iridotomy between April 1, 2016 and April 30, 2019. Parameters investigated included patient demographics, ocular history, laser settings, surgeon experience, and medication compliance. Persistent inflammation was defined as the presence of cells and/or flare at the first follow-up visit despite prophylactic treatment with prednisolone acetate 1% four times a day for 1 week. We performed Fisher's exact tests and two-sample t-tests to identify risk factors associated with persistent inflammation.

Results: 203 patients were included of which 16 (7.9%) met criteria for persistent inflammation following LPI. Risk factors associated with persistent inflammation included pre-treatment with argon laser (p=0.045). The mean argon energy and exposures was 741.9 ± 108.9 mW and 262 ± 242 counts in the persistent inflammation group verses and 533.8 ± 302.4 mW and 113 ± 131 counts in the no inflammation group (p<0.001 and p=0.027 respectively). Persistent inflammation was associated with ≤ 5 years of surgeon experience (p=0.03615, OR=3.19). Patient demographics (age, gender or race), LPI location and Nd-Yag laser parameters were not associated with persistent inflammation.

Conclusion: We demonstrate an association between mean argon laser energy, exposure count and surgeon inexperience and persistent inflammation following LPI. We hope identifying risk factors can help guide interventions to minimize post-operative inflammation in a common laser procedure in glaucoma.

Keywords: Persistent inflammation, Risk factors, Laser peripheral iridotomy.

Abbreviations: LPI: Laser Peripheral Iridotomy; IOP: Intraocular Pressure.

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Introduction

Primary angle closure glaucoma can be a devastating eye disease affecting as much as 4 in 1,000 patients ≥ 40 years old [1]. Patients with narrow anterior chamber angles can be classified as primary angle closure suspects and those that subsequently develop evidence of optic neuropathy are diagnosed with primary angle closure glaucoma [2]. The primary prophylactic treatment of primary angle closure is laser peripheral iridotomy (LPI) [3-6]. In the USA alone, nearly 50,000 LPI procedures are done annually, mostly in primary angle-closure suspects [7].

Although LPIs are generally safe and effective, they can be associated with several postoperative complications which include iritis, transient elevation of Intraocular Pressure (IOP), hyphema, dysphotopsia, etc [8-12]. Hence it is important to evaluate if persistent inflammation occurs post LPI because if not treated appropriately and in a timely manner, persistent inflammation can lead to peripheral iridotomy closure, posterior anterior synechiae, cataract development and even elevated intraocular pressure. Knowing the risk factors that lead to persistent inflammation can help identify susceptible patients prior to LPI and accordingly plan treatment and follow-up before and after their LPI.

While a number of studies have reported typical power use and complication rates associated with LPIs, no prior study to our knowledge has looked at identifying risk factors for persistent inflammation post-LPI in patients with narrow angles.

The purpose of this study is to retrospectively compare patients with and without persistent inflammation after LPI to identify risk factors for this persistent inflammation.

Materials and Methods

The study protocol was approved by the Institutional Review Board of Boston University and adhered to the Declaration of Helsinki. We retrospectively reviewed the medical records of patients treated with LPI between April 1, 2016 to April 30, 2019 at Boston Medical Center (Boston, MA). Patients were identified through a query of CPT code 66761 for LPI within the study time period. We excluded patients without any follow-up visits after LPI, inadequate information in chart review, prior intraocular surgery, and history of known intraocular inflammation. Demographic data collected from each patient included: patient age, gender, operative eye, race, diagnostic indication of LPI, pre-LPI Visual Acuity (VA), pre-LPI intraocular pressure (IOP), and any history of ocular inflammation or intraocular surgery. Variables pertaining to

laser parameters were collected including: surgeon performing LPI, date of LPI, iris treatment location, type of laser used, argon pretreatment power (mW) and exposure count, Nd-Yag power (mJ) and exposure count. Lastly, the presence of inflammation as noted by the documented presence of cells or flare in the anterior chamber at each subsequent follow-up, the number of days since the LPI of first follow-up, the number of days of the most recent follow-up visit, medication compliance, and any other complications were recorded.

Typically, patients received topical drops of proparacaine 0.5% and pilocarpine 1%. All iridotomies were performed using an Abraham lens (Ocular Abraham Iridectomy YAG laser lens; Ocular Instruments, Bellevue, WA, USA) in conjunction with hydroxypropyl methylcellulose as a coupling agent to focus the laser. The LPIs were performed using the Lumenis selecta trio argon laser and a neodymium: yttrium-aluminum-garnet (Nd:YAG) laser (Lumenis inc. CA, USA). A drop of prednisolone acetate 1% and apraclonidine 0.5% were administered after the treatment. Post-laser IOP was checked 30 minutes after the laser procedure using either a tonopen or Goldmann Applanation (Reichert, Depew, NY, USA). Post-operatively, all patients were prescribed prednisolone acetate 1% four times a day for 4-7 days.

Statistical analysis was performed comparing the groups of patients with and without persistent inflammation. Persistent inflammation was defined as the presence of cells and/or flare in the anterior chamber at the first follow up visit. Risk factor analyses for categorical variables were performed using Fisher's exact tests and continuous using two sample t-tests in univariate analysis. A multivariate logistic analysis was also performed between variables. All statistical tests were conducted using R-studio Version 3.5.1 with a significance of $p<0.05$.

Results

A total of 203 patients who underwent LPIs were reviewed. We found 187 patients with no inflammation and 16 patients with persistent inflammation. All inflammation was limited to the anterior segment.

The patient characteristics are shown in Table 1. Patients in the persistent inflammation group had a mean ± SD age of 65.3 ± 14.1 years, 81% were female, 63% received an LPI in the right eye, 69% had a diagnosis of primary angle-closure suspect, 56% were Hispanic, 25% African American, 13% Asian, 6% Caucasian, baseline mean ± SD VA was 0.528 ± 0.741, baseline mean ± SD IOP was 22.1 ± 11.4 mmHg, average number of days to the first follow-up after LPI was 15.6 ± 11.5, average number of days to the most recent follow-up after LPI was 214.7 ± 204.6, and 75% were compliant with their medications.

	No inflammation (n=187)	Persistent inflammation (n=16)	p-value
Age			
Mean ± SD	63.0 ± 10.8	65.3 ± 14.1	0.5344
Range	36-97	27-86	
Gender			
Male (%)	55 (29%)	3 (19%)	0.5648
Female (%)	132 (71%)	13 (81%)	
Eye			
Right Eye (%)	114 (61%)	10 (63%)	1
Left Eye (%)	73 (39%)	6 (38%)	
Race			
Hispanic or Latino (%)	84 (45%)	9 (56%)	0.8713
African American (%)	41 (22%)	4 (25%)	
Caucasian (%)	29 (16%)	1 (6%)	
Asian (%)	30 (16%)	2 (13%)	
Middle Eastern (%)	2 (1.1%)	0	
Pacific Islander (%)	1 (0.5%)	0	
Diagnosis			
Narrow Angles Without Glaucoma (%)	144 (77%)	11 (69%)	0.4616
Narrow Angles with Ocular Hypertension and Glaucoma Suspects (%)	30 (16%)	3 (19%)	
Glaucoma (%)	13 (7%)	2 (12%)	
Visual Acuity (logMAR)			
Mean ± SD	0.181 ± 0.341	0.528 ± 0.741	0.08276
Range	0-2	0-2	
IOP (mmHg)			
Mean ± SD	17.3 ± 5.94	22.1 ± 11.4	0.1184
Range	Sep-46	Oct-45	
Time to 1st follow-up (days)			
Mean ± SD	49.7 ± 68.0	15.6 ± 11.5	2.11 E-08
Range	0-483	Jan-36	
Time to last follow-up (days)			
Mean ± SD	193.8 ± 152.2	214.7 ± 204.6	0.7262
Range	8-713	36-644	
Medication compliance			
Yes (%)	102 (86%)	9 (75%)	0.3822
No (%)	16 (14%)	3 (25%)	

Table 1. *Patient characteristics.*

Patients in the no inflammation group had a mean ± SD age of 63.0 ± 10.8 years, 71% were female, 61% received an LPI in the right eye, 77% had a diagnosis of primary angle-closure suspect, 45% were Hispanic, 22% African American, 16% Caucasian, 16% Asian, baseline mean ± SD VA was 0.181 ± 0.341, baseline mean ± SD IOP was 17.3 ± 5.94 mmHg, average number of days to the first follow-up after LPI was 49.7 ± 68.0, average number of days to the most recent follow-up after LPI was 193.8 ± 152.2.

There were no statistically significant differences in age, gender, eye operated on, diagnosis, race, VA, IOP, medication compliance, and time to most recent follow-up between the persistent inflammation group and the group with no inflammation. The time to the first follow-up was statistically significant when compared between the persistent inflammation group and the group with no inflammation ($p \leq 0.001$).

	No inflammation (n=187)	Persistent inflammation (n=16)	p-value
Type of laser			
Argon and YAG	147 (79%)	16 (100%)	0.04544
YAG only	40 (21%)	0	OR=∞
Argon energy (mJ)			
Mean ± SD	533.8 ± 302.4	741.9 ± 108.9	5.93 E-07
Range	0-1000	500-900	
Argon exposure (# shots)			
Mean ± SD	112.5 ± 130.8	261.7 ± 242.3	0.02731
Range	0-673	33-772	
YAG energy (mJ)			
Mean ± SD	60.2 ± 126.4	112.3 ± 107.6	0.2244
Range	1.78-1236	6.6-264.28	
YAG exposure (# shots)			
Mean ± SD	29.4 ± 47.8	42.3 ± 57.9	0.4002
Range	1-381	3-171	
LPI location			
Nasal	24 (18%)	5 (42%)	0.06294
Temporal	105 (78%)	6 (50%)	
Superior	2 (2%)	1 (8%)	
Inferior	3 (2%)	0	

Table 2. *Laser peripheral iridotomy parameters.*

The laser parameters are shown in Table 2. In the persistent inflammation group, the mean ± SD argon energy was 741.9 ± 108.9 mW, the mean ± SD number of argon shots was 261.7 ± 242.3, the mean ± SD Nd:YAG energy was 112.3 ± 107.6 mJ, the mean ± SD number of Nd:YAG shots was 42.3 ± 57.9. In the no inflammation group, the mean ± SD argon energy was 533.8 ± 302.4 mW, the mean ± SD number of argon shots was 112.5 ± 130.8, the mean ± SD Nd:YAG energy was 60.2 ± 126.4 mJ, the mean ± SD number of Nd:YAG shots 29.4 ± 47.8. As shown in Table 2, pretreatment with argon laser before using Nd: YAG laser lead to statistically significant persistent inflammation (p=0.045). The amount of argon energy and argon exposure (number of laser shots) was also statistically significant when compared between the persistent inflammation group and the no inflammation group (p<0.001, p=0.027 respectively). However Nd:YAG energy and Nd:YAG exposure (number of laser shots) were not significant between the persistent inflammation group and the no inflammation group.

LPI locations were recorded in 134 patients out 203 (72%). However, in the persistent inflammation group, out of 12 recorded LPIs, 42% were located nasally, 50% temporally, 8% superiorly, and 0% inferiorly. In the no inflammation group out of 134 recorded cases, 18% were located nasally, 78% temporally, 2% superiorly, and 2% inferiorly. There was no statistically significant difference noted when comparing LPI locations between persistent inflammation group and the no inflammation group (p=0.063).

	No inflammation (n=187)	Persistent inflammation (n=16)	p-value
Glaucoma specialization			
Glaucoma Surgeon	119 (64%)	12 (75%)	0.4264
Non-glaucoma Surgeon	68 (36%)	4 (25%)	
Surgeon experience			
5 Years or Less	76 (41%)	11 (69%)	0.03615
>5 Years of Experience	111 (59%)	5 (31%)	OR=3.19

Table 3. *Surgeon parameters.*

A total of 12 different attending surgeons were included in the study as shown in Table 3. Subgroup analysis was performed by stratifying the surgeons by glaucoma specialization and years of experience. There were 5 glaucoma specialists and 7 comprehensive ophthalmologists in this study. There were 6 surgeons with five or less years of post-residency practice experience and 6 surgeons with more than five years of experience. Patients with persistent inflammation compared to patients with no inflammation did not show any significant differences when stratified by surgeons with glaucoma training (p=0.426). However, the number of patients with persistent inflammation compared to patients with no inflammation was statistically significant when stratified by surgeon's years of experience (p=0.0362). Surgeons with five or less years of training were almost three times more likely to have a patient with persistent inflammation compared to surgeons with more than five years of training (OR=3.195).

Multivariate analysis was performed between selected variables and shown in Table 4. Statistically significant risk

factors included number of argon exposures (p=0.007) and pre-operative vision (p=0.023).

Characteristic	Odds ratio	95% confidence interval	p-value
Age	1	0.95 to 1.06	0.94
Gender	0.24	0.03 to 1.16	0.08
African American	1.59	0.23 to 11.9	0.63
Hispanic	4.02	0.86 to 27.2	0.079
Pre-LPI Vision (logMAR)	4.02	1.18 to 13.7	0.023
Pre-LPI IOP (mmHg)	1.04	0.96 to 1.12	0.3
Number of Argon Exposures	1.05	1.01 to 1.09	0.0007
Number of Nd:YAG Exposures	1.01	0.86 to 1.12	0.89
Glaucoma Provider	3.04	0.71 to 18.2	0.64
Surgeon Inexperience	1.38	0.35 to 5.53	0.64

Table 4. *Multivariate analysis of risk factors.*

Discussion

In this retrospective, single-center case-control study we investigated the incidence and risk factors for persistent inflammation in patients with narrow anatomical angles treated with LPI procedures.

We found that the incidence of persistent inflammation was 7.9% (16 out of 203 patients) post-LPI after receiving a 1-week course of topical prednisolone acetate 1% four times a day. Baig et al., reported post-LPI transient uveitis with anterior chamber reaction in 32 eyes out of 40 eyes which settled within 48-72 hours of topical steroids treatment four times a day for 5-7 days. However, out of 40 eyes post-Nd:YAG laser, one eye experienced anterior uveitis for about 4 weeks while another eye developed posterior synechiae [13]. In a seven year retrospective observational case series by Ali et al., all 5 non-uveitic patients developed a chronic anterior uveitis following Nd:Yag LPI treatment [12].

In our study, we found a statistically significant association between pretreatment with argon laser and persistent inflammation. All of the patients in the persistent inflammation group had some degree of argon laser treatment whereas patients with Nd:YAG treatment alone did not have persistent inflammation. We found a statistically significant association between argon exposure count and persistent inflammation, however a similar association was not observed for the Nd:YAG laser. Our findings suggests that pretreatment with argon may be a risk factor for developing persistent inflammation. We hypothesize that the additional intraocular energy administered and perhaps specifically the argon laser coagulative process caused more damage to the iris tissue, which might be pro-inflammatory and increase the release of pigmented cells. In addition, there may also be a cumulative/synergistic effect of both the coagulative process of argon laser and pigment dispersion of the photo-disruptive nature of Nd:YAG laser leading to more inflammation which would require further studies to explore.

We did not appreciate any statistically significant association between LPI location and persistent inflammation. In our center, nasal and temporal LPI locations are most common and thus this study may not be powered enough to draw conclusions about superior/inferior treatment locations. However, a study by Ahmadi et al., looked at efficacy and safety of Nd:Yag iridotomies in the superior and inferior LPI locations, and found that inferior LPIs required less use of mean total laser energy to perforate the tissue and resulted in a lower post-LPI iritis risk [14]. Further work is recommended to compare the four different quadrants with a higher sample size to evaluate which quadrant, if any, is associated with the lowest inflammation post-LPI.

To the best of our knowledge, this is the first study to evaluate surgeon's experience as a possible risk factor in persistent inflammation post-LPI. LPIs done by more experienced providers were associated with lower persistent inflammation compared to the inexperienced providers. Kam et al., states that increased power settings may be more effective by creating a concentrated force to penetrate the iris stroma rather than multiple weak ineffective shots that just disperse pigment with minimal penetrating tissue [15]. It is possible that the less power and increased shots used by the inexperienced surgeon might be contributing to the persistent inflammation. Based on this finding it is more important for the inexperienced surgeons to look back at their data and reassess their techniques and methods to prevent this persistent inflammation. LPIs done by glaucoma specialists versus other subspecialists showed no difference between the persistent inflammation group and no inflammation group.

To our surprise there was no significant difference between the races when comparing the persistent inflammation group to the no inflammation group. However most of the patient population at this center are non-Caucasian and the sample of Caucasian is very small which likely could be confounding the result. As such, iris color was not recorded consistently however, given the majority of Hispanic and African American subjects, dark irides are likely to represent our patient sample. Saim et al., reported a retrospective review on 4 darkly pigmented irides patients with marked inflammation post-LPI and concluded that these cases were possibly predisposed to this reaction by the heavy pigmentation of their irides based on race [11], however no other studies are reported in the literature that examine the correlation between persistent inflammation and dark irides post-LPI. Kam et al., also did not find any significant differences in complication rates between Caucasian and non-Caucasian subjects which is consistent with our study as race was not found to be a risk factor for persistent inflammation.

Kam et al., did however find a significant increase in repeat LPI among non-Caucasian subjects. Notably in their study, argon laser treatment was not performed prior to photodisruptive Nd:YAG laser. Kam et al., argues that the higher rate of iridotomy closure and inflammation is due to the greater amount of pigment dispersed in darker, thicker irides among non-Caucasian eyes [15,16]. However, this closure is likely related to the use of only Nd:YAG laser without argon laser pretreatment. Ho et al., reported 20 eyes from 13 patient with dark irides pretreated with argon laser prior to Nd:Yag laser worked well with low rates of iridotomy closure which agrees with our experience. In this report they conclude that sequential argon-Nd:YAG is a safe and effective procedure however they did not report persistent inflammation one week post-operatively with prophylactic treatment. One can argue that their sample size is small and all of the cases are done by single surgeon while in our study, we have reviewed 203 patients with about 12 different surgeons who have different levels of experience. No significant correlation was found between pre-laser IOP and persistent inflammation. Likewise, we did not identify a correlation between pre-laser IOP or VA and persistent inflammation. We also did not find statistical significance when looking at medication non-compliance as a risk factor to persistent inflammation. This could be explained by overall good medication compliance between the two groups.

We found that time to first follow-up visit was statistically significant between the groups. Patient with persistent inflammation came back much earlier to their first follow-up visit compared to the patients in the no inflammation group. This statistically significant difference in the first follow-up visit could be explained by the fact that patients present earlier due to symptoms from the persistent inflammation to seek medical attention. However, due to the retrospective nature of the study, we are unable to control for the difference in the time point of when each post-operative visit occurred and if individuals with persistent inflammation had earlier scheduled follow ups. Therefore, there may be a cohort of patients without inflammation with later scheduled follow-up who had inflammation which self-resolved prior to their appointment.

One limitation of this study is that most of the LPIs are done by residents with different levels of training even though each resident is under supervision of the attending surgeon. At our institution traditionally LPIs are done solely with first year residents however, given the nature of the close supervision by the attending surgeon, we feel that the experience of the attending surgeon is still paramount. A second limitation of the study is that not all surgeons consistently document the LPI location but out of the documented cases, most are located nasally or temporally in this study and only handful of performed LPI are located superiorly or inferiorly. In the future we recommend performing a large population-based study to evaluate all four quadrants to find the location associated with the least complication rate. The third limitation is lack of standard post-LPI assessment for safety and timeline of follow up to assess for anterior chamber inflammation.

Future prospective studies may standardize the window of follow-up to ensure an exam while the inflammation remains present. The fourth limitation in this study is that we evaluated one of many laser machines that are currently available on the market, and the energy and power may not be extrapolated to other machines. Lastly, given the retrospective nature of the study, there was no consistency to account for lack of documentation for some of the variables including the total Nd: YAG energy which restricted our ability to evaluate this important laser parameter. Often the energy is estimated by using the total work and exposures recorded. This limitation is also reflected in the multivariate analysis as many variables are collinear including laser parameters. Additionally, the two significant variables in the multivariate analysis are number of argon laser exposure and pre-operative visual acuity however their odds ratio of the likelihood of inflammation seem clinically unintuitive as an additional 10 counts of argon laser increase odds of persistent inflammation by 1.04 or each logmar change in initial visual acuity would increase odds of persistent inflammation by 4.02.

Finally, even though these persistent inflammation patients did not have any known intraocular inflammation prior to LPI, it would be interesting to correlate their medical history with any predisposing systemic autoimmune diseases.

Conclusion

In conclusion, we found the following risk factors for persistent inflammation post-LPI: 1) the use of both Argon; 2) the amount of argon energy; 3) the amount of argon exposure; 4) surgeon inexperience (less than five years). We did not find any statistical significance when comparing race which was surprising as dark irides were reported to be prone to inflammation since they require more power compared to light irides. Additionally, the following risk factors were also not significant: age, gender, eye operated on, visual acuity, pre-LPI IOP, Nd-Yag laser energy, Nd-Yag laser exposure, and glaucoma specialist training. These results indicate that surgeons especially those who use argon pretreatment and with less experience need to be more cognizant of the increased inflammation risk in their patients. They may need to consider changing their standard of care in order to avoid this complication by implementing a more intensive post-LPI treatment regimen such as a slower steroid taper or closer follow-up visits.

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Conflicts of interest

None of the authors have any conflicts of interest

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References

1. Day AC, Baio G, Gazzard G, et al. The prevalence of primary angle closure glaucoma in European derived populations: A systematic review. Br J Ophthalmol. 2012;96:1162-7.
2. Foster PJ, Buhrmann R, Quigley HA, et al. The definition and classification of glaucoma in prevalence surveys. Br J Ophthalmol. 2002;86:238-42.
3. Robin AL, Pollack IP. Argon laser peripheral iridotomies in the treatment of primary angle closure glaucoma. Arch Ophthalmol. 1982;100:919–23.
4. Ang LP, Aung T, Chew PT. Acute primary angle closure in an Asian population: Longterm outcome of the fellow eye after prophylactic laser peripheral iridotomy. Ophthalmology. 2000;107:2092-6.
5. Laser peripheral iridotomy for pupillary-block glaucoma. American Academy of Ophthalmology. Ophthalmology. 1994;101:1749-58.
6. Robin AL, Pollack IP. A comparison of neodymium: YAG and argon laser iridotomies. Ophthalmology. 1984;91:1011-6.
7. Arora KS, Robin AL, Corcoran KJ, et al. Use of various glaucoma surgeries and procedures in Medicare beneficiaries from 1994 to 2012. Ophthalmology. 2015;122:1615-24.
8. Corriveau LA, Nasr Y, Fanous S. Choroidal and retinal detachment following argon laser iridotomy. Can J Ophthalmol. 1986;21:107-8.
9. Hiroshi S, Michiko Y, Miyako S. Recurrent uveal effusion after laser iridotomy. Case Rep Ophthalmol. 2017;8:26-30.
10. Sakai H, Ishikawa H, Shinzato M, et al. Prevalence of ciliochoroidal effusion after prophylactic laser iridotomy. Am J Ophthalmol. 2003; 136:537-8.
11. Siam GA, de Barros DS, Gheith ME, et al. Post-peripheral iridotomy inflammation in patients with dark pigmentation. Ophthalmic Surg Lasers Imaging. 2008;39:49-53.
12. Ali A, Rosenfeld C, Rosenberg E, et al. Chronic uveitis following neodymium-doped-yttrium-aluminum garnet laser peripheral iridotomy. 2017;26.
13. Baig R, Khan A. Clinical outcome of iridotomy with Argon-YAG laser at a tertiary care center in Karachi, Pakistan. J Pak Med Assoc. 2010;60:220-3.
14. Ahmadi M, Naderi BZ, Naderi BA, et al. Efficacy of neodymium-doped yttrium aluminum garnet laser iridotomies in primary angle-closure diseases: Superior peripheral iridotomy versus inferior peripheral iridotomy. Curr Med Res Opin. 2017;33:687-92.
15. Kam JP, Zepeda EM, Ding L, et al. Resident-performed laser peripheral iridotomy in primary angle closure, primary angle closure suspects, and primary angle closure glaucoma. Clin Ophthalmol. 2017;11:1871-6.
16. Ho T, Fan R. Sequential argon –yag laser iridotomies in dark irides. British Journal of Ophthalmology, 1992;76:329-31.

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