To PI or not to PI—That is the Question: When is a Prophylactic Laser Peripheral Iridotomy Justified?

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INTRODUCTION

Angle closure glaucoma is uniquely different from open angle glaucoma and many eye diseases—it is preventable. Early diagnosis of a narrow or closed angle before irreversible damage to the trabecular meshwork (TM) has occurred can, in many cases, lead to treatment that changes the anatomy around the angle and its widening, prevention of further damage to the TM and ultimately prevention of elevated IOP and glaucoma. In most cases of angle closure, there is a dominant mechanism of pupillary block that can be treated with laser peripheral iridotomy (LPI). Since often the clinician is not certain whether a prophylactic LPI is indicated, in this review I shall present current data on this subject and some practical recommendations.

When to do a LPI?

It is sometimes argued that LPI should be performed in all eyes diagnosed with an angle that is felt to be at high-risk for closure. To make this argument it is assumed that such an angle is relatively uncommon in the general population and that LPI is extremely safe. I challenge these assumptions. Angle closure glaucoma is very common in East Asia and individuals from this region, especially Chinese, as shown in recently published epidemiological research. Prevalence among Caucasians has been less studied, and according to current literature it is estimated at 0.05 to 0.5%. Prevalence of “narrow angle” that deserves prophylactic LPI is even less well defined, and is likely a few percent. In a recently published survey of an Indian population (3850 subjects), angle closure glaucoma was found in 0.88% whereas angle closure—without peripheral anterior synchiae (PAS) and IOP < 21 mmHg—was found in 7.2%. In this report “angle closure” was precisely defined as an angle whose details were not seen in more than 180° of the circumference. From these findings we can conclude that a “narrow” or “occludable” angle, as is broadly determined by clinicians in everyday practice, is diagnosed even more frequently. Rather than treat all these eyes, it would be more appropriate to identify only those eyes truly at high-risk and therefore in need of LPI. (I note that this scenario would indeed occur only if gonioscopy is performed as part of a routine eye examination, whereas it seems that currently this is not the situation, and gonioscopy is not performed frequently enough, even in subjects diagnosed with glaucoma.)

Regarding the safety of LPI, this is indeed a simple and very safe procedure. However, like any medical procedure, it is not completely without possible complications. In addition to the well-known temporary short-term complications such as elevated IOP and hyphema, there may be longer-term complications that have not been adequately investigated. LPI may accelerate the formation of cataract. Significant damage to the corneal endothelium cannot be excluded. And, although infrequent, a single patient who was “healthy” before the procedure and chronically suffers visual disturbances following it, is enough to cause us to re-evaluate the need for prophylactic LPI.

Before concluding when I think LPI is appropriate, I should briefly describe why it is considered to prevent glaucoma. The fundamental idea behind LPI is the understanding that in the presence of relative pupillary block there is resistance to aqueous flow between the lens and iris at the pupillary border, increased hydrostatic pressure is created behind the iris, and the iris becomes convex. In eyes with predisposing anatomy (short axial length, shallow anterior chamber) iris convexity leads to narrowing or closure of the iridocorneal angle. LPI creates a bypass of the peripupillary area of resistance and allows free flow of aqueous from the posterior to the anterior chambers. The pressure gradient is eliminated, the iris becomes flat, and the angle widens. These anatomic changes can be demonstrated by several imaging modalities, and are seen in most treated eyes. However, clinicians prefer clinical proofs, and here there is rather little. Lowe reported that after having had an attack of acute angle closure in one eye, there was a 50% chance of a similar episode in the fellow eye during the following 25 years if this eye was not treated surgically; but if preventive surgical iridotomy was performed, only 1 of 54 eyes experienced an attack. Today it is ethically difficult to observe these
eyes and perform a prospective comparative study. In one study in which 80 such fellow eyes were treated with prophylactic LPI and followed for 5 years on average, acute angle closure did not occur in 100% of the eyes. Clinical proof that LPI is effective in preventing chronic angle closure, a condition that is far more common than acute closure, is lacking. There are however a few studies that followed eyes with “occludable angles” and demonstrated that indeed some of them progressed in the disease process. In one, glaucoma experts identified on routine examination 129 patients with narrow angles “believed to be capable of closure” or shallow chamber (less than 2 mm).

During average follow-up of 3 years, acute angle closure had occurred in 8 (6.2%) patients (11 eyes) and chronic angle closure, defined as gonioscopic observation of iridotrabecular contact, was observed in 17 (13.2%) patients (27 eyes). In a large survey in India, researches identified in 1995, 118 subjects with closed angle—an angle in which more than 180° was not seen, with no PAS or elevated IOP—in one or both eyes. Fifty of these were examined again after 5 years. Of these 11 (22%) progressed—in 7 cases PAS were seen and in 4 IOP was measured > 21 mmHg. Similar “progression” was observed in 1 of 110 controls without a closed angle at baseline, with a calculated relative risk of 24.2 in the eyes with closed angles. None of the eyes that progressed showed glaucomatous changes in the optic nerve or visual field. In another publication based on this cohort, 32 eyes had been diagnosed in 1995 with a closed angle in the presence of either PAS and/or IOP > 21 mmHg but without glaucomatous neuropathy. Five years later 28 subjects were re-examined, of whom 8 (28.5%) had progressed to frank angle closure glaucoma. One of 9 who had LPI in 1995 progressed, compared with 7 of 19 who did not have LPI (the procedure had been offered but they refused). If LPI is performed when glaucomatous damage has already occurred, the TM is severely damaged and LPI by itself is not likely to lead to sufficiently reduced IOP, even if the angle widens. In a retrospective analysis of 80 such eyes followed for 5 years on average, all eyes required IOP-lowering therapy following LPI, 70% medications with or without laser and 30% filtration surgery. It seems than, that the recommendation to perform prophylactic LPI in order to prevent angle-closure glaucoma is based more on pathophysiological evidence and logical thinking than on clinical evidence. This is reminiscent of the situation regarding the question of need to treat isolated elevated IOP prior to the publication of the ocular hypertension treatment study.

So who are we going to treat with prophylactic LPI? There is one category of eyes for which there is clear agreement— fellow eye in a patient who has had an episode of acute angle closure. In virtually all cases the anatomy of both eyes is similar, the angle in the fellow eye is very narrow or closed, and as mentioned above is at a significant risk to develop a similar episode, this risk potentially reduced to zero with an LPI.

Another category of eyes for which there is consensus on the need for LPI consists of those eyes in which there is clear evidence for a process of chronic angle closure, namely the presence of PAS. PAS are unequivocal evidence for chronic touch between the iris and inner eye wall. In most eyes with PAS, the rest of the angle circumference is narrow or appositionally closed, and so it is logical to perform LPI in order to cause widening of these parts of the angle, prevent further formation of PAS, and ultimately elevation of IOP and glaucoma. (I refer to eyes with primary chronic angle closure, not when PAS is secondary to such conditions as uveitis or anterior segment neovascularization).

Questions usually arise when the above 2 scenarios are not present, and the angle is diagnosed as “narrow” or “occludable”. These are subjective terms used to describe an angle perceived to be at high-risk for closure. Since these eyes are much more than those in the above 2 groups, I would like to provide some clarifications that shall facilitate the diagnostic and therapeutic decision making. Again, this situation can be analogized to one from the open angle glaucoma world, namely that until recently an IOP higher than 21 mmHg was considered an indication for treatment, whereas with progressive accumulation of data and its understanding, today we recognize the importance of central corneal thickness, diagnosis of optic nerve status, etc. Similarly, regarding angle closure and its treatment, there are findings and nuances that can guide us in limiting the number of eyes for which to recommend a prophylactic LPI.

I recommend that clinicians abandon the above terms in everyday clinical practice, since they are vague, do not clearly describe the clinical condition, and thus do not allow a common language among different clinicians and investigators. If an angle is “narrow” but never closes it should be diagnosed and treated differently from an angle in which the iris is actually touching the trabecular meshwork, intermittently or constantly, in part or all the circumference. Hence, I suggest that the clinician determine not whether the angle is “narrow” but rather “closed”, and only in the latter case consider treatment. A closed angle is diagnosed when the clinician observes during gonioscopy a clear apposition of the iris and inner eye wall, even if only in part of the circumference. Rather than hint at an arbitrary risk of angle closure, this finding provides unequivocal evidence of an abnormal anatomic condition with a logical potential for TM damage from continued friction with the iris, and irreversible closure if PAS is formed. This conceptual definition relies on correctly performed and interpreted gonioscopy.

When gonioscopy is performed, if the observer does not see the anatomic angle landmarks, this does not necessarily imply that the angle is closed. Rather, the convex iris may hide the open angle from view. To distinguish between these 2 conditions the examinee must be asked to look in the direction of the mirror at which the examiner is looking. Often, this simple
maneuver—which should be performed routinely—will reveal that an angle which was considered closed is actually open; this is why sometimes a patient is referred for LPI and is returned with the recommendation for observation only. Conversely, the examiner may diagnose the angle as open when observing a pigmented band and thinking it is the TM, when in fact the angle is closed and the band being visualized—deposition of pigment on the cornea—is anterior to the TM. This occurs mainly in the inferior angle, and may easily be distinguished using indentation gonioscopy, detailed below, that opens the angle and exposes the true TM. In addition, I recommend that gonioscopy be performed in a darkened room, with the height of the slit lamp beam reduced to 1 mm and aimed at the angle in such a manner that it does not cross the pupil border. The logic behind this is clear—light caused pupil constriction, hence iris thinning, and widening of the angle. It is possible that an angle will be closed in dark but open in light ambient conditions. In these eyes the examiner will erroneously diagnose the angle as open only because gonioscopy was performed under incorrect conditions. Such changes in the angle between dark and light conditions have been documented by several imaging devices.\(^{19,20,21}\) Importantly, even when an open angle is seen during gonioscopy, intermittent closure may be suspected if irregular pigment spots are observed on the TM. This is especially true if immediately next to such a spot on the TM there is a pigmented patch or nevus on the iris, thus clearly implying intermittent touch of the two. Again, I presume that chronic intermittent touch may potentially lead to formation of PAS and TM dysfunction.

I recommend that a Zeiss-type gonioscopy lens rather than a Goldmann-type one be used. It’s easier for routine use since it does not require the use of a coupling gel, the area of contact with the cornea is smaller, and the 4 mirrors provide a full-circumference view of the angle with minimal or no rotation of the lens. Some skill is required in maintaining the lens on the eye, but after this is acquired, the examination is quick and easy, and only as such can potentially be incorporated into the “routine” of eye examination. Only this type of lens allows indentation gonioscopy. When iris-TM touch is observed, the examiner presses the lens against the cornea (“indents” the cornea), thus increasing the pressure in the anterior chamber and forcing aqueous into the angle. If Iris-TM touch is reversible (appositional) this maneuver will force it open and angle details will be revealed to the examiner. If closure is irreversible (synechial) there will be no change in angle visualization. This distinction is mandatory, since performing LPI is sensible only if there are parts of the angle that are closed reversibly; if all the angle circumference is closed with synechiae LPI is not expected to bring about any change. Indentation also allows distinction between angle closure that is secondary to pupillary block or other different mechanisms, such as plateau iris syndrome or phacomorphic angle closure. In addition to observing an iris contour that is characteristic of each of these conditions, when angle closure is secondary mainly to pupillary block, the examiner performing indentation pushes the iris back against aqueous in the posterior chamber—this requires little manual force. When the examiner needs to push the iris back together with the ciliary body (plateau iris) or lens (phacomorphic), greater force is required and this can be appreciated by the experienced examiner. The presence of these other mechanisms does not negate performance of LPI, since some component of pupillary block frequently coexists. But their recognition provides a better diagnostic and prognostic picture, namely anticipation that LPI may not bring about angle opening.\(^{22}\) Such cases are not rare, and so following LPI the angle must be reassessed. If LPI did not widen the angle, other treatment modalities may be considered such as laser peripheral iridoplasty, pilocarpine, lens removal or goniosynechialysis.

Performance and interpretation of gonioscopy are subjective. Objective and quantitative assessment of the angle is possible with imaging instruments such as ultrasound biomicroscopy or anterior segment OCT. One picture is worth a thousand words when it allows the clinician not only precise objective diagnosis but also clear demonstration to the patient of his problematic anatomy, the need for LPI, and subsequently its beneficial effect. However, gonio-phobes can hardly rejoice, since these instruments are expensive and not widely available and so currently are, at best, complimentary to gonioscopy but certainly not a replacement.

In conclusion, chronic angle closure is much more common than acute episodes of angle closure, and so is the main prevention target by LPI. Routine use of gonioscopy allows us to identify those eyes that are at high-risk to develop these conditions. With skillful performance of gonioscopy and correct interpretation of its findings, specifically unequivocal demonstration of iris touching the inner eye wall, we may be able to recommend the performance of prophylactic LPI only in those eyes that truly need it.

REFERENCES


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