

Glaucoma — Optic Nerve Head Changes and Treatment

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Morphologic Risk Factors for Progressive Glaucomatous Optic Nerve Head Changes

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A prospective clinical observational study was performed to evaluate which morphologic features of the optic disc are risk factors or indicators for progressive neuroretinal rim loss in chronic open angle glaucoma (COAG). Progression of glaucoma was defined as loss of neuroretinal rim.

The study included 394 eyes of 257 patients with COAG and the mean follow up was 31.8 months. All patients underwent repeated qualitative and morphometric evaluation of colour stereo optic disc photographs.

Progression of optic nerve changes was detected in 42 eyes (11%). At baseline, the neuroretinal rim was significantly smaller ($p = 0.03$) and the beta zone of parapapillary atrophy was significantly greater ($p = 0.04$) in the patients who progressed than in the non-progressing group. However, there were no significant differences between the groups in size and shape of the optic disc, optic cup depth, alpha zone of the parapapillary atrophy, and diameter of the retinal blood vessels. Multiple Cox-regression analysis revealed that glaucoma progression was significantly dependent on the area of the neuroretinal rim and the beta zone of parapapillary atrophy.

Conclusion

Important morphological risk factors or indicators of progression of the glaucomatous appearance of the optic nerve head are small size of the neuroretinal rim and large area of the beta zone of parapapillary atrophy.

Blue Arc Entoptic Phenomenon for Detecting Glaucomatous Visual Field Loss

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The blue arc phenomenon is an entoptic response from the nerve fibre layer. Although patients with advanced monocular glaucoma and good visual acuities who are able to see blue arcs in their healthy eyes but not in their glaucomatous eyes have been reported, the potential utility of the blue arc phenomenon in testing for nerve fibre layer function has been largely ignored. The

purpose of this study was to determine the specificity and sensitivity of the blue arc test for detecting perimetric visual field loss due to glaucoma in a clinical setting.

Seventy eight non-consecutive patients from a tertiary care glaucoma clinic who met the inclusion criteria were selected. Glaucomatous visual field loss was defined as one or both eyes demonstrating an abnormal hemifield test on the Humphrey visual field analyser.

The stimulus for the blue arc test was presented on a standard computer video display. A demonstration program was run first, showing the stimulus and simulated blue arcs. Each patient was then light-adapted for 2 minutes and dark adapted for 1 minute. Patients were then asked to fixate on an X while the stimulus was presented. The stimulus was a vertical red slit on a black background, located 2.3° nasal to fixation, 0.86° wide, and extending 5° vertically above and below the horizontal. The stimulus intensity was 5 Cd/m^2 . The stimulus was presented 10 times for 0.5 seconds at 2-second intervals. The patient was again light- and dark-adapted, and the fellow eye tested in the same manner.

Results

The sensitivity and specificity of the blue arc test for detecting glaucomatous visual field loss were 78% and 73%, respectively. The probability of seeing the blue arcs decreased with increasing cup/disc ratio (Figure 1),



Figure 1. Effect on the blue arc test of cup/disc ratio.

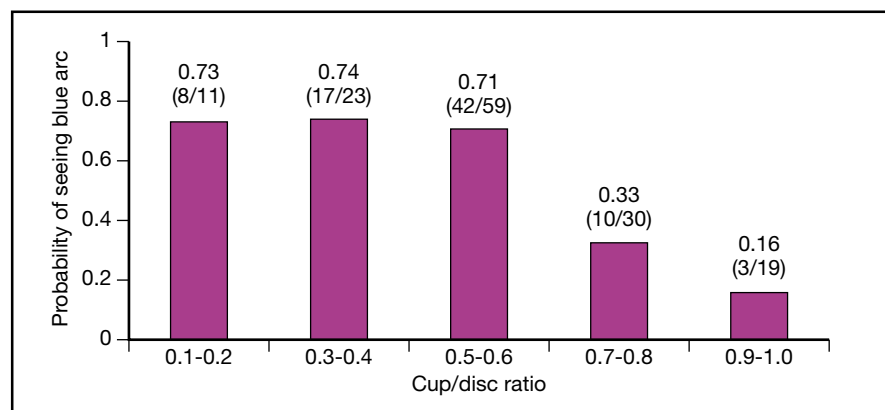


Figure 2. Effect on the blue arc test of mean deviation.

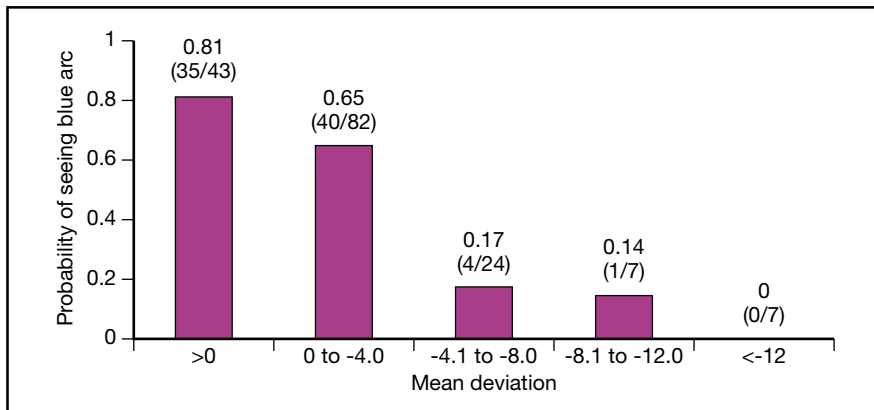
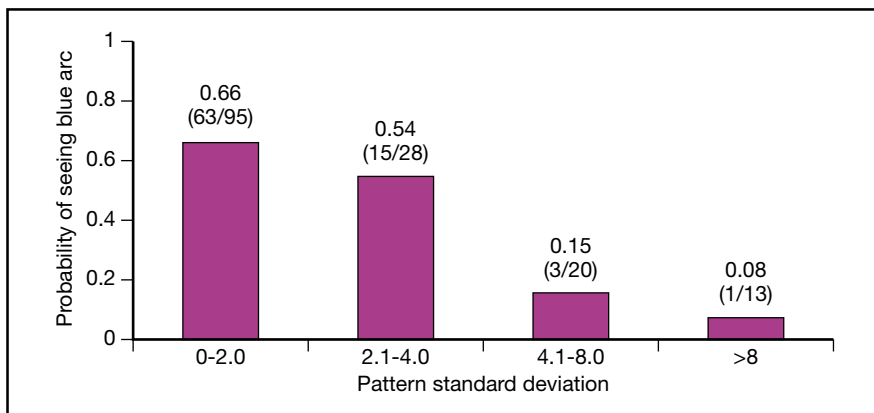


Figure 3. Effect on the blue arc test of pattern standard deviation.



increasing mean deviation (Figure 2), and increasing pattern standard deviation (Figure 3). Patients with pupils of 1 to 2 mm were less likely to see the blue arcs than those with larger pupils. However, this may be confounded by the higher proportion of patients with glaucoma in this subset of patients. Patients with cataract were also less likely to see the blue arcs.

The effect on the blue arc test of cup/disc ratio, mean deviation, and pattern standard deviation, as well as its good sensitivity and specificity in detecting an abnormal glaucoma hemifield test suggests that this test correlates with nerve fibre layer function. Indeed, this test may be uniquely suited to early detection of central glaucomatous nerve fibre layer damage since blue signals travel in larger diameter axons that are more susceptible to damage from high IOPs than small axons. The fact that the entoptic arcs are blue suggests

that this test may be selectively testing for the function of these more susceptible axons enabling the detection of early dysfunction.

In addition, the blue arc test stimulates nerve fibre layer and retinal cells within the central 3° of the visual field. Although central glaucomatous visual field defects tend to occur in the later stages, it is known that ganglion cell loss occurs centrally even early in the disease. Central visual field defects are likely to occur later in the course of the disease than peripheral defects because of the higher density of ganglion cells in this area, suggesting that a higher proportion of ganglion cell loss is required centrally compared with peripherally for equal field losses. The fact that some patients with glaucomatous visual field defects not involving the central 5° are unable to see the blue arcs suggests that this test may be more sensitive for detecting

central nerve fibre layer dysfunction than Humphrey visual field testing.

Conclusion

The blue arc entoptic phenomenon is suppressed in eyes with nerve fibre layer dysfunction secondary to glaucoma, showing good sensitivity and specificity for detecting glaucomatous visual field defects. The test is easy and rapid for patients to perform, taking less than 30 seconds for each eye.

Latanoprost for Normal Tension Glaucoma



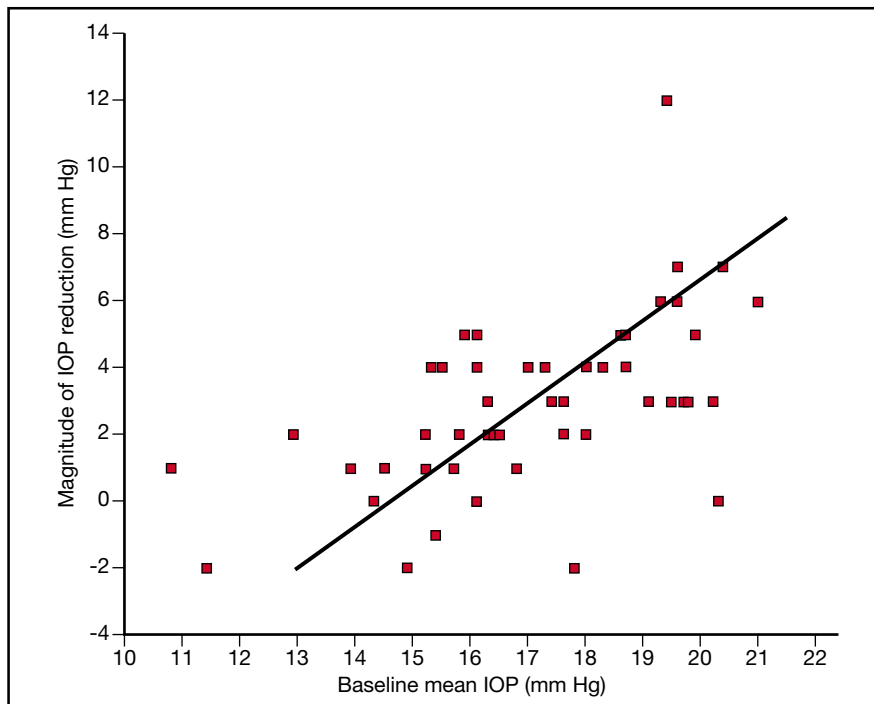
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Normal tension glaucoma (NTG) accounts for approximately 25 to 30% of open angle glaucoma. While the pathogenesis of NTG remains an enigma, a reduction in intra-ocular pressure (IOP) by 20 to 30% from baseline has been shown to slow the rate of progression. Fistulising surgery, with or without antimetabolites is an effective method of lowering IOP. However, potential complications, particularly cataract, may mask the overall visual benefit of IOP reduction and medical treatment represents a potentially safer alternative without the adverse visual effects.

Treatment with pilocarpine, timolol, or betaxolol has had limited success due to side effects and/or a poor ocular hypotensive effect in NTG. Latanoprost has been found to be effective for the treatment of high pressure open angle glaucoma and studies in NTG show that latanoprost 0.005% daily reduces IOP by 18 to 21.3% in the short-term. This study was performed to determine the long-term effect of latanoprost on diurnal IOP in NTG.

In this trial, 81 patients with the following features were randomised to receive latanoprost 0.05% (n = 55) once daily or no treatment (n = 26):

Figure 1. Decrease in mean diurnal intraocular pressure (IOP) following treatment with latanoprost for normal tension glaucoma.



- glaucomatous optic disc changes and visual field defects characteristic of glaucoma
- no recorded IOP >24 mm Hg in either eye during a period of routine baseline daytime IOP phasing.

After a minimum of 6 months, each patient underwent a second IOP phasing period. Phasing consisted of hourly IOP measurements between 08.00 and 17.00 hours using an electronic hand held tonometer.

Results

The mean duration of treatment was 10.5 months (range, 6 to 29 months). A 17.4% decrease in mean diurnal IOP and a 19.5% decrease in maximum diurnal IOP was found in the group receiving latanoprost. Forty percent of treated patients achieved at least a 20% decrease in mean diurnal IOP and the magnitude of reduction in mean IOP correlated with the pretreatment baseline mean IOP (Figure 1). Interestingly, treated patients with a higher initial mean diurnal IOP tended to achieve a

greater magnitude of IOP reduction. No statistically significant IOP changes were found in the control group.

In Conclusion

Latanoprost appears to have a sustained IOP lowering effect in patients with NTG, with 40% of treated patients achieving a satisfactory decrease in mean diurnal IOP of at least 20%. This result, together with its favourable side effect profile, suggests that latanoprost represents a suitable treatment option for NTG.

Concomitant Antiglaucoma Medications and Oral Non-steroidal Anti-inflammatory Drugs

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Prostaglandin F_{2α} potentiates uveoscleral outflow. However, endogenous production of prostaglandins can be interrupted by

non-steroidal anti-inflammatory drugs (NSAIDs) via cyclo-oxygenase inhibition. Indeed, indomethacin has been shown to block the ocular hypotensive effect of epinephrine.

NSAID use is widespread among elderly populations, who may also be taking anti-glaucoma medications. For this reason, a trial was performed to ascertain whether any clinically relevant changes in intraocular pressure (IOP) lowering, visual function, or ocular perfusion occur in patients taking latanoprost or brimonidine plus NSAID therapy.

All ocular hypotensive medication was stopped 3 weeks prior to baseline, at which point 20 patients were randomised to receive either latanoprost 0.005% once daily or brimonidine 0.2% twice daily in the right eye. After 1 week, the left eye was given the other treatment (i.e. that not used for the right eye), while the right eye continued with the original regimen. After 1 week of bilateral topical treatment, oral indomethacin 25 mg 4 times daily was introduced and coadministered with the eye drops for 2 weeks.

Results

After 1 week of treatment, the IOPs in the treated eyes had decreased by 14% with brimonidine ($p = 0.004$) and by 25% with latanoprost ($p < 0.0001$) [Table 1]. After 2 weeks of co-therapy with indomethacin, brimonidine-treated eyes had a slightly raised IOP, not significantly different to the baseline value ($p = 0.3$), while latanoprost-treated eyes retained an IOP similar to that prior to the start of indomethacin therapy ($p = 0.02$) [Table 1].

Pulsatile ocular blood flow (POBF) increased significantly with latanoprost by $296 \pm 82 \mu\text{l}/\text{min}$ ($p = 0.002$), while there was no significant change with brimonidine ($72 \pm 57 \mu\text{l}/\text{min}$) after 1 week.

After 2 weeks coadministration with indomethacin, POBF had further increased



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Table 1. Intraocular pressure lowering effect of latanoprost and brimonidine with concomitant indomethacin.

	Latanoprost (mm Hg)	Brimonidine (mm Hg)
Baseline	18.5 ± 1.3	19.1 ± 0.9
Week 1	13.8 ± 1.1	16.5 ± 1.3
Week 4	13.9 ± 1.3	16.9 ± 1.3

in the latanoprost-treated eyes, to 1164 ± 94 µl/min, but not in the brimonidine treated eyes, resulting in a difference

of 303 µl/min between the 2 groups (p = 0.004).

Retinal microcirculation increased

significantly with latanoprost alone, but was not significantly elevated with either drug during co-therapy with NSAIDs.

Conclusion

Indomethacin therapy coincided with a loss of significance of the ocular hypotensive effect of brimonidine, but did not appear to alter that of latanoprost.



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2nd Biennial Meeting of the South East Asian Glaucoma Interest Group (SEAGIG)

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