Research Article

Glaucoma with hypertension: a comparison of treatment modality

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Abstract

Background: As the intraocular pressure (IOP) increases, the risk of open-angle glaucoma (OAG) substantially increases. However, individuals with systemic hypertension at baseline exhibit one-half of the relative risk. The range of IOP fluctuation is larger in patients with untreated glaucoma; but, as some other studies deny it as independent risk factor, further studies are warranted. It is also established that drug effect varies with the timing of application. Thus, effect of amlodipine and atenolol should also vary.

Objective: To compare the efficacy/safety of amlodipine versus atenolol in the glaucomatous hypertensive people using IOP control as the primary endpoint.

Materials and Methods: IOP maxima, minima, and fluctuation were chosen as three separate outcome variables while minima, maxima, and fluctuations of systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean ocular perfusion pressure (MOPP) as predictor variables. The criteria and methods were decided as in the study by Choi et al. (Invest Ophthalmol Vis Sci 2006;47(3):831–6), with some modifications. We included freshly diagnosed patients of coexistent "hypertension + glaucoma" and used amlodipine (5 mg OD)/atenolol (50 mg OD) + timolol (1 drop; 0.25% solution twice daily).

Result: Baseline parameters showed no significant difference among the two groups. Amlodipine significantly changed all the three IOP and SBP parameters. SBP and DBP fluctuations increased but circadian MOPP fluctuation (CMF) decreased. Atenolol significantly changed all the three IOP parameters. Although peak SBP did not vary significantly, least SBP decreased and SBP fluctuation increased. DBP fluctuation and CMF decreased. Amlodipine and atenolol differed significantly in fluctuation effects—while amlodipine bettered on DBP fluctuation, atenolol bettered on IOP, SBP, and CMF fluctuations.

Conclusion: Instead of SBP or DBP, their fluctuations show more effects. Atenolol is better than amlodipine in glaucomatous hypertensive people.

KEY WORDS: Glaucoma, hypertension, intraocular pressure (IOP), treatment modalities

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Introduction

As the baseline intraocular pressure (IOP) increases, the risk of open-angle glaucoma (OAG) substantially increases. However, individuals with systemic hypertension at baseline exhibit one-half of the relative risk, suggesting that hypertension does not increase (and may decrease) the 4-year risk of OAG.[1]

Numerous studies of patients with normotensive glaucoma (NTG) support the hypothesis that other vascular factors are significantly involved in the development of the disease.[2] Migraine, blood transfusion, Raynaud's phenomenon, and nocturnal blood pressure (BP) decrease are some of the vascular risk factors.[3]

Lower perfusion pressure (PP = BP – IOP) at baseline leads to about threefold increase in the relative risk, which confirms the vascular supposition of OAG pathogenesis.[1] At the same time, most ocular hemodynamic parameters are significantly lower in the primary OAG/ocular hypertension (POAG) group compared with the healthy control group ($p < 0.001$ each).[4]

Recent studies have suggested that the range of IOP fluctuation is larger in patients with untreated glaucoma and that large diurnal variation in IOP is an independent risk factor for the development of glaucoma.[5,6] But, as some other studies deny it as independent risk factor,[7] further studies are warranted.

A 5% to 10% decrease or dipping in the physiological nocturnal BP is exhibited by almost two-thirds of the individuals of the general public. The rest of the population is categorized as either nondippers or overdippers.[2]

It is also known that IOP increases significantly with increasing systolic and diastolic blood pressures (SBP and DBP, respectively).[8] However, diurnal mean ocular perfusion pressure (MOPP) remains higher in overdippers.[2]

It has been established that when compared with healthy people, patients with NTG exhibit significantly higher nocturnal BP reductions and that higher decreases in nocturnal BP result in enhanced advancement of glaucoma.[2]

Thus, it can be hypothesized that, comparatively, extreme decrease in OPP in overdippers can result in short-term ischemia of ocular tissue and subsequently by reperfusion damage,[2] resulting in worse prognosis in overdippers.[9]

Moreover, compared with healthy persons, there is a reversible vasospasm, especially within the ocular vasculature of patients with NTG. It could be hypothesized that variation in blood flow can play a causal role in the pathogenesis of NTG.[2]

A decrease in sympathetic activity at nights with a decreased level of circulating catecholamine hormones can lead to nocturnal BP reduction.[10] Therefore, enhanced level (such as in atherosclerosis, vasospastic disorders, or insufficient antihypertensive therapy) or absence (such as in patients with diabetes mellitus, orthostatic hypotension, or corticosteroid therapy) of nocturnal BP reduction can be observed in patients with sympathetic dysfunction.[2]

Patients with untreated POAG reveal higher percentage of reduction in diurnal MOPP than that by the normal subjects, which attributes that comparative alteration in diurnal MOPP may be a threat factor for POAG.[11]

It is also established that drug effect varies with the timing of application. At sleeping time, there is a reduction in the cardiovascular sympathetic activity and enhancement in the renin secretion that can possibly act as the controller of BP.[12]

The usage of drugs with comparatively nondependent action (e.g., diuretics and calcium blockers) of these systems can be predicted to elicit alike responses during the asleep and awake hours, which forms the scenario in the elderly male patients with hypertension.[12]

Conversely, the reactions to drugs that impact these systems can differ depending on a person's status, that is, awake or asleep. This seems to be the scenario in which there is less reaction to β-blockers and enhanced reaction to ACE inhibitors during the sleeping period.[12]

The aim of this study was to compare the efficacy/safety of amlodipine versus atenolol in the glaucomatous hypertensive people using IOP control as the primary endpoint. The drugs were especially chosen to ascertain the chronopharmacology of antihypertensive drugs, if any, as already discussed.

Lower central corneal thickness (CCT) indicates POAG or NTG, while higher CCT indicates ocular hypertension as per established norms.[13] But, some recent studies correlated CCT only to IOP and not to POAG/primary angle-closure glaucoma.[14] This is the reason for age and CCT were planned to be recorded to ascertain as probable confounders.

Materials and Methods

In the valued opinion in Choi et al.,[2] "whether antihypertensive treatment has beneficial effect on circadian MOPP fluctuation (CMF) by flattening circadian BP fluctuation or not could be another subject for future research" and here it is!

For the study, IOP maxima, minima, and fluctuation were chosen as three separate outcome variables while minima, maxima, and fluctuations of SBP, DBP, and MOPP as predictor variables.

Mostly, the criteria and methods were decided as in Choi et al.[2] The *planned basic difference* of the approach was in the effect of antihypertensive treatment and/or modulation of BP otherwise, which, as a known bias, was overlooked in the reference study.[2] Moreover, we included only cases of POAG (not NTG as in the reference study).

Inclusion/Exclusion Criteria

The eligible criteria included patients with glaucomatous optic nerve form, with diffuse or focal neural rim thinning, hemorrhage, enlarged cupping, or nerve fiber layer faults suggestive of glaucoma along with equivalent visual field loss, on several measurements (at least three independent results).[2]

Goldmann applanation tonometry (GAT) was involved in tonometer model AT900 from Haag-Streit Diagnostics. Slit-lamp and gonioscopic examination (Viewlight CSL-4U from optivision2020) were supplemented.

Exclusion criteria included patients on antihypertensive or other hemodynamically active drugs, with indication of intracranial or otolaryngeal lesion, with history of massive hemorrhage or hemodynamic crisis, with previous or current use of antiglaucoma drugs, with any other ophthalmic disease that could lead visual field faults, or with a history of diabetes mellitus.

We tried to find out freshly diagnosed patients of coexistent "hypertension + glaucoma" and used the "antihypertensive + antiglaucoma" treatment as an intervention, while Choi et al.[2] included patients already diagnosed of hypo/hypertension and being treated accordingly.

Patients of mild or moderate glaucoma (code 365.71 and 365.72) were included, while those of severe glaucoma (code 365.73) and/or those requiring surgery were excluded from the study.[15] All procedures conformed to the Declaration of Helsinki and involved due informed consent from patients and permission from institutional ethical committee.

Thus, finally 50 patients were selected who were randomly assigned to two groups: receiving calcium channel blocker amlodipine (Amlosun® from Sun Pharma; 5 mg OD, at 10 a.m.) and receiving selective β_1-blocker atenolol (Atezen® from Zenith Pharmaceuticals; 50 mg OD, at 10 a.m.), with 25 subjects in each group. For glaucoma, 1 drop (0.25% solution) of timolol maleate (Timoptic from Merck) was instilled in the affected eye(s) twice daily (10 a.m. and 10 p.m.) in all the subjects.

Central Corneal Thickness (CCT)

It was measured three times by ultrasonic pachymetry (DGH-550; DGH Technology, Inc., Exton, PA) in all the patients on the first and the last (after a month) visits, and the average in each patient was calculated.

The affected eye was selected in patients with unilateral disease. In bilateral disease, for this study purpose, only right eye was selected for the study to avoid the statistical problem of inherently correlated data and selection bias.

Visual field examinations

They were performed with the 24-2 full-threshold program on the Humphrey field analyzer (Carl Zeiss Meditec, Inc., Dublin, CA). Visual field data for analysis included mean deviation and corrected pattern standard deviation (CPSD).

CPSD is a measure of how much the total shape of the patients' hill of vision deviates from the shape of the hill of vision of healthy people of the patients' age.[16] The criteria for glaucomatous visual field defects were defined as by Choi et al.[2]:

1. a cluster of three points (excluding rim area) with a probability of <5% on a pattern deviation map in at least one hemifield and comprising at least one point with a probability of <1%;
2. a cluster of two points with a probability of <1%;
3. glaucoma hemifield test more than 99% of age-specific normal limits; or
4. CPSD outside 95% of the normal limit.
5. Reliable visual field was defined as having a false-positive error less than 33%, a false-negative error less than 33%, and a fixation loss less than 20%.

IOP/BP measurement

Twice 24-h admission was made mandatory for inclusion—first, at the start of study and, second, after a month. All the IOP measurements were taken with a slit lamp-mounted GAT with the patient in the sitting position. IOP was measured after the subject had been seated for at least 3 min and was adjusted by 3 mm Hg for every 50 m that the CCT deviated from 530 m.

SBPs and DBPs were measured with a brachial Diamond Mercury Free LED Deluxe Blood Pressure Apparatus BPDG141. Sphygmomanometer was positioned on the upper left arm after resting the subject for at least 3 min. Patients were advised to cease any physical actions that could impact BP for half an hour before the reading.

Meals were given at two timings: at 6:30 p.m. and 7:30 a.m., which were devoid of any alcohol or caffeine content. The estimation of mean arterial pressure (MAP) was done applying the formula: MAP = DBP + [1/3 (SBP – DBP)]. Thereby, the estimation of MOPP at an indicated time was enabled from the difference between MAP and IOP replaced for venous pressure as follows: MOPP = 2/3 * (MAP – IOP).

The average MOPP from 8 p.m. to 6 a.m. of the next day gave the nocturnal MOPP values, while the diurnal MOPP was given by the average MOPP at the remaining times. The difference between the greatest and the least MOPPs noted during the 24-h period is known as CMF.

However, our study design included parameters much different from the reference[2] research—we took the mean and standard deviation of maximum and minimum IOP, SBP, DBP, and MOPP and mean thereof but not taken mean IOP (averaging maximum and minimum) and so on as our parameters.

Second, we used unpaired *t* test to find the baseline difference in the two groups to be experimented and outcome difference after 1 month. Our subjects were not categorized as overdipper or nondipper; instead, a continuum of measurement was maintained in the calculation.

Arithmetic mean ± standard deviation was taken for each variable and *p*-value with confidence interval was calculated using online GraphPad.[17] Regression analysis for correlation was not done as the sample size was small, and any trend could be, by the best, only an indicator of correlation.

Results

Baseline parameters showed no significant difference among the two groups. Amlodipine significantly changed all the three IOP and SBP parameters. SBP and DBP fluctuations increased but CMF decreased. Atenolol significantly changed all the three IOP parameters. Although peak SBP did not vary significantly, the least SBP decreased and SBP fluctuation increased. DBP fluctuation and CMF decreased. Amlodipine and atenolol differed significantly in fluctuation effects—while amlodipine bettered on DBP fluctuation, atenolol bettered on IOP and SBP fluctuations and CMF.

Discussion

As we observe in Table 1, age and CCT varied very little in the given study duration of 1 month. CCT was on high-normal

Table 1: Group amlodipine (AM) and atenolol (AT) at baseline

	AM ($\mu \pm$ SD) (0 month)	AT ($\mu \pm$ SD) (0 month)	Two-tailed *p*-value	Confidence interval
Age	35.57 ± 8.34	39.34 ± 5.12	0.0600	–7.7 & 0.16
CCT(μ)	566 ± 89.85	563 ± 82.72	0.9028	–46.1 & 52.11
Peak IOP	29.16 ± 2.97	28.72 ± 4.47	0.6837	–1.7 & 2.6
Least IOP	22.92 ± 3.01	22.8 ± 3.53	0.8976	–1.7 & 1.99
IOP Fluct	6.24 ± 2.14	5.92 ± 2.23	0.6071	–0.9 & 1.56
Peak SBP	166.24 ± 24.39	167.56 ± 42.32	0.9042	–23.2 & 20.6
Least SBP	159.84 ± 21.91	160.72 ± 29.73	0.9057	–15.7 & 13.9
SBP fluct	6.4 ± 0.98	6.84 ± 1.99	0.3263	–1.3 & 0.45
Peak DBP	94.24 ± 18.26	95.12 ± 21.51	0.8767	–12.2 & 10.5
Least DBP	91.84 ± 20.02	92.8 ± 21.05	0.8695	–12.6 & 10.7
DBP fluct	2.4 ± 0.35	2.32 ± 0.50	0.5153	–0.16 & 0.32
DMOPP	48.7 ± 7.49	48.9 ± 7.61	0.9258	–4.5 & 4.1
NMOPP	46.6 ± 6.38	45.9 ± 6.13	0.6942	–2.8 & 4.2
CMF	2.78 ± 0.47	3 ± 0.45	0.1364	–0.51 & 0.07

Table 2: Group amlodipine (AM) at baseline and after a month

	AM ($\mu \pm$ SD) (0 month)	AM ($\mu \pm$ SD) (1 month)	Two-tailed *p*-value	Confidence interval
Age	35.57 ± 8.34	35.58 ± 8.31	0.9966	–4.7 & 4.7
CCT(μ)	566 ± 89.85	569 ± 90.04	0.9066	–54.1 & 48.1
Peak IOP*	29.16 ± 2.97	22.12 ± 3.33	0.0001	5.2 & 8.8
Least IOP*	22.92 ± 3.01	18.52 ± 3.86	0.0001	2.4 & 6.3
IOP Fluct*	6.24 ± 2.14	3.6 ± 1.76	0.0001	1.5 & 3.7
Peak SBP*	166.24 ± 24.39	147.16 ± 27.56	0.0126	4.3 & 33.8
Least SBP*	159.84 ± 21.91	136.72 ± 24.44	0.0010	9.9 & 36.3
SBP fluct*	6.4 ± 0.98	10.44 ± 2.25	0.0001	–5.03 & –3.05
Peak DBP	94.24 ± 18.26	88.32 ± 16.55	0.2356	–4.0 & 15.8
Least DBP	91.84 ± 20.02	84.64 ± 22.09	0.2331	–4.8 & 19.2
DBP fluct*	2.4 ± 0.35	3.68 ± 0.46	0.0001	–1.5 & –1.04
DMOPP	48.7 ± 7.49	46.9 ± 8.54	0.4321	–2.77 & 6.3
NMOPP	46.6 ± 6.38	45.4 ± 5.89	0.4929	–2.3 & 4.7
CMF*	2.78 ± 0.47	1.5 ± 0.36	0.0001	1.04 & 1.52

*indicate significant values ($p < 0.05$) and confidence interval not across 0.

Table 3: Group atenolol (AT) at baseline and after a month

	AT ($\mu \pm$ SD) (0 month)	AT ($\mu \pm$ SD) (1 month)	Two-tailed *p*-value	Confidence interval
Age	39.34 ± 5.12	39.35 ± 5.11	0.9945	–2.9 & 2.9
CCT(μ)	563 ± 82.72	562 ± 88.71	0.9673	–47.7 & 49.8
Peak IOP*	28.72 ± 4.47	21 ± 5.29	0.0001	4.9 & 10.5
Least IOP*	22.8 ± 3.53	19.04 ± 4.16	0.0012	1.56 & 5.95
IOP Fluct*	5.92 ± 2.23	1.96 ± 0.79	0.0001	3.0 & 4.9
Peak SBP	167.56 ± 42.32	147.56 ± 39.24	0.0896	–3.2 & 42.2
Least SBP*	160.72 ± 29.73	138.92 ± 41.52	0.0379	1.3 & 42.3
SBP fluct*	6.84 ± 1.99	8.64 ± 2.48	0.0068	–3.08 & –0.52
Peak DBP	95.12 ± 21.51	88.88 ± 19.78	0.2910	–5.5 & 17.99
Least DBP	92.8 ± 21.05	84.39 ± 20.96	0.1634	–3.5 & 20.3
DBP fluct*	2.32 ± 0.50	4.24 ± 0.38	0.0001	–2.2 & –1.67
DMOPP	48.9 ± 7.61	46.3 ± 8.36	0.2559	–1.9 & 7.1
NMOPP	45.9 ± 6.13	45.9 ± 6.87	1.000	–3.7 & 3.7
CMF*	3 ± 0.45	0.4 ± 0.01	0.0001	2.4 & 2.8

*indicate significant values ($p < 0.05$) and confidence interval not across 0.

Table 4: Group amlodipine (AM) and atenolol (AT) after a month

	AM ($\mu \pm$ SD) (1 month)	AT ($\mu \pm$ SD) (1 month)	Two-tailed *p*-value	Confidence interval
Age	35.58 ± 8.31	39.35 ± 5.11	0.0592	–7.6 & 0.15
CCT(μ)	569 ± 90.04	562 ± 88.71	0.7830	–43.8 & 57.8
Peak IOP	22.12 ± 3.33	21 ± 5.29	0.3748	–1.4 & 3.6
Least IOP	18.52 ± 3.86	19.04 ± 4.16	0.6489	–2.8 & 1.8
IOP Fluct*	3.6 ± 1.76	1.96 ± 0.79	0.0001	1.86 & 2.41
Peak SBP	147.16 ± 27.56	147.56 ± 39.24	0.9708	–22.2 & 21.4
Least SBP	136.72 ± 24.44	138.92 ± 41.52	0.8204	–21.6 & 17.2
SBP fluct*	10.44 ± 2.25	8.64 ± 2.48	0.0099	1.45 & 3.15
Peak DBP	88.32 ± 16.55	88.88 ± 19.78	0.9140	–10.9 & 9.8
Least DBP	84.64 ± 22.09	84.39 ± 20.96	0.9674	–11.99 & 12.5
DBP fluct*	3.68 ± 0.46	4.24 ± 0.38	0.0001	–0.79 & –0.32
DMOPP	46.9 ± 8.54	46.3 ± 8.36	0.8029	–4.2 & 5.4
NMOPP	45.4 ± 5.89	45.9 ± 6.87	0.7835	–4.1 & 3.1
CMF*	1.5 ± 0.36	0.4 ± 0.01	0.0001	1.95 & 2.24

*indicate significant values ($p < 0.05$) and confidence interval not across 0.

side in both the groups, which might be a reason of the retarded progression of POAG.[14] The difference in AM (amlodipine) and AT (atenolol) group is not significant on these redundant variables or any other count considered so far.

Regarding the individual drug, as per Table 2, 1-month application of amlodipine significantly benefitted all the three IOP parameters. Although peak and trough of SBPs were significantly lowered by 1 month of amlodipine, SBP and DBP fluctuations were rather significantly increased.

Increase in SBP fluctuation might indcate uncompensatory decrease in maximum and minimum SBP. Here may lie the crux—the fluctuation of BP might be more important for ischemia at lower end and reperfusion injury at the upper end.[2] However, CMF was lowered significantly.

As per Table 3, all the IOP parameters significantly improved with atenolol, similar to amlodipine, as given in Table 2. Again, similar to amlodipine [Table 2], atenolol action [Table 3] on fluctuations in SBP and DBP showed significant increases. In addition, atenolol, being a selective β_1-blocker, is less active in the night,[10] and could be avoiding overdipping, which could have induced worse prognosis.[2]

As per Table 3, the significant decrease in CMF owing to 1-month atenolol therapy was similar to that of 1-month amlodipine treatment [Table 2]. But, unlike amlodipine, which significantly decreased maximum and minimum SBPs, maximum SBP is spared by atenolol. This isolated depression of lowest SBP might be owing to selective blockade of β_1, while β_2 action of vasodilation was least effected.

Despite this discrepancy, the quantity of fluctuation in SBP, after 1 month of amlodipine therapy, was greater (10.44 ± 2.25 mm Hg) than that of atenolol (8.62 ± 2.48 mm Hg). Moreover, it might, at least partially, explain why atenolol was significantly better than amlodipine on the three fluctuations including that of IOP, while amlodipine significantly bettered only on DBP fluctuation, as per Table 4.

As Table 4 shows, after 1 month, the difference of amlodipine versus atenolol was seen only in fluctuations—as mentioned earlier—while amlodipine controlled the DBP fluctuations more significantly, atenolol was significantly better on IOP and SBP fluctuations and CMF.

Limitations of the Study

There were several limitations of our study, mostly similar to the base study already done.[2] First, we did not include the data on the progression of glaucomatous damage in the patients. Rather, our data represent the results of IOP inspection alone.

Second, although only patients with dependable visual field indices were included, there is a probability of certain inconsistency in the results, as few patients could have experienced some struggle in carrying out the visual field examination for the first time.

Third, the theoretical formula-based estimation of MOPP might not reveal the actual physiological position of ocular perfusion. The ocular blood flow was estimated directly, which could lead to diverse outcome. There is a possibility of a role of autoregulation impact that affects the real blood flow.

Fourth, slight selection bias in our study subjects could have occurred as patients approaching a tertiary hospital exhibit more advanced glaucomatous damage.

Fifth, estimating BP and IOP in the sitting position during nocturnal readings would not reveal reflect the paramount possible physiological status, which we were aware of. A slight variation in results could have been observed if BP and IOP readings were measured in a seamless physiological form at these hours.

Moreover, our sample size was small and relatively less sick overall; in extreme cases, the study finding might differ owing to emergency interventions, problem of attrition, and many other physiological confounders.

Conclusions

In the outcome of amlodipine versus atenolol as antihypertensive drugs in glaucoma patients, instead of SBP or DBP values as such, their fluctuations exhibit more effects, especially the fluctuation of SBP. Once this parameter is better controlled, we can have better control over IOP and CMF too. On that count, atenolol is better than amlodipine in glaucomatous hypertensive people.

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