e-ISSN: 0975-5160, p-ISSN: 2820-2651

Available online on www.ijtpr.com

International Journal of Toxicological and Pharmacological Research 2022; 12(2); 07-12

Original Research Article

Prospective Assessment of the Clinical Correlation of Glaucoma with Systemic Hypertension and its Effect on Visual Morbidity

Nandani Priyadarshini¹, Vikash Vaibhav², Uday Narayan Singh³

¹Senior Resident, Department of Ophthalmology, Nalanda Medical College and Hospital, Patna, Bihar, India

²Senior Resident, Department of Ophthalmology, Anugrah Narayan Magadh Medical College and Hospital, Gaya, Bihar, India

³Associate Professor, Department of Ophthalmology, Nalanda Medical College and Hospital, Patna, Bihar, India

Received: 31-10-2021 / Revised: 28-11-2021 / Accepted: 09-01-2022

Corresponding author: Dr. Nandani Priyadarshini

Conflict of interest: Nil

Abstract

Aim: To evaluate the association between hypertension and occurrence of glaucoma.

Methodology: A prospective study was conducted in the Department of Ophthalmology, NMCH, Patna, Bihar, India from November 2017 to October 2018. including 72 patients between the age groups of 30 to 70 years diagnosed with hypertension. Patients with other systemic diseases or vascular pathologies were excluded from the study. All the patients were followed up for atleast 6 months and the need for regular review visits was explained to them. During the first visit and each follow up opinions regarding the progress of hypertension was obtained from departments of cardiology and internal medicine. The oral hypertensive medication taken by patients were categorized into 5 groups as calcium channel blockers (CCB), diuretics, angiotensin converting enzyme inhibitors (ACE), angiotensin receptor blockers and beta blockers. A detailed history of age, sex, duration of hypertension, history of other co-morbidities and treatment were collected. The participants then underwent a detailed ophthalmological evaluation. IOP measurement was done by applanation tonometry with Goldman Applanation Tonometer. The same procedure was repeated in the other eye. Phasing technique of repeating recordings was done and the average IOP was used in the study.

Results: Among the 72 hypertension patients involved in the study, 40 patients (55.5%) were found to have glaucoma. 39 patients (54.2%) were female, and 33 patients (45.8%) were male. Age group affected was 9.7% between 30-40 years, 15.3% between 41 to 50 years, 23.6% between 51 to 60 years and 51.4% between 61 to 70 years. 51.7% patients taking CCB, 58.8% taking ACE inhibitors, 54.6% taking ARB, 55.6% taking beta blockers, and 66.7% taking diuretics had reduced IOP. The range of IOP in the treated population was between 10-16mmHg and this difference in those on hypertension medications was statistically significant.

Conclusion: It can be concluded from this study that there is a correlation between higher systemic blood pressure and higher intraocular pressure, and this also produces effects to be regarded as risk factors for glaucoma. Patients taking oral hypertensive drugs also had reduced intraocular pressure and prevent further progression of glaucoma.

Keywords: Glaucoma, Ocular, Hypertension, Angiotensin.

ISSN: 0975-5160, p-ISSN: 2820-2651

This is an Open Access article that uses a fund-ing model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0) and the Budapest Open Access Initiative (http://www.budapestopenaccessinitiative.org/read), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction:

Glaucoma is a group of eye diseases which result in damage to the optic nerve and cause vision loss. The most common type is openangle (wide angle, chronic simple) glaucoma, in which the drainage angle for fluid within the eye remains open, with less common types including closed-angle (narrow angle, acute congestive) glaucoma and normaltension glaucoma [1]. Glaucoma is a progressive optic neuropathy with characteristic changes in the optic nerve head and corresponding visual field Currently, glaucoma accounts for 12% of all global blindness with 4.5 million people affected worldwide [2]. Primary open angle glaucoma (POAG) is more frequent in Caucasian and African populations whilst primary angle closure glaucoma (PACG) is more common in Asian ethnicity. Normal tension glaucoma (NTG) is especially prevalent in Japan and Korea [3-5].

Risk factors for glaucoma include increasing age, high pressure in the eye, a family history glaucoma, and use of steroid medication.[1] For eye pressures, a value of 21 mmHg or 2.8 kPa above atmospheric pressure (760 mmHg) is often used, with higher pressures leading to a greater risk.[6, 7] However, some may have high eye pressure for years and never develop damage.[6] Conversely, optic nerve damage may occur with normal pressure, known as normal-tension glaucoma.[8] Intraocular pressure is a function of production of liquid aqueous humor by the ciliary processes of the eye, and its drainage through the trabecular meshwork. Aqueous humor flows from the ciliary processes into the posterior chamber,

bounded posteriorly by the lens and the zonules of Zinn, and anteriorly by the iris. It then flows through the pupil of the iris into the anterior chamber, bounded posteriorly by the iris and anteriorly by the cornea. From here, the trabecular meshwork drains aqueous humor via the scleral venous sinus (Schlemm's canal) into scleral plexuses and general blood circulation.[9] The mechanism of open-angle glaucoma is believed to be the slow exit of aqueous humor through the trabecular meshwork, while in closed-angle glaucoma the iris blocks the trabecular meshwork.[6]

Hypertension and raised intra ocular pressure share an overlapping pathophysiology [10]. Altered sodium transport in distal nephrons ciliary epithelium mediated corticosteroid receptors and enzyme 11βhydroxysteroid dehydrogenase has been found to be a common biochemical process in the occurrence of hypertension and glaucoma [11, 12]. Increasing age is a common risk factor in primary open angle glaucoma and hypertension [13]. Drugs used in the treatment of hypertension are also known to produce changes in the intraocular pressure. Hence the present study was conducted with the aim to evaluate the association between hypertension occurrence of glaucoma.

Materials and Methods:

A prospective study was conducted in the Department of Ophthalmology, NMCH, Patna, Bihar, India from November 2017 to October 2018 .including 72 patients between the age groups of 30 to 70 years diagnosed

with hypertension. Patients with other systemic diseases or vascular pathologies were excluded from the study. Those with hypertension but less than 30 years of age were not enrolled into the study as both glaucoma and hypertension could be due to congenital causes in young individuals.

Methodology

All the patients were followed up for at least 6 months and the need for regular review visits was explained to them. During the first visit and each follow up opinions regarding the progress of hypertension was obtained from departments of cardiology and internal medicine.

Patients were classified as hypertensive based on elevated BP readings of >120/80 mm Hg on two separate occasions according to current American Heart Association [14]. Blood pressure measurements were made over 3 visits and the average of last two measurements was used for analysis. Recording was done with manual sphygmomanometer. The oral hypertensive medication patients taken by categorized into 5 groups as calcium channel angiotensin blockers (CCB), diuretics, converting enzyme inhibitors (ACE),

angiotensin receptor blockers and beta blockers. A detailed history of age, sex, duration of hypertension, history of other comorbidities and treatment were collected.

ISSN: 0975-5160, p-ISSN: 2820-2651

The participants then underwent a detailed ophthalmological evaluation including visual acuity, anterior segment evaluation using slit-lamp bio-microscopy and fundus evaluation using a + 90 D lens/ indirect ophthalmoscope. IOP measurement was done by applanation tonometry with Goldman Applanation Tonometer. Fluorescein was instilled in each eye and the tonometer was set at 10mmHg. Mires were viewed through the prism and measurements were read from the rotating dial. The same procedure was repeated in the other eye. Phasing technique of repeating recordings was done and the average IOP was used in the study.

Results:

Among the 72 hypertension patients involved in the study, 40 patients (55.5%) were found to have glaucoma. 39 patients (54.2%) were female, and 33 patients (45.8%) were male. Age group affected was 9.7% between 30-40 years, 15.3% between 41 to 50 years, 23.6% between 51 to 60 years and 51.4% between 61 to 70 years.

Table 1: Demographic details and presence of glaucoma in hypertensive patients

Variables		Number	%
Gender	Male	33	45.8
	Female	39	54.2
Age (in years)	30-40	7	9.7
	41-50	11	15.3
	51-60	17	23.6
	61-70	37	51.4
Glaucoma	Present	40	55.5
	Absent	32	44.5

The increased incidence of OHT among hypertensives was statistically significant (p value<0.005). In those with OHT,

predominant fundus changes were seen as increased cup disc ratio in 14% and neuroretinal thinning in 8%. Corneal

thickness in patients diagnosed with ocular hypertension was on an average 0.740 +/-0.03mm. Thicker cornea was noted in 28%. Thinner cornea was noted in 4% of patients.

The oral hypotensive medication taken by patients were categorized into 5 groups as calcium channel blockers (CCB), diuretics, angiotensin converting enzyme inhibitors (ACE inhibitors), angiotensin receptor

blockers (ARB) and beta blockers. 51.7% patients taking CCB, 58.8% taking ACE inhibitors, and 54.6% taking ARB, 55.6% taking beta blockers, and 66.7% taking diuretics had reduced IOP. The range of IOP in the treated population was between 10-16mmHg and this difference in those on hypertension medications was statistically significant.

ISSN: 0975-5160, p-ISSN: 2820-2651

Table 2: Correlation between hypertensive medication and IOP reduction

Medication	Gla	Glaucoma		
	Yes	No		
CCB	14 (48.3%)	15 (51.7%)	29 (100%)	
ACE inhibitors	7 (41.2%)	10 (58.8%)	17 (100%)	
ARB	5 (45.4%)	6 (54.6%)	11 (100%)	
Beta blockers	4 (44.4%)	5 (55.6%)	9 (100%)	
Diuretics	2 (33.3%)	4 (66.7%)	6 (100%)	
Total	40 (55.5%)	32 (44.5%)	72 (100%)	

Discussion:

According to the vascular or ischemic hypothesis, glaucomatous damage is caused by, or at least facilitated by, inadequate perfusion of the proximal portion of the optic nerve. In systemic hypertension, this may be the result of increased peripheral resistance in the small vessels. It is often stressed, however, that a reduction in systemic blood pressure may also have a deleterious effect by creating insufficient perfusion pressure in the optic disc. In other words, both systemic hypertension and hypotension could, with different mechanisms, be risk factors for glaucoma. More than one mechanism has been postulated for the possible effects of blood pressure in the pathogenesis of glaucoma. On the one hand, various investigators have found a positive correlation between systemic hypertension and OH [15, 16] Although we realize that blood pressure in the eye is different from blood pressure in the arm, we used brachial diastolic pressure for calculating ocular

perfusion pressure in our study, as was used by other investigators [17-19].

Although high BP seems to be related to increased IOP, low BP, that is, nocturnal hypotension, is a known risk factor in the development and progression of GON [20-23]. But several studies also have demonstrated a weak positive correlation between IOP and blood pressure (BP) [20]. In a prospective long-term study by Tokunaga et al. [24], the relation between nocturnal BP dip and the progression of the visual field defect in NTG and HTG over a period of 4 vears was examined. The degree of nocturnal BP reduction was classified as nondipper (dip of 20%). Interestingly, there was a tendency for the visual field defect to progress not only in the extreme dipper group, but also in the nondipper group. Kashiwagi et al. [25] demonstrated that a lack of the physiologic dipping might be an independent risk factor for progression. The fact that both nondipping and over dipping are associated with GON indicates that an underlying

vascular dysregulation, and not simply a low perfusion pressure, might be causal. Such a general vascular dysregulation might interfere with OBF regulation.

In a report by Wilson et al [26], systemic hypertension, along with being black, would appear to be the main risk factors for glaucoma. In contrast to this, Leske et al [19] believe that glaucoma is more often associated with low levels of systemic blood pressure. They found in their study population that glaucoma was more frequent in patients with minor differences between systemic pressure and IOP. Tielsch et al [17] convincingly demonstrated the existence of an inverse correlation between glaucoma and diastolic perfusion pressure of the ocular tissues.

Systemic hypertension exerts an oxidative stress to the arterial wall which in addition to atherosclerosis can then impair autoregulation such that an elevation of IOP above a criteria level will comprise the ocular blood flow and induce optic nerve damage even though IOP may still be within normal range [27]. Additionally, when hypertensive patients are started on systemic antihypertensive medication, the diurnal variation. postural hypotension, nocturnal dips induced by the systemic medication can further comprise ocular perfusion pressure contributing to further optic nerve hypo perfusion [28].

Conclusion:

It can be concluded from this study that there is a correlation between higher systemic blood pressure and higher intraocular pressure, and this also produces effects to be regarded as risk factors for glaucoma. Patients taking oral hypertensive drugs also had reduced intraocular pressure and prevent further progression of glaucoma.

References:

- "Facts About Glaucoma". National Eye Institute. Archivedfrom the original on 28 March 2016. Retrieved 2 January 2022
- 2. Priority Eye Disease, http://www.who.int/blindness/c auses/priority/en/index7.html, 2011.Retrieved 2 January 2022
- 3. A. Iwase, Y. Suzuki, M. Araie et al., "The prevalence of primary open-angle glaucoma in Japanese: The Tajimi Study," Ophthalmology, vol. 111, no. 9, pp. 1641–1648, 2004.
- 4. C. S. Kim, G. J. Seong, N. H. Lee, and K. C. Song, "Prevalence of primary openangle Glaucoma in central South Korea: The Namil study," Ophthalmology, vol. 118, no. 6, pp. 1024–1030, 2011.
- 5. M. A. Kass, "Normal-pressure glaucoma," American Journal of Ophthalmology, vol. 125, no. 2, pp. 242–244, 1998.
- 6. Mantravadi AV, Vadhar N (September 2015). "Glaucoma". Primary Care. 42 (3): 437–49.
- 7. Rhee DJ (2012). Glaucoma (2 ed.). Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins. p. 180.
- 8. Mi XS, Yuan TF, So KF (16 September 2014). "The current research status of normal tension glaucoma". Clinical Interventions in Aging. 9: 1563–71.
- 9. Alguire P (1990). "The Eye Chapter 118 Tonometry>Basic Science". In Walker HK, Hall WD, Hurst JW (eds.). Clinical methods: the history, physical, and laboratory examinations (3rd ed.). London: Butterworths.
- 10. Langman MJS. Systemic hypertension and glaucoma: mechanisms in common and co-occurrence. Br J Ophthalmol. 2005;89(8):960–3.
- 11. Omoti AE, Enock ME, Okeigbemen VW, Akpe BA, Fuh UC. Vascular risk factors for open angle glaucoma in African eyes.

- Middle East Afr J Ophthalmol. 2009;16(3):146.
- 12. Maurya R. Biomarkers of primary openangle glaucoma. Ind J ClinExpOphth. 2017;3(4):387–388.
- 13. Deokule S, Weinreb RN. Relationships among systemic blood pressure, intraocular pressure, and open-angle glaucoma. Can J Ophthalmol. 2008;43(3):302–7.
- 14. Costa VP, Arcieri ES, Harris A. Blood pressure and glaucoma. Br J Ophthalmol. 2009;93(10):1276–82.
- 15. Leske MC. The epidemiology of openangle glaucoma: a review. Am J Epidemiol 1983; 118:166–91.
- 16. McLeod SD, West SK, Quigley HA, Fozard JL. A longitudinal study of the relationship between intraocular and blood pressures. Invest Ophthalmol Vis Sci 1990; 31:2361–6.
- 17. Tielsch JM, Katz J, Sommer A et al. Hypertension, perfusion pressure, and primary open-angle glaucoma. A population-based assessment. Arch Ophthalmol 1995; 113:216–21.
- 18. Dielemans I, Vingerling JR, Algra D, et al. Primary openangle glaucoma, intraocular pressure, and systemic blood pressure in the general elderly population. The Rotterdam Study. Ophthalmology 1995;102:54–60.
- 19. Leske MC, Connell AM, Wu SY et al. Risk factors for open-angle glaucoma. The Barbados Eye Study. Arch Ophthalmol 1995;113:918–24.
- 20. Bonomi L, Babighian S, Bonadimani M, et al. Correlation between glaucoma and vascular factors, and circumstances leading to the diagnosis of glaucoma. ActaOphthalmolScandSuppl 2000, (232):34—35.
- 21. Leske MC, Wu SY, Nemesure B, et al. Incident open-angle glaucoma and blood

- pressure. Arch Ophthalmol 2002; 120:954—959.
- 22. Hayreh SS, Zimmerman MB, Podhajsky P, et al. Nocturnal arterial hypotension and its role in optic nerve head and ocular ischemic disorders. Am J Ophthalmol 1994; 117:603—624.
- 23. Hayreh SS. Role of nocturnal arterial hypotension in the development of ocular manifestations of systemic arterial hypertension. CurrOpinOphthalmol 1999; 10:474—482
- 24. Tokunaga T, Kashiwagi K, Tsumura T, et al. Association between nocturnal blood pressure reduction and progression of visual field defect in patients with primary open-angle glaucoma or normaltension glaucoma. Jpn J Ophthalmol 2004; 48:380—385
- 25. Kashiwagi K, Hosaka O, Kashiwagi F, et al. Systemic circulatory parameters. comparison between patients with normal tension glaucoma and normal subjects using ambulatory monitoring. Jpn J Ophthalmol 2001; 45:388—396.
- 26. Wilson MR, Hertzmark E, Walker AM, et al. A case-control study of risk factors in open-angle glaucoma. Arch Ophthalmol 1987; 105:1066–71.
- 27. V. P. Costa, E. S. Arcieri, and A. Harris, "Blood pressure and glaucoma," British Journal of Ophthalmology, vol. 93, no. 10, pp. 1276–1282, 2009.
- 28. J. M. Tielsch, J. Katz, A. Sommer, H. A. Quigley, and J. C. Javitt, "Hypertension, perfusion pressure, and primary openangle glaucoma: a population-based assessment," Archives of Ophthalmology, vol. 113, no. 2, pp. 216–221, 1995.