Section: Ophthalmology

Original Research Article

BIOMETRIC ASSESSMENT OF OCULAR RIGIDITY IN GLAUCOMA PATIENTS AND CONTROLS

Sunita Bisht[1], Vivekanand Satyawali[2], Shanti Pandey[3], Vimlesh Sharma[4]

[1]Post Graduate 3rd Year, Department of Ophthalmology, Government Medical College, Haldwani, Nainital, Uttarakhand, India
[2]Associate Professor, Department of Medicine, Government Medical College, Haldwani, Nainital, Uttarakhand, India
[3]Professor, Department of Ophthalmology, Government Doon Medical College, Dehradun, Uttarakhand.
[4]Associate Professor, Department of Ophthalmology, GMC Haldwani, Nainital, Uttarakhand, India

Received : 08/03/2023
Received in revised form : 05/04/2023
Accepted : 18/04/2023

Keywords:
CNS tumors, grading, diagnosis.

Corresponding Author:
Dr. Vimlesh Sharma
Email: drvimlesh.stm@gmail.com

DOI:10.47009/jamp.2023.5.3.209

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2023; 5 (3); 1021-1024

Abstract

Background: Biometric Assessment of ocular rigidity in Glaucoma patient's and control: Objective: To compare oral acetazolamide induced IOP and axial length change in normal and glaucomatous subjects. **Material and Methodology:** The comparative study of "Biometric Assessment of ocular rigidity in Glaucoma patients and controls" was conducted after clearence from Board of Studies and Ethical committee in the Department of ophthalmology, Dr. Sushila Tiwari Government Hospital, Haldwani, Nainital during the period 2020-2022.Hundred subject (50 patients with primary open angle glaucoma and 50 control patients) underwent axial eye length measurements using partial coherence laser interferometry and measurement of IOP using Goldmann tonometry before and two hours after oral intake of 500 mg acetazolamide. Unparied t-test was used to compare the difference in the means. **Result:** A significantly greater (p=0.004) drop in IOP was in glaucoma (mean=2.64) group compared with control (mean= 1.63) groups. The change in axial length was significantly smaller (p=0.022) in the glaucoma group (mean= 0.02) compared with the control (mean= 0.02).**Conclusion:** Our results strongly suggests that the ocular rigidity has increased in patient with glaucoma in comparison to control subjects. Ocular rigidity could play a role in pathogenesis and pathophysiology of glaucoma, determination of ocular rigidity could helpful in detection of glaucoma.

INTRODUCTION

Ocular rigidity is a biomechanical parameter of the eye expressing the elasticity of the globe. It depends mainly on the properties of the cornea, sclera and other components of the outer shell of the eye. Ocular rigidity relates intraocular pressure changes to the corresponding volume changes and is a measure of the resistance that the eye exerts to distending forces.[1] Ocular rigidity is inversely proportional to the eye volume. On the other hand, ocular rigidity is directly proportional to intraocular pressure.[2-4] In a higher pressure state, it is more elevated than in a lower pressure state.The development of non-invasive technologies for estimating ocular stiffness (OR) will have far-reaching ramifications for ocular illness research. Importantly, glaucoma continues to be a leading cause of blindness due to tremendous hurdles in both diagnosis and treatment, and its origin is unknown.The most often used clinical treatment for slowing the development of open angle glaucoma(OAG) is to reduce intraocular pressure (IOP). However, the relationship between IOP and OAG development is not clear.[5,6] Recent data from experimental research in primates and mathematical modelling shows that ocular biomechanics may play a significant role in the aetiology of glaucoma.[7-10]
As per finite element modelling, IOP is a primary predictor of optic nerve head stress and strain that leads to glaucoma damage, but so does scleral elasticity and other biomechanical parameters.In fact, scleral flexibility is thought to be the most essential driver of optic nerve head stress and strain, even more important than IOP[11] implying that other variables, such as ocular biomechanics, must play a role.

MATERIALS AND METHODS

Patients visiting eye OPD diagnosed as POAG during study period time except falling under the exclusion criteria was taken as cases and controls

ISSN (O): 2687-5365; ISSN (P): 2753-6556

was taken on the basis of matching of age and gender with respect to cases.

Inclusion and Exclusion Criteria

The study subjects were chosen as per the inclusion and exclusion criteria:

Inclusion Criteria

Newly diagnosed cases of POAG and control subjects except under exclusion criteria.

Exclusion Criteria

Unclear ocular media,High myopia (> 4 Dioptre), High hyperopia (> 4 Dioptre), Patient having anterior segment pathology,Patient having any kind of posterior segment chorioretinal degeneration and chorioretinopathy, any active infection in eye, Corneal scar, Nystagmus, Recent trauma to eye, Uveitis, Uncooperative patients, Allergic to sulfa drugs, Age less than 15, Subjects with hepatic and renal disease.

Study Procedure

After obtaining the informed written consent, patients was taken for comprehensive ocular examination that include best corrected visual acuity, refraction, slit lamp biomicroscopy, IOP measurement, pachymetry, posterior segment evaluation and visual field examination. Patients are divided into two groups.

GLAUCOMATOUS SUBJECTS	CONTROLS
Selected on the basis of IOP> 21mmHg with glaucoma disc changes. Only one eye is considered having more glaucomatous damage on the basis of visual field defect and optic disc changes.	Normal subjects whose IOP <21, optic disc and visual fields are normal are considered as control. Patient presenting to eye OPD for complaints other than related to glaucoma except the subjects having the exclusion criteria. Only right eye is taken for the study.

Following the division of the participants into two groups, the basal axial length of the eyes of all of the subjects was measured using IOL master, and spectral domain OCT was done on both of the groups in order to observe changes in the posterior segments of both groups. Following the administration of 500 mg of acetazolamide orally to both groups, the axial length of the eye was measured using IOL Master and IOP measured using Goldmann Applanation Tonometry(GAT) after a period of two hours, and a correlation was established between ocular rigidity and changes in the posterior segment as revealed by OCT.

Statistical Analysis

After the data were input into the spreadsheet using Microsoft excel, the statistical analysis was performed utilising the statistical application SPSS version 21.0. The information pertaining to the quantitative variables (numerical variables) was presented in the form of the mean and standard deviation, whereas the information pertaining to the qualitative variables (categorical variables) was presented in the form of the frequency and percentage of each category.

The student t-test was utilised in the process of analysing the differences in mean values between the two groups, while the chi-square test was utilised in the process of analysing the frequency differences between the two groups. If the p-value was less than 0.05, then it was considered to be statistically significant. If the p-value was greater than 0.05, then it was not.

RESULTS

Table 3: Distribution of study population according to axial length

	Glaucomatous Case		Controls			
Axial length	Mean	Std. Deviation	Mean	Std. Deviation	t-test value	p-value
Pre-treatment	22.49	0.54	23.79	1.40	-6.154	0.001*
Post-treatment	22.46	0.53	23.70	1.41	-5.808	0.001*
Difference from Pre to post treatment	0.02	0.02	0.09	0.20	-2.330	0.022*

The mean pre-treatment, post-treatment and difference from pre to post treatment was compared between glaucomatous cases and controls using the unpaired t-test. The mean axial length pre-treatment, post-treatment and difference from pre to post treatment was significantly more among controls compared to glaucomatous cases.

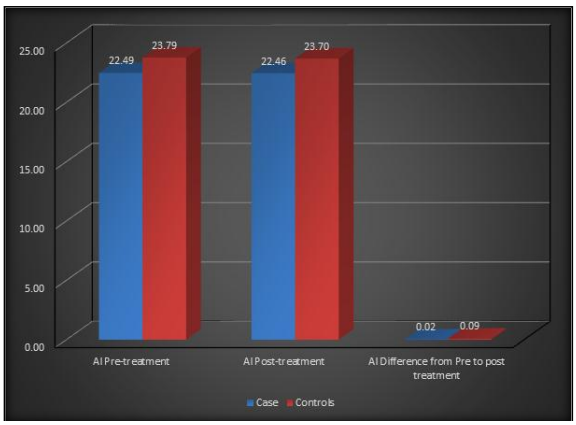
Table 6: Distribution of study population according to intra ocular pressure

	Glaucomatous Case		Controls			
Intra Ocular Pressure	Mean	Std. Deviation	Mean	Std. Deviation	t-test value	p-value
Pre-treatment	15.08	4.94	12.88	2.68	2.769	0.007*
Post-treatment	12.44	3.65	11.28	2.46	2.086	0.046*
Difference from Pre to post treatment	2.64	1.63	1.60	1.92	2.926	0.004*

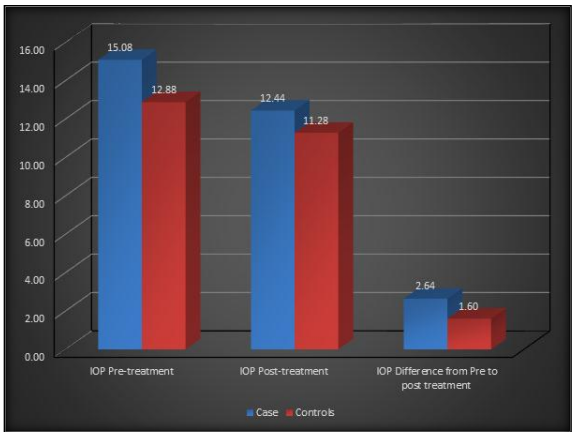
The mean intra ocular pressure pre-treatment, post-treatment and difference from pre to post treatment was compared between glaucomatous cases and controls using the unpaired t-test. The mean intra

ISSN (O): 2687-5365; ISSN (P): 2753-6556

ocular pressure pre-treatment, post-treatment and difference from pre to post treatment was significantly more among glaucomatous cases compared to controls.



Graph 6: Showing mean axial length pre-treatment post treatment and difference in mean axial length pre and post treatment in glaucomatous cases and controls



Graph 9: Showing mean intra ocular pressure pretreatment, post treatment and difference in mean intra ocular pressure pre and post treatment in glaucomatous cases and controls

DISCUSSION

Ocular rigidity is biomechanical parameter of the eye expresses the elasticity of the globe. It depends mainly on the properties of the cornea, sclera and other components of the outer shell of the eye. Ocular rigidity relates intraocular pressure changes to the corresponding volume changes and is a measure of the resistance that the eye exerts to distending forces. Accumulating clinical and scientific evidence has confirmed the critical roles of biomechanics in ocular health and disease, specifically in glaucoma.[12-15] Glaucoma is the second leading cause of blindness worldwide.[16] and represents a significant health and financial burden on the economy. Glaucomatous axonal damage initiates at the optic nerve head (ONH) where the retinal nerve fibers (axons of ganglion cell) exit the eye.[17-18] Mathematical modelling and animal studies have suggested that scleral stiffness is a major determinant of the ONH susceptibility to the damage.[19]

Axial Length

On pharmacologically inducing IOP change effect on axial length in glaucomatous eye and healthy eye we have found that lesser axial length change in glaucomatous group as compared to controls(healthy individuals) group suggestive of greater ocular rigidity in glaucomatous patients as compared to controls.

Ebneter et al demonstrated an identical drop in IOP was induced in both the glaucoma (mean±SEM: 2.90±0.44mmHg, n¼19) and the control group (mean±SEM: 3.17±0.32mmHg, n¼23).The change in axial eye length was significantly smaller (P¼0.026) in the glaucoma group (mean±SEM: _14.2±3.2 lm,n¼19) compared with the control group (mean±SEM: _23.0±2.98 lm, n¼23). Results strongly suggest that the ocular rigidity is increased in patients with established glaucoma in comparison to control subjects.[20] Ocular rigidity could play a role in the pathogenesis and pathophysiology of glaucoma. Determination of ocular rigidity could be helpful in detection of glaucoma.[20]

In our study we found that after inducing IOP fall pharmacologically the change in axial length was significantly smaller (p=0.022) in the glaucoma group (mean= 0.02) compared with the control (mean= 0.02). Our results were suggestive of increased ocular rigidity in the glaucomatous group compared to control group. The results of our study were similar to Ebneter study.

Intra Ocular Pressure

The mean IOP Pre-treatment, Post-treatment and difference from Pre to post treatment was significantly more among glaucomatous cases compared to controls. A significantly greater (p=0.004) drop in IOP was in glaucoma (mean=2.64) group compared with control (mean= 1.63) groups. Friedenwald JS et al. found that ocular rigidity describes the change in intraocular pressure (IOP) in response to a change in ocular volume. The ocular volume fluctuates due to the pulsatile vascular filling that occurs with each heartbeat, and for a given volume change, stiffer eyes will have a correspondingly larger increase in IOP, and vice versa for less stiff eyes.[21]Findings were similar to our study.

In Ebneter et al. on pharmacologically inducing IOP drop, an identical drop in IOP was induced in both the glaucoma (mean±SEM: 2.90±0.44mmHg, n¼19) and the control group (mean±SEM: 3.17±0.32mmHg, n¼23).Our findings were different as significantly greater (p=0.004) drop in IOP was in glaucoma (mean=2.64) group compared with control (mean= 1.63) groups in our study.

CONCLUSION

Our results strongly suggests that the ocular rigidity has increased in patient with glaucoma in

ISSN (O): 2687-5365; ISSN (P): 2753-6556

comparison to control subjects. Pliability of ocular tissue is increased to bear the raise pressure.If see it in reverse manner increased ocular rigidity could be one of the cause of glaucomatous damage .So, ocular rigidity could play a role in pathogenesis and pathophysiology of glaucoma, determination of ocular rigidity could helpful in detection of glaucoma.

REFERENCES

1. Collins R, van der Werff TJ. Mathematical Models of the Dynamics of the Human Eye. Vol. 34. Berlin: Springer, 1980;29–38.
2. Eisenlohr JE, Langham ME, Maumenee AE. Manometric studies of the pressure-volume relationship in living and enucleated eyes of individual human subjects. Br J Ophthalmol 1962; 46: 536–548.
3. Ytteborg J. Further investigations of factors influencing size of rigidity coefficient. Acta Ophthalmol (Copenhagen) 1960; 38:643–657.
4. Ytteborg J. The effect of intraocular pressure on rigidity coefficient in the human eye. Acta Ophthalmol (Copenhagen) 1960; 38: 548–561.
5. Lesk MR, Hafez AS, Descovich D. Relationship between central corneal thickness and changes of optic nerve head topography and blood flow after intraocular pressure reduction in open-angle glaucoma and ocular hypertension. Arch Ophthalmol. 2006 Nov;124(11):1568-72.
6. Morgan WH, Chauhan BC, Yu DY, Cringle SJ, Alder VA, House PH. Optic disc movement with variations in intraocular and cerebrospinal fluid pressure. Invest Ophthalmol Vis Sci. 2002 Oct;43(10):3236-42.
7. Bellezza AJ, Rintalan CJ, Thompson HW, Downs JC, Hart RT, Burgoyne CF. Deformation of the lamina cribrosa and anterior scleral canal wall in early experimental glaucoma. Invest Ophthalmol Vis Sci. 2003 Feb;44(2):623-37.
8. Levy NS, Crapps EE. Displacement of optic nerve head in response to short-term intraocular pressure elevation in human eyes. Arch Ophthalmol. 1984 May;102(5):782-6.
9. Sigal IA, Ethier CR. Biomechanics of the optic nerve head. Exp Eye Res. 2009 Apr;88(4):799-807.
10. Lesk MR, Spaeth GL, Azuara-Blanco A, Araujo SV, Katz LJ, Terebuh AK, Wilson RP, Moster MR, Schmidt CM. Reversal of optic disc cupping after glaucoma surgery analyzed with a scanning laser tomograph. Ophthalmology. 1999 May;106(5):1013-8.
11. Beaton L, Mazzaferri J, Lalonde F, Hidalgo-Aguirre M, Descovich D, Lesk MR, Costantino S. Non-invasive measurement of choroidal volume change and ocular rigidity through automated segmentation of high-speed OCT imaging. Biomed Opt Express. 2015;6(5):1694-706.
12. Burgoyne CF, Downs JC, Bellezza AJ, Suh J-KF, Hart RT. The optic nerve head as a biomechanical structure: a new paradigm for understanding the role of IOP-related stress and strain in the pathophysiology of glaucomatous optic nerve head damage. Prog Retin Eye Res. (2005) 24:39–73. 10.1016/j.preteyeres.2004.06.001 [PubMed] [CrossRef] [Google Scholar]
13. Downs JC, Roberts MD, Sigal IA. Glaucomatous cupping of the lamina cribrosa: a review of the evidence for active progressive remodeling as a mechanism. Exp Eye Res. (2011) 93:133–40. 10.1016/j.exer.2010.08.004 [PMC free article] [PubMed] [CrossRef] [Google Scholar]
14. Ma Y, Pavlatos E, Clayson K, Pan X, Kwok S, Sandwisch T, et al. Mechanical deformation of human optic nerve head and peripapillary tissue in response to acute IOP elevation. Invest Ophthalmol Vis Sci. (2019) 60:913–20. 10.1167/iovs.18-26071 [PMC free article] [PubMed] [CrossRef] [Google Scholar]
15. Boote C, Sigal IA, Grytz R, Hua Y, Nguyen TD, Girard MJA. Scleral structure and biomechanics. Prog Retin Eye Res. (2020) 74:100773. 10.1016/j.preteyeres.2019.100773 [PMC free article] [PubMed] [CrossRef] [Google Scholar]
16. Quigley HA, Broman AT. The number of people with glaucoma worldwide in 2010 and 2020. Br J Ophthalmol. (2006) 90:262–7. 10.1136/bjo.2005.081224 [PMC free article] [PubMed] [CrossRef] [Google Scholar]
17. Quigley HA, Addicks EM, Green WR, Maumenee AE. Optic nerve damage in human glaucoma: II. The site of injury and susceptibility to damage. Arch Ophthalmol. (1981) 99:635–49. 10.1001/archopht.1981.03930010635009 [PubMed] [CrossRef] [Google Scholar]
18. Nickells RW, Howell GR, Soto I, John SWM. Under pressure: cellular and molecular responses during glaucoma, a common neurodegeneration with axonopathy. Annu Rev Neurosci. (2012) 35:153–79. 10.1146/annurev.neuro.051508.135728 [PubMed] [CrossRef] [Google Scholar]
19. Sigal IA, Flanagan JG, Ethier CR. Factors influencing optic nerve head biomechanics. Invest Ophthalmol Vis Sci. (2005) 46:4189–99. 10.1167/iovs.05-0541 [PubMed] [CrossRef] [Google Scholar]
20. A Ebneter, B Wagels and MS Zinkernagel, Non-invasive biometric assessment of ocular rigidity in glaucoma patients and controls. Eye (Lond) 2009 Mar;23(3):606-11.
21. Friedenwald JS. Contribution to the theory and practice of tonometry. Am J Ophthalmol. (1937) 20:985–1024. 10.1016/S0002-9394(37)90425-2 [CrossRef] [Google Scholar].

ISSN (O): 2687-5365; ISSN (P): 2753-6556