

Correlation of Intraocular Pressure and Random Blood Glucose Level in Patients with Primary Open-Angle Glaucoma

Azuamah, Y.C.¹, Chioma, M.C.², Esenwah, E.C.³, Ikoro, N.C.⁴, Megwas, A.U.⁵

^{1,2,3,4,5}Department of Optometry, Federal University of Technology, Owerri, Nigeria

Corresponding Author: Azuamah, Y.C.

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ABSTRACT

Primary open angle glaucoma (POAG) is a multifactorial chronic optic neuropathy, characterized by progressive loss of retinal ganglion cells (RGC), leading to structural damage to the optic nerve head (ONH), retinal nerve fiber layer (RNFL), with visual field defects. This study was carried out at the Department of Optometry Teaching Clinic, Federal University of Technology, Owerri, Imo State, Nigeria to determine the correlation between intraocular pressure and blood glucose levels in patients with primary open-angle glaucoma. A total of 37 subjects, 14 males and 23 females were used for the study. The Accu Chek glucometer was used to measure the random blood glucose level and the intraocular pressure was measured using the Perkins applanation tonometer. Results showed that the mean intraocular pressure was 16.57 ± 3.97 while the mean random blood glucose level was 168.43 ± 108.48 . SPSS version 23 data analysis using the Pearson Product Moment Correlation Coefficient at 0.05 level of significance and 95% confidence interval revealed that there was no correlation between intraocular pressure level and random blood glucose level ($P > 0.05$). It was recommended that patients with primary open-angle glaucoma should frequently monitor their blood glucose levels.

Keywords: Intraocular Pressure, Random Blood Glucose, Primary Open Angle Glaucoma, Diabetes Mellitus

INTRODUCTION

Glaucoma is characterized by a progressive optic neuropathy, including chronic axonal damage and retinal ganglion cell (RGC) loss. [1] It affects the eye and is associated with an increased intraocular pressure (IOP), which also constitutes the only modifiable risk factor [2] in glaucoma management. When left untreated, patients may gradually experience a visual field loss, and even lose their sight completely. It is the number one cause of irreversible vision loss and the second leading cause of blindness around the globe. [3,4] Glaucoma involves a progressive loss of retinal ganglion cells (RGC) [5] and characteristic changes in neuroretinal rim tissue in the optic nerve head (ONH) which are accompanied by visual field (VF) constriction. [6] This eye disease, which is a leading cause of irreversible blindness worldwide, has generated a major public health problem. Intraocular pressure (IOP) is determined by the balance between aqueous humour production and outflow, and IOP homeostasis is primarily maintained by changes in aqueous humour outflow resistance. [7] Epidemiological studies [8] have suggested that IOP is affected by several factors, including non-modifiable risk factors, such as age, race, refraction, and central corneal thickness (CCT), and modifiable risk factors, such as blood pressure (BP), physical activity, and obesity. Elevated IOP may cause glaucomatous optic

nerve damage and subsequent visual field deficits.^[9] Evaluation of the optic disc is an indicator for progression of glaucoma disease.^[10] The optic disc is a small blind spot on the surface of the retina and is composed of RGC axons, which at the surface of the disc bend highly to exit the eye through the lamina cribrosa.^[11]

In Primary open-angle glaucoma, the trabecular meshwork undergoes morphological changes over time, resulting in progressive impairment of drainage although it remains anatomically open. Primary open angle glaucoma (POAG) is the most common type and accounts for 74% of all glaucoma cases.^[10] The consequent rise in IOP is transmitted to the optic disc and is eventually responsible for atrophy and nerve fibre damage.^[11] POAG is characterized by major risk factors of high IOP often greater than 21 mmHg, strong genetic tendency and age, mostly adults and older patients. It is chronic, generally bilateral, slowly progressive and insidious in onset.^[6] POAG is mostly asymmetrical, with difference in cupping of more than 0.2, more severe in one eye than the other and with absence of underlying cause.^[12] It is asymptomatic until visual impairment occurs. The irido-cornea (filtration) angle in POAG is usually open or wide, devoid of any narrowing tendency, with a normal anterior chamber depth. It is mainly characterized by glaucomatous optic neuropathy, with thinning of the temporal disc margin. Owing to the slow progression of the disease, POAG remains asymptomatic until central vision is affected. It does not present with headache, eye pain or loss of visual acuity. This increases the challenge to diagnose and treat open-angle glaucoma.^[13]

Diabetes mellitus is a chronic disease caused by the inability of the body to produce insulin or consume sugar.^[14] In diabetics, either the pancreas is not capable of producing sufficient insulin, or cells might not be able to appropriately respond to insulin. Hyperglycaemia, also called raised blood glucose or raised blood sugar,

is a common effect of uncontrolled diabetes and over time leads to serious damage to many of the body's systems, especially the nerves and blood vessels.^[15] Diabetes Mellitus (DM) is a major public health problem and the disease burden is increasing day by day. It has been estimated that there were 451 million people (Age 18-99 years) with diabetes in 2017 and this figure might increase to 693 million by 2045 globally.^[14] According to WHO^[16], the number of people with diabetes rose from 108 million in 1980 to 422 million in 2014. Prevalence has been rising more rapidly in low- and middle-income countries than in high-income countries.^[17] The global prevalence of diabetes was estimated to be 476 million people in 2017 and is expected to increase to 571 million people in 2025.^[18] The International Diabetes Federation states that the prevalence of DM will be 9.9% and the number of people with DM will rise to 552 million people by 2030.^[18] The objective of this study is to investigate the correlation of intraocular pressure and random blood glucose levels in patients with Primary Open-Angle Glaucoma.

MATERIALS AND METHODS

This study was a clinical study carried out at the Department of Optometry Teaching Clinic, Federal University of Technology, Owerri, Imo state, Nigeria. An informed consent was gotten from all the subjects who were part of the study. Ethical approval for the study was obtained from the Ethics Committee, School of Health Technology, Federal University of Technology Owerri, Imo State, Nigeria. Patients who were diagnosed with Primary Open Angle Glaucoma were selected for the study. Their Intraocular Pressure was measured using the Perkins applanation tonometer while their blood glucose level was measured using the Accu Chek glucometer.

Statistical Methods

The data obtained from the study was uploaded into the Statistical Package for

Social Sciences (SPSS) version 23 software. The Pearson Product Moment Correlation Coefficient was used to test the correlation of intraocular pressure and blood glucose levels at 0.05 level of significance and 95% confidence interval.

RESULTS

A total of 37 patients diagnosed with Primary Open Angle Glaucoma were used for this study. Table 1 showed the age and gender distribution of the subjects. There were 14 males and 23 females. Males aged between 39 and 58 (middle-aged adults) had a percentage frequency of 13.51%. Subjects within the age range of 79-98 had the lowest frequency of 2.7% for both male and females. Subjects in the age range of 19 - 38 (young adults) had a percentage frequency of 10.81% for both male and female subjects. Table 2 showed the frequency of intraocular pressure values and mean intraocular pressure values across various ranges. IOP was measured in 70 eyes out of the 37 patients. One (1) eye was in the range of 26 - 30mmHg; 3 eyes were within the range of 5 - 10mmHg with a mean of 9.67mmHg; 29 eyes were in the range of 16 - 20mmHg with a mean IOP of 17.76mmHg. Table 3 showed the frequency and mean Random Blood Glucose Levels of subjects. Out of the 21 subjects whose RBG was measured, 3 subjects were in the lowest range of 1 - 100 mg/dl with a mean random BGL of 74.33mg/dl; 2 subjects were within the range of 401 - 500mg/dl with mean random BGL of 431.50mg/dl; 14 subjects were in the range of 100 - 200mg/dl with a mean fasting BGL of 132.57mg/dl. Table 4 showed the descriptive statistics values for

intraocular pressure and IOP. The minimum IOP value was 9mmHg; the maximum value was 26mmHg giving a range of 17mmHg, the mean was 16.57mmHg and the standard deviation was 3.97mmHg. The Random Blood Glucose Levels were measured in 21 subjects. The minimum value was 42mg/dl, maximum was 458mg/dl, giving a range of 416mm/dl. The mean was 168.43mg/dl and standard deviation, 108.48mg/dl. Table 5 showed the SPSS data analysis using the Pearson Product Moment Correlation Coefficient at 0.05 level of significance and 95% confidence interval to test the correlation of RBG and IOP. It revealed a P value of 0.745 and a Pearson correlation of -0.076. There was no correlation ($P > 0.05$) between the IOP and the RBG levels among the subjects.

Table 1: Age and Gender Distribution of Subjects

Age (Years)	Male		Female		Total	
	n	%	n	%	n	%
19 – 38	4	10.81	4	10.81	8	21.62
39 – 58	5	13.51	12	32.43	17	45.95
59 – 78	4	10.81	6	16.22	10	27.03
79 – 98	1	2.70	1	2.70	2	5.41
TOTAL	14	37.84	23	62.16	37	100.00

Table 2: Intraocular Pressure Distribution of Subjects

IOP (mmHg)	n	Mean (mmHg)
5 – 10	3	9.67
11 – 15	26	13.19
16 – 20	29	17.76
21 – 25	11	22.45
26 – 30	1	26.00
Total	70	

Table 3: Distribution of Random Blood Glucose Level of Subjects

Random BGL (mg/dl)	n	Mean (mg/dl)
1 – 100	3	74.33
101 – 200	14	132.57
201 – 300	1	242.00
301 – 400	1	353.00
401 – 500	2	431.50
Total	21	

Table 4: Descriptive Statistics for Intraocular Pressure and Random Blood Glucose Levels

Variable	n	Range	Min	Max	Mean	S.D
IOP (mmHg)	70	17	9	26	16.57	3.97
RBG (mg/dl)	21	416	42	458	168.43	108.48

n = number; IOP = Intraocular Pressure, Min = Minimum, Max = Maximum, S.D. = Standard Deviation

Table 5: SPSS data analysis result showing P value for testing of correlation

Variables	Pearson Correlation	P-value
RBG - IOP	-0.076	0.745

DISCUSSION

The results of the study showed that the random blood glucose levels had no correlation with intraocular pressure ($P > 0.05$). The mean IOP for the various age

groups indicated a gradual increase of IOP with age. Increased IOP values are a major predictor of Primary Open-Angle Glaucoma. A study [20] on diabetes and intraocular pressure showed that with increased age, prevalence of glaucoma also increases. However, the same study also showed no correlation between age and IOP. The difference in IOP between male subjects and female subjects in our study revealed that female subjects with a mean IOP value of 17.51mmHg showed higher IOP values than male subjects with mean IOP value of 14.88mmHg. Although Khalaj, et al. [20] showed insignificant difference in mean IOP between genders both with diabetic and non-diabetic patients, Khachatryan, et al. [21] asserts that the male gender was significantly associated with risk of Primary Open-Angle Glaucoma among African-Americans 35 years and older. A study [22] on genetically predicted fasting blood glucose provides evidence for a causal role for genetically determined fasting blood glucose level in the development of increased IOP. IOP should be monitored in non-diabetic patients with increased fasting blood sugars. Increased IOP levels are a major predictor of Primary Open-Angle Glaucoma as well as the cause of ocular hypertension. Choi, et al. [23] in a study on fasting glucose level and the risk of Open-Angle Glaucoma, showed that subjects with the highest fasting blood glucose level ($\geq 160\text{mg/dL}$), as against the lowest ($< 80\text{mg/dL}$), showed a higher hazard ratio (HR 2.189) and a correlation ($P < 0.001$) for Open-Angle Glaucoma both for non-diabetic patients and patients with presence of type 2 diabetes. This is contrary to our study and might be because none of our subjects recorded such high blood glucose levels. We did not also consider other factors such as body mass index (BMI), presence of hypertension, smoking, drinking, and exercise habits, etc. From the statistical analysis, while testing for the first hypothesis, a negative Pearson correlation value (-0.076) showed an inverse relationship between intraocular pressure

and Blood Glucose Level and vice versa. The negative value however, was closer to zero and showed that the correlation was insignificant.

Elevated IOP has been validated as an independent risk factor for POAG, and it is currently the only treatable risk factor. There are many other risk factors that are involved in glaucoma pathogenesis, as IOP is not the only risk factor that can increase a patient's risk for POAG. Other factors that have been shown to be associated with POAG development include cup-to-disc ratio, visual field patterns, disc haemorrhages, myopia, central corneal thickness (CCT), ocular perfusion pressure, intracranial pressure (ICP), translamina cribrosa pressure gradient, diabetes, systemic blood pressure, ocular blood flow, vascular dysregulation, genetic factors and age. [24-27] Gender and ethnicity are known modifiers of the relative risk for POAG, although how they contribute to an individual's POAG risk is yet to be established. Several studies [23,26] have indicated that diabetic patients with glaucoma have a stronger vascular component to their glaucomatous progression than non-diabetic POAG patients. Specifically, diabetes is shown to affect structural and hemodynamics factors associated with ocular blood flow. In POAG patients with diabetes, changes in inferior retinal blood flow were significantly correlated with changes in the ONH (e.g., rim volume, cup to disc area ratio, rim area, cup area, cup shape, linear c/d ratio) after four years. [28] In addition, the changes in retinal blood flow in these patients were significantly correlated with changes in macular thickness after four years. This suggests that POAG patients with diabetes may have a stronger vascular contribution to their glaucomatous structural damage. It is also important to note that changes in vascular resistance were strongly correlated with deterioration of the RNFL and macula in POAG patients with DM. [29] From a study carried out by Amato, et al. [30], to clarify the effect of diabetes in the early

progression of glaucomatous RGC dysfunction preceding intraocular pressure (IOP) elevation, using the DBA/2J mouse (D2) model of glaucoma, intraocular pressure (IOP) was not influenced by time or diabetes. In contrast, RGC activity progressively decreased in the D2 group independently from intraocular pressure (IOP) elevation and outer retinal dysfunction.

CONCLUSION

In conclusion, this study showed that there was no significant correlation between intraocular pressure and random blood glucose levels in patients with Primary Open-Angle Glaucoma attending the Optometric Teaching Clinic, Federal University of Technology, Owerri, Imo State, Nigeria. However, measurement of blood glucose levels for patients with Primary Open Angle Glaucoma was recommended.

Declaration by Authors

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REFERENCES

1. Davis BM, Crawley L, Pahlitzsch M, Javaid F, Cordeiro MF. Glaucoma: the retina and beyond. *Acta Neuropathol.* 2016; 132(6): 807–826.
2. Vijaya L, Rashima A, Panday M, Choudhari NS, Ramesh SV, Lokapavani V, Boddupalli SD, Sunil GT, George R. Predictors for incidence of primary open-angle glaucoma in a south Indian population: The Chennai eye disease incidence study. *Ophthalmol.* 2014; 121(7): 1370–1376.
3. Arthur S, Cantor LB. Update on the role of alpha-agonists in glaucoma management. *Exp Eye Res.* 2011; 93(3): 271-283.
4. Conlon R, Saheb H, Ahmed IIK. Glaucoma treatment trends: A review. *Canadian Ophthalmological Society. Can J Ophthalmol.* 2017; 52(1): 114-124.
5. Akaiwa K, Namekata K, Azuchi Y, Guo X, Kimura A, Harada C, Mitamura Y, Harada T. Edaravone suppresses retinal ganglion cell death in a mouse model of normal tension glaucoma. *Cell Death Dis.* 2017; 8(7): 29-34.
6. Shon K, Wollstein G, Schuman JS, Sung KR. Prediction of glaucomatous field progression: pointwise analysis. *Curr Eye Res.* 2014; 39(7): 705–710.
7. Crawley L, Zamir SM, Cordeiro MF, Guo L. Clinical options for the reduction of elevated intraocular pressure. *Ophthalmol Eye Dis.* 2012; 4(4): 43–64.
8. Perez CI, Singh K, Lin S. Relationship of lifestyle, exercise, and nutrition with glaucoma. *Curr Opin Ophthalmol.* 2019; 30(2): 82–88.
9. Quigley HA. Glaucoma. *Lancet.* 2011; 377: 1367–1377.
10. Bajwa MN, Malik MI, Siddiqui SA, Dengel A, Shafait F, Neumeier W, Ahmed S. Two-stage framework for optic disc localization and glaucoma classification in retinal fundus images using deep learning. *BMC Med Inform Dec Mak.* 2019; 19(1): 136-142.
11. Salazar JJ, Ramírez AI, De Hoz R, Salobar-García E, Rojas P, Fernández-Albarral JA, López-Cuenca I, Rojas B, Triviño A, Ramírez JM. Anatomy of the Human Optic Nerve: Structure and Function. *Optic Nerve.* 2018; 203: 22–30.
12. Umezurike BC, Akhimien MO, Udeala O, Green UG, Agbo O, Ohaeri MO. Primary Open Angle Glaucoma: The Pathophysiology, Mechanisms, Future Diagnostic and Therapeutic Directions. *Ophthalmic Res.* 2019; 10(3): 1-17.
13. Saade CE, Lari HB, Berezina TL, Fechtner R, Khouri AS. Topical glaucoma therapy and ocular surface disease: a prospective, controlled cohort study. *Can J Ophthalmol.* 2015; 50(2): 132-6.
14. Cho NH, Shaw JE, Karuranga S, Huang Y, da Rocha Fernandes JD, Ohlrogge AW, Malanda B. IDF Diabetes Atlas: Global estimates of diabetes prevalence for 2017 and projections for 2045. *Diabetes Res Clin Pract.* 2018; 138: 271-281
15. World Health Organization. Global status report on non-communicable diseases. 2010. Available at: http://www.who.int/nmh/publications/ncd_report2010/en/. [Accessed November 12, 2022].

16. World Health Organization. Diabetes. 2022. Available at: <https://www.who.int/news-room/fact-sheets/detail/diabetes> [Retrieved November 3, 2022]
17. Lin X, Xu Y, Pan X, Xu J, Ding Y, Sun X, Song X, Ren Y, Shan P. Global, regional, and national burden and trend of diabetes in 195 countries and territories: an analysis from 1990 to 2025. *Sci Rep.* 2020; 10(1): 14-20.
18. Lin H, Stein JD, Nan B, Childers D, Newman-Casey PA, Thompson DA, Richards JE. Association of Geroprotective Effects of Metformin and Risk of Open-Angle Glaucoma in Persons with Diabetes Mellitus. *JAMA Ophthalmol.* 2015; 133(8): 915-923.
19. Whiting DR, Guariguata L, Weil C, Shaw J. IDF Diabetes Atlas: Global estimates of the prevalence of diabetes for 2011 and 2030. *Diabetes Res Clin Pract.* 2011; 94(3): 311-321.
20. Khalaj M, Fereydooni S, Barikani A. Relationship between Diabetes and Intraocular Pressure. *Acta medica Iranica.* 2015; 53(6): 363-368.
21. Khachatryan N, Pistilli M, Maguire MG, Salowe RJ, Fertig RM. Primary Open-Angle African American Glaucoma Genetics (POAAGG) Study: gender and risk of POAG in African Americans. *PloS one.* 2019; 14(8): 18-24.
22. Tan X, Zhong Z, Wang Q, Su G, Cao Q, Kijlstra A, Yang P. Genetically predicted fasting blood glucose level plays a causal role in intraocular pressure: A Mendelian randomisation study. *Clin Experiment Ophthalmol.* 2022; 50(5): 534-542.
23. Choi JA, Park YM, Han K, Lee J, Yun JS, Ko SH. Fasting plasma glucose level and the risk of open angle glaucoma: Nationwide population-based cohort study in Korea. *PLoS One.* 2020; 15(9): 23-29.
24. Varma R, Lee PP, Goldberg I, Kotak S. An assessment of the health and economic burdens of glaucoma. *Am J Ophthalmol.* 2011; 152(4): 515-522.
25. Cherecheanu AP, Garhofer G, Schmidl D, Werkmeister R, Schmetterer L. Ocular perfusion pressure and ocular blood flow in glaucoma. *Curr Opin Pharmacol.* 2013; 13(1): 36-42.
26. Memarzadeh F, Ying-Lai M, Chung J, Azen SP, Varma R. Blood pressure, perfusion pressure, and open-angle glaucoma: the Los Angeles Latino Eye Study. *Invest Ophthalmol Vis Sci.* 2010; 51(6): 2872-2877.
27. Jonas JB, Wang N. Association between arterial blood pressure, cerebrospinal fluid pressure and intraocular pressure in the pathophysiology of optic nerve head diseases. *Clin Experiment Ophthalmol.* 2012; 40(4): 233-234.
28. McIntyre N, Harris A, Amireskandari A, Eckert G, WuDunn D, Abrams J, Catoira-Boyle Y, Siesky BA. Changes in retinal capillary blood flow correlate with changes in macular thickness in glaucoma patients with diabetes. *Invest Ophthalmol Vis Sci.* 2014; 55(13): 29-29.
29. Schaab T, Harris A, Amireskandari A, Eckert G, Wirostko B, Ling J, Kanakamedala P, Siesky BA. Retrobulbar blood flow in glaucoma patients with and without diabetes. *Invest Ophthalmol Vis Sci.* 2014; 55(13):29-34.
30. Amato R, Lazzara F, Chou TH, Romano GL, Cammalleri M, Dal Monte M, Casini G, Porciatti V. Diabetes Exacerbates the Intraocular Pressure-Independent Retinal Ganglion Cells Degeneration in the DBA/2J Model of Glaucoma. *Invest Ophthalmol Vis Sci.* 2021; 62(9): 9-16.

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