European Journal of Scientific Research
ISSN 1450-216X Vol.49 No.2 (2011), pp.293-299

http://www.eurojournals.com/ejsr.htm

Biochemical Changes in Primary Open Angle Glaucoma Patients in a Nigerian Teaching Hospital

Adekunle B Okesina
Departments of Chemical Pathology and Immunology
University of Ilorin Teaching Hospital, Ilorin. Nigeria

Joshua FA Owoeye
Corresponding Author, Departments of Ophthalmology
University of Ilorin Teaching Hospital, Ilorin. Nigeria
E-mail: jfa_owoeye@yahoo.com or foluowoeye@gmail.com
Tel: +234 8033557763

Ahmed A Jimoh
Departments of Chemical Pathology and Immunology
University of Ilorin Teaching Hospital, Ilorin. Nigeria

Abdulkabir A Ayanniyi
Departments of Ophthalmology
University of Ilorin Teaching Hospital, Ilorin. Nigeria

Sikiru K Biliaminu
Departments of Chemical Pathology and Immunology
University of Ilorin Teaching Hospital, Ilorin. Nigeria

Abstract

Intraocular pressure is currently the main recognised modifiable risk factor in glaucoma - a leading cause of irreversible blindness worldwide. There is an established association between glaucoma, systemic hypertension and diabetes. There is the need to examine the relationship between glaucoma and other metabolic abnormalities. Different biochemical parameters in newly diagnosed primary open-angle glaucoma patients (POAG) were evaluated. Sixty nine POAG patients and 27 age and sex matched controls were included in this prospective, cross sectional hospital-based study. Ophthalmic examinations included visual field analysis, disc changes and Gonioscopy. Patients on anti-glaucoma medication and drugs that can affect serum metabolic parameters were excluded. Both the mean systolic and mean diastolic blood pressures were elevated in POAG patients compared with controls. Mean Fasting Blood Sugar (FBS) was also significantly higher in patients than controls. Mean creatinine level (94.56±35.8 vs 76.85±9.7mmol/l, p=0.013), serum total cholesterol (4.3±0.9mmol/l vs 3.7±1.3mmol/l, p=0.01) was significantly higher in the patients than controls. Both mean total protein and albumin levels were lower in patients compared with controls but only that of albumin was significant (Total protein 71.6±9.6mg/dl vs 72.0±6.8mg/dl; albumin 38.1±5.4 vs 42±5.8mg/dl p=0.003) when compared with the control subjects. The mean BMI was significanly higher among the control than the patients ((25.8±5.5 vs 23.5±4.5kg/m^2, p=0.04). In this study, POAG was

associated with significant changes in the FBS level, creatinine, total cholesterol and albumin. The observed increase in cholesterol - a risk factor of atherosclerosis - calls for further investigation, more so that homocysteine which is another risk factor was recently reported to be elevated in POAG patients.

Keywords: Metabolic Abnormalities, Diabetes, Glaucoma

Introduction

POAG is a spectrum of disorder typified by a characteristic optic neuropathy and visual field loss in eyes with open drainage angles. Of the glaucomas, POAG is the most prevalent in the African population [1] and in Nigeria [2, 3]. A genetic cause for about 5% of POAG has been identified in myocilin / TIGR (trabecular meshwork inducible glucocorticoid response) gene located at the GLCIA locus on chromosome 1q21 – q31 [4]. An elevated IOP is an important modifiable risk factor for the development of glaucoma and by far the most common risk factor for vision loss in glaucoma. However, it is not the only factor involved, because people with normal IOP have been shown to experience vision loss from glaucoma. On the other hand, some people with high IOP never develop the optic nerve head damage of glaucoma.

There is an association between systemic hypertension and glaucoma, with the common mechanism related to sodium handling being responsible [5]. It has been suggested, although disputed, that diabetes mellitus and blood pressure changes may play a role in POAG pathogenesis [6, 7, 8, 9]. This finding was complemented by Elisaf et al [10] who observed that the disturbance of carbohydrate and uric acid metabolism could play a role in glaucoma damage and pathogenesis.

This study was intended to determine the biochemical parameters in recently diagnosed glaucoma patients, which might be a factor in glaucoma damage.

Materials and Method

All newly diagnosed glaucoma patients attending the eye clinic of our Teaching Hospital, between June and December 2007, were screened for this prospective, cross sectional study. Informed consent, in accordance to the tenets of the Declaration of Helsinki, was obtained from all subjects. Aproval to carry out the study was also obtained from the Institutional Review Board. The purpose of the research was made known to the patients and responses to an interview were recorded for all the patients. The body weight and height were measured to determine the body mass index (BMI) and the ophthalmic examination findings were recorded for each of them. The criteria for diagnosis of POAG were based on the presence of visual field defects, optic disc damage and an open-angle of the anterior chamber, without the features characteristic of congenital, secondary, rubeotic or angle-closure glaucoma [8, 11, 12, 13]. None of the patients was on any anti-glaucoma drug or has had an eye surgery or ocular trauma in the past.

Blood samples for biochemical analysis such as: fasting blood sugar (FBS), total protein, albumin, urea, creatinine, uric acid, total cholesterol and triglyceride, were taken after an overnight fast of at least 12 hours. Patients with dyslipidemia necessitating the use of hypolipidemic drugs were excluded. Additionally, subjects on drugs that potentially affect serum metabolic parameters (allopurinol, uricosuric drugs, corticosteroids, thiazides, β-blockers, losartan, etc) were also excluded from the study as well as known diabetic patients, who were on treatment. Blood pressure for each subject was determined after 10 minutes of rest on arrival in the hospital, with Accouson sphygmomanometer. Subjects were classified as hypertensive based on the Joint National Committee on Prevention, Diagnosis and Treatment of Hypertension (JNC) – 7 classification [20]. Hypercholesterolemia was defined as a total cholesterol level of >5.2mmol/l, hypertriglyceridemia as a

serum triglyceride level of >2.24mmol/l. Hyperuricemia was defined as serum uric acid levels 360µmol/l for women and 420µmol/l for men. The diagnosis of diabetes mellitus relied on a fasting serum glucose level of 7.0 mmol/l, Impaired fasting glucose was defined as a fasting serum glucose level greater than 6.1mmol/l but less than 7.0mmol/l.

Laboratory Analysis

The serum total cholesterol and triglyceride were determined by enzymatic method using Randox kit. Serum uric acid was determined by the uricase / PAP method, while glucose was determined using the glucose oxidase method. Total protein was determined using the Biuret method, while albumin was determined using the BCG method. Urea was determined using the urease enzymatic method, while creatinine was determined using the Jaffe (Picric acid) method. All the kits for the analysis were purchased from Randox Laboratory UK. The Spectronic 20 spectrophotometer was used for reading the absorbance of the color developed.

Results

A total of 69 POAG patients (46 males and 25 females) and 27 controls (11 males and 16 females) were included in the study. The demographic and clinical parameters of the studied population are as shown on Table 1.

Table 1: Clinical Parameters of the Study Population

Parameters	POAG, n=69	Controls, n=27	p value
Age (yrs)	56.9±13.2	54.7±13.6	0.016
Sex M/F	46/23	11/16	-
Systolic Blood Pressure (mmHg)	136±24	124.7±12.4	0.017
Diastolic Blood Pressure (mmHg)	85.8±11.5	81.1±9.7	0.056
Body weight (kg)	62.9±12	65.14±8.4	0.021
Body mass index (kg/m2)	23.5±4.5	25.8±5.5	0.040

The mean age of the patients with POAG was comparable with that of contols (56.9 ± 13.2 vs 54.7 ± 13.6 years).

The mean systolic blood pressure of the patients was significantly higher than that of control (136 ± 24mmHg vs 124.7 ± 12.4mmHg, p=0.017). Also the mean diastolic blood pressure of patients was significantly higher than that of controls (85.8 ± 11.5mmHg vs 81.1±9.7mmHg). The controls had a significantly higher mean BMI than the subjects.

As shown in Table 2; POAG patients had a significantly higher concentration of the FBS (5.58 ± 2.5mmol/l vs 3.9±0.8mmol/l, p=0.001). Also 13 (19%) of the patients were diabetic and there was non among the controls. So also 4.3% of the patients had impaired glucose tolerance while only 3.7% among the controls.

Table 2: Biochemical Parameters of the Study Population

Parameters	Patients with POAG	Controls	p value
Fasting plasma glucose (mmol/l)	5.58±2.5	3.9±0.8	0.001
Uric acid (µmol/l)	340±130	340±100	0.322
Creatinine (µmol/l)	94.56±35.8	76.85±9.7	0.013
Urea (mmol/l)	5.1±1.4	5.3±1.2	0.249
Total protein (mg/dl)	71.6±9.6	72±6.8	0.160
Albumin (mg/dl)	38.1±5.4	42.0±5.8	0.003
Triglyceride (mmol/l)	1.0±0.58	1.3±1.5	0.717
Total Cholesterol (mmol/l)	4.3±0.9	3.7±1.3	0.010

Mean serum urea and uric acid levels were similar in the patients and the controls (urea 5.1±1.4mmol/l vs 5.3 ± 1.2mmol/l; Uric acid 340 ± 130μmol/l vs 340 ± 100μmol/l respectively). Mean creatinine level was significantly higher in the patients than controls (94.56±35.8 vs 76.85±9.7mmol/l, p=0.013). Similarly mean Total cholesterol was significantly higher among patients than the controls (4.3 ± 0.9mmol/l vs 3.7±1.3mmol/l, p=0.01). Both mean total protein and albumin levels were lower in patients compared with controls but only that of albumin was significant (Total protein 71.6 ± 9.6mg/dl vs 72.0 ± 6.8mg/dl; albumin 38.1±5.4 vs 4.2±5.8mg/dl p=0.003) when compared with the control subjects.

Table 3 displayed the pattern of biochemical abnormalities in patients and controls. Prevalence of both diabetes and impaired glucose tolerance were higher among patients compared with controls.

Table 3: Incidence of Metabolic Abnormalities of the Study Population

Disturbance	Patients with POAG, n=69	Controls, n=27	LS
	No (%)	No (%)	
Diabetes mellitus	13 (19)	0 (0)	0.005
Impaired fasting glucose	3 (4.3)	1 (3.7)	NS
Hyperuricemia	20 (30)	10 (37)	NS
Hypercholesterolemia	9 (13)	3 (11)	NS
Hypertriglyceridemia	2 (2.9)	1 (3.7)	NS

LS=level of significance, NS=not significant

In this study, POAG was associated with significant changes in the FBS level, creatinine, total cholesterol and albumin. The observed increase in cholesterol; a risk factor of atherosclerosis calls for further investigation, more so that homocysteine which is another risk factor was recently reported to be elevated in POAG patients.

There was no difference in the serum mean value of triglycerides when the patients were compared with the controls (1.0± 5.58mmol/l vs 1.3± 1.5mmol/l).

Discussion

The need to study the effect of biochemical changes in glaucoma patients cannot be over emphasized, more so that there had been problem of determining the appropriate treatment for the disease. Researchers in US, working with rats with elevated eye pressure were able to prevent loss of retinal ganglion cells by inhibiting enzyme that synthesizes nitric oxide [21]. They suggested that inhibition of nitric oxide might preserve vision in those who do not respond to current therapies and also could be used with drugs that lower intra ocular pressure in a sub-set of patients who have normal-tention glaucoma.

The present study has to a large extent emphasized the presence of disturbance of carbohydrate metabolism in POAG patients. This tally with previous suggestion of a relationship between diabetes and POAG [10]; though this association is said to be contentious [7, 8]. We observed a significantly higher level of mean plasma glucose in our POAG patients when compared with controls. These results point out the potential significance of carbohydrate intolerance in the pathogenesis of POAG. A number of explanations have been proposed for the association between disturbances of the carbohydrate metabolism and glaucoma, including optic nerve damage as a result of the vascular or other effects of diabetes [23], autonomic dysfunction leading to increased IOP [18, 19] and genetic factors [19].

Our result disagrees with the finding of Elisaf et al [10] who reported that abnormalities of urate metabolism were more common among glaucoma patients compared with the normal population (with values of 6.2+1.9mmol/l vs 5.0+1.2mmol/l). Also, their result showed that 37% of their patients

developing country. Br J Ophthalmol 1976; 60: 665 668.
13. Morgan R. E. Pattern of eye diseases in children seen at Jos University Teaching Hospital. Dissertation for the award of Fellowship Diploma of the National Postgraduate Medical College of Nigeria in Ophthalmology, 1995.
14. Yorston D. Measles and childhood blindness. Community Eye Health Journal 1991; 8: 2 4.
15. Osahon A. I. Consequencies of traditional eye medication in UBTH. Benin City Nigerian Journal of Ophthalmology 1995; 3: 39 41.
16. Ayanniyi A. A., Olatunji F. O., Mahmoud A. O., Ayanniyi R. O. Clinical findings among Nigerian paediatric glaucoma suspects during a school eye health survey. The Open Ophthalmology Journal 2008; 2: 137 140.
17. Araoye M. O. Research methodology with statistics for health and social sciences. Ilorin. Nathadex Publishers, 2003; 117 118.
18. Isawumi M.A. Ocular disorders among school children in Osun State, Nigeria. Dissertation for the award of Fellowship Diploma of the National Postgraduate Medical College of Nigeria in Ophthalmology, 2003.
19. Balogun B. G. Vision Screening among primary school children in the Mainland Local Government Area of Lagos State. Dissertation for the award of Fellowship Diploma of the National Postgraduate Medical College of Nigeria in Ophthalmology, 1999.
20. Onyekwe L. O. Visual impairment among school children in Jos, Plateau State, Nigeria. Dissertation for the award of Fellowship Diploma of the National Postgraduate Medical College of Nigeria in Ophthalmology, 1995.
21. Yoloye M. O. Pattern of visual defects and eye disease among primary school children in Ibadan. Dissertation for the award of a Fellowship Diploma of the National Postgraduate Medical College in Ophthalmology, 1993; 70 91.
22. Courtright P., Chirambo M., Lewallen S., Harjinder C., Kanjaloti S. Collaboration with African traditional healers for the prevention of blindness. Singapore. World Scientific Publication Co. Pte. Ltd.; 2000.
23. Omolase C. O., Fadamiro C. O., Omolase B. O., Ajibido J., Adepoju F. G. Barriers to uptake of orthodox eye care in a rural Nigerian community. Tropical Journal of Health Sciences 2007; 14: 23 25.
24. Ayanniyi A. A. A 39-year-old man with blindness following the application of raw cassava extract to the eye. Digital Journal of Ophthalmology 2009; 15(2): 1 4.
25. Paczka J. A., Ochoa-Tabares J. C., Giono-Sandoval L. A., Topete-Jiménez J., Sánchez-Castellanos E., Villaseñor-García D. G., Vázquez-Martínez L. Knowledge and perceptions of glaucoma among adults living in an urban area. Gac Med Mex 2006; 142: 303 8.
26. Nirmalan P. K., Sheeladevi S., Tamilselvi V., Victor A. C., Vijayalakshmi P., Rahmathullah L. Perceptions of eye diseases and eye care needs of children among parents in rural south India: the Kariapatti pediatric Eye Evaluation Project (KEEP). Indian J Ophthalmol 2004; 52: 163 7.
27. Dandona R., Dandona L., John R. K., McCarty A. C., Rao G. N. Awareness of eye diseases in an urban population in southern India. Bull World Health Organ. 2001; 79: 96 102.
28. Sannapaneni K., Vilas K., Marmamula S., Bindiganavale S. R., Gullapalli R. N., Ravi T. Awareness of glaucoma in the rural population of Southern India. Community Ophthalmology 2005; 33: 205 208.
29. Yun L. S., Hassan Y., Aziz N. A., Awaisu A., Ghazali R. A comparison of knowledge of diabetes mellitus between patients with diabetes and healthy adults: a survey from north Malaysia. Patient Educ Couns 2007; 69: 47 54.
30. Al-Shafaee M A., Al-Shukaili S., Rizvi S. G., Al-Farsi Y., Khan M. A., Ganguly S. S., Afifi M., Al-Adawi S. Knowledge and perceptions of diabetes in a semi-urban Omani population. BMC Public Health 2008; 8: 249.
31. Olurin O. National Postgraduate Medical College of Nigeria. Third Faculty of Ophthalmology lecture. Glaucoma Revisited: 18 August, 2000; 3 8.