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Comparative evaluation of intraocular pressure in diabetic and non-diabetic patients admitted in Patna medical college & hospital, Patna, Bihar

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Abstract
Diabetics. Diabetes mellitus is an important ocular risk factor. Diabetes has been found to be associated with elevated intraocular pressure (IOP), particularly primary open angle glaucoma (POAG). IOP has been associated with different systemic, familial, anthropometric and demographic factors by several studies Several studies have reported factors including age, sex, African ancestry, blood pressure, BMI, alcohol, smoking, myopia and family history of glaucoma to be positively associated with elevated IOP in general. Diabetes mellitus is an important risk factor associated with the occurrence of retinopathy, certain types of cataract, and glaucoma during its course. Hence based on above findings the present study was planned for Comparative Evaluation of Intraocular Pressure in Diabetic and Non-Diabetic Patients Admitted in Patna Medical College & Hospital, Patna, Bihar.
The present study was planned in Department of Ophthalmology Patna Medical College& Hospital, Patna, Bihar, India. The study was conducted from July 2018 to Oct 2018. Total 40 cases of the diabetic and non-diabetic patients were enrolled in the present study. The 20 cases of the diabetes were enrolled in Group A and the remaining 20 cases of the normal, non-diabetic patients were enrolled in the Group B. IOP was measured in both eyes using Schiotz tonometer and recorded after putting lignocaine drops in both eyes and recorded after 5 minutes. IOP measurements were always performed between 9: 00 am-12: 00 pm by Schiotz tonometry to minimize the diurnal variation. Blood glucose was estimated using Glucometer.
The data generated from the present study concludes that mean intra ocular pressure was higher in diabetic group as compared to non-diabetic group. Mean intra ocular pressure was higher in uncontrolled diabetic patients (HbA1c >6.5) when compared with controlled diabetic patients (HbA1c < 6.5). In diabetics IOP should be monitored regularly so as to prevent complications.

Keywords: intraocular pressure, diabetic and non-diabetic, IOP, etc

Introduction

Intraocular pressure (IOP) is the fluid pressure inside the eye. Tonometry is the method eye care professionals use to determine this. IOP is an important aspect in the evaluation of patients at risk of glaucoma. Most tonometers are calibrated to measure pressure in millimeters of mercury (mmHg).

Intraocular pressure is determined by the production and drainage of aqueous humour by the ciliary body and its drainage via the trabecular meshwork and uveoscleral outflow. The reason for this is because the vitreous humour in the posterior segment has a relatively fixed volume and thus does not affect intraocular pressure regulation.

Intraocular pressure is measured with a tonometer as part of a comprehensive eye examination. Measured values of intraocular pressure are influenced by corneal thickness and rigidity. As a result, some forms of refractive surgery (such as photorefractive keratectomy) can cause traditional intraocular pressure measurements to appear normal when in fact the pressure may be abnormally high. A newer transpalpebral and transscleral tonometry method is not influenced by corneal biomechanics and does not need to be adjusted for corneal irregularities as measurement is done over upper eyelid and sclera [1].

Current consensus among ophthalmologists and optometrists define normal intraocular pressure as that between 10 mmHg and 20 mmHg. The average value of intraocular pressure is 15.5 mmHg with fluctuations of about 2.75 mmHg. Ocular hypertension (OHT) is defined by intraocular pressure being higher than normal, in the absence of optic nerve damage or visual field loss. Ocular hypotension, hypotony, or ocular hypotony, is typically defined as intraocular pressure equal to or less than 5 mmHg. Such low intraocular pressure could indicate fluid leakage and deflation of the eyeball.

Intraocular pressure varies throughout the night and day. The diurnal variation for normal eyes is between 3 and 6 mmHg and the variation may increase in glaucomatous eyes. During the night, intraocular pressure may not decreasedespite the slower production of aqueous humour. In the general population, IOP ranges between 10- and 21-mm Hg with a mean of about 15- or 16-mm Hg (plus or minus 3.5 mm Hg during a 24-hour cycle). Glaucoma patients' 24-hour IOP profiles may differ from those of healthy individuals [2].

There is some inconclusive research that indicates that exercise could possibly affect IOP (some positively and some negatively). Playing some musical wind instruments has been linked to increases in intraocular pressure. A 2011 study focused on brass and woodwind instruments observed "temporary and sometimes dramatic elevations and fluctuations in IOP". Another study found that the magnitude of increase in intraocular pressure correlates with the intraoral resistance associated with the instrument and linked intermittent elevation of intraocular pressure from playing high-resistance wind instruments to incidence of visual field loss. The range of intraoral pressure involved in various classes of ethnic wind instruments, such as Native American flutes, has been shown to be generally lower than Western classical wind instruments [3].

Intraocular pressure also varies with a number of other factors such as heart rate, respiration, fluid intake, systemic medication and topical drugs. Alcohol and marijuana consumption leads to a transient decrease in intraocular pressure and caffeine may increase intraocular pressure. Taken orally, glycerol (often mixed with fruit juice to reduce its sweet taste) can cause a rapid, temporary decrease in intraocular pressure. This can be a useful initial emergency treatment of severely elevated pressure.

The depolarising muscle relaxant succinylcholine, which is used in anaesthesia, transiently increases IOP by around 10 mmHg for a few minutes. This is significant for example if the patient requires anaesthesia for a trauma and has sustained an eye (globe) perforation. The mechanism is not clear but it is thought to involve contraction of tonic myofibrils and transient dilation of choroidal blood vessels. Ketamine also increases IOP.

Ocular hypertension is the most important risk factor for glaucoma. Intraocular pressure has been measured as a secondary outcome in a systematic review comparing the effect of neuroprotective agents in slowing the progression of open angle glaucoma. Differences in pressure between the two eyes are often clinically significant, and potentially associated with certain types of glaucoma, as well as iritis or retinal detachment.

Intraocular pressure may become elevated due to anatomical problems, inflammation of the eye, genetic factors, or as a side-effect from medication. Intraocular pressure laws follow fundamentally from physics. Any kinds of intraocular surgery should be done by considering the intraocular pressure fluctuation. Sudden increase of intraocular pressure can lead to intraocular micro barotrauma and cause ischemic effects and mechanical stress to retinal nerve fiber layer. Sudden intraocular pressure drop can lead to intraocular decompression that generates micro bubbles that potentially cause multiple micro emboli and leading to hypoxia, ischemia and retinal micro structure damage [4].

Ocular hypertension (OHT) can be used as a generic term referring to any situation in which intraocular pressure (IOP) is greater than 21 mm Hg, the widely accepted upper limit of normal intraocular pressure in the general population. The term makes no mention of whether or not glaucomatous nerve damage is present. It also depicts no particular time frame during which the elevated pressure has been measured.

The formal definition of ocular hypertension evolved in the latter part of the 20th century [1]. It was used as early as 1962 by Drance, but was not defined in English language publications until 1966 by Perkins and others.

Ocular hypertension is a condition in which the following criteria are met: An intraocular pressure greater than 21 mm Hg in one or both eyes, as measured by applanation tonometry on 2 or more occasions, Absence of glaucomatous defects on visual-field testing, Normal appearance of the optic disc and nerve fiber layer, Anatomically normal, open angles on gonioscopy, Absence of ocular conditions contributing to the elevation of pressure, such as narrow angles, neovascular conditions, and uveitis.

Despite early definitions, ocular hypertension has historically meant different things to different ophthalmologists. Some glaucoma experts such as Hitchings stressed the point of not reading too much into the term. Others, including Spaeth, advocated total disuse of the term secondary to its inherent ambiguity, preferring the term glaucoma suspect to more adequately convey uncertainty regarding the diagnosis and prognosis [5].

The Ocular Hypertension Treatment Study (OHTS) is a multicenter, prospective, randomized, controlled clinical trial studying more than 1800 research subjects, evaluating the safety and efficacy of medical treatment in preventing or delaying the onset of visual-field loss and/or optic nerve damage in patients with ocular hypertension who are at moderate risk for developing primary open-angle glaucoma (POAG) [6].

In this article, ocular hypertension refers to a state in which the eye(s) meet the above 5 criteria, in the absence of identifiable causes or cardinal signs of POAG. Ocular hypertension is a condition requiring closer observation for the potential development of glaucomatous damage.

The exact pathophysiology of elevated intraocular pressure (IOP) in ocular hypertension is not known. In primary open-angle glaucoma, myocilin (MYOC) gene mutations have been found and determined to cause protein misfolding, making trabecular meshwork cells dysfunctional, with subsequent decrease in outflow facility and marked elevation of IOP [7].

Elevated IOP is of great concern because it is the most established risk factor for the development of glaucoma [9]. Two theories of how IOP initiates glaucomatous damage include (1) onset of vascular dysfunction causing ischemia to the optic nerve and (2) mechanical dysfunction via cribriform plate compression of the neuronal axons.

In addition to vascular compromise and mechanically impaired axoplasmic flow, contemporary hypotheses of possible pathogenic mechanisms that underlie glaucomatous optic neuropathy include excitotoxic damage from excessive retinal glutamate, deprivation of neuronal growth factors, peroxynitrite toxicity from increased nitric oxide synthase activity, immune-mediated nerve damage, and oxidative stress.

The exact role that IOP plays in combination with these other factors and its significance in the initiation and progression of subsequent glaucomatous neuronal damage and cell death over time is still under debate [8].

Population studies such as the Framingham, Beaver Dam, Baltimore, Rotterdam, Barbados, and Egna-Neumarkt studies have estimated that 4-10% of the population older than 40 years will have IOPs of 21 mm Hg or higher without detectable signs of glaucomatous damage. Ocular hypertension has a 10-15 times greater prevalence than POAG.

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Although black individuals are considered to have a 3-4 times higher prevalence of POAG and larger cup-to-disc ratios compared with white individuals, the data are less clear concerning ocular hypertension. The Barbados Eye Study found the incidence of IOPs greater than 22 mm Hg to be 5 times higher in blacks than in whites [10]. The Baltimore Eye Survey found no difference in mean IOP between blacks and whites [11]. The Los Angeles Latino Eye Study found Latinos to be at higher risk of ocular hypertension than non-Latino whites but lower than blacks [12].

The Barbados Eye Study found ocular hypertension present more frequently in women. Mean IOP slowly rises with increasing age. Age older than 40 years is considered a risk factor for the development of ocular hypertension and POAG.

Prospective studies in the 1980s showed that among patients with elevated IOP, roughly 0.5-1% per year developed glaucoma over a period of 5-10 years. The OHTS suggests that progression to glaucoma increases with higher IOPs and lower central corneal thickness (CCT) and that certain patient characteristics are associated with a greater than 2% annual risk of developing glaucoma [13].

The Gutenberg Health Study found positive associations with higher IOP in an adult European cohort for systemic cardiovascular risk factors such as hypertension, diabetes, smoking, and obesity. Systemic morbidity and mortality can also result from the possible cardiopulmonary adverse effects of IOP-lowering medications. With regard to ocular morbidity and mortality, retinal vascular occlusion may occur in approximately 3% of ocular hypertensive patients [14].

Progression to glaucoma is the main source of ocular morbidity and mortality. Studies have shown that over a 5-year-period, the incidence of glaucomatous damage in ocular hypertensive patients increases with increasing IOP levels:

- IOP of 21-25 mm Hg - Approximately 2.6-3%
- IOP of 26-30 mm Hg - Range from 12-26%
- IOP higher than 30 mm Hg - Approximately 42%

The Ocular Hypertension Treatment Study (OHTS) states that over a 5-year-period, patients with ocular hypertension and IOP levels of 24 mm Hg or more have a 10% overall risk of developing glaucoma. This risk can be cut in half by medical treatment. In 2004, more than 2 million individuals in the United States were diagnosed as having open-angle glaucoma. This number is projected to increase to more than 3 million by 2020 [15].

Elevated intraocular pressure, which is a main risk factor for glaucoma, is a major concern among diabetics. Diabetes mellitus is an important ocular risk factor [16]. Diabetes has been found to be associated with elevated intraocular pressure (IOP), particularly primary open angle glaucoma (POAG). IOP has been associated with different systemic, familial, anthropometric and demographic factors by several studies [17] Several studies have reported factors including age, sex, African ancestry, blood pressure, BMI, alcohol, smoking, myopia and family history of glaucoma to be positively associated with elevated IOP in general [18]. Diabetes mellitus is an important risk factor associated with the occurrence of retinopathy, certain types of cataract, and glaucoma during its course. Hence based on above findings The present study was planned for Comparative Evaluation of Intraocular Pressure in Diabetic and Non-Diabetic Patients Admitted in Patna Medical College & Hospital, Patna, Bihar.

Methodology

The present study was planned in Department of Ophthalmology Patna Medical College& Hospital, Patna, Bihar, India. The study was conducted from July 2018 to Oct 2018. Total 40 cases of the diabetic and non-diabetic patients were enrolled in the present study. The 20 cases of the diabetes were enrolled in Group A and the remaining 20 cases of the normal, non-diabetic patients were enrolled in the Group B. IOP was measured in both eyes using Schiotz tonometer and recorded after putting lignocaine drops in both eyes and recorded after 5 minutes. IOP measurements were always performed between 9: 00 am-12: 00 pm by Schiotz tonometry to minimize the diurnal variation. Blood glucose was estimated using Glucometer.

All the patients were informed consents. The aim and the objective of the present study were conveyed to them. Approval of the institutional ethical committee was taken prior to conduct of this study.

Following was the inclusion and exclusion criteria for the present study.

Inclusion Criteria: Patients with diabetes mellitus, Age group20-70 years, Non-diabetic individuals.

Exclusion Criteria: Diabetes Mellitus with hypertension, thyroid disorder and other systemic diseases, Patients having glaucoma, Patients having corneal pathology and any other ocular abnormalities like pterygium, entropionj, trichiasis.

Results & Discussion

Glaucoma is the world's leading cause of acquired blindness [19]. Glaucoma is an optic neuropathy characterized by progressive degeneration of retinal ganglion cells and their axons, manifested by increasing optic disc cupping and deterioration of visual function [20]. The round firm shape to the eyeball is caused by the intra ocular pressure (IOP) within the eyeball which is caused by the aqueous humour and vitreous body. Importance of IOP is in maintaining the structural and functional integrity of the eye. High intraocular pressure is more often associated with glaucomatous optic nerve damage. IOP is not the only risk factor for optic nerve damage but is one of the modifiable risk factor for emergence of glaucoma and is the only amendable risk factor that can be treated [21].

An abnormally high IOP reading indicates that either the eye is producing too much fluid, or that it is not draining properly [22]. The IOP can be influenced by different systemic factors such as hypertension atherosclerotic diseases, body mass index, and diabetes can be explained by the numerous factors affecting IOP. Previous studies have shown that the factors associated with elevated IOP include smoking blood pressure, family history of glaucoma diabetes (elevated glycosylated alcohol usage, race (African), nuclear sclerosis, body mass index (BMI) and iris colour associated with higher IOP values in most population studies, the underlying mechanisms are still unclear Recent studies have suggested that changes in corneal biomechanics (increased corneal hysteresis) in diabetic eyes would lead to overestimated IOP measurements [23].

Table 1: Age & Sex

Groups	Group A	Group B
Type of Cases	Diabetic	Non-Diabetic
Parameters		
Sex		
Males	11	15
Females	9	5
Age		
20 – 30 years	0	1
31 – 40 years	2	2
41 – 50 years	5	7
51 – 60 years	8	5
61 & above years	5	5

Table 2: Mean IOP

Groups	Group A	Group B
Type of Cases	Diabetic	Non-Diabetic
Parameters		
Mean IOP (mm Hg)	17.1 ± 0.13	12.7 ± 0.21

Table 3: HbA1c & IOP

HbA1c:	No. of Cases
Non-Diabetic: Less than 6.5	15.2 ± 1.2
Diabetic: More than 6.5	17.1 ± 1.3

Shital A. Patel *et al.* [24] in his study of profile of primary open angle glaucoma patients reported that there was no significant gender difference in prevalence of POAG. Anhchuong Le *et al.* [25] in his study of risk factors associated with the incidence of open-angle glaucoma showed that development of OAG was not gender related. The Barbados Incidence Eye Study [26] had shown a higher incidence in men, whereas the Dalby Sweden Study [27] showed a higher incidence in women. Naila Ali *et al.* (2007) had reported that males are more prone to glaucomatous optic neuropathy, whether a gender difference exists in the prevalence of POAG has been controversial. Overall proportion of POAG cases observed was 4.0% (4 out 100).

High glucose levels can induce excess extracellular matrix (ECM) synthesis by trabecular meshwork cells leading to accumulation of ECM and decreases aqueous outflow. Trabecular meshwork represents a specialized tissue composed of various ECM components including fibronectin, laminin and collagen IV [28]. The composition of these ECM components in the trabecular meshwork can influence the meshwork ultra-structure and function including maintenance of outflow facility. The trabecular meshwork cells grown in high glucose conditions up regulates mRNA and protein synthesis of fibrolactin and the excess deposition of fibrolactin may be a common biochemical link that on one hand contributes to the development of thickened vascular basement membranes in diabetics microangiopathy and on other hand alters the structural contents, compromises resiliency, reduces cellularity, blocks the aqueous outflow in the trabecular meshwork and leads to increased IOP leads to the development of POAG in persons with diabetics [28, 29]. besides increased IOP is associated with death of retinal ganglion cells [30, 31] and leads to optic neuropathy which progressively damages optic nerve head due to mechanical compression causing progressive loss of optic nerve fibre and visual file loss.

The association between Diabetes and POAG is not new. In 1971 Becker [32] stated “Diabetes Mellitus occurs more often in patients with Primary Open Angle Glaucoma than in nonglaucomatous populations. Similarly, Glaucoma is more prevalent in diabetic than in non-diabetic population”. Considerable controversy exists in literature. While several studies show an association between the two diseases, several others fail to show any significant association. Most of these studies were comparatively small, used differing definitions of glaucoma and were clinical, rather than community based. The disparity in results of those denying a correlation between diabetes mellitus and POAG and those supporting it could be due to various reasons. One may be glaucoma case misclassification among subjects (as development of visual field loss in diabetics may mimic glaucoma) or different definitions and varying criteria for diagnosis. Another may be the variations in diagnostic criteria for diabetes such as self-reported cases, medication use and fasting / non-fasting / postlunch blood glucose levels. Besides this, selection bias in cases could also be a factor.

Diabetes mellitus is a major health problem in India, with its incidence increasing every day. Diabetes is associated with long-term damage to various organs such as eye, kidney, heart, blood vessels and nerves. Diabetes mellitus has emerged as a major cause of vision loss and visual disability, not only in developed countries but also in developing countries. Besides its other ocular manifestations, diabetes also affects intra ocular pressure. The mechanism that causes higher intraocular pressure is not clear, but various etiologies have been postulated as genetic, autonomic dysfunction, and osmotic diffusion.

This study shows significantly higher intraocular pressures in patients with Type 2 diabetics. Since intraocular pressure is a known risk factor for glaucoma and considering that there is a greater prevalence of diabetes mellitus in patients diagnosed with glaucoma, this would suggest that diabetics be monitored regularly for intraocular pressure to detect an early onset of glaucoma in susceptible patients. Institution of early treatment could thus limit visual morbidity due to glaucoma in diabetic patients.

Conclusion

The data generated from the present study concludes that mean intra ocular pressure was higher in diabetic group as compared to non-diabetic group. Mean intra ocular pressure was higher in uncontrolled diabetic patients (HbA1c >6.5) when compared with controlled diabetic patients (HbA1c < 6.5). In diabetics IOP should be monitored regularly so as to prevent complications.

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