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Understanding and Managing Steroid-Induced Glaucoma : An Urgent Call for Awareness and Personalized Care

M N Islam[1]

In this issue of the JBGS, Dr. Anisur Rahman Khan, Dr. Bipul Kumar Dey Sarker et al has published their work on "Outcome of Topical steroid induce ocular hypertension in a tertiary eye care center in Bangladesh"

They have shown 60 patients who used topical steroid for 6 months developed steroid-induced glaucoma in 20% of the patients of which 10% underwent glaucoma surgery (Trab + MMC). The authors concluded that simple precautions can help avoid glaucoma brought on by steroids. By recognizing risk factors (such as POAG, family history, excessive myopia, diabetes mellitus, and connective tissue disease) and monitoring for ocular pressure, irreversible glaucomatous optic neuropathy can be avoided. To avoid self-medication, it is essential to monitor for IOP after any kind of steroid prescription and to provide prompt care.

Steroid-induced glaucoma is a concerning yet often under-recognized complication arising from the use of corticosteroids in clinical practice. Though corticosteroids are a cornerstone of treatment for a wide range of inflammatory and autoimmune conditions, they present significant risks for ocular health, particularly in susceptible populations. This editorial seeks to shed light on the pathophysiology, risk factors, clinical management, and preventive strategies for steroid-induced glaucoma. Greater awareness, early detection, and personalized approaches to corticosteroid prescribing and monitoring can help mitigate the risk and protect patients' long-term visual outcomes.

Corticosteroids have long been valued in medicine for their potent anti-inflammatory effects. Their versatility, evident in systemic, topical, inhaled, and even intraocular forms, makes them invaluable in managing inflammatory diseases, asthma, allergies, and autoimmune conditions. However, these therapeutic benefits can come with significant ocular side effects, most notably the development of steroid-induced glaucoma, an iatrogenic form of secondary open-angle glaucoma caused by corticosteroid therapy. Despite its serious implications, steroid-induced glaucoma remains under-recognized, often leading to delays in diagnosis and, consequently, irreversible visual impairment.

Pathophysiology of Steroid-Induced Glaucoma

The primary mechanism underlying steroid-induced glaucoma is increased resistance to aqueous humor outflow, attributed to corticosteroid-induced remodeling of the trabecular meshwork. Corticosteroids induce structural and functional changes in trabecular meshwork cells, including altered gene expression, deposition of extracellular matrix, and inhibition of phagocytosis. These changes lead to increased intraocular pressure (IOP), creating an environment conducive to optic nerve damage if unrecognized and untreated. The prolonged use of corticosteroids, even at moderate doses, can exacerbate these effects, underscoring the need for cautious and informed prescribing practices.

Clinical Presentation and Diagnosis

Patients with steroid-induced glaucoma may be asymptomatic until the disease has progressed significantly, making regular IOP monitoring crucial for those on long-term corticosteroid therapy. Clinicians should maintain a high index

of suspicion, especially in patients with identifiable risk factors or those exhibiting unexplained visual changes. A thorough examination, including visual field testing, optic nerve head assessment, and imaging to evaluate retinal nerve fiber layer thickness, should be performed in cases where steroid-induced glaucoma is suspected.

Management and Therapeutic Approaches

The cornerstone of managing steroid-induced glaucoma is reducing or discontinuing corticosteroid use, when feasible. This should be done under close medical supervision, especially if the steroids are essential for managing underlying systemic conditions. Alternative therapies, such as non-steroidal anti-inflammatory drugs, may be considered to maintain disease control while minimizing ocular risks. When corticosteroid therapy cannot be reduced, the use of IOP-lowering medications, such as prostaglandin analogs or beta-blockers, is indicated. In refractory cases, surgical interventions, including trabeculectomy or glaucoma drainage devices, may be necessary to achieve adequate IOP control.

Preventive Strategies

Preventing steroid-induced glaucoma begins with a conscientious approach to corticosteroid prescription. Shorter treatment durations, lower doses, and consideration of steroid-sparing agents can all reduce the risk. Patient education is equally important; individuals prescribed corticosteroids should be informed about the potential risk of glaucoma and encouraged to report any vision changes promptly. For patients requiring long-term therapy, regular ophthalmic assessments should be integrated into their care plans to ensure early detection and intervention if IOP begins to rise.

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Conclusion

Steroid-induced glaucoma exemplifies the delicate balance between therapeutic benefit and potential risk in modern medicine. As the use of corticosteroids continues to be widespread, so does the responsibility of healthcare providers to recognize, monitor, and manage the associated ocular complications. Personalized treatment approaches, informed by a careful assessment of patient-specific risk factors and ongoing ophthalmic monitoring, can help safeguard patients against this preventable cause of vision loss. With heightened awareness, timely intervention, and collaborative patient care, the medical community can better address steroid-induced glaucoma and improve long-term outcomes for those who rely on corticosteroids for their health and quality of life.

References

1. Chang, T.C., & Kass, M.A. (2012). The mechanism of steroid-induced IOP elevation and glaucoma. Ophthalmology Clinics of North America, 25(2), 197–203.
2. Vijaya, L., Asokan, R., & Panday, M. (2017). Steroid-induced glaucoma: What we need to know. Indian Journal of Ophthalmology, 65(6), 412–415.
3. De Moraes, C.G., & Liebmann, J.M. (2016). Detection and management of steroid-induced glaucoma. Journal of Current Glaucoma Practice, 10(1), 27–35.
4. Leung, D.Y., & Tham, C.C. (2015). Corticosteroid-induced ocular hypertension and glaucoma: A review of treatment options. Expert Opinion on Pharmacotherapy, 16(16), 2457–2467.
5. Lam, D.S.C., Leung, D.Y.L., & Wong, A. (2014). Importance of early detection and management of corticosteroid-induced glaucoma in at-risk patients. Clinical and Experimental Ophthalmology, 42(1), 94–96.
6. Adelman, R.A., Wei, E., & Adelman, R.V. (2013). Corticosteroids and their use in ophthalmology. Ophthalmology and Therapy, 2(2), 91–101.

Original Article

An Evaluation of Disease Burden and Prevalence of Glaucoma in Bangladesh

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Abstract

Background: Glaucoma is the second leading cause of blindness worldwide.Being asymptomatic, a significant proportion of these patients do not present at the appropriate time. This study was carried out to estimate the epidemiology of glaucoma using a representative population-based sample across Bangladesh.This study also aims to estimate the health and social burden of glaucoma in the country.

Methods: A cross-sectional household survey was performed to identify target population aged 35 and over. A multi-stage stratified cluster random sampling was done to select households, the survey being conducted on 13791 residential households, with 17,002 completed interviews. International Society for Geographic and Epidemiological Ophthalmology (ISGEO) definitions were used to define glaucoma and glaucoma suspect cases.

Results: 12000 patients were screened with a male to female ratio of approximately 1:1. A larger percentage (3.9%) of males were affected with glaucoma, compared to 2.5% of females (3.19%). 1206 patients (10.0%) were diagnosed as glaucoma suspect, with females (12.0%) comprising a majority over males (8.1%). Approximately 6% of patients had an occludable angle. Primaryglaucoma comprised 94.6% of all diagnosed glaucoma, with normotensive glaucoma forming almost 83% of open angle glaucoma.The survey included self-reported visual disability to assess glaucoma's impact on daily activities and quality of life.

Conclusions: Extrapolating the results of the population-based survey, this survey estimates that there are about 2 million glaucoma patients and nearly 6 million glaucoma suspect cases in Bangladesh. Primary open angle glaucoma (POAG) is far more common, affecting over 4 out of 5 patients, with males more affected. Primary angle closure glaucoma (PACG) is less common, but appears to affect females more. It found that glaucoma often impairs reading and seeing nearby objects, with nearly half of patients reporting difficulty walking in the dark.

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Introduction

Glaucoma is the second most common cause of blindness worldwide and is the leading cause of irreversible blindness, accounting for 8% of total blindness[1]. Since glaucomatous damage is irreversible, and asymptomatic in the early stages, appropriate detection and monitoring is essential.

With a population of 165 million,Bangladesh has a potentially significant glaucoma burden[2]. Ahospital-based glaucoma survey in Bangladesh carried out in 2004estimated that approximately 586,000 people in the country would have glaucoma, out of a population of approximately 26 million adults aged 35 years and older[3]. Prevalence data for glaucoma in the South-East Asian region are relatively scant. No population-based data for glaucoma is currently available in Bangladesh. However, while hospital-based studies have assumed approximately equal numbers for angle closure and open angle glaucoma, studies in different states have indicated regional differences. Therefore, we have planned our current study ensuring representation from different divisions of Bangladesh, as well as a mix of rural and urban populations, to identify potential differences therein.

Thecurrent glaucoma survey was done to outline the epidemiology of glaucoma using a large, nationally representative population-based sample. Specific objectives of the study include determination of the prevalence of glaucoma in

people 35 years and above, and determining socio-demographic attributes such as gender, social class, occupational categories, and literacy levels, among others. To the best of our knowledge, this is the first and only population-based survey to estimate the prevalence and epidemiology of glaucoma in Bangladesh andto assess the disease burden of glaucoma patients in terms of status of disability and quality of life.

Methods

Study design

A cross sectional household survey was conducted to estimate the prevalence of glaucoma. Consecutive households were interviewed, and socioeconomic characteristics recorded. Eligible population (35 years and above) were registered and invited to attend adesignated facility for glaucoma screening at different time points.

Sampling methods and sample size

A multi-stage stratified cluster random sampling was employed, stratified by place of usual residence (urban/rural), and sampling frames were derived from the 2011 national census. The frame was divided into two domains: urban areas and rural areas. Selection was then independently conducted for each domain in each of the eight divisions. However, the proportion of people living in rural areas varied by division,which was also considered during stratification.

A total of 140 cluster sample sites were selected (89 rural, 51urban), with an average of 100 households per cluster. All households within the cluster were interviewed. With this design, the survey selected 14,000 residential households, and estimated to interviewabout 21,000 adult individuals. The sampling weight was applied to ensure the actual representation of the survey results at the national and domain levels. The sampling weights were adjusted for distribution of urban-rural households in the survey based on the urban-rural distribution in the 2011 population census[4].

The survey was conducted in 13791 residential households (5002 urban, 8789 rural), with a sample of 17,002 completed interviews with individual 35 years and above (6057 urban, 10945rural).

All invited participants for this survey were brought to the particular venue or survey spot for clinical examination (58 spots in 66 thanas/upazilas). Participants were examined only after obtaining Informed consent.

Teams and quality control

The study employed 9 qualified and experienced field staff (6 Field Investigators, 2 Field Supervisors, and 1 Field manager) for field level data collection, who received hands on training with role playing, interviewing techniques and record keeping from glaucoma specialists, ophthalmologists, and statisticians. Two sets of staff were involved – the advance enumeration team and the eye examination team.Field data collectors were supervised by field managers and supervisors. The supervisorsled performance reviews for team members, and ophthalmic nurses and optometrists were supervised by the glaucoma specialists (designated as team leaders). Investigators made periodic visits to field sites for monitoring and performance checks.

Clinical examinations

Best corrected visual acuity was recorded using a Snellen distance vision chart at 6 metres by an optometrist. All slit lamp (Haag-Streit, Haag-Streit, Bern, Switzerland) examinations were performed by the ophthalmologist. Intraocular pressure (IOP) was measured by Goldmann applanation tonometry following corneal anaesthesiaby oxybuprocainehydrochloride. Gonioscopy was carried out on all eyes of all participants by Goldmann four mirror gonio lens and the angle of the anterior chamber graded by Shaffer's angle grading system. Angles were defined as either open or narrow, depending on whether less than onethird of posterior (usually pigmented) trabecular meshwork circumference was seen.Central corneal thickness (CCT) (Ocuscan, Alcon Laboratories, USA) was measured in all patients by and CCT corrected IOP was used for analysis.

The optic disc was examined after dilating each

pupil (if angles graded as "open") with one drop of a mixture of tropicamide 0.8 percent and phenylephrine 5 percent (Aristopharma, Dhaka, Bangladesh). Disc was examined through an undilated pupil if angles were judged narrow or closed. A +90D lens (Volk) was used for slit lampbiomicroscopy.

The ratio of the longest vertical diameter of the cup to the longest vertical diameter of the disc was estimated as the VCDR for each eye. Any asymmetry of the VCDRs between the two eyes, narrowest part of neuroretinal rim, any notching at the cup margin, disc pallor, and disc haemorrhage were noted. A red free light was used to check for any defect in the nerve fibre layer.

Threshold visual field testing was carried with Humphrey visual field analyzer (ZEISS, USA. An automated threshold related single stimulus supra-threshold program was used to check 68 points in the central 25 degrees in each eye.

Criteria for Glaucoma Diagnosis

International Society for Geographic and Epidemiological Ophthalmology (ISGEO) definitions were used, with three levels of evidence[5].

Category 1: VCDR 0.7 in either eye or asymmetry of 0.2 or more between eyes and a visual field defect consistent with glaucoma;

Category 2: VCDR 0.9 in either eye or right-left asymmetry of 0.3 or more, and a dependable field test result could not be done;

Category 3: IOP 26 mm Hg and visual acuity worse than 3/60, or evidence of previous glaucoma filtering surgery, when optic disc could not be examined because of media opacity;

Criteria for glaucoma suspect diagnosis

- Disc suspect: Met category 1 (but not category 2) disc criteria, but no definite field defects.
- Field suspect: Definite field defects, but not meeting category 1 disc criteria
- Optic disc margin haemorrhages
- IOP>97.5 percentile
- Occludable drainage angle (defined as one in which the pigmented/posterior trabecular meshwork could be seen for less than 120^{o} of the angle circumference)

Visual field tests were be judged to be reliable if fixation losses 20 percent, false positives 25 percent and false negatives 20percent.

Type of glaucoma

Glaucoma was classified as primary and secondary glaucoma. Primary glaucoma was classified as primary open-angle glaucoma (POAG) or primary angle-closure glaucoma (PACG) according to angle morphology viewed by gonioscopy. IOP less than 21mmHg in patients fulfilling other POAG criteriawere considered as Normal tension glaucoma (NTG).Glaucoma was classified as secondary where there was an identifiable underlying cause such as AC angle neovascularization, exfoliation, pigment dispersion, trauma, surgical procedure or uveitis.

Wealth index and Wealth Quintile: The survey used wealth index to measure inequalities in household characteristics, in the use of health and other services, and in health outcomes. This index used in many demographic and health survey (DHS) and other country-level surveys (Rutstein et al. 2000). It serves as an indicator of household-level wealth that is consistent with expenditure and income measures (Rutstein 1999). The wealth index is constructed using household asset data via principal components analysis. After computed the index, wealth quintiles (from lowest to highest) are obtained by assigning thehousehold score to each household member, ranking each person in the population by his or herscore, and then dividing the ranking into five equal categories, each comprising 20 percent of the population.

Measurement of eye diseases burden: There are several methods are largely used in measuring the personal burden of eye diseaseand vision impairment on health and functional well-being (Cynthia & Gerald 2007). This study used questionnaires method with patient's own self-reported perspective on difficulty in engaging in

everyday activities, psychological well-being, and/or health status to assessthe personal burden of eye disease.

Data Analysis

Data entry was done using the2 Census and Survey Processing System (CS-Pro) software and exported to SPSS 23 (SPSS Inc.) for analysis. The prevalence for glaucoma and glaucoma suspect were estimated. Bi-variate analysis was performed to investigate the associations of gender, age, area of residence, education and occupational status with occurrence of glaucoma. The 95% confidence intervals for the prevalence estimates were also determined.

Ethical Approval

The Bangladesh Medical Research Council (BMRC) provided written ethical clearance for this survey in February 2021. Directorate General of Health Services (DGHS) approval was obtained in April 2021 after ethical clearance.

Results

Out of a total of 21387 households, 13791 households with an eligible population of 27205 were contactable. 17002 consented to the survey, and 12000 attended the final eye exam at the designated venue.

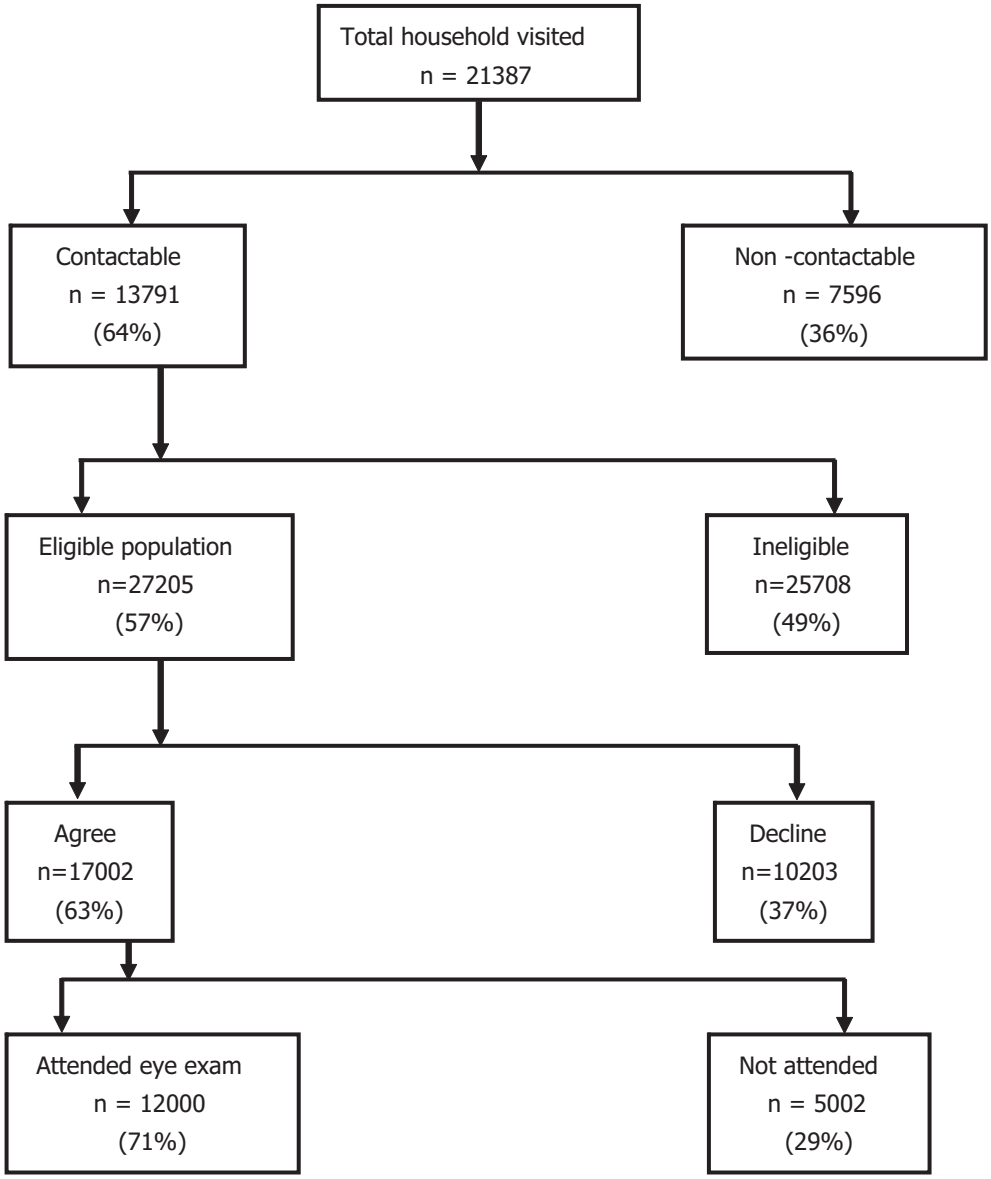

Table 1 : Socio-demographic characteristics of participants with glaucoma in the study population

		Total	Participants with glaucoma		
		N	N	%	95% CI
Sex					
	Male	5953	233	3.9	3.41-4.39
	Female	6047	153	2.5	2.07 - 2.74
					P <0.000
Age group					
	<40	1639	33	2.1	1.41-2.79
	40-49	3617	90	2.5	1.99-3.01
	50-59	3057	106	3.5	2.85-4.15
	60-69	2392	107	4.5	3.67-5.33
	>=70	1296	47	3.7	2.67-4.73
					P<0.000
Residence					
	Urban	2777	100	3.6	2.91-4.29
	Rural	9223	286	3.1	2.75-3.45
					P = 0.19
Region					
	Barisal	767	31	4.1	2.7-5.5
	Chattogram	2182	66	3.0	2.28-3.72
	Dhaka	3142	126	4.0	3.31-4.69
	Khulna	1395	41	2.9	2.02-3.78
	Mymensingh	955	23	2.4	1.43-3.37
	Rajshahi	1519	46	3.0	2.14-3.86
	Rangpur	1375	29	2.1	1.34-2.86
	Sylhet	682	21	3.0	1.72-4.28
					P = 0.01
Education					
	None	6170	186	3.0	2.57-3.43
	Primary level	1985	48	2.4	1.73-3.07
	Secondary level	2616	111	4.2	3.43-4.97
	More than secondary	1229	38	3.0	2.05-3.95
					P = 0.003

Males and females formed approximately equal halves among the respondents (50.4% female, 49.6% male), but had a slightly lower percentage of glaucoma overall (2.5%) compared to males (3.9%). Dhaka division accounted for the highest proportion of glaucoma patients, and over half of the patients had no primary education. Participants were evenly divided across all wealth quintiles.

Table 2 : Percentage of Glaucoma and glaucoma suspect among male and female

Characteristics	Male		Female		Total	
	Percentage Glaucoma	Number	Percentage Glaucoma	Number	Percentage Glaucoma	Number
Glaucoma examined						
Glaucoma	3.9	233	2.5	153	3.2	386
No Glaucoma	96.1	5720	97.5	5894	96.8	11614
Glaucoma						
	3.9	233	2.5	153	3.2	386
VCDR 0.7 & Visual field defect or VCDR Diff. 0.2	3.5	206	2.1	125	2.8	331
VCDR 0.9 & NVFD or VCDR Diff. 0.3 & NVFD	0.2	13	0.2	13	0.2	26
IOP > 26 & visual acuity worse than 3/60 or visual acuity worse than 3/60 & previous glaucoma filtering surgery	0.2	14	0.3	15	0.2	29
Glaucoma Suspect	8.1	480	12.0	726	10.0	1206
Disc suspect [a]	2.3	136	2.1	126	2.2	262
Field suspect [b]	1.0	59	1.4	87	1.2	146
IOP>97.5 percentile [c]	1.2	71	1.2	71	1.2	142
Occludable angle [d]	3.6	214	7.3	442	5.5	656
Total	100.0	5953	100.0	6047	100.0	12000

a. Disc suspect: Those who met category 1 (but not category 2) disc criteria, but no definite visual field defects.

b. Field suspect: Those with definite visual field defects, but not meeting category 1 disc criteria

c. Those with IOP>97.5 percentile

d. Those with occludable drainage angle (defined as one in which the pigmented/ posterior trabecular meshwork could be seen for less than 120^{o} of the angle circumference)

Overall, 3% of participants were found to be affected with glaucoma (Males 3.9%, Females 2.5%). The vast majority (85.7%) of patients were diagnosed based onVCDR 0.7 & Visual field defect or VCDR Diff. 0.2. 1206 patients (10.0%) were diagnosed as glaucoma suspect, with females (12.0%) comprising a majority over males (8.1%). This is a reversal of the pattern seen with diagnosed glaucoma patients, where males formed a higher proportion of diagnosed cases. Approximately 6% of patients had an occludable angle, with females almost double the number of male patients.

Table 3: Proportions of the different types of Glaucoma

Glaucoma Type	**Proportion of Glaucoma**	
	N	%
POAG	303	78.4
Normal tension	251	83.3
PACG	62	16.2
Secondary Glaucoma	21	5.5
Neovascular Glaucoma	5	1.3
Lens-induced glaucoma	4	1.04
Pseudoexfoliation Glaucoma	2	0.52
Steroid-Induced Glaucoma	2	0.52
Traumatic Glaucoma	4	1.04
Uveitic glaucoma	4	1.04
Total Glaucoma	**386**	**100%**

Primary glaucoma formed most of the glaucoma patients (94.6%). Primary open angle glaucoma was the most common, forming 78.4% of the population, with angle closure glaucoma forming 16.2% of the population. Within primary open angle glaucoma, it is important to note that the vast majority of patients (83.3%) were diagnosed as normal tension POAG, indicating IOP was in the normal range. Of the secondary glaucomas, there were approximately equal proportions of neovascular, lens-induced, uveitis and traumatic glaucomas. This study exhibits the relationship between corneal thickness and IOP. The results show that median IOP and median CCT were 12 and 552 respectively.

Among participants, 13.5% had no difficulty reading. About 64.3% faced some difficulty, and nearly 1% had severe difficulty. Among glaucoma patients, 11.7% had no difficulty, compared to 15.5% of normal participants and 10.6% of glaucoma suspects. Severe difficulty affected 3% of glaucoma patients, slightly higher than normal participants (2.5%) and glaucoma suspects (2.8%).

Table 4 : Difficulty in reading books or newspaper due to visual impairment of respondents

Percentage distribution of population age 35 and above had level of difficulty in reading books or newspaper according to diagnosis

Level of difficulty	Normal	Glaucoma	Glaucoma suspect	Other eye disease	Total
No difficulty	15.5	11.7	10.6	8.9	13.5
A little bit difficulty	28.6	24.2	32.6	23.0	27.7
Moderate difficulty	35.0	32.8	32.2	39.5	35.7
Quite more difficulty	18.4	28.3	21.8	26.5	20.7
Severe difficulty	2.5	3.0	2.8	2.1	0.8
Total	100.0	100.0	100.0	100.0	100.0
Number of Participants	7880	386	1206	2528	12000

Nearly half (46.3%) of participants had no difficulty walking in the dark due to visual impairment, while about one-third (34.3%) experienced slight difficulty. Only 3.2% faced significant challenges, with glaucoma patients showing the highest rates: 1.7% had severe difficulty, and 4.4% had moderate difficulty.

Around 36.2% of glaucoma patients reported no difficulty.

Table 5 : Difficulty to walk in the dark due to visual impairment of respondents

Percentage distribution of population age 35 and above had level of difficulty to walk in the dark according to diagnosis

Level of difficulty	Normal	Glaucoma	Glaucoma suspect	Other eye disease	Total
No difficulty	51.4	36.2	44.1	32.5	46.3
A little bit difficulty	31.3	37.2	37.5	42.1	34.3
Moderate difficulty	14.2	20.5	16.2	21.5	16.2
Quite more difficulty	2.2	4.4	1.6	3.3	2.4
Severe difficulty	0.9	1.7	0.6	0.6	0.8
Total	100.0	100.0	100.0	100.0	100.0
Number of Participants	7880	386	1206	2528	12000

Nearly half (51%) of participants had difficulty seeing at night due to visual impairment, with 33.1% reporting slight difficulty and 1% severe difficulty. About 54.2% had no trouble walking on uneven ground, while 46% of glaucoma patients reported the same. Severe difficulty in walking on uneven ground was higher among glaucoma patients (2.3%). Over 30% of all participants had trouble seeing objects from the sides, compared to over 40% of glaucoma patients. Around 30% of all participants had difficulty crossing roads, while 40% of glaucoma patients faced this issue, with 24.1% reporting slight difficulty.

Discussion

The current survey was the largest, population-based survey of glaucomaand aimed to give precise estimates of the disease burden in the general population. Because the areas of study were randomly selected, we believe the sample was nationally representative by age, gender, ethnicity, rural and urban residence and socioeconomic status, which coupled with the high response rate are generalizable to the whole country.

A total of 386 glaucoma patients were diagnosed, with 1206 patients estimated to be glaucoma suspects. Extrapolating this data, it has been estimated that in people over 35 years of age (based on a 2022 census carried out by the Bangladesh Bureau of Statistics), there arealmost 2 million (19,43,470) glaucoma patients in the country (11,99,980 male; 7,43,490 female) and almost 6 million (59,25,030) were glaucoma suspect patients.

The prevalence of glaucoma is 3.2 percent (95 percent CI: 2.79–3.64), with a higher prevalence among males (3.9%)compared to females (2.5%). The prevalence of glaucoma suspectsis 10.1 percent (95 percent CI: 9.05–11.12), which conversely were more among females than males (12.0 percent versus 8.1 percent). A hospital-based survey carried out by Rahman et al had an age-standardized prevalence of 2.4 percent, which is quite comparable with the current study [3].

Studies carried out in India has had varied outcomes, ranging from 3.2 to 6.1 % based on different populations [6].A study carried out in Nigeria estimated an overall prevalence of 5.02%, further proof of the variability of prevalence across different studies [7].

The prevalence of glaucoma was slightly higher in urban areas than in rural areas (3.6 percent versus 3.1 percent). The patterns were similar, with higher proportions among males (4.6 percent in urban and 3.7 percent in rural) compared to females (2.6 percent versus 2.4 percent). A study carried out in Hyderabad in a predominantly urban population found a prevalence of 6.1% [8,9], against another study carried out in Tamil Nadu in a rural population which found a prevalence of 3.2% [10]. Our study did not elicit such marked differences between urban and rural populations.

The majority of participants had primary open angle glaucoma (78.4 percent), which was higher among males (83.7 percent) than females (70.6 percent). Primary angle closure glaucoma (PACG) comprised 16.6 percent of total glaucoma patients, and was more common among females (21.6 percent) than males (12.4 percent). A study in West Bengal [5], an ethnically similar population, found predominantly POAG patients, and strikingly low PACG patients, with a ratio of 10:1. POAG also formed the majority in the current study, with a

POAG to PACG ratio of approximately 4:1. This is also consistent with the previous hospital-based study carried out in Dhaka, which found similar proportions. Among the glaucoma suspect patients, 54.4 percent patient had angle closure and 45.6 percent patients had open angles.Since POAG outnumbers PACG by far, it can be assumed that most patients with occludable angles do not progress to PACG. The prevalence (10.0%) of glaucoma suspect underscores the importance of appropriate screening as a significant proportion of these patients will develop glaucoma with consequent visual morbidity.

Another finding of note is that a large proportion of the patients (83.3%) had pressures in the normal range, i.e. POAG with low tension. This shows that IOP alone cannot determine glaucoma in participants, and is an important consideration for potential screening programs. The prevalence of glaucoma has also been shown to be significantly associated with increasing age, correlating with the other studies carried out in the region.

The survey showed that 97.7percent of the patient population of both sexes of age 35 above reported experiencing one or more eye problems prior to the survey. Commonly reported eye problems included dimness of vision (90.4 percent), watering (30.5 percent), and itching (21.6 percent).

Regarding awareness, only 2.9 percent of total participants had previous knowledge of the disease. Awareness was found to be some what higher among the younger respondents. Urban residence and education status have a very strong positive association with glaucoma awareness. Six percent of urban participants had heard about glaucoma compared with only 2.0 percent of rural participants. Knowledge of glaucoma ranged widely from only 0.2 percent among illiterate participants to 23 percent among those with at least secondary education. One percent of the participants reported their family member had diagnosed glaucoma, while 93 percent did not know about it.Study limitations include a low response rate due to difficulties in reaching patients in rural areas, exacerbated by COVID-19 restrictions. The survey included self-reported visual disability to assess glaucoma's impact on daily activities and quality of life.

Conclusions

The survey estimates around 2 million glaucoma patients and nearly 6 million glaucoma suspect cases in Bangladesh. Primary open-angle glaucoma (POAG) is more common, affecting 4 out of 5 glaucoma patients, with a higher prevalence in males, while primary angle-closure glaucoma (PACG) is less common but more frequent in females. Glaucoma prevalence increases with age. Secondary glaucoma, though rare, has varied causes such as trauma and uveitis complications, warranting further research.

The study also highlights a high prevalence of glaucoma suspect cases and low public awareness, emphasizing the need for national health programs to implement effective screening methods. Glaucoma often impairs reading, seeing nearby objects, and nearly half of patients report difficulty walking in the dark. With this survey, appropriate resources can be allocated to reduce avoidable blindness in the country.

References

1. Pascolini D, Mariotti SP. Global estimates of visual impairment: 2010. Br J Ophthalmol. 2012 May;96(5):614-8. doi: 10.1136/bjophthalmol-2011-300539

2. Bangladesh Population and Housing Census 2022, preliminary result, Statistics and Information Division, Ministry of Planning, Dhaka [online]. 2022.http://www.bbs.gov.bd/site/page/47856ad0-7e1c-4aab-bd78-892733bc06eb/Population-and-Housing-Census (accessed April 2022).

3. Rahman MM, Rahman N, Foster PJ, Haque Z, et al. The prevalence of glaucoma in Bangladesh: a population based survey in Dhaka division. Br J Ophthalmol. 2004 Dec;88(12):1493-7. doi: 10.1136/bjo.2004.043612

4. Bangladesh Population and Housing Census 2011, Statistics and Information Division, Ministry ofPlanning, Dhaka [online].https://bbs.gov.bd/site/page/47856ad0-7e1c-4aab-bd78-892733bc06eb/Population-and-Housing-Census

5. Foster PJ, Buhrmann R, Quigley HA, et al. The definition and classification of glaucoma in prevalence surveys. Br J Ophthalmol. 2002 Feb;86(2):238-42. doi: 10.1136/bjo.86.2.238

6. Raychaudhuri A, Lahiri SK, Bandyopadhyay M, et al. A population based survey of the prevalence and types of glaucoma in rural West Bengal: the West Bengal Glaucoma Study. Br J Ophthalmol. 2005 Dec;89(12):1559-64. doi: 10.1136/bjo.2005.074948

7. Kyari F, Entekume G, Rabiu M, Spry P et al. Nigeria National Blindness and Visual Impairment Study Group. A Population-based survey of the prevalence and types of glaucoma in Nigeria: results from the Nigeria National Blindness and Visual Impairment Survey. BMC Ophthalmol. 2015 Dec 12;15:176. doi: 10.1186/s12886-015-0160-6

8. Dandona L, Dandona R, Srinivas M, et al. Open-angle glaucoma in an urban population in southern India: the Andhra Pradesh eye disease study. Ophthalmology. 2000 Sep;107(9):1702-9. doi: 10.1016/s0161-6420(00)00275-x

9. Dandona L, Dandona R, Mandal P, et al. Angle-closure glaucoma in an urban population in southern India. The Andhra Pradesh eye disease study. Ophthalmology. 2000 Sep;107(9):1710-6. doi: 10.1016/s0161-6420(00)00274-8

10. Ramakrishnan R, Nirmalan PK, Krishnadas R, et al. Glaucoma in a rural population of southern India: the Aravind comprehensive eye survey. Ophthalmology. 2003 Aug;110(8):1484-90. doi: 10.1016/S0161-6420(03)00564-5. Erratum in: Ophthalmology. 2004 Feb;111(2):331

Original Article

"Outcome of Topical steroid induce ocular hypertension in a tertiary eye care center in Bangladesh"

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Abstract

Introduction: Corticosteroids are anti-inflammatory drugs commonly used to treat various ocular and systemic conditions. The use of steroids can lead to significant ocular side effects. The more common ocular side effects are cataracts and ocular hypertension. This study aimed to determine the outcome of topical steroid-induced ocular hypertension in a tertiary care center.

Methods: This prospective observational study was conducted in the Department of Glaucoma, Ispahani Islamia Eye Institute and Hospital from October 2018 to March 2019. In this study, we included 60 patients with steroid-induced ocular hypertension who fulfilled the inclusion criteria and attended the glaucoma department Ispahani Islamia Eye Institute, and Hospital.

Result: The mean age was 32.2±8.5 years. The majority of the patients 26 (43%) had dimness of vision, followed by eye aches with headaches (15%). The majority of the patients' IOP was more than 24 mmHg. At the last follow-up, IOP was 14 mmHg. The majority of the patients' IOP was controlled with anti-glaucoma medication. Most of the patients required two drops (Brimonidine+ Timolol 0.5%) plus PG analogue. Among all patients, 20% developed steroid-induced glaucoma of which 10% underwent glaucoma surgery (Trab + MMC).

Conclusion: The use of different forms of corticosteroids is becoming more common and can induce a significant elevation of IOP. It could develop steroid-induced glaucoma in case of prolonged therapy or could not be recognized early. To prevent SIG, it is recommended that the choice of corticosteroid, duration, and frequency of instillation should be chosen wisely best on the desired effects.

Keywords: Outcome, Corticosteroid,Steroid-induced ocular hypertension

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Introduction

Corticosteroids are anti-inflammatory drugs that are commonly used to treat a variety of systemic and ocular conditions. The usage of steroids can have serious adverse consequences on the eyes. The more common ocular side effects are cataracts and ocular hypertension. Intraocular pressure (IOP) elevation after steroid use is well documented.[1,2] Steroids induce ocular hypertension when administered with topical, periocular, and even systemic or inhalational routes.[3] Most studies describing steroid-induced glaucoma have focused on adults.[4,5]

The most prevalent risk factor for progressive optic neuropathy in glaucoma is IOP, which is impacted by oral and topical corticosteroids. Around 4% to 6% of the population are "steroid responders" who develop an increase in IOP of >15 mm Hg or with IOPs of >31 mm Hg after daily topical corticosteroid use for 4–6 weeks.[6]

OHT is characterized as having a full visual field and a healthy optic disc with an IOP of more than 21 mmHg. Secondary ocular hypertension (OHT) can be brought on by external steroids that are injected topically (via peri- and/or intraocular injections) or taken orally. [7,8] The risk of inhaled nasal sprays causing secondary OHT is less clearly defined.[9] The risk of steroid-induced OHT varies by route of administration, duration of treatment, type of steroid, and

preexisting history of glaucoma, among other factors. For example, approximately 40% of the general population developed OHT after a 4–6week course of topical 0.1% dexamethasone, so-called steroid responders, compared with nearly 100% of patients with primary open-angle glaucoma (POAG) or normal-tension glaucoma. [10-12] The etiology of steroid-induced OHT has been linked to the myocilin gene that is upregulated by steroid treatment in cultured trabecular meshwork cells.[13] Stone et al reported that myocilin gene mutations were also associated with the development of POAG.[14] Although steroid-induced OHT usually reverses after cessation of steroid administration, it remains an important risk factor for the development of glaucomatous optic neuropathy.[10,15]

In susceptible individuals, topical steroids induce a rise in intraocular pressure (IOP), probably by increasing resistance to aqueous flow through the trabecular meshwork.[16,17] If not recognized and treated, steroid-induced intraocular hypertension may progress to glaucoma with optic nerve damage and visual field defects.[18,19] Around 5% of adults may be at risk of developing a 'high' pressure response (an elevation of more than 15 mm Hg from baseline) and 35% an 'intermediate' response (6–15 mm Hg elevation above baseline).[7] Children may be more susceptible than adults, with up to 60% developing a 'high' IOP response.[20-23] This study aimed to determine the outcome of topical steroid-induced ocular hypertension in a tertiary care center.

Methodology & Materials

This prospective observational study was conducted in the Department of Glaucoma, Ispahani Islamia Eye Institute, and Hospitalfrom October 2018 to March 2019. In this study, we included 60 patients with steroid-induced ocular hypertension who fulfilled the inclusion criteria and attended the glaucoma departmentIspahani Islamia Eye Institute, and Hospital.

These are the following criteria to be eligible for enrollment as our study participants: a) Patients aged more than 18 years; b) Patients with a history of topical steroid use; c) Patients with IOP > 21 mmHgwere included in the study And a) Patients with all primary glaucoma; b) Patients with any history of acute illness (e.g.,renal or pancreatic diseases, ischemic heart disease, asthma, COPD, etc.); c) Patients who were unwilling to participatewere excluded from our study.

Data collection procedure: The patients had a routine glaucoma workup including IOP measurement, assessing visual acuity, Goniouscopy to see ant chamber angle, and fundoscopy for disc evaluation. BCVA was recorded using the Snellen chart. IOP measurement was performed using Goldman applation tonometry. Gonioscopy was performed by 4 mirror Goniolens. Demographic elements of age, gender, occupation, name of topical steroid medical history, Family history of glaucoma as well as patient's address were recorded. All the subjectshad been explained the benefits and risks of the procedure before obtaining informed consent. Follow-up visits will be scheduled for 7 days 1st follow-up, 1 month 2nd follow-up, and 3 months 3rd follow-up. Patients were followed up for 3 months and evaluated for IOP, BCVA, optic disc, number of anti-glaucoma medications, and requirement of glaucoma surgery of Trabeculectomy and Mitomycin C (Trab MMC).

Statistical Analysis: All data were recorded systematically in preformed data collection form. Quantitative data was expressed as mean and standard deviation. Qualitative data was expressed as frequency distribution and percentage. A p-value <0.05 was considered as significant. Statistical analysis was performed by using SPSS 20 (Statistical Package for Social Sciences) for Windows version 10. The Ethical Review Committee of Ispahani Islamia Eye Institute and Hospital approved the study.

Results

Table 1: Demographic characteristics of our study patients (n=60)

Variables	N	P(%)
Age (Years)	32.2(±8.5)	
Gender		
Male	46	77.00
Female	14	23.00
Occupation		
Student	15	25.00
Businessman	21	35.00
Job holder	12	20.00
Farmer	5	8.33
Housewife	7	11.67
Complaints		
Redness and itching	31	51.67
Dimless of vision	26	43.33
Headache and eyeche	13	21.67
Halos around light	6	10.00
Co-morbidities		
Hypertension	4	6.67
Diabetes	10	16.67
Diabetes with hypertension	5	8.33

In Table 1 we found the mean age was 32.2 (±8.5) years. Most patients were male (77%) compared to female (23%). Most patients were businessmen (35%). The majority of the study patients were present with redness and itching (51.67%), followed by 26 (43%) patients who had dimness of vision, 13(21.67%) patients had headaches and eye aches, and 6(10%) patients had complaints of seeing halos around the light. The common comorbidity was diabetes (16.67%).

Table 2: Distribution of our study patients by Corticosteroid drop

Name of Steroid	Number of Patients	Percentage	Duration (Month)
Dexamethason	24	40	7.73
Prednisolone	9	15	5.94
Loteprednol	16	26.7	4.44
Fluromethanol	4	6.7	2.29
Difluprednol	7	11.7	2.13
Total	n=60		4.51

Most of the patients 24(40%) used Dexamethason eye drops, followed by 16(26.7%) patients who used Loteprednol drops, 9(15%) patients used Prednisolone drops, and 7(11.7%) patients used Difluprednol drops. The average duration of all steroid drops was 4.51 months. The average frequency of all drops is 3-4 times per day.

Table 3: Record of Intraocular Pressure (IOP) of our study patients

IOP(mmHg)	No. of patient	Presentation	1st F/U	2nd F/U	3rd F/U
21-24	12	21	16	19	15
>24	48	26	21	16	14

Out of 60 patients, 12 patients have IOP 21-24 mmHg. Initially, these patients were treated without anti-glucoma medication. All patients' IOPs were controlled up to the last 3 months' follow-up period, Except 2 patients. One patient's baseline IOP was Rt eye 21, and Lt eye 13. We treated him just to stop steroid drop and F/U after 7 days. At 1st F/U, the patient's IOP was 25-13 after using an anti-glaucoma drop (Brimonidine+Timolol 0.5%), and IOP was 12-13 at 2nd F/U. Anti-glaucoma drop was stopped and F/U the patient after one month, without drop IOP was not controlled and the patient developed steroid-induced glaucoma. Another patient without anti-glaucoma drop IOP was not controlled at the last F/U (3 Months) period and this patient needed glaucoma surgery (Trab+MMC).

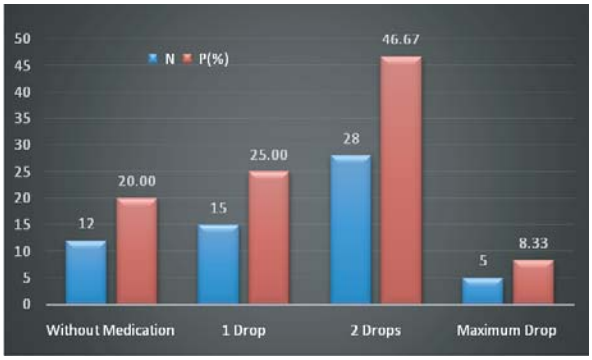
Table 4: Distribution of our study patients by visual outcome

Presentation	1st F/U(7 days)		Last F/U(3 months)	
NPL, PL, HM	Rt	Lt	Rt	Lt
1/60	2	2	1	1
2/60	2	2	1	1
3/60	6	8	2	3
6/60	8	6	4	2
6/36	2	2	2	2
6/24	2	2	3	3
6/18	5	8	5	5
6/12	8	5	10	10
6/9	14	11	16	11
6/6	11	14	16	17

Table 4 shows thatmost of the patients had a PVA of 6/9. Most patients had well-controlled IOP with anti-glaucoma medication. IOP-

controlled patients had improved BCVA 1-2 line of Snellen's chart. A total of 12 patients(20%) developed steroid-induced glaucoma and decreased or no improvement of vision.

Figure 1: Distribution of our study patients by number of anti-glaucoma medication



Among all 60 patients, 12 patients (20%) IOP was 24 mmHg. Upto 1st F/U (7 days) these patients were treated without anti-glaucoma medication. The majority of the patients 28 (47%) required 2 drops (Brimonidine + Timolol 0.5%) combined preparation with PG analog to control IOP, and 5 (25%) patients required 1 drop of either Brimonidine, Timolol 0.5%, or combination of both.

Discussion

The ocular hypertensive response is relatively dose-dependent and often returns to normal after stopping corticosteroid use in a month. However, if medication is continued, the response may not recover.

When steroid-induced glaucoma or OHT is identified, the triggering medication should be discontinued or, if that is not feasible, the dosage lowered. An alternative steroid formulation could be recommended. Stepwise care of steroid-induced open-angle glaucoma (OHT) is similar to that of primary-open-angle glaucoma in cases where the condition is permanent. Alpha-agonists, topical beta-blockers, systemic and topical carbonic anhydrase inhibitors, and prostaglandin analogues are all part of medical antiglaucomatous therapy. However, in situations when cystoid macular oedema or uveitic glaucoma coexist, prostaglandin analogues might not be helpful. Selective laser trabeculoplasty (SLT) may potentially work as a temporizing measure in patients with steroid-induced OHT.[24] Whilst trabeculectomy and tube shunts are the preferred surgical option in adults with corticosteroid-induced OHT or glaucoma, goniotomy should be considered for initial surgical treatment in children with persistent steroid-induced glaucoma.[25]

Steroid-induced intraocular pressure rise can occur at any age. Most investigations on steroid-induced glaucoma have focused on adults. Children have a more severe ocular hypertensive response to topical steroids than adults, and considerable IOP rise has been documented in newborns treated with nasal and inhalational steroids.[26,27]

The average present IOP 24 was 21 and more than 24 was 26 mmHg. After a 3months F/U period IOP was 14 mmHg. This represented a 43% reduction in average IOP from the presentation level. The initial course of treatment is stopping the steroid's use. The majority of the time, an acute elevation in IOP caused by steroids normalizes within a few days of stopping the medication, but chronic versions take one to four weeks. In rare instances, the IOP may not decrease, in which case antiglaucoma drugs or surgery would be required. The IOP elevation's reversibility appears to be influenced by the length of steroid therapy.

The majority of the patients' IOP was controlled with or without anti-glaucoma medication. 12 patients developed glaucoma out of this, 6 patients' IOP were controlled with anti-glaucoma medication. The next 6 patients needed glaucoma surgery (Trab +MMC) and after surgery, IOP was well-controlled in all of the 6 patients.At the end of the 3-month F/U period, the success rate was 80% with an average IOP 21mmHg with no additional medication or interventions. The qualified success rate was 20% with a mean IOP 21 mmHg with anti-glaucoma medications.This is similar to a previous study, Harju etal reported long-term use of steroids may develop optic nerve damage

(SIG) or cataracts. VA is not a good indicator of the success of glaucoma because there might be other confounding factors affecting the vision.[28]

Limitations of the study

Our study was a single-center study. We took a small sample size due to our short study period. After evaluating those patients, we did not follow upwith them for the long term anddo not know other possible interference that may happen in the long term with these patients.

Conclusion and recommendations

In our study, steroid-induced glaucoma is an iatrogenic illness that is preventable. Patients' unmonitored self-administration and local clinicians' unwarranted and unreasonable use of steroids, especially in underdeveloped countries, are signs of ignorance about the disease. Simple precautions can help avoid glaucoma brought on by steroids. By recognizing risk factors (such as POAG, family history, excessive myopia, diabetes mellitus, and connective tissue disease) and monitoring for ocular pressure, irreversible glaucomatous optic neuropathy can be avoided. To avoid self-medication, it is essential to monitor for IOP after any kind of steroid prescription and to provide prompt care.

So further study with a prospective and longitudinal study design including a larger sample size needs to be done to determine the long-term effect of topical steroid-induced ocular hypertension.

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References

1. Covell LL. Glaucoma induced by systemic steroid therapy. Am J Ophthalmol. 1958;45:108–109.
2. Goldmann H. Cortisone glaucoma. Arch Ophthalmol. 1962;68: 621–626.
3. Urban RC Jr, Dreyer EB. Corticosteroid-induced glaucoma. Int Ophthalmol Clin. 1993;33:135–139.
4. Kitazawa Y. Increased intraocular pressure induced by corticosteroids. Am J Ophthalmol. 1976;82:492–495.
5. Armaly MF, Becker B. Intraocular pressure response to topical corticosteroids. Fed Proc. 1965;24:1274–1278.
6. Clark AF. Steroids, ocular hypertension, and glaucoma. J Glaucoma 1995;4:354-69.
7. Armaly MF. Statistical attributes of the steroid hypertensive response in the clinically normal eye. I. The demonstration of three levels of response. Invest Ophthalmol. 1965;4: 187—97
8. Yamamoto Y, Komatsu T, Koura Y, et al. Intraocular pressure elevation after intravitreal or posterior subTenon triamcinolone acetonide injection. Can J Ophthalmol. 2008;43:42—7
9. Yuen D, Buys YM, Jin YP, et al. Effect of beclomethasone nasal spray on intraocular pressure in ocular hypertension or controlled glaucoma. J Glaucoma [Internet]. 2011 Jun 28. [cited 2012 Jan 10];Available from: http://www.ncbi. nlm.nih.gov/pubmed/21716127.
10. Lews JM, Priddy T, Judd J, et al. Intraocular pressure response to topical dexamethasone as a predictor for the development of primary open-angle glaucoma. Am J Ophthalmol. 1988;106:607—12
11. Armaly MF. Effect of corticosteroids on intraocular pressure and fluid dynamics. I. The effect of dexamethasone in the normal eye. Arch Ophthalmol. 1963;70:482-91
12. Armaly MF. Effect of corticosteroids on intraocular pressure and fluid dynamics. II. The effect of dexamethasone in the glaucomatous eye. Arch Ophthalmol. 1963;70: 492-9
13. Polansky JR, Fauss DJ, Chen P, et al. Cellular pharmacology and molecular biology of the trabecular meshwork inducible glucocorticoid response gene product. Ophthalmologica. 1997;211:126—39
14. Stone EM, Fingert JH, Alward WL, et al. Identification of a gene that causes primary open-angle glaucoma. Science. 1997;275:668—70
15. Wordinger RJ, Clark AF. Effects of glucocorticoids on the trabecular meshwork: towards a better understanding of glaucoma. Prog Retin Eye Res. 1999;18:629—67

16. Rhen T and Cidlowski JA. Antiinflammatory action of glucocorticoids – new mechanisms for old drugs. N Engl J Med 2005; 353(16): 1711–1723.
17. Zhang X, Clark AF and Yorio T. FK506-binding protein 51 regulates nuclear transport of the glucocorticoid receptor beta and glucocorticoid responsiveness. Invest Ophthalmol Vis Sci 2008; 49(3): 1037–1047.
18. Kaur S, Dhiman I, Kaushik S, et al. Outcome of ocular steroid hypertensive response in children. J Glaucoma 2016; 25(4): 343–347.
19. Spaeth GL, Monteiro de Barros DS and Fudemberg SJ. Visual loss caused by corticosteroid-induced glaucoma: how to avoid it. Retina 2009; 29(8): 1057–1061.
20. Brookes JL and Khaw PT. Steroid response in children. Clin Exp Ophthalmol 2005; 33: 229–230.
21. Ohji M, Kinoshita S, Ohmi E, et al. Marked intraocular pressure response to instillation of corticosteroids in children. Am J Ophthalmol 1991; 112(4): 450–454.
22. Kwok AK, Lam DS, Ng JS, et al. Ocular-hypertensive response to topical steroids in children. Ophthalmology 1997; 104: 2112–2116.
23. Lam DS, Fan DS, Ng JS, et al. Ocular hypertensive and anti-inflammatory responses to different dosages of topical dexamethasone in children: a randomized trial. Clin Exp Ophthalmol 2005; 33(3): 252–258.
24. Rubin B, Taglienti A, Rothman RF, Marcus CH, Serle JB. The effect of selective laser trabeculoplasty on intraocular pressure in patients with intravitreal steroid-induced elevated intraocular pressure. Journal of glaucoma. 2008 Jun 1;17(4):287-92.
25. Choi EY, Walton DS. Goniotomy for steroid-induced glaucoma: clinical and tonographic evidence to support therapeutic goniotomy. Journal of Pediatric Ophthalmology & Strabismus. 2015 May 1;52(3):183-8.
26. Desnoeck M, Casteels I, Casteels K. Intraocular pressure elevation in a child due to the use of inhalation steroids—a case report. Bull Soc Belge Ophtalmol. 2001;(280):97–100.
27. Ohji M, Kinoshita S, Ohmi E, Kuwayama Y. Marked intraocular pressure response to instillation of corticosteroids in children. Am J Ophthalmol. 1991 Oct 15;112(4):450–454.
28. Harju M, Suominen S, Allinen P, Vesti E. Long term results of deep sclerectomy in normal tension glaucoma. Acta ophthalmologica. 2018 Mar;96(2):154-60.

Original Article

Association of Serum Complement C3 with Primary Open Angle Glaucoma and its severity

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Abstract

Background: Primary open angle glaucoma (POAG) is the most common type of glaucoma, which may lead to irreversible blindness. Identifying risk factors for its development can aid disability prevention. While elevated intraocular pressure (IOP) is a key risk, some patients still experience vision loss despite IOP reduction. Evidence shows that low serum complement C3 is linked to early nerve damage and retinal ganglion cell (RGC) loss post IOP increase, implying C3's protective role against early glaucomatous damage.

Aims: To assess the association of serum complement C3 level with primary open angle glaucoma and its severity

Methods: This cross sectional comparative study was carried out into Department of Ophthalmology, Bangabandhu Sheikh Mujib Medical University (BSMMU) during January, 2022 to August, 2023. A total of 40 diagnosed cases of Primary open angle glaucoma was the study population and 40 patients were selected considering similar age group as comparison group without glaucoma. Complete clinical evaluation was done. Visual field analysis, color fundus photography (CFP), optical coherence tomography (OCT) Retinal nerve fibre layers (RNFL) were done in Ophthalmology department and serum complement C3 level was measured by immunonephelometry system in Department of Microbiology, BSMMU. Statistical analyses of the results were obtained by using Statistical Packages for Social Sciences (SPSS-26).

Results: The mean serum complement C3 level was 1.02 ± 0.12 (g/l) in POAG group and 1.09 ± 0.15 (g/l) in control group. The difference was statistically significant ($p < 0.05$) between groups. Most of the patients (47.5%) had severe glaucomatous damage, 27.5% mild and 25.0% moderate glaucomatous damage on the basis of Mean deviation (MD) of visual field. The mean serum complement C3 level was 1.07 ± 0.12 (g/l) in mild glaucomatous damage, 1.03 ± 0.14 (g/l) in moderate glaucomatous damage and 0.99 ± 0.99 (g/l) in severe glaucomatous damage. The mean Serum complement C3 was not statistically significant ($p > 0.05$) in different grading of severity of POAG patients.

Conclusion: The majority of patients displayed pronounced signs of severe glaucomatous damage. Furthermore, the analysis revealed reduced serum concentrations of C3 in individuals diagnosed with POAG than comparison group. However, there was no correction between serum complement C3 levels and the severity of POAG.

Keywords: Aqueous humor, Complement C3, Complement system, Primary open angle glaucoma.

Introduction

Primary glaucoma, which is characterized by cupping of the optic nerve head and impairment to the visual field, is one of the most common causes of irreversible blindness globally.[1-3] More than 70 million patients worldwide have visual impairment as a result of it.[4, 2] The most prevalent kind of glaucoma is primary open angle, which is around 6 times more prevalent than angle closure glaucoma. The prevalence of glaucoma worldwide among people aged 40 to 80 years is approximately 3.54%.[2, 5] There are, 64.3 million persons in the world between the ages of 40 and 80 were predicted to have glaucoma in 2013.[2] This figure is expected to rise to 76.0 million by 2020 and 111.8 million by 2040. There are 2.1% cases of definite glaucoma in Bangladesh. 70% of cases of primary glaucoma are Primary Open Angle Glaucoma (POAG) and there are around 1 million sufferers' worldwide.[6]

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Although increased intraocular pressure is a significant glaucoma risk factor[7], the precise mechanisms of increased IOP that contribute to the degeneration of retinal optic ganglion cells have not yet been identified.[8] Ageing, black ethnicity, male gender, genetic make-up, and high intraocular pressure have all been recognized as risk factors for POAG.[7, 2, 9, 10] IOP reduction is the mainstay of current treatment for glaucoma because it slows the disease progression in general.[8,11] Nevertheless, even when the IOP is successfully reduced, visual field loss continues in a sizable portion of individuals. This shows that elements other than IOP are involved in the development of the disease. In order to create new therapies that can enhance the present approach of IOP reduction, it is critical to identify these components and mechanisms.[12]

Evidence has been building over time that suggests increased IOP alters complement system pathways in non-neuronal cells like astrocytes, microglia, monocytes and RGCs.[13] There is mounting evidence that the immune system, and more specifically the complement system, play a role in glaucoma.[13-16] About 20 proteins, which are present in typical human serum, make up the complement system. Among them one is serum complement C3. The ability of these proteins to supplement the effects of other immune system components is referred as complement. In other words, the complement system is a component of the innate immune response. Complements' primary effects include lysing cells including bacteria, allografts, and tumour cells, producing inflammatory mediators that draw neutrophils, and opsonizing (improving phagocytosis). The liver produces complement proteins. A C3 deficit is thought to be the root cause of severe, recurrent pyogenic sinus and respiratory tract infections, according to Warren Levinson's study in Medical Microbiology and Immunology.[17] Genetics of complement component C3 has a significant role in the development of age-related macular degeneration.[18] In glaucoma and animal models with retinal degeneration, the complement system component activation has been reported.[19] According to several experimental studies, the level of C3 and C3 split products was aberrant in the aqueous humor of patients with exfoliation glaucoma[20] and the retinas of mice used as glaucoma models.[21] Recent research suggests that C3 protects against early glaucomatous damage and that C3 deficiency dramatically increased the number of eyes with nerve damage and retinal ganglion cells loss at an early time point following IOP elevation. According to this research, C3 may be linked to glaucomatous neurodegeneration.[22]

POAG is a neurodegenerative illness that may include intricate connections between retinal ganglionic cells (RGSc), glial cells, and the immune system.[23] The innate immune system's complement system is a crucial component. According to animal models of glaucoma[24, 25], complement activation and the control of it by ocular complement regulatory proteins play a role in the disease's pathogenesis. Previous research has demonstrated that low serum C3 levels can cause RGC destruction in the early stages of glaucoma, and that POAG patients are more likely to experience severe POAG when their C3 levels are low. The most likely causes of a low level of C3 in POAG patients may be structural abnormality or C3 gene mutations. Complement-mediated apoptosis is crucial in the loss of RGCs in chronic ocular hypertension.[24] Additionally, it was found that suppression of the classical complement cascade can stop early RGC degradation in glaucoma.[25] Due to the subsequent activation of complement components 5 to 9 and the development of the membrane attack complex, C3 activation in glaucoma patients can also contribute to the degeneration of RGCs. Ocular auto antibodies have been identified in glaucoma patients' serum[26, 27] and retinas[28], according to a number of investigations.

Complex IgG auto-antibody levels have been reported to be greater in glaucoma patients'

serum than in healthy individuals.[26] Similarly, it was shown that glaucoma patients had retinal autoantibodies that were at least twice as high as those of healthy individuals. IgG can activate the complement system and the activation of autoantibodies in glaucoma may be a factor in serum C3 depletion. Patients with a C3 deficiency may be more likely to experience recurrent infections; as a result, POAG patients may be more prone to infection, which could lead to a further drop in C3 levels. Infected POAG patients will see faster RGC deterioration. As a result, there is a vicious cycle of activation, consumption, infection, and activation. These indicate a potential link between POAG at disease onset and a low serum C3 level.[29]

Materials and method

A cross sectional comparative study was carried out in Bangabandhu Sheikh Mujib Medical University, Shahbag, Dhaka, from January 2022 to August 2023. Patients attended in the glaucoma clinic and out patient department of Department of Ophthalmology, BSMMU, were the study population.

There were two groups; Study group (POAG): Patients who came to Glaucoma clinic, age between 40-60 years, of all sexes, IOP >21mm of Hg or <21mm of Hg having definite glaucomatous optic nerve head damage, receiving

anti glaucoma medications, glaucomatous optic nerve head changes and visual field defect, open irido-corneal angle assessed with gonioscopy and normal anterior chamber depth were diagnosed as POAG and considered as study group. Comparison group: Patients without POAG were selected from Ophthalmology Out Patient Department .Patients who have secondary glaucoma, congenital glaucoma, neovascular glaucoma, angle closure glaucoma, diabetic retinopathy, macular generation were excluded from the study. As well as patients having autoimmune diseases like SLE, kidney disease like membranoproliferative glamerolonephritis, serum sickness, acute glomerulonephritis, cancer, metabolic syndrome & acute infectious diseases were excluded from the study. The consecutive sampling technique was applied to collect sample from the study population. Because liver is the source of serum complement C3, serum SGPT and complete blood count were performed in the same session along with serum complement C3 in order to rule out altered liver function and any pre-existing infection. Complete clinical evaluation including history, clinical examination, relevant ocular examinations, fundus examination, some special ocular examination like IOP, visual field analysis, gonioscopy were done in Ophthalmology department, Bangabandhu Sheikh Mujib University. Ocular examinations includes: BCVA, Pupillary light reaction, RAPD, Hirchberg reflex, Ocular motility, Color vision test, Slit lamp examination of anterior segment & fundus examination was done with the help of + 78 D condensing lens. Intraocular pressure was measured by Goldmann applanation tonometer. Normal value is 11-21 mm of Hg. Angle assessment by gonioscopy (Sussman four mirror diagnostic gonioscope) was done under low ambient illumination. Serum complement C3 level, SGPT, CBC, Color fundus photograph (CFP)- B/ E, Humphrey visual field analysis 24-2 B/E, Optical Coherence Tomography ONH and RNFL.

Measurement of serum complement C3 level: A peripheral blood sample was collected from each participants at baseline and tested for complement C3 level. After collection of blood, it was sent into Microbiology department BSMMU within 1 hour. Ensure complete clot formation before centrifugation. Then it was centrifuged at room temperature at 2000 rpm for 10minutes. After that serum and blood clot was separated and 20 times dilution of the serum was done. Then serum was placed in SIEMENS BN Pro Spec system and result was obtained digitally by Nephelometric method or the sample was preserved under-4^0

temperature for any future measurement. The principal of immunonephelometric method is- Protein contained in human body from immune complexes with specific antibodies, which scatter a beam of light passed through the sample. The intensity of the scattered light is proportional to the concentration of the relevant protein of the sample. Then compared with a standard of known concentration.The reference ranges of serum complement C3 was given .9-1.8 g/L

Measurement of Serum SGPT level: After blood collection, the sample also sent into Biochemistry department BSMMU for measurement of SGPT. Following the separation of the serum and the clot, the serum was added to the SIEMENS Atellica solution for SGPT testing. The reference ranges in case of male <45 IU/L and female <34 IU/L. Measurement of CBC: The blood sample was sent into Laboratory medicine department BSMMU and CBC is done by sysmex automated heamatological analyzer machine.

Statistical Analysis

The demographic information, relevant history, examination findings, investigation report, of all the study subjects was recorded in the data collection sheet. After compilation, the data was presented in the form of tables, figures and graphs, as necessary. Statistical analysis of the results was done by using computer-based software, SPSS version 26 (IBM Corp. Released 2019.IBM SPSS statistics for windows, version 26.0. Armonk, NY: IBM corp). Descriptive statistics were expressed as Mean, Standard deviation, frequency, percentage. Un-paired t-test was used to compare two means of two groups, chi-square test was used to measure the association between two qualitative variables, ANOVA test was used to compare three means of three groups. The association laboratory parameter and ocular parameter of POAG had analyzed using multiple logistic regression analysis. 'P' value of 0.05 or less was considered as significant.

Table I: Distribution of the study patients by demographic profile (n=40)

Demographic profile	Group I (n=40)		Group II (n=40)		P value
	n	%	n	%	
Age (in year)					
40-50	16	40.0	21	52.5	
51-60	24	60.0	19	47.5	
Mean ± SD	52.75±6.42		51.18±7.44		$^{a}0.315^{ns}$
Range (min, max)	40,60		40,60		
Gender					
Female	15	37.5	19	47.5	$^{b}0.365^{ns}$
Male	25	62.5	21	52.5	

ns= not significant

^{a}p value reached from Unpaired t-test ^{b}p value reached from Chi-square test Group I= POAG group

Group II= Comparison group

Table I shows the distribution of the study patients by demographic profile. It was observed that almost two third (60.0%) patients belonged to age 51-60 years in group I and 19(47.5%) in group II. The mean age was 52.75±6.42 years in group I and 51.18±7.44 years in group II. Almost two third (62.5%) patients were male in group I and 21(52.5%) in group II. The difference was statistically not significant (p>0.05) between group.

Table II: Distribution of the study patients by Serum complement C3 level (n=40)

Serum Complement (C3 level (g/L)	Group I (n=40)		Group II (n=40)		P value
	n	%	n	%	
Decreased C3 level (<0.9 g/L)	3	7.5	0	0.0	
Normal C3 level (0.9 – 1.8 g/L)	37	92.5	40	100.0	
Mean ± SD	1.02±0.12		1.09±0.15		0.026^{S}
Range (min, max)	(0.8, 1.38)		(1.00, 1.50)		

p value reached from Unpaired t-test

The mean serum complement C3 level was 1.02±0.12 (g/l) in group I and 1.09±0.15 (g/l) in group II. The difference was statistically significant (p<0.05) between group.

Figure 1 shows the distribution of the study patients having Primary open angle glaucoma (POAG) by severity of the glaucomatous damage graded according to mean deviation in visual field. MD <-6dB is considered as mild damage, MD between -6 to -12dB is consider as moderate damage and MD >-12 dB is considered as severe damage. Most of the patients that is 47.5% had severe glaucomatous damage, 27.5% mild and 25.0% moderate glaucomatous damage.

Table III: Comparison between Serum complement C3 status of different grading of primary open Angel glaucoma (POAG) based on severity of the glaucomatous damage graded according to mean deviation in visual field (MD) (n=40)

Serum Complement (C3 level (g/l)	POAG grading Mild (n=11)		Moderate (n=10)		Severe (n=19)		P value
	n	%	n	%	n	%	
Decreased C3 level (<0.9 g/L)	0	0.0	1	10.0	2	10.5	
Normal C3 level (0.9 – 1.8 g/L)	10	100.0	9	90.0	18	89.5	
Mean ± SD	1.07±0.12		1.03±0.14		0.99±0.99		0.311^{ns}
Range (min, max)	(0.92, 1.38)		(0.83, 1.27)		(0.80, 1.23)		

ns= not significant

p value reached from ANOVA test

Table VI shows the mean value of Serum complement C3 level of the patients with different grading of primary open Angel glaucoma (POAG) based on severity of the glaucomatous damage graded according to mean deviation in visual field (MD). The mean serum complement C3 level was 1.07±0.12 (g/l) in mild glaucomatous damage,

1.03±0.14 (g/l) in moderate glaucomatous damage and 0.99±0.99 (g/l) in sever glaucomatous damage II. The mean Serum complement C3 was not statistically significant (p>0.05) in different grading of severity of POAG patients.

Table IV: Multiple linear regressions for association between C3 levels and ocular parameters in patients with POAG

Variable	B	p value	95% CI
Right			
IOP	0.001	0.841	0.001-0.009
VCDR	0.102	0.573	0. 263-0.467
MD	0.003	0.131	0.001-0.007
Left			
IOP	0.002	0.628	0.007-0.011
VCDR	0.001	0.980	0.141-333
MD	0.004	0.074	0.001-0.009

Table IV contains multiple linear regressions for association between C3 levels and ocular parameters in patients with POAG adjusting IOP, VCDR and MD of visual field, which shows there is no association of severity of POAG with serum complement C3 level.

Discussions

This cross sectional comparative study was carried out with an aim to measure the level of serum complement C3 level in Primary open angle glaucoma (POAG) and without POAG and also to compare the serum complement C3 level in two groups; as well as to see the demographic variables of those patients. A total of 80 patients attending in the Department of Ophthalmology of Bangabandhu Sheikh Mujib Medical University, Dhaka, during January, 2022 to August, 2023, were included in this study. Among them, 40 patients with POAG were considered as group I and the rest 40 normal control as group II.

In this current study it was observed that 60.0% patients belonged to age 51-60 years in group I and 47.5% in group II. The mean age was 52.75±6.42 years and 51.18±7.44 years in group I and group II respectively. The mean difference was statistically not significant (p>0.05) between groups. The mean age was 75.7 ± 7.5 years in POAG group and 74.0±5.1 years in control group[12], which is higher than the present study but there was no significant

differences in age between groups. In another study observed the mean age was 47.08±17.39 years in POAG group and 47.65±17.26 years in control group. There was also no statistical difference in mean age between the POAG and control subjects, which is lesser than the present study.[30] The higher and lesser mean age and age ranges obtained by the above authors maybe due to higher age group and lower age group population were included in those studies, geographical variations, racial, ethnic differences and genetic causes may have significant influence on their study subjects.

In this particular study it was seen that 62.5% patients were male in group I and 52.5% in group II. The difference was statistically not significant (p>0.05) between groups. Similarly, it was also showed that male predominant in their study and no statistical difference in sex difference between the POAG and control subjects.[30] Similar observations also observed by another study.[12]

In this study, POAG subjects were divided into male (age between 40-50 years, 51-60 years), female (age between 40-50 years, 51-60 Years). Which shows there is significant low level of serum C3 in male participants in group I than then group II(p<0.05) and male patients age between 51-60 years there was significant low level of serum C3 in POAG patients than comparison group(p value<0.05). In a similar study, the mean serum C3 level was significantly lower [female (93.05 ± 19.07 vs.118.25 ± 24.86 mg/dL), male (96.61 ± 17.13 vs.114.48 ± 20.87 mg/dL)] (p value<0.001) [30]both which closely resembles this study.

But in case of female participants female patients, mean serum C3 in group I is 1.03±0.12 and in group II 1.09±0.13, which is not statistically significant between groups (p value 0.111), between age 40-50 years in group I it was1.08±0.1 and in group II it was 1.06±0.11, which is not statistically significant between groups (p value 0.700). The mean serum C3 between age 51-60 years in group I is 1.03±0.13 and in group II 1.09±0.15. Which is not statistically significant between groups (p value 0.259), which does not align with[30] may be because higher number of male patients were included in this study.

In this current study it was observed that the mean IOP in right eye was 15.83±4.78 mm of Hg in group I and 13.73±2.73 mm in Hg in group II. The mean IOP in left eye was 16.6±4.02 mm in Hg in group I and 13.5±2.75 mm in Hg in group II. The mean IOP in right eye and left eye was significantly (p<0.05) higher in group I.[12] Noted that both stable and progressive POAG patients used ocular medication to lower IOP, with the progressive group tending to use more. While treated IOP didn't significantly differ (p>0.05) from controls in both glaucoma groups, progressive POAG patients had somewhat higher IOP. Correspondingly, raised IOP triggers complement system activation in retinal animal models.[31-33] Recent research indicates immune cell infiltration in AH[34] and retina[35] due to elevated IOP. While heightened intraocular pressure (IOP) stands as a prominent risk element for glaucoma[7], the specific mechanisms through which elevated IOP contributes to the degeneration of retinal optic ganglion cells (RGCs) remain to be elucidated.[36] Over time, a gradual accumulation of evidence has suggested that elevated IOP triggers alterations within the complement system pathways not only in non-neuronal cells like astrocytes and microglia/monocytes[37-38], but also in RGCs themselves.[22]

This present study showed that decreased C3 level was found 7.5% in group I and not found in group II. The mean serum complement C3 level was 1.02±0.12 (g/l) in group I and 1.09±0.15 (g/l) in group II. The mean serum complement C3 level was significantly (p<0.05) lower in group I than comparative group. As serum C3 level may be abnormal in altered liver function and infectious conditions, that's why serum SGPT and CBC were done along with serum complement C3. Participants who had abnormal SGPT and CBC results, were excluded from the study. It was found that glaucoma

patients displayed a tendency toward elevated concentrations of complement factor C3a in aqueous humor (AH) with no statistically significant difference ($p>0.05$).[39] The estimated mean concentration of C3a was 15 ng/ml in individuals with glaucoma, in contrast to 8.5 ng/ml in cataract control subjects. Another investigation conducted revealed a noteworthy reduction ($p<0.05$) in complement C3 levels in the serum of glaucoma patients, and this reduction correlated with the severity of the disease.[30]Similarly identified a significant ($p<0.05$) and inverse relationship between serum complement C3 levels and disease severity. Essentially, patients with lower serum complement C3 concentrations exhibited more severe forms of the disease[12].

Primary open-angle glaucoma (POAG) stands as a prominent neurodegenerative ailment characterized by intricate interplays involving retinal ganglion cells (RGCs), glial cells, and the immune system.[23] The complement system, a vital facet of the innate immune defense, remains enigmatic regarding its impact on systemic C3 levels in POAG patients due to potential ocular tissue complement activation, deposition and consumption. A study revealing markedly diminished mean serum C3 levels in POAG subjects in contrast to controls, with the most pronounced decrease noted in severe POAG cases, followed by moderate and mild cases.[30] These findings suggest a potential disarray in the complement system within POAG patients. Subsequent research by[40, 24, 20] also documented aberrant C3 levels and activation products in glaucomatous eyes of both animals and humans. Correspondingly[30, 32, 40] highlighted significantly lower plasma C3 levels in primary angle-closure glaucoma (PACG) subjects compared to controls. A comprehensive grasp of peripheral C3 levels and its potential roles in POAG management emerges as clinically beneficial. This cascade of evidence poses fundamental inquiries: What underpins the reduced serum C3 concentrations in POAG patients, and what roles does C3 play in the POAG context. It was proposed that complement-mediated apoptosis substantially contributes to chronic ocular hypertension-induced RGC loss in glaucoma models.[32] It was demonstrated that quelling the classical complement cascade pathway can forestall early RGC degeneration.[25] Consequently, C3 activation might culminate in RGC degeneration via downstream complement component activation (C5-C9) and membrane attack complex formation. The involvement of IgG-triggered complement activation[41] coupled with autoantibody-driven C3 depletion could intensify the predicament. Diminished C3 levels could augment infection vulnerability, further diminishing C3, thereby creating a self-perpetuating cycle of activation, consumption, infection, and renewed activation. These findings collectively underscore C3's role in POAG pathomechanisms, albeit the specific facets of this involvement remain elusive.

The grading of severity among patients with primary open-angle glaucoma (POAG) involves a systematic assessment that aims to categorize the extent of disease progression and its impact on visual function. This process enables clinicians to tailor appropriate treatment strategies and monitor the condition's advancement over time. Various factors contribute to the grading, including intraocular pressure (IOP) measurements, optic nerve head appearance, visual field testing results, and the presence of other risk factors. Typically, severity is categorized into different stages, such as mild, moderate, and severe, based on the extent of optic nerve damage, visual field loss, and the degree of IOP control. Grading systems provide valuable insights for both medical decision-making and patient communication, allowing healthcare professionals to develop personalized management plans that address the specific needs of individuals at different stages of POAG. Regarding the grading of severity of POAG patients in this present study it was observed that most (47.5%) of the patients had severe glaucomatous damage followed by 27.5% mild and 25.0% had moderate

glaucomatous damage.

Regarding the association between serum complement C3 status of different grading of primary open angle glaucoma (POAG) based on severity of the glaucomatous damage graded according to mean deviation in visual field (MD) in this present study it was observed that decreased C3 level was not observed in mild glaucomatous damage, 10.0% in moderate and 10.5% in severe glaucomatous damage. The mean serum complement C3 level was 1.07±0.12 (g/l) in mild glaucomatous damage, 1.03±0.14 (g/l) in moderate glaucomatous damage and 0.99±0.99 (g/l) in severe glaucomatous damage. Serum complement C3 was not significantly ($p>0.05$) associated with grading of severity of POAG. In the research conducted by Li et al. (2018), it was observed that the average serum C3 levels exhibited a descending trend across different severity groups of primary open-angle glaucoma (POAG). The severe POAG group had the lowest mean level of serum complement C3 level is 85.18 ± 19.62 mg/dL, followed by the moderate POAG group is 96.62 ± 12.63 mg/dL and the mild POAG group is 110.44 ± 14.89 mg/dL($p<0.001$). There were also non significant association in multiple linear regression analysis adjusting IOP, VCDR and MD in POAG group. Logistic regression analyses revealed that serum C3 levels were associated with severity of POAG. These findings does not align with the current study's observations. The reason behind this disparity could be higher population were included in the other studies. The outcomes of this investigation indicated an apparent disruption in the complement system among individuals with POAG. The role of complement C3 in POAG has been approached from two distinct perspectives. One viewpoint asserts that C3 has a safeguarding effect against early glaucomatous damage, as established by the work of.[22] Conversely, an alternative stance posits that the activation of C3 might trigger apoptosis in retinal ganglion cells (RGCs) through downstream activation of complement components 5 to 9, culminating in the formation of the membrane attack complex. This notion is supported by research from.[25, 42, 43] In a study by[22], it was determined that early responses involving complement C3 and epidermal growth factor receptor signaling by astrocytes are advantageous, with C3 deficiency notably increasing the incidence of RGC damage in DBA/2J eyes. Furthermore, the protective role of C3 in a different neurodegenerative disorder was also highlighted.[44] Investigation revealed a reduction in serum C3 levels, correlating inversely with the severity of POAG. Given its vital role in humoral immunity[30], deficiencies in C3 could potentially render patients more susceptible to frequent infections, as suggested by previous studies.[45] Multiple studies have reported elevated counts of oral bacteria[46], and there is a statistically significant association between glaucoma and Helicobacter pylori infections.[47, 48] This prompts the hypothesis that diminished serum C3 concentrations might trigger early-stage RGC damage in individuals with POAG. Consequently, a lower C3 level could elevate the risk of severe POAG for these patients. This viewpoint suggests that C3 gene mutations or structural abnormalities could plausibly underlie the reduced C3 levels observed in individuals with POAG. However, there is a dearth of comprehensive data available to definitively establish the link between C3 gene mutations, structural abnormalities, and the susceptibility to POAG.

Conclusion

This cross sectional comparative study was undertaken to assess the association of serum complement C3 level with primary open angle glaucoma and it's severity It included 40 POAG patients as study group and 40 patients without POAG as comparison group. Demographic as of the participants, clinical findings of POAG and their serum complement C3 level were compared between the two groups. Within the POAG group, the mean serum level of complement C3 was significantly diminished than the comparison group. These findings suggest a possible involvement of serum complement C3 in the pathogenesis of primary

open angle glaucoma. However, there was no correlation between serum complement C3 levels and the severity of POAG. Visual field indices were used to

determine the severity of POAG. However, other non glaucomatous factors may also contribute to the visual field. Duration of POAG and effects of anti glaucoma medication were not considered in this study. Although standard inclusion criteria and standard recruitment method were used, curtained biases of this study should be considered.

Recommendations

Longitudinal studies are necessary to evaluate the topographic and temporal relationship between changes in serum C3 levels and glaucomatous changes in standard structural and functional measures in healthy participants, and those with POAG. Future research can be done to determine the relationship between the duration of POAG and the complement C3 level. Further studies can also be undertaken by including large number of patients. Serum IgG level auto antibody tests from POAG patients can be done to further explore the causal association between serum complement C3 and POAG. Further research can be done on the levels of C3 and C3a in the aqueous humor of POAG patients. Our findings further demonstrate the significance of the complement system in POAG, suggesting that complement-modulating therapies may provide a novel strategy for reducing RGC damage.

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All authors contributed to the article's drafting, revision, drafting and finalizing to fulfill authorship criteria. Funding

This study had received thesis grant from BSMMU grant committee. Conflict of interest

The author has no conflict of interest to declare. Ethical Approval

The institutional review board has approved this research protocol, on 2nd july 2022 before starting this study.

References

1. Jonas JB, Aung T, Bourne RR, Bron AM, Ritch R, Panda-Jonas S. Glaucoma–Authors' reply. The Lancet. 2018 Feb 24;391(10122):740.
2. Tham YC, Li X, Wong TY, Quigley HA, Aung T, Cheng CY. Global prevalence of glaucoma and projections of glaucoma burden through 2040: a systematic review and meta-analysis. Ophthalmology. 2014 Nov 1;121(11):2081-90.
3. Bourne RR, Stevens GA, White RA, Smith JL, Flaxman SR, Price H, Jonas JB, Keeffe J, Leasher J, Naidoo K, Pesudovs K. Causes of vision loss worldwide, 1990–2010: a systematic analysis. The lancet global health. 2013 Dec 1;1(6):e339-49.
4. Gupta N, Weinreb RN. New definitions of glaucoma. Current opinion in ophthalmology. 1997 Apr 1;8(2):38-41.
5. Ha Q. The number of people with glaucoma worldwide in 2010 and 2020. Br j ophthalmol. 2006;90:262-7.
6. Rahman MM, Rahman N, Foster PJ, Haque Z, Zaman AU, Dineen B, Johnson GJ. The prevalence of glaucoma in Bangladesh: a population based survey in Dhaka division. British journal of ophthalmology. 2004 Dec 1;88(12):1493-7.
7. Leske MC, Wu SY, Hennis A, Honkanen R, Nemesure B, BESs Study Group. Risk factors for incident open-angle glaucoma: the Barbados Eye Studies. Ophthalmology. 2008 Jan 1;115(1):85-93.
8. La Croix N, Wayne J. Ophthalmic Lasers for the Treatment of Glaucoma. Laser Surgery in Veterinary Medicine. 2019 Jun 4:129-40.
9. Janssen SF, Gorgels TG, Ramdas WD, Klaver CC, van Duijn CM, Jansonius NM, Bergen AA. The vast complexity of primary open angle glaucoma: disease genes, risks, molecular mechanisms and pathobiology. Progress in retinal and eye research. 2013 Nov 1;37:31-67.
10. Kapetanakis VV, Chan MP, Foster PJ, Cook DG, Owen CG, Rudnicka AR. Global variations and time trends in the prevalence of primary open angle glaucoma (POAG): a systematic review and meta-analysis. British Journal of Ophthalmology. 2016 Jan 1;100(1):86-93.
11. Weinreb RN. IOP and the risk of progression to glaucoma. Graefe's Archive for Clinical and Experimental Ophthalmology. 2005 Jun;243:511-2.
12. Hubens WH, Beckers HJ, Gorgels TG, Webers CA.

Increased ratios of complement factors C3a to C3 in aqueous humor and serum mark glaucoma progression. Experimental Eye Research. 2021 Mar 1;204:108460.

13. Adav SS, Wei J, Terence Y, Ang BC, Yip LW, Sze SK. Proteomic analysis of aqueous humor from primary open angle glaucoma patients on drug treatment revealed altered complement activation cascade. Journal of proteome research. 2018 Jun 14;17(7):2499-510.
14. Beutgen VM, Schmelter C, Pfeiffer N, Grus FH. Autoantigens in the trabecular meshwork and glaucoma specific alterations in the natural autoantibody repertoire. Clinical & translational immunology. 2020;9(3):e01101.
15. Clark SJ, Bishop PN. The eye as a complement dysregulation hotspot. InSeminars in immunopathology 2018 Jan (Vol. 40, No. 1, pp. 65-74). Berlin/Heidelberg: Springer Berlin Heidelberg.
16. Tsai T, Reinehr S, Maliha AM, Joachim SC. Immune mediated degeneration and possible protection in glaucoma. Frontiers in neuroscience. 2019 Sep 2;13:931.
17. Warren L. Review of medical microbiology and immunology.
18. Yates JR, Sepp T, Matharu BK, Khan JC, Thurlby DA, Shahid H, Clayton DG, Hayward C, Morgan J, Wright AF, Armbrecht AM. Complement C3 variant and the risk of age-related macular degeneration. New England Journal of Medicine. 2007 Aug 9;357(6):553-61.
19. Rutar M, Natoli R, Kozulin P, Valter K, Gatenby P, Provis JM. Analysis of complement expression in light-induced retinal degeneration: synthesis and deposition of C3 by microglia/macrophages is associated with focal photoreceptor degeneration. Investigative ophthalmology & visual science. 2011 Jul 1;52(8):5347-58.
20. Doudevski I, Rostagno A, Cowman M, Liebmann J, Ritch R, Ghiso J. Clusterin and complement activation in exfoliation glaucoma. Investigative ophthalmology & visual science. 2014 Apr 1;55(4):2491-9.
21. Ahmed F, Brown KM, Stephan DA, Morrison JC, Johnson EC, Tomarev SI. Microarray analysis of changes in mRNA levels in the rat retina after experimental elevation of intraocular pressure. Investigative ophthalmology & visual science. 2004 Apr 1;45(4):1247-58.
22. Harder JM, Braine CE, Williams PA, Zhu X, MacNicoll KH, Sousa GL, Buchanan RA, Smith RS, Libby RT, Howell GR, John SW. Early immune responses are independent of RGC dysfunction in glaucoma with complement component C3 being protective. Proceedings of the National Academy of Sciences. 2017 May 9;114(19):E3839-48.
23. Burda JE, Sofroniew MV. Reactive gliosis and the multicellular response to CNS damage and disease. Neuron. 2014 Jan 22;81(2):229-48.
24. Jha P, Banda H, Tytarenko R, Bora PS, Bora NS. Complement mediated apoptosis leads to the loss of retinal ganglion cells in animal model of glaucoma. Molecular immunology. 2011 Sep 1;48(15-16):2151-8.
25. Williams PA, Tribble JR, Pepper KW, Cross SD, Morgan BP, Morgan JE, John SW, Howell GR. Inhibition of the classical pathway of the complement cascade prevents early dendritic and synaptic degeneration in glaucoma. Molecular neurodegeneration. 2016 Dec;11:1-3.
26. Joachim SC, Reichelt J, Berneiser S, Pfeiffer N, Grus FH. Sera of glaucoma patients show autoantibodies against myelin basic protein and complex autoantibody profiles against human optic nerve antigens. Graefe's Archive for Clinical and Experimental Ophthalmology. 2008 Apr;246:573-80.
27. Wax MB, Barrett DA, Pestronk A. Increased incidence of paraproteinemia and autoantibodies in patients with normal-pressure glaucoma. American journal of ophthalmology. 1994 May 1;117(5):561-8.
28. Gramlich OW, Beck S, von Thun und Hohenstein-Blaul N, Boehm N, Ziegler A, Vetter JM, Pfeiffer N, Grus FH. Enhanced insight into the autoimmune component of glaucoma: IgG autoantibody accumulation and pro-inflammatory conditions in human glaucomatous retina. PloS one. 2013 Feb 25;8(2):e57557.
29. Ehrnthaller C, Ignatius A, Gebhard F, Huber-Lang M. New insights of an old defense system: structure, function, and clinical relevance of the complement system. Molecular medicine. 2011 Mar;17(3):317-29.
30. Li S, Li D, Shao M, Cao W, Sun X. Decreased serum levels of complement C3 reflect complement system dysregulation in patients with primary open-angle glaucoma: results from a pilot study. Journal of Glaucoma. 2018 Sep 1;27(9):761-8.
31. Becker S, Reinehr S, Burkhard Dick H, Joachim SC. Complement activation after induction of ocular hypertension in an animal model. Der Ophthalmologe. 2015 Jan;112:41-8.

32. Jha P, Banda H, Tytarenko R, Bora PS, Bora NS. Complement mediated apoptosis leads to the loss of retinal ganglion cells in animal model of glaucoma. Molecular immunology. 2011 Sep 1;48(15-16):2151-8.

33. Kuehn MH, Kim CY, Ostojic J, Bellin M, Alward WL, Stone EM, Sakaguchi DS, Grozdanic SD, Kwon YH. Retinal synthesis and deposition of complement components induced by ocular hypertension. Experimental eye research. 2006 Sep 1;83(3):620-8.

34. Nair AP, Sahu GR, Tejwani S, Ghosh A, Sethu S. Increased infiltration of immune cell subsets and altered soluble factor profile in aqueous humor of glaucoma patients correlates with disease severity. Investigative Ophthalmology & Visual Science. 2019 Jul 22;60(9):674-.

35. Chen J, Jiang C, Huang Q, Lin X, Wu W, Li J. Detection of plasma complement and immune globulin in different sorts of glaucoma. European Journal of Ophthalmology. 2022 Sep;32(5):2907-12.

36. La Croix N, Wayne J. Ophthalmic Lasers for the Treatment of Glaucoma. Laser Surgery in Veterinary Medicine. 2019 Jun 4:129-40.

37. Howell GR, Soto I, Zhu X, Ryan M, Macalinao DG, Sousa GL, Caddle LB, MacNicoll KH, Barbay JM, Porciatti V, Anderson MG. Radiation treatment inhibits monocyte entry into the optic nerve head and prevents neuronal damage in a mouse model of glaucoma. The Journal of clinical investigation. 2012 Apr 2;122(4):1246-61.

38. Tezel G, Yang X, Luo C, Peng Y, Sun SL, Sun D. Mechanisms of immune system activation in glaucoma: oxidative stress-stimulated antigen presentation by the retina and optic nerve head glia. Investigative ophthalmology & visual science. 2007 Feb 1;48(2):705-14.

39. Gallenkamp J, Spanier G, Wörle E, Englbrecht M, Kirschfink M, Greslechner R, Braun R, Schäfer N, Bauer RJ, Pauly D. A novel multiplex detection array revealed systemic complement activation in oral squamous cell carcinoma. Oncotarget. 2018 Jan 1;9(3):3001.

40. Tezel G, Yang X, Luo C, Kain AD, Powell DW, Kuehn MH, Kaplan HJ. Oxidative stress and the regulation of complement activation in human glaucoma. Investigative ophthalmology & visual science. 2010 Oct 1;51(10):5071-82.

41. Ehrnthaller C, Ignatius A, Gebhard F, Huber-Lang M. New insights of an old defense system: structure, function, and clinical relevance of the complement system. Molecular medicine. 2011 Mar;17(3):317-29.

42. Kumari R, Astafurov K, Genis A, Danias J. Differential effects of C1qa ablation on glaucomatous damage in two sexes in DBA/2NNia mice. PloS one. 2015 Nov 6;10(11):e0142199.

43. Howell, G.R., Macalinao, D.G., Sousa, G.L., Walden, M., Soto, I., Kneeland, S.C., Barbay, J.M., King, B.L., Marchant, J.K., Hibbs, M. and Stevens, B., 2011. Molecular clustering identifies complement and endothelin induction as early events in a mouse model of glaucoma. The Journal of clinical investigation, 121(4), pp.1429-1444.

44. Maier M, Peng Y, Jiang L, Seabrook TJ, Carroll MC, Lemere CA. Complement C3 deficiency leads to accelerated amyloid â plaque deposition and neurodegeneration and modulation of the microglia/macrophage phenotype in amyloid precursor protein transgenic mice. Journal of Neuroscience. 2008 Jun 18;28(25):6333-41.

45. Ram S, Lewis LA, Rice PA. Infections of people with complement deficiencies and patients who have undergone splenectomy. Clinical microbiology reviews. 2010 Oct;23(4):740-80.

46. Astafurov K, Elhawy E, Ren L, Dong CQ, Igboin C, Hyman L, Griffen A, Mittag T, Danias J. Oral microbiome link to neurodegeneration in glaucoma. PloS one. 2014 Sep 2;9(9):e104416.

47. Kim JM, Kim SH, Park KH, Han SY, Shim HS. Investigation of the association between Helicobacter pylori infection and normal tension glaucoma. Investigative Ophthalmology & Visual Science. 2011 Feb 1;52(2):665-8.

48. Zeng, J., Liu, H., Liu, X. and Ding, C., 2015. The relationship between Helicobacter pylori infection and open-angle glaucoma: a meta-analysis. Investigative Ophthalmology & Visual Science, 56(9), pp.5238-5245.

Original Article

Demographic and clinical status of patients with refractory glaucoma who underwent Ahmed valve surgery

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Abstract

Background: Globally, glaucoma is the most common irreversible cause of blindness. The Ahmed glaucoma valve (AGV) is a popular glaucoma drainage implant used for the control of intraocular pressure in patients with glaucoma.It prevents postoperative hypotony, allowing for AH drainage when IOP is in the range of 8–12 mmHg. Preconception on the demographic, especially clinical features ofrefractory glaucoma cases is very important for the surgeons in managing such patients by Ahmed valve surgery or even by any other methods.

Aim of the study: This studyaimed to assess the demographic and clinical status of patients with refractory glaucoma who underwent Ahmed valve surgery.

Methods: This cross-sectional observational study was conducted at the Ispahani Islamia Eye Institute and Hospital, Dhaka, Bangladesh from March 2021 to July 2022. In total 35 patients (Single eye)who underwent Ahmed valve implantation for refractory glaucoma to the maximum tolerated therapy and/or prior surgery failure since January 2016 were selected as the study subjects. A convenient purposive sampling technic was used in sample selection. All data regarding the demographic status, type of glaucoma, prior ocular surgeries, intraocular pressure (IOP) value and the number of drops needed before and after surgery, during several follow-up visits and postoperative complications were recorded.All data were processed, analyzed and disseminated by using the MS Office program.

Results: In this study, 35 eyes of 35 patients were analyzed; the male-female ratio of the participants was 2:1. The majority of our patients (40%) were from the 19-35 years age group. The majority of the participants (54%) were with aphakic glaucoma; 14% and 11% were with neovascular glaucoma (NVG) and proliferative diabetic retinopathy with glaucoma respectively. In most of the cases (51%), the visual acuity was ＜6/60, in 34% of cases the range was 6/36-6/12 and in 14% of cases the range of VA was 6/9-6/6. At the baseline, the mean intraocular pressure (IOP) of our participants was 37.72%.

Conclusion: The frequency of refectory glaucoma among the younger population (At 19-35 years) is alarming. Younger male individuals are mainly prone to refectory glaucoma. The most frequent type of glaucoma among such cases is aphakic glaucoma which contributed more than fifty percent.

Keywords: Ahmed glaucoma valve, AGV, Refractory glaucoma, Demographic, Clinical status

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Introduction

The Ahmed glaucoma valve (AGV), a GDI device, was permitted by the FDA in 1993 for use in glaucoma patients with uncontrolled IOP[1]. Worldwide, glaucoma is one of the leading causes of irreversible blindness, which can affect up to 111 million people globally by the year 2040[2,3]. Refractory glaucoma is defined as uncontrolled intraocular pressure (IOP) withevidence of optic nerve and/or visual field deterioration despite maximally tolerated anti-glaucoma medications, previouslyfailednon-seton surgicaltreatment or a combination of surgery and medicines or a high risk of failure of trabeculectomy[4]. The cases that do not respond to any conventional glaucoma treatment, maybe treated with Ahmed glaucoma valve implants literature[5,6]. Ishida and Netland[7] reported that African American race had a higher risk of failure of AGV. For failed AGV, previous glaucoma surgery was reported as a risk factor. The outcomes of Ahmed glaucoma valve implantation in the managementof various forms of refractory glaucoma are variable. To our knowledge, very few studies have been conducted on the adult Arab population with

refractory glaucoma[8,9]. Al-Shahwan[8] reported a single case of tube erosion following Ahmed glaucoma valve implantation. Eid[9] reporteda positive role of intravitreal bevacizumab in improving the success of Ahmed glaucoma valve (AGV). Shah[10] found 12% absolute and 78% relative success rates of intraocular pressure (IOP) control following Ahmed glaucoma valve surgery in Oman. Ahmed glaucoma valve implantation was reserved for glaucoma cases low controlled after one or other filtration procedures, recent evidence has encouraged its use as a primary surgery in refractory glaucoma like secondary to neovascular glaucoma, penetrating keratoplasty, pars plana vitrectomy (PPV) and uveitis[11]. The objective of this study was to assess the demographic and clinical status of patients with refractory glaucoma who underwent Ahmed valve surgery.

Methodology

This cross-sectional observational was performed at the Ispahani Islamia Eye Institute and Hospital, Dhaka, Bangladesh from March 2021 to July 2022. In total 35 patients (Single eye) who underwent Ahmed valve implantation for glaucoma refractory to maximum tolerated therapy and/or prior surgery failure since January 2016 were enrolled in this study as the study subjects. The study was approved by the ethical committee of the mentioned hospital. Properly written consent was taken from all the participants before data collection. The whole intervention was conducted following the principles of human research specified in the Helsinki Declaration[12] and executed in compliance with currently applicable regulations and the provisions of the General Data Protection Regulation (GDPR)[13]. According to the exclusion criteria of this study, patients with less than 6 months of follow-up were excluded.All data regarding the demographic status, type of glaucoma, prior ocular surgeries, intraocular pressure (IOP) value and the number of drops needed before and after surgery, during several follow-up visits and postoperative complications were recorded. The patient data were collected by using a predesigned questionnaire and rechecked by the hospital register. All data were processed, analyzed and disseminated by using the MS Office program and described by mean and standard deviation.

Result

In this study, 35 eyes of 35 patients were analyzed. Among the total participants, 66% were male whereas the rest 34% were female. So, the male-female ratio of the participants was 2:1. The majority of our patients (40%) were from the 19-35 years age group, 31% were from the 36-65 years age group, 23% were from 18 years age group and only 6% were from >65 years age group. In analyzing the diagnostic findings among our participants, we observed that the majority of the participants (54%) were with aphakic glaucoma. Besides 14% and 11% were with neovascular glaucoma (NVG) and proliferative diabetic retinopathy with glaucoma respectively which is also noticeable. Among the majority of the participants (51%) the visual acuity was found <6/60, in 34% of cases the range was 6/36-6/12 and in 14% of cases the range of VA was 6/9-6/6. We found that the mean intraocular pressure (IOP) of our participants was 37.72% at the baseline. But the mean IOPs were 13.7%, 13.9%, 19.4% 18.43%, 16.5% and 17.6% on 1st day of surgery, at 1week, at 1 month, at 3 months, at 6 months and the end of 1 year respectively.As the most frequent complication, hyphemia was found in 11% of the total cases. Besides. vertical heterophoria, Descemet folds, shallow anterior chamber and ciliochoroidal detachment were found in some cases.

Table 1:Demographic status of participants (N=35)

Characteristics	n	%
Age (Year)		
<18	8	23%
19-35	14	40%
36-65	11	31%
>65	2	6%
Gender		
Male	23	66%
Female	12	34%

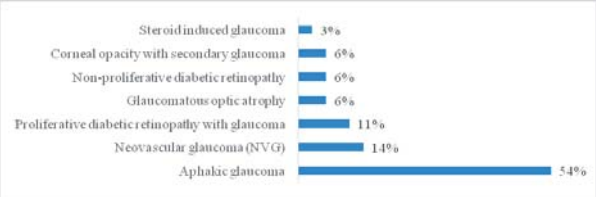

Figure 1: Distribution of participants as per the diagnostic findings(N=35)

Table 2: Visual acityof participants (N=35)

Visual acuity	n	%
<6/60	18	51%
6/36-6/12	12	34%
6/9-6/6	5	14%

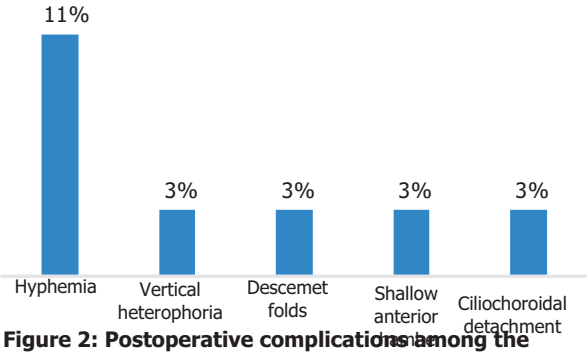

Figure 2: Postoperative complications among the participants (N=35)

Table 3 : The mean IOP changes eyes by AVG procedure (N=35)

Period	IOP (Mean)
Baseline	37.72
1st day	13.7
1 Week	13.9
1 Month	19.4
3 Month	18.3
6 Month	16.5
1 Year	17.6

Discussion

This study aimed to assess the demographic and clinical status of patients with refractory glaucoma who underwent Ahmed valve surgery.In this study, among the total participants, 66% were male whereas the rest 34% were female. The majority of our patients (40%) were from the 19-35 years age group.In this study, the majority of the participants (54%) were with aphakic glaucoma. Besides 14% and 11% were with neovascular glaucoma (NVG) and proliferative diabetic retinopathy with glaucoma respectively which was also noticeable.In some studies, it was found that refractory glaucoma was very common among eyes with secondary glaucoma especially neo-vascular glaucoma and aphakic/pseudo-phakic glaucoma.[14,15] In this study, the mean intraocular pressure (IOP) was 37.72% at the baseline. But the mean IOPs were 13.7%, 13.9%, 19.4% 18.43%, 16.5% and 17.6% on 1st day, 1week 1 month, 3 months 6 months and 1 year respectively. In another study[16], the mean preoperative intraocular pressure was 39.3±13.8 mm Hg.In our study, the majority of the participants (51%) were found in <6/60 at visual acuity, and 34% as well as 14% were found in 6/36-6/12 and 6/9-6/6 at visual acuity respectively.The best range of near visual acuity quantified with LogMar was 0.1 or better in 80% of the eyes studied[17]. In a study conducted in Ghana, there was a reduction of visual acuity in 23% of cases, but also a similar reduction in fellow eyes treatedwith medications alone[18]. In our study, as the most frequent complication, hyphemia was found in 11% of the total cases. So, hyphemia is a potential complication.Several complications such ashyphema, hypotony, corneal decompensation, cataract, and failure to control intraocular pressure, trans-scleral erosion as well as endophthalmitis (1.7%) werefound after AGV implantation[19,20]. On the other hand, Shunji Nakatake et al.found in their research that post-operative hyphema or increased concentration of some cytokines may lead to the failure of conjunctival bleb formation[24]. All the findings of this current study may be helpful in further similar studies.

Limitation of the study

This was a single-centered study with small-sized samples. Moreover, the study was conducted over a very short period. So, the findings of this study may not reflect the exact scenario of the whole country.

Conclusion & Recommendation

As per the findings of this current study, we can conclude that the frequency of refectory glaucoma among the younger population (At 19-35 years) is alarming. Younger male individuals are mainly prone to refectory glaucoma. The most frequent type of glaucoma among such cases is aphakic glaucoma which contributed more than fifty percent. To reduce the disease burden of refractory glaucoma, awareness development among the general population regarding the prevention and prompt treatment is very necessary. For getting more specific results, we would like to recommend conducting similar studies in several places with larger-sized samples.

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References

1. Coleman AL, Hill R, Wilson MR, et al. Initial clinical experience with the Ahmed glaucoma valve implant. Am J Ophthalmol. 1995; 120: 23–31.
2. Quigley HA, Broman AT. The number of people with glaucoma worldwide in 2010 and 2020. BrJOphthalmol. 2006; 90: 262-7.
3. Jonas JB, Yang D, Wang N. [Effect of intraocular pressure on glaucomatous damage to the optic nerve]. Ophthalmology. 2014; 111: 181-8; quiz 9-90.
4. Nassiri N, Kamali G, Rahnavardi M, Mohammadi B, Nassiri S, Rahmani L, Nassiri N. Ahmed glaucoma valve and single-plate Molteno implants in the treatment of refractory glaucoma: a comparative study. 2010;149(6):893-902
5. El Afrit MA, Trojet S, Mazlout H, Hamdouni M, Kraiem A. Efficacy of the Ahmed glaucoma valve implant in eyes with refractory glaucoma.2007;85(11):941-944
6. Souza C, Tran DH, Loman J, Law SK, Coleman AL, Caprioli J. Long-term outcomes of Ahmed glaucoma valve implantation in refractory glaucomas. 2007;144(6):893-900
7. Ishida K, Netland PA. Ahmed glaucoma valve implantation in African American and white patients. 2006;124(6):800-806
8. Al-Shahwan S. Transcorneal tube erosion of an Ahmed valve implant in an adult. 2010;17(4):377-378
9. Eid TM, Radwan A, el-Manawy W, el-Hawary I. Intravitreal bevacizumab and aqueous shunting surgery for neovascular glaucoma: safety and efficacy. 2009;44(4):451-456
10. Shah MR, Khandekar RB, Zutshi R, Mahrooqi R. Short-term outcome of Ahmed glaucoma valve implantation in management of refractory glaucomain a tertiary hospital in Oman. 2013;6(1):27-32
11. Desai MA, Gedde SJ, Feuer WJ, Shi W, Chen PP, Parrish RK, 2nd. Practice preferences for glaucoma surgery: a survey of the American Glaucoma Society in 2008. Ophthalmic Surg Lasers Imaging. 2011; 42: 202-8.
12. World Medical Association. (2001). World Medical Association Declaration of Helsinki. Ethical principles for medical research involving human subjects. Bulletin of the World Health Organization, 79 (4), 373 - 374. World Health Organization. https://apps.who.int/iris/handle/ 10665/268312.
13. Voigt, Paul, and Axel von dem Bussche. "Enforcement and fines under the GDPR." The EU General Data Protection Regulation (GDPR). Springer, Cham, 2017. 201-217.
14. Ishida K, Netland PA. Ahmed glaucoma valve implantation in African American and white patients. Arch Ophthalmol 2006; 124:800-6.
15. Montañez FJ, Laso E, Suñer M, Amaya C. Ahmed drainage device implant. Our experience between 1995 and 2003. Arch Soc Esp Oftalmol 2005; 80:239-44.
16. Alasbali T, Alghamdi AA, Khandekar R. Outcomes of Ahmed valve surgery for refractory glaucoma in Dhahran, Saudi Arabia. Int J Ophthalmol. 2015 Jun 18;8(3):560-4. doi: 10.3980/j.issn.2222-3959.2015.03.22. PMID: 26086008; PMCID: PMC4458663.
17. Javitt JC, Steinert RF. Cataract extraction with multifocal intraocular lens implantation; a multinational clinical trial evaluating clinical, functional, and quality-of-life outcomes. Ophthalmology 2000; 107: 2040-2048.
18. Egbert PR, Fiyadoyor S, Budenz DL, Dadzie P, Byrd S. Diode laser transscleral cyclophotocoagulation as a primary surgical treatment for primary open angle glaucoma. Arch Ophthalmol 2001; 119: 345–350.
19. Al-Torbak AA. The outcome of combined Ahmed glaucoma valve implant and penetrating keratoplasty in refractory congenital glaucoma with corneal opacity. 2004; 23(6):554-559.
20. Wilson MR, Mendis U, Paliwal A, Haynatzka V. Long-term follow-up of primary glaucoma surgery with Ahmed glaucoma valve implant versus trabeculectomy. 2003;136(3):464-470.
21. Nakatake S, Yoshida S, Nakao S, Arita R, Yasuda M, Kita T, et al. Hyphema is a risk factor for failure of trabeculectomy in neovascular glaucoma: a retrospective analysis. BMC Ophthalmol. 2014; 14: 55.

Original Article

Factors Related to Raised Intraocular Pressure: In a Tertiary Hospital in Bangladesh

M S Ahmed[1], S M Noman[2], Q S Mahnur[3]

Abstract

Glaucoma is the leading cause of irreversible blindness in the world, accounting for 8% of total blindness in the world (WHO, 2012) and systemic hypertension and diabetes mellitusare thought to increase the risk of development of glaucoma. Raised intraocular pressure is the only modifiable risk factor of glaucoma development and progression that can be treated medically and or surgically. This study was a hospital-based cross-sectional study, conducted in Ophthalmology department of BSMMU during the period of July 2021 to June 2022 The total sample size for this study was 153. Most of the respondents age group was 41-50 years. The Mean ±SD age was 37.15±17.14. 64(41.8%) were male and most 89(58.2%) were female. Refractive Error was found in 11(7.2%) cases. POAG was diagnosed in 51 cases(60%)NTG in 17cases(11.1%), PACG in 16 cases(10.5%), secondary glaucoma in 3 cases(2%), NVG in 1case(0.7%). There were 3(4.3%) patients in 20-29 years age group with the most frequent (IOP) in(mmHg) range 12-12 and followed by 29(42%) were 30-50 years with (IOP) in(mmHg) range 18-22 and 37(53.6%) were >50 years with (IOP)in(mmHg) range 22-36. There were 1(1.7%) patients in 20-29 years age group with the most frequent (IOP)A(mmHg) range 10-12 and followed by 22(37.3%) were 30-50 years with (IOP)A(mmHg) range 18-20 and 36(61%) were >50 years with (IOP)A(mmHg) range 20-40. There were 1(1.6%) patients in 20-29 years age group with the most frequent (IOP)A(mmHg) range 10-10 and followed by 25(39.7%) were 30-50 years with (IOP)A(mmHg) range 18-18 and 37(58.7%) were >50 years with (IOP)A(mmHg) range 20-36.50 % of the stablished glaucoma had diabetismalitus and 30 % had hypertension.10 % had the history of steroid therapy

Raised intraocular pressure (IOP) is an important risk factor for the incidence and progression of glaucoma. Diabetismalitus hypertension and steroid using are also important risk factors for glaucoma

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Key words : Glaucoma; IOP; POAG; Tonometer; Refractive error.

Introduction

Raised Intraocular pressure (IOP) is proven to be the most important risk factor for developing glaucoma, around the world. Hypertensive patients with IOP are more prone to develop Open Angle Glaucoma (OAG) which is one of the major causes of blindness globally (Langman et al, 2005). Glaucoma is not a single complication rather it refers to a group of diseases having common characteristics of optic neuropathy in relation with visual function loss. Although studies have found elevated intraocular pressure (IOP) as one of the primary risk factors for developing glaucoma but in reality, its presence or absence has less impact on the development of the disease (Foster et al, 2002). Age and genetic predispositions are the leading risk factors for developing glaucoma and further progression of the disease but they have a little interventional potential hence, IOP is left as the only parameter subject to treatment procedure (Kown et al, 2009). Studies have claimed glaucoma as the second common cause of blindness around the world specially after cataracts and hypertension is supposed to increase the risk of glaucoma development and progression (Resnikoff et al, 2002).

The risk factors for high ocular pressure are aging, sex, African race, hypertension, pulse rate, diabetes, obesity, alcohol use, smoking, myopia, colorblind, nuclear sclerosis and a family history of glaucoma (Leske et al, 1983). A Hypertension and other cardiovascular risk factors can affect many patients who have other comorbidities like ophthalmic disorders which leaded to rise of intra ocular pressure. A study conducted in Africa reported that glaucoma is responsible for 15% blindness and the region is found with the highest prevalence of blindness

relative to other places in the world. Another study also claimed that Open Angle Glaucoma (OAG) is more predominant among Africans than Europeans comparing to other regions (Park et al, 2009). Systemic hypertension has already affected more than 25% of the adult population globally and it is estimated that by 2025 more than 1.5 billion individuals will be affected globally (Kearny et al, 2005). A community-based survey conducted in Uganda reported the prevalence of hypertension at 7% of patients with IOP (Guwatudde et al, 2015). Besides, several population-based studies had also found a positive correlation between IOP and systemic blood pressure (Leske et al, 1983). Although raised IOP can easily be treated, its prevalence among hypertensive patients is still unknown. Because of the asymptomatic nature of Open Angle Glaucoma, most of the patients remains unknown about it until and hence they can develop irreversible complications. Therefore, the aim of this study was to investigate the prevalence of raised IOP and associated factors among hypertensive patients.

Objective of the Study

The objective of this study was to investigate factors relatedto raised IOP among the aged people.

Materials and Methodology

This study was a hospital-based cross-sectional study, conducted in Ophthalmology department of BSMMU during the period of June 2021 to July 2022. The total sample size for this study was 153.

All diagnosed case of chronic glaucoma were included

Patients with corneal irregularities that could affect the IOP readings e.g., in anterior segment pathology like corneal opacities and ulcers, collagen disorders like keratoconus, Endothelial-based corneal dystrophies (e.g., Fuchs), Previous corneal surgery involving Central cornea, Previous cornea trauma/injury, Previous refractive surgery, Corneal edema, Corneal astigmatism (3.00 D), Contact lens wear with induced corneal edema with severe surface disorder.

After the admission the blood pressure (B.P) of all patients was measured using a manual sphygmomanometer and when systolic blood pressure was > 130 mm Hg or a diastolic blood pressure > 80 mm Hg the blood pressure was considered to be raised or high. The sugar level of every patient was also measured. The Intraocular Pressure (IOP) was assessed with the applanation tonometry after applying tetracaine Hcl 0.1%. Three consecutive readings were taken and the average recorded as the measured IOP in mmHg. The IOP measurements were taken from 9 am to 12 noon by the principal investigator to avoid diurnal variation. IOP levels between 10 and 21 mmHg were considered normal. Values higher than 21 mmHg were considered raised IOP and below 10 mmHg were considered as ocular hypotension. Statistical Analysis was done using the SPSS version 21 software to establish the relationship between the dependent variable (IOP) and each independent variable.

Result:

Table I: Age Distribution of the Respondents

Age	N=153	Percentage (%)
0-10	8	5.2
11-20	20	13.1
21-30	25	16.3
31-40	21	13.7
41-50	42	27.5
51-60	24	15.7
61-70	9	5.9
71-80	4	2.6
Mean ±SD	37.15±17.14	

Table 1 shows the age distribution of the respondents. Most of the respondents 42(27.5%) were in the age group 41-50 years and followed by 8(5.2%) in 0-10 years, 20(13.1%) in 11-20, 25(16.3%) in 21-30, 21(13.7%) in 31-40, 24(15.7%) in 51-60, 9(5.9%) in 61-70, 4(2.6%) in 71-80 years. The Mean ±SD age was 37.15±17.14.

Figure 1: Age Distribution of the Respondents

Table II : Gender Distribution

Gender	N=110	Percentage (%)
Male	64	41.8
Female	89	58.2

Table II shows the gender distribution of the study patients where 64(41.8%) were male and most 89(58.2%) were female.

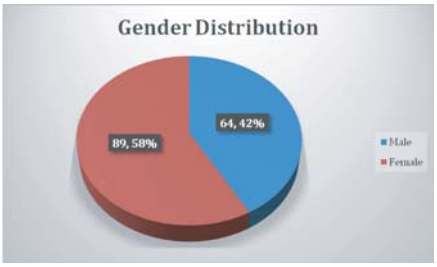

Figure 2: Gender Distribution

Table III: Diagnosis

DX	N=110	Percentage (%)
Refractive Error	11	7.2
NTG	17	11.1
PACG c-Catarect	16	10.5
NTG	3	2.0
	1	0.7

Table III shows the diagnosis done among the study patients. Refractive Error was found in 11(7.2%) cases and followed by NTG e-ps in 17(11.1%), PACG c-Cataract in 16(10.5%), ONTG in 3(2%), (S/P) 1(0.7%).

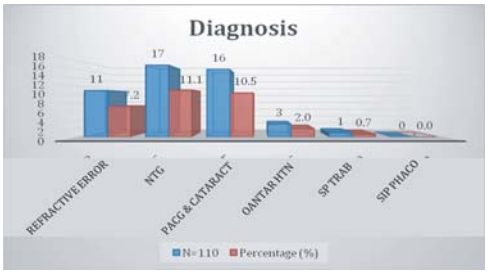

Figure 3: Diagnosis

Table IV: IOP Range

(IOP) (MHg)	N=153	Percentage (%)
8-14	21	13.7
12-20	9	5.9
14-36	7	4.6
15-16	5	3.3
16-20	19	12.4
17-18	6	3.9
18-48	27	17.6
20-22	9	5.9
21-22	6	3.9
22-36	8	5.2
24-40	8	5.2
25-23	4	2.6
26-18	3	2.0
Others	21	13.7

Table IV shows the (IOP)(MHg) in different ranges. Most of the study patient's (IOP)(MHg) 27(17.6%) was ranged between 18-48 and followed by 21(13.7%) was 8-14, 9(5.9%) was 12-20, 7(4.6%) was 14-36, 5(3.3%) was 15-16, 19(12.4%) was 16-20, 6(3.9%) was 17-18, 9(5.9%) was 20-22, 6(3.9%) was 21-22, 8(5.2%) was 22-36, 8(5.2%) was 24-40, 4(2.6%) was 25-23, 3(2%) was 26-18 and 21(13.7%) ranged others.

Age Group in Years	Diabetic Patients		Most Frequent (IOP)A(MHg) Range
	N=69	Percentage (%)	
20-29	3	4.3	12-12
30-50	29	42.0	18-22
>50	37	53.6	22-36

Table V shows the most frequent (IOP)(mmHg) range in diabetic patients in relation to age. There were 3(4.3%) patients in 20-29 years age group with the most frequent (IOP)(mmHg) range 12-12 and followed by 29(42%) were 30-50 years with (IOP)(mmHg) range 18-22 and 37(53.6%) were >50 years with (IOP)(mmHg) range 22-36.

Table VI: Most Frequent (IOP)(mmHg) Range in HTN Patients in Relation to Age

Age Group in Years	Hbp Patients		Most Frequent (IOP)(MHg) Range
	N=59	Percentage (%)	
20-29	1	1.7	10-12
30-50	22	37.3	18-20
>50	36	61.0	20-40

Table VI represents the most frequent (IOP)A(mmHg) range in HTN patients in relation to age. There were 1(1.7%) patients in 20-29 years age group with the most frequent (IOP)A(mmHg) range 10-12 and followed by 22(37.3%) were 30-50 years with (IOP)A(mmHg) range 18-20 and 36(61%) were >50 years with (IOP)A(mmHg) range 20-40.

Table VII: Most Frequent (IOP)(mmHg) Range in Cardiac Patients in Relation to Age

Age Group in Years	Cardiac Patients		Most Frequent (IOP)(mmHg) Range
	N=63	Percentage (%)	
20-29	1	1.6	10-10
30-50	25	39.7	18-18
>50	37	58.7	20-36

Table VII denotes the most frequent (IOP)(mmHg) range in cardiac patients in relation to age. There were 1(1.6%) patients in 20-29 years age group with the most frequent (IOP)(mmHg) range 10-10 and followed by 25(39.7%) were 30-50 years with (IOP)(mmHg) range 18-18 and 37(58.7%) were >50 years with (IOP)(mmHg) range 20-36.

Discussion

Studies had reported IOP as a good predictor for the onset of POAG, and proved that topical ocular hypotensive medication was effective in delaying or preventing the onset of POAG in individuals with elevated IOP(Gordon et al, 2002). Several studies reported that the glaucoma prevalence rate is increased with age(Song et el, 2011). Most of the respondents 27.5% were in the age group 41-50 years and followed by 5.2% in 0-10 years, 13.1% in 11-20, 16.3% in 21-30, 13.7% in 31-40, 15.7% in 51-60, 5.9% in 61-70, 2.6% in 71-80 years. The Mean ±SD age was 37.15±17.14. [table I].Most of the respondents 64(41.8%) were male and most 89(58.2%) were female[table II]. Similar finding like 42.25% male and 57.75%female were seen in other study (Hedner et al, 2005).

Refractive Error was found in 7.2% cases and followed by NTG e-ps in 11.1%, PACG c-Cataract in 10.5%, NTG in 2%, (B\E) 0.7%. [table III].Most of the study patient's (IOP)(mmHg) 17.6% was ranged between 18-48 and followed by 13.7% was 8-14, 5.9% was 12-20, 4.6% was 14-36, 3.3% was 15-16, 12.4% was 16-20, 3.9% was 17-18, 5.9% was 20-22, 3.9% was 21-22, 5.2% was 22-36, 5.2% was 24-40, 2.6% was 25-23, 2% was 26-18 and 13.7% ranged others[table IV].

This study showed statistically significant correlation among the patient with diabetics, HTN and cardiac diseases with raised IOP. There were 4.3% diabetic patients in 20-29 years age group with the most frequent (IOP)(mmHg) range 12-12 and followed by 42% were 30-50 years with (IOP)(mmHg) range 18-22 and 53.6% were >50 years with (IOP)(mmHg) range 22-36. [table V]. There were 1.7% HTN patient in 20-29 years age group with the most frequent (IOP)(mmHg) range 10-12 and followed by 37.3% were 30-50 years with (IOP)(mmHg) range 18-20 and 61% were >50 years with (IOP)(mmHg) range 20-40 [table VI]. There were 1.6% cardiac patient in 20-29 years age group with the most frequent (IOP)(mmHg) range 10-10 and followed by 39.7% were 30-50 years with (IOP)(mmHg) range 18-18 and 58.7% were >50 years with (IOP)(mmHg) range 20-36[table VII].

Conclusion

Glaucoma is proved to be second commonest cause of blindness globally which is accounting for 8 % of worldwide blindness. Raised intraocular pressure (IOP) is an important risk factor for the incidence and progression of glaucoma. Hypertensive patients are at a risk of developing and progression of primary Open Angle Glaucoma (OAG) which can be a leading

cause of blindness. Besides, the patients with diabetics and cardiac disease are also at high risk of developing glaucoma. Hence, hypertension, diabetics and cardiac disease is considered to be the alarming global health problems because of their chronic effects, devastating nature and cost of management among the patients with IOP. Although studies had shown an association between these diseases with IOP but there is no specific evidence yet of a causative link among them. Routine screening and monitoring of Intraocular pressure in patients with diabetics, HTN and cardiac diseases is required for the early recognition of raised IOP and early measurement of prevention of Glaucoma-associated with blindness.

References

1. Foster, P. J., Buhrmann, R., Quigley, H. A. &Johnson, G. J. 2002. The definition and classification of glaucoma in prevalence surveys. British journal of ophthalmology, 86**,** 238-242.
2. Gordon, M. O., Beiser, J. A., Brandt, J. D., Heuer, D. K., Higginbotham, E. J., Johnson, C. A., Keltner, J. L., Miller, J. P., Parrish, R. K. & Wilson, M. R. 2002. The Ocular Hypertension Treatment Study: baseline factors that predict the onset of primary open-angle glaucoma. Archives of ophthalmology, 120**,** 714-720.
3. Guwatudde, D., Mutungi, G., Wesonga, R., Kajjura, R., Kasule, H., Muwonge, J., Ssenono, V. & Bahendeka, S. K. 2015. The epidemiology of hypertension in Uganda: findings from the national non-communicable diseases risk factor survey. PloS one, 10**,** e0138991.
4. Hedner, T., Narkiewicz, K. & Kjeldsen, S. E. 2005. Hypertension control–A global challenge. Blood Pressure, 14**,** 4-5.
5. Kearney, P. M., Helton, M., Reynolds, K., Muntner, P., Whelton, P. K. & He, J. 2005. Global burden of hypertension: analysis of worldwide data. The lancet, 365**,** 217-223.
6. Kwon, Y. H., Fingert, J. H., Kuehn, M. H. & Alward, W. L. 2009. Primary open-angle glaucoma. New England Journal of Medicine, 360**,** 1113-1124.
7. Langman, M.J.S, Lancashire, R.J, Cheng, K. & Stewart, P. 2005. Systemic hypertension and glaucoma: mechanisms in common and co-occurrence. British Journal of Ophthalmology, 89**,** 960-963.
8. Leighton, D. & Phillips, C. 1972. Systemic blood pressure in open-angle glaucoma, low tension glaucoma, and the normal eye. The British journal of ophthalmology, 56**,** 447.
9. Leske, M. C. & Podgor, M. J. 1983. Intraocular pressure, cardiovascular risk variables, and visual field defects. American journal of epidemiology, 118**,** 280-287.
10. Park, S. B., Nam, Y. P., Sung, K. R. & Kook, M. S. 2009. Structural and functional relationships in glaucoma using standard automated perimetry and the humphrey matrix. Korean Journal of Ophthalmology, 23**,** 176-182.
11. Resnikoff, S., Pascolini, D., Etya'ale, D., Kocur, I., Pararajasegaram, R., Pokharel, G. P. & Mariotti, S. P. 2004. Global data on visual impairment in the year 2002. Bulletin of the world health organization, 82**,** 844-851.
12. Song, W., Shan, L., Cheng, F., Fan, P., Zhang, L., Qu, W., Zhang, Q. &Yuan, H. 2011. Prevalence of glaucoma in a rural northern china adult population: a population-based survey in Kailu county, inner Mongolia. Ophthalmology, 118**,** 1982-1988.

Original Article

Association of Hypothyroidism with Primary Open Angle Glaucoma in Diabetic patients at tertiary hospital

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Abstract

Background: Hypothyroidism and primary open-angle glaucoma (POAG) are prevalent medical conditions with significant implications for public health. However, the potential association between these two conditions has been a subject of debate and limited research. This study aims to investigate the relationship between hypothyroidism and the risk of developing POAG.

Objective: The aim of this study was to observe the association of hypothyroidism with POAG in diabetic patients.

Methodology: In this cross-sectional study at BIRDEM General Hospital, Dhaka, from March 2022 to April 2023, 185 diabetic respondents aged 40 to 60, with and without hypothyroidism were examined. Respondents were consecutively selected from the Ophthalmology & Endocrinology OPD, excluding hyperthyroid cases and those with ocular surgery, inflammation, trauma, secondary glaucoma, or angle closure glaucoma history. Data was collected via ocular examination and specialized tests, analyzed using SPSS. Ethical considerations included informed consent, confidentiality, and Institutional Review Board approval and hospital records were used for data collection.

Results: Among total 185 Diabetic respondents, 17.3 % had POAG and 37.8 % had hypothyroidism. Among POAG respondent 34.4 % hypothyroid and 65.6 % normal. Mean age 54.2±6.06 years and 51.03±6.6 years, male 53.1 % and 30.7%, duration of DM 12.56±5.7 and 9.69±6.25, family history of glaucoma 9.4 % and 0.7 %, HTN 37.5% and 26.1 % in POAG and normal respondent respectively. OR with 95 % CI of hypothyroid and POAG of the respondent 0.83(95% confidence interval, 0.03-1.85).

Conclusion: In this study, no significant association was observed between hypothyroidism and primary open angle glaucoma.

Keywords: Hypothyroidism, Primary open angle glaucoma, Diabetes Mellitus

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Introduction

One of the common reason for preventable blindness worldwide is glaucoma[1]. When the condition is recognized, it manifests with an irreversible loss of vision because it is asymptomatic in the early stages[2]. Primary Open Angle Glaucoma (POAG) is defined as a progressive, chronic optic neuropathy where intraocular pressure (IOP) and some other unknown factors contribute to damage resulting in atrophy of the optic nerve and loss of retinal ganglion cells and their axons and occurred in age more than 40 years.

Although the mechanism of retinal ganglion cell death is not fully understood, elevated IOP is considered the most important risk factor. Several large randomized clinical trials showed a relationship between IOP and glaucoma development and progression. Besides the mechanical effect of raised IOP on the optic nerve head (ONH), several vascular and systemic factors have also been identified as risk factors. Such factors can lead to hypoperfusion of the ONH and may thus play an important role in the pathogenesis and progression of primary open-angle glaucoma (POAG)[3].

About 60 million people worldwide are affected by glaucoma. POAG predominates over PACG in most populations, with a prevalence of 3.54% in those between 40 and 80 years old[4]. It is estimated that 3.6 million people worldwide are blind due to POAG (Collaborate GBD, 2019). Thyroid problem have been reported in over 110 countries of the world with 1.6 billion at risk and most of these are in developing countries. The

prevalence rate of primary hypothyroidism is 1.02% and subclinical hypothyroidism is 13.93%[8].

Glaucoma is correlated with various systemic disorders like diabetes mellitus, hypertension, thyroid disorders making it worthwhile to look for a relationship between it and any systemic disease[5].

Hypothyroidism is one of these systemic disorders. It was reported that 23.4% of the patients with primary open-angle glaucoma (POAG) had hypothyroidism. The same authors also reported the possibility of a decrease in intraocular pressure (IOP) after treatment of hypothyroidism[6].

The thyroid gland, under hypothalamic pituitary control, secretes 93% thyroxine (T4) and 7% triiodothyronine (T3). These hormones increase transcription of several genes known to affect catabolism. Iodine deficiency is the most common cause of hypothyroidism worldwide, but in iodine-sufficient regions, autoimmune disease (Hashimoto's thyroiditis) and iatrogenic causes (After thyroid surgery or anti-thyroid drug treatment) are more frequent. Eye involvement in thyroid disease ranges from well-documented thyroid-associated orbitopathy to hypothyroid eye changes including conjunctival and periorbital oedema, blepharoptosis, and nyctalopia[7].

Although, a well-described association exists between Open-Angle Glaucoma (OAG) and thyroid-related orbitopathy, the association of hypothyroidism with open angle glaucoma is conflicting. Many population based survey showed an association of hypothyroidism with primary open angle glaucoma. In this study, we evaluated association of hypothyroidism with primary open angle glaucoma at tertiary hospital so that we can play some role to prevent the one of most common cause of preventable blindness worldwide for the benefit of the patients. In this way, it will be easier to create a nationwide recommendation to check every relevant hypothyroid patient for glaucoma and perform early intervention to restore eyesight, which may open up new possibilities for glaucoma prevention and therapy.

Materials and Method

A cross sectional analytical study was carried out at the Department of Ophthalmology in collaboration with Department of Endocrinology, BIRDEM General hospital, Dhaka from March 2022 to April 2023. The study population comprised of 185 type 2 diabetic patients in total. Patients either admitted or attending OPD at the department of Ophthalmology & Endocrinology OPD of BIRDEM General Hospital, Dhaka were included in this study. Consecutive type of sampling technique was applied to enroll the patients. Inclusion criteria were Diabetic patients age 40-60 years old, who were with and without diagnosed case of hypothyroidism. Hyperthyroid, patients with history of trauma, any ocular surgery, angle closure glaucoma, secondary glaucoma were excluded. POAG were defined as age between 40-60 years, of all sexes, IOP > 21mm of Hg or 21mm of Hg having definite glaucomatous optic nerve head damage, receiving anti glaucoma medications, glaucomatous optic nerve head changes and visual field defect, open irido-corneal angle assessed with gonioscopy and normal anterior chamber depth.

Each of them underwent comprehensive ocular examination like Visual acuity, Slit lamp Bio microscopy, IOP (done by Goldmann applanation tonometer. Normal value is 10-21 mm of Hg), gonioscopy (with the help of Zeiss 4 mirror lens and anterior chamber was designated as open on the basis of Shaffer grading system grade 2 or more i.e. posterior Trabecular meshwork visible at least 270º) and fundoscopy (done with the slit lamp via the help of Ocular +78 D condensing lens).

After detailed ophthalmic examination, those patients who were glaucoma suspect underwent Color Fundus Photography for documentation, CCT (Central corneal thickness) to measure the actual IOP, OCT of optic nerve head and Retinal nerve fibre layer analysis for early glaucoma diagnosis and Visual field analysis for assessing functional damage.

Statistical analysis

Statistical analysis was conducted using SPSS 27.0 software. Numerical data were represented as mean and SD, while categorical data were shown as frequency and percentage. The Chi-square test was used for categorical data, and the unpaired t-test for numerical data. Significance was defined as $p < 0.05$. Data were displayed in tables and graphs. Descriptive statistics included frequency tables, and associations were depicted using cross tables with test statistics noted in table footnotes.

Table I: Distribution of the respondents according to POAG and Thyroid status (n=185)

Characteristics	Frequency	Characteristics
POAG		
Yes	32	17.3
No	153	82.7
Thyroid status		
Hypothyroid	70	37.8
Euthyroid	115	62.2

Table I showing among the 185 respondent, 32 (17.3%) had POAG. And among the 32 POAG respondent 11(34.4 %) had hypothyroidism and 21(65.6%) had euthyroid.

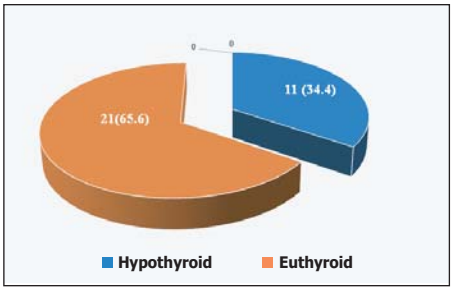

Figure 1: Pie diagram showing total 32 POAG in hypothyroid (34.4%) and Euthyroid (65.6%) respondents

Table II: OR with 95% CI of hypothyroid and POAG of respondent (n=185)

Thyroid status	POAG (n=32)	No POAG (n=153)	P value	OR (95% CI)
Hypothyroid	11 (34.4)	59 (38.6)	0.694[a]	0.83 (0.03-1.85)
Euthyroid	21 (65.6)	94 (61.4)		

[a] Chi square test

Table II showing among POAG respondent 34.4 % had hypothyroidism and 65.6 % had euthyroid. Similarly, among No POAG respondents 38.6 % had hypothyroid and 61.4 % had euthyroid. This result was statistically not significant (P=0.694). In this study, hypothyroid respondents had 0.83 times lower chance to have POAG (OR=0.83, 95% CI=0.03-1.85)

Table III: Comparison of Age and Sex among POAG and normal respondents (n=185)

Characteristics	POAG (n=32)	Normal (n=153)	P value
Patient's Age (years)			
40-50	10 (31.3)	76 (49.7)	0.049 [a]
51-60	22 (68.8)	77(50.3)	
Mean ±SD	54.2±6.06	51.03±6.6	0.013
Gender			
Male	17 (53.1)	47 (30.7)	0.031 [a]
Female	15(46.9)	106(69.3)	

[a] Chi square test

Table III: Comparison of Age and Sex among POAG and normal respondents shows significant difference between POAG and normal respondents. The mean age of POAG is 54.2±6.06 and 51.03±6.6 in normal respondents. Among the respondent's male were 17 and female were 15 in POAG group, 47 were male and 106 were female in normal group.

Table IV: Comparison of risk factors among POAG and normal respondents (n=185)

Characteristics	POAG (n=32)	Normal (n=153)	P value
Duration of DM			
<10 years	10 (31.2)	85 (55.6)	0.041 [a]
³10 years	22 (68.8)	68 (44.4)	
Mean ± SD	12.56±5.7	9.69±6.25	0.017 [c]
HTN			
No	20 (62.5)	113 (73.9)	0.201 [a]
Yes	12 (37.5)	40 (26.1)	
F/H of Glaucoma			
No	29 (90.6)	152 (99.3)	0.017 [b]
Yes	3 (9.4)	1(0.7)	
Hypercholesterolemia			

No	25 (78.1)	120 (78.4)	1.000 [a]
Yes	7 (21.9)	33 (21.6)	
F/H of Hypothyroidism			
No	31 (96.9)	144 (94.1)	0.696 [a]
Yes	1 (3.1)	9 (5.9)	
IHD			
No	30 (93.8)	126 (82.4)	0.119 [a]
Yes	2 (6.3)	27 (17.6)	

[a] chi square test, [b] fisher's exact test, [c] unpaired t test

Table IV: Comparison of risk factors among POAG and normal respondents shows significant result in duration of DM and family history of glaucoma. The mean duration of DM for POAG 12.56±5.7 years and in normal group 9.69±6.25 years. Family history of glaucoma suggests that about majority of the patients from both groups (90.6% & 99.3%) had no history of glaucoma but POAG respondents (9.4%) tend to have a strong family history of glaucoma than normal respondents (0.7%). About 37.5% and 26.1% respondents in both groups had hypertension, 21.9% and 21.6% had hypercholesterolemia, 3.1% and 5.9% had positive family history of hypothyroidism and 6.3% and 17.6% respondents had IHD.

Table V: Comparison of ocular parameter of the respondents (n = 185)

Characteristics	POAG (n=32)	Normal (n=153)	P value
VA (Log MAR score) Mean ± SD	0.30±0.14	0.29±0.13	0.526c
IOP (Intraocular pressure) Mean ± SD	18.63±3.81	14.08±2.33	<0.001c
CD ratio (Cup to Disc) Mean ± SD	0.72±0.09	0.41±0.09	<0.001c

[c] unpaired t test

Table V: Comparison of ocular parameter of the study population shows visual acuity in POAG group and normal group 0.30±0.14 and 0.29±0.13 (p value 0.526), IOP 18.63±3.81 and 14.08±2.33 in both groups, CD ratio 0.72±0.09 and 0.41±0.09 in both groups respectively which showed significant results (p value <0.001).

Discussion

This study was carried out to see association of hypothyroidism with POAG in diabetic patients. Although several risk factors for the development of POAG, such as diabetes, increased age, IOP, and family history of glaucoma have been assessed, this research area remains the subject of ongoing investigation. In order to confirm this potential association, several epidemiological studies have tried but failed to reach a consistent conclusion. Thus, it remains controversial whether or not hypothyroidism increases the risk of POAG.

Comparison of Age and Sex among POAG and normal respondents showed significant difference. The mean age of POAG is 54.2±6.06 years and 51.03±6.6 years in normal respondents. A study showed that mean age in glaucomatous patient were 44.10±9.17 and non-glaucomatous patient were 40.50±8.50 [2]. Another study showed that the mean age of similar groups was 68.0±7.35 and 67.28±10.05 years [8]. Among the respondents, 17 were male and female were 15 in POAG group, 47 were male and 106 were female in normal group. A study showed that males were dominant in both groups (glaucomatous and non-glaucomatous patients) [2] and in other study where females were also the majority in similar groups (61.7% and 61.7% respectively) [9]. On the contrary, another study found that male and female ratios were same [16].

Comparison of risk factors among POAG and normal respondents shows significant result in duration of DM and family history of glaucoma. The mean duration of DM for POAG 12.56±5.7 years and in normal group 9.69±6.25 years. Family history of glaucoma suggests that about majority of the patients from both groups (90.6% & 99.3%) had no history of glaucoma. In a study it was showed that most patients had hypertension and hyperlipidemia [2]. In other study, about 5.4% patients had positive family history of glaucoma [10]. However, other revealed that individuals with DM have an increased risk of developing POAG [11] [12]. But there was no statistically significant difference in the duration of HTN between the two groups. In a study, it was showed that HTN was able to increase the

risk of OAG and DBP (Diastolic blood pressure) instability [13].

Distribution of ocular parameter of the study population shows visual acuity in POAG group and normal group 0.30±0.14 and 0.29±0.13 (p value 0.526), IOP 18.63±3.81 and 14.08±2.33 in both groups, CD ratio 0.72±0.09 and 0.41±0.09 in both groups respectively which showed significant results (p value <0.001). In a study, it was found that, the comparison of IOP in Glaucoma (+) and Glaucoma (-) patients were 15.8±3.1 mmHg and 15.0±3.0 mm Hg. IOP was 15.8±3.1 in glaucomatous group and 15.0±3.0 in non-glaucomatous patients [14]. And other showed in their study visual acuity in non-glaucomatous and open angle glaucoma patients were 0.71±0.09 and 0.80±0.08, IOP was 19.90±4.60 and 17.06±4.20, CD ratio was 0.60±0.10 and 0.70±0.17 respectively[15].

OR with 95% CI of hypothyroid and POAG of respondent shows non-significant result. In this study, hypothyroidism patient had 0.83 times lower chance to have POAG. OR 0.83(95% CI=0.03-1.85). A study showed that OR (95% CI) for diagnosed hypothyroid 0.97 (0.89-1.06) and for untreated hypothyroidism is 1.09 (0.96-1.24) [9].

Conclusion

This cross sectional analytical study was undertaken to find out the association of hypothyroidism with primary open angle glaucoma in 185 diabetic patients. This study revealed there was no significant association of hypothyroidism with POAG among diabetic population. Older age, male gender, family history of glaucoma, and longer diabetes duration as significant POAG risk factors for diabetic population.

Authors Contributions

All authors contributed to the article's drafting, revision, drafting and finalizing to fulfill authorship criteria.

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This study has been done by self-finance .

Conflict of interest

The author has no conflict of interest to declare.

Ethical Approval

The institutional review board has approved this research protocol, on 28 February 2022 before starting this study.

References

1. Steinmetz JD, Bourne RR, Briant PS, Flaxman SR, Taylor HR, Jonas JB, Abdoli AA, Abrha WA, Abualhasan A, Abu-Gharbieh EG, Adal TG. Causes of blindness and vision impairment in 2020 and trends over 30 years, and prevalence of avoidable blindness in relation to VISION 2020: the Right to Sight: an analysis for the Global Burden of Disease Study. The Lancet Global Health. 2021 Feb 1;9(2):e144-60.
2. Lee JY, Kim TW, Kim HT, Lee MY, Min HW, Won YS, Kwon HS, Park KH, Kim JM, Epidemiologic Survey Committee of the Korean Ophthalmological Society. Relationship between anthropometric parameters and open angle glaucoma: The Korea National Health and Nutrition Examination Survey. PloS one. 2017 May 8;12(5):e0176894.
3. Chung HJ, Hwang HB, Lee NY. The association between primary open-angle glaucoma and blood pressure: two aspects of hypertension and hypotension. BioMed Research International. 2015 Oct 18;2015.
4. Quigley HA, Broman AT. The number of people with glaucoma worldwide in 2010 and 2020. British journal of ophthalmology. 2006 Mar 1;90(3):262-7.
5. Tham YC, Li X, Wong TY, Quigley HA, Aung T, Cheng CY. Global prevalence of glaucoma and projections of glaucoma burden through 2040: a systematic review and meta-analysis. Ophthalmology. 2014 Nov 1;121(11):2081-90.
6. Smith TJ, Murata Y, Horwitz AL, Philipson L, Refetoff S. Regulation of glycosaminoglycan synthesis by thyroid hormone in vitro. The Journal of clinical investigation. 1982 Nov 1;70(5):1066-73.
7. Ferman SS. New discoveries in diabetes and thyroid related eye disease. Curr Opin Ophthalmology 2007; 8(6): 61-65
8. Li J, Li Y, Shi X, Teng D, Teng X, Teng W, Shan Z. Prevalence and risk factors of hypothyroidism after universal salt iodisation: a large cross-sectional study from 31 provinces of China. BMJ open. 2023 Feb 1;13(2):e064613.
9. Motsko SP, Jones JK. Is there an association

between hypothyroidism and open-angle glaucoma in an elderly population?: an epidemiologic study. Ophthalmology. 2008 Sep 1;115(9):1581-4.

10. Lee AJ, Goldberg I. Emerging drugs for ocular hypertension. Expert opinion on emerging drugs. 2011 Mar 1;16(1):137-61.
11. Zhou M, Wang W, Huang W, Zhang X. Diabetes mellitus as a risk factor for open-angle glaucoma: a systematic review and meta-analysis. PloS one. 2014 Aug 19;9(8):e102972.
12. Shen LQ, Kloek CE, Turalba AV. Assessing the effect of a glaucoma surgical curriculum in resident physicians. JAMA ophthalmology. 2015 Sep 1;133(9):1077-80.
13. Nislawati R, Zainal AT, Ismail A, Waspodo N, Kasim F, Gunawan AM. Role of hypertension as a risk factor for open-angle glaucoma: a systematic review and meta-analysis. BMJ Open Ophthalmology. 2021 Sep 1;6(1):e000798.
14. Won YS, Kim DY, Kim JM. Glaucomatous optic nerve changes and thyroid dysfunction in an Urban South Korean Population. Journal of Ophthalmology. 2017 May 2;2017.
15. Kakigi C, Kasuga T, Wang SY, Singh K, Hiratsuka Y, Murakami A, Lin SC. Hypothyroidism and glaucoma in the United States. PLoS One. 2015 Jul 31;10(7):e0133688.
16. Jonas JB, Aung T, Bourne RR, Bron AM, Ritch R, Panda-Jonas S. Glaucoma–Authors' reply. The Lancet. 2018 Feb 24;391(10122):740.
17. Kapetanakis VV, Chan MP, Foster PJ, Cook DG, Owen CG, Rudnicka AR. Global variations and time trends in the prevalence of primary open angle glaucoma (POAG): a systematic review and meta-analysis. British Journal of Ophthalmology. 2016 Jan 1;100(1):86-93.
18. La Croix N, Wayne J. Ophthalmic Lasers for the Treatment of Glaucoma. Laser Surgery in Veterinary Medicine. 2019 Jun 4:129-40.
19. Khan AA, Mishra A, Khan AA. Association between Hypothyroidism and Primary Open Angle Glaucoma. Int J Acad Med Pharm. 2023;5(3):339-43.

Management of Secondary Angle Closure Glaucoma

S M Noman[1], A S M M Quadir[2]

Introduction

In secondary angle closure glaucoma, the underlying cause can close the angle directly by local iris and angle factors or by acting to move the crystalline lens forward causing pupillary block (secondary pupillary block). This is important since some of these patients with secondary pupillary block will respond to laser iridotomy. They are common causes of glaucoma and can produce high elevations of intraocular pressure (IOP) and ocular morbidity. This review will discuss the risk factors, signs and symptoms, pathophysiology, imaging and the treatment modalities of secondary angle closure glaucomas.

Aqueous Misdirection Syndrome

The aqueous misdirection syndrome is a form of secondary angle closure glaucoma occurring postsurgery with raised intraocular pressure (IOP), shallow or fat anterior chamber (AC) in the presence of a patent peripheral iridectomy (PI)[1] (Figs 1A to toC).C). It is unresponsive to miotic or fltering surgery. It can occur after fltering surgery,[2] cataract[3]/combined surgery, surgical peripheral iridectomy, following suturelysis,[4] glaucoma drainage device implantation[5] or even after laser iridotomy.[6] The predisposing factors are preexisting angle closure glaucoma, shallow anterior chamber due to wound leak or overflteration.

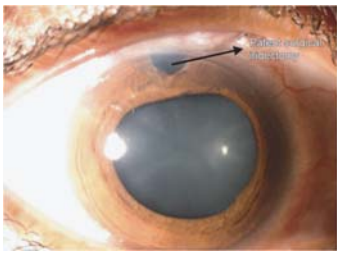

Fig. 1A : Aqueous misdirection post fltering surgery showing a patent surgical iridectomy

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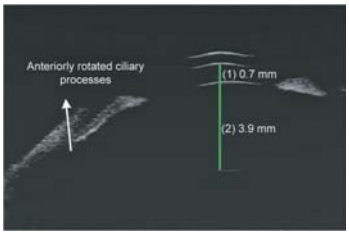


Fig. 1C : UBM showing shallow anterior chamber with anterior rotation of ciliary processes

The pathophysiology is not completely understood, but is believed that the primary mechanism is a blockage of anterior aqueous fow at the level of the ciliary body combined with an inherent impermeability defect in the anterior hyaloid.[7] Recently, choroidal expansion has been proposed as a contributory factor as evidenced by ultrasound biomicro-scopy (UBM) studies showing fuid in the suprachoroidal space in some patients with ciliary block glaucomas.[8]

Clinical Features

It is seen in the postoperative period anytime from the 1st day to weeks, sometimes months later. The features are axial shallowing of AC, high IOP or normal IOP in case of functioning blebs, patent PI, closed angle on gonioscopy and ciliary processes can be seen rotated forward pressing against the base of the iris in case of choroidal effusions. UBM shows anterior rotation of ciliary processes pressing against the lens equator (or anterior hyaloid in aphakia).[9]

The principles of treatment are to relieve the obstruction of aqueous fow and restore normal intraocular pressure by medical therapy, to surgically correct the block to aqueous fow, re-establish a normal aqueous fow pathway and drain the aqueous from its abnormal location.

Medical Therapy

Rule out pupillary block by verifying or creating a patent iridectomy/iridotomy. Start mydriatic-cycloplegic therapy[10] consisting of 1% atropine and 5% phenylephrine twice a day in phakic and

pseudophakic eyes. This tightens the lens–zonular diaphragm to resist the force from behind and also dilate the ciliary body ring to move the ciliary body away from peripheral anterior hyaloid. Fifty percent success rate has been noted with this therapy. Additionally, topical steroids, aqueous suppressants are used to reduce infammation and IOP. Osmotics help to lower the IOP as well as to reduce the vitreous volume. Conservative treatment is advised for 5 days to see for resolution.

YAG hyaloidotomy disrupts the anterior hyaloid face allowing aqueous to drain out of the vitreous. It's an elective procedure done in pseudophakics and aphakics through the surgical iridectomy, first perforating the posterior lens capsule and then the hyaloid face. Slight deepening of the anterior chamber is noticeable immediately, dramatic deepening is noticeable after 12 to 24 hours.

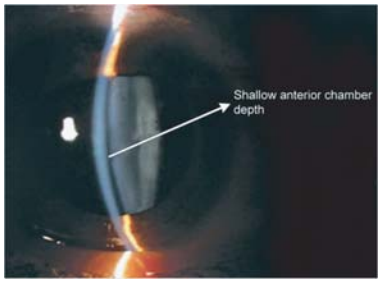


Fig. 1B : Slit photo showing shallow anterior chamber

Surgical Therapy

In cases of medical and laser therapy failure, vitreous surgery almost always cures this condition. The surgery is intended to establish an opening through the anterior hyaloid membrane and the vitreous body for aqueous humour to escape forward to the posterior and anterior chambers. Pars plana vitrectomy[2,3] is done in pseudophakic and aphakic eyes and IOP control is achieved in 67 to 100% of cases. Vitrectomy-phacoemulsificaton-vitrectomy[11] was studied in 5 phakic eyes, the preliminary vitrectomy was limited to core vitrectomy to debulk the vitreous, the residual vitrectomy, zonulohyaloidectomy and peripheral iridectomy was performed to create a free communication between the anterior and posterior chambers. Cyclodiode laser photocoagulation has also been proposed in patients with failed medical therapy to break the cycle of aqueous misdirection and prevent future recurrences.[12]

YAG PI done preoperatively in occludable eyes can reduce aqueous misdirection. In patients with increased risk like in decompression related shallowing of the anterior chamber, using viscoelastics during surgery, tighter closure and cycloplegics after surgery prevents aqueous misdirection. It must be remembered that recurrence can occur so vigorous follow-up and chronic atropine drops may be necessary. The fellow eye is also at a high-risk for aqueous misdirection.

Neovascular Glaucoma

Neovascular glaucoma (NVG) arises in response to retinal ischemia, the common predisposing factors being central retinal vein occlusion and diabetic retinopathy. An NVG patient requires a broad diagnostic workup to determine the underlying cause[13] and also a predisposed patient requires careful monitoring to detect NVG in its earliest stages.

The pathogenesis of NVG is retinal ischemia which produces angiogenic factors (VEGF, angiogenin, PDGF, TGF-á, â, TNF-á) which in turn causes new vessel formation on the iris and angle leading to formation of fibrovascular membrane which eventually contracts to form peripheral anterior synechiae (PAS) and ultimately complete closure of the angle. VEGF levels are elevated to 40 to 100 times normal in aqueous and vitreous of patients with rubeosis and NVG.[14] The clinicopathologic course and treatment may be described in the following stages.[15]

Rubeosis Stage (Preglaucoma)

Neovascularization is typically first seen in the pupillary border;[16] however, gonioscopy should be done in all patients under the risk to look for angle neovascularization, since it can precede

iris neovascularization.[17] The IOP is usually normal, rubeosis is seen as fine randomly oriented vessels near pupillary border or may be first seen in the angle in diabetic and CRVO patients.[18] Pan retinal photocoagulation (PRP) is indicated which decreases the retinal oxygen demand, thereby reducing the stimulus for release of angio genesis factors.[19]

Open-angle Glaucoma Stage

The rubeosis is more forid in this stage with elevated IOP and open angles with intense neovascularization of the angle. PRP can reverse IOP elevation in this stage. Aqueous suppressants, topical corticosteroids and atropine are used.

Angle Closure Glaucoma Stage

PAS formation can eventually close the angle with ectropion uveae formation resulting in severe glaucoma requiring surgical intervention. PRP is still useful in this stage to reduce IOP if synechial closure is less than 270°[20] and even if the angle is closed for more than 270° PRP is useful in reducing anterior segment neovascularization before any surgical intervention.[21]

Filtering surgery is rarely successful because of the risk of bleeding and postoperative progression of fibrovascular membrane. In a study in NVG patients, mitomycin C (MMC) augmented trabeculectomy yielded success rates of 62.6, 58.2 and 51.7% at 1, 2 and 5 years respectively.[22] Modifed trabeculectomy can be performed with intraocular bipolar cautery of peripheral iris and ciliary processes to prevent bleeding.[23] Intracameral Bevacizumab (avastin)[24] has been tried in NVG patients resulting in rapid regression of the iris and angle neovascularization and could act as an adjunct in the surgical treatment of these patients. Preoperative intravitreal bevacizumab combined with mitomycin C augmented trabeculectomy has been studied to be a safe and effective method for controlling IOP in NVG, though long-term effects are not known.[25] Even comparative studies between combined intravitreal bevacizumab (IVB) and trabeculectomy with MMC verses trabeculectomy with mitomycin C alone concluded that IVB significantly reduces postoperative hyphema and controls early postoperative IOP with no difference in long-term results.[26]

GDDS have gained wide acceptance as a primary procedure for NVG patients because the tube bypasses the fibro-vascular membrane. The aqueous tube shunts have reported success rates of 22 to 97% in these patients.[27,28] Molteno tube shunts[29] Ahmed glaucoma valve[30] and Baerveldt glaucoma implants[31] have shown comparable success rates. Successful IOP control was achieved with com bined pars plana vitrectom y and glaucoma drainage implant in selected patients with refractory NVG.[32]

In eyes with little or no visual potential, treatment is mainly with topical cycloplegics, steroids and aqueous sup-pressants. Ablation of the ciliary body is undertaken in cases of symptomatic patients. Bloom et al reported a mean IOP reduction of 53% in 25 patients with diode laser for refractory glaucoma.[33] However of note is the study by Ramli et al[34] that underlying NVG was a significant risk factor for hypotony post transscleral cyclophoto-coagulation. A less aggressive energy setting and a more limited treatment was advised to prevent hypotony.

Iridocorneal Endothelial Syndromes

Iridocorneal endothelial (ICE) syndrome is a spectrum of ocular diseases characterized by corneal endothelial abnormalities, unilateral glaucoma and iris stromal abnor-malities.[35] They include progressive iris atrophy, Chandler syndrome and Cogan-Reese syndrome (Figs 2A to toC).C). They may be regarded as different manifestations of the same disease process. It is caused by an abnormal corneal endo-thelium which forms a membrane (ICE membrane) over the anterior surface of the iris and the angle structures, which on contraction distorts the iris, forms peripheral anterior synechiae and closes the angle leading to glaucoma.[36] Half of all the patients of ICE syndrome develop glaucoma.[37]

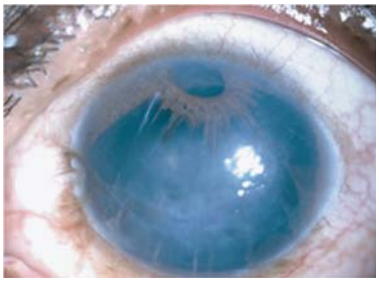


Fig. 2A : Progressive iris atrophy

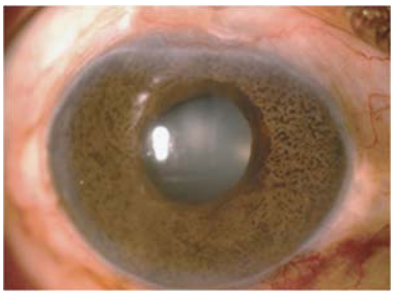


Fig. 2C : Cogan-Reese syndrome

Management of glaucoma in ICE syndromes is difficult and medical therapy is restricted to aqueous suppressants which is often unsuccessful.[38] The success rate of fltering surgery is also lower than most other forms of glaucoma.[39,40] A study of surgical outcome of trabeculectomy with MMC in 10 ICE syndrome patients showed 8 of 10 patients having adequate IOP control after fltering surgery with mitomycin-C, with a mean follow-up of 14.9 months. The results of their study suggested that trabeculectomy with adjunctive mitomycin-C may offer better intermediate-term success than trabeculectomy alone or trabeculectomy with 5-fluorouracil.[41] Failure of fltering surgery is often due to the continued growth of the endothelial membrane over the fltration site.

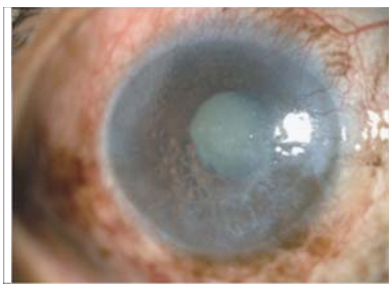


Fig. 2B : Chandler's syndrome

With GDDs the growth of ICE membrane over the ostium is bypassed. A long-term study of 10 patients with ICE syndrome[42] showed IOP control of less than 21 mm Hg in four patients and in three required tube repositioning due to membrane formation. They proposed that in ICE patients the tube should be left long in the anterior chamber away from the potential source of ICE cells and entry through pars plana can be used in pseudophakicvitrectomized eyes. Cyclophotocoagulation is indicated in eyes with refractory glaucoma who have failed trabeculectomy or shunt surgery.

Infammatory Glaucoma

Glaucoma is a potentially devastating complication of uveitis and remains a therapeutic challenge despite availability of new modalities of treatment in both the conditions.[43]

Inflammation can produce secondary angle closure glaucoma with pupillary block mechanism due to posterior synechiae formation or without pupillary block due to infam matory peripheral iris swelling, exudates in the angle contracting to form PAS or due to forward rotation of the ciliary body. PAS form easily in eyes with shallow anterior chamber and in eyes with chronic infammatory processes.

Infammatory glaucoma can occur after trauma, surgery, idiopathic infammatory condition or due to specific uveitic entities.

Principles of Management

Usually control of infammation alone leads to normalization of IOP. Management of infammation is done by topical or systemic corticosteroids, mydriatics-cycloplegics and systemic immunosuppressives. Medical therapy to control IOP basically involves aqueous suppressants. Intracameral tissue plasminogen activator has been reported to be of use in impending pupillary block glaucoma due to acute fibrin-ous HLA-B27 uveitis.[44]

YAG PI is done for pupillary block glaucomas, although fibrin can close small iridotomies in an infamed eye.[45] When this is unsuccessful surgical iridectomy is useful if less than 75% of

the angle is closed by PAS.[46]

If medical or laser therapy is unable to control the IOP then fltration surgery has to be considered. Trabeculectomy in uveitic eyes generally carries less success due to acceleration of wound healing by postoperative fibrinous and cellular responses.[47] Antimetabolites like 5FU and Mitomycin C are used to increase the success of trabeculectomy in uveitic eyes (5FU, MMC). A success of 95% (IOP less than 21 mm Hg with 1 or no medication) was achieved with MMC augmented trabeculectomy.[48] Aggressive anti-infammatory therapy should be given before and after trabeculectomy in these patients.

All the three glaucoma drainage implants (Molteno, Baerveldt and Ahmed valve) have been tried with a success of 79% with Molteno implants,[49] 94% with Ahmed valve[50,51] and 91.7% with Baerveldt implant.[52]

Trabeculodialysis[53] (a modifed goniotomy) was studied in children with infammatory glaucomas with a success of 60%. Goniotomy in chronic childhood uveitis was reported to be successful in 75% of their patients by Freedman et al.[54]

Trans scleral diode cyclophotocoagulation is considered in low visual potential eyes; however, the rate of hypotony in uveitis eyes can be higher (19%) than other refractory glaucomas.[55]

Ciliary Body Cysts

Benign iris/ciliary body cysts can cause glaucoma if they are multiple by pushing the peripheral iris forward to close the angle and cause raised intraocular pressure. The onset of glaucoma can mimic acute angle closure glaucoma. It is also called as pseudoplateau iris since it produces a clinical picture identical to plateau iris. Cysts should be suspected when plateau iris configuration appears more in one segment of the angle giving a characteristic bumpy contour to the peripheral iris. They are often multiple and diffusely distri-buted.[56] Around 10% of the iris cysts can because glaucoma associated with or without pupillary block glaucoma. They can be confirmed and their extent can be noted by UBM.[57]

Laser iridotomy,[58] Argon laser iridoplasty,[59] Laser cystostomy and intermittent pilocarpine therapy[60-62] have been described as therapeutic approaches.

Glaucoma following Scleral Buckling Procedures

Here angle closure glaucoma is produced by swelling of the ciliary body due to impaired venous drainage from the vortex veins by the scleral buckle. The incidence of angle closure glaucoma after scleral buckling procedures range from 1.4 to 4.4%.[63] The risk factors are pre-existing narrow angles, use of an encircling band, placement of the band anterior to the equator and high myopia.

It usually resolves spontaneously over several days to weeks. Cycloplegics are used to shift the lens-iris diaphragm posteriorly by relaxing the ciliary muscle and topical steroids to reduce the infammation. IOP is reduced by means of aqueous suppressants. Miotics should be avoided. Laser PI is not beneficial; rather laser iridoplasty is useful in opening the angle in some of these patients.[64]

GDDs offer an alternative approach in uncontrolled glaucoma as evidenced by the study done by Scott Ingrid U et al with Baerveldt glaucoma implant in eyes with preexisting episcleral bands where IOP control was achieved in all 16 patients with or without medications during the follow-up period of 19.1 to 45.5 months with none of the usual complications reported with glaucoma drainage devices (GDD).[65]

Intraocular gases have been increasingly used in vitreo-retinal surgeries for tamponade effect on the retinal breaks. The gases used are air, Perfluoropropane (C3F8), sulphur hexafluoride (SF6). SF6[66] expands to twice its volume within 24 to 48 hours and stays in the eye for 10 to 14 days, C3F8 expands to four times its volume in 48 to 72 hours and stays for 55 to 65 days.[67] The increase in the volume of the gas causes anterior displacement of the lens-iris diaphragm even when prone position is maintained. The IOP rise is high during the period of maximum expansion of gas. The incidence of IOP elevation

with SF6 has been reported to range from 6.1 to 67%, maximum with 100% SF6 and minimum with 20% SF6.[68] C3F8 causes IOP elevation of 18 to 59%, minimum when 14% of C3F8 is used.[69] Patients should be instructed to maintain head down position to prevent forward movement of iris lens diaphragm. They should be warned against air travel due to IOP variation during atmospheric changes.[70]

Medical therapy consists of aqueous suppressants. If IOP is not responsive to medical therapy then aspiration of a portion of gas may be performed to lower IOP. Laser PI is useful in pupillary block.

Glaucoma after Silicone Oil Injection

Silicone oil is used as a vitreous substitute for retinal tampo-nade. It can produce glaucoma by pupillary block, infa-mmation, synechial closure, neovascularization, migration of oil into anterior chamber or by open angle mechanism. Secondary glaucoma after silicone oil injection has been reported to be in 6 to 30% of eyes.[71] Angle closure glaucoma can occur after silicone oil injection due to synechial closure. Overflling of the oil in the eye has to be avoided to prevent secondary glaucoma. Inferior prophylactic iridectomy prevents pupillary block in pseudophakic and aphakic eyes. Medical therapy consists of aqueous suppressants, cortico-steroids and cycloplegics. Postoperative closed iridectomies due to fibrin can be relieved by laser PI.

Silicone oil removal[72] with or without concurrent glaucoma surgery has been performed to lower IOP, but oil removal carries some risk of retinal detachment. In patients with complete synechial closure silicone oil removal alone cannot be expected to lower IOP, rather glaucoma surgery is indicated in such cases and the decision to remove silicone oil or not depends on the relative risk of redetachment on oil removal and emulsification of oil.[73] If silicone oil remains in the eye, the GDD should be positioned in the inferior quadrant.[74]

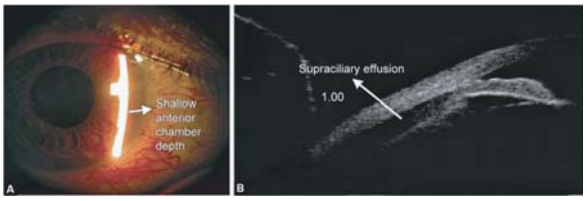
Cyclophotocoagulation by transscleral cytophotoco-agulation (TSC) or endo cytophotocoagulation (ECP) can be used[75] in refractory cases. Eyes with relatively intact central visual acuity are appropriate candidates for ECP and can be combined with intraocular surgery.[76]

Ciliary Body Swelling

Secondary angle closure glaucoma can occur due to ciliary body (CB) swelling secondary to vortex vein obstruction (venous stasis), drugs or infammation.

Vortex vein obstruction occurs secondary to extensive PRP or CRVO producing angle closure glaucoma similar to scleral buckling procedures. It is a self limiting condition and will resolve with or without medical therapy. Laser PI has no role in this condition.

Ciliary body swelling secondary to drug use is an idiosyncratic reaction causing relaxation of zonules, anterolateral rotation of the ciliary body leading to anterior displacement of iris–lens diaphragm producing induced myopia. Cho-roidal detachment and supraciliary effusions are frequently present detected by UBM. Sulfa-based drugs are associated with this type of secondary angle closure glaucomas like topiramate[77] (Figs 3A and B), acetazolamide,[78] hydrochloro-thiazide and contrimoxazole. It presents as bilateral angle closure glaucoma. The management requires stopping of the drug, instituting aqueous suppressants and cycloplegics.[79] Miotics, topical and systemic carbonic anhydrase inhibitors are contraindicated and laser PI has no role.



Figs 3A and B : (A) Shallow anterior chamber depth due to topiramate induced angle closure, (B) UBM showing supraciliary effusion following topiramate intake causing secondary angle closure

Conclusion

Secondary angle closure glaucomas require

meticulous history taking, clinical examination, if necessary anterior segment imagings like UBM to identify the specific cause if possible and formulate a treatment plan to avoid ocular morbidity.

References

1. Luntz MH, Rosenblatt M. Malignant glaucoma. SurvOphthal-mol. 1987 Sep-Oct;32(2):73–93. [PubMed] [Google Scholar]
2. Harbour JW, Rubsamen PE, Palmberg P. Pars plana vitrectomy in the management of phakic and pseudophakic malignant glaucoma. Arch Ophthalmol. 1996 Sep;114(9):1073–1078. [PubMed] [Google Scholar]
3. Lynch MG, Brown RH, Michels RG, Pollack IP, Stark WJ. Surgical vitrectomy for pseudophakic malignant glaucoma. Am J Ophthalmol. 1986 Aug;102(2):149–153. [PubMed] [Google Scholar]
4. DiSclafani M, Liebmann JM, Ritch R. Malignant Glaucoma following argon laser release of scleral flap sutures after trabeculectomy. Am J Ophthalmol. 1989 Nov;108(5):597–598. [PubMed] [Google Scholar]
5. Greenfield DS, Tello C, Budenz DL, Liebmann JM, Ritch R. Aqueous misdirection after glaucoma drainage device implantation. Ophthalmology. 1999 May;106(5):1035–1040. [PubMed] [Google Scholar]
6. Cashwell LF, Martin TJ. Malignant glaucoma after laser iridotomy. Ophthalmology. 1992 May;99(5):651–659. [PubMed] [Google Scholar]
7. Epstein DL. The malignant glaucoma syndromes. In: Epstein DL, Allingham RR, Schuman JS, editors. Chandler and Grant's Glaucoma. 4th ed. Baltimore: Williams & Wilkins; 1997. pp. 285–303. [Google Scholar]
8. Lieberman MF., Lee DA. Diagnosis and management of malignant glaucoma. In: In Higginbotham EJ, Lee DA, editors. Clinical guide to glaucoma management, Woburn, MA, Butterworth Heinemann/ Elsevier, 2004. [Google Scholar]
9. Trope GE, Pavlin CJ, Bau A, Baumal CR, Foster FS. Malignant glaucoma: Clinical and ultrasound biomicroscopic features. Ophthalmology. 1994 Jun;101(6):1030–1335. [PubMed] [Google Scholar]
10. Chandler PA, Grant WM. Mydriatic-cycloplegic treatment in malignant glaucoma. Arch Ophthalmol. 1962 Sep;68:353–359. [PubMed] [Google Scholar]
11. Sharma A, Sii F, Shah P, Kirkby GR. Vitrectomy-phacoemulsif cation-vitrectomy for the management of aqueous misdirection syndromes in phakic eyes. Ophthalmology. 2006 Nov;113(11):1968–1973. [PubMed] [Google Scholar]
12. Stumpf TH, Austin M, Bloom PA, McNaught A, Morgan JE. Transscleral cyclodiode laser photocoagulation in the treatment of aqueous misdirection syndrome. Ophthalmology. 2008 Nov;115(11):2058–2061. [PubMed] [Google Scholar]
13. Sivak-Callcott JA, O' Day DM, Gass JD, Tsai JC. Evidence-based recommendations for the diagnosis and treatment of neo vascular glaucoma. Ophthalmology. 2001 Oct;108(10):1767–1776. [PubMed] [Google Scholar]
14. Tripathi RC, Li J, Tripathi BJ, Chalam K V, Adamis AP. Increased level of vascular endothelial growth factor in aqueous humour of patients with neovascular glaucoma. Ophthalmology. 1998 Feb;105(2):232–237. [PubMed] [Google Scholar]
15. Allingham RR., Karim F., Sharon DF . Shields Textbook of Glaucoma. New Delhi, India: Wolters Kluwer Pvt Ltd; 2011; Chapter 19, Glaucoma associated with disorder of the retina, vitreous and choroid. [Google Scholar]
16. Browning DJ. Risk of missing angle neovascularization by omitting angle screening gonioscopy in patients with diabetes mellitus. Am J Ophthalmol. 1991 Aug 15;112(2):212. [PubMed] [Google Scholar]
17. Blinder KJ, Friedman SM, Mames RN. Diabetic iris neovascularization. Am J Ophthalmol. 1995 Sep;120(3): 393–395. [PubMed] [Google Scholar]
18. Browning DJ, Scott AQ, Peterson CB, Warnock J, Zhang Z. The risk of missing angle neovascularization by omitting angle screening gonioscopy in acute central retinal vein occlusion. Ophthalmology. 1998 May;105(5):776–784. [PubMed] [Google Scholar]
19. Little HL, Rosenthal AR, Dellaporta A, Jacobson DR. The effect of pan–retinal photo-coagulation on rubeosis iridis. Am J Ophthalmol. 1976 Jun;81(6):804–809. [PubMed] [Google Scholar]
20. Flanagan DW, Blach RK. Place of panretinal photocoagulation and trabeculectomy in the management of neovascular glaucoma. Br J Ophthalmol. 1983 Aug;67(8):526–528. [PMC free article] [PubMed] [Google Scholar]
21. Goodart R, Blankenship G. Panretinal photocoagulation infuence on vitrectomy results for complications of diabetic retinopathy. Ophthalmology. 1980 Mar;87(3):183–188. [PubMed] [Google Scholar]
22. Takihara Y, Inatani M, Fukushima M, Iwao K, Iwao M, Tanihara H. Trabeculectomy with mitomycin C for neovascular glaucoma: prognostic factors for surgical failure. Am J Ophthalmol. 2009 May;147(5):912–918. [PubMed] [Google Scholar]
23. Herschler J, Agness D. A modified filtering operation for neovascular glaucoma. Arch Ophthalmol. 1979 Dec;97(12):2339–2341. [PubMed] [Google Scholar]
24. Duch S, Buchacra O, Milla E, Andreu D, Tellez J. Intracameral bevacizumab (Avastin) for neovascular glaucoma: a pilot study in 6 patients. J Glaucoma. 2009 Feb;18(2):140–143. [PubMed] [Google Scholar]
25. Fakhraie G, Katz JL, Prasad A, Eslami Y, Sabour S, Zarei R, Moghimi S. Surgical outcomes of intravitreal bevacizumab and guarded fltration surgery in neovascular glaucoma. J Glaucoma. 2010 Mar;19(3):212–218. [PubMed] [Google Scholar]
26. Takihara Y, Inatani M, Kawaji T, Fukushima M, Iwao K, Iwao M, Tanihara H. Combined intravitreal bevacizumab and trabeculectomy with mitomycin C versus trabeculectomy with

mitomycin C alone for neovascular glaucoma. J Glaucoma. 2011 Mar;20(3):196–201. [PubMed] [Google Scholar]

27. Hong CH, Arosemena A, Zurakowski D, Ayyala RS. Glaucoma drainage devices: a systematic literature review and current controversies. SurvOphthalmol. 2005 Jan-Feb;50(1):48–60. [PubMed] [Google Scholar]

28. Assaad MH, Baerveldt G, Rockwood EJ. Glaucoma drainage devices: pros and cons. CurrOpinOphthalmol. 1999 Apr;10(2):147–153. [PubMed] [Google Scholar]

29. Mermoud A, Salmon JF, Alexander P, Straker C, Murray AD. Molteno tube implantation for neovascular glaucoma. Long-term results and factors infuencing the outcome. Ophthalmology. 1993 Jun;100(6):897–902. [PubMed] [Google Scholar]

30. Park UC, Park HK, Kim MD, Yu HG. Ahmed glaucoma valve implantation for neovasuclar glaucoma after vitrectomy for proliferative diabetic retinopathy. J Glaucoma. 2011 Sep;20(7):433–438. [PubMed] [Google Scholar]

31. Sidoti PA, Dunphy TR, Baerveldt G, LaBree L, Minckler DS, Lee PP, Heuer DK. Experience with the Baerveldt glaucoma implant in treating neovascular glaucoma. Ophthalmology. 1995 Jul;102(7):1107–1118. [PubMed] [Google Scholar]

32. Scott IU, Alexandrakis G, Flynn HW Jr, Smiddy WE, Murray TG, Schiffman J, Gedde SJ, Budenz DL, Fantes F, Parrish RK. Combined pars plana vitrectomy and glaucoma drainage implant placement for refractory glaucoma. Am J Ophthalmol. 2000 Mar;129(3):334–341. [PubMed] [Google Scholar]

33. Bloom PA, Tsai JC, Sharma K, Miller MH, Rice NS, Hitchings RA, Khaw PT. "Cyclodiode". Trans-scleral diode laser cyclophotocoagulation in the treatment of advanced refractory glaucoma. Ophthalmology. 1997 Sep;104(9):1508–1520. [PubMed] [Google Scholar]

34. Ramli N, Htoon HM, Ho CL, Aung T, Perera S. Risk factors for hypotony after transscleral diode cyclophotocoagulation. J Glaucoma. 2012 Mar;21(3):169–173. [PubMed] [Google Scholar]

35. Eagle RC Jr, Font RL, Yanoff M, Fine BS. Proliferative endotheliopathy with iris abnormalities. The iridocorneal endothelial syndrome. Arch Ophthalmol. 1979 Nov;97(11):2104–2111. [PubMed] [Google Scholar]

36. Campbell DG, Shields MB, Smith TR. The corneal endothelium and the spectrum of essential iris atrophy. Am J Ophthalmol. 1978 Sep;86(3):317–324. [PubMed] [Google Scholar]

37. Laganowski HC, Kerr Muir MG, Hitchings RA. Glaucoma and the iridocorneal endothelial syndrome. Arch Ophthalmol. 1992 Mar;110(3):346–350. [PubMed] [Google Scholar]

38. Patel A, Kenyon KR, Hirst LW, Quigley HA, Stark WJ, Meyer RF, Green WR. Clinicopathologic features of Chandler's syndrome. SurvOphthalmol. 1983 Mar-Apr;27(5):327–344. [PubMed] [Google Scholar]

39. Kidd M, Hetherington J, Magee S. Surgical results in Iridocorneal endothelial syndrome. Arch Ophthalmol. 1988 Feb;106(2):199–201. [PubMed] [Google Scholar]

40. Wright MM, Grajewski AL, Cristol SM, Parrish RK. 5-Fluorouracil after trabeculectomy and the iridocorneal endothelial syndrome. Ophthalmology. 1991 Mar;98(3):314–316. [PubMed] [Google Scholar]

41. Lanzl IM, Wilson RP, Dudley D, Augsburger JJ, Aslanides IM, Spaeth GL. Outcome of trabeculectomy with mitomycin-C in the iridocorneal endothelial syndrome. Ophthalmology. 2000 Feb;107(2):295–297. [PubMed] [Google Scholar]

42. Kim DK, Aslanides IM, Schimdt CM Jr, Spaeth GL, Wilson RP, Augsburger JJ. Long-term outcome of aqueous shunt surgery in 10 patients with iridocorneal endothelial syndrome. Ophthalmology. 1999 May;106(5):1030–1034. [PubMed] [Google Scholar]

43. Moorthy RS, Mermoud A, Baerveldt G, Minckler DS, Lee PP, Rao NA. Glaucoma associated with uveitis. SurvOphthalmol. 1997 Mar-Apr;41(5):361–394. [PubMed] [Google Scholar]

44. Skolnick CA, Fiscella RG, Tessler HH, Goldstein DA. Tissue plasminogen activator to treat impending pupillary block glaucoma in patients with acute fibrinous HLA-B27 positive iridocyclitis. Am J Opththalmol. 2000 Mar;129(3):363–366. [PubMed] [Google Scholar]

45. Schwartz LW, Moster MR, Spaeth GL, Wilson RP, Poryzees E. Neodymium-YAG laser iridectomies in glaucoma associated with closed or occludable angles. Am J Ophthalmol. 1986 Jul;102(1):41–44. [PubMed] [Google Scholar]

46. Kanski JJ., McAllister JA. Glaucoma associated with uveitis diagnosis and therapy. In: Cairns JE, editor. Glaucoma. Vol. 11. London: Grune and Stratton; 1986. pp. 851–859. [Google Scholar]

47. Skuta GL, Parrish RK 2nd. Wound healing in glaucoma fltering surgery. SurvOphthalmol. 1987 Nov-Dec;32(3):149–170. [PubMed] [Google Scholar]

48. Prata JA Jr, Neves RA, Minckler DS, Mermoud A, Heuer DK. Trabeculectomy with mitomycin C in glaucoma associated with uveitis. Ophthalmic Surg. 1994 Sep-Oct;25(9):616–620. [PubMed] [Google Scholar]

49. Hill RA, Nguyen QH, Baerveldt G, Forster DJ, Minckler DS, Rao N, Lee M, Heuer DK. Trabeculectomy and Molteno implantation for glaucomas associated with uveitis. Ophthalmology. 1993 Jun;100(6):903–908. [PubMed] [Google Scholar]

50. Gil-Carrasco F, Salinas-VanOrman E, Recillas-Gispert C, Paczka JA, Gilbert ME, Arellanes-García L. Ahmed valve implant for uncontrolled uveitic glaucoma. Ocul Immunol Infamm. 1998 Mar;6(1):27–37. [PubMed] [Google Scholar]

51. Da Mata A, Burk SE, Netland PA, Baltatzis S, Christen W, Foster CS. Management of uveitic glaucoma with Ahmed glaucoma valve implantation. Ophthalmology. 1999 Nov;106(11):2168–2172. [PubMed] [Google Scholar]

52. Ceballos EM, Parrish RK 2nd, Schiffman JC. Outcome of Baerveldt glaucoma drainage im plants for the treatment of uveitic glaucoma. Ophthalmology. 2002 Dec;109(12): 2256–2260. [PubMed] [Google Scholar]

53. Kanski JJ, McAllister JA. Trabeculodialysis for infammatory glaucoma in children and young adults. Ophthalmology. 1985 Jul;92(7):927–930. [PubMed] [Google Scholar]

54. Freedman SF, Rodriguez-Rosa RE, Rojas MC, Enyedi LB. Goniotomy for glaucoma secondary to chronic childhood uveitis. Am J Ophthalmol. 2002 May;133(5):617–621. [PubMed] [Google Scholar]

55. Murphy CC, Burnett CA, Spry PG, Broadway DC, Diamond JP. A two centre study of the dose-response relation for transscleral diode laser cyclophotocoagulation in refractory glaucoma. Br J Ophthalmol. 2003 Oct;87(10):1252–1257. [PMC free article] [PubMed] [Google Scholar]

56. Azuara-Blanco A, Spaeth GL, Araujo SV, Augsburger JJ, Terebuh AK. Plateau iris syndrome associated with multiple ciliary body cysts. Report of three cases. . Arch Ophthalmol. 1996 Jun;114(6):666–668. [PubMed] [Google Scholar]

57. Ritch R, Liebmann JM. Role of ultrasound biomicroscopy in the differentiation of block glaucomas. CurrOpinOphthalmol. 1998 Apr;9(2):39–45. [PubMed] [Google Scholar]

58. Bron AJ, Wilson CB, Hill AR. Laser treatment of primary ring-shaped epithelial iris cyst. Br J Ophthalmol. 1984 Dec;68(12):859–865. [PMC free article] [PubMed] [Google Scholar]

59. Crowston JG, Medeiros FA, Mosaed S, Weinreb RN. Argon laser iridoplasty in the treatment of plateau-like iris configuration as result of numerous ciliary body cysts. Am J Ophthalmol. 2005 Feb;139(2):381–383. [PubMed] [Google Scholar]

60. Lois N, Shields CL, Shields JA, Mercado G. Primary cysts of the iris pigment epithelium. Clinical features and natural course in 234 patients. Ophthalmology. 1998 Oct;105(10):1879–1885. [PubMed] [Google Scholar]

61. Vela A, Rieser JC, Campbell DG. The heredity and treatment of angle-closure glaucoma secondary to iris and ciliary body cysts. Ophthalmology. 1984 Apr;91(4):332–337. [PubMed] [Google Scholar]

62. Kuchenbecker J, Motschmann M, Schmitz K, Behrens-Baumann W. Laser iridocystotomy for bilateral acute angle-closure glaucoma secondary to iris cysts. Am J Ophthalmol. 2000 Mar;129(3):391–393. [PubMed] [Google Scholar]

63. Kreiger AE, Hodgkinson BJ, Frederick AR Jr, Smith TR. The results of retinal detachment surgery. Analysis of 268 operations with a broad scleral buckle. Arch Ophthalmol. 1971 Oct;86(4):385–394. [PubMed] [Google Scholar]

64. Burton TC, Folk JC. Laser iris retraction for angle-closure glaucoma after retinal detachment surgery. Ophthalmology. 1988 Jun;95(6):742–748. [PubMed] [Google Scholar]

65. Scott IU, Gedde SJ, Budenz DL, Greenfield DS, Flynn HW Jr, Feuer WJ, Mello MO Jr, Krishna R, Godfrey DG. Baerveldt drainage implants in eyes with a preexisting scleral buckle. Arch Ophthalmol. 2000 Nov;118(11):1509–1513. [PubMed] [Google Scholar]

66. Chang S. Intraocular gases. In: Ryan SJ, editor. Retina. St Louis, MO: Mosby; 1994. pp. 2115–2129. [Google Scholar]

67. Lincoff A, Haft D, Liggett P, Reifer C. Intravitreal expansion of perfluorocarbon bubbles. Arch Ophthalmol. 1980 1646 Sep;98(9) [PubMed] [Google Scholar]

68. Lean JS, Boone DC, Azen SP et al. Vitrectomy with silicone oil or sulfur hexafluoride gas in eyes with severe proliferative vitreoretinopathy: Results of a randomized clinical trial. Silicone Study Report 1. Arch Ophthalmol. 1992 Jun;110(6):770–779. [PubMed] [Google Scholar]

69. McCuen B, Azen SP, Boone DC et al. Vitrectomy with silicone oil or perfluoropropane gas in eyes with severe proliferative vitreoretinopathy: results of a randomized clinical trial. Silicone Study Report 2. Arch Ophthalmol. 1992 Jun;110(6):780–792. [PubMed] [Google Scholar]

70. Mills MD, Devenyi RG, Lam WC, Berger AR, Beijer CD, Lam SR. An assessment of intraocular pressure rise in patients with gas-filled eyes during simulated air fight. Ophthalmology. 2001 Jan;108(1):40–44. [PubMed] [Google Scholar]

71. Honavar SG, Goyal M, Majji AB, Sen PK, Naduvilath T, Dandona L. Glaucoma after pars plana vitrectomy and silicone oil injection for complicated retinal detachments. Ophthalmology. 1999 Jan;106(1):169–177. [PubMed] [Google Scholar]

72. Budenz DL, Taba KE, Feuer WJ, Eliezer R, Cousins S, Henderer J, Flynn HW Jr. Surgical management of secondary glaucoma after pars plana vitrectomy and silicone oil injection for complex retinal detachment. Ophthalmology. 2001 Sep;108(9):1628–1632. [PubMed] [Google Scholar]

73. Nguyen QH, Lloyd MA, Heuer DK, Baerveldt G, Minckler DS, Lean JS, Liggett PE. Incidence and management of glaucoma after intravitreal silicone oil injection for complicated retinal detachments. Ophthalmology. 1992 Oct;99(10):1520–1526. [PubMed] [Google Scholar]

74. Gedde SJ. Management of glaucoma after retinal detachment surgery. CurrOpinOphthalmol. 2002 Apr;13(2):103–109. [PubMed] [Google Scholar]

75. Lima FE, Magacho L, Carvalho DM, Susanna R Jr, Avila MP. A prospective, comparative study between endoscopic cytophotocoagulation and the Ahmed drainage implant in refractory glaucoma. J Glaucoma. 2004 Jun;13(3):233–237. [PubMed] [Google Scholar]

76. Uram M. Combined phacoemulsification, endoscopic ciliary process photocoagulation and intraocular lens implantation in glaucoma management. Ophthalmic Surg. 1995 Jul-Aug;26(4):346–352. [PubMed] [Google Scholar]

77. Fraunfielder FW, Fraunfielder FT, Keates EU. Topiramate-associated acute, bilateral, secondary ange-closure glaucoma. Ophthalmology. 2004 Jan;111(1):109–111. [PubMed] [Google Scholar]

78. Narayanaswamy AK, Antrolikar M, Vijaya L. Acetazolamide-induced glaucoma. Asian J Ophthalmol. 2007;9(5):213–215. [Google Scholar]

79. Lachkar Y, Bouassida W. Drug-induced acute angle closure glaucoma. CurrOpinOphthalmol. 2007 Mar;18(2):129–133. [PubMed] [Google Scholar]

Case Report: Trabebeculectomy and cataract surgery in a patient with primary open angle glaucoma and high myopia

M Z Hassan[1], R B Ahsan[2], B K D Sarker[3], T R Chhara[4], M I Iqbal[5]

Abstract

The rising prevalence of myopia presents significant challenges in glaucoma management. We report a 50-year-old male with hypertension and diabetes who underwent combined cataract and trabeculectomy due to primary open-angle glaucoma and advanced myopia. Postoperatively, his visual acuity remained 6/36, with an IOP of 16 mmHg. This case highlights the complexities of surgery in high myopia, emphasizing the need for meticulous planning to improve patient outcomes.

Introduction

The rising prevalence of myopia, particularly among younger populations due to lifestyle changes and increased near work, poses significant challenges in ophthalmic practice[1]. The association between myopia and primary open-angle glaucoma (POAG) is well documented, with numerous studies indicating that the risk of POAG increases amyopia worsens. For instance, the Blue Mountains Eye Study found that low myopes (1.0 D to 3.0 D) are twice as likely to develop glaucoma compared to non-myopes, while those with moderate-to-high myopia (3.0 D or worse) face a threefold increase in risk [2].

This case report details the clinical presentation, surgical approach, and outcomes of a 50-year-old male patient with hypertension, diabetes, and advanced myopia undergoing combined cataract and trabeculectomy surgery.

Case Report:

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A 50-year-old hypertensive and diabetic male presented with a three-month history of gradually worsening vision in both eyes, unresponsive to frequent changes in spectacles, accompanied by headaches. His medical history included a diagnosis of primary open-angle glaucoma (POAG), for which he was on three anti-glaucoma medications.

At presentation, his best-corrected visual acuity (BCVA) was 6/36 in both eyes, with refractive errors of -14.00 D in the right eye and -13.00 D in the left eye. Intraocular pressure (IOP) measurements were 23 mmHg in the right eye and 18 mmHg in the left eye, despite the patient being treated with four anti-glaucoma medications (Dorzolamide 2%, Timolol 0.5%, Brinzolamide 1%, and Tafluprost 0.001%).

Fundoscopic examination revealed cupping of 0.9:1 in the right eye and 0.85:1 in the left eye, along with peripapillary atrophy in both eyes. Additionally, both eyes exhibited posterior subcapsular (PSC) and nuclear sclerosis (NSII) cataracts. The intraocular lens (IOL) power was calculated at +7.00 D, with an axial length of 27.10 mm measured by IOL Master.

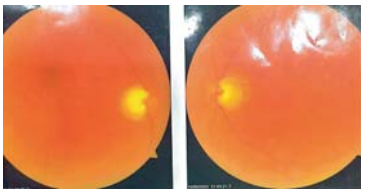
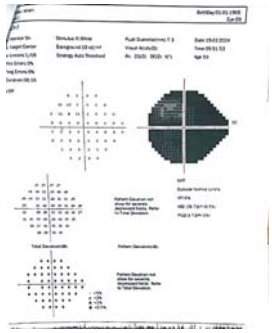
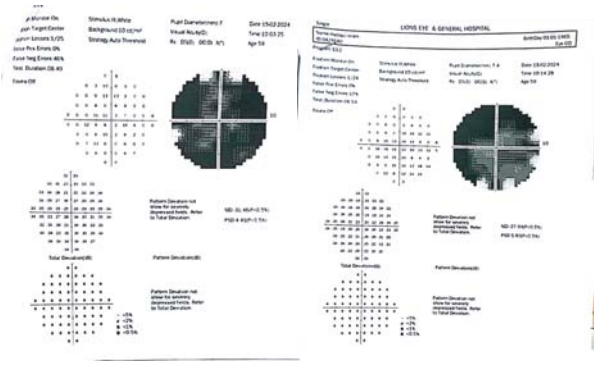
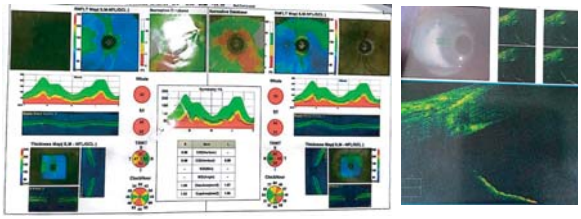


Fig: Colour fundus photography







Surgical Intervention:

Given the advanced glaucoma and cataracts, combined surgery was recommended for the right eye, followed by the left eye, under mannitol coverage to manage IOP though this procedure carries significant risks of both peroperative and postoperative challenges and complications.

We did the procedure under topical anaesthesia avoiding peribulbar block as central fixation was threatened in 10'-2 field. We Used Mitomycin C (0.02%) to prevent excessive postoperative scarring and thus reduce the risk of failure.

During the surgery, the surgeon encountered several challenges, including elevated IOP and difficulties in creating a scleral flap due to the thin sclera associated with high myopia. A small hexagonal flap was fashioned, and the anterior chamber was notably deep due to the myopic condition.The flap was stitched tightly

Postoperative Outcome

On the first postoperative day (POD), the BCVA in the operated eye remained 6/36, and the IOP was measured at 16 mmHg without any anti-glaucoma medications. No abnormalities were detected in the macular region during examination.

Discussion

When considering surgical interventions for glaucoma in high myopic patients, the balance between safety and efficacy becomes paramount. Trabeculectomy, while a common choice, presents unique challenges in this demographic. The inherent scleral thinning associated with high myopia increases the risk of hypotony-related complications following surgery. Notably, hypotony maculopathy can manifest years after the procedure, even in the absence of a bleb leak .[3]

The presence of cataracts further complicates the surgical landscape. In combined cataract and trabeculectomy procedures, careful planning is essential. Surgeons must counsel patients about the heightened risk of intraoperative and postoperative complications, including unexpected refractive outcomes. Accurate axial length measurement is critical; studies indicate that ultrasound biometry can overestimate axial length in eyes with posterior staphylomas, leading to hyperopic surprises[4]. Optical biometry, when feasible, often yields more reliable results [5].

The selection of intraocular lenses (IOLs) is also crucial. Newer calculation formulas, such as the Barrett Universal II and Kane formulas, have shown promise in improving refractive accuracy for high myopic eyes[6]. Adjustments to standard IOL formulas may be necessary in patients with a history of refractive surgery, and intraoperative aberrometry can provide additional guidance for achieving optimal outcomes.

High myopic patients are at increased risk for retinal detachment post-cataract surgery, making thorough preoperative retinal assessments essential[7]. During surgery, maintaining the anterior chamber through

careful wound construction and the use of viscoelastic substances is vital to prevent sudden decompression. Employing techniques that reduce stress on the zonules, such as a chopping approach for nuclear disassembly, is advisable. Surgeons should remain vigilant for signs of lens–iris diaphragm retropulsion syndrome, which can be exacerbated in myopic eyes[8].

Finally, meticulous irrigation and aspiration techniques are crucial to prevent postoperative intraocular pressure spikes, and anterior capsular polishing may help reduce the risk of anterior capsular contraction syndrome, which is more common in myopic eyes [9]. By addressing these multifaceted considerations, surgeons can enhance outcomes in high myopic patients undergoing trabeculectomy, ultimately improving the management of glaucoma in this challenging cohort.

Conclusion

As myopia prevalence increases globally, it is essential to enhance our understanding of its complex relationship with glaucoma to reduce functional vision loss in myopic eyes. Myopic eyes are at higher risk for intraoperative and postoperative complications during cataract surgery, necessitating careful surgical planning and modifications to standard phacoemulsification techniques to optimize outcomes.

References

1. Holden B.A., Fricke T.R., Wilson D.A., Jong M., Naidoo K.S., Sankaridurg P., Wong T.Y., Naduvilath T.J., Resnikoff S. Global Prevalence of Myopia and High Myopia and Temporal Trends from 2000 through 2050. Ophthalmology. 2016;123:1036–1042. doi: 10.1016/j.ophtha.2016.01.006.
2. Knapp A. Glaucoma in Myopic Eyes. Trans. Am. Ophthalmol. Soc.
3. Fong D.S., Epstein D.L., Allingham R.R. Glaucoma and Myopia: Are They Related? Int. Ophthalmol. Clin. 1990;30:215–218. doi: 10.1097/00004397-199030030-00009.
4. Xu L., Wang Y., Wang S., Wang Y., Jonas J.B. High Myopia and Glaucoma Susceptibility the Beijing Eye Study. Ophthalmology. 2007;114:216–220. doi: 10.1016/j.ophtha.2006.06.050.
5. Abe RY, Oltramari L, Vessani RM, Schimiti RB, Magacho L, Kanadani FN, Costa VP. Trabeculectomy in Eyes With High Myopia. J Glaucoma. 2024 Feb 1;33(2):110-115. doi: 10.1097/IJG.0000000000002293. Epub 2023 Aug 17. PMID: 37671507.
6. Zhang ML, Hirunyachote P, Jampel H. Combined surgery versus cataract surgery alone for eyes with cataract and glaucoma. Cochrane Database Syst Rev. 2015 Jul 14;2015(7):CD008671. doi: 10.1002/14651858.CD008671.pub3. PMID: 26171900; PMCID: PMC4730948.
7. Tanaka D, Nakanishi H, Hangai M, Akagi T, Morooka S, Ikeda HO, Yoshimura N. Influence of high myopia on outcomes of trabeculectomy with mitomycin C in patients with primary open-angle glaucoma. Jpn J Ophthalmol. 2016 Nov;60(6):446-453. doi: 10.1007/s10384-016-0468-7. Epub 2016 Aug 12. PMID: 27517556.
8. Koh V, Chen D, Aquino CM, Aduan J, Sng C, Chee LS, Chew P. Success rates of 2-site phacoemulsification combined with fornix-based trabeculectomy using mitomycin C for primary angle-closure glaucoma and primary open-angle glaucoma in an Asian population. Jpn J Ophthalmol. 2017 May;61(3):245-252. doi: 10.1007/s10384-017-0502-4. Epub 2017 Feb 10. PMID: 28188405.
9. Lai JS, Tham CC, Chan JC, Lam DS. Phacotrabeculectomy in treatment of primary angle-closure glaucoma and primary open-angle glaucoma. Jpn J Ophthalmol. 2004 Jul-Aug;48(4):408-11. doi: 10.1007/s10384-003-0075-2. PMID: 15295672.

Battling neovascular glaucoma: A case report

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Abstract

Purpose: To report a case of neovascular glaucoma (NVG) managed with trabeculectomy along with suppressing the ongoing ischemic process.

Case: A 39yrs old patient with uncontrolled diabetes, presented with right eye neovascular glaucoma and intraocular pressure of 55mmHg, which came down to 38mmHg with antiglaucoma medications. He underwent right eye combined phacoemulsification with intraocular lens (IOL) implantation with trabeculectomy with Mitomycin C (MMC), along with intravitreal Anti VEGF (Bevacizumab) injection on the same sitting. On 2nd and 3rd post operative week, he received panretinal photocoagulation (PRP) using laser indirect ophthalmoscope. IOP was under control after the procedure.

Conclusion: Trabeculectomy may be successful in NVG patient to reduce IOP to desirable levels, with proper pre and post operative management especially combatting the ongoing ischemic process at the same time.

Keywords: Neovascular glaucoma, Trabeculectomy, Intraocular pressure, panretinal photocoagulation (PRP), Anti-Vascular Endothelial Growth Factor (Anti-VEGF) injections.

Introduction

Neovascular glaucoma (NVG) is a type of secondary glaucoma resulting from the neovascularisation of the angle due to posterior segment ischemia, causing elevation of intraocular pressure (IOP) and optic nerve damage. Common causes include central retinal vein occlusion (CRVO), central retinal artery obstruction (CRAO), proliferative diabetic retinopathy (DR) and ocular ischaemic syndrome (OIS)[1]

Managing NVG is challenging. It includes controlling the underlying posterior segment ischemia by panretinal photocoagulation (PRP) or administration of intravitreal injections of Anti-Vascular Endothelial Growth Factor (Anti-VEGF) with simultaneous reduction of IOP by medical therapy or surgical interventions[2]. Surgical options include filtering surgery, glaucoma drainage devices (GDDs) and cyclodestructive procedures[2]. Theselection of the surgical procedure is based primarily on the individual surgeon's judgement and consideration of patients' variables[3].

In this paper, we report a young diabetic gentlemen who had uncontrolled IOP due to neovascular glaucoma, being managed with trabeculectomy with antifibrotic angent, along with reduction of posterior segment ischemia to reduce IOP to desirable level.

Case

A 39yrs old male, with uncontrolled diabetes mellitus (HbA1c 11.9%), diagnosed with Advance Diabetic Eye Disease (ADED) presented to glaucoma outpatient department as a referred case form retina department. He had history of multiple intravitreal Anti Vascular Endothelial Growth Factor (VEGF) injections and Pan Retinal photocoagulation (PRP) laser in both the eyes and has undergone Pars Plana Vitrectomy (PPV) with endolaser (EL) with Silicone oil insertion (SOI) in left eye about 6 months before presentation. One month before presentation, his intraocular pressure (IOP) recorded was 18mmHg and 14mmHg in right and left eye respectively, with 4AGMs (Timolol, Brimonidine, Brinzolamide and Netarsudil) in right eye and 2 AGMs (Timolol and Brimonidine) in left eye. Few days before presentation, he has stopped the

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AGMs by self.

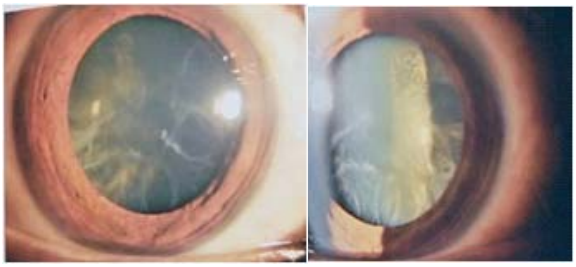

Figure 1: Anterior segment photo of right eye with NVI and cataract.

On presentation, he had best corrected visual acuity (BCVA) of 5/60 in right eye and 6/36 in left eye with IOP of 55mmHg in both the eyes. He had hazy, edematous cornea in both the eyes with neovascularization of iris (NVI) in right eye and cataractous lens in both the eyes. Angle structures were not clearly visible on gonioscopy due to hazy cornea. He was diagnosed with right eye neovascular glaucoma, cataract and proliferative diabetic retinopathy with left eye cataract with ADED, status post PPV with EL with SOI in left eye, status post anti VEGF and PRP laser in both the eyes.

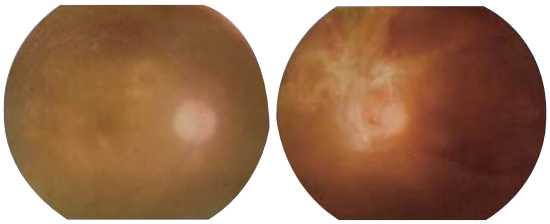

Figure 2: Fundus photo of right and left eye: Note the hazy view due to cataract in both the eyes

He was started on Timolol with Brimonidine combination three times a day, Brinzolamide three times a day, Netarsudil once a day along with dexamethasone eye drop three times a day in both the eyes and tablet Acetazolamide 250mg three times a day with tablet potassium chloride 600mg twice a day. After 5 days with this treatment, his IOP dropped to 38mmHg and 16 mmHg in right and left eye respectively and he was planned for right eye combined phacoemulsification with intraocular lens (IOL) implantation with trabeculectomy with Mitomycin C (MMC), along with intravitreal Anti VEGF (Bevacizumab) injection on the same sitting.

The following day, patient underwent the planned procedure in right eye uneventfully. After scleral flap, MMC 0.02% was used for 2 minutes under the conjunctival and scleral flap. Then phacoemulsification with IOL implantation was done from superior approach as routinely and trabeculectomy was completed with an air bubble kept in anterior chamber (AC). Lastly intravitreal Inj Bevacizumab 0.025mg/ml was given.

On 1st post operative day, he had BCVA of counting finger at 1 foot and IOP of 15mmHg in right eye. Bleb was diffuse and satisfactory. Cornea was clear and air bubble was in AC, where air fluid meniscus was around the level of visual axis, which explains the visual acuity of the patient. He was advised to continue all preoperative topical AGMs along with topical steroids and antibiotics postoperatively.

One week postoperatively, he had BCVA of 6/9 and IOP of 08 in his eye. Bleb was diffuse and satisfactory. He was advised to stop Netarsudil and continue Timolol with Brimonidine combination three times a day and Brinzolamide three times a day along with topical antibiotics and tapering dose of steroids inhis right eye. He was advised for RE PRP laser using Laser Indirect Ophthalmoscope (LIO) to avoid trauma to the bleb by contact lens used in regular PRP laser.

He underwent right eye PRP by LIO on 2nd and 3rd post operative week. On 3rd post operative week, he had BCVA of 6/9 and IOP of 12mmHg in his right eye. Neovascularization of iris has regressed than before. The three AGMs were continued in right eye as before. The conjunctival sutures in right eye were removed. He was kept in close follow-up with both retina and glaucoma department.

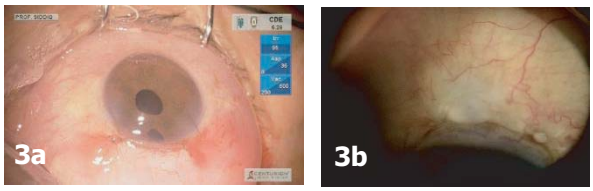

Figure 3: Intraoperative (3a) and one month postoperative (3b) picture of the bleb in right eye.

Discussion

The management of neovascular glaucoma is challenging and gives varying degree of success. The management on NVG focuses on reducing IOP and inhibiting the neovascular drive in the posterior segment[4]. If medical management alone fails to achieve desirable IOP levels, various surgical procedures including conventional filtering surgery, glaucoma drainage devices (GDDs) and cyclodestructive procedures can be tried to reduce IOP (2,4). However, NVG itself is a risk factor for failure of these surgical procedures[4].

A randomized controlled trial has shown that the intravitreal injections of bevacizumab reduce NVI and IOP in NVG and therefore it may be considered as an adjunct to more definitive surgical procedures for NVG[5]. The expected time for new vessel regression is 3 weeks post PRP and 2 days–2 weeks post Bevacizumab[6].

A multicentric retrospective study from Japan compared surgical outcomes between the Baervaldt implant and trabeculectomy with MMC and showed a higher success probability in patients with implants over trabeculectomy(7). Both groups showed similar postoperative complications; however, the Trab group had a higher re operation rate[7]. Another retrospective multicenter study has shown to achieve a successful IOP control in all three procedures, with trabeculectomy showing a notable improvement in postoperative vision compared to GDD and trans scleralcyclophotocoagulation (TSCPC)[6]. This emphasizes the importance of individualized treatment selection and vigilant postoperative care for every NVG patient.

In the case we presented here, the primary aim of reduction of IOP was achieved with trabeculectomy with MMC along with repeated intravitreal Bevacizumab and postoperative PRP laser to control the ischemic process in the posterior segment. On short term follow-up, the patient was able to maintain desirable IOP with one AGM less than the preoperative medications. However, the patient needs to be followed up long time to know the long-term effectiveness of the procedure and the management.

Conclusion

Trabeculectomy with MMC along with intravitreal anti VEGF and post operative retinal lasers to control the ischemia in posterior segment, can be used as an option to bring down IOP to desirable levels and/or reduce AGMs in patients with NVG. Careful patient selection and close follow-up is necessary to have a good outcome.

References

1. Shazly TA LM. Neovascular glaucoma: etiology, diagnosis and prognosis. . Semin Ophthalmol . 2009;24(2):113–21.
2. Shchomak Z, Cordeiro Sousa D, Leal I, Abegão Pinto L. Surgical treatment of neovascular glaucoma: a systematic review and meta-analysis. Vol. 257, Graefe's Archive for Clinical and Experimental Ophthalmology. Springer Verlag; 2019. p. 1079–89.
3. Havens SJ, Gulati V. Neovascular glaucoma. Dev Ophthalmol . 2016;196–204.
4. Ramji S, Nagi G, Ansari AS, Kailani O. A systematic review and meta-analysis of randomised controlled trials in the management of neovascular glaucoma: absence of consensus and variability in practice. Graefe's Archive for Clinical and Experimental Ophthalmology. 2023 Feb 1;261(2):477–501.
5. Yazdani S, Hendi K, Pakravan M, Mahdavi M, Yaseri M. Intravitreal Bevacizumab for Neovascular Glaucoma. J Glaucoma. 2009 Oct;18(8):632–7.
6. Pegu J, Garg P, Shakya R, Dubey S, Dash D, Ahluwalia N, et al. Long-term outcome of surgical management in neovascular glaucoma: A retrospective, multicentric study. 2024; Available from: https://journals.lww.com/ijo
7. Iwasaki K KSWROTYYIT et al. Comparing surgical outcomes in neovascular glaucoma between tube and trabeculectomy: A multicenter study. Ophthalmol Glaucoma. 2022;5:672–80.

Case Report: Management of a case of Repeated Elevation of Intraocular Pressure Due to Anterior Chamber Intraocular Lens

S A Nipa[1], S Rahman[2], Z J Ruma[3], S A Nipu[4]

Abstract

Purpose : To report a case with repeated increase intraocular pressure in a pseudophakic eye by anterior chamber intraocular lens.

Case summary: A 65 years old male presented with the complaints of redness and dimness of vision in right eye. He had a history of redness and pain in right eye one month back. On examination best corrected visual acuity in right eye was PL and 6/6 in left eye. By slit lamp examination revealed conjunctival congestion, pupil mid dilated, cornea clear, intra ocular lens in anterior chamber, peripheral iridectomy in right eye and pseudophakia in left eye. IOP on his right eye was 34 mm of Hg and left eye was 10 mm of Hg. Considering the rise of the IOP, patient received pressure lowering topical and systemic therapy and also with IV mannitol support patient underwent surgery for explanation of AC IOL with trabeculectomy with SFIOL implantation. His post operative best corrected visual acuity was HM and IOP was 14 mm of Hg in right eye.

Key word: Anterior chamber intraocular lens (ACIOL), increase intraocular pressure (IOP), Scleral fixation intra ocular lens (SFIOL).

Introduction

After uneventful cataract surgery, ideally posterior chamber IOL is implanted in the capsular bag. However, this is not always possible, as capsular bag-associated complications may already exist preoperatively (loose zonula, IOL luxation) or occur intraoperatively (anterior or posterior capsular tear). In these cases, IOL has to be implanted in other positions such as anterior chamber (AC), iris, sulcus, or the sclera.

In cases of IOL-related complications or secondary aphakia, a secondary intraocular lens implantation is a good preferable surgical procedure. IOL luxation, incorrect IOL power, IOL opacification, uveitis-glaucoma-hyphemia (UGH) syndrome, patient dissatisfaction, or secondary aphakia indicate the major reasons for such surgery. Recently Secondary IOL implantations have increased, and this surgical procedure is now a days considered very common [1,2].

Case report

A 65 years old male with history of pain redness in right eye one month back presented with these clinical finding.

Indications	Right Eye	Left Eye
VA	PL	6/6
Cornea	Clear	Clear
Anterior chamber	Intra ocular lens	Normal
Lens	Pseudophakia	Pseudophakia
Pupil	Mid dilate	RRR
Iris	Peripheral iridotomy	Normal
Fundus	Optic atrophy	NAD
IOP	34 mm of Hg	10 mm of Hg

No abnormality found in B-scan ultrasound of his right eye. Systemic examination revealed no abnormality involved. Topical Brinzolamide, Timolol and Brimonidine were given to the patient 3 times daily. Additionally, he received oral Acetazolamide 250 mg and Potassium chloride 3 times daily. Difluorinated 4 times daily was used as topical anti-inflammatory agent.

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Under the IOP lowering agent patient went to surgery for explanation of ACIOL. During operation ACIOL was extracted with synechiolysis and trabeculectomy with MMC was done, also anterior vitrectomy was done and SFIOL was implanted with I/V mannitol support. On 1st post operative day his VA was HM and IOP was 14 mm of Hg in right eye. However, on 7th post operative day his IOP was 12 mm of Hg. On 30th post operative day his VA was 3/60 and IOP was 14 mm of Hg without any anti glaucoma medication.

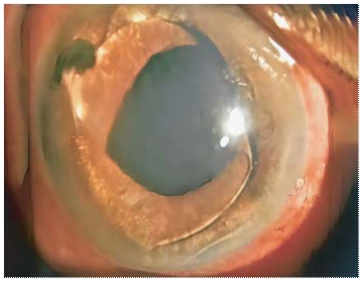

Fig.-1: Pseudophakia with anterior chamber

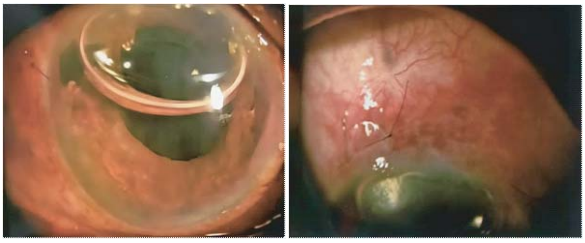

Fig-2, 3: After Exaplantation of intraocular lens (AC IOL) and implantation of SFIOL and Trabeculectomy with air in AC.

Discussion

In case of complicated cataract surgery or trauma with insufficient or any no capsular support intraocular lens (IOL) implantation can be scleral fixation, iris claw or anterior chamber IOL (ACIOL) is the option.

Early closed-loop ACIOLs were initially reported a good short-term result, but later associated with several complications, including cystoid macular edema, uveitis, glaucoma, irreversible endothelial cell loss, hyphemia, and corneal decompensation[3,4,5,6,7,8].

After ACIOL implantation in non-vitrectomies eyes the rate of both short and long-term rise IOP variation in different studies. For example, in one study the primary ACIOL implantation was done with anterior vitrectomy found 100% patients had a long-term IOP rise[9].

Glaucoma in pseudophakia is a secondary glaucoma in which intraocular pressure (IOP) is elevated following cataract removal. This diagnosis is occurred only if there was no glaucoma before to cataract removal[10]. Transient or permanent intraocular pressure elevation occurs in pseudophakic eyes may occur due to vitreous filling the anterior chamber, peripheral anterior synechia, aqueous misdirection syndrome, capsular block syndrome, haptic-induced post-operative pseudophakic glaucoma, post-operative chronic inflammation, uveitis-glaucoma-hyphemia syndrome, pupillary block glaucoma.

The causes of elevation of IOP in cases with ACIOL were presumed due to inflammation, HUG syndrome and pigment dispersion. this is also noted by Sousa et al 2016 and Peralba et al 2018[11]. Pupillary block may also occur due to peripheral iris blow forward around the ACIOL and occlude the angle. Scleral fixation intra ocular lens (SFIOL) may often be the most appropriate for certain patient. SFIOL may be idea for the patient with an IOL that must be exchanged, or an aphakic patient who requires secondary IOL placement[13].

Most patient of pseudophakic glaucoma managed medically. Surgical intervention is needed for cases that are uncontrolled on maximum tolerable medical therapy.

Trabeculectomy without antimetabolites success rate is 27% reported byGross and associates. Prata Junior and associateshad a 70%

success rate after trabeculectomy with mitomycin C (considering both complete and qualified success) in a study that compared postoperative 5-FU to intraoperative mitomycin C[12].

In summary,we experienced a repeated episode of IOP elevation by ACIOL. This case was welled managed by SFIOL and trabeculectomy with MMC. Now patient IOP is well control by this treatment option. Like this case we apply this

treatment option in elevated IOP by secondary IOL specially in ACIOL.

Conclusion

Repeated episodes of elevated IOP by ACIOL, explantation of IOL and SFIOL implantation is a good option. Trabeculectomy with MMC also add this case, the condition was well managed. SFIOL and Trabeculectomy with MMC is a very good treatment option for elevated IOP due to ACIOL.

References

1. Dabrowska-Kloda K, Kloda T, Boudiaf S, Jakobsson G, Stenevi U. Incidence and risk factors of late in-the-bag intraocular lens dislocation: evaluation of 140 eyes between 1992 and 2012. J Cataract Refract Surg. 2015;41(7):1376–1382. doi: 10.1016/j.jcrs.2014.10.040. [PubMed] [CrossRef] [Google Scholar]
2. Ascaso FJ, Huerva V, Grzybowski A. Epidemiology, etiology, and prevention of late IOL-capsular bag complex dislocation: review of the literature. J Ophthalmol. 2015;2015:805706. doi: 10.1155/2015/805706. [PMC free article] [PubMed] [CrossRef] [Google Scholar]
3. Smith PW, Wong SK, Stark WJ, Gottsch JD, Terry AC, Bonham RD. Complications of semiflexible, closed-loop anterior chamber intraocular lenses. Arch Ophthalmol. 1987;105:52–7. [PubMed] [Google Scholar]
4. Biro Z. Results and complications of secondary intraocular lens implantation. J Cataract Refract Surg. 1993;19:64–7. [PubMed] [Google Scholar]
5. Drolsum L. Long-term follow-up of secondary flexible, open-loop, anterior chamber intraocular lenses. J Cataract Refract Surg. 2003;29:498–503. [PubMed] [Google Scholar]
6. Hennig A, Johnson GJ, Evans JR, Lagnado R, Poulson A, Pradhan D, et al. Long term clinical outcome of a randomised controlled trial of anterior chamber lenses after high volume intracapsular cataract surgery. Br J Ophthalmol. 2001;85:11–7. [PMC free article] [PubMed] [Google Scholar]
7. Donaldson KE, Gorscak JJ, Budenz DL, Feuer WJ, Benz MS, Forster RK. Anterior chamber and sutured posterior chamber intraocular lenses in eyes with poor capsular support. J Cataract Refract Surg. 2005;31:903–909. [PubMed] [Google Scholar]
8. Ravalico G, Botteri E, Baccara F. Long-term endothelial changes after implantation of anterior chamber intraocular lenses in cataract surgery. J Cataract Refract Surg. 2003;29:1918–23. [PubMed] [Google Scholar]
9. Kwong YY, Yuen HKL, Lam RF, Lee VY, Rao SK, Lam DS. Comparison of outcomes of primary scleral-fixated versus primary anterior chamber intraocular lens implantation in complicated cataract surgery. Ophthalmology. 2007;114:80–5. [PubMed] [Google Scholar]
10. Dosunmu EO, Freedman SF. Aphakic/ Pseudophakic Glaucoma. Practical Management of Pediatric Ocular Disorders and Strabismus. Springer.N Y; 2016:459–470. [Google Scholar]
11. Peralba RT, Lamas-Francis D, Sarandeses-Diez T, Martínez-Perez L, Rodríguez-Ares T. Iris-claw intraocular lens for aphakia: can location influence the final outcomes? J Cataract Refract Surg. 2018;44(7):818–826. doi: 10.1016/j.jcrs.2018.05.010 [PubMed] [CrossRef] [Google Scholar]
12. Prata Junior JA, Minckler DS, Baerveldt G, Lee PP, LaBree L, Heuer DK. Trabeculectomy in pseudophakic patients: postoperative 5-fluorouracil versus intraoperative mitomycin C antiproliferative therapy. Ophthalmic Surg 1995;26:73–77. 23. Lamping KA, Belkin JK. 5-Fluorouracil and mitomycin C
13. Stem MS, Todorich B, Woodward MA, Hsu J, Wolfe JD. Scleral-fixated intraocular lenses: past and present. J Vitreoretin Dis. 2017;1(2):144-152.