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Bangladesh Glaucoma Society

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How can we make a model Glaucoma practice in Bangladesh

M N Islam[1]

About 70 million people have glaucoma globally[1].It is the second leading cause of blindness after cataract. It occurs more commonly among older people.[2] Closed-angle glaucoma is more common in women.[1] Glaucoma has been called the "silent thief of sight", because the loss of vision usually occurs slowly over a long period of time.[3]

In Bangladesh, a population based survey in Dhaka division conducted by M MustafizurRahman, et al[4]showed among people aged 40 years and older, the prevalence ofdefinite glaucoma was 2.1%, definite and probable glaucoma was 3.1%, POAG was the most common form of glaucoma, 5% of all blindness were caused by glaucoma.

In last decade, Glaucoma awareness has been increased overwhelmingly in Bangladesh. Definitely it's a primary success of the members of Bangladesh Glaucoma Society. Our patient come to our chamber and many of them wish to know if they have glaucoma or not.

In a recent study by Nazneen Khan et al conducted from March 2010 -February 2011, showed that POAG - 51.4%,PACG -48.7%,Secondary Glaucoma 15% and most importantly awareness has been increased among our population[5].

In Bangladesh there are more than 1000 ophthalmologists, but how many of them practice glaucoma? Bangladesh Glaucoma Society has only 84 members. Our total population is more than 165 million but with that kind of ratio we do not even haveminimum number of ophthalmologists or glaucoma specialists.

I will outline what I believe are the necessary steps to build a large, successful glaucoma practice so that our ophthalmologists can effectively prevent glaucoma blindness.

We all know, glaucoma is a devastating disease and it has no complete cure. Ophthalmologists can control the disease after its diagnosis. So earliest diagnosis is most important factor to prevent glaucoma blindness. All ophthalmologists should have facility to see the optic disc, angle of anterior chamber and applanation tonometry. In any suspected glaucoma the patient's colour fundus photography (CFP), Optical Coherence Tomography (OCT)[6], Central Corneal Thickness (CCT) measurementi.e. Pachymetry and Visual Field Analysis (VFA) should be done. If that is not available, the patient should be referred to any centre where all these facilitiesare available. Someone might think most of these advanced imaging technologies are very necessary for a glaucoma practice.No, as our teachers 20 years before did not need these imaging, but they could diagnose glaucoma. Nearly two decades imaging technology have been in use to diagnose early glaucoma only. Of course, these investigations help us to be more confident on our clinical diagnosis and to evaluate prognosis, but they are not absolutely necessary.

Patient Communication is of utmost importance

Being able to communicate effectively with patients is crucial to their long-term management as well as the long-term growth of our practice.Wemay look at the entire diagnosis of glaucoma, including the treatment process, from the patient's perspective. To them, they have a potentially blinding disease. Their visual outcome rests completely in our hands. Even though the patient may think it is easy, in reality the actual management of chronic disease – 'glaucoma' is not as simple as the patient feels.

While the non-verbal communication of our chamber and staff is important, it pales in comparison to the verbal and non-verbal communication from us, the practitioner. While everyone's chairside manners are different, we simply must gain the patient's trust the first time any mention of glaucoma is made. Once the patient trusts you, he or she will follow your

instructions, be more compliant, stay with your practice and tell others about your services.

Without question, one of the more common mistakes new glaucoma practitioners make is the manner - how they explain a diagnosis to a patient. While trying to be compassionate, an inexperienced clinician may say, "Well, Mrs. R, I think that you may have glaucoma." The patient heard two things: "think" and "may." In other words, the patient might believe that the clinicianhas no idea what is wrong. And, while you may not yet have all the diagnostic puzzle pieces in place, you must concentrate on the known facts.So, be sure to say something like, "Mrs. R, all of your symptoms and investigations indicate that you have some risk factors for glaucoma development, and you will need to return to my chamber within the next few weeks for further evaluation."

One thing that I often do during the initial discussion with particularly nervous patients who are at risk for developing glaucoma is put my hand on top of their hand and speak directly to them while looking into their eyes. Physical touch and eye contact portray not only compassion and concern, but also firmness and confidence.

If a patient ultimately wants a second opinion, I encourage him or her to do so. I'll make recommendations to ophthalmologic colleagues whom I believe will best handle the patient at home or abroad. I encourage second opinion, because if both the first and second opinions are correct, both providers will come to the same medical conclusion, itincreases the patient's confidence of the initial opinion.

Once they become your patient for the long run, it is your responsibility to watch them closely and monitor their condition. You need to give them an idea that their medications may need to be changed and inform them that they may require surgery. Offer them reassurance. Give them confidence. Provide them with expert care.

Counselling with Photograph/ digital camera

Counselling is the integral part of glaucoma practice. Counselling should be audio visual. Patients understand better by watching any documents. Even if you do not have facility of photography, draw a picture of optic disc, show him/her the cupping. What is normal and why you are suspecting. If you have the facility, show them the changes in visual field, retinal nerve fibre layer (RNFL) thickness change. Patient is definitely going to have more confidence in your diagnosis.

Referring glaucoma patients

In different parts of the world, licensed optometrists and general practioners can see patient and they can refer glaucoma patient. They have medical models designed specifically to facilitate the day-to-day management of glaucoma patients in an optometric setting. In Bangladesh we do not have such kind of optometrists so our all ophthalmologists should diagnose glaucoma confidently. Fortunately, we have ophthalmic diagnostic centres not only in our capital and major cities, but also in some districts, where OCT, VFA and CCT measurement are available. With good clinical examination and with help of investigations, glaucoma can be diagnosed confidently. In case of suspicion, the patient should be referred to a tertiary hospital.

If all our ophthalmologists, along with our glaucoma specialists, can try sincerely and if we can commit to undertaking all these responsibilities properly, we would look around one day and find that we have a successful glaucoma practice in Bangladesh. And we can make the dream of preventing glaucoma blindness a reality.

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A TALE OF DISCS.... case series study

S M Noman[1], S R Roy[2]

Abstract

Purpose : To document and describe different patterns of non-glaucomatous atypical discs and to identify their other ocular and systemic associations.

Design : A hospital-based prospective observational case series done during the period from 1st November 2009 to 31st October 2011.

Method : Patient particulars, history and complaints were recorded. Ophthalmic examination included visual acuity, refraction, slit-lamp examination, pupillary reaction, tonometry, gonioscopy and funduscopy. The relevant investigations were done and documented. Similar relevant details were recorded on each follow-up.

Results : 30 patients were included in this case series. 13 patients were female, 12 were male. 5 patients were diagnosed as myelinated optic nerve head in both eyes, 5 patients had bilateral titled discs without refractive error; 4 patients had unilateral optic disc pit; 4 patients had bilateral optic disc drusens, 3 patients had bilateral disc coloboma, 2 patients had morning glory disc, 1 patient had optic nerve hypoplasia, 5 patients had macrodisc and 1 patient was diagnosed with post-traumatic optic nerve head avulsion.

Conclusion : Proper ophthalmic examination and investigations are essential for the diagnosis of atypical discs and congenital anomalies of the optic disc. Documentation not only helps clinicians in their clinical practice but also helps in teaching, patient counseling and planning for rehabilitation.

Introduction

The optic nerve begins anatomically at the optic disc but physiologically and functionally from the ganglion cells that cover the entire retina. It is a collection of 1.1–1.2 million ganglion cell axons that traverse the sclera through lamina cribrosa. The optic nerve develops from the neural tube (neuroectoderm). Due to developmental anomalies, infection and trauma there is damage to the ganglionic cells, as well as nerve fibres, which in turn affects the patient's visual acuity and visual field. A normal disc is 1.5 mm in diameter, has a 0.3:1 – 0.5:1 cup disc ratio and a healthy neuroretinal rim.

Traumatic, inflammatory and vascular toxic optic disc abnormalities are not uncommon in this subcontinent but atypical optic disc changes like congenital optic disc anomalies are very rare and usually these patients remain undiagnosed and without rehabilitation. With accurate diagnoses at early age, this can aid us in identifying associated ophthalmic and systemic anomalies so that treatment and rehabilitation can be started.

Methods

This is a hospital-based combined non-concurrent and concurrent prospective case series. Cases were identified throughout a two-years period. All patients were referred from the outpatient department of the Chittagong Eye Infirmary and Training Complex (CEITC). Details of history included maternal and birth history, biographical details and family history. History of trauma was ascertained. Ophthalmic examination was done on all patients and examination details included visual acuity, tonometry, gonioscopy, indirect ophthalmoscopy and optic disc photography. B-scan ultrasonogram was done if necessary.

Results

A total number of 30 patients with atypical optic discs were included in this case series. Amongst them, 29 cases were either congenital or developmental in nature. 1 case was traumatic.

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Myelinated Optic Nerve

Of the 5 myelinated optic nerve cases, 3 were female and 2 were male. Symmetrical involvement was observed in these cases. No other ocular and systemic associations were present in these cases. In all cases, visual acuity was normal. 2 cases had peripapillary myelination and 3 cases had myelination along the superior arcuate area. On Humphrey's Visual Field (HVF) analysis 4 cases showed corresponding visual field loss such as enlargement of blind spot and arcuate scotomas.

Bilateral Tilted Discs

There were 5 bilateral titled disc cases. 3 were female and 2 were male. They all had normal visual acuity with no refractive errors. They all has small, oval and D-shaped discs that were tilted inferior-nasally.

Optic Disc Pit

4 optic disc pit cases were found; 3 were female and 1 was male with an average age of 40 ± 10 years. Their visual acuity was normal with no refractive errors. HVF analysis revealed caecocentral scotomas in 2 cases. Neither macular oedema nor serous macular detachment was observed in any of these cases. The pit was present in the temporal aspect of the disc.

Optic Disc Drusen

4 patients were diagnosed with optic disc drusen. Of these, there were 3 female and 1 male. All cases were bilateral with minimal disturbance in visual acuity (6/9 – 6/12). 2 cases revealed an enlargement of the blind spot on HVF analysis. All cases had some common features such tortuous blood vessels, increased blood vessels, scalloped disc margin and refractile bodies over the disc. B-scan ultrasonography revealed hyperacoustic shadows in optic nerve head suggestive of calcification.

Optic Disc Coloboma

In the optic disc coloboma cases all were male with an average age of 50 ± 10 years. 2 of them had isolated disc colobomas and 1 was associated with retino-choroidal coloboma. Visual acuity was diminished in all of the affected eyes (6/60–CF).

Morning Glory

Of the 2 morning glory cases 1 was male and 1 was female. Visual acuity was impaired in both cases. Typical signs of morning glory seen were seen in both cases (such as large optic discs, funnel-shaped excavation, central glial tissue and spoke-like blood vessel configuration).

Optic Nerve Hypoplasia

There was one patient of optic nerve hypoplasia who was a boy of 6 years. It was a bilateral case in both optic disc and macular hypoplasia. Visual acuity was diminished in both eyes.

Macrodisc

5 macrodisc cases were found (1 female and 4 male) with an average age of 30 ± 10 years. All of them had normal visual acuities without refractive errors. Normal IOP and normal visual fields were observed in all cases. Average cup:disc ratio was 0.6 ± 0.2.

Optic Nerve Avulsion

There was a single case of optic nerve avulsion which had a history of trauma and loss of vision for 2 days.

Discussion

The first portion of the optic nerve contains the nerve fibres of 1 to 1.2 million ganglionic cells that traverse the sclera through the lamina cribrosa. Developmentally, the optic nerve is neural tube derivative. The ganglionic cells of the retina develop axons, to a point where the optic nerve leaves the posterior surface of optic cup. Developmentally this will become the optic disc. In the inferior aspect of the optic stalk, normally there is a groove or fissure which contains mesenchymal tissues and the hyaloid atrery. This fissure becomes the optic canal by fusion at the seventh week of gestation. Failure of the fissure to close completely results in coloboma formation in the retina, choroid and also in the optic nerve. The cells of the optic

stalk form neuroglial supporting cells for the axons, and the cavity of the stalk disappears. The stalk together with the ganlionic cell axons, form the optic nerve. The axon of the optic nerve begin to develop their myelin sheaths just before birth, but the process of myelinetion continues for sometime after birth.

In myelinated optic disc, the optic disc is surrounded by bright white streaky and irregular patches. The myelination may extend to the periphery or cover part of the disc. It may be unilateral or bilateral with asymmetrical involvement. Myelinated nerve patches are often seen in the arcuate bundles occasionally abutting the disc. When they are contiguous, these nerve patches may be confused with disc oedema or cotton wool spots1 (Figure 1).

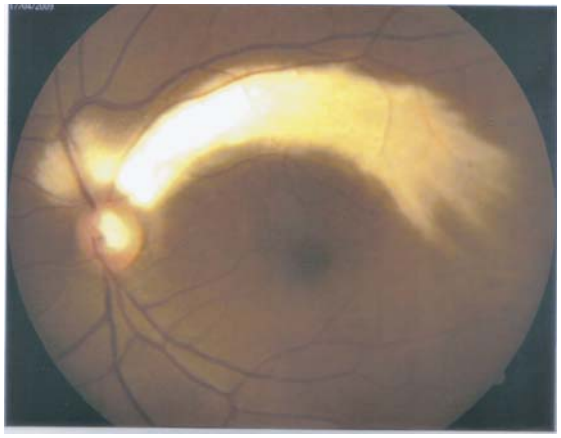

Figure 1: Myelinated optic disc involving superior arcuate area.

Of the 5 myelinated optic nerve cases, 3 were female and 2 were male. Symmetrical involvement was observed in these cases. No other ocular and systemic associations were present in any of the cases. In all cases visual acuity was normal. In 2 cases peripapillary meylination was present and in 3 cases meylination was along the superior arcuate area. On HVF analysis, 4 cases showed corresponding visual field loss such enlargement of blind spot and arcuate scotomas. Usually with extensive nerve fibre myelination, myopia, anisometropia and amblyopia are associated. Neurofibromatosis-1 and multiple basal cell naevus are some systemic associations. Neither systemic nor ocular associations were found in our series.

Tilted discs are usually bilateral. They are caused by a developmentally oblique entry of the optic nerve into the globe. For this, one portion of the neuroretinal rim is elevated and the other is depressed. Usually it looks D-shaped and is directed inferonasally. The disc margin may be indistinct and temporal vessels deviate nasally before turning temporally. The visual field may show a superior temporal field defect2. Visual acuity and visual field were normal in all of our cases. Titled discs positioned inferonasally was present in all cases. Temporal elevation of the neuroretinal rim with nasal shifting of temporal vessels were observed in all cases (Figure 2).

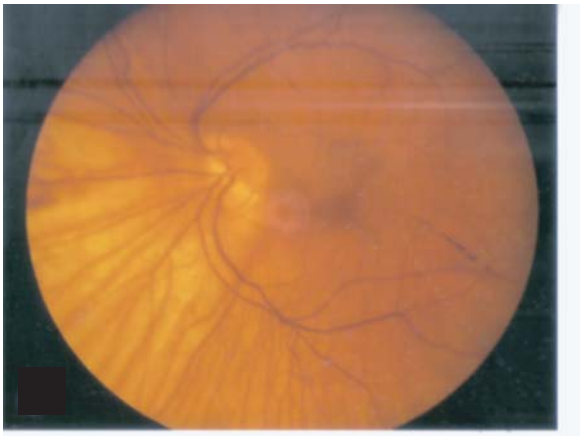

Figure 2 : Titled optic disc.

Optic disc pit is a depressed area within the optic nerve head and usually lies temporally. Visual acuity is normal in the absence of complications. Visual filed defects are common and mimic glaucoma. Serous macular detachment develops in about 45% of eyes with non-central disc pits. The subretinal fluid is thought to be derived from the vitreous. Less likely sources are the subarachnoid space and from abnormal vessels within the base of the pit.2 Visual acuity was normal in all of our cases. In 2 cases caecocentral scotomas were detected on HVF examination. Discs were normal in shape, no subretinal fluid nor serous macular detachment was observed in our cases (Figure 3).

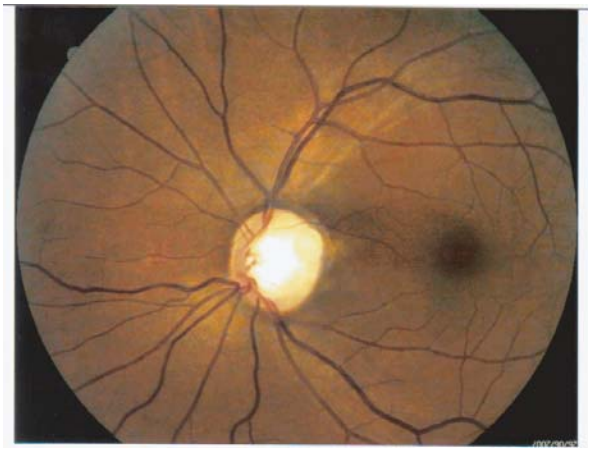

Figure 3: Optic disc pit.

Optic disc drusen are composed of hyaline-like calcific material within the optic nerve head. They are often bilateral. The buried drusen cannot be seen over the surface. Elevated disc, hyperaemia and early branching of the blood vessels are characteristics of buried drusens. Exposed drusens are usually seen over the surface of the disc. The drusen may be associated with angioid streak. They may also be associated with retinal bleeding.3 All of our cases were bilateral with minimal disturbance in visual acuity (6/9-6/12) and with no improvement with pinhole. 2 cases revealed enlargement of blind spot on HVF analysis. In all cases, there were some common features like tortuous blood vessels, increased blood vessels, scalloped disc margin and retractile bodies all over the disc. B-scan ultrasonography revealed hyperacoustic shadows in the optic nerve head suggestive of calcifications (Figures 4a and 4b).

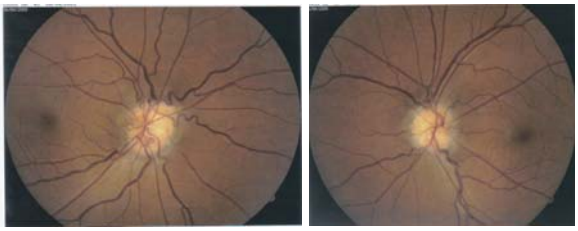

Figure 4a: Optic disc drusen

Optic disc coloboma is a developmental defect due to non-closure of optic fissure at the 7th week of gestation. It may be associated with retinochoroidal coloboma. Sometimes it looks like duplication of the optic disc and is called pseudo duplication4. Visual acuity is often decreased. A bowl-shaped excavation (decentered inferiorly) and the remaining superior portion of neuroretinal rim are the clinical features of optic disc coloboma. Perimetry usually shows superior visual field defect. It may be associated with microphthalmos and retinochoroidal coloboma. Complications such as serous retinal detachment and peripapillary choroidal neurovascularization may occur. Some chromosomal anomalies and CHARG association (heart defects, choanal atresia, retarded growth and development, genital and ear anomalies) are associated systemically. In our 3 optic disc coloboma cases, 2 were isolated disc colobomas and 1 was associated with retinochoroidal coloboma. Visual acuity was diminished in all affected eyes (6/60-CF). The coloboma involved almost the entire disc except for a tiny superior neuroretinal rim. There were no systemic associations in any of the cases. HVF examination revaeled a caecocentral scotoma in 2 cases and an arcuate scotoma-like defect in 1 case which was associated with retinochoroidal coloboma (Figure 5).

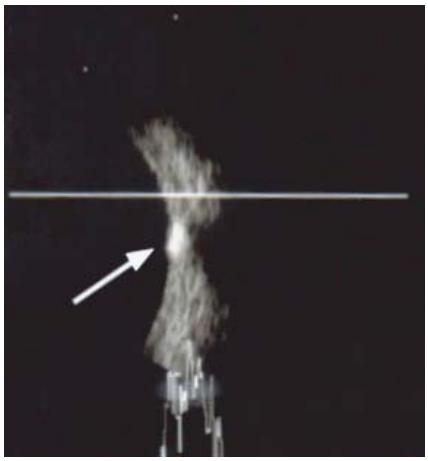

Figure 4b: Hyperacoustic shadow of drusen in optic disc.

Morning glory is a unilateral and rare case which affects women more than men. Patients may present with poor vision or strabismus. A funnel-shaped excavation of the optic disc surrounded by a depigmented area, centrally elevated hyperplastic glial tissue and normal straight blood vessels radiating from disc margin are the

characteristics of the morning glory anomaly. Non-rhegmatogenous retinal detachment may be associated as a complication with this disease.

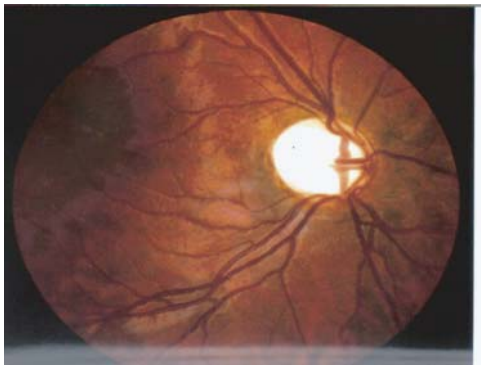

Figure 5: Optic disc coloboma

Frontonasal displasia that includes mid-facial abnormalities with hypertelorism, basal encephalocele and the absence of corpus callosum are the systemic associations.5 In our cases one was male and one was female. Both of them presented with severely depressed visual acuity (CF). Both of them were unilateral and all of the features of morning glory anomalies (such as large optic disc, funnel-shaped excavation, central glial tissue and spoke-like blood vessels were present). No other ocular and systemic associations were found in our cases (Figure 6).

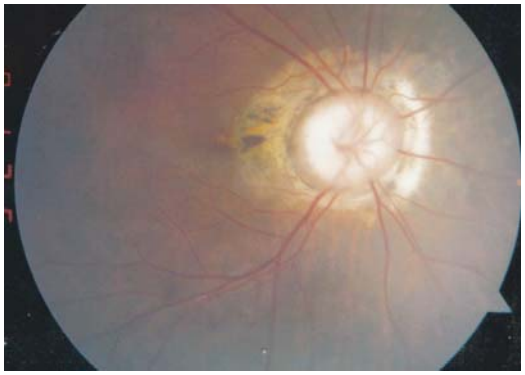

Figure 6 : Morning glory anomaly

Optic nerve hypoplasia is a condition that results in under development of the optic nerve. The optic disc appears abnormally small due to immature and decreased optic nerve axons. It may be unilateral or bilateral. Maternal use of alcohol, quinine, protaminzinc insulin, steroids, diuretics, cold remedies and anti-convulsants may cause such a defect. Blindness in early infancy in bilateral cases and squint in unilateral cases are also associations.2 In our single case, visual acuity was very poor since birth and later esotropia developed gradually. Macular hypoplasia was diagnosed in both eyes. No other ocular nor systemic associations

were found. No history of maternal adverse drug use during pregnancy was observed. (Figure 7).

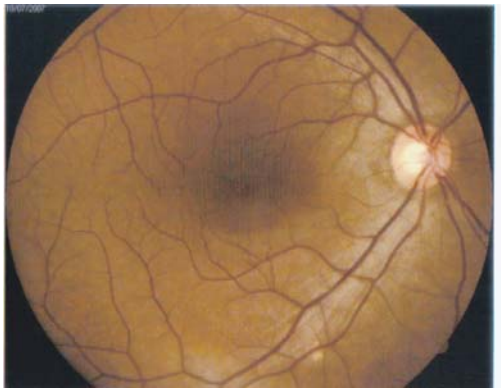

Figure 7: Optic nerve hypoplasia

Developmentally bilateral large discs (more than 1.5mm) is not very uncommon. There is an increased cup:disc ratio that resembles glaucomatous optic disc. Optic neuropathy looks like normal pressure glaucoma in teenagers with congenital macrodisc.6 In our macrodisc cases, all of them had normal visual acuity without refractive errors. Intraocular pressure and visual field analysis were normal. The average cup:disc ratio was 0.6 0.2 (Figure 8).

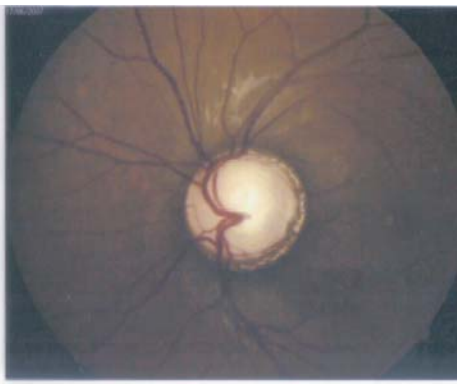

Figure 8: Congenital macrodisc

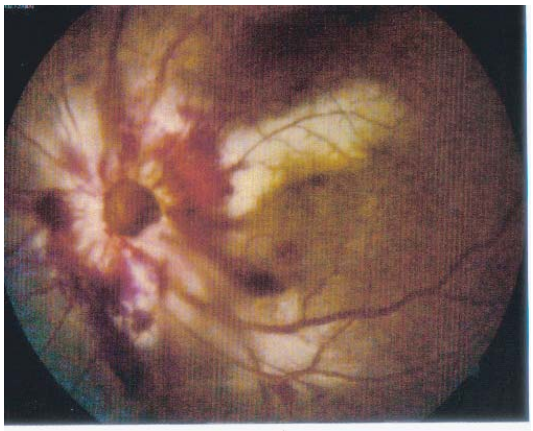

Figure 9: Optic nerve avulsion

Optic nerve avulsion is caused by severe blunt trauma which is associated with loss of vision, optic disc and retinal hemorrhage and total afferent pupillary defect. Our patient had a history of severe blunt trauma with loss of vision, intra-retinal hemorrhage and restricted ocular motility due to entrapment of muscle within a blow-out fracture (Figure 9).

Conclusion

Different types of optic nerve abnormalities like developmental, inflammatory, traumatic, idiopathic and vascular can be found in clinics. Proper history taking and appropriate examination (like pupillary reaction, visual field analysis, direct or indirect ophthalmoscopy) are very important for the diagnosis of atypical discs. After diagnosis, proper evaluation, counseling and rehabilitation are mandatory.

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Original Article

Incidence of POAG and PACG in Patients attending the Eye OPD in a Tertiary Military Hospital in Dhaka

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Abstract

Introduction : Glaucoma is one of the leading causes of irreversible blindness. Proper diagnosis and early intervention can save the sight and reduced social burden worldwide.

Objective: To Estimate the incidence of Primary Open Angle Glaucoma (POAG) and Primary Angle Closure Glaucoma (PACG) in the eye OPD of tertiary military hospital, Dhaka.

Materials and Method : This population based Cross sectional observational study was carried out in patients attending the eye OPD from March 2018 to February 2019. Individuals attending the OPD aged 40 years and above were included in this study using multistage random cluster sampling. All subjects underwent a detailed ophthalmic examination including applanation tonometry, ultrasound pachymetry, gonioscopy, and frequency doubling technology perimetry. Glaucoma suspects also underwent perimetry. Data collected were analyzed using SPSS 23.

Result : Of 172 cases diagnosed with glaucoma,103(59.88%) had POAG and 69(40.12%) had PACG. 9(5.24%)subjects newly detected with glaucoma [3.49% POAG; 1.75% PACG].In POAG group,4(66.67%) were male and 2(33.33%) female. In PACG group it was 1(33.33%) male and 2(66.67%) female. Average age was 58±9.2 and 60±11.3 years for POAG and PACG respectively.The average IOP at presentation was 24.66±4.02mmHg for POAG whereas 29.71±6.81 mmHg for PACG.

Conclusion : Incidence of POAG and PACG is2:1 with mean age lower for POAG than PACG.A longer study with a larger population is recommended.

Key-words : Glaucoma, Incidence,Gonioscopy, Frequency doubling perimetry, Humphrey visual field analysis, Primary angle closure glaucoma, Primary open angle glaucoma.

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Introduction

Glaucoma is the leading cause of global irreversible blindness[1] and an important public health issue[2]. Population-based studies are important for assessment of disease burden, health-care policy planning, and appropriate resource allocation[2]. Asia alone accounts for almost 60% of the world's total glaucoma cases[2–5].Unless detected at an early stage the prognosis for sight will be poor[4]. Once detected, effective delivery of care still presents dilemmas that are specific to the individuals and their social environment. So far maximum study carried out only the prevalence of POAG and PACG , few studies only incidence.

This study is a population-based cross-sectional study[1] in a tertiary military hospital, Dhaka, which spanned from February 2018to January 2019. In this study, primary angle closure glaucoma (PACG), and primary open angle glaucoma (POAG) were defined as per ISGEO guidelines[3].

Materials and Methods

The International Society of geographical and epidemiological Ophthalmology (ISGEO) guidelines for detection of glaucoma in cross sectional epidemiological research are based on three levels of evidence (Foster et al[3]):

1. Category 1 Diagnosis (structural and functional evidence) : The highest level of certainty requires optic disc abnormalities (VCDR or VCDR asymmetry >97.5th percentile for the normal population, or a neuroretinal rim width reduced to <0.1 CDR between 11 to 1 o' clock or 5 to 7 o'clock) and visual field defect compatible with glaucoma.
2. Category 2 diagnosis (advanced structural damage with unproved field loss) : In the

second level, if a visual field test could not be performed satisfactorily, a severely damaged optic disc (VCDR > 99.5th percentile of the normal population) would be sufficient to make the diagnosis.

3. In diagnosing cases based on Category 1 or 2, there should be no alternate pathology to explain the disc damage (like dysplastic disc or marked anisometropia) or the visual field change (like macular degeneration, retinal vaso-oclusive events or cerebrovascular accident).

4. Category 3 diagnosis (optic disc not seen; field testing not possible) : Lastly, if the optic disc could not be examined because of media opacity / visual acuity <3/60 (and, hence, no field test was also possible), an IOP exceeding the 99.5th percentile of the normal population, or evidence of previous glaucoma filtering surgery, may be taken as sufficient for a diagnosis of glaucoma.

The following definitions, based on the ISGEO guidelines[3] were used for the current work:

(1) Primary angle closure suspect (PACS): An eye in which appositional contact was present on gonioscopy between the peripheral iris and posterior trabecular meshwork and more than 270° of posterior trabecular meshwork could not be visualized5.

(2) Primary angle closure (PAC): An eye with an occludable drainage angle on gonioscopy (posterior trabecular meshwork seen for less than 90°) and features indicating that trabecular obstruction by the peripheral iris had occurred, such as peripheral anterior synechiae, elevated intraocular pressure, iris whorling (distortion of the radially orientated iris fibers), "glaucomflecken" lens opacities, or excessive pigment deposition on the trabecular surface. The optic disc did not have glaucomatous damage.

(3) PACG: PAC, along with evidence of glaucoma with characteristic disc and field changes.

(4) POAG:A bilateral disease of-

- ⇨ Adult onset.
- ⇨ IOP >21 mmHg at some stage.
- ⇨ Glaucomatous optic nerve damage.
- ⇨ An open anterior chamber angle.
- ⇨ Characteristic visual field loss as damage progresses.
- ⇨ Absence of signs of secondary glaucoma or a non-glaucomatous cause for the optic neuropathy.

Selection criteria

1) Inclusion criteria

a. All patients attending OPD, age over 40 were included for enumeration of glaucoma.

b. ISGEO guidelines were used for enumeration subjects with glaucoma.

2. Exclusion criteria

a. Any corneal disease/opacities preventing accurate applanation tonometry.

b. Ocular trauma.

c. One eyed and bilaterally blind patients.

d. Subjects with dense media opacity precluding adequate view of the posterior pole.

e. Best corrected visual acuity <6/60, spherical refraction outside 5.0 diopters, cylinder correction outside 3.0 diopters, or a combination thereof.

f. Any other ocular or systemic disease that could affect the optic nerve or the visual field.

Ethical measures

Informed written consent was taken from all study subjects after full explanation of the nature, purpose and potential risk of all procedures needed for the study. The tenets of the declaration of Helsinki were observed

Instrumentation and procedure

After determination of the best corrected visual acuity, a complete ocular examination was performed. As is normal procedure in clinic IOP was measured using the GAT with topical anesthetic and fluorescein. Pupillary evaluation and gonioscopy carried out all the subjects. Grading of lens opacities also done as or when required.

Data analysis

Data was analyzed and processed as frequency and percentage in tables using Micro-Soft Excel statistical program & IBM SPSS 23 (Statistical Package for Social Sciences) program.

Results

A total of 8,613 subjects aged 40 years or older were enumerated from patient attending eye OPD, CMH Dhaka and 3683 (42.76%)subjects fulfilled the laid down inclusion and exclusion criteria. Data from these eligible subjects were included for tabulation and analysis.Out of that 172(4.67%) was diagnosed with glaucoma. Figure1 shows distribution of subjects according to age group. The average age of the subjects in our study was 61.23 (±9.90) and mean IOP was 24.66±4.02mmHg and 29.71±6.81 mmHg for POAG andPACG. In male POAG64(62.13%), PACG 28(40.59%)and in female it was 39(37.86%) and 41(59.41%) for POAG and PACG (Table1). Total 9(5.24%)subjects newly detected glaucoma(Figure2).Out of 9 , 6(5.82%) having POAG and 3(4.34%) PACG (Table II). In POAG group male was 4(3,88%), female was 2(1.94%) and PACG group 1(1.44%) male and 2(3.17%) was female.

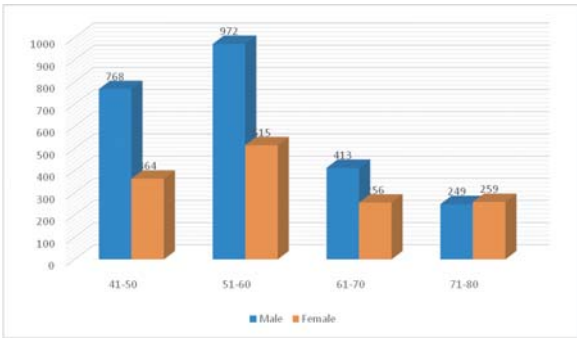

Fig-I: Distribution of subjects according to age group

Table-I : Distribution of subjects according to age group

Age (in year)	POAG		PACG	
	Male (%)	Female(%)	Male(%)	Female(%)
Total	64 (62.13)	39(37.86)	28(40.59)	41(59.42)
41-50	14(15.10)	5(6.30)	5(5.40)	7(8.90)
51-60	19(22.60)	9(11.40)	8(8.60)	13(16.50)
61-70	21(20.40)	17(21.50)	10(10.80)	15(19.00)
71-80	11(11.80)	7(8.90)	5(5.40)	6(7.60)

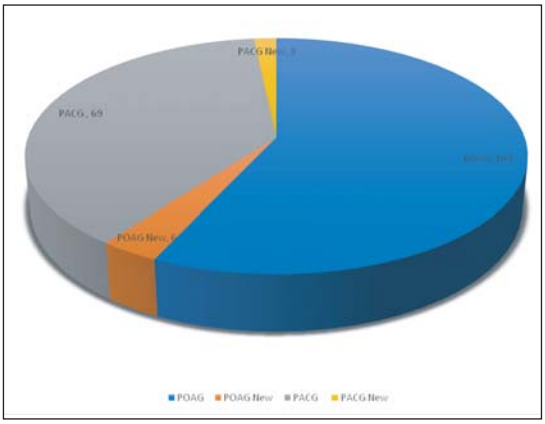

Fig II: Distribution of respondents diagnosed with glaucoma

Table-II: Distribution of newly detected glaucoma (incidence)

Sex	POAG	PACG	Total
Male	4(3.88%)	2(1.94%)	6(5.82%)
Female	1(1.44%)	2(3.17%)	3(4.34%)

Discussion

79.6 million people projected to have glaucoma by 2020, and of these, 74% will have open angle glaucoma (OAG)[6]. The worldwide prevalence of glaucoma is further expected to increase to 111.8 million people by 2040, causing extensive economic and quality-of-life burdens[7].Among those detected with glaucoma, approximately 26% have angle closure glaucoma, which accounts for half of the cases blinded from glaucoma[8].In 2013, the overall prevalence of glaucoma was 3.54%. POAG was the predominant glaucoma subtype, accounting for 65.2% of all glaucoma. There was a higher PACG risk in individuals residing in East Asia, and higher POAG risk in urban compared with rural habitations. Among the subregions, South-Central Asia will record the steepest increase in number of glaucoma individuals from 17.06 million to 32.90 million, from 2013 to 2040. These ndings have implications on the prioritization and implementation of future public health initiatives for glaucoma in different countries and regions across Asia.Quigley and Broman[9] estimated that the prevalence of POAG and PACG were 0.98% and 0.60%, respectively, in Asians aged 40 years and older in 2010.Rudnicka and colleagues found apooled

POAG prevalence of 1.4% in Asians[10]. These analyses likely underestimated the actual glaucoma prevalence, as these were based predominantly on population studies with non-standardized glaucoma diagnostic procedures and criteria, for example, lacking visual eld testing, or relying only on intraocular pressure to diagnose glaucoma. Accurate glaucoma assessment involves a complete evaluation of structural (ie, glaucomatous optic disc changes) and functional damage (ie, visual eld defects). Gonioscopy is essential to differentiate between POAG and PACG, while alternative assessment techniques, for example, van Herrick, fail to identify a signi cant number of PACG cases[11]. East and South-Central Asia harbour the highest glaucoma burden in 2013. These regions comprise the two most highly populated Asian countries, that is, China and India, which account for 61% of Asia's population[12].Overall glaucoma prevalence rates for East Asia are attributed to the high prevalence of PACG in China[13-20] and high POAG prevalence in Japan and South Korea in the form of normal tension glaucoma[21-24]. The overall and region-speci c glaucomaburdens are higher than previous projections for Asia[3] and India[8]. There is a substantial increase of people with glaucoma (57.6%) in our projection up to 2040. The increase in overall number of people with glaucoma is principally due to the ageing transition in population structure, that is, improved life expectancy and disproportionate increase in elderly individuals over time[12].While East Asia had the highest number of people with glaucoma in 2013, South-Central Asia will eventually surpass East Asia and record the highest number of people with POAG, secondary glaucoma and overall glaucoma in 2040. In addition, better representation across Asia in the meta-analysis is evidenced by pooling more recent studies from varied ethnicities, including those not previously analyzed (ie, South Korea, Iran, Nepal, Myanmar, Sri Lanka and Qatar)[25-28]. There are limitations to this analysis. First, the lack of age-speci c and gender-speci c data from several studies allowed fewer studies to be included in the Bayesian meta-regression, particularly for secondary glaucoma. Second, prevalence estimates for West Asia used data from the Qatar Eye Study only. Third, prevalence estimates for South-Central Asia were derived from South Asian population-based studies due to the unavailability of data in Central Asia. However, considering the geographic proximity between South and Central Asia, and the larger demographic of South Asia in that subregion, it may be reasonable to extrapolate the estimates of South Asia to the entire subregion in order to provide the best available estimate for this subregion.

Conclusion

Our analysis provides the basis to guide rationalisation and implementation of sustainable healthcare infrastructure to screen, monitor, treat and rehabilitate people affected with glaucoma.The lack of an acceptable screening technique, limitations in access to care and available medical expertise need to be suf ciently addressed to ensure the success of any public health programme.

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Original Article

Anterior Segment Biometric Parameter Changes Following Laser Peripheral Iridotomy In Primary Angle Closure Suspect Patients Assessed by Anterior Segment Optical Coherence Tomography (Oct): A Tertiary Center Study

R Mannan[1], S Adhikary[2], R Hasan[3], I M Munir[4]

Abstract

Purpose : To measure anterior segment biometric parameter changes in primary angle-closure suspect (PACS) patients after laser peripheral iridotomy (LPI) using anterior segment optical coherence tomography (AS OCT) in a tertiary center.

Methods : Twenty eight primary angle closure suspect (PACS) patients were examined with AS-OCT before and after performing LPI (after 2 weeks & after 1 year). Anterior chamber depth (ACD), anterior chamber volume (ACV), and Anterior chamber angle (ACA) parameters (eg. angle opening distance [AOD750], angle recess area [ARA750], and trabecular iris space area [TISA750]) were measured. Anterior chamber parameters were compared before and after performing LPI. Statistical analysis was performed to find out possible factors associated with narrow angle of anterior chamber.

Results : Mean ACD(from 2.09 mm pre LPI to 2.56 mm post LPI) ,mean ACV (from pre LPI 163.23 mm^3 to post LPI 185.35 mm^3) and all the three ACA parameters (AOD750 [from 0.16mm to 0.27 mm], TISA750 [from 0.08 mm to 0.13 mm], and ARA750 [from 0.09 mm to 0.14 mm]) were found to be increased at 2 weeks post-LPI (all $P < 0.001$). As after 1 year post-LPI, all three ACA parameters remained almost unchanged from their 2 weeks post-LPI value, possibly the Lens Volume significantly might have increased and was associated with ACA narrowing in long term .

Conclusions : The ACA appeared to become a little bit narrow after 1 year post-LPI, which may be due to inceased lens volume. So the study results suggests continuous follow-up is required for narrow-angle patients even after LPI.

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Keywords : anterior segment optical coherence tomography; laser peripheral iridotomy ; primary angle closure suspect.

Introduction

Measurement of the anterior chamber parameters is very significant in various aspects of ophthalmology, such as glaucoma risk evaluation[1,2] and surgical planning and intraocular lens (IOL) power calculation[3,4]. Accurate measurement of anterior chamber volume (ACV), anterior chamber depth (ACD) or the anterior chamber angle (ACA) has been difficult historically due to technology limitations. Development of Anterior Segment Optical Coherence Tomography (AS-OCT) allows rapid and precise anterior chamber measurement[7,8,9]. Currently, the primary angle-closure glaucoma (PACG) classification is primary angle-closure suspects (PACS), primary angle-closure (PAC) and primary angle-closure glaucoma(PACG). Primary angle-closure suspects (PACS) are those having greater than 180° to 270° of iridotrabecular contact. In addition to the same degree of iridotrabecular contact, PAC cases would have elevated IOP as well and PACG cases would have elevated IOP with glaucomatous optic neuropathy[5,6]. Limited studies have evaluated the effect of LPI in eyes with PACS measured with AS-OCT. In this study, we used the AS-OCT, to investigate the anterior segment biometric parameters in PACS patients for better understanding factors that impact AC measurements after LPI.

Method : This prospective study was performed from February 2017 to July 2018 at the Glaucoma Clinic,National Institute of Ophthalmology and Hospital, Dhaka. All procedures adhered to the Declaration of Helsinki and were conducted in accordance with the approved research protocol. Informed consent was obtained from each patient. The

study comprised of 28 PACS patients.

Inclusion& Exclusion criteria : diagnosis of PACS was based on gonioscopy, i.e. posterior trabecular meshwork not visible in at least three quarters. Absence of glaucomatous optic neuropathy or visual field defects or visually significant cataracts prior to enrollment. Exclusion criteria consisted of a history of an acute attack of primary angle closure. Neovascular, inflammatory, or other secondary types of angle closure.Patients with history of previous ocular surgery or trauma were excluded as well.

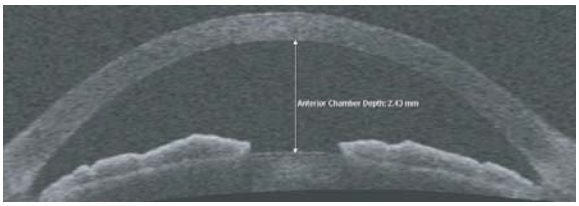


Fig.1 anterior segment imaging with AS-OCT

Before performing LPI, all participants underwent a comprehensive ophthalmologic evaluation including best corrected visual acuity (BCVA) measurement, slit lamp biomicroscopy, IOP (mmHg) measurement with a calibrated Goldmann applanation tonometer, gonioscopy, dilated fundus examination with a 78 diopter lens and 24-2[2] Humphrey visual field examination (Carl Zeiss Meditec AG, Jena, Germany). Preoperative values of anterior chamber depth, volume and angle of anterior chamber were measured in all cases using Anterior Segment OCT (SL-OCT,Heidelberg Engineering,Germany). The anterior chamber angle was graded as: 4, a wide open angle with visible ciliary body band; 3, an angle with visible scleral spur; 2, an angle with visible anterior trabecular meshwork; 1, an angle in which only the Schwalbe line is visible. When AS-OCT was used. Patients were instructed to fixate on the target. All eyes were imaged in room light without pupil dilation. The instrument software automatically detected the boundaries of cornea, iris, and lens for each image, as shown in Figure 1. Manual adjustment was made if the software failed to detect the boundaries at the correct location. Angle width parameters included the angle opening distance 750 (AOD 750), the trabecular iris space area 750 (TISA 750), the angle recess area 750 (ARA 750). LPI was performed with laser machine from Carl Zeiss Meditec, Germany. After performing LPI; after 2 weeks & after 1 year Anterior chamber depth (ACD), anterior chamber volume (ACV), and Anterior chamber angle (ACA) parameters (eg. angle opening distance [AOD750], angle recess area [ARA750], and trabecular iris space area [TISA750]) were measured again . Anterior chamber parameters were compared before and after performing LPI.Statistical Analysis: Commercial software was used for statistical analysis. P values of 0.05 or less were labeled as statistically significant.

Results

Twenty eight eyes of 28 PACS patients including 18 female and 10 male subjects were included in this study.When both eyes had same diagnosis,right eye was considered for studyMean age: Mean age of the study patients was 44.2±6.2 (range: 38-51) years.

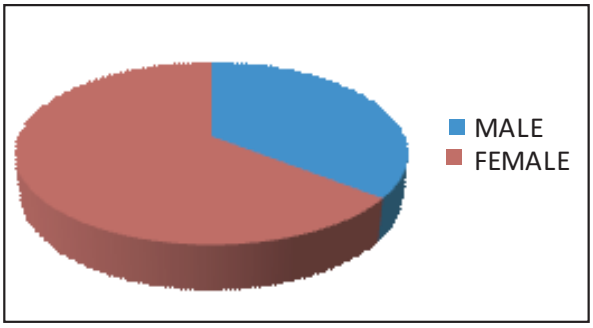


Fig-2 : distribution of study subjects according to gender

Refractive statusof the study subjects :among the 28 study subjects , 22 patients were hypermetropic,4 were emmetropic and 2 were myopic according to their refractive evaluations.

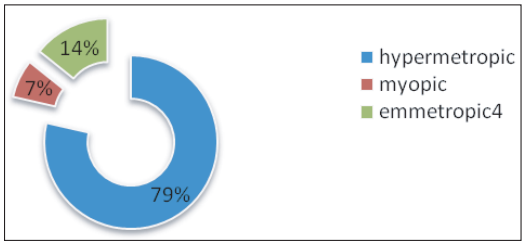
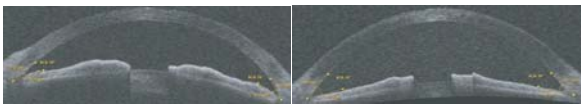
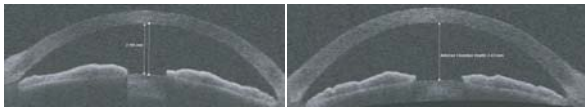


fig:pattern of refractive status among study subjects

Anterior chamber angle (ACA): anterior chamber angle width increased significantly from grade 1to 2 preLaser to grade 2 to 3 post-laser in most patients . All the three ACA parameters (AOD750 [from 0.16mm to 0.27 mm], TISA750 [from 0.08 mm to 0.13 mm], and ARA750 [from 0.09 mm to 0.14 mm]) were found to be increased at 2 weeks post-LPI (all P<0.001).The measurements decreased a little bit at one year post LPI.



Anterior Chamber depth (ACD): There was significant increase in anterior chamber depth after LPI in study.Mean ACD changed from 2.09 mm±0.16 pre LPI to 2.56±0.40 mm post LPI(P<0.001).



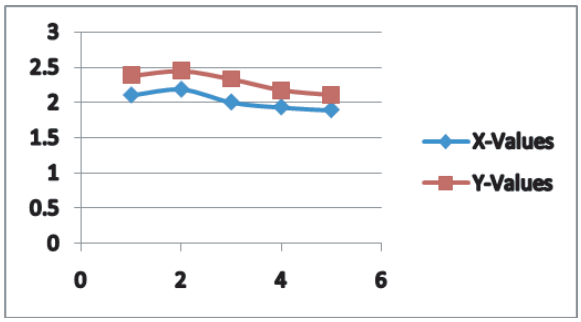
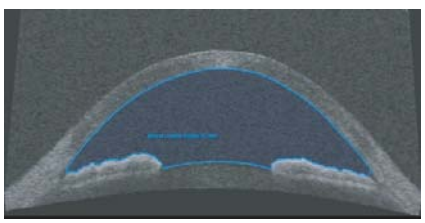


diagram : 1 Represents the mean pre & post operative anterior chamber depth(the X-values represent the prelaser AC depth and Y- values represent the average post laser AC depths .

Anterior chamber volume(ACV) : after performing LPI the mean ACV changed from pre LPI 163.23±0.26mm^3 to post LPI 185.35±0.16mm^3.



Discussion : In our study, majority of the PACS patients were female which matches with several other study findings. We also found that a significant number (78%) of patients were hypermetropic among the patients. This finding also comes in accordance with previous studies as well. After performing LPI although the angle opened and AC volume increased compared to that of the pre LPI measurements, we could not differentiate specifically which factors was more significant. As found in previous studies on PACS patients, we have also observed an increase in AC depth after LPI. As after 1 year post-LPI, all three ACA parameters remained almost unchanged from their pre-LPI value, possibly the Lens Volume significantly might have increased and was associated with ACA narrowing in long term. The study has few limitations. Such as , the results may not be randomized to racial groups other than Bangladeshi subjects. If the follow up period as well as number of study subjects were increased the outcome could be studied more effectively. Use of imaging techniques such as ultrasound biomicroscopy (UBM) or newer versions of anterior segment OCT could be done to evaluate the biometric values of the anterior segment more efficiently .

Conclusion : In this study, we have noticed a significant increase in anterior chamber depth and widening of anterior chamber angle width as well as increase in anterior chamber volume after laser peripheral iridotomy in PACS eyes as measured by anterior segment OCT. The anterior chamber angle (ACA) appeared to become a little bit narrow after 1 year post-LPI, which may be due to inceased lens volume. So continuous follow-up is required for narrow-angle patients even after LPI.

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Original Article

Relationship of Vessel Density by Optical Coherence Tomography Angiography (OCT-A) in Healthy, Glaucoma Suspect and Glaucoma Eyes

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Abstract

Purpose : To evaluate optic nerve vascular density using optical coherence tomography angiography (OCTA) in patients with primary open angle glaucoma (POAG), glaucoma suspect and normal eyes

Methods : This is a prospective, observational study including 50 eyes in total and divided into 3 groups; 20 eyes with POAG, 20 glaucoma suspect eyes, and 10 age-matched healthy eyes as controls. Vessel density was assessed as the ratio of the area occupied by the vessels in 3 distinct regions: 1) within the optic nerve head; 2) in the 3 mm papillary region around the optic disc; and 3) in the whole image (4x4). The potential associations between vessel density and structural, functional measures were analyzed.

Results : There was a statistically significant difference for the peripapillary vessel density and optic nerve head vessel density among all the groups ($p < 0.001$). Control eyes showed a significant difference for all measured vessel densities compared to glaucomatous eyes (p values from 0.001 to 0.024). The optic nerve head vessel density, superior and inferior papillary area vessel density were highly correlated with mean overall, superior and inferior RNFL thickness in POAG eyes ($p = 0.04$, $p = 0.02$ and $p = 0.04$ respectively).

Conclusions : Eyes with glaucoma could be differentiated from glaucoma suspect eyes, which also could be differentiated from normal eyes using OCTA-derived retinal vessel density measurements. This OCT angiography may provide new structural parameters that could potentially be used by clinicians to diagnose glaucoma at earlier stages.

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Introduction

Glaucoma is a progressive optic neuropathy with unknown etiology characterized by degeneration of retinal ganglion cells (RGC) and their axons resulting in a characteristic appearance of the optic disc and visual field loss[1]. There is increasing evidence that optic nerve blood flow impairment and microcirculatory deficiency may have a role in the pathogenesis of glaucoma. Although the details of this relationship have not been established precisely. This is in part due to the instrumentation that has been available and their difficulty of accurately measuring ocular blood flow[2,3] .

Glaucoma suspect patients represent a group of individuals with risk factors such as elevated IOP or ocular findings such as optic disc cup enlargement that are suspicious for glaucoma, but often do not have the classic optic nerve neuroretinal rim loss or characteristic visual field defects to meet the formal definition of definite glaucoma[4]. In terms of functional assessments, visual field (VF) testing using standard automated perimetry remains the standard criterion for glaucoma diagnosis and assessment, but it has substantial variability, often with poor reproducibility[5]. Optical coherence tomography (OCT) gives an objective measurement of retinal nerve fiber layer (RNFL) thickness and/or ganglion cell complex (GCC) which is important for glaucoma assessment but is limited utility in advanced disease and does not relate to cause of disease as opposed to the final presentation[6].

Previously, microvascular changes of optic nerve head and peripapillary area have been demonstrated patients with glaucoma[7,8]. Recent studies suggested that another possible imaging

modality OCT angiography (OCTA), may be used for early diagnosis and monitoring of glaucoma[9].

Although no current technology including Laser Doppler Flowmetry can provide flow rate in smaller retinal vessels, OCTA has been developed as a non-invasive imaging technique that generates three-dimensional, depth encoded images of small and large caliber retinal vasculature within the eye by using motion contrast. It is based on comparison of repeat scans acquired at the same position in the retina to look for changes in the scan of blood flow. It does mapping by A scan to A scan comparison of two or more OCT volumetric cubes which provides detailed vasculature of the retina and optic nerve head, in a noninvasive manner, using OCT scanning alone and without the use of any kind of exogenous dye needed in other vascular imaging techniques like fluorescein angiography [10].

Understanding the relationship between retinal vessel density and glaucoma may increase our understanding of the role of retinal blood flow in the glaucoma cascade and in the pathophysiology of glaucoma. The purpose of this study was to measure the retinal vessel density using OCT angiography in POAG eyes vs glaucoma suspect eyes vs normal eyes, and to investigate correlations between retinal vessel density measurements to other structural parameters like RNFL thickness and functional VF parameters.

Methods

This prospective, observational study was performed between April 2019 and September 2019 at the Ispahani Islamia Eye Institute and Hospital glaucoma clinic. Written informed consent was obtained from each participant. Initially, a total of 25 eyes with POAG, and 23 glaucoma suspect eyes were age matched with 10 eyes from normal controls. Five eyes from the POAG group and 3 eye from the glaucoma suspect group were not analyzed because of poor OCT angiography quality, leaving 50 eyes for statistical analysis.

The diagnostic criteria for glaucoma included all of the following: 1) the presence of characteristic glaucomatous optic disc damage and abnormal thinning of the circumpapillary RNFL; 2) visual field defects consistent with glaucoma, confirmed on at least two visual field examinations; 3) normal open angles on gonioscopy; and 4) no history of any other ocular or systemic diseases causing non-glaucomatous optic nerve damage. Only POAG eyes were included in our study.

Glaucoma suspects who did not meet the aforementioned definition of glaucoma but had ocular hypertension (IOP > 21 mmHg) and an absence of characteristic glaucomatous optic nerve damage or detectable visual field defects[11].

The inclusion criteria for the normal subjects were defined as IOP of _21 mmHg, normal appearing optic nerve head, intact neuroretinal rim and normal RNFL thickness, and normal standard automated perimetry (defined as a glaucoma hemifield test within normal limits and a pattern standard deviation within 95% confidence-interval limits)[12].

The exclusion criteria for all eyes were the following: (1) best-corrected visual acuity less than 6/18, (2) age younger than 30 years or older than 80 years, (3) refractive error greater than +3.00 diopter (D) or less than 6.00 D, (4) previous intraocular surgery except for uncomplicated cataract extraction with posterior chamber intraocular lens implantation, (5) any non-glaucomatous conditions that may cause VF loss or optic disc abnormalities, or (6) inability to perform reliably on automated VF testing. One eye from each participant was imaged and analyzed in a random manner.

All subjects were interviewed regarding their medical history. Thorough ophthalmic examinations included refractive status, slit-lamp biomicroscopy, fundus examination, IOP, central corneal thickness (CCT), and gonioscopy, performed by glaucoma specialists. The RNFL thickness, from a 3.4-mm diameter circle scan centered on the disc, was assessed with Heidelberg SD-OCT. IOP was measured using Goldmann applanation tonometry and CCT was

measured with a handheld ultrasound pachymeter. Visual field tests were performed with the Humphrey Field Analyzer II (Carl Zeiss Meditec, Inc). The system was set for the 24±2 threshold test, size III white stimulus, SITA-standard algorithm.

The OCT-A imaging system provides a noninvasive method for visualizing the optic nerve head and retinal vasculature. The image acquisition technique is optimized for the Split-Spectrum Amplitude-Decorrelation Angiography (SSADA) algorithm described in detail elsewhere[12]. The SSADA method captures the dynamic motion of moving scatters such as red blood cells in a flowing blood vessel and computes a high-resolution three-dimensional (3D) visualization of perfused vasculature. The OCT-A characterizes vascular information at each retinal layer as an en face angiogram, a vessel density map, and quantitatively as vessel density (%), calculated as the percentage area occupied by flowing blood vessels in the selected region.

For this study, we utilized vessel density measurements within the peripapillary RNFL in scans with a 4.5 × 4.5 mm field of view centered on the optic nerve head. Vessel density within the RNFL was measured from the internal limiting membrane (ILM) to RNFL posterior boundary using standard AngioVue software (version 2018.1.0.90). Measurements were calculated in three areas. Whole image vessel density was obtained over the entire 4.5 × 4.5 mm scan field, and within optic nerve head and circumpapillary vessel density (cpVD) was measured in a 750-μm-wide elliptical annulus extending outward from the optic disc boundary, where the inner elliptical contour is obtained by fitting an ellipse to the disc margin on the OCT en face retinal angiogram and the ring width between inner and outer elliptical contour is defined as the circumpapillary region.

For statistical analysis, data are shown as a mean value with the standard deviation. The Kruskal Wallis test was used to analyze the significance of differences among the 3 groups and if there was a difference T test was used to check where the difference came from. Multiple linear regression analysis was used to determine the relationships between the vessel density and traditional glaucoma measurements of structure (OCT-derived RNFL thickness) and function (VF mean deviation, VF pattern standard deviation [PSD]) in glaucomatous eyes. The significance level was set at $p < 0.05$. All analyses were performed with statistical software (SPSS for Windows, version 19.0; SPSS, Inc., Chicago, IL, USA).

Results

A total of 40 eyes from 40 patients were age-matched with 10 eyes from 10 normal controls. Kruskal Wallis testing was first performed to evaluate for overall differences among the groups.

Table 1 summarized the baseline clinical and ocular characteristics of each group. As expected, the POAG patients were treated with medications, whereas the glaucoma suspect and normal eyes were not on glaucoma medications. For treatment in the POAG group, prostaglandin analogue once daily at bedtime was used in 8 eyes, while brimonidine-timolol combination twice daily was used in 7 eyes, and brimonidine tartrate twice daily was used in 5 eyes. The duration of medication use was 2.5±2 years.Overall differences were also found amongst the groups for visual field function (MD [$p = 0.01$] and PSD [$p = 0.03$]) as well as OCT-measured RNFL thickness ($p = 0.004±0.04$).

However, pairwise t-tests with statistically significant differences between both POAG vs suspects ($p = 0.02$) and controls vs suspects ($p = 0.012$) for mean RNFL thickness, help confirm that structural loss often precedes perimetric loss (since glaucoma suspect eyes had lower RNFL thickness compared to normal) (Table 1). There was no otherwise statistically significant difference found for VF MD, VF PSD, and C/D area ratio between the normal and glaucoma suspect patients.

Table 1. Characteristics of the study groups- Demographic and ocular

Variables	POAG (n: 20)	Glaucoma suspect (n:20)	Control/ Healthy(n:10)	P value*
Age (years)	55.375±5.2	51.375±5.2	45.375±5.2	0.01
Gender (Male/Female)	12/8	13/7	5/5	0.01
Central corneal thickness (m)	535±16	524±16	544±16	0.03
Number of IOP lowering drops	1.6±0.5	0	0	0.01
Visual ®eld mean deviation (dB)	-1.6±1.8	-0.78±1.86	-0.25±1.6	0.01
Visual ®eld PSD (dB)	2.6±1.9	1.55±2.25	1.35±1.4	0.02
C/D	0.73±0.17	0.48±0.12	0.3±0.1	<0.001
Mean RNFL thickness (m)	75.9±11.5	87.8±11	98.3 ± 5.9	<0.001
Superior RNFL thickness (m)	93±19.9	108.56±18.8	176±16.3	<0.001
Inferior RNFL thickness (m)	97.5±19.04	113.66±20.35	125 ± 15.8	<0.001
Optic nerve head vessel density (%)	60.1±7.8	72.04±7.2	81.6 ± 4.7	<0.001
Peripapillary vessel density (%)	65.03±5.63	76.83±6.64	82.03± 3.95	<0.001
Whole area vessel density (%)	72.44±6.03	80.7 ± 5.3	83.4±1.96	<0.001

In OCTA-measured vessel density, overall differences were found between the groups for whole area, optic nerve head, and peripapillary vessel density (all p<0.001, Table 1).

Figure 1 showing the OCT A vessel density. This showed that optic nerve head vessel density in POAG group were more strongly linked to RNFL thickness than to any other variables (p = 0.65 for peripapillary vessel density, p = 0.04 for optic nerve head vessel density and p = 0.112 for papillary area vessel density).

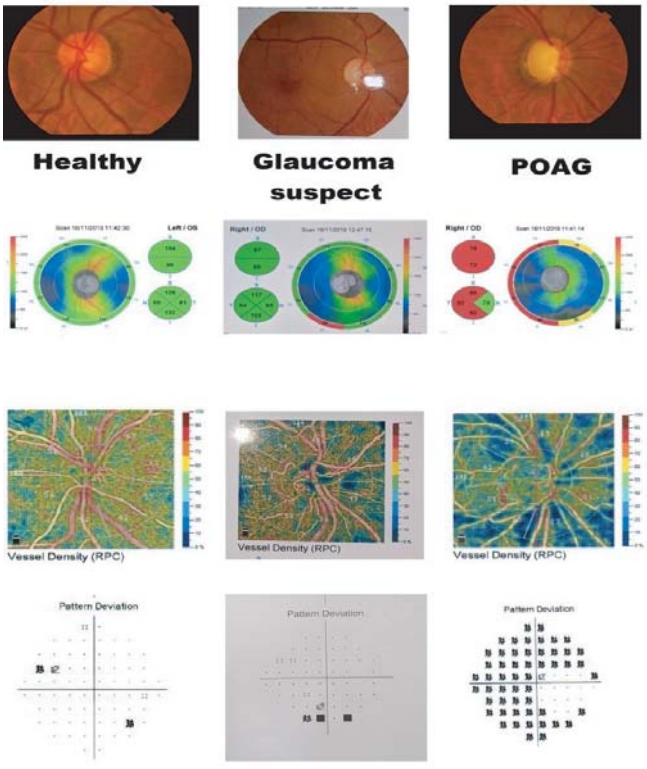

Figure 1: Vessel density map in healthy, glaucoma suspect, and glaucoma eyes. First row (from top): Colour fundus photo, Second row (from top): RNFL thicknes by OCT, Third row (from Top): Vessel density map measurement by OCTA. Bottom row: Pattern deviation in HVFA

Discussion

This study using OCT angiography, we were able to demonstrate lower retinal vessel densities for eyes with glaucoma compared with normal eyes, as well as, compared with glaucoma suspect eyes. In addition, one of the most revealing findings in our study is that we were able to demonstrate lower retinal vessel densities in our glaucoma suspect group (which includes patients with essentially normal perimetry testing but mild reduction in OCT-derived RNFL thickness measurements) compared with normal eyes. Thus, we were able to distinguish patients with pre-perimetric glaucoma from age-matched normal eyes using OCTA-derived vessel density measurements, and found correlation with mild reduction in OCT-derived RNFL thickness measurements. This may have important implications in increasing our understanding of the pathophysiology of glaucoma and its relationship with retinal vasculature, as previously suggested and in agreement with previous studies[11,12,13].

Functional loss in our study as seen in standard automated perimetry which is widely used in the clinical setting and it has been shown previously that structural parameters such as loss of RNFL thickness can be typically identified before perimetric loss is detected since structural loss can precede functional loss by years[14,15] . A lower vessel density found in our cases of early POAG and glaucoma suspect eyes suggests that the retinal vasculature attenuation may start early in the course of glaucomatous disease cascade. Previously, Kerr et al[16] reported that patients with untreated POAG had a reduction in lamina cribrosa and temporal neuroretinal rim blood flow compared to patients with ocular hypertension.

They also indicated that reduced ONH blood flow may be an early event in glaucoma which is consistent with many studies. Additionally, Pareira et al[17] evaluated retinal vessel density in a 3.46 mm circle with scanning laser ophthalmoscope from a Fourier domain OCT and concluded that vessel density may have a clinically relevant influence on the RNFL distribution. Thus, it is possible that retinal vessel attenuation may become a parameter that is particularly useful for detection of early glaucomatous disease. Future studies that include more advanced levels of glaucoma and longitudinal data analyses are needed to determine whether vessel density continues to be correlated to functional loss.

Most patients in our glaucoma group were receiving multiple ocular antihypertensive eye drops. In our statistical analyses of our sample, use of an ocular antihypertensive eye drop or any subclass was not correlated with the vessel density measurements but our study may not have been powered to detect such a difference. Therefore, it is not possible to determine their individual effects on vessel density with our small sample size, and we cannot entirely rule out the possibility that the glaucoma drops could somehow be responsible for the vascular changes. We consider this less likely because glaucoma medications were previously reported to have been no significant effect[18] . We are planning to perform a study in the future to remove the confounding effects of the drugs.

In addition, although our results noted statistically significant differences between groups, our sample size was not very large and additional patient recruitment will likely address this shortcoming. All these measurements were taken on initial visits with the patient before treatment was initiated, if any.

The current study is cross-sectional and needs longitudinal follow up to further assess associations over time. Furthermore, we included both large and small vessel measurements in our data analysis, and thus cannot comment on the relative contributions of micro- vs. macro-vessels towards their individual contributions to glaucoma.

Conclusion

This study reports that retinal blood vessel density using non-invasive OCT angiography showed a stepwise decrease from normal eyes to glaucoma suspect eyes to glaucoma eyes. Furthermore, this difference in vessel density

was seen in all three of the anatomic sites measured. This shows that OCT angiography may provide new structural parameters that could potentially be used by clinicians to diagnose glaucoma at earlier stages.

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Original Article

Primary Selective Laser Trabeculoplasty for open-angel glaucoma: Primary versus Secondary treatment outcomes

S M Hossain[1], Z. S. Shahid[2]

Purpose

To compare the outcomes of selective laser trabeculoplasty (SLT) on treatment-naïve, open angle glaucoma(OAG) patients with those of SLT on patients previously treated medically and / surgically, as well as to establish age and gender influenced SLT outcomes.

Design

A retrospective review of patients who received SLT therapy for OAG between October 2016 and September 2019.

Participants

Group A: Treatment-naïve patients(n=59), Group B: Prior medical therapy and / or prior surgery(n=125)

Methods

Group A: Patients were treated with SLT therapy as first line, with medical treatment added as needed. Group B: Patients were treated with SLT therapy as additional therapy to medication and / or surgery. All patients were followed up for minimum period of 12 months.

Out Come Measures

A reduction in Intraocular pressure (IOP) of at least 20% from base line was considered significant.

Results

A total of 320 eyes of 184 patients were evaluated at Deen Mohd. Eye Hospital & Research Center, Dhaka. Group A consisted 137 eyes, while Group B had 183 eyes. A greater than 20% reduction in IOP was achieved in 72% of eyes at 1 month, 78% at 3 month, 75% at month 6 and 79% at 1 year. The following percentage reduction in IOP was found in different groups : Group A 50.42% , Group B 29.10%.

Conclusion

Primary SLT achieved significant reductions in IOPs in treatment -naïve eyes as well as previously medically and surgically treated eys with OAG. Statistically significant higher reduction in IOP at 1 year after SLT was observed in treatment-naïve eyes, patients older than 60 years when compared with younger patients, female patients (51%) when compared with male patients (47%).

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Introduction

Glaucoma is a progressive multifactorial disease characterized by damage to the Optic nerve. It is strongly associated with elevated intraocular pressure (IOP) but may also occur with IOP in the normal range. Glaucoma results in progressive Visual field loss and is the leading cause of blindness worldwide, second only to Cataract. It is predicted that by 2020, close to 80million people will have glaucoma, the majority open-angle glaucoma (OAG).[1]

The mainstay of glaucoma treatment is lowering of IOP to slow or prevent further progression and visual loss. There is no simple answer to how to treat open-angle glaucoma (OAG).

To date, the only proven modality to slow down the disease progression is to lower the intraocular pressure (IOP). This may be achieved by either medical, laser, or surgical means.

This study was conducted to determine if there is a difference in the outcomes of selective laser trabeculoplasty (SLT) therapy in the treatment-naive OAG patients compared with those who have had prior medical or surgical therapy. The study also looked at the possible influence of age and gender on the outcomes of SLT.[2]

To slow down or halt the progression of glaucomatous nerve fibre loss, a target IOP should be set before commencing any treatment.3 This is usually accepted as a reduction in IOP of 20% from baseline.

The European Glaucoma Society (EGS) has shown that first-time SLT is effective in 85% of

cases and reported drop in IOP of 6 mmHg – 9 mmHg was observed[3].

Since receiving FDA approval in 2001, SLT has increasingly been adopted into practice[4]. SLT has proven its safety and repeatability as seen in the EGS guidelines[3], building on the research done by the Glaucoma Laser Trial.

Research Methods and Design

This study was conducted as a retrospective chart review of patients who received SLT Therapy for OAG over a 3–year period, spanning from Oct-2016 to Sept-2019, at Deen Mohd. Eye Hospital and Research Center, Dhaka, Bangladesh.

AIMS

§ To determine the outcome of SLT on treatment-naive OAG Patients (Group A).

§ To determine the outcome of SLT on OAG patients previously receiving topical and/or systemic IOP-lowering agents and/or previously operated upon (Group B).

§ To determine if SLT outcomes are influenced by gender, age or previous glaucoma treatment.

Statistical Analysis

The primary outcome of interest was SLT induced IOP reduction; successful SLT was defined as having a reduction of 20% of pre treatment IOP at 1 year post treatment. Appropriate student's t tests were used to detect statistical significance; for categorical variables X2 tests was used. All statistical analyses were performed using the stata 7.0 software. One hundred & eight four patients (320 eyes) were enrolled in the study.

Table 1 : Age and gender distribution.

Age group	Male	Female	Total
30-39	10	3	13
40-49	9	15	24
50-59	27	35	62
60-69	30	34	64
70-79	9	10	19
80-89	1	1	02
Total	86	98	184

Results

A total of 320 eyes of 184 patients were evaluated at Deen Mohd. Eye Hospital & Research Center, Dhaka. Group A consisted 137 eyes ,while Group B had 183 eyes. A greater than 20% reduction in IOP was achieved in 72% of eyes at 1 month, 78% at 3 month, 75% at month 6 and 79% at 1 year. The following percentage reduction in IOP was found in different groups: Group A 50.42%, Group B 29.10%.

Table-2 : Mean Intraocular Pressure Drop Group A versus Group B

RE, Right eye; LE, Left eye; SLT, selective laser trabeculoplasty

Group (P<0.001)	Time	Mean IOP (mmHg)	Mean IOP reduction	Reduction from baseline (%)
Group A	Pre SLT Therapy	26.14	-	-
	1/12 post SLT	15.86	-10.28	39.32
	3/12 post SLT	15.42	-10.72	41.00
	6/12 post SLT	13.34	-12.80	48.96
	12/12 post SLT	12.96	-13.18	50.42
Group B	Pre SLT Therapy	19.72	-	-
	1/12 Post SLT	14.45	-5.27	29.11
	3/12 Post SLT	15.54	-4.18	21.20
	6/12 Post SLT	15.86	-3.86	19.57
	12/12 Post SLT	13.98	-5.74	29.10

In group A the mean pre SLT was 26.14 mmHg; it was 15.86 mmHg at 1 month after SLT, 15.42 mmHg at 3 months, 13.34 mmHg at 6 months and 12.96 mmHg at 1 year. There was a 50% reduction in mean IOP from the base line value. (Table 2 & Figure 1)

In group B the mean pre SLT IOP was 19.70 mmHg it was 14.45 mmHg at 1 month, 15.44 mmHg at 3 months, 15.86 mmHg at 6 months and 13.98 mmHg at 1 year. There was a reduction in mean IOP of 29% at 1 year (Table 2 & Figure 1).

Evaluating for age it was observed that patients from the 50 - year age group had a reduction in IOP of 38% at 1 year, those in the 60 – year group had a reduction of 48% and those in the 70 - year had a reduction of 50%.

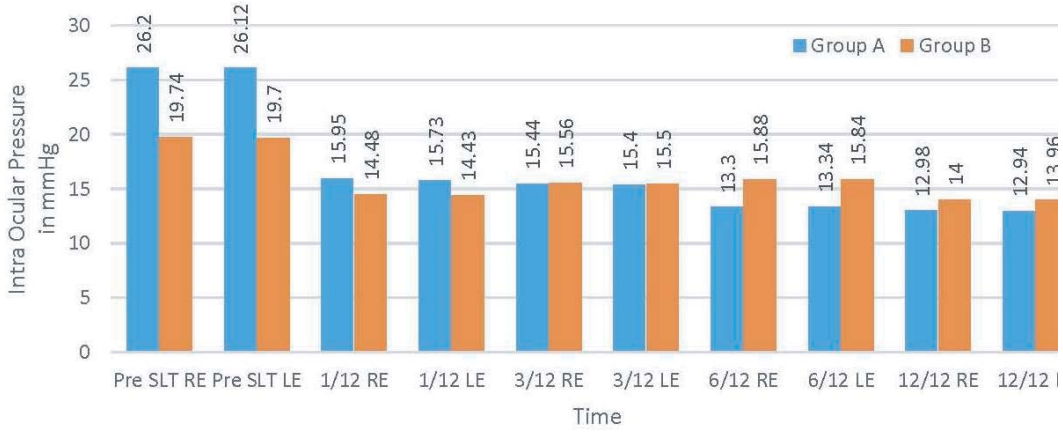

Figure 1: Mean intraocular Pressure Group A versus Group B

RE, Right eye; LE, Left eye; SLT, Selective Laser Trabeculoplasty

Analyzing the differences in gender, 80% of male patients (n=86) had a reduction in IOP of at least 20% at 1 year compared with 78% of female patients (n=98).

Analyzing the differences in gender, 80% of male patients (n=86) had a reduction in IOP of at least 20% at 1 year compared with 78% of female patients (n=98).

In SLT literature, the most commonly measure of success is a minimum IOP reduction of 20% from baseline IOP after SLT at a specified time point, without the need for further intervention.

These analyses are exploratory but support primary SLT to be effective and safe in treatment – naive OAG eyes.

Discussion

Laser settings

The laser is focus at the pigmented trabecular meshwork and 100 non – overlapping shots are applied to 3600 of the pigmented trabecular meshwork. The spot size is 400-microns and pulse duration 0.3 ns. The large spot size results in low fluencies (ms/cm2).

Our study shows SLT only affects pigmented trabecular meshwork cells5 explaining the greater affects in pigmented population like ours. In vitro investigation showed that non-pigmented cells did not experience collateral thermal damage 6.

SLT laser energy recruits macrophages to the trabecular meshwork by increasing the expression of cytokines and cellular mediator activities. Macrophages remove obstructive proteins and remodel the trabecular meshwork, thus improving aqueous outflow and reducing the IOP[7].

It is also noted that these biological changes may take up to 6 weeks to take effect[8].

In this study, only patients with a minimum of 1-year follow up were included. We in and et al. looked at the survivability of SLT and found that around 50% of patients had treatment failure at 2 years[9], and this finding was reproduced by Bovell et al[10].

There was no disparity between genders. The percentage of patients who had a reduction in IOP of more than 20% were similar in both groups, with 80% of male patients and 78% of female patients reaching the target.

There are known complications of SLT therapy. The EGS guidelines list anterior chamber inflammation, iritis, anterior chamber bleed and IOP spikes as possible complications3. None of the eyes treated during the trial were noted to have any of the side effects mentioned.

This study did show that SLT as the first line was a viable option, as seen by Waisbourn[11].

Our study had 320 eyes of 184 patients. Though

relatively small sample size, but comparable with international studies.

Conclusion

In evaluating the efficacy, ease of application and favorable risk profile, this study shows, primary SLT to be effective and safe in treatment-naïve open angle glaucoma (OAG).

Acknowledgements

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Disclosures

The Author has nothing to disclose.

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Outcomes of Traumatic Glaucoma in Military Tertiary Hospital

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Abstract

Background : Traumatic eye injury is one of the most common causes of unilateral blindness worldwide as well as in our country and itremains a core root of avoidable blindness. Traumatic glaucoma can result in severe visual impairment even it may land to total blindness.Recognition of factors related to poor visual outcome, appropriate medical therapy, surgical intervention whenindicated and careful follow up will help in preserving vision in these patients. This study is aimed at determining thefrequency, clinical forms and types of therapy of traumatic glaucomas as seen in our hospital.

Objective : To report the incidence,modes of injury, treatment, and short term outcomes in eyes with post-traumatic elevated intraocular pressure (IOP) with optic neuropathyin a tertiary military Hospital.

Methods : This is a retrospective study where all consecutive patients with ocular injury who received treatment at the Eye Department, Combined Military Hospital, Dhaka from January 2017 to September 2019 were selected for the study. All subjects had thorough ophthalmic examinations and related ocular and systemic investigations.The patients with ocular injury and subsequently developedglaucoma were selected. Extracting from the medical records the demographic data, visual acuity at presentation,clinical form of glaucoma , various therapies for management.

Result : A total of 5357 patients were seen during this study at the Glaucoma clinic of CMH Dhaka, 259 of them had oculartrauma out of which 33(15.06%) was traumatic glaucoma. Rupture globe, Hyphaema, Angle recession glaucoma were the common clinical manifestations of traumatic glaucoma.The clinical types of traumatic glaucoma are secondary open angle glaucoma (54.55%) which is higher than thesecondary closed angle type (45.45%). 22 (66.66%) of these patients had medical interventions with antiglaucoma drugs and rest 11 (33.33%) needs surgical intervention in addition to medical intervention.

Conclusion : Secondary Glaucoma after Ocular trama is a common consequence. Timely intervention can save permanent visual impairment of patient.Glaucoma is 15.06%, this glaucoma may result in severe visual impairment if treatment is not started early and follow up protocols is not maintained as scheduled.

Keywords : Traumatic glaucoma; Visual impairment; Secondary open angle; Secondary close angle; Hyphaema.

Table 01 : Age distribution of patients

Age	Male	Female	Frequency
20-29	14	3	17 (51.52%)
30-39	11	1	12 (36.37%)
40-49	3	-	3 (9.09%)
50-59	1	-	1 (3.03%)
Total	29	4	33

Table 02 : Types of ocular trauma

Type of trauma	Number of trauma	Number of traumatic glaucoma	Percentage of traumatic glaucoma
Open globe injury	48	9	18.75
Close globe injury	211	24	11.37
Total	259	33	15.06

Table 03 : Presenting visual acuity

Visual acuity at presentation	Category of vision	Frequency
6/6-6/9	Normal vision	3
6/12-6/24	Moderate visual impairment	5
6/36-6/60	Severe visual impairment	16
<6/60- NPL	blindness	9
		Total - 33

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Table 04 : Angle status in traumatic patients

Clinical type of traumatic glaucoma	Medical	Surgical	Total / Frequency
Open angle glaucoma	15	3	18(54.55%)
Close angle glaucoma	7	8	15(45.45%)
Total	**22**	**11**	**33**

Table 05 : Visual acuity after treatment Visual acuity

after treatment	Category of vision	Frequency
6/6-6/9	Normal vision	11
6/12-6/24	Moderate visual impairment	11
6/36-6/60	Severe visual impairment	8
<6/60- NPL	blindness	3
		Total - 33

Table 06 : Types of closed globe injury

Types of Close globe injury	Frequency N %
Hyphaema	12 (50%)
Lens dislocation/subluxation	4 (16.66%)
Angle recession glaucoma	7 (29.16%)
Others	1 (4.1%)
Total	24

Table 07 : Types of open globe injury

Types of open globe injury	Frequency N %
Penetrating injury with iris prolapse	2 (22.22%)
Penetrating injury with retained intraocular FB	2 (22.22%)
Rupture globe	5 (55.55%)
Total	09

Introduction

Traumatic eye injury is one of the most common causes of unilateral blindness worldwide as well as in our country and it remains a core root of avoidable blindness worldwide.Ocular trauma is an event witnessed very frequently by most ophthalmologists with documenting an incidence of 4.5%, 0.4% being bilateral[1]. Maximum injuries are related to domestic accidents and sporting activities with young males being most afflicted.[2]It can occur in a shape of closed globe injury or open globe injury. Traumatic glaucoma can occur soon after the injury or many years later with some series issues to address. Traumatic glaucoma refers to a heterogeneous group of posttraumatic ocular disorders with different underlying mechanisms that lead to the common pathway of abnormal elevation of intraocularpressure (IOP) and increased risk of optic neuropathy.[3]

The most common types of blunt trauma are the following:

- ✓ Sports injuries (eg, boxing, ball, airsoft gun toys)
- ✓ Motor vehicle accidents (eg, airbag deployment, otherfacial trauma)
- ✓ Assaults
- ✓ Falls from height
- ✓ Military combat injuries
- ✓ Accidents (eg, industrial, farm, home) by rocks or stones, fists

Penetrating or perforating ocular injuries can be due to injury from any sharp or high velocity object. The most common sharp objects were sticks, knives, scissors, screwdrivers, fish hooks and nails. High velocity bullet, pellet and grenade splinters are common in military services causing penetrating injuries. In addition road traffic accidents also contributes a bulk amount of reporting to emergency department having penetrating injuries.

Traumatic glaucoma represents a number of conditions in which an eye injury leads to the development of secondary glaucoma. This subtype of glaucoma usually entails several mechanisms that inter play to produce increased IOP. It is important to consider all the pathophysiological mechanisms leading to increased IOP after blunt or penetrating trauma of the eye as they have a direct bearing on the management of patients.

It appears in 5% to 15% of the injured eyes. When traumatic eye injury occurs, glaucomatic optic nerve damage may complicate it either early or late. Recognition of factors related to poor visual outcome, appropriate medical therapy, surgical intervention when indicated and careful follow up will help in preserving vision in these patients. Furthermore, a high index of suspicion will be needed to help in the accurate diagnosis and management of this potentially blindingdisease. Blunt eye injury is commoner in unilateral cases and

traumaticglaucoma in unilateral occurrence can be seen in any type such asangle recession glaucoma, phacolytic glaucoma, red cell glaucoma andHyphaema. Approximately one-third of all hyphema patients exhibit increased intraocular pressure in the early period[4] which increases to 65% in cases of rebleed[5].

Hyphaema and concomitant injuries to ocular structuresfollowing blunt trauma are frequent cause of presentation to the emergency unit in many hospitals.[6,7] Blood and cells from theinjury can block the trabecular meshwork, damaging the trabecularmeshwork and resulting trabeculitis. These can cause increase dintraocular pressure which eventually leads to glaucomatous opticnerve damage. Glaucoma may appear in different clinical forms and indifferent periods after eye injury. The causes of poor vision after bluntinjury include black ball hyphema, secondary glaucoma, cataract,vitreous haemorrhage, commotio retinae and retinal detachment.

Angle-recession glaucoma is classified as a type of traumaticsecondary open-angle glaucoma.[8] There is a 4-9% risk in patients with angle recession to develop secondary glaucoma. [9]It may be underdiagnosed due tothe fact that onset is often delayed and because a history of eye injurymight have been forgotten. It is a common sequela of blunt ocular trauma and one characterized by a variable degree of cleavage betweenthe circular and the longitudinal fibers of the ciliary muscle.This study is aimed at determining the frequency, clinical forms andtypes of therapy of traumatic glaucomas as seen in our hospital.

Background Information on Study Location

The Combined Military Hospital Dhaka is the tertiary hospital centrally located in the capital city. Huge no of ocular trama sustained patients are reporting to this Hospital everyday. We run Glaucoma Clinic twice in a week. All the ocular injury patient with high IOP are routinely evaluated by Glaucoma specialist. The implication of this is that all the patients that were included for the purpose of this study were seen without any bias.

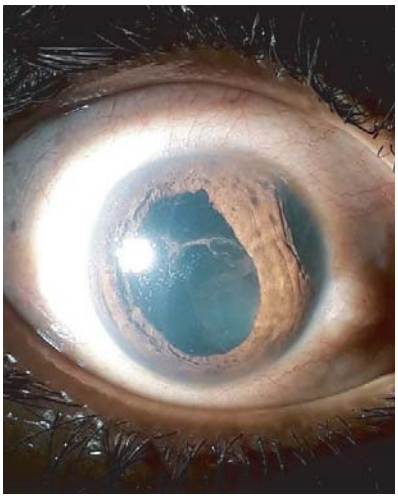

Fig-01 : Traumatic Subluxated lens, iridodialysis with vitreous in AC causing Secondary glaucoma

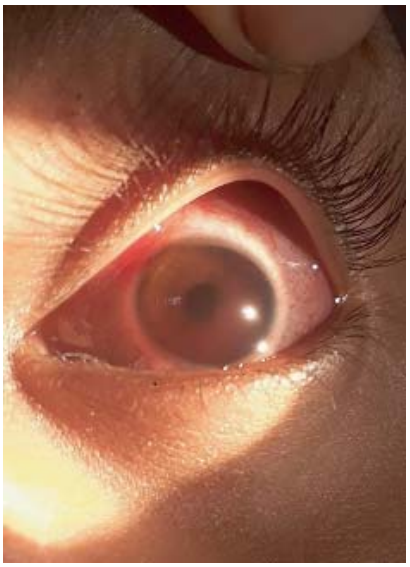

Fig-02 : Traumatic hyphaema causing raised intraocular pressure

Methods

All consecutive patients with ocular injury who received treatment at the Glaucoma clinic of combined military hospital, Dhaka, from January 2017 to September 2019 were selected for the study. Approval for the study was obtained from the institution ethics and review board. Data were not used in any other purpose. All the patients had thorough ocular examinations; Visual acuity assessment with Snellen's chart,

slit lamp biomicroscopeassessment of the anterior segment, intraocular pressure measuredwith Goldman applanation tonometer and Non contact Tonometer (NCT) ,optic disc stereoscopicevaluation with +78D lens, gonioscopy (with 4 mirror goniolens), perimetry (except in patients with open globe injury in which it wastechnically difficult to carry out these procedures). The patients with ocular injury and subsequently developedglaucoma were selected extracting from the medical records thedemographic data, visual acuity at presentation, clinical form ofglaucoma and various therapies for management and follow up after treatment all were recorded and statistical analysis was done.

Inclusion criteria:

Key inclusion criteria included:

1. Patient reported to Emergency &Casualty of CMH Dhaka having history of ocular trauma and was found a rise of IOP during follow up.
2. Adult patients from 20 to 69 years of age
3. Both gender with no prior ocular disease
4. Patients having an easy access to CMH Dhaka with motivation to follow up in scheduled time.
5. Patients only having the background of military services and their dependants.

Exclusion criteria:

Key exclusion criteria included:

1. Patients who are on antiglaucoma drugs predating the ocular injury or who had trabeculectomy before the injury
2. Extremes of age such as children and elderly citizens.
3. Patients having an issue of irregular follow up schedule.

Discussion

Traumatic glaucomas represent a very heterogeneous group of disorders due to a variety of pathomechanisms which increase the intraocular pressure in the early or late phase after traumatic injury (blunt or penetrating injury). Ocular trauma is a major cause of preventable monocular blindness and visual impairment in the world. The visual impairment caused by ocular trauma may arise from traumatic glaucoma. In this study, the frequency of traumatic glaucoma was 15.06% which is slightly higher than value reported by Stanic et al. This showed that contribution of traumatic glaucoma to the global burden of glaucoma blindness particularly in military services in bangladesh is increasing.

The male gender is usually associated with high physical, outdoor activities and so may be prone to injuries and various ocular injuries particularly. This may explain the male preponderance seen in this study in the age range of 20 to 29 years. A good number of males are also affected in the age range of 30 to 39 years. Majority of the ocular injuries resulting in traumatic glaucoma in our study were basically closed globe injuries. This is similar to findings in study conducted by Sujatha et al. Proper health education in prevention of ocular injury should focus on this age group of masculine gender during routine health awareness issues in schools, public places and eye clinics.

Twenty one (63.63%) of traumatic glaucoma had low vision at presentation, while 9(27.27%) were blind out of which one was no light perception (NLP).

In this study, Rupture globe, Hyphaema and Angle recession glaucoma were the common clinical manifestations of traumatic glaucoma. This is similar to what was reported by other authors.

Clinical findings of secondary glaucoma associated with ocular trauma may remain complex; however a good history taking and thorough ocular examination will afford the clinician an opportunity to institute early treatment for traumatic glaucoma. In our study, secondary angle closure due to rupture globe leading to glaucoma in patients with penetrating eye injury while on the other hand; it is

secondary open angle due to hyphaema in patients with closed globe injury. Traumatic glaucoma occurs more frequently as secondary open angle clinical type and in closed globe injury which is similar to what was reported in India.

The onset of early treatment intervention for traumatic glaucoma is aimed at lowering the intraocular pressure which when elevated,because if it remains untreated may contribute to the development of visual impairment. In this study, majority of the subjects, 66.66% were offered medical antiglaucoma treatment while less than 20% had surgical intervention in addition to medical ones.

In a study by Bai et al, it was concluded that for traumatic secondary glaucoma, antiglaucoma medication should be used at the early stage, and surgery should be carried out when medical treatment does not reduce the elevated IOP, or in difficult cases to avoid severe complications.

Results

A total of 5357 patients were seen during the duration of this studyat the Glaucoma clinic, 259 of them had ocular trauma out ofwhich 33(15.06%) were traumatic glaucoma. The age range was 20 to 69years (median 28.73 years). There was a male preponderance inthe age range of 20 to 29 years. Male to female ratio was 7:1. The cause of such male preponderance is due to the patients having the military background where males are more prone to injury. Table 1 showed that 17 (51.52%) of the subjects with traumatic glaucoma werein the 20 to 29 years age group. Majority of the traumatic eye injurywere closed globe (Table 2) among which 24 were found of secondary glaucoma. The percentage in this group is 11.37%.

Table 3 showed twenty one (63.64%) of traumatic glaucoma had Low Vision(moderate and severe visual impairment) at presentation, while9 (27.27%) were blind out of which one (1) was No light perception(NLP). Rupture globe (55.55%), Hyphaema (50%) and Angle recession (29.16%) were the commonclinical manifestation of traumatic glaucoma considering the open & close globe injury.

Traumatic glaucoma being a secondary glaucoma had the clinical type of open angle glaucoma occurring (54.55%) which is higher than theclosed angle type (45.45%).Table4 showed that 22(66.66%) of the patient with traumatic glaucomahad medical treatment only while 11(33.34%) had surgery offered to themas treatment in addition to medical management. After the treatment and follow up protocols only 3(9.09%) patientseneded up in blindness but none required extensive surgery like evisceration.

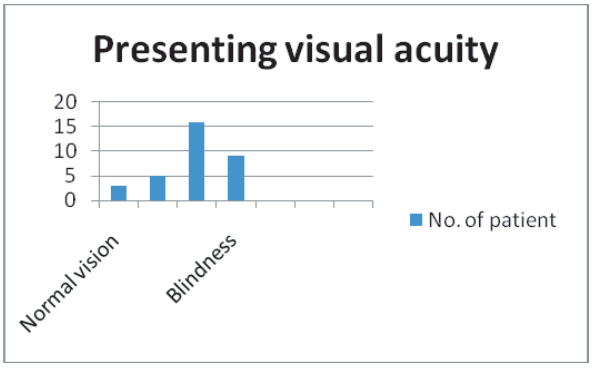

Fig:03 – Types of presenting visual acuity

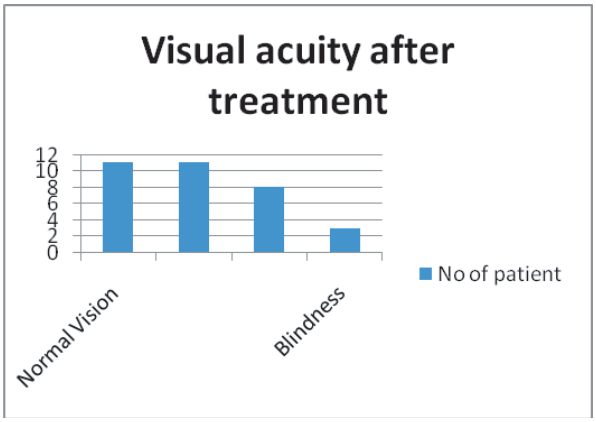

Fig o4 : Visual acuity after treatment

Conclusion

The prevalence of traumatic glaucoma is 15.06%, this glaucoma may result in severe visual impairment if treatment is not started in an early stage. Traumatic glaucoma presents one of the most difficult complications of eye injuries with severe loss of vision. Secondary

open angle glaucoma was the most common clinical type of traumatic glaucoma seen in our study. Both medical and surgical treatment can be offered to these patients in order to prevent visual complications. Regular follow-up is a must for all cases of ocular trauma and evaluation of both eyes should be done, keeping in mind the fact that glaucoma can develop even 15 years post trauma.

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Original Article

Relationship of IOP with Refractive Error

M R A Khan[1]

Abstract

The purpose of this study was to determine the variation of the intraocular pressure (IOP) with spherical equivalent refractive error. A total of sixty (N=60) subjects of which male 42(n=42) and female 18(n=18) within 15-66 years with mean age of 43.6 ±13.2 years were taken for this study. Among the subjects, 30 had myopia and 30 had hypermetropia. IOP was measured with the slit-lamp mounted Goldmann applanation tonometer and refraction done by Topcon autorefractometer and trial lens set. The mean IOP was 18.23 mmHg,with a standard deviation of 4.95 (range 16.64-20.00) mmHg in right eyes and 17.83 mmHg with a standard deviation of 4.95 (range 16.06-19.60) mmHg in left eyes in myopia & in hypermetropia that was 16.80 mmHg,with a standard dediation of 4.16 (range 15.31-18.29) mmHg in right eyes and 18.00 mmHg with a standard deviation of 5.99 (range 15.86-20.14) mmHg in left eyes. The mean myopic refractive error was -3.55D,with a standard deviation of -2.24D (range -2.75 to -4.35) diopter in right eyes and -3.43 D with a standard deviation of -2.46 (range -2.55 to -4.31) diopter in left eyes in myopia & in hypermetropia that was +150 D, with a standard deviation of +0.89 (range +1.18 to +1.82) D in right eyes and +1.44 D with a standard deviation of +0.30 (range +1.33 to +1.55) diopter in left eyes. Result reveals that there were no significant association between IOP and myopia (r=0.18;p=0.17) but there are slight association between IOP and hypermetropia (r=0.33;p=0.01) although no linear correlation exist between IOP and spherical equivalent refractive errors.The intraocular pressure was not affected by gender ('t'=1.60,df=58;p>0.12).

Introduction

Background

Increased IOP is known to be a potent risk factor for developing glaucomaThe ocular hypertension treatment study (OHTS) identified reduced central corneal thickness as a risk factor for glaucoma in patients with IOP between 24 mmHg and 32 mmHg (Ehlers et al,1975).

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Primary open angle glaucoma represents a significant public health problem. WHO has undertaken an extensive analysis of the literature to estimate the prevalence, incidence and severity of the different types of glaucoma on a worldwide basis . Using data collected predominantly in late 1980s and early 1990s, WHO estimated the global population of people with high IOP (>21 mmHg) at 104.5 million. The incidence of POAG was estimated at 2.4 million people per year. Blindness prevalence for all types of glaucoma was estimated at more than 8 million people; with 4 million cases caused by POAG. The different types of glaucoma were theoretically calculated to be responsible for 15 % of blindness, placing glaucoma as the third leading cause of blindness worldwide following cataract. (Glaucoma; AAO: 2004-2005)

Identifying risk factors is important because this information may lead to development of strategies for disease screening and prevention and may be useful in identifying persons for whom close medical supervision is indicated. Strictly defined, a factor can be considered a risk factor only if it predates disease occurrence. Several risk factors and not all risk factors are known; increase the likelihood of the development of POAG. Besides increased IOP, factors known to be associated with an increased risk for the development of glaucoma include advanced age, decreased corneal thickness, racial background and a positive family history.

Over time, it has been shown that myopic refractive errors are associated with thin CCT. Duch et al posited that high ametropia may bias the measurement of CCT(Duch et al ,2001). Hidek et al in their investigation found a significant relationship between IOP and refractive error (Hidek et al ,2004). However, Daubs and Crick found no relationship between refractive error and ocular hypertension (Daubs et al,1981). Studies that have attempted to

investigate the effect of refractive errors on IOP have reported conflicting results. One report showed no correlation between corneal thickness and the level of myopia, whereas another study found the cornea to be thinner in more myopic eyes (Journal of Glaucoma ,June 2006). High myopia is a moderate risk factor of ocular hypertension because myopic discs are more susceptible to glaucomatous damage at a lower IOP than emmetropic discs. Hypermetropia is also related to PACG. In a study, 10 ocular hypertensive patients who developed glaucomatous field defects during observation, 6 were myopic,3 were emmetropic and 1 was hyperopic. But in another study, there are no linear co-relation between CCT and spherical equivalent errors (Cho et al ,1994)

Another study suggests higher responsibility to glaucoma of highly myopic eyes versus non-highly myopic eyes (Investigative Ophthalmology and Visual Sciences ,2000).

Since refractive error especially myopia may influence the amount and rate of progression of glaucomatous optic nerve damage and increased IOP is directly related to glaucomatous damage, it is the purpose of present study to evaluate the variation of IOP with spherical equivalent refractive error.

Rationale of the study

● Case-control and population based studies have reported an association between myopia and POAG.. We can identify patient for developing POAG who are more likely to benefit from early medical treatment and regular follow up. Measuring association between IOP and spherical equivalent refractive error may aid the ophthalmologist in identification of glaucoma patient at high risk of progression and also to estimate target pressure.

Since there are enormous controversy of published papers on relationship of IOP with refractive error and also lack of sufficient related study in Bangladesh, so it was the rationale ground of adopting the current study.

1.4 Objective of the study :

General objective

To investigate the association of IOP with refractive error.

Specific objectives

● To examine relationship between IOP and refractive error

● To estimate association between refractive error with POAG

Ultimate Objectives

● To determine risk factors for developing POAG by measuring IOP and refractive error

3. Materials And Methods

3.1 Type of study: Prospective observational study

3.2 Place of study: National Institute of Ophthalmology, Dhaka and Ahmed Medical Centre, Dhaka.

3.3 Period of study: January, 2007 to December, 2008.

3.4 Study population: Patients attending in OPD of NIO&H, Dhaka and Ahmed Medical Center, Dhaka during the above mentioned period.

5.5 Sample size: A total of 60 patients were selected irrespective of sex; of them 30 are myopic and 30 subjects are hypermetropic.

5.6 Inclusion criteria

● Age between 15 -66 years

● Healthy patients except refractive errors and ocular hypertension

5.7 Exclusion criteria

● Previous ocular surgery

● Ocular pathology such as keratoconus

● Recent contact lens wearer in previous 2 weeks

● Patients with significant astigmatism

3.8 Methods

The ocular parameters of 60 consecutive eyes were measured:

● All the subjects were first refracted to determine their refractive status. Measurements of refractive status were taken with the autorefractor and subjective refraction after adequate preparation of subjects.

● The IOP was assessed with the Slit-lamp mounted Goldmann applanation tonometer.

Three consecutive readings are taken and the average recorded as measured IOP (mIOP) in mmHg.

- Analysis was carried out to examine the relationship:
- Between myopia and IOP
- Hypermetropic and IOP

9.9 Data management analysis

- Data taken from both eyes but right eyes was used for the analysis to avoid duplication
- Pearson correlation analysis was carried out to examine the relationship between IOP or refractive error
- The unpaired t test and linear regression were used to compare the refractive status and IOP
- Data were analyzed using MS Excel and above mentioned test of significance and significance will be assumed at $p<0.05$

3.10 Ethical issues: The ethical committee for approval of the protocol of National Institute of Ophthalmology, Dhaka has approved this protocol and written informed consent were obtained from all patients.

4. Results

A total of sixty (N=60) subjects consisting of males (n=42) and females (n=18) within 15-66 years with mean age of 43.6 ± 13.3 years were used for this study.

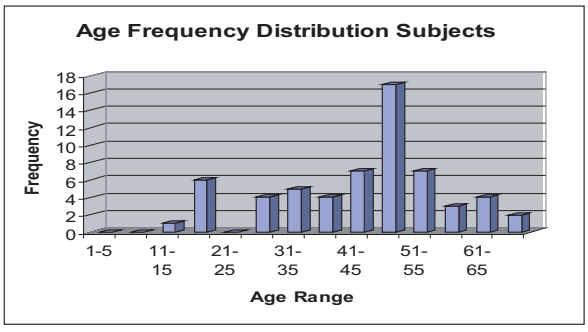

Figure 1 : Age Frequency Distribution of Subject

Of all the subjects, 30 had hyperopia and 30 had myopia.The mean hyperopia was +1.50 ± 0.89D (OD) and +1.44± 0.30D (OS) while the mean myopia was -3.55 ± 2.24D (OD) and -3.43 ± 2.46 D (OS) [Table 1].

Table 1: Mean, SD and Confidence Interval of Refractive Error

Mean, standard deviation and confidence interval of Refractive Error

Refractive Error	Mean ± SD RE (D)	95% confidence interval Mean ± SEM (D)
Myopia		
OD	-3.55 ± 2.24	-2.75 to -4.35
OS	-3.43 ± 2.46	-2.55 to -4.31
Hyperopia		
OD	+1.50 ± 0.89	+1.18 to +1.82
OS	+ 1.44± 0.30	+1.33 to +1.55

Although readings were obtained for both eyes, to avoid duplication of results only the readings of the right eye (OD) were used for analysis.

Table 2 : Mean, SD and Confidence Interval of IOP with Refractive Error

Mean, standard deviation and confidence interval of IOP of subjects with Refractive Error

Refractive Error IOP (mmHg)

Refractive Error	Mean ± SD RE (D)	95% confidence interval Mean ± SEM (D)
Myopia:	OD 18.23 ±4.95	16.64-20.00
	OS 17.83 ± 4.95	16.06 -19.60
Hyperopia:	OD 16.8 ±4.16	15.31-18.29
	OS 18.0±5.99	15.86-20.14

Table shows the mean, confidence interval of IOP of hyperopes and myopes. Pearson correlation coefficient showed that there was no significant association (p=0.16) between IOP and mean spherical equivalent myopia (MSEM), but there was a correlation (p=0.01) between IOP and mean spherical equivalent hyperopia (MSEH), but the linear regression was not significant (ANOVA=0.7, df=29,29,p>o.o5).

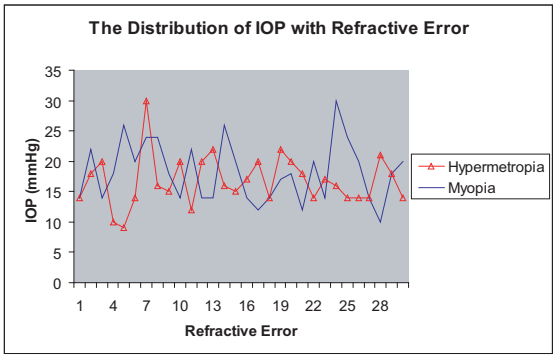

Figure 5 : Distribution of IOP with Refractive Error

Figs shows the scatter plot of IOP vs. Refractive Error .

The difference in mean IOP between males and females was not statistically significant (t=1.60, df=58, p=0.12). Summarily, IOP was not affected by gender.

Discussion

The result obtained from this study showed that refractive error has no effect on the IOP. Pearson correlation coefficient showed that there was no significant association between IOP and MSEM (r=0.18, p=0.17) but there was a correlation between IOP and MSEH (r=0.33, p<0.05) although the linear regression was not statistically significant (ANOVA: F=0.7, df=29, 29, p>0.08).

This finding was consistent with the study of Lee et al who assessed whether IOP is associated with refractive error or axial length and found that neither spherical equivalent refractive error (r=0.014, p>0.05) nor axial length (r=0.027, p>0.05) were significantly associated with IOP(Lee et al,2004) . Lleo et al also showed in their study that there was no significant correlation between mean spherical equivalent refraction and IOP (r=0.054, p=0.231) (Lleo et al,2003). However, Wong et al reported that in their study population, subjects with myopia were 60% more likely to have prevalent glaucoma in contrast to subjects with hyperopia who were 40% more likely to have incident ocular hypertension (Wong et al,2003) .

The difference in mean IOP between males (16.9 ±4.18 mmHg) and females (18.9 ± 5.29 mmHg) was not significant ('t'=1.60, p>0.05) and the 95% confidence intervals were 15.50-18.31 and 18.81 – 21.08 mmHg respectively. Summarily, IOP was not affected by gender. This was consistent with the finding of Lleo et al who reported no significant difference in mean IOP between males (15.47 ± 2.21mmHg) and females (15.37 ± 2.23mmHg) (Lleo et al,2003).

Conclusion

In conclusion, this study has shown that there was no linear correlation between IOP and MSEM and MSEH. This means that IOP is neither affected by MSEM nor MSEH. It was also shown that IOP was not influenced by gender.

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Review Article

Phaco-Goniosynechialysis by using Mori Upright Surgical Gonio Lens

R Raju[1], M S I Bhuiyan[2], O Faroque[3], M Haque[4], I M Munir[5], M H Rahman[5]

Abstract

Phaco-Goniosynechialysis is the procedure of separating the peripheral anterior synechiae (PAS) from the trabecular meshwork using a spatula or microforceps under direct visualization with a goniolens during phaco surgery.

Phaco-GSL was initially suggested as an alternative to trabeculectomy in patients with cataract and primary angle-closure glaucoma (PACG).By performing this procedure in combination with cataract surgery, GSL addresses the root of the problem in PACG—the crowded anterior chamber due to the lens and also iridotrabecular meshwork contact from the PAS. With GSL, the conjunctiva is not incised, allowing for the possibility of a subsequent trabeculectomy if necessary in future.

This procedure is traditionally been done by Swan-Jacob Goniolens which needs tilting of microscope & patients head for proper visualization of angle. So 360º goniosynechialysis is difficult and sometimes not possible. But newer Ocular Mori Upright Surgical GonioLens (OMUSG) provides better approach of that procedure in normal phaco surgery position with easy instrumentation where 360º goniosynechialysis is possible with better outcome.

Keywords : primary angle closure glaucoma, goniosynechialysis, surgical goniolenses.

Introduction

Goniosynechialysis (GSL) is an intraocular surgical procedure designed to mechanically break peripheral anterior synechie (PAS) in eyes with primary angle closure (PAC) or primary angle closure glaucoma (PACG). The aim is to provide long-term lowering of intraocular pressure (IOP).[1]

GSL was first described in conjunction with cataract surgery in 1984 by Campbell and Vella.[2] This procedure physically separates peripheral anterior synechiae (PAS) from the trabecular meshwork using a spatula or microforceps under direct visualization with a goniolens. GSL was initially suggested as an alternative to trabeculectomy in patients with cataract and primary angle-closure glaucoma (PACG).

By performing this procedure in combination with cataract surgery, GSL addresses the root of the problem in PACG—the crowded anterior chamber due to the lens and also iridotrabecular meshwork contact from the PAS. With GSL, the conjunctiva is not incised, allowing for the possibility of a subsequent trabeculectomy if necessary.

Indications

Typically, a phakic patient with primary angle closure, PACG, or CACG with elevated IOP and at least 50% of the angle sealed with PAS is a good candidate for phaco-GSL. The amount of PAS necessary is debatable. If there is only a small amount of PAS, however, the IOP-lowering effect of GSL is likely to be negligible.

In early studies, GSL was initially performed on patients with PACG who had synechiae formation within the past 6 to 12 months.[3-5]

It is generally accepted that GSL is less effective in eyes with CACG. Eyes with CACG that have had PAS for a long period of time likely have greater underlying trabecular dysfunction than eyes with "fresh" PAS from a recent acute angle-closure attack. The eyes with chronic IOP elevation are likely to have more advanced optic nerve damage as well and may not tolerate the postoperative IOP spike that can accompany

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phaco-GSL.

Contraindications

Subjects with advanced glaucoma and marked visual field loss involving fixation may not be suitable to undergo phaco-GSL because of the possibility of postoperative pressure spike. It is advised in such cases in which IOP spike needs to be avoided at all costs to avoid the risk of "wipe-out", that alternative procedure be performed, e.g. combined phacotrabeculectomy.[6-8]

Patients with hazy corneas, especially in the periphery, are a relative contraindication because the view of the angle is more difficult in such eyes.

Furthermore, this procedure should be used with caution in patients who are on anticoagulants due to the increased risk of uncontrolled bleeding and hyphema with GSL.

Surgical steps

Peribulber or sub-Tenon's anesthesia is preferred since some patients do complain of pain during the GSL itself. Phacoemulsification with IOL implantation should be performed as normal. Once the IOL is implanted the viscoelastic should not be removed- in fact it is usually necessary to fill the AC with more viscoelastic as a deep AC improves visualization. Visco-cohesive agents are preferred.

There are several goniolenses available. The commonly used lens is the Swann-Jacob goniolens. But I prefer the Mori Upright Surgical Gonio Lens which has the distinct advantages over the Swann-Jacob goniolens. There is no need of manipulating the patient's head and tilting the microscope in Mori Gonio Lens which is mandatory in Swann-Jacob goniolens. GSL is done in normal phaco position of patient head & microscope which provide a great comfort both for the patient & the surgeon. Moreover it is very easy to approach in all quadrant of AC to release PAS by Mori Gonio Lens which is not possible by Swann-Jacob goniolens in most of the circumstances. Due to less manipulation, less time is consumed in the procedure by Mori Gonio Lens. The main disadvantage of Mori Gonio Lens is less magnification of angle which can be overcome by magnification of microscope. In this literature I am going to describe the Phaco-GSL procedure by Mori Upright Surgical Gonio lens.

After completion of phacoemulsification with IOL implantation, viscoelastic is placed on the concave surface of the goniolensto act as a coupling agent and the lens is placed on the cornea. Then magnification should be increased & focus should be adjusted to see the angle in sufficient detail so that GSL can be performed. Once areas of PAS are identified, a plane spatula is inserted into the AC via either the main corneal incision or the side port incision, depending on where the PAS is located. Sometimes, it is necessary to create another one or two paracentesis opposite the area of PAS is access is difficult via the main and side port incisions.

The tip of the spatula should be positioned over the most periphery (i.e. most anterior) part of the area of PAS and with a gentle downward motion, the PAS is broken, revealing the TM. The goniolens is then rotated in order to visualize other quadrant and if areas of PAS are identified, they are broken by the spatula in a similar way. In this way, 360° of the angle is visualized and all areas of PAS are broken with the spatula.

Once this has been done, viscoelastics is removed from the AC in the usual manner and corneal wounds are hydrated to seal them. Finally intracameralCeftazidime& sub-conjunctival Dexamethason injections are given.

Post-operatively oral Acetazolamide twice daily for 3-5 days, oral Prednisolone for 7 days, topical steroid 2 hourly for 7 days, tapered over 6 weeks along with oral & topical antibiotic are given. The eye is often inflamed for a few days but after that both inflammation and IOP decreases so that after 1 week post-operatively the IOP is often at the desired level.[1]

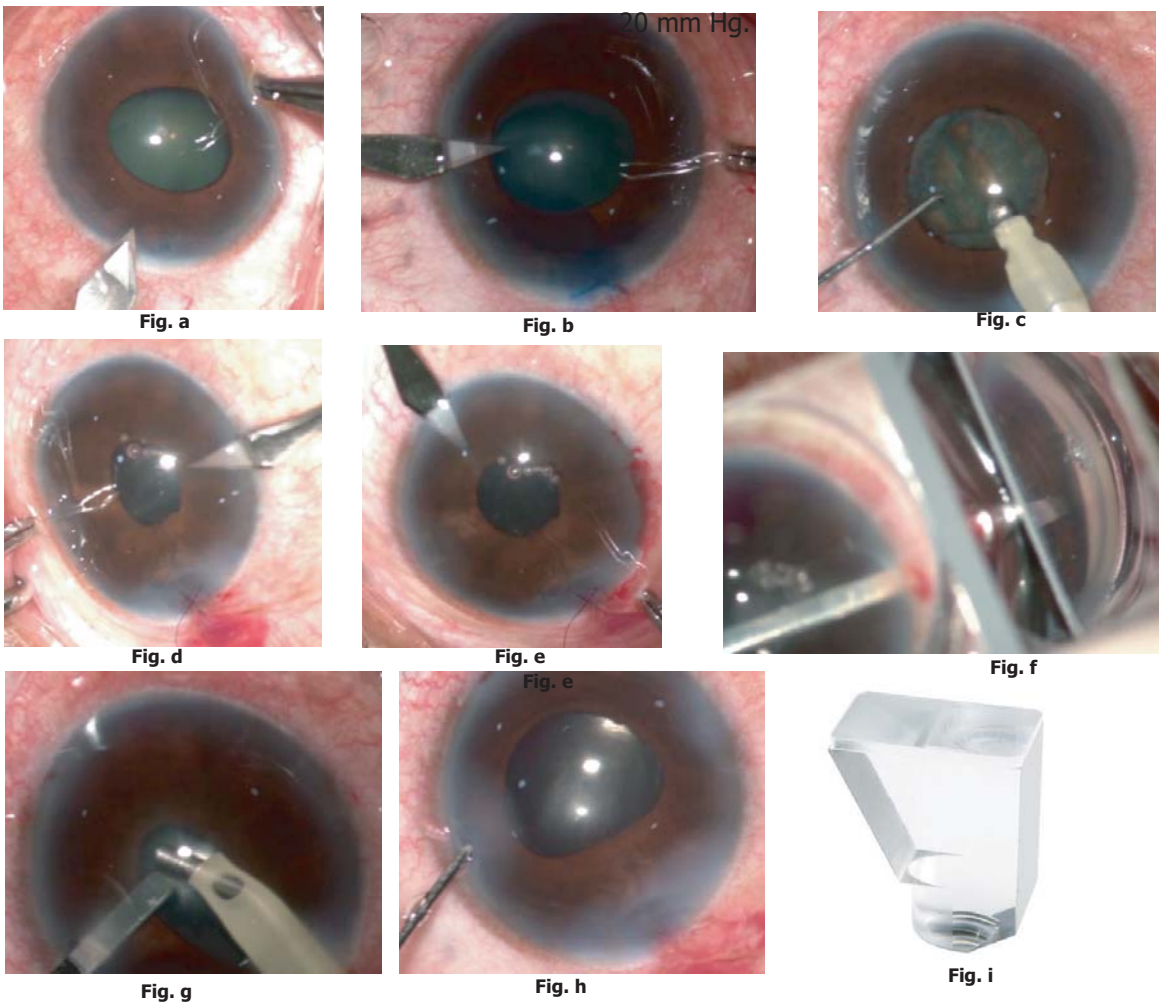

Figure: Steps of Phaco-goniosynechialysis. (a) main port [temporal] construction of phaco surgery, (b) side port [superior] construction of phaco surgery, (c) phacoemulsification of nucleas, (d) 3rd side port [inferior] after implantation of IOL and suturing of main port without washing of viscoelastic (e) 4thside port [nasal], (f) goniosynechialysis by plain spatula in all four quadrant from four different ports, (g) releasing of main port sutureand washing of viscoelastic substance, (h) stromal hydration of all ports, (i) Mori Upright Surgical Gonio Lens.

Results

Most clinical studies on GSL have been retrospective case series and/or less than 1 year follow up. Analyzing the results overall mean IOP reduction from these studies has been over

In Teekhasaenee and Ritch's papere (1999), they prospectively followed up 52 eyes of 48 patients for up to 6 years (mean follow-up 21 months). All eyes had an episode of acute angle closure with persistently raised IOP despite of a patent peripheral iridotomy and all had phaco-GSL within 6 months of the acute angle closure attack. Mean IOP decreased from 29.7 ±7.9 mm Hg to13.2 ±2.9 mm Hg ($p < 0.001$) and mean number of medications decreased from 2.4 to 0.1.The amount of PAS also significantly decreased from 309° to 56°. No patient experienced a decrease in visual acuity compared to preoperative levels.[3]

In a recent study, Campbell et al demonstrated that phaco-GSL lowered IOP effectively from 19.8 ±4.4 mm Hg to 14.4 ±2.1 mm Hg in 34

eyes with CACG. The 17 eyes with recent PAS showed a more significant IOP-lowering effect from 30.4 ±2 mm Hg to 12.1 ±2.3 mm Hg.5 Both the CACG and acute ACG groups demonstrated a similar decrease in postoperative glaucoma medications.[2]

In the most recent publication by Zhang et al., IOP was reduced from a median of 45 mm Hg to 15 mm Hg at last follow-up, in a series of 17 eyes – all of who had had acute angle closure attack.[9]

There has been only one study which compares phaco-GSL to arguably the gold standard, namely trabeculectomy. In this retrospective analysis, the authors compared 19 eyes that underwent phaco-GSL to 20 eyes that had trabeculectomy. All subjects had PACG. At a mean follow-up of 10 months they found that the phaco-GSL group had significantly increased success rate (IOP criterion) compared totrabeculectomy group. Although this study had small sample size with short follow-up, this result is significant. Because it suggests that phaco-GSL not only had the advantage of not creating a filtering bleb and avoids all the postoperative complications of trabeculectomy, but it also results in superior IOP lowering. This finding needs to be verified by other larger studies before more definite conclusions can be drawn, and comparison to phacotrabeculectomy is also needed.[10-11]

Complications and management

Intraoperatively, the main risk during GSL is hemorrhage. It seems that the longer the PAS have been present, the more likely to occur hemorrhage. In most instances, the bleeding will cease spontaneously, and this can be facilitated by increasing IOP with injecting more viscoelastic.

Another potential complication is iridodialysis or cycodialysis during GSL. It mainly occurs if the angle view is poor, pulling the iris rather than tapping or sweeping and mostly occurs by inexperienced surgeon. Careful handling can prevent this complication significantly.

Sometimes, iatrogenic trauma can occur to peripheral corneal endothelium which can be avoided by careful surgical technique by experienced surgeon.

Postoperatively, most patients will have significant AC inflammation in the first few postoperatively days. For this region, it is advisable to inject subconjunctival dexamethasone at the end of surgery and to prescribe topical steroid every 2 hourly for first 5-7 days. Similarly, IOP is often high in the first few days after surgery and postoperatively oral Acetazolamide is advisable for few days after surgery. Generally, about 5-7 days after surgery, the IOP will reduce and AC inflammation will settle down.

Conclusion

Phaco-GSL is a relatively old procedure which has enjoyed a recent resurgence of interest, probably due to increased recognition of the morbidity associated with PACG and the obvious advantages of "phaco-plus" procedures in contrast to conjunctival incising surgery such as trabeculectomy. Recent studies show that Phaco-GSL surgery is safe and produces marked IOP reduction.

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Pigment Dispersion Syndrome Masquerading as Acute Bilateral Anterior Uveitis- A Case Series

B K Sarker[1], Z Hassan[2], A R Khan[3], P Musa[4], R Sultana[5]

Abstract

Introduction : Clinical features of pigment dispersion syndrome (PDS) may be misdiagnosed with those of acute anterior uveitis. The purpose of this case series is to aid the ophthalmologist in the clinical differentiation between these two disorders.

Discussion : PDS may present with blurred vision, redness, ocular pain, and photophobia, all of which are also symptoms of acute anterior uveitis. These symptoms, plus the fact that pigment floating in the aqueous humor can be mistaken for inflammation, make diagnosis challenging. Moreover, the possible co-existence of true anterior uveitis and pigment dispersion makes the diagnosis and treatment more difficult. This study presents a series of 5 patients with pigment dispersion who were initially diagnosed as having acute anterior uveitis and treated with anti-inflammatory medication, including corticosteroids. The patients were referred for a second opinion due to poor or no response to therapy and were found to have pigment dispersion instead of uveitis.

Keywords : pigment dispersion syndrome, pigmentary glaucoma, uveitis, masquerading syndrome, acute anterior uveitis.

Introduction

Ocular pigment dispersion may be seen in the general population. During aging process a variable amount of uveal pigment is chronically released and dispersed into the anterior ocular segment[1]. As might be anticipated, pigment dispersion may also be found among individuals with various forms of glaucoma, although pigment in most of these patients is not believed to have a major role in the pathogenesis of the disease[1,2], However, there are several ocular conditions that are associated with an unusual, heavy dispersion of pigment that might significantly increase resistance to aqueous outflow with secondary elevation of the intraocular pressure and glaucomatous neuropathy. This condition is known as pigmentary glaucoma[2,3].

Pigment dispersion syndrome (PDS) is a distinct clinical entity characterized by an acute pigment scattering from the pigment epithelium of the iris and/or the ciliary body. Pigment dispersed in the anterior segment usually deposits on corneal endothelium, trabecular meshwork, iris, and lens.

Patients with PDS may present with a transient elevation of intraocular pressure[1,4,5]. Patients with an acute attack of pigment dispersion usually suffer from bilateral red eye, moderate to severe pain, photophobia, and blurred vision. These symptoms easily resemble those of an acute attack of anterior uveitis (AAU). Moreover, ciliary injection and pigmented particles floating in the anterior chamber may be confused with inflammation and so might easily lead to misdiagnose as AAU.

The purpose of this case series is to aid the general ophthalmologist in the clinical differentiation between a truly acute anterior uveitis episode and that of pigment dispersion syndrome, as well as to aid in the recognition of when these syndromes overlap.

Case Description

Case 1

A 32-year-old female was referred to our clinic with a 1-month history of persistent bilateral anterior uveitis. The patient was first seen by a

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general ophthalmologist for ocular pain, redness, and severe photophobia in the right eye; 10 days later the same symptoms appeared in the left eye. She was diagnosed as having AAU and was started on dexamethasone eye drop every hour and homatropine 2% eye drop every 6 h with no response to therapy. At that time, diagnostic investigation was negative. She reported that she was told that cells did not decrease in spite of topical corticosteroid therapy. When first seen in our clinic, she continued with the same, significant symptoms. On examination, her visual acuity was 6/9 OD and 6/12 OS.

Intraocular pressures were 25 mmHg OD and 31 mmHg OS. Slit-lamp examination disclosed a Krukenberg spindle (Figure 1), scant pigment deposition over the anterior lens capsule, and 1+ pigment floating in the anterior chamber OU. Irides were slightly concave with moderate peripheral stromal atrophy and irregular pupillary contour no transillumination defects were seen OU.Gonioscopy, while on cyclopegics, revealed an open angle with heavy pigment deposition on the trabecular meshwork, especially on the inferior quadrants. Dilated fundus exam was normal OU.

Based on ocular findings, pigment dispersion syndrome with secondary ocular hypertension was diagnosed. She was asked to stop topical steroids and cycloplegics; she was also started on timolol maleate 0.5% twice a day OU, brinzolamide three times a day OU, and acetazolamide 250 mg twice a day. One week later, most symptoms had disappeared. Trace pigment floating in the anterior chamber OU was noted as well as a decrease in intraocular pressures to 15 mmHg OD and 17 mmHg OS. The patient continued on ocular hypotensive medications for 2 months before stopping them. At her last visit, 12 months after initial presentation, no pigment dispersion was noted, and intraocular pressure had remained 15 mmHg OU without medication.

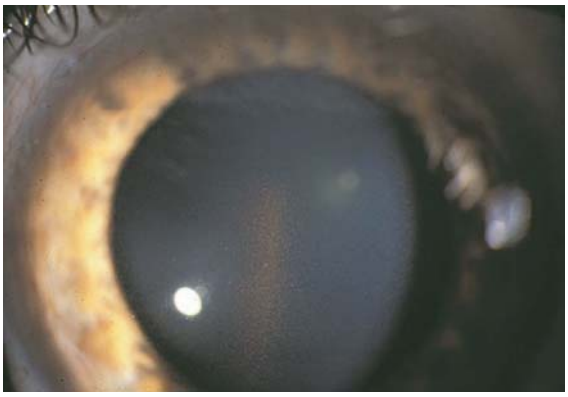

Figure 1: Showing diffuse pigmentation on corneal endothelium with Krukenberg spindle

Case 2

A 33 year-old female came to our clinic with a 2-week history of bilateral blurred vision, extreme photophobia, and redness of abrupt onset. The patient was previously diagnosed as having bilateral acute anterior uveitis (AAU). All laboratory workup performed at that time was unrevealing, and the patient was treated with prednisolone acetate 1% every 3 hour, and tropicamide 1% four times a day with no response or improvement in symptoms.

She was referred to our hospital for diagnosis and treatment of her AAU. On examination, visual acuity was 6/6 in both eyes (OU), and intraocular pressures were 15 mmHg OD, and 11 mmHg OS. The patient had 1+ ciliary injection OU. Slit-lamp examination disclosed 1+ pigment granules in the anterior chamber, papillary contour irregularity, and mid peripheral iris transillumination defects OU.

Gonioscopic exam showed an open angle with moderate pigment accumulation on the trabecular meshwork (figure 2), in the inferior quadrant OU.

The patient was diagnosed as having pigment dispersion syndrome, and no additional diagnostic workup was performed. She was asked to stop all medications and advised to continue steroid for 2 weeks 2 times daily. After a 5-month follow-up period, the patient was asymptomatic, intraocular pressures remained normal, and no further pigment dispersion was detected.

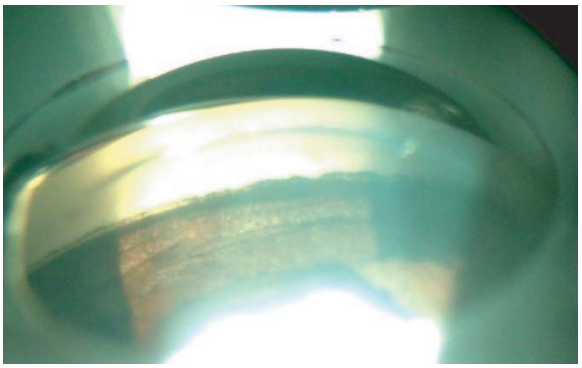

Figure 2 : Showing an open angle with pigment accumulation on the trabecular meshwork

Case 3

A 36-year-old male was seen for a 15 day history of redness, ocular pain, photophobia, blurry vision, and tearing, starting in the left eye and followed almost immediately by the right eye. The patient was diagnosed with bilateral AAU. He was started on prednisolone acetate 1% every hour, his symptoms worsened over time, with development of severe photophobia, intense ocular pain, and decreased vision.

The patient was told that he had persistent inflammation with no improvement to therapy. The patient decided to look for a second opinion. We found visual acuity of 6/9 OD and 6/24 OS. Intraocular pressures were 25 mmHg OD and 31 mmHg OS. At slit-lamp examination, there was 1+ ciliary injection, 2+ Krukenberg spindle, scant pigment deposition over the anterior lens capsule, and 2+ pigment floating in the anterior chamber OU; a slight concavity of the irides was also noted. Gonioscopy showed heavy pigment deposition on the trabecular meshwork in all quadrants OS > OD (Figure 3).

The patient was diagnosed as pigment dispersion syndrome with secondary ocular hypertension OU. All previous medications were stopped and the patient was started on timolol maleate 0.5% twice a day and brinzolamide three times OU, and acetazolamide 250 mg three times a day. Five days after this therapy was commenced the intraocular pressures were 12 mmHg OU. Three weeks after initial visit no pigment dispersion was seen in either eye, visual acuity was 6/9 OU, and IOP was 12 mmHg OU. Irregular pupils and extensive midperipheral transillumination defects OU were noted .After several months the patient achieved a best-corrected visual acuity of 6/6 OU and IOP remained normal; however, the patient still complained of moderate photophobia, especially while exercising.

Figure 3: Gonioscopy showed heavy pigment deposition on the trabecular meshwork in all quadrants

Case 4

A 35-year-old female was referred to our clinic with a 1-month history of ocular pain, photophobia, and redness OU. The patient recalled that symptoms began OS, followed by OD weeks after. She stated that headache and ocular pain were variably present. No other signs or symptoms were positive in the complete clinical history and physical exam. She was previously treated with, brinzolamide hydrochloride 2% twice a day, and dexamethasone 0.1% every 2 h alternating with loteprednol etabonate 0.5% every 2 h.

Best corrected visual acuities were 6/9 OD and 6/6 OS. Intraocular pressure was 14 mmHg OU. Remarkable findings OD were 1+ ciliary injection 1+ cells and 1+ pigment in the anterior chamber, and 2+ flare (figure 4). The left eye showed 1+ ciliary injection, trace cells and pigment in anterior chamber, and 1+ flare. No posterior or peripheral anterior synechiae were found in either eye. Lens and anterior vitreous were clear without relevant findings. Dilated fundus exam in both eyes revealed no abnormalities.

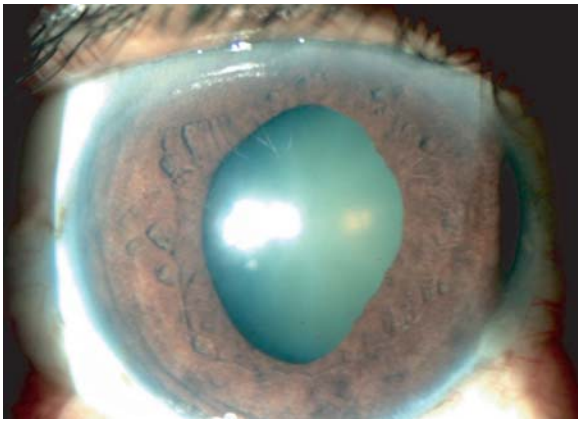

Figure 4: Presenting as uveitis with dilated pupil due to use of cycloplegic

The patient was diagnosed with diffuse anterior scleritis OD and bilateral anterior uveitis. During the following days, cycloplegics were stopped. Two days later, she presented to our clinic with visual acuities of 6/9 OU. IOP was 14 mmHg OD and 13 mmHg OS. Anterior segment examination was remarkable for Krukenberg spindle OU, 1+ pigment clumps in the anterior chamber, trace cells, and no flare in both eyes. Gonioscopy revealed slightly pigmented trabecular meshwork. A diagnosis of overlapping pigment dispersion syndrome and anterior uveitis was made. A very slow tapering of steroids and oral NSAIDs was performed, achieving no inflammatory activity within 3 months. At her last visit, her best-corrected visual acuity was 6/6 in both eyes. At slit-lamp examination no anterior inflammatory activity, no pigment floating, no iris transillumination defects, and no pupillary irregularities were found.

Case 5

A 32-year-old male was presented severe ocular pain, photophobia, redness, and blurred vision, starting in the left eye followed by the right eye 3 days later. The patient was initially diagnosed as having acute bilateral anterior uveitis. He was given atropine 1% once a day OU, and dexamethasone phosphate 0.1% every 3 h. Laboratory workup for uveitis was unrevealing.

During the acute phase, intraocular pressures rose to 32 mmHg OD and 31 mmHg OS.

Because no response to treatment was observed during a period of 3 weeks and anterior uveitis was noted, the patient was referred to our clinic for a second opinion and treatment. On first visit, the patient had a 2+ ciliary injection, 3+ pigment floating in the anterior chambers, and 4+ prominent Krukenberg spindle OU . Gonioscopy showed 360° heavy pigment deposition on the trabecular meshwork of both eyes, especially in the inferior quadrants. Postdilation gonioscopy was not documented. Intraocular pressures were 32mmHg OD and 33 mmHg OS. No optic nerve cupping was observed during dilated fundus examination.

The patient was diagnosed as having pigment dispersion syndrome with secondary ocular hypertension OU. All medicationss were stopped, and the patient was started on pilocarpine 2% three times a day OU, timolol maleate 0.5% twice a day OU, and acethazolamide 250 mg three times a day orally. He was also asked to reduce physical activity to a minimum. After a period of 3 weeks pigment dispersion subsided, IOP dropped to 13 mmHg OD and 14 mmHg OS, and all symptoms disappeared.

Discussion

The PDS is an autosomal dominant disorder with phenotypic onset beginning in most persons in mid-20s[7]. The disorder is characterized by disruption of iris pigment epithelium and deposition of pigment granules on the anterior segment structures. The incidence of PDS is 4-8 per 100,000 population. This condition is more commonly seen in Caucasians and is considered to be rare in Indians [8] .PDS is typically a bilateral disease, although asymmetry may occur. Men and women are equally affected by PDS. Men are significantly younger than women at the time of diagnosis of the disease.

About 60% - 80% of patients are myopes and 20% are emmetropes[5] . The syndrome is characterized by the triad of deposition of pigment on the posterior corneal surface in a vertical line (Krukenberg's spindle), wide open angles on gonioscopy with uniform and heavy pigmentation of trabecular meshwork, slit like

radial transillumination defects in the iris. Other ocular findings commonly observed are: relatively flat cornea, deep anterior chamber,and wide open angles.2 The iris is inserted posteriorly and shows a concave configuration in the mid periphery. This feature is best demonstrated by ultrasound biomicroscopy (UBM) or anterior segment OCT[8,9] .

Pigment accumulation on the anterior surface of the iris often appears as concentric rings within the iris furrows. Pigment is also deposited on the posterior surface of the lens in the region of contact between the anterior hyaloids face and posterior lens capsule. Visualization of this circular ring or arc of pigmentation requires pupillary dilatation and is considered pathognomonic of PDS[8] . Patients with PDS and pigmentary glaucoma are at increased risk for retinal detachment and this may occur in 6%-7% of individuals. Retinal breaks and retinal detachment may occur twice frequently in these eyes·[9].

One of the main reasons that PDS masquerades as acute anterior uveitis is that pigment granules mimic inflammatory cells floating in the anterior chamber[8]. The wide spectrum of clinical presentation of patients with PDS varies from silent dispersion and ocular hypertension to symptoms resembling those of an acute anterior uveitis attack such as blurred vision, redness, ocular pain, and extreme photophobia[9,10] . In this report of 3 cases of bilateral acute depigmentation of the iris, the main symptoms were a sudden onset of ocular discomfort and red eye, with or without ciliary injection and Krukenberg spindle[11] .

These overlapping symptoms make misdiagnosis even more probable. Persistent anterior inflammatory cells in spite of steroid therapy should prompt one to consider pigment dispersion as a possible diagnosis. The presence of a Krukenberg spindle, pigmentation of the trabecular meshwork, or backward bowing of the iris is highly suggestive of this pathology.

The diagnosis of PDS is even more challenging when the patient presents on topical steroids, rising the questionable presence of previous inflammation or an overlapping syndrome. However, little or no response to topical, regional, or systemic steroids, associated with no improvement or worsening of ocular symptoms and signs, should raise a suspicion of a masquerade syndrome. Moreover, the rapid response and significant improvement in inflammation after discontinuing anti-inflammatory therapy and initiating PDS therapy is confirmatory of the diagnosis.

On the other hand, it is remarkable that two clinical entities might be present in the same patient. In the case of patient 5, all clinical features of pigment dispersion syndrome overlapped with a typical presentation of recurrent, alternating iridocyclitis. In this particular case, it is interesting that in some visits the patient presented only with pigment dispersion without ocular hypertension compared to other visits when mild, obvious anterior uveitis with no pigment dispersion was present. Thus, we believe these entities in fact can overlap, making a definite diagnosis even more difficult.

The management of PDS depends on the severity of the pigment dispersion and the association with secondary ocular hypertension or glaucoma. Miotics like pilocarpine have been recommended for limiting pigment dispersion as they are thought to reduce iridolenticular contact and friction by changing iris configuration[12] ·

In summary, therapy in patients with pigmentary ocular hypertension or glaucoma must be individualized, considering that aggressive therapy might be necessary. If target ocular pressures are not achieved with medical therapy, laser trabeculoplasty and filtering surgery might be of help.

Conclusion

Pigment dispersion syndrome can lead to severe visual field loss and poor quality of life. Therefore, it is very important to consider the diagnosis of PDS in any young patient presenting with symptoms suggestive of acute anterior uveitis. A detailed ophthalmological

examination, including gonioscopy, is mandatory to make the correct diagnosis and to avoid the need for extensive laboratory workup that eventually proves to be useless.

It also will eliminate inappropriate use of corticosteroids that, apart from not resolving the condition, further complicate the clinical scenario, either by causing secondary ocular hypertension and glaucoma or by inducing secondary cataract formation. The case series also highlights the importance of looking for subtle signs like phacodonesis and iridodonesis which may be due to increased axial length of the eyeball or may be an associated feature of PDS.

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Medulloepithelioma...with Secondary Glaucoma : A Case Report

S M Noman[1]

Background : To report a rare case of Medulloepithelioma that presented with secondary glaucoma.

Method : After taking history from the patient, ocular and systemic examinations were done. A boy of 8 years came with the complaints of gradual dimness of vision right eye for 2 years. He also complaints of sectoral redness and occasional pain in the right eye for last 1 year. He developed sudden decrease of vision as well as painful right eye for the last 10 days. Occular examinations revealed visual acuity in the right eye was 6/60, sentinel vessels over one sector, mass in front and behind iris that touched the lens, ectropion uvae in the same that sector. IOP was raised in that eye. Mass was seen in the indirect ophthalmoscopic examination. No abnormalities found in the systemic examination. Histopathology done after enucleation.

Result : B scan showed a echogenic shadow looked like a mass. Ribbons and cords of tumous cells of ciliary body origin, lack of differentiation, multi layered nucleus, round/oval cells were ascertained on histopathology suggestive of mudulloepithelioma.

Conclusion : Ciliary body tumour in a young age group is very rare but present with some typical features. Detailed ocular examinations and investigations are mandatory to diagnose such case. Histopathology confirms the diagnosis.

Introduction

Intraocular medulloepithelioma is a rare primary intraocular neoplasm derived from neuroectoderm. This type of tumor characteristically arises from the nonpigmented epithelium of the ciliary body. On rare occasions, medulloepithelioma also arises from the iris, retina, or optic disc. The tumors range from benign proliferations to malignant neoplasms with unequivocal invasive capacity but limited metastatic potential. This would correspond to a cumulative lifetime incidence of approximately 1 case per 450,000–1,000,000 persons. Intraocular medulloepithelioma is usually a congenital or infantile tumor,[1] but juvenile- and even adult-onset cases have been reported. The average age of the affected individual at diagnosis is about 5 years in most series.

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Case History :

Baby Masud of 8 years came to CEITC with the complaints of gradual dimness of vision & occasional pain in his right eye for the last 1 year. According to his parent's statement, they have been observing a white mass in the right side of the right eye for the last 2 month which is gradually increasing in size. They have also observed continuous redness of the right side of the right eye for the last 1 year. Suddenly he developed severe painful right eye with sudden decrease of vision for the last 10 days. On examination his visual acuity of right eye was 6/60 pinhole no improvement & of left eye was 6/6. His intraocular pressure was 30 Hgmmhg in right eye & 12mmHg in the left eye. Slit lamp examination revealed sectoral vascular engorgement in the temporal side of the right eye (sentinel vessels) & a redishwhite mass in front and behind the iris that touched the mild cataractous lens from the back, ectropion uvae in the same that sector. There was moderate chronic inflammation in the anterior & posterior chamber. Indirect ophthalmoscopic examination revealed a cystic mass behind the right side of the lens (Ciliary body mass). B scan showed a echogenic shadow looked like a mass. No other abnormality is found in the retina of the right eye. On examination of the left eye revealed no abnormality. No hepatic or lymphatic enlargement was found on systemic examination. The patient was diagnosed as Ciliary body mass (medulloepithelioma or dictyoma). It had a malignant potentiality, enucleation of the right eye with cutting of

10mm of optic nerve was done. Tissue sent for histopathology. Histopathology revealed Ribbons and cords of tumour cells of ciliary body origin, lack of differentiation, multi layered nucleaus, round/oval cells were ascertained on histopathology suggestive of medulloepithelioma.

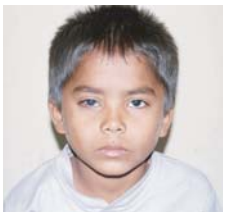

Fig-1 : Redness of right eye

Fig-2: Sentinel vessels

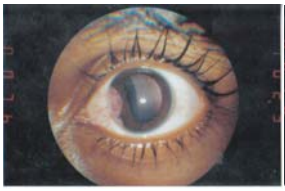

Fig-3 : Mass in front of the iris

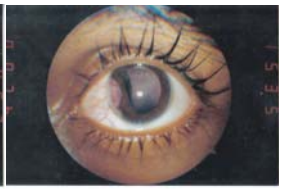

Fig-4 : Mass in front & back of the iris

Discussion

Ciliary body medulloepithelioma is usually an amelanotic, fleshy mass with an intralesional cystic component, it can occur as a pigmented solid tumor that may resemble a melanoma or a neoplasm of the pigment epithelium[2]. It is the commonest ciliary body neoplasm in childhood. Grinker coined this term "medulloepithelioma" in 1931 as it best describes the cellular derivation of this neoplasm from the undifferentiated medullary epithelium of the embryonic retinal epithelium destined to form the nonpigmented ciliary body epithelium during the later years of life.

This case highlights the extent at which a diagnosis was delayed, when a ciliary body medulloepithelioma in a eight-year-old child masquerades as a chronic uveitis, cataract, and secondary glaucoma.

The usual presenting symptoms of medulloepithelioma are a red eye, change in color of the iris, visible mass in the iris, and (in adults and some older children) visual impairment.[1] Medulloepithelioma of the ciliary body typically appears as a tan to white lesion of the extreme peripheral fundus. Because of its peripheral location, the tumor may be detectable only by binocular indirect ophthalmoscopy with scleral depression during ophthalmic examination under anesthesia. A tumor of this type frequently appears intrinsically cystic or has prominent neuroepithelial cysts on its surface.[1,3]

In this case patient presented with localized red eye due to engorged sectoral blood vessels, ectropion uvae that changed the colour of the iris, redish white fleshy mass in front & behind the iris. The mass was causing cronic uveitis followed by secondary glaucoma & touching the lens that caused cataract. Indirect ophthalmoscopy revealed that cystic mass with scleral depression. An intraocular medulloepithelioma that involves the iris usually appears as a tan to pink mass that replaces the peripheral iris and fills the angle. In our case that pink mass encroached over the iris & close the angle almost 120^0 that was revealed on gonioscopic examination. A common complication of medulloepitheliomas of the ciliary body is development of neovascular glaucoma.[4] In some cases, glaucoma of this type develops even when the tumor is limited in extent. In other cases, non-neovascular angle closure develops in response to the ciliary body tumor.[5] In our case raised intraocular pressure was due to combined effects of both gradual angle closure & chronic uveitis. No neovascularization was observed over the iris or in the angle. In exceptional cases, intraocular medulloepithelioma arises from the retina or optic disc. All reported cases of this type have occurred in children younger than 7 years. Because of this, such tumors have uniformly been misdiagnosed clinically as retinoblastoma. But in our case no abnormality was seen in the retina & optic nerve that excluded the possibility of retinoblastoma. But as the tumor was in the extreme periphery that touched the lens & encroached over & behind the iris that indicated the tumor as ciliary body origin.

Although iridocyclectomy and episcleral plaque

radiotherapy have both been employed in some cases of iridociliary or ciliary body medulloepithelioma, such treatments have frequently failed to eradicate the tumor. In fact, local failure of such treatments appears to be almost the rule in medulloepitheliomas judged to be malignant by histopathological criteria. Eyes that have an extremely large intraocular tumor at presentation, those that are blind and painful as a result of tumor-related complications, and most eyes that develop local recurrence of medulloepithelioma after primary attempted resection or plaque radiotherapy eventually require enucleation[6]. In our case patient developed intractable uveitis with refractory glaucuma followed by severe painful eye. Before tumor invasion in to the sclera & to prevent periocular tumor invasion, enucleation of the right eye was done & specimen was sent for histopathology.

The characteristic histopathological feature of intraocular medulloepithelioma is a structural arrangement of cells that closely resembles that of neural medullary epithelium. The degree of cellular differentiation differs widely from case to case. Many well-differentiated medullo epitheliomas contain prominent rosettes and cystic spaces filled with hyaluronic acid. Medulloepitheliomas that contain heterotopic elements such as hyaline cartilage, striated muscle, or brain are referred to as teratoid medulloepitheliomas. Those that do not contain such elements are termed nonteratoid medulloepitheliomas. About two thirds of intraocular medulloepitheliomas are categorized as malignant pathologically,[1, 2] largely on the basis of invasiveness and extraocular extension of the tumor, especially if associated with numerous mitotic figures and undifferentiated cells.

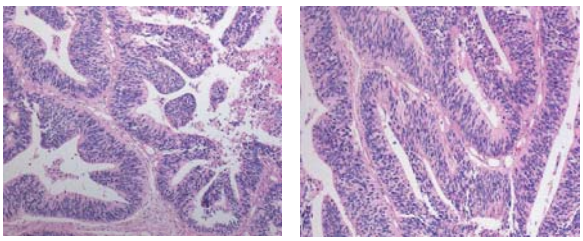

Fig-5 : Histopathological slide of Ciliary body tumor

In our case histopathology of the mass from the enucleated eye showed ribbons and cords of tumous cells of ciliary body origin. There was lack of differentiation but no mitototic figure. Central cystic space field by fluid (hyaluronic acid). Multi layered nucleus, round/oval cells without heterotopic elements suggestive of non teratogenic mudulloepithelioma.

Surgical removal of the tumor provides a definitive histological diagnosis[7]. The decision for enucleation is usually based on a large tumor size, a painful eye with poor visual potential, and a strong clinical suspicion of malignancy for instance rapid tumor growth as in our case. As ciliary body medulloepitheliomas are generally locally invasive, complete excision is curative and associated with a good survival prognosis. In our case, the delayed diagnosis might have contributed to the local scleral invasion of the tumor, thereby necessitating adjuvant chemotherapy after enucleation.

Conclusion

Medulloepithelioma, even in its benign varieties, tends to be a relentlessly progressive tumor. This can lead ultimately to destruction of the eye, profound visual loss, and even trans-scleral tumor extension. As long as the tumor is still contained within the eye at the time of enucleation, survival generally is assured. Detailed ocular examinations and investigations are mandatory to diagnose such case.

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Review Article

A case series of short term surgical outcome of combined surgery for secondary angle-closure glaucoma and cataract as a complication of Vogt-Koyanagi-Harada (VKH) disease

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Abstract

Purpose : To evaluate short-term surgical outcome of VKH secondarily complicated with cataract and angle-closure glaucoma.

Methods : All case files of patients who presented with bilateral VKH complicated with cataract and secondary angle closure glaucoma were reviewed. After confirmation of diagnosis of VKH by the vitreo-retina department, medical treatment was initiated with intravenous Methylprednisolone and subsequently with high dose oral corticosteroids, Azathioprine, Antiglaucoma medications, cycloplegics, and/or topical steroids, as necessary. The patients developed cataract and secondary angle closure glaucoma with very high intraocular pressures (IOP) and were consequently referred to the glaucoma department. All cases had trabeculectomy with intraocular lens (IOL) implantation for immediate IOP control. The outcome measures were visual acuity(VA) and IOP. Postoperatively, the follow-ups after 1day, 7days and at 1month were recorded.

Result : All patients had immediate IOP control with an excellent improvement in VA following surgery. During follow up IOP was well controlled and a notable improvement in vision was maintained over the follow-up period. However, the trabeculectomy failed in one case, with subsequent implantation of an Ahmed glaucoma valve (AGV), which restored IOP parity.

Conclusion : Bilateral VKH complicated with cataract and secondary glaucoma can be managed immediately by combined surgery (Trabeculectomy+IOL) and if trabeculectomy fails AGV can be implanted to control the IOP trabeculectomy and cataract surgery at the same time will improve vision dramatically. However, long term follow up is needed for future comparison.

Keywords : VKH, Secondary glaucoma, complicated cataract, Trabeculectomy, Ahmed glaucoma valve.

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Introduction

Vogt-Koyanagi-Harada (VKH) disease is a severe bilateral granulomatous posterior or panuveitis associated with serous retinal detachments, discoedema, and vitritis, with eventual development of a sunset glow fundus.[1] VKH disease is a globally distributed condition that has a predilection for dark-skinned individuals such as Asians, Native Americans and Hispanics.[1] The incidence of VKH in Bangladesh has been reported to be 8.4% of all uveitis, based on presentation at a tertiary center.[2] It usually occurs in patients between the ages of 20 and 50 years, but children may also be affected, with females twice as affected as males.[3] Diagnosis is mainly clinical and aided by Bscan ultrasonography, colour fundus photograph and optical coherence tomography and fundus fluorescein angiography in selected cases. Treatment modalities mainly include systemic corticosteroids and more recently steroid-sparing immunomodulating agents.[3]

In this case series, we present six eyes of four patients who underwent combined trabeculectomy and cataract surgery between March 2018 and May 2019 for VKH with secondary angle-closure glaucoma and cataract. Their initial presentation and subsequent management will be discussed.

Case One

A 19-year-old female presented with complaints of diminished vision, redness and pain in both

eyes of 4months duration, and diagnosed as a case of with bilateral VKH with complicated cataract and secondary angle closure glaucoma. It had unfortunately been managed as a case of anterior uveitis with only topical corticosteroids, and other past medical history, review of systems and ocular history were unremarkable. Initial examination revealed vision of counting fingers at 0.5m in both eyes. Other remarkable findings were bilateral mild conjunctival congestion, keratic precipitates, shallow anterior chambers with 360 synechial angle closure on gonioscopy. There was also Iris neovascularization, complicated cataract, significant vitritis and sunset glow fundus bilaterally. Intraocular pressures were 29 and 30mmHg in the right and left eye respectively.

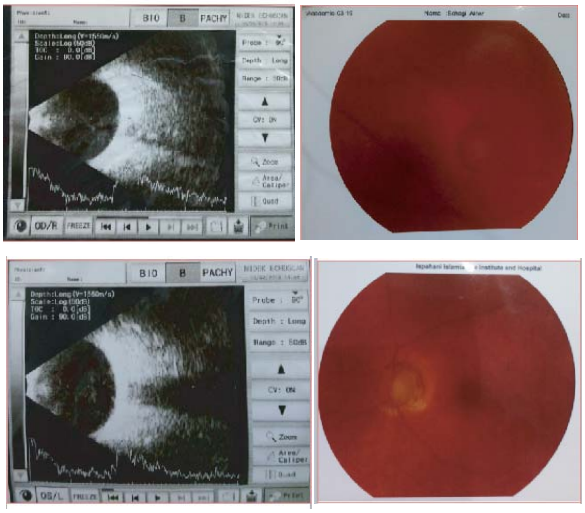


Fig: B Scan and Colour fundus photograph (CFP) demonstrating vitritis and choroidal thickening (B Scan) and sunset glow (CFP)

Medical treatment was immediately initiated with intravenous methylprednisolone (one gram) for 3days, and subsequent oral corticosteroids (1mg/kg/day with a slow taper), oral Azathioprine, topical steroid and antiglaucoma medication. There was an initial improvement with vision improving by 3 lines of Snellens chart in both eyes, with mild reduction of intraocular pressures. However, after 3 months there was deterioration of visionwith sustained raised IOP was 48mmHg(RE) and 46mmHg(LE). Inflammation was relatively well controlled, and she had bilateral Trabeculectomy+ IOLwithin2 weeks, with IOP at the 1st week and 1st month 9mmHg and 20mmHg respectively in the right eye, and 14mmHg and 12mmHg in the left eye. Her VA was 6/60 and 6/18 which improved to 6/36 and 6/12 with refraction in the right and left eyes respectively as of last follow up, with normal IOP.

Case Two

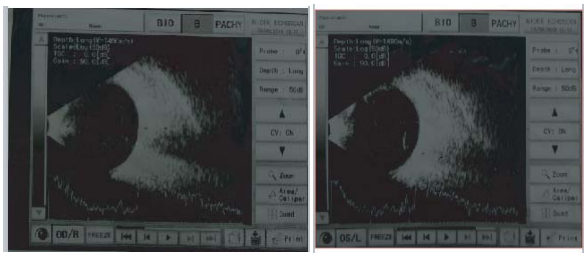
A 25years female presented with complaints of diminished vision and pain in both eyes of 6months duration. She was diagnosed with bilateral VKH with right pthisis bulbi (presumably from chronic inflammation with ciliary shutdown) and a left complicated cataract with secondary angle closure glaucoma. Initial examination revealed unaided visual acuity of NPL in the right eye and 6/60 in the left eye. The conjunctiva,cornea and anterior chamber were normalin the left eye.The pupil was irregularwith 360 degrees posterior synechiae with segmental synechial closure on gonioscopy. There was also complicated cataract andevidence of posterior inflammation on B-Scan USG. The IOP in the right eye could not be measured but was 37mmHg in the left eye.

Medical treatment was immediately initiated with Intravenous methylprednisolone for 3days, oral corticosteroids, oral Azathioprine, and antiglaucoma medication. She had left Trab + IOL performed. There was an improvement with vision improving by 4 lines of Snellens chart and the IOP on the 1st week and at 1month was 17 and 12mmHg. Her vision was 6/9 which improved to 6/6 as of the last follow up.

Case Three

A 35-year-oldmale presented with complaints of poor vision in both eyes of 1year, with a sudden deterioration vision in both eyes of 2weeksduration.The past medical history, review of systems were unremarkable. Hehad cataract surgery done in the right eye 2years back. Intraocular pressures at presentation were 11mmHg and 10mmHg in the right and left eye respectively. Initial examination revealed vision of PL in both eyes, with bilateral mild

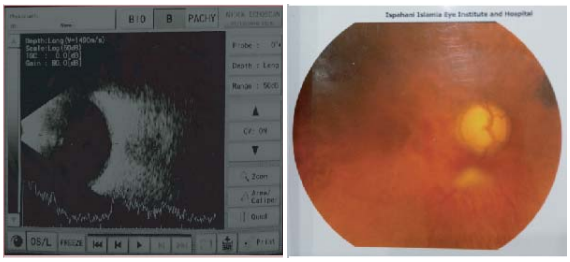
conjunctival congestion, keratic precipitates , cells (++) and flare (++), and open angles with hyperpigmentation of the trabecular meshwork. Other findings were posterior synechiae, early cataract, and severe vitritis. The optic nerve head had a cup to disc ratio of 0.6:1 in both eyes. Bscan ultrasonoraphy revealed severe bilateral vitritis with choroidal thickening.



Medical treatment was immediately initiated with Intravenous methylprednisolone (1gm) for 3days, with switch to oral corticosteroids as before, oral azathioprine and topical steroids. There was an initial improvement with VA of 3/60 (RE) and 6/60 (LE) After 3months,IOP was markedly elevated to 50mmHg and 48mmHg in the right and left eyes respectively and was commenced on medical antiglaucoma regimen. He subsequently developed shallow anterior chambers with 360 degree synechial angle closure on gonioscopy bilaterally. He hadRt Trabeculectomy done, and vision at 1week postoperative visit was 6/18 with IOP of 15mmHg and at 1month, the vision was 6/24 with IOP of 12mmHg. He also underwent Lt Trab + IOL and vision at 1week postoperative visit vision was HM with IOP of 17mmHg. At 1month VA in LE was 6/60 and IOP 18mmHg. However, at 6months (although not within the reported period) there was worsening clinical signs evidenced by VA of 6/60 and HM, IOP of 34 and 32 in right and left eye respectively. Ahmed glaucoma valve implantation was done in RE and at 1week, VA was 6/36 with IOP of 10mmHg and at 1month, VA and IOP remained unchanged.

Case Four

A 50 year old male who presented with complaints of diminished vision in both eyes of 3years duration was diagnosed as a case of with bilateral old VKH with left complicated cataract and secondary angle closure glaucoma. The past medical history and review of systemswere unremarkable. He had right cataract surgery with IOL implantation done previously elsewhere. Intraocular pressures were 10mmHg in both eyes. Initial examination revealed VA of HM and 3/60 in right and left eye respectively. Other remarkable findings were keratic precipitates, shallow anterior chamber with 360 synechial angle closure on gonioscopy in LE. The RE was pseudophakic while the LE had complicated cataract. Funduscopy revealed a sunset glow fundus in RE, but no fundal view in the LE. Colour Fundus photograph revealed the grossly tessellated fundus, and Bscan ultrasonography revealed severe bilateral vitritis.



Medical treatment was immediately initiated as before with Intravenous methylprednisolone for 3days followed by tapering oral corticosteroids, oral Azathioprine & antiglaucoma medication. He had Lt. Trab+ IOL performed, with an immediate improvementevidenced by VA of 6/36 and IOP of 15mmHg at 1week postoperative visit . At 1 month postoperative visit the VA was 6/24, with an IOP of 06mmHg.

Discussion

Glaucoma is one of the most common complications associated with uveitis due to VKHthat usually necessitates therapeutic intervention.[4] The incidence of glaucoma in VKH varies from 6% to 45% in literature.[4] Risk factors for the development of glaucoma are uveal effusion and increased number of recurrences.[4] Treatment is mainly surgical, though initial medical treatment is recommended to reduce the intraocular pressure preoperatively. No matter the clinical stage, it is

a progressive disease and when complicated with glaucoma,controlling the IOP medically is usually a challenge and surgical management is usually indicated. However, there are few reports on immediate short-term postoperative outcome of combined surgery in VKH. Trabeculectomy has been tried in glaucoma due to VKH and Faisal A Almobarak et al reported that the cumulative probability of success was 77.8% at 12 months postoperatively.[5] In the intermediate term, the IOP was reduced from 40.15±7.0 to 13.12±6.9mmHg as of the last follow up as well as the whole follow up period.[5]In a study by Felix Gil Carrasco et al, success was achieved in 8 out of 14 eyes(57.14%) with AGVs.[6] Another study by Pandey Amit et al, surgical success was achieved in 50% at 12 months follow up period.[4] In our small series, four (approximately 66%) out of the six(6) Trab+ IOL surgeries performed had good VA and immediate IOP control following surgery while one required AGV implantation to achieve control (with the other eye awaiting valve implantation).

Conclusion

Secondary glaucoma is, unfortunately, quite common in VKH.As the IOP rise is chronic in nature and usually angle-closure, early surgical intervention has been shown to have a good outcome.[6] Trabeculotomy remains a good option for initial management, with Ahmed Glaucoma Valve implantation a safe alternative in high risk patients with uncontrolled uveitic glaucoma who have had multiple previous ocular surgeries.[6]We therefore recommend early glaucoma surgical intervention and where necessary, removal of the cataract to aid IOP control and optimize visual outcome. Our case series suggests that the immediate surgical outcome is excellent. However, a long term follow up is needed for the long term maintenance of intraocular pressures and vision, and continuous monitoring of IOP with long-term follow-up is essential.

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