Journal Bangladesh Glaucoma Society

July 2018 Volume 06 Number 02

JBGS

Contents

Journal
Bangladesh Glaucoma Society

Volume - 06 Number - 02 July 2018

Journal
Bangladesh Glaucoma Society (JBGS)

Volume - 06, Number-02, July 2018

Published by :
Dr. Md. Safiul Islam Prodhan
Publication & Publicity Secretary
On behalf of the
Bangladesh Glaucoma Society
House # 12A, Road # 05, Dhanmondi, Dhaka
email : bangladeshglaucomasociety@gmail.com
website : www.bgsbd.net

Printed at :
New Ekata Computer & Printers
435/A-2, Baro Moghbazar, Dhaka-1217
Tel : 01715444644

Courtesy : ARISTOVISION

Bangladesh Glaucoma Society

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Angle closure glaucoma – a public health problem

M N Islam[1]

In this issue of the JBGS, 2 articles as case presentation has been published on Bilateral Angle-Closure Glaucoma. Both the article cases are not common but significantly medical important. If timely managed by medical or surgical treatment angle closure can be treated and vision can be saved. The 1st article Bilateral Acute Angle Closure from Topiramate toxicity – 3 case reports by M. Nazrul Islam is of public health importance asTopiramate is prescribed by medicine specialists, Neurologists, Otolaryngologist and also by Ophthalmologist.

2nd Case Presentation: Bilateral Angle-Closure Glaucoma by Dr. Shams Mohammad Noman and Dr. Nahid Sultana is more of ophthalmologist importance. We need to know the pathophysiology of angle closure diseases and how to manage this public health problem

Acute angle closure glaucoma (AACG) is regarded as an ocular emergency around the world. The most accepted terminology now is acute primary angle closure (APAC). AACG remains – in general– a useful trigger term to exhume a pattern recognition for the non specialist.

Primary angle closure spectrum (PACsp) eyes can be classified as primary angle closure suspects (PACS), primary angle closure (PAC) or primary angle closure glaucoma (PACG). The common denominator is an overcrowding of the anterior segment, with appositional contact of the iris and trabecular meshwork. Relative to normal eyes, these eyes typically have a shallow anterior chamber depth (ACD), a small corneal diameter (W2W) and a thick, anteriorly situated lens.

A misconception is that PACsp is exclusive to short, hyperopic eyes. In fact, it can be found in eyes of normal or long axial length (AL). An increasing prevalence of PAC/PACG in axial myopes has been observed, especially in Asian patients.

Mechanisms underlying acute angle closure is described by many authors. The identification of the mechanisms and/or causative factor(s) underlying acute angle closure (AAC) can lead to better management of the disease. Ritch and Lowe[1] have described a classification system for the mechanisms of angle closure that is based on the forces acting at four successive anatomic levels: the iris (pupil block), the ciliary body (plateau iris), the lens (phacomorphic glaucoma) and forces posterior to the lens (malignant glaucoma). Pupil block, considered the primary mechanism of AAC, is alleviated by laser peripheral iridotomy (LPI), which eliminates the pressure differential between the anterior and posterior chambers, flatten the iris and widen the angles[2,3]. However, the variable efficacy of LPI to open the angles coupled with reports of disease progression in the presence of a patent iridotomy suggests that mechanisms other than pupil block may be important in many cases of AAC[4,5].

In the last decade, we have witnessed rapid advancement in imaging techniques for the anterior segment. Greater and more precise imaging provided by ultrasound biomicroscopy (UBM) and anterior segment optical coherence tomography (ASOCT) has revealed characteristics of the anterior segment that have brought about new insights to our understanding of the various mechanisms involved in angle closure. For example, using standardized UBM based criteria, plateau iris was found to be an important mechanism of angle closure in about 30% of cases in a predominantly Chinese population[6,7]. Using ASOCT imaging, several novel anatomical risk factors were identified including greater iris convexity, area and thickness; smaller anterior chamber width and volume; and a large lens vault.

Understanding the precise mechanism(s) leading to AAC may aid in deciding the appropriate approach to treatment. The presence of a non PB component may be recalcitrant to an iridotomy and may be relieved only by a subsequent lens extraction. However, it is

still difficult to identify which eyes with AAC would benefit from an LPI and which would not. At present, LPI remains the primary treatment option for eyes with AAC in order to first eliminate PB. It is also not known why some eyes with AAC progress to chronic angle closure glaucoma stage while others do not. A clearer understanding of the mechanisms involved in AAC is the initial step towards development of personalized care aimed at improving management of AAC and prevention of disease progression and blindness from this disorder.

As we watch the ebb and flow of the debate over the relative contributions of pressure versus vascular factors in the pathogenesis of glaucoma, it might perhaps be prudent not to be too dogmatic about the term acute angle closure glaucoma (AACG).

Microspherophakia with swollen lens can produce acute glaucoma in younger population. Anterior Segment OCT and UBM can show the shallow AC , increased ant-post diameter of the lens and cofirm the diagnosis. As authors described it can only be managed by filtration surgery .

As I mentioned bilateral Acute Angle Closure from Topiramate toxicity – 3 case reports, published in this issue of the JBGS, is a public health problem.

Topiramate, a sulfamate-substituted monosaccharide(Etopira, Topirva, Topmate) primarily used in the management of migraine, Seizure disorders and bipolar disease and may produce acute bilateral angle closure.

The mechanism consists of ciliochoroidal effusion with anterior rotation of the ciliary body and displacement of the lens-iris diaphragm. Topiramate may also disrupt the blood-brain barrier, leading to increased protein content in cerebrospinal fluid and simultaneous blood ocular barrier breakdown, suggesting a common inflammatory mechanism. Initial treatment consisted of discontinuing topiramate and administering ocular hypotensive medications. Use of topical cycloplegia, systemic high-dose steroids, mannitol, peripheral iridotomy, and choroidal drainage have been described.

Whatever mechanisms is involved either primary or secondary from drugs or other diseases, acute angle closure is a public health problem. The mechanisms and pathophysiology should be understood even by non ophthalmologist so they can refer the cases immediately to proper clinics and hospitals. Early diagnosis and its judicious treatment can save the patients eye and save vision almost completely. We need to act accordingly.

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A Study of Retinal Nerve Fiber Layer (RNFL) Thickness Changes in Patients of Type 2 Diabetes Melitus with Chronic Kidney Disease (CKD) Measured by Optical Coherence Tomography

R Mannan[1], F Hossain[2], S Adhikary[3], A Sayeed[4]

Abstract

Purpose: To evaluate the retinal nerve fiber layer (RNFL) thickness changes in patients with chronic kidney disease (CKD)with type 2 diabetes mellitus by using optical coherence tomography (OCT).

Methodology: Thirty six eyes of eighteen patients were evaluated. Among them 18eyes of 9 patients had type 2 diabetes mellitus with chronic kidney disease (CKD)and were included in (group 1) and 18 eyes of 9 ,age-matched, normal control subjects were included in (group 2). The RNFL thicknesses were assessed in both the groups. An RNFL thickness protocol was used to acquire circular scans of 3.4 mm in diameter around the optic nerve. For each eye, RNFL thicknesses were evaluated in 4 quadrants. All the patients underwent comprehensive ophthalmologic examination. The mean and quadrant RNFL thickness values in patients with CKD were compared with the control group.

Result: In group 1 (CKD with type 2 DM): the mean superior, inferior, nasal, and temporal RNFL thickness at the disc margin was 164,172,118 and 102 μm, respectively. In group 2 :the mean superior, inferior, nasal, and temporal RNFL thickness at the disc margin was 262 ,286,146,and 124 μm, respectively. The RNFL thickness in CKD with type 2 DM, measured by OCT, was found to be significantly reduced compared to that of group 2. The average superior and inferior RNFL thickness was inversely related to age (P = 0.031, P = 0.048, respectively).

Conclusion: The RNFL thickness in chronic kidney disease (CKD)with type 2 diabetes mellitus (DM), measured by OCT, was found to be significantly reduced. The presence of CKD and type 2 diabetes mellitus may lead to inappropriate measurement of glaucomatous optic neuropathy.

Keywords : Chronic kidney disease , Optical coherence tomography, Retinal nerve fiber layer thickness

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Introduction

The retinal nerve fiber layer (RNFL) is formed by retinal ganglion cell axons and represents the innermost layer of the retina. The nerve fiber layer collects the visual impulses that begin with the rods and cones. The thickness of the RNFL increases toward the optic disc.

Chronic kidney disease (CKD) is usually defined as kidney damage or reduced glomerular filtration rate (GFR). Here kidney damage implies pathological abnormalities or presence of markers of damage. Normal individuals usually excrete very small amounts of protein in the urine. Persistently increased protein excretion is usually a marker of kidney damage. The excretion of specific types of protein, such as albumin or low molecular weight globulins, depends on the type of kidney disease that is present. Increased excretion of albumin is a sensitive marker for chronic kidney disease due to diabetes, glomerular disease, hypertension etc. The risk of cardiovascular disease, retinopathy, and other diabetic complications is higher in patients with diabetic kidney disease than in diabetic patients without kidney disease. The presence of CKD can be a source of false positive results and lead to overestimation of glaucomatous optic neuropathy. Optical coherence tomography (OCT) is one promising technology which is non-invasive, non-contact method of giving a cross sectional image of the retina and its substructures in a real time mode and in vivo. The resolution of the OCT image is at about 1–15 µm. It provides details 10 times superior to an ultrasound-B scan. Using the OCT, the retinal thickness is given by the distance between the first high reflective layer (that is, the

vitreoretinal interface) and the retinal pigment epithelium. The RNFL thickness is calculated from the reflectivity distribution within the retina, using a special algorithm.

This particular study was conducted to evaluate the retinal nerve fiber layer (RNFL) thickness in patients with chronic chronic kidney disease with type 2 diabetes mellitus by using optical coherence tomography (OCT).

Methodology

Thirty six eyes of eighteen patients were evaluated in this study .Among them 9 patients had type 2 diabetes mellitus(DM) with chronic kidney disease (CKD), were included in (group 1) and 9, age-matched normal control subjects were included in (group 2). The RNFL thicknesses were assessed in both of the groups. An RNFL thickness protocol of Optovue OCT machine was used to acquire circular scans of 3.4 mm in diameter around the optic nerve. For each eye, RNFL thicknesses were evaluated in 4 quadrants. The mean and quadrantic RNFL thickness values in patients with CKD were compared with the control group.

It was an observational study, performed at BIRDEM General Hospital, Dhaka from January 2016 to December 2016. The study protocol was approved by the ethical committee of the institute and strictly followed the Declaration of Helsinki. All the study subjects were explained the use of OCT. An informed, written consent was taken from all the study patients. The study patients had no previous intraocular surgery, ocular inflammation, trauma, optic neuropathy, glaucoma or retinopathy. All the patients underwent comprehensive ophthalmologic examination at the beginning of the study including measurement of BCVA, anterior sement evaluation by slit lamp biomicroscope, gonioscopy, tonometry and fundoscopy. IOP was measured by using Goldmann Applanation Tonometer and gonioscopy was performed by using a Sussman's 4 mirror gonioscope.

Statistical Analysis

Statistical analysis was performed using commercial software. Both the study groups were compared using student's t test. A P value of < 0.05 was considered statistically significant.

Result

36 eyes of eighteen individuals were studied. Age (mean ± standard deviation) was 55.3 ± 19.7 in group 1 and 55.2 ± 17.8 in group 2 (range: 30-76 years). For the eyes studied, the disc area (mean ± standard deviation) and cup:disc ratio (mean ± standard deviation) were 1.93 ± 0.4 mm^2 and 0.3 ± 0.16, respectively in group 1 and 1.88 ± 0.5 mm^2 and 0.35 ± 0.21, respectively in group 2. In group 1 (CKD with type 2 DM) the mean superior, inferior, nasal, and temporal RNFL thickness at the disc margin was 164 ,172,118 ,and 102 µm, respectively. In group 2 the mean superior, inferior, nasal, and temporal RNFL thickness at the disc margin was 262 ,286,146 and 124 µm, respectively. The RNFL thickness in CKD with type 2 DM, measured by OCT, was found to be significantly reduced. The retinal nerve fibre layer thickness were significantly correlated with age. The average superior and inferior RNFL thickness was inversely related to age (P=0.031, P=0.048, respectively).

Table 1 : Distribution Of Age, Sex, Bcva, Mean Iop Among the Study Patients

	GROUP 1	GROUP 2
Age (mean ± standard deviation)(years)	55.3 ± 19.7	55.2 ± 17.8
Sex (M:F)	5:4	4:5
BCVA	6/6 – 6/9	6/6-6/9
Mean IOP (mmHg)	14.3±5.4	15.4±4.3

Table 2 : The RNFL thickness measured by OCT

Mean RNFL thickness (Micro meters)	GROUP 1	GROUP 2
Superior	164	262
Inferior	172	286
Nasal	118	146
Temporal	102	124

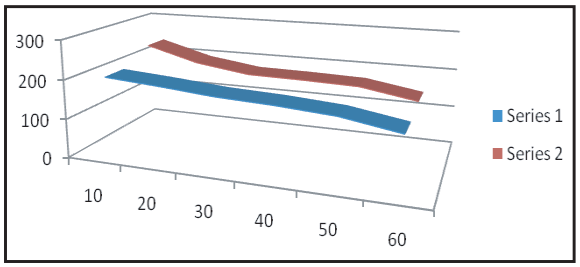

Fig.1 showing changes in RNFL thickness with age. Here, (series 1 : patients with CKD & DM, series 2: normal subjects)

Discussion

Our study confirms the excellent reproducibility of retinal thickness measurements using the OCT. Our reproducibility values are in good agreement with several other studies already published in the literature,[2,4,5] Our study was designed to evaluate the retinal nerve fiber layer (RNFL) thickness changes in patients with chronic kidney disease (CKD) with type 2 diabetes mellitus by using optical coherence tomography (OCT). When it occurs over time, a glaucomatous narrowing of the rim could be missed or incorrectly measured in primary diagnosis or in follow up examinations. For the eyes studied the disc area (mean ± standard deviation) and cup:disc ratio (mean ± standard deviation) were 1.93 ± 0.4 mm^2 and 0.3 ± 0.16, respectively in group 1 and 1.88 ± 0.5 mm^2 and 0.35 ± 0.21, respectively in group 2. In group 1 (CKD with type 2 DM): the mean superior, inferior, nasal, and temporal RNFL thickness at the disc margin was 164,172,118 and 102 µm respectively. In group 2 : the mean superior, inferior, nasal, and temporal RNFL thickness at the disc margin was 262, 286,146 and 124 µm, respectively. The RNFL thickness in CKD with type 2 DM, measured by OCT, was found to be significantly reduced. Although there is paucity of literature and supportive studies of the same subject, there are some studies that have considered patients of chronic renal failure. Additionally, we found a significant deterioration of the OCT. RNFL thickness measurements deteriorate with increasing age. This is not due to lens opacity, because lens opacity also would affect the measurements of the retinal thickness. The mean values of RNFL thickness are within the range already described in the literature. Schumann[5] for example found a RNFL thickness of 91.5 µm in the temporal parapapillary area. In the study he used a circular OCT scan around the optic disc with a diameter of 3.37 mm. In another study he found a RNFL thickness of 126 µm in the temporal area, but this study included only 26 eyes.[6] We found a highly significant correlation of the RNFL thickness with age. The RNFL thickness decreased by 0.44.µm per year. Obviously, about 80% of the changes in retinal thickness over time are caused by a shrinkage of the RNFL.The question, whether there is a decrease in RNFL thickness with age has already been addressed in several other studies.[1,5,9-11] A direct comparison is only possible with the study of Schumann et al,[5] because they used OCT. Schumann[5] examined 59 eyes of 33 subjects. He found a RNFL thickness decrease for the peripapillary RNFL thickness ($p < 0.03$) and the temporal RNFL thickness ($p < 0.0001$). Like in our study he did not find any differences between men and women. Poinooswamy et al[11] examined 150 healthy volunteers of different ages using scanning laser polarimetry. They found a progressive reduction of the RNFL thickness with increasing age. The data presented in their study indicate a significant reduction of the RNFL thickness of 0.38 µm/year. In our study we found a very similar value of 0.43 µm per year. Balazsi et al[1] and Mickelberg et al[4] counted the axons of 16 respectively 22 normal human eyes. They found an axon fibre loss in the optic nerve of 4909 and 5637, respectively, per year. This may be interpreted as a qualitative confirmation of our findings, because any loss of axons should lead to a decrease in RNFL thickness.Jonas et al,[9] using red free photographs, also found a correlation between the visibility of the retinal nerve fibre bundles and age. But there are a few studies that did not find a

correlation between RNFL/retinal thickness and age. Varma et al[8] performed histological examinations of 10 normal enucleated human eyes. They only found a significant correlation with age in the superior-nasal and inferior-temporal region. This, however, may be explained by the small number of eyes examined.

Limitation of the study

If our sample size were bigger, the statistical outcome would have been more efficient.

Conclusions

The RNFL thickness in chronic kidney disease (CKD) with type 2 diabetes mellitus (DM), measured by OCT, was found to be significantly reduced. If our sample size were larger,we could have concluded the study more effectively and the statistical outcome would have been more efficient.The presence of CKD can be a source of false positive results and lead to inaccurate measurement of glaucomatous optic neuropathy.

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Original Article

Visual outcome after manual small incision cataract surgery for Phacomorphic glaucoma

S M Noman[1]

Abstract

Aim : To evaluate the visual outcome after manual small incision cataract surgery (MSICS) as a treatment of phacomorphic glaucoma.

Method : The study included 44 patients with phacomorphic glaucoma treated by manual small incision cataract surgery with intraocular lens implantation. Preoperative and postoperative visual acuity and intraocular pressure have been recorded and compared at the end of six weeks after surgery.

Result : The mean preoperative intraocular pressure was 35.20 (± 10.86) mm of Hg. There were no significant intraoperative complications such as posterior capsular tear or expulsive hemorrhage. Post operative mean intraocular pressure (IOP) was 12.48 (± 3.45) mm Hg. There was a statistically significant difference between IOP at presentation and IOP at last presentation.(P· < 0.0001).Pre operative visual acuity in all the affected eyes were perception of light with projection of rays in all quadrant. Postoperative best corrected visual acuity was 6/6-6/18 in 28 patients (62.80%), 6/24- 6/36 in 10 patients (23.25%) and 6/60 in 6 patients (13.95%).

Conclusion : Manual small incision cataract surgery is a safe and effective method of treatment with minimal or no complications for phacomorphic glaucoma and the visual outcome and IOP reduction is satisfactory.

Introduction

Cataract is the most common cause of avoidable blindness in the world. Bangladesh is one of the densely populated developing countries having about 700 thousand people blind. Cataract contributes 80 % of total blindness in Bangladesh[1]. Limited health care facility and other socioeconomic factors influence the patients for late presentation, sometimes with complication like phacolytic and phacomorphic glaucoma.

The definitive treatment for phacomorphic glaucoma is cataract extraction[6,8]. Surgery in a patient of phacomorphicglaucomahastofacesomechallenges. High intraocular pressure, increases the risk of expulsive haemorrhage, hypermaturity of lens is often associated with zonulolysis which makes surgery technically more difficult. Shallow anterior chamber may cause corneal touch that makes the cornea hazy.. Phacoemulsification is not possible for high intraocular pressure as well as for the inflammation. Conventional extracapsular cataract extraction(ECCE) has some limitations. Manual small incision cataract surgery is suitable for such phacomorphic glaucoma cases as it is relatively easy to approach and manage. The aim of our study is to evaluate the visual outcome and intraocular pressure after manual small incision cataract surgery in the management of phamorphic glaucoma.

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FCPS, DCO

Methods

This is a retrospective review of case series and was conducted at the glaucoma clinic of Chittagong Eye Infirmary and Training Complex, Bangladesh and was approved by institutional review board. A total of 43 patients with phacomorphic glaucoma were included in this study. The patients with inaccurate perception of light, combined mechanism where primary angle closure glaucoma was also associated, zonular dialysis and subluxation of lens where intraocular lens implantation was not possible were excluded from the study. Most of the patients in this study group came from remote area with poor socioeconomic background.

All of the patients presented with gradual loss of vision followed by sudden acute onset of pain with sudden loss of residual vision, redness in the affected eye. The diagnostic features were shallow anterior chamber and hypermature cataract with perception of light and raised intraocular pressure. Conjunctival congestion, corneal edema and inflammation in the anterior chamber were found in all cases. Lenticular changes were capsular calcification or thinning, cortical liquefaction, swelling of the lens, phacodonesis due to zonular weakness. All patients were treated medically prior surgery to reduce

inflammation and intraocular pressure.

All 44 surgeries were done by a single surgeon. Raised intraocular pressure was usually with systemic carbonic anhydrase inhibitor or hyperosmotic agents prior to surgery to soften the eye ball. Ocular inflammation was reduced with frequent topical steroid usage.

Peribulbar block was given in all cases with short and long acting anesthetic agents. Superior rectus briddle suture was placed and superior limbus and adjacent conjunctiva were exposed. Fornix based conjunctival flap was made in the superior part and bleeding blood vessels were cauterized with wet field bipolar cautery. A partial thickness 6mm scleral incision was made 2mm behind the limbus and scleral tunnel was created up to 1mm of the clear cornea. Anterior chamber entry was done with 3.2 mm keratome. Reformation of the anterior chamber was done to create an environment for easy manipulation for the next step. A small perforation was made in the upper part of anterior capsule using a bent 26 G needle attached with a syringe and aspiration of the liquid cortex was done. The capsular bag was then inflated with viscoelastic substance and either continuous curvilinear capsulorrhexis (where possible) or canopener capsulotomy was done.

The tunnel was enlarged on either side up to 6 mm with the help of crescent knife. The nucleus was prolapsed in the anterior chamber by rotation technique and removed by irrigating vectis. After aspiration of remaining cortex with simco cannula, anterior chamber and capsular bag was reformed with viscoelastic substances. A 6 mm PMMA lens was then inserted in the capsular bag and proper positioning was done by dialer. Aspiration of viscoelastic material was done and anterior chamber was reformed with ringer lactate solution. Self sealed limbal wound was covered with conjunctival flap.

Post operatively all patients were treated with topical cycloplegic, steroid and antibiotic. Total ophthalmic examination was done on first post operative day and then one week and six weeks after operation. Detailed ophthalmic examination was done in each follow up.

Results

The demographic details of the 43 patients are showed in Table 1: Mean age of the patients was 59.65 (± 10.44) years and among them 24 were male and 20 female. The fellow eyes of the study patients were cataract in 24 cases (53.49 %), pseudophakic in 15 cases (34.88 %) and aphakic in 5 cases (11.63 %). Mean duration of acute symptoms like pain redness and photophobia was 8.18(±3.60SD) days. Among them most of the patients have a duration of 0-10 days. Preoperative visual acuity was perception of light with projection of rays in all quadrants in all affected cases.

Table 1: Demographic characteristics of the study population

Demographics	Number of Patients (%)
Age (Years)	
Mean (± SD)	59.65 (± 10.44)
Range	40-85
Gender	
Male	23
Female	20
Operated Eye	
Right Eye	20 (46.52 %)
Left Eye	23 (53.48 %)
Status of Fellow Eye	
Cataract	23 (53.49)
Pseudophakia	15 (34.88)
Apahakia	5 (11.63)
Duration of Symptoms (Days)	
Mean (± SD)	9.18 (± 3.60)
0-10 Days	31 (69.8)
11-16 days	11 (25.6)
17-22 days	2 (4.7)
Visual acuity	
Light perception with projection of rays	100%

Pre-operative and post-operative visual acuity was shown in Table-2: Best corrected visual acuity at six week follow up was ≥ 6/18 in 62.80% cases, between 6/24-6/36 in 23.25% cases and < 6/60 in 13.95% cases. Among the all patients 86% cases had postoperative best corrected visual acuity ³ 6/36. The mean refractive status at six weeks follow up was +1.50D cylinder (range +0.5 to +2.5 D) and the median axis was 180^{O}.

Table 2 : Comparison of pre-operative and postoperative visual acuity

Visual Acuity	Pre-Op Uncorrected	Post-op Best Corrected
6/6-6/18	0 (0.0%)	28 (62.80%)
6/24-6/36	0 (0.0%)	10 (23.25%)
< 6/60	43 (100.0%)	6 (13.95%)

The pre operative intraocular pressure ranges from 22-60 mm Hg with the mean of 36.23 (± 10.86) mm Hg. Post operative intraocular pressure ranges from 5-22 mm Hg with the mean of 12.5 (±3.45) mm Hg. (Table: 3).

Table 3: Comparison of pre-operative and postoperative IOP

IOP mmHg	Preoperative
Mean	35.20 (± 10.86)
Range	22 - 60
20 - 29	11 (25.6 %)
30 - 39	13 (30.2 %)
40 - 49	13 (30.2 %)
50 - 60	6 (14.0 %)
IOP mmHg	**Postoperative**
Mean	12.48 (± 3.45)
Range	5 - 22
5 - 8	6 (14.0%)
9 - 12	18 (41.9%)
13 - 16	16 (37.2%)
17 - 22	3 (7.0%)

Discussion

The result of this study showed good visual outcome after manual small incision cataract surgery in patients with phacomorphic glaucoma. Phacomorphic glaucoma is caused by an obstruction of pupil by swollen lens. Extracapsular cataract extraction needs large incision and more surgical manipulation. So there is always a high risk of expulsive hemorrhage and severe post operative inflammation.

Phacoemulsification is not suitable in phacomorphic glaucoma, because the nucleus is swollen, very shallow anterior chamber, high IOP, compromised zonules as well as severe inflammation in almost all cases. There is also a risk of endothelial damage, zonular dialysis, and posterior capsular tear. But in MSICS causing less stress on the zonules, does not need expensive equipments and anterior chamber is more stable due to shelving scleral wound. In this study MSICS gives satisfactory uncorrected vision as it has a low range of post operative astigmatism.

At six weeks visit, 28 patients (62.80%) had best corrected visual acuity (BCVA) of ≥ 6/18, 10 patients (23.25%) had BCVA between 6/24-6/36 and 6 patients had<6/60. This compares favorably with other series in which ECCE was performed in the lens induced glaucoma[4,12,19,20]. Post operative visual acuity was not appreciable in 6 patients in comparison to others. The reason behind the poor visual

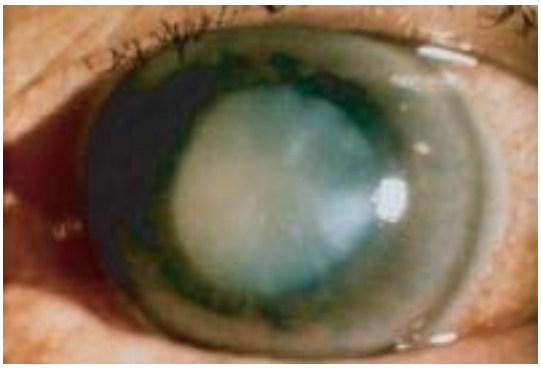

Figure-1 : Phacomorphic glaucoma at presentation

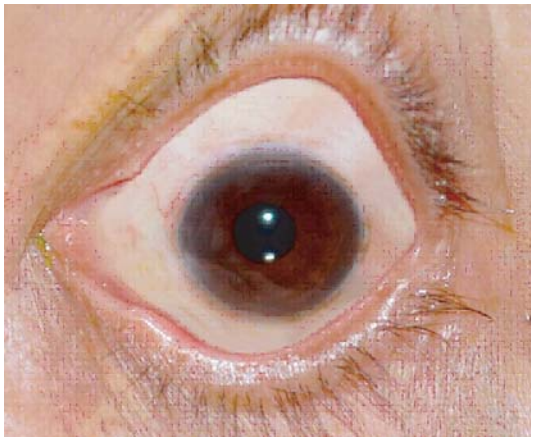

Figure-2 : Phacomorphic glaucoma -6 weeks after surgery

outcome in this group was late presentation of the patient which causes more inflammation and corneal decompensation due to prolong raised pre operative intraocular pressure. The mean post operative astigmatism of our patient is comparable to a series where MSICS was performed in 191 eyes of lens induced glaucoma where the mean astigmatism was 1.20D[20]. Most of the cases in our study the steep axis was 180^0, whose vision were improved with refraction, possibly due to relaxation caused by the superior scleral incision.

The result of post operative visual acuity in our study group is also similar to the result of Venkatesh et al where they showed the post operative outcomes of 33 patients after MSICS in lens induced glaucoma cases.[23] The result is also very much similar with the study of Ramakrishanan R who showed the post operative visual outcome as well as IOP control after MCECS in phacomorphic glaucoma cases.24 Post operative IOP in all cases was controlled without the need for long term anti-glaucoma medications. This is similar to other studies where ECCE performed for lens induced glaucoma.[4,12,19]

Conclusion

In a developing country like Bangladesh, phacomorphic glaucoma is not an uncommon disease due to limited eye care facilities, ignorance and also economical barrier. Our study demonstrates that, MSICS is a safe and effective treatment for the patient with phacomorphic glaucoma due to satisfactory post operative visual outcome and adequate control of intraocular pressure without anti glaucoma medication.

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Original Article

Reduction of IOP & other associated ocular parameters following Phaco-emulsification in ACG

S M Hossain[1]

Purpose : To evaluate the ocular bio-metric parameters in conjunction with Intraocular Pressure (IOP) reduction after phaco-emulsification.

Design: Prospective Observational study.

Methods: A total of 25 patients who had undergone uneventful phacoemulsification were included in the study. IOP and ocular bio metric parameters were checked pre-operatively and four months post-operatively using Goldmann applanation tonometry, optical biometry and anterior segment optical coherence tomography. The relationship between IOP change and the parameters, including preoperative IOP, anterior chamber depth, axial length, angle opening and lens thickness was evaluated.

Results: The mean patient age was 62.3 years. The average change in IOP was - 3.24 mm Hg. Preoperative IOP, anterior chamber depth, angle opening in degrees and lens thickness were significantly associated with IOP change. The axial length was not associated with IOP reduction.

Conclusion: Preoperative IOP, lens thickness and parameters like anterior chamber depth and angle opening in degrees were significantly associated positively with reduced IOP after phaco-emulsification cataract surgery.

Financial Disclosure: The author has no proprietary or commercial interest in any of the materials discussed in this article.

Cataract or Clear lens Extraction has been suggested as a treatment option for different spectra of Primary angle closure diseases. Terminology to describe PACG is confusing and this lack of clarity influences how we think about the disease. Currently 4 angle closure categories. Three requires specific Gonioscopic findings. There is no firm agreement on how many quadrants must have ITC for angle closure to be present, but current consencus appears to be that at least 180^{O} is required.

Progression of Angle Closure with/ or glaucoma decreases by helping to open the angle and control the IOP. Besides PACG, the other glaucoma subsets which have shown best pressure - lowering effects following phacoemulsification are OHT, PXS, POAG

Dr. Shingleton said "Phaco offers Surgeons another option individualizing Glaucoma treatment." However few studies have examined the relationship between a reduction in IOP and ocular biometric or pathologic changes

Aging crystalline lens > Enlarging lens compress the TMW > Collapses schlemm's canal as it presses forward > Outflow channel slowly begins to fail > Raised IOP> Phaco + PCIOL > Compression released > Opening of TMW > changes anterior segment configuration > decrease IOP.

Here we attempt to determine effects of cataract surgery on IOP, identify ocular biometric parameters that effect the reduction in IOP after phaco in ACG

Methods

All patients and their parents were informed accordingly and gave written informed consent to participate in this study in accordance with institutional guidelines. In this hospital based prospective observational study, we included 25 consecutive patients who had non hypertensive IOP values (10-19 mmHg) before surgery and who were scheduled to undergo cataract surgery between 3rd December, 2015 and 15th November, 2016 in Glaucoma department at National Institute of Ophthalmology & Hospital, Dhaka, Bangladesh. Subjects who have been diagnosed with uveitis, retinal diseases, congenital anomalies, Ocular trauma, Intra Ocular Surgeries were excluded. 2.2 mm clear corneal temporal incision phaco-emulsification with posterior chamber foldable intra ocular lens surgeries were singularly performed in all subjects. IOP was measured using Goldmann applanation tonometry 1 day before and 4 months after surgery. Best Corrected Visual Acuity (BCVA) was measured in treated eyes 1 day before and 4 months after surgery. Anterior Chamber Depth (ACD) and Anterior Chamber Angle Distance (ACAD) were measured using Anterior Segment Optical Coherence Tomography (AS-OCT). Lens thickness and axial length were measured using an IOL Master. All biometric values were assessed by single physician.

Anterior chamber depth in millimeter and angle opening distance in degree were measured using AS-OCT (Heidelberg Engineering, Germany). Axial length and lens thickness were measured using IOL master

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(Carl Zeiss, Germany).

Statistical analysis was performed using a commercially available statistical software package. The mean values are presented as means (±) standard deviation. Paired 't' tests were performed to determine the significance of changes in IOP.

Results

Among the 25 patients Scheduled in the analysis 17 were male and 8 female; the average age was 62.3 years. The average BCVA before surgery was 6/6 - 6/18 6 (24%), 6/24 -6/36 8 (32%), 6/60 –hand movement 11 (44%) and the average BCVA 4 month after surgery was 6/6 -6/18 23 (92%), 6/24 -6/36 2(8%), 6/60 – HM 00 respectively.

Table1: Patients characteristics and ocular parameters Pre-surgery and 4 months after Phaco emulsification

Variables	Before surgery	After surgery	p value
Age(Y)	62.3	62.3	
M/F	17/8	17/8	
IOP (mm Hg)	16.56±2.9	13.3±2.0	0.001
BCVA			
Mode	6/60	6/6	
6/6 – 6/18	6(24%)	23(92%)	
6/24- 6/36	8(32%)	2(8%)	
6/60 – Hand movement	11(44%)	00	
ACD(mm)	2.6±0.1	4.0±0.1	0.001
Angle opening in degree	17.0±2.4	30.4±3.1	0.001
Lens thickness(mm)	4.31	N/A	
Axial length(mm)	23.12	N/A	

Table 2: Postoperative Intraocular Pressure Reduction After Phacoemulsification in Primary Angle Closure Suspects

Pre op IOP intervals (mmHg)	Eyes	Mean IOP(mmHg)±SD			Paired t Test value	p value
		Preop IOP	Postop IOP	Change at 4 months		
10-11	2	10.0±0.0	11.5±0.05	1.50±0.05	3.0	0.205
12-15	7	14.14±1.21	10.85±1.21	-3.28±1.1	-7.81	**0.001**
16-19	16	18.4±0.89	14.6±1.02	-3.81±0.20	-18.282	**0.001**
All eyes	25	16.56±2.91	13.32±2.05	-3.24±1.69	-9.58	**0.001**

Graph: Mean IOP differences between before and after phaco emulsification (mm Hg)

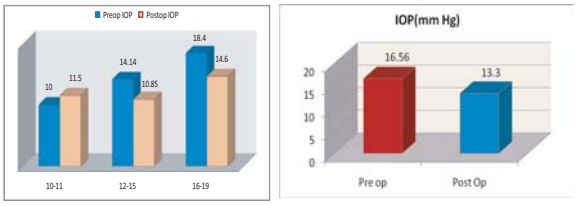


Fig:1a Pre op

Graph: Changes of IOP before and after surgery

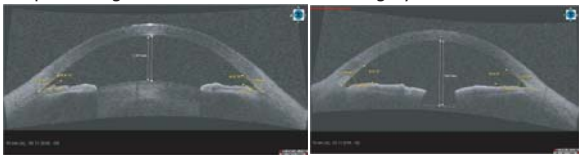
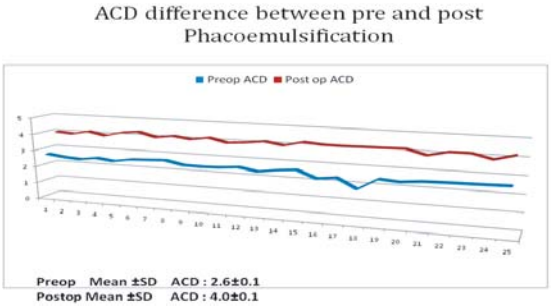


Fig: 1b post op

Table - 1 shows the average axial length before surgery was 23.12 ± 0.21 mm. The average lens thickness before surgery was 4.31 ± 0.19 mm. The average anterior chamber depth (ACD) before surgery was 2.6 ± 0.1 (fig 1a) and post surgery 4.0 ± 0.1 mm (fig 1b). Pre operative average angle opening in degree was 17.0 ± 2.4 (fig 1a) and post operative was 30.4 ± 3.1 (fig 1b).



The average IOP among all the subjects was 16.56 ± 2.91 mmHg and the average change in IOP after 4 months of surgery was -3.24 ± 1.69 mmHg (Table 2).

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Steroid Induced Ocular Hypertension in Post -LASIK Patients: A Matter of Great Concern

U Kawsar[1], S Rahman[2], I Anwar[3], H Rahman[4]

Abstract

Aim : To investigate the fluctuation of intraocular pressure (IOP) due totopical steroidafter laser in situ keratomileusis (LASIK).

Methods : LASIK was performedon 156 eyes of 78 patients for correction of myopia. IOP was measured by Goldmannapplanation tonometer before and after LASIK. All 78 patients who underwent LASIK were enrolled as study group. These aimed at comparing the IOP differences in pre and post-operative changes.

Results : In study group, comparison of preoperative and postoperative IOP, the amplitude of abnormal mean IOP fluctuations reached14.4mmHg (ranged from 18. 2 to 32.6 mmHg) for 19 patients. After receiving treatment with Timolol 0.5%,18 patients got back their intra ocular pressure within normal range (18.1 mmHg) at 5th month visit.

Conclusion : Steroid is important for post LASIK inflammation management. But it induces IOP fluctuations beyond normal range that we observed in our study. So it is matter of great concern to evaluate IOP not only before LASIK but also after LASIK. Immediate treatment to lower IOP can save the sight against POAG (Primary Open Angle Glaucoma). Unfortunately we noticed POAG for case due to steroid use after LASIK.

Keywords : Intraocular Pressure; Primary Open Angle Glaucoma; LASIK, Steroid

Introduction

Refractive procedures have become a popular surgical option for the treatment of myopia. Laser in Situ Keratomileusis (LASIK) has become the most accepted surgical modality for the correction of a wide range of myopia. LASIK is performed routinely in more and more patients. LASIK corrects myopia by altering the thickness and curvature of the central cornea. Continuous innovations and incorporating new technology such as latest generation microkeratomes, femtosecond laser[1], and wavefront technology[2] to this field enabled the prosperity and progress of LASIK over the world. And clinical outcomes of LASIK also gained traction and studied amply. Among the vast majority of publications, postoperative outcomes and visual quality assessment, especially postoperative complications have drawn much attention.[3,4]

Ocular hypertension is such a hot topic in this domain. Topical corticoid is routinely prescribed for corneal wound healing in early post - LASIK patients. However, it may induce elevation of intraocular pressure (IOP), and serious consequences in steroid-sensitive patients, such as steroidinduced lamellar keratitis[5], interface fluid syndrome[6], refractive regression[7], visual acuity loss[8], and even visual field defects[9]. Early recognition of these signs and symptoms, followed by proper treatment might be a brake to ocular hypertension and reverse deteriorating consequences. Recently, IOP fluctuation and variation was highlighted as a risk factor for glaucoma progression[10,11]. However, the influence of abnormal IOP fluctuations on visual performance has received little attention[12]. We conduct this prospective study to evaluate greater-than-normal IOP fluctuations in steroid responders after refractive surgery.

Subjects And Methods

78 patients who had LASIK during January 2016 to August 2018 in Vision Eye Hospital were enrolled as study group. Patients previous ocular surface diseases such as corneal injury or illness, ocular surgery, any sign of keratoconus, soft contact lens wear during the 2 week prior to presentation, and those who were pregnant were excluded. None of the 78 patients reported a history of systemic or ophthalmic diseases.

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All procedures were performed in accordance with the ethical standards, and informed consent was obtained from all patients prior to the study.

Laser in Situ Keratomileusis Procedure:LASIK surgery consisted of two major steps: flap creation and laser ablation.Flap diameters of 8. 5 to 9. 0 mm were created with Moria One Plus microkeratome (Moria, Antony, France) or Amadeus II (Zeimer Group)or Wavelight FS200 femtosecond laser (Alcon, USA), and the optical zones ranged from 6.0 to 8. 0 mm in diameter. All eyes underwent LASIK profiles (WavelightOculyzer II pentacam) and standard Lasik Procedure by Allegretto EX500 excimer laser (Alcon, USA).

Postoperative Management: Routine postoperative management included topical Levofloxacin 0.5% sterile eye drop 4 times a day for 4 weeks, Dexamethasone 0.1% sterile eye drop 4 times a day for 4 weeks and Carboxymethylcellulose Sodium 1% sterile eye drop4 times daily for 4 weeks. After the first post-operative day visit, all patients scheduled next two follow-up visits at post – 1st week and 4thweek. But patients identified with ocular hypertension had scheduled additional two more visit at2ndmonth& 5th month and each of them had received immediate hypotension therapy, such as discontinuation of topical corticosteroidsin a rapid tapering mode within 7 days and adding topical 0. 5% Timololmaleate twice daily till to normal tension.

Postoperative Outcomes Assessment: Patients were consecutively evaluated at postoperative 1week and 1month. IOP was measured with Goldmann applanation tonometry (GAT) and then the corrected IOP was calculated according to the Ehlers method by taking into account the postoperative Pachymetry (measured by ultrasonic Pachymetry: OcuscanRxP, Alcon, USA) and the IOP measured with GAT[13]. All instruments involved were the same.

Results

All the patients were divided into two category. Category 1, those who had elevated IOP within normal range and Category 2, who showed elevated IOP beyond normal. At the 1st monthexaminations, 19cases (out of 78) showed steroid induced ocular hypertension. Compared with preoperative value, the amplitude of postoperative abnormal IOP fluctuations reached 14.4mmHg(ranged from 18. 2 to 32.6 mmHg). (Table 1).

Table 1: Preoperative and postoperative Mean IOP analysis

Category	No. of patients	Preoperative Mean IOP	Postoperative Mean IOP (1st month visit)
Category-1	59	17.3 mmHg	19.7 mmHg
Category-2	19	18.2 mmHg	32.6 mmHg

Category-2 was then treated with topical 0. 5% Timolol maleate twice daily. Two more visit was scheduled at 2nd and 5th month respectively. Except one case all 18 patients got their ocular hypertension within normal range (Table 2)

Table 1: Postoperative Mean IOP analysis of Category-2 treated with Timolol 0.5%

Category	No. of patients	Preoperative Mean IOP (2nd month visit)	Postoperative Mean IOP (5th month visit)
Group-1	18	26.2 mmHg	18.1 mmHg
Group-2	1	31.7 mmHg	30.8 mmHg

Discussion

The demand for better visual outcomes after LASIK has led to the emphasis on the postoperative management, especially patients in the first postoperative month with topical corticoid eye drops to regulate corneal wound healing[1, 14]. Some steroid - sensitive patients may risk ocular hypertension, even worse consequences. Fortunately, the greater- than - normal IOP fluctuations could be limited if identified and managed in time. So weconducted this controlled study to monitor postoperative variation of IOP. And we found that abnormal IOP fluctuations might cause remarkable changes in optical and visual performance after LASIK. We could safely conclude that abnormal IOP fluctuations could deteriorate visual performance in post-LASIK patients. Our study found 19 cases who had elevated IOP due to steroid. After receiving treament with Timolol 0.5% 18 cases got their IOP within normal range. One case was different as steroid induced ocular hypertension had led to POAG. So it is very much important to analyze IOP before and after LASIK in treament of Myopia. And also limiting the IOP fluctuations could improve the visual performance in those steroid responders.

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Pediatric Glaucoma and its Medical Management

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Abstract

Childhood glaucomas are challenging to diagnose. This group of disease which is heterogeneous in type can cause a rapid loss of vision of children or even blind. So timely recognition proper and optimal treatment of pediatric glaucoma is very important. Fortunately, ophthalmologists often have at their disposal the tools needed to diagnose and manage these children.

Successful control of IOP is crucial and challenging .Most often achieved surgically, with medical therapy as a supportive role.

Key Words : Pediatric glaucomas, medical therapy, amblyopia, IOP.

Introduction

Pediatric glaucoma signs vary largely among children depending on the severity of the IOP (Intraocular Pressure) rise, suddenness, and the age of the child. Clinical studies indicate common signs such as corneal edema, megalo-cornea, buphthalmos, optic nerve cuping, conjunctival injection, anisometropia, myopia, strabismus, and amblyopia. At the first year of life, glaucoma is normally supposed owing to the symptoms that are associated with secondary corneal changes. Equally, children who are considerably older are identified with the disease through the loss of vision caused by chronic glaucoma as well as signs of vomiting and pain associated with acute glaucoma. Similarly, the elevation of IOP is needed to ascertain the presence of pediatric glaucoma as the range of normal IOP in childhood approximates the normal adult range, and it is rare to have measurements greater than 22 mm Hg or below 10 mm Hg, and these measurements are essential in the management of children with glaucoma[10]. In infantile glaucoma, the iris indicates a more anterior insertion than one of a normal infant, showing altered translucency of the angle face yielding a unique ciliary body band, sclera spur and trabecular mesh. Furthermore, corneal opacification and swelling are common signs of glaucoma in infants. Children with pediatric glaucoma can also be seen to withdraw from light, burying their heads to prevent exposure to light as well as rubbing of eye.

This article reviews the current and up to date standards in the treatment of glaucoma among the pediatric population, focusing on ongoing researches on medications used in pediatric patients, the current guidelines of treatment and a provision of recommendatory effective medication.

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Classification

There are many classifications of childhood glaucoma, but they can simply be classified as Primary, in which a developmental abnormality of the anterior chamber (AC) angle only exists. Primary glaucoma in children is generally divided based on age of onset into primary congenital glaucoma (PCG), from birth to early childhood, and juvenile primary open-angle glaucoma (JOAG), from 4 years to early adulthood. Secondary glaucoma includes a variety of conditions resulting from damage to the aqueous outflow system due to congenital or acquired ocular diseases or systemic disorders.

The worldwide prevalence of childhood blindness ranges from 0.03% in high-income countries to 0.12% in undeveloped countries. The causes of severe visual impairment and blindness are varied and complex: glaucoma accounts for 4.2%–5.0% of blindness in the pediatric population.[2,4]

Medical therapy of Pediatric glaucoma

Medical therapy is usually allocated to a supportive role in the management of pediatric glaucoma patients. In children, medical therapy is used to reduce intraocular pressure temporarily or to clear the cornea so that surgical therapy, the definitive treatment for primary congenital glaucoma, can be undertaken. Those patients who do require long-term medical therapy usually have intractable disease that

has not responded adequately to surgical therapy. Our purpose is to review medical therapy of pediatric

patients. Some children with congenital glaucoma and elevated intraocular pressure respond well to medical therapy. In 161 eyes with congenital glaucoma, medical therapy by itself reduced the intraocular pressure to less than 21 mm Hg in 12% of eyes in the short term and 10% of eyes in the long term [1]. When considering medical therapy in pediatric patients, clinicians should evaluate the risks and benefits of specific medications, use the minimum dosages required for therapeutic efficacy, and follow the patient closely for ocular and systemic side effects.[2,3] Although regulatory agencies worldwide do not typically include children in antiglaucoma drug approval studies, clinicians have found several medications to be useful in treating children with elevated intraocular pressure.

As a primary approach to childhood glaucoma, medication alone usually has a provisional efficacy, particularly in PCG. However, medical treatment should be considered first-line for JOAG, uveitis-related glaucoma, and glaucoma after cataract removal. Different kinds of childhood glaucoma may respond differently to topical hypotensive drug. In the case of PCG, universally recognized as a surgical condition, pharmacologic therapy may help to alleviate pain and photophobia and to recover corneal transparency for a better view of the AC angle, especially before angle surgery.

a) Beta - Blockers

Beta-blockers can be an excellent choice for lowering IOP in older children because the drugs may be dosed bd , depending upon their formulation, and usually reduce IOP by approximately 25%. In infants and young children, practitioners should start with the lowest available dose

Plasma timolol levels in children after treatment with 0.25% timolol greatly exceed those in adults after instillation of 0.5% timolol, particularly in infants.[13] Increased plasma timolol levels in children are explained by the volume of distribution of the drug, which is much smaller in children compared with adults. The ocular volume of the neonate is approximately half that of the adult, reaching full size by approximately 2 years, whereas the blood volume of the neonate as a function of body weight is only a small fraction of that of the adult

In addition, the infant's immature metabolic enzyme systems may prolong the half-life of drugs in the neonate from two to six times beyond that of the typical adult.[14] Higher plasma levels of drug in infants and children would be expected to increase the risk of systemic side effects as compared with adults.

Alarming side effects of timolol therapy, such as Cheyne-Stokes breathing and apneic spells, have been reported, especially in infants and younger children.[15-17] Provocation of asthma has been associated with timolol treatment. Betaxolol, a selective b1 antagonist, reduces the risk of pulmonary side effects in adults as compared with timolol, but its effect on children is not known. Likewise, the effects of long-term use of any of the topical beta-blockers in children have not been reported. Timolol in 0.25% and 0.5% solutions should be used cautiously in young glaucoma patients. Because of the possibility of apnea, the drug should be used with extreme caution in neonates. A detailed pediatric history and examination to elicit the presence of systemic abnormalities, such as bronchial asthma and cardiac disease, should precede the use of timolol. In such cases, therapy with a beta-blocker is contraindicated. The use of 0.25% timolol instead of 0.5% timolol is strongly recommended to reduce the risk of side effects. Additionally, a significant reduction in the systemic absorption of timolol has been observed when performing punctal occlusion and simple eyelid closure after drop administration.[13] Although the practice of punctal occlusion with the eyelids closed for at least 1 minute may be considerably more difficult to execute in children than in adults, simply blotting off excess drops from the child's lids may help to minimize unwanted systemic absorption.[13] Once-daily dosing with timolol 0.25% in a gelforming solution may similarly help to simplify the medical regimen.

2. Cholinergic drugs or Miotics

Miotics are generally useful in children only for constricting the pupil in order to protect the crystalline lens during surgery and to keep the cleft open after angle surgery in infants and young children. Pilocarpine or echothiophate iodide may be used in select cases of aphakic glaucoma.

Although miotic drugs enhance aqueous outflow through the trabecular meshwork in normal patients as well as in glaucoma patients, thereby lowering

intraocular pressure, these drugs are likely not as effective in developmental glaucoma. This reduced efficacy in developmental glaucoma is believed to be attributable to the abnormal insertion of the ciliary muscle into the trabecular meshwork. In pediatric patients, the use of pilocarpine (2% applied every 6–8 hours) has been limited.[6] These drugs may benefit aphakic and pseudophakic children with elevated intraocular pressure, however. In addition, cholinergic drugs serve a useful purpose in the surgical management of pediatric glaucoma by causing pupillary miosis before and after goniotomy.[22] The induced myopia produced by miotic therapy can cause disabling visual difficulties. A slow-release pilocarpine membrane delivery system, Ocusert (Alza Pharmaceuticals, Palo Alto, California), currently not available from the manufacturer, was helpful in some young patients[24] and the sudden release of pilocarpine (burst effect) seldom induced myopic spasms. The long-acting anticholinesterase drugs are not readily available, are associated with serious adverse effects, and offer no advantages over pilocarpine for use in pediatric glaucoma. Echothiophate iodide (phospholine iodide), usually administered topically every 12 to 24 hours, is a potent and relatively irreversible inhibitor of the enzyme cholinesterase. Ciliary spasm and angle-closure glaucoma have been

precipitated by the use of echothiophate iodide to treat esotropia in a child.[25] Also, the systemic absorption of anticholinesterase agents can significantly reduce the serum cholinesterase and pseudocholinesterase levels. Affected patients, particularly children, may show signs of excessive parasympathetic nervous system activation. These signs can include generalized weakness, diarrhea, nausea, vomiting, excessive salivation, and decreased heart rate. Significantly reduced systemic levels of cholinesterase and pseudocholinesterase can be particularly dangerous when surgery is contemplated, because succinylcholine is commonly used as a muscle relaxant during general anesthesia. Succinylcholine is normally quickly hydrolyzed by systemic cholinesterase at nerve endings. When serum cholinesterase levels are low, however, prolonged apnea can result because of this excess of unhydrolyzed succinylcholine.

3. Alpha-2 Agonists

Brimonidine is absolutely contraindicated in infants and young children due to its depression of the central nervous system. The drug also causes sedation and fatigue in school-aged children, so it must be used with caution in this population, if at all[12,13] In contrast, even fairly young children usually tolerate apraclonidine 0.5% well, and the drug can help achieve a short-term reduction in IOP and can decrease bleeding at the time of angle surgery.

Several noncomparative case series describing the use of brimonidine in pediatric glaucoma patients exist in the ophthalmic literature, whereas the pediatric use of apraclonidine has not been described. In 30 patients with a mean age of 10 years, brimonidine treatment achieved a mean 7% reduction in intraocular pressure from baseline.[18] Two young children (aged 2 and 4 years) were transiently unarousable after administration of brimonidine, and five other children experienced severe fatigue.[18] In a study of 23 patients with a mean age of 8 years, 18% experienced serious systemic adverse effects necessitating cessation of the drug.[19] Four pediatric patients have been reported to develop somnolence after treatment with brimonidine.[20] Additionally, a 1-month-old infant experienced repeated episodes of coma, characterized by unresponsiveness, hypotension, hypotonia, hypothermia and bradycardia after treatment with brimonidine.[21] The a 2 agonists are less often used in pediatric patients compared with adult patients. The possibility of central nervous system–mediated side effects is greater with lipophilic drugs (eg, brimonidine) than for more hydrophilic drugs (eg, apraclonidine), which are less likely to cross the blood-brain barrier. Iopidine may help to minimize intraoperative hyphema precipitated by goniotomy.[22] Brimonidine should be used cautiously in pediatric patients, and its use should be restricted to older children.

4. Prostaglandin-related drugs

Latanoprost seems to be quite useful for the treatment of juvenile open-angle glaucoma and select cases of primary congenital and aphakic glaucoma.

Prostaglandin-related drugs, specifically latanoprost, have been evaluated in studies of a variety of glaucoma diagnoses, including glaucoma associated with Sturge-Weber syndrome.[26-30] In 31 eyes with a variety of glaucoma diagnoses, 6 (19%) of the treated eyes responded with a mean reduction in intraocular pressure of 8.5 mm Hg, which represented

a 34% decrease from baseline. Most eyes were nonresponders, however. Subjects who responded favorably were more likely to have juvenile onset open-angle glaucoma and to be older than nonresponders.[26] The drug was well tolerated in this short-term study. In glaucoma associated with Sturge-Weber syndrome, 17% to 28% of eyes treated with latanoprost responded with a reduction in intraocular pressure.[27-28] Increased episcleral venous engorgement was noted, and one patient (6%) discontinued latanoprost therapy because of intolerable hyperemia of the conjunctiva.[28] Although a decline in success over time was noted, half of the patients were controlled at 1 year of follow-up after a trial of latanoprost as adjunctive therapy.[29]

Although most children do not respond well to latanoprost therapy, some children may experience an appreciable hypotensive effect with treatment.[30] Likewise, the once-daily dosing schedule for latanoprost is convenient. Although local side effects are infrequent and mild, parents and patients should be warned about them, including iris pigmentation changes, eyelash growth, and hyperemia. When short-term medical therapy is planned, such as before surgery, these local side effects are usually not a problem. The prevalence and types of side effects associated with long-term latanoprost use are not known, however.

According to Wright, primary infantile glaucoma is usually treated surgically, although medications have a definite role as adjuvant therapy, and, rarely, as the sole form of intervention.[7] Enyedi and Freedman study proposes Latanoprost as a treatment for pediatric glaucoma. The study indicates that Latanoprost is a prostaglandin F2 analog, which decreases IOP by 20-40% with ocular hypertension in adults or with open-angle glaucoma.[12] According to Enyedi and Freedman, juvenile-onset and older children with open-angle glaucoma showed significant effects of ocular hypotensive when treated latanoprost.[12] Similarly, Black et al, study endeavors to examine the effectiveness of latanoprost in the treatment of pediatric glaucoma[2]. Their study focuses on assessing the lowering effect of IOP when using Latanoplast and its safety in children, exposing a total of 115 children with pediatric glaucoma for a period of 1 month while recording any side-effect information. Black et al. found out that out of the 115 children participants, 63 of them had IOP data that could be interpreted and side effects were mild, confirming the safety of using the drug in pediatric glaucoma[2]. Accordingly, Black et al. indicate that latanoprost has an IOP lowering ability in juvenile and old children with open-angle glaucoma and a small percentage of those with aphakic glaucoma[2].

5. Carbonic anhydrase inhibitors

In adult patients, the side effects of systemic carbonic anhydrase inhibitors are well known to clinicians. In children, growth suppression has been associated with oral acetazolamide therapy, and infants may experience severe metabolic acidosis.[4,5] Side effects from the use of systemic carbonic anhydrase inhibitors in infants and young children are not commonly reported, although these patients may not verbalize the occurrence of side effects to their parents or health care providers. Oral administration of acetazolamide suspension at a dosage of 10 (range: 5–15) mg/kg/d in divided doses (three times daily) is usually tolerated by children, reduces intraocular pressure, and diminishes corneal edema before surgery.[6,7] Topical versus oral carbonic anhydrase inhibitor therapy has been evaluated for pediatric glaucoma in a crossover design study.[8] The mean intraocular pressure was reduced by 36% and 27% compared with baseline after treatment with oral acetazolamide and topical dorzolamide, respectively. Although not as effective as acetazolamide in this group of patients, topical dorzolamide caused a significant reduction in intraocular pressure.[8] Furthermore, treatment with topical dorzolamide caused few side effects. Topical carbonic anhydrase inhibitors are more widely prescribed compared with systemic carbonic anhydrase inhibitors. Many clinicians prefer twice-daily dosing to minimize the discomfort and inconvenience to the parent and the child associated with three times daily dosing. Taking the fixed combination of dorzolamide with timolol twice a day can simplify the medical regimen by reducing the number of drops instilled per day and may be more appropriate for older children.

5. Osmotic drugs

Glycerol is administered orally at a dose of 0.75 to 1.5 g/kg of body weight in a 50% solution.[31] The excessively sweet taste may be partially masked by chilling the solution over ice or by using fruit juice (commonly lemon or orange) or flavored water as a diluent. This drug is not commonly used in the

treatment of developmental glaucoma. Mannitol (20% solution) is dosed intravenously at 0.5 to 1.5 g/kg of body weight at approximately 60 drops per minute. A rapid fall in intraocular pressure occurs in 20 to 30 minutes after drug administration and can last for 4 to 10 hours. Mannitol may also be used to reduce markedly elevated intraocular pressure before surgery in patients with developmental glaucomas refractory to standard medical therapy.

Discussion

Various treatment options have been suggested by various studies in the treatment of pediatric glaucoma.[1,3-5] As an adjunctive therapy, latanoprost is tolerated with good IOP response, and although the response rate is low in the pediatric population, in those that do respond it is very effective and offers good 24-hour control. According to Netland, the frequency of these side effects in children on long-term therapy is not well known.[9] It is also noted in the studies that timolol has been tested as an additional medication in uncontrolled pediatric glaucoma. As in adults, systemic levels of timolol can be found in pediatric patients after topical dosing, but at much higher levels and much of the plasma level increase can be explained by the much smaller volume of distribution in children as compared to adults. Furthermore, various studies indicate that oral carbonic anhydrase inhibitor administration can cause growth retardation and metabolic acidosis.[9] Moreover, a study of children between age 3 and 12 showed an effective lowering of IOP in pediatric glaucoma when comparing the use of systemic administration of topical dorzolamide and acetazolamide.

Notably, based on the study, epinephrine and dipivefrin nonspecific adrenergic agonists, are rarely used in pediatric patients, since, when considered for medical therapy, systemic side effects may limit their use in this population. Various researchers have studied the use of brimonidine, an alpha-2-selective agonist in the treatment of pediatric populations. Brimonidine has shown a slight decrease in IOP with a high rate of central nervous system depression while in other studies, side effects were sufficient to merit discontinuation of the therapy.[5]

In the use of cholinergic drugs, they were the first medical treatment for glaucoma but are now seldom used for pediatric patients. Topical use of both pilocarpine and carbochol can be associated with cholinergic side effects, including diarrhea, gastrointestinal cramping, hypotension, headaches, salivation, syncope, and sweating.[4] The degree to which side effects are experiences is highly dependent on systemic absorption, which can be greater in pediatric patients. Since the majority of pediatric glaucomas result from structural and developed mental abnormalities of the angle and associated structures, these drugs may be less effective in lowering IOP. Furthermore, pilocarpine has been used to a limited degree in pediatric patients, and it may be used for induction of miosis pre and postoperatively for surgical goniotomy. Equally, long acting anticholinesterases such as echothiophate iodide are used mostly for the treatment of accommodative esotropia. Netland indicates that, since the agents are of poor availability, with no advantages over pilocarpine and with more serious side effects, they are seldom used for glaucoma therapy.[9]

Conversely, medical treatments of pediatric glaucoma could include oral and topical carbonic anhydrase inhibitors, adrenergic agents, prostaglandins, and miotics. Often, oral carbonic anhydrase inhibitors are used to lower intraocular pressure in an attempt to clear the cornea for goniotomy surgery.

Summary

Goals of medical therapy in pediatric glaucoma is to decrease intraocular pressure temporarily, to clear an edematous cornea, and to facilitate surgical intervention.

Most pediatric patients who require long-term medical therapy have severe disease that has not responded sufficiently to surgical therapy, and these patients may experience additional intraocular pressure reduction with a medical treatment. Oviously before commencing medical glaucoma therapy in a child, clinicians need to consider the potential for side effects very carefully. During treatment, children need to be closely monitored because they may be at increased risk of systemic side effects compared with adults as a result of their reduced body mass and blood volume for drug distribution. Similar caution should be exercised when treating the pregnant glaucoma patient or the nursing mother.

Conclusion and Recommendations

Pediatric glaucomas associated with a relatively poor surgical outcome, such as secondary glaucoma associated with aphakia, a medication trial is

frequently warranted prior to surgical treatment. Medical therapy is also valuable postoperatively in controlling glaucoma in children in whom surgery is only moderately successful. Furthermore, physicians should be aware of the potential side effects. Topical carbonic anhydrase inhibitors are both effective and save adjunctive agents in the treatment of pediatric glaucoma. Physicians need to be wary when using topical -blockers during the neonatal period and in premature infants since there is a definite risk of bronchospasm and bradycardia. Furthermore, beta-blockers should not be used in infants or children with a history of reactive airway disease. In the treatment of pediatric glaucoma, the consultation with the patient's pediatrician and pulmonologist or cardiologist may be worthwhile if surgical or other medical options are limited. Parasympathomimetics and carbon anhydrase inhibitors have also been used systematically. In addition, side effects noted in newborns treated with glaucoma medications provide help in choosing treatment in late stages of pregnancy.

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Review Article

Angle-closure Glaucoma : The Role of the Lens in the Pathogenesis, Prevention and Treatment

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Abstract

Primary angle-closure glaucoma is a major cause of blindness worldwide. It is a disease of ocular anatomy that is related to pupillary-block and angle-crowding mechanisms of filtration angle. Eyes at increased risk for primary angle-closure are small with decreased axial length, anterior chamber depth, and filtration angle width, associated with a proportionately large lens. Angle-closure glaucoma afflicts Asian and Eskimo eyes more frequently than eyes in other races with similar predisposing dimensions. The treatment of primary angle closure addresses its causal mechanisms. Laser peripheral iridotomy equalizes the anterior and posterior pressures and widens the filtration angle by reducing the effect of pupillary block. Argon laser peripheral iridoplasty contracts the iris stroma to reduce angle crowding and is helpful for some affected eyes. Lensectomy dramatically widens the angle and eliminates pupillary block. Clinical reports of lensectomy with posterior chamber intraocular lens implantation in the treatment of acute, chronic, and secondary angle-closure glaucoma describe very favorable results. The appropriate role for lensectomy in the management of primary angle closure, however, remains unproven. Prospective, randomized clinical trials are ongoing to determine the value and comparative risks and efficacy of lensectomy versus medical therapy, laser peripheral iridotomy, laser iridoplasty, and filtration procedures for the treatment of acute and chronic primary angle closure and for the prevention of chronic angle-closure glaucoma, both after and in place of laser peripheral iridotomy.

Key words : angle-closure glaucoma, blindness, goniosynechialysis, iridotomy, lensectomy, phacoemulsification.

I. Introduction

Angle closure is a disorder of ocular anatomy characterized by closure of the drainage angle by appositional or synechial approximation of the iris against the trabecular meshwork, blocking its access to aqueous humor. The final common result in related disorders is an elevation of the intraocular pressure (IOP), due to the secondary impairment of aqueous humor outflow from the eye, followed by the development of glaucomatous optic neuropathy.This review will focus on the role of the lens in the pathogenesis and treatment of primary and secondary angle closure.

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II. Clinical Types of Angle-closure Glaucoma

Angle-closure disorders can be divided into primary and secondary groups. Primary angle closure includes those that are caused by pupillary block, angle crowding (from plateau iris configuration or anterior lens position) or a combination of both[50]. A classification endorsed by the American Academy of Ophthalmology subdivides the primary group into primary angle-closure suspect, primary angle closure without optic neuropathy (PAC), and primary angle-closure glaucoma with neuropathy (PACG)[4]. Secondary angle-closure disorders are those that occur in the presence of a second ocular disease such as with iris neovascularization, uveitis, trauma, or lens related conditions[18].

III. Epidemiology of Primary Angle-closure Glaucoma

It has been estimated that 67 million people worldwide are affected with a primary glaucoma and that one-third have PACG[95]. In European and African populations primary open-angle glaucoma (POAG) occurs approximately five times more frequently than PACG; in Chinese[20,21], Mongolians[19], and Indians[15], however, the rates of PACG may equal or be greater than POAG. In Eskimos/ Inuit the prevalence of PACG is felt to be higher than any other ethnic group[12]. In Bangladesh 2% population suffers from glaucoma. Among these PACG is almost equal to POAG. A prevalence study reported PACG as occurring more frequently in Italy than in the rest of the European population (0.6 - 0.1%)[22] In China alone an estimated 3.5 million people are afflicted with the disease, and 28 million are estimated to have occludable drainage angles[20]. The incidence of PAC increases with age and is greater in females[3]. The annual rate of APAC in Singapore was highest (68.5/100,000) for elderly Chinese women with a two times higher rate than in males[107]. Angle closure is a disease of older persons, peaking in incidence between 55 and 70 years of age[96]. The risk of APAC

in elderly Singaporeans greater than 60 years of age was nine times greater compared with the 30 to 59 year old population[109]. PACG is more visually destructive than POAG, and it is responsible for the majority of the bilateral glaucoma-related blindness in Singapore[22]. The proportion of those with PACG who become blind is over 25%, which is double that of POAG[20]. Angle closure glaucoma patients, presenting with an acute attack, are felt to represent only a minority of patients potentially visually affected by PACG[19]. The predominant form of PACG is the chronic, asymptomatic type, and because the diagnosis does require gonioscopy, it is likely that a large proportion of those at risk remain undiagnosed and untreated[93]. Even in Singapore, where glaucoma is responsible for 60% of blindness and the incidence of angle closure is high, awareness of the problem is low[108].

IV. Mechanisms in Angle-closure Glaucoma

Pupillary block is the most frequent and important mechanism responsible for angle closure[14,97], but in many cases it is not the only mechanism involved. Iris angle-crowding may co-exist with pupillary block to cause the angle closure. In the plateau iris configuration, the iris is held anteriorly by the ciliary processes, but a pupillary block component may also be present[132]. These pathologic mechanisms exist because of primary anatomic variations in the size, position, and relationship of the anterior segment structures (cornea, iris, ciliary body, lens), or occur secondary to other acquired ocular pathology. For example, the lens may shift anteriorly secondary to trauma or to drugs that result in the formation of fluid in the supraciliary space[107], or the lens may move anteriorly secondary to expansion of posterior segment structures (vitreous, subretinal space, choroid)[94]. Anterior traction on the peripheral iris may pull the iris over the trabecular meshwork with resultant blockage of access to aqueous. This mechanism of trabecular obstruction occurs with contraction of neovascular, inflammatory, or proliferative fibrocellular membranes and is common in clinical ocular conditions including rubeosis irides, chronic anterior uveitis, aniridia, and after cataract surgery in infancy. Because this article will only review the role of the lens in the pathogenesis of the angle closure, these mechanisms related to anterior traction on the iris, which cause angle obstruction independent of the lens, will not be further discussed.

A. PUPILLARY BLOCK MECHANISM

Aqueous humor (AH) is produced into the posterior chamber and normally flows anteriorly between the posterior surface of the iris and the anterior lens capsule, to enter the anterior chamber through the pupil, and exits through the trabecular meshwork (TM). Relative resistance to flow of AH from the posterior chamber (PC) into the anterior chamber (AC) is normal and reflected by an estimated pressure drop of 0.23 mm Hg between the PC and AC[35]. This pressure differential may increase greatly when the dimensions of the iris-lens channel are changed in such a manner that flow of AH is more impeded[113]. The pressure difference between the PC versus the AC is an important variable that determines the iris contour. As this pressure increment increases, the iris becomes more convex. Clinically significant pupillary-block is present when the increased iris convexity brings the iris into apposition with the TM or appears close enough to do so in the future. Extreme anterior iris-bulging, iris bombe, would be expected with pressure differentials of 10-15 mm Hg[35]. The variables that influence the AH flow through the "pinch region" (iris-lens channel) and influence the pressure differential and related iris contour have been studied extensively.[35,48,78,103,113] Changing pupillary size within the normal range of 3 to 7 mm was determined to have little effect, however, miosis of 2 mm was predicted to significantly increase the pressure differential especially in the presence of increased channel length (O1 mm) or decreased height (!5 mm)[35,113]. Increased channel length and decreased height were associated with increased pressure increments and were accompanied by the expected increased iris convexity. Movement of the iris insertion posteriorly or the lens anteriorly also was associated with an expected increase in the pressure differential and iris convexity, which itself lessens the area of iris--lens canal. Other variables exist and interact to determine the iris contour, including eye size, especially the dimensions of the anterior segment[78], lens size and position, iris stroma and iris musculature characteristics[35], ciliary body anatomy, and physiologic parameters including aqueous humor flow rate, facility of outflow, vitreous--aqueous fluid flow, and the effects of accommodation and blinking. These potentially significant dimensional and physiologic parameters vary and may become risk factors of more or less importance in determining iris contour and the development of pupillary-block in

eyes which become at risk for angle closure. It should be clear that the lens plays a pivotal role in the pathogenesis of angle closure secondary to the pupillary block mechanism. If an iridotomy or iridectomy is performed the pressure differential between the AC and PC becomes minimal and iris deformation secondary to pupillary-block is relieved. If the lens is removed and posterior synechiae lysed, the prerequisite anatomical relationship for irislenticular (pupillary) block is eliminated, and the anterior chamber angle will widen as the iris leaf becomes flat and rotates to a more posterior position.

B. ANGLE CROWDING

This mechanism for angleclosure may exist alone, but more often co-exists with pupillary-block. Angle crowding can be thought of as the sandwiching of the peripheral iris between the trabecular meshwork and some other structure, compared to the pupillary-block related anterior iris shift secondary to the pressure differential between the anterior and posterior chamber. The clinical primary condition possessing this mechanism is the plateau iris configuration[132]. In this condition, anteriorly positioned

ciliary processes prop up the iris anteriorly resulting in the peripheral iris being held forward in approximation with the trabecular meshwork[103]. Depending on the amount of trabecular obstruction that develops, acute or chronic angle closure can occur. Indentation gonioscopy reveals a characteristic double iris hump, and ultrasound biomicroscopy (UBM) of the ciliary body reveals the anterior position of the ciliary processes filling the ciliary sulcus[88,133]. Gonioscopy after an iridotomy (LPI) will reveal persistence of the narrow and occludable peripheral angle. This helps explain why creating a patent LPI to relieve the pupillary block component in the presence of this cause of angle crowding

may not prevent progression to ACG. In plateau iris configuration, removal of the lens with intraocular lens implantation increases ACD but does not change iridociliary apposition. This explains why the elimination of iris support by the lens also does not cause the angle to significantly widen[126]. Age-related lens changes and other ocular conditions associated with a forward shift of the anterior lens plane and decreased ACD can also cause significant angle-crowding, as seen clinically in nanophthalmos, microphthalmia, retinopathy of prematurity (ROP), spherophakia, and axial anterior ectopia lentis.

V. Pathogenesis of Angle-closure Glaucoma and the Role of the Lens

Eyes with primary angle closure have significant anatomic differences from normal eyes[13,75]. The most significant clinical hallmarks of an eye with angle-closure are the shallow AC and narrow angle. The mean anterior chamber depth (ACD) in PAC eyes is approximately 1.8 mm, which is 1 mm shorter than in normal eyes[75,125]. Angle closure becomes a rarity when anterior chamber depth exceeds 2.5 mm[76]. Decreased AC volume[68,79], small corneal diameter[16,124], and short axial lengths[16,124], are all characteristic of eyes with PACG. The most satisfactory explanations for the more shallow AC is the age related increase in lens thickness and more anterior position of the lens[75,76,90]. The axial lens thickness is greater than in normal subjects[16,75,76], and the thicker lenses are significantly more anteriorly positioned than in normal eyes[76,116]. Lowe[75,76], estimated that increased lens thickness causes 0.35 mm of AC shallowing, and forward lens position causes 0.65 mm of shallowing, accounting for the total of 1 mm difference in AC depth of the smaller eye compared to the normal eye. Growth of the lens, with an increase in the number of lens fibers continuing throughout adult life, results in an increase in lens thickness and anterior curvature[68]. Ocular biometry of Alaskan Eskimos, an ethnic group at high risk for PAC, confirmed the presence of decreased anterior chamber angle width, depth, and axial length associated with increased hyperopia and lens thickness[139]. The age-adjusted angle width and ACD were significantly less than other ethnic groups. When the biometry of contralateral eyes of patients having an APAC were studied and compared to population-based controls, unfavorable dimensions were found consisting of more shallow anterior chambers and narrow angles, and thicker lenses. These differences were considered to explain in part the estimated 50% risk for APAC in these eyes[24]. These observations also explain the tendency of PAC to affect older patients and its relative rarity in young adults. Decreased ACD is accelerated in women between the fourth and fifth decades, which may explain their greater propensity for PAC[71]. Biometry and clinical examination of PAC patients identifies anatomic risk factors for angle closure and supports the pivotal role of the lens position and size in the active or potential mechanism of closure[22]. These assessments, however, have not conclusively explained its more

frequent occurrence in the eyes of certain ethnic groups (Eskimos and Chinese) with dimensions similar to eyes in other populations (whites and blacks) with a less frequent rate of angle closure[13,139]. Lens disorders of position and size as seen with lens dislocation and spherophakia can also result in pupillary-block and secondary angle-closure. Unlike PAC, which usually afflicts older patients aged 50 years and above, lens-related secondary angle closure occurs in patients of all ages. Shallowing of the anterior chamber occurs from increased thickness and curvature of the lens and/or forward shifting of the dislocated lens in the pupil to cause crowding of the angle and potential pupillary block. With an intumescent age-related cataract or with lens swelling after a perforating lens injury, the sheer increased thickness and bulk of the lens can push the peripheral iris (angle crowding) against the trabecular meshwork. As with the plateau-iris syndrome, lens-induced angle crowding can lead to acute or chronic angle closure despite a patent LPI[102]. Argon laser peripheral iridoplasty (ALPI) has been used to manage acute phacomorphic angle-closure with favorable results[123,145]. Extraction of the abnormal lens in these conditions is ultimately the only way to definitively eliminate both the angle-crowding and co-existing pupillary block.

VI. Current Surgical Treatment Options for Primary Angle-closure Glaucoma Understanding and caring for patients with APAC requires repetitive careful clinical ocular examinations including evaluation of the filtration angle to determine the mechanism of the angle-closure and the active stage of the disease. The treatment of a patient with acute disease should be followed by care to prevent the development or worsening of chronic angle closure glaucoma. In a patient with established synechial angle closure and advanced glaucomatous optic neuropathy (GON), active management of the IOP is essential. Appropriate surgical decisions for angle closure should be congruous with the patient's anatomic defects, offending pathophysiology, and the stage of disease. Randomized clinical trials are lacking to support the efficacy of procedures for primary angle closure glaucoma.

A. LASER PERIPHERAL IRIDOTOMY

Laser peripheral iridotomy (LPI) eliminates the pressure difference between the anterior and posterior chambers and is the current standard treatment to correct pupillary block. It is also a safe and effective prophylaxis in suspect eyes with occludable angles secondary to pupillary block, including fellow eyes of APAC patients at risk for bilateral angle closure[5]. Although LPI is an effective treatment for APAC, with resultant widening of the filtration angle and reduction of elevated IOP, it is not reliably protective against chronic angle closure[6,84,105]. In a study of the benefit of LPI in Asian eyes, 100% had resolution of the acute attack after the LPI, but 58.1% subsequently developed elevated IOP requiring treatment and 32.7% eventually needed trabeculectomy for pressure control[6]. The median time interval for increase in IOP was 5.8 months, and most of these eyes which developed CACG had more than 180 degrees of peripheral anterior synechiae (PAS). The failure of LPI to prevent recurrent elevation of the IOP has been correlated with the amount of PAS present in these eyes[51,84,143].

B. ARGON LASER PERIHERAL IRIDOPLASTY

ALPI is a procedure to induce immediate focal iris stromal contraction to pull the iris root away from the angle wall thus widening it. In the long term, it produces thinning and contraction of the peripheral iris, giving it a flatter contour. Histopathology suggests that heat shrinkage of collagen may account for its short-term effect and contraction of the induced fibroblastic membrane may be responsible for its long term effect[1]. Hence, it is used to reduce angle crowding in nanophthalmos[52], PAC[60], lens-induced angle closure[100], and reported to be very effective in the plateau iris configuration by eliminating and reducing the amount of residual appositional angle closure after LPI[99]. In the initial treatment of APAC it can assist to lower the IOP by pulling open the angle, and it can be useful also when initial medical treatment fails[101]. A study of 10 APAC patients treated with ALPI after administration of pilocarpine and timolol showed a remarkable decrease in IOP from 59.5 mm Hg to 21.7 mm Hg at 30 minutes and 16 mm Hg at 1 hour post-ALPI[65]. A follow-up study was done to confirm the effect was predominantly from the ALPI by managing eight APAC patients with ALPI alone and produced similar results[122]. In 2000, a randomized controlled interventional trial comparing ALPI against conventional systemic IOP-lowering medications in the first-line treatment of APAC showed the ALPI treated group had lower IOP than the medically treated group at 15 minutes, 30 minutes, and 1 hour after the start of treatment. The differences were statistically significant. The differences in IOP became

statistically insignificant from 2 hours onwards[64]. In chronic PACG, a study with a followup period of 6 months suggested that in eyes which had failed medical therapy, the effect of iridoplasty may not be sustained in some eyes even though at least half the angle was opened at the time of the procedure[11]. In another report on the short-term benefit of iridoplasty after 180 degrees goniosynechialysis in 5 eyes with chronic PAC and total synechial angle closure, satisfactory IOP was achieved in 80% (4 eyes), with a mean follow-up period of 7.6 months[61]. Re-closure of the angle occurred in the remaining eye with IOP elevation. There is a paucity of data on the long-term effect of iridoplasty in PAC. ALPI alters the peripheral configuration permanently and may help prevent subsequent angle closure from peripheral anterior synechiae formation.

C. ANTERIOR CHAMBER PARACENTESIS

Immediate anterior chamber paracentesis is a maneuver that rapidly lowers IOP in APAC

providing instantaneous relief of symptoms and prevention of further optic nerve and trabecular meshwork damage secondary to the acutely elevated IOP. The IOP-lowering benefit of a paracentesis may decrease by 1 hour after the procedure and thus anti glaucoma medications will become necessary to sustain the IOP control[63]. Paracentesis may not break the pupillary block but can allow the LPI to be performed sooner. Possible complications include excessive shallowing of the anterior chamber, lens trauma, choroidal effusion, and hemorrhage due to the sudden decompression[63].

D. GONIOSYNECHIALYSIS

When extensive synechial angle-closure has occurred, goniosynechialysis (GSL) is an option for removal of PAS of recent onset; long-standing PAS are likely to be associated with permanent trabecular damage[10,59]. On its own, the long-term success of GSL for PAC is unlikely because the procedure does not address the underlying cause for the synechial angle closure, be it pupillary-blockor angle-crowding. Hence, goniosynechialysis is often performed with other procedures such as LPI[120], ALPI61,119 or lens extraction [62,121] to open the angle in eyes with minimal to moderate neuronal damage. It can also be complicated by hyphema, fibrinous inflammation, and synechial re-closure of the angle[120].

E. TRABECULECTOMY

After the onset of permanent trabecular dysfunction, the surgical management of PACG is in principal option like POAG[59]. In medically unresponsive APAC, trabeculectomy is associated with high risk of post-operative complications such as shallow anterior chamber and surgical failure[7]. Trabeculectomy in chronic PACG is also associated with higher risk of failure, postoperative anterior chamber shallowing, malignant glaucoma, and a significant rate of cataract formation [1,7,41,45,127] compared to POAG. Even when the filtration surgery has successfully reduced the IOP, the ailing trabecular meshwork does not regain its function, and so the disease is not cured. The eye must be followed for life to ensure that the aqueous outflow remains adequate and the IOP sufficiently controlled to prevent further progression of the glaucomatous optic neuropathy.

F. LENSECTOMY

Lensectomy in the treatment of PACG has been actively studied and reported in recent years[30,34,39]. Cataract surgery in PACG is generally more challenging and complicated than in normal eyes or eyes with POAG because of the shallow AC, large cataractous lens, and a sometimes atonic pupil after an acute angle closure attack. The phacoemulsification procedure offers the advantages of a smaller self-sealing incision, better maintenance of the AC intraoperatively with less risk of iris prolapse, less iris manipulation, better intraocular maneuver when the pupil is small, faster postoperative visual rehabilitation, and better preservation of the superior conjunctiva for future trabeculectomy if needed.

A. BIOMETRY FOLLOWING LENSECTOMY

Hayashi has shown that after phacoemulsification and PCIOL implantation, the ACD and angle width in ACG eyes approximates that of POAG eyes and control eyes without glaucoma, even though differences of 1.0 mm of anterior chamber depth and 10 degrees of angle width existed preoperatively[34]. They thought that these changes contributed to the significant IOP reduction seen in the postoperative follow-up period of 12 months. Another study[144], done in chronic PACG eyes, also showed a mean increase in AC depth, from 2.04 mm to 3.44 mm, after cataract extraction and IOL implantation followed by control of IOP postoperatively. This increase in ACD and angle width was created by the exchange of the thickened lens (5 mm) for a 1-mm acrylic or polymethylmethacrylate lens[111].

B. CLINICAL STUDIES AND IOP CONTROL AFTER LENSECTOMY

In Table-1, 22 clinical studies, reported from 1988 to 2007, are summarized, which describe the results of lensectomy in PAC suspects, PAC, and PACG patients as well as control groups, who were selected in a nonrandom manner. In the early studies[2,28,30,138], extracapsular cataract extraction (ECCE) was performed with posterior chamber IOL implant (PCIOL). The cataract extractions were made through a corneal incision, except in Acton's study[2], where some were performed via a limbal corneoscleral incision after a fornix-based conjunctival incision. In most of these studies, the patients included had visually significant cataracts in addition to PACG of different severity, chronicity, amount of PAS, and various extents of IOP control with medications, previous filtration surgeries, or laser procedures. A high proportion (65%) of these patients were considered successes post-cataract extraction with normal IOP without medications, whereas preoperatively glaucoma medications were needed. In the studies by Gunning and Greve[29], Roberts[104], and Jacobi[44], clear lens extractions were performed mainly for glaucoma control. High preoperative IOPs, in these cases of uncontrolled glaucoma, were followed by high rates (67-72%) of IOP control without medications postoperatively, even though the patients in the different studies were variable in terms of the stage of their disease, chronicity, and amount of PAS present preoperatively. The decision to do clear lens extraction for angle closure is even more controversial considering that patients' vision often improves when the corneal edema and inflammation settles. In one study, more than half of test subjects recovered good vision (6/12 or better) within a few days of an acute angle closure attack[118]. The greatest IOP reduction after lens removal occurred in

TABLE 1 : Summary of Clinical Studies: Lens Extraction for Primary Angle-Closure Glaucoma

Study (year)	Lens Procedure	Glaucoma Type (# of eyes)	Preop Gonioscopy	Follow - up (months)	Preop/Postop IOP (mm Hg)	Success % IOP <22	Qualified Success % IOP <22 on Rx	Complications
Greve 28 (1988)	ECCE +PCIOL	AACG (5) CACG (14)	Near or complete closure	Range 6--42	31/16	76%	24%	Early IOP spikes
Wishart 138 (1989)	ECCE +PCIOL	CACG (23) POAG (21)	PAS (8) Open (21)	Mean 11.2	19.1/15.6 19.4/19.6	65% 5%	35% 95%	9% IOP spikes 14% IOP spikes
Gunning 30 (1991)	ECCE +PCIOL	CACG (41) PAC AACG (18) suspects (8)	No data	Mean 14.3	22.6/15.6	65%	34%	60% IOP spikes
Yang 144 (1997)	ECCE +PCIOL	CACG (20) Control (10)	No data	Mean 53 Mean 59	Controlled 17 Controlled 15	0% No Data	100%	IOP spikes; iritis
Acton 2 (1997)	ECCE +PCIOL	AACG (9) CACG (10)	Mean PAS	Mean 19	17/ 15.6	68%	26%	32% IOP spikes 16% iritis
Gunning 29 (1998)	ECCE +PCIOL vs Trabeculectomy	CACG (22) CACG (25)	PAS 77%eyes	Mean 53 Mean 59	28/17 29/ 15	68%	68%	45% IOP spikes 48% shallow ACD 36% CD
Teekhasaenee121 (1999)	PHACO +PCIOL	CACG (52)	PAS mean 310 _ to 60_ Postop	Mean 21	30/ 13	90%	8%	20% Plasmoid iritis
Roberts104 (2000	PHACO +PCIOL	AACG (3)	360_ PAS (2)	36, 24 mos, and no data	39/ 17	67%	33%	10% IOP spikes No data
Ge26 (2000)	PHACO +PCIOL	AACG (18) CACG (14)	Narrow angles	Mean 9	23/12	84%	16%	Early IOP spike
Lai 62 (2001)	PHACO + PCIOL+GSL+ DLPI	CACG (7)	360_ PAS (7)	Mean 9	33/ 13	100%	---	28% IOP spikes hyphema
Hayashi 33 (2001)	PHACO +PCIOL	CACG (68) POAG (68)	No data	Mean 25	21/ 15	41% 19%	51% 53%	No data
Jacobi44 (2002)	PHACO +PCIOL CSI	AACG (43) AACG (32)	Partial closure (7)	Mean 10	41/18 40/20	72% 35%	16% 31%	9% IOP spikes 21% IOP spikes 65% additional surgery
Staso117 (2002)	PHACO +PCIOL	CACG (12) Control (12)	Angle width 19 _	9	21/15 16/16	100%	100%	No data
Zhi 148 (2003)	PHACO +PCIOL	AACG (18)	No data	7 days	48/ 13	No Data	No Data	No data
Yoon 147 (2003)	PHACO +PCIOL	AACG(12)	No data	Mean 6	50/12	70%	30%	20% IOP spikes

acute PAC eyes with uncontrolled IOP preoperatively[44,104,120]. This is expected as this group of patients would have the greatest amount of pupillary block and appositional angle closure, as well as the highest baseline IOP. IOP reduction occurred to an extent that medications were not required postoperatively, even in eyes with extensive PAS preoperatively[28,121,138]. Many eyes were found to have less PAS after surgery[2], suggesting that gonioscopic examination preoperatively may overestimate the extent of PAS, when the angle is narrow. Another possible explanation for finding less PAS postoperatively, when no additional maneuver was done during the lens extraction to open the angle, is the positive pressure of viscoelastic material and fluid as it is introduced into the eye during the surgery[58]. Although gonioscopic details of those who failed to have IOP control were not described, we suspect that a residual open angle does not guarantee successful IOP control. Acton et al's failure to achieve IOP control in some subjects occurred even though only eyes with a maximum of two quadrants of PAS were included in the study[1]. The residual open trabecular meshwork can be potentially damaged by high IOP, inflammation associated with an acute attack, or by the adverse effect of appositional closure. Phacoemulsification and PCIOL combined with GSL, performed within 6 months of chronic ACG with an acute attack despite LPI and argon laser peripheral iridoplasty, has been found to control IOP without medications in 90.4% of eyes[121]. Another study combining phacoemulsification and PCIOL with limited (inferior) GSL and diode laser peripheral iridoplasty in the treatment of seven eyes with cataract and CACG with total synechial angle closure found a short-term success rate of 100% during a mean follow-up period of 8.9 months[62]. The study by Gunning and Greve [29]compared the results of lens extraction to trabeculectomy without antimetabolites, except for use of postoperative 5-fluorouracil in 6 of 25 eyes following surgery. Longterm IOP control was found to be better in the trabeculectomy group, but there were also moresight-threatening complications such as hypotony and central field loss, poorer visual outcome, and more surgical reintervention in this group. Sixty percent of the trabeculectomy eyes required cataract extraction after a mean postoperative period of 32 months. When phacoemulsification was compared to conventional surgical iridectomy (CSI) in the study by Jacobi, phacoemulsification was found to have more than doubled the success rate in terms of IOP control and much lower rate of surgical reintervention than the CSI group[44]. Imaizumi subdivided his study groups into: 1) APAC at first visit without prior treatment, 2) PACG with earlier laser iridotomy (LI), and 3) a control group. His results show that the post-cataract surgery IOPs of group 1 are significantly lower than the preoperative IOP of group 2 even with glaucoma medications[40]. This suggests that lens extraction lowers IOP as well as, if not better, than LPI. Lens extraction in PAC, whether clear or cataractous, not only deepens the AC and opens the angle but also decreases diurnal IOP variation[74] and improves facility of outflow[81]. This IOP reduction following cataract surgery is not observed to the same extent in POAG eyes. Some studies have found that IOP control in POAG eyes are largely unaffected by cataract surgery[80,138], whereas others found a reduction in IOP in the short term that is smaller [104] and less sustained[87,104] than that seen in PACG eyes. The mechanism for IOP reduction in POAG eyes is unclear.

C. COMPLICATIONS OF LENSECTOMY

Intraocular surgery in patients with angle closure is more challenging than regular surgery because of the shallow AC, atonic pupil from the acute attack, and residual corneal edema. The reviewed studies (Table-1) of lensectomy for treatment of angle closure glaucoma report lensectomy, by either ECCE or PHACO, to be potentially safe in the hands of a skilled cataract surgeon. The complication causing the most frequent concern was the immediate postoperative pressure spike, which occurred in 9--60% of eyes. Significant postoperative inflammation was seen in 16-40% of eyes reported in at least four studies[2,121,144,147]. The addition of GSL may be associated with increased rates of hyphema, fibrinoid anterior chamber reaction, IOP spikes, and cystoid macular edema postoperatively, secondary to the iris manipulation[121]. Kubota described the successful addition of GSL to PHACO and PCIOL, in the presence of PAS of more than two quadrants, and reported no significant complications in 5 eyes[55]. Endothelial cell damage is common after acute angle closure and elevated IOP. One study shows that endothelial cell counts are not significantly diminished following lens surgery compared to preoperatively[40].

VIII. Lens Extraction for Secondary Angle-closure Glaucoma

The lens plays a significant role in the development of angle closure in other important eye conditions, which

occur less frequently than PACG but in patients of all ages[96]. It is of value to appreciate that the same mechanisms of pupillary block and angle crowding, as seen in patients with PACG, also occur in these other conditions. Angle closure develops in these conditions when the lens is disproportionately large, when the eye is abnormally small, when the lens is thickened, or when the lens becomes subluxated and blocks the flow of AH through the pupil and/or closes the narrowed angle by crowding. Treatment choices should be determined by the underlying pathology and can include lensectomy when irreversible anatomic abnormalities exist that are unresponsive to medical therapy, laser iridoplasty or iridotomy, or surgical iridectomy.

A. Nanophthalmos

Nanophthalmos is a rare, bilateral, sporadic or familial condition characterized by small eyes, with adult axial lengths of less than 20 mm, shallow anterior chambers with narrow angles and convex irides, small corneal diameters, high hyperopia, thick sclera and choroid, high lens/eye volume ratio, high corneal refractive power, absence of other congenital malformations, and the frequent occurrence of angle closure[114]. The mechanism of the angle-closure in nanophthalmos relates to the angle-crowding secondary to the normal-sized lens in a small eye and anterior segment. This leads to progressive PAS formation as the iris is forced anteriorly against the trabecular meshwork by the worsening pupillary-block and expansion in the posterior segment as the lens enlarges with age. The mechanism explains the rarity of long term success in IOP control with LPI alone, and the beneficial effect of combined LPI and ALPI in some eyes[8]. The thickened sclera is felt to impede venous drainage from the choroid and is

responsible for the uveal effusion that can be corrected by therapeutic or prophylactic sclerossclerostomy procedures[46]. The potential role for lensectomy in the management of nanophthalmos is shadowed by an awareness of vision loss and complications after intraocular surgery in the past[9,86]. The potential goals of lensectomy are for cataract removal, to deepen the AC, and widen the angle to halt and prevent progressive closure.

Successful cataract removal with phacoemulsification and PCIOL implantation in nanophthalmos is now reported[77,142]. If medical therapy, LPI, ALPI, combined with therapeutic sclerostomy procedures for uveal effusions fail to halt progressive angle closure[53], lensectomy with PCIOL implantation with prophylactic sclerostomy procedures should be considered.

B. Retinopathy of Prematurity

Retinopathy of prematurity (ROP) occurs in premature infants with the risk of occurrence inversely related to the birth weight, reaching 90% for infants less than 750 g. Affected eyes may be small depending on the stage of the active disease[67], possess steep corneas[36], shallow anterior chamber depths[36], narrow angles[32], proportionately large lenses, and exhibit progressive lenticular myopia[27]. Secondary angle closure associated with anterior displacement of the iris-lens diaphragm is common (30%) in advanced ROP with onset in infancy. Closure of the angle is secondary to angle crowding, a belief supported by the clinical failure of iridectomy only to open the angle and improve IOP control[91,131]. The angle-closure may be of acute onset or occur more slowly and cause CACG[82]. Progressive growth and forward movement of the lens secondary to contraction of posterior retrolenticular membranes[96] are also felt to contribute to the development of pupillary-block related angle closure[82,91,128,131]. Posterior diversion of AH (ciliary block glaucoma) and neovascular glaucoma also have been seen in ROP glaucoma patients[17,56,82]. In the study by Hartnett and coworkers of 26 untreated stage IV or V ROP eyes in 17 children between 4 and 35 months, 65% had open angles, 35% had 90_ or more of closure with associated glaucoma in only 3 (12%) eyes[32]. The treatment of angle closure associated with ROP depends on the clinical indication and stage of the illness. When glaucoma is acquired later in childhood and in adults, active therapy to treat the pupillary block with iridectomy[69], LPI[115], lensectomy[82,91], and with glaucoma procedures [82] have been successful. PHACO with PCIOL has been performed in adults with ROP resulting in improved vision and IOP control in glaucoma patients[54].

C. Phacomorphic Glaucoma

In phacomorphic glaucoma, angle-closure and secondary glaucoma is caused by the enlargement of the swollen lens as seen after lens trauma and more frequently in the intumescent age-related cataract. The need and benefit of lens removal for improvement of vision and correction of the pupillary block and crowding mechanisms for angle-closure is obvious.

D. Ciliochoroidal Expansion Syndromes

Ciliochoroidal expansion places even an eye of normal size at risk for angle-closure glaucoma. In the average human eye the vitreous volume is 5,000 ml, the choroidal volume is 480 ml, and the anterior chamber volume is about 150 ml[94]. Hence, it can be appreciated that minimal sustained expansion of the choroid can decrease the AC volume and ACD significantly and create the prerequisite anterior segment configuration for angle closure. Furthermore, in these syndromes, separation and thickening of the ciliary body has been documented by UBM[130]. The related anterior rotation of the ciliary body and relaxation of the zonules would be expected to contribute further to the development of the angle closure. Ciliochoroidal expansion syndromes occur secondary to inflammation, choroidal venous congestion, choroidal metastases, trauma, drugs, systemic diseases, from aggressive Panretinal Photocogulation (PRP), or may be idiopathic[23,49,130]. Treatment is directed to the underlying illness and to management of the related angle closure. Lensectomy is rarely indicated because often these conditions can be treated medically or can resolve spontaneously.

E. Lens Subluxation Glaucoma

Eyes with lens subluxation or dislocation are at increased risk for secondary glaucoma[42]. When the lenticular zonular attachments weaken or detach, the lens thickens and may move from its normal position. If the weakened zonules are confined to less than half of the lens circumference, the thickening of the lens will occur asymmetrically and create refractive astigmatism and the lens will remain approximately in its normal position. This circumstance is seen in young patients with Marfan syndrome whose eyes are rarely complicated by lens related glaucoma[43]. In older patients with Marfan disease, or in those conditions where circumferential weakening of the zonules occurs[70,136], the lens becomes mobile and moves anteriorly with resultant shallowing of the anterior chamber, increased iris convexity, increased pupillary block, and risk for acute or chronic ACG[137]. The subluxated or dislocated lens may enter the anterior chamber or acutely obstruct the pupil and cause an acute attack associated with a flat anterior chamber. Treatment of glaucoma caused by lens subluxation is best directed to the lens itself. Surgical iridectomy or LPI can help by decreasing the iris convexity and increasing the angle width, and will prevent the occurrence of acute angle closure but can not be relied on to definitively treat or prevent this secondary ACG[47,136]. These measures can be considered for prophylaxis for selected young patients[141]. Angle-crowding is an important component causing the angle closure and can be marginally benefited by ALPI[98]. The definitive treatment for lens subluxation related glaucoma is lensectomy[42,47,137]. The indication for implantation of anterior chamber IOL or PCIOL is controversial[47,112,129,137]. The addition of successful GSL combined with lensectomy for the treatment of this PAC has been reported[47], and deserves continued clinical study.

IX. Conclusion

PACG is a leading cause of blindness and is potentially preventable. It is projected that 15.7 million will have ACG in 2010 and 3.9 million will be bilaterally blind from it[92,]. The lens plays an essential and pivotal role in the pathogenesis of primary and secondary ACG. Clinical studies suggest that lensectomy and PCIOL implantation for ACG patients may offer successful IOP control, and maintenance of improved vision. Lensectomy eliminates pupillary block, widens the angle to lessen angle crowding thus reducing the irido-trabecular proximity. Medical management and LPI remain the most common modes of treatment of an acute attack but newer approaches including early lens removal are gaining popularity because of their potential long-term success in IOP control. After an acute attack lensectomy is most appropriate or it should be combined with a filtration procedure. Randomized, controlled studies are ongoing in Hong Kong and Singapore and should help clarify this surgical decision[66,83]. Although lens extraction for angle closure is biologically plausible, as of this time, there is no evidence from good quality randomized trials or non-randomized studies of the effectiveness of lens extraction for CACG[25]. More longitudinal biomorphometrical studies of PACG eyes treated with and without lensectomy are needed to determine its role in the prevention of progressive angle closure and to determine which patients will benefit from lens extraction[37].

X. Method of Literature Search

A search of the PubMed database was conducted for the years 1988-2007, using the following key words: angle closure glaucoma, pupillary block, angle

crowding, lensectomy, cataract extraction. Additional references were recovered from bibliographies of the references.

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Bilateral Acute Angle Closure from Topiramate toxicity– 3 case reports

Islam MN[1]

Abstract

Purpose : To report 3 cases of topiramate-induced bilateral acute angle closure .

Methods : Three case report of otherwise healthy womanwho developed bilateral acute angle closure with decreased vision,choroidal thickening, bilateral suprachoroidal effusions,and induced myopia after taking topiramate group of drugs.

After confirmation of diagnosis, treatment was initiated with high-dose oral steroid, intravenous mannitol, topical difluprednate ophthalmicemulsion, atropine sulfate, Brinzolamide and the combination ofbrimonidine and timolol. Patient were followed after 1 day, 7 days and after 3 weeks. At the 3-week follow-up visit, vision, IOP and angle anatomy returned to normaland there was resolution of choroidal and ciliary body effusions onultrasound biomicroscopy.

Conclusions : Bilateral acute angle closure may develop after ingestion of topiramate for the treatment of migraine. Early diagnosis and treatment returns the vision completely

Key Words : topiramate, angle closure, angle closure glaucoma

Introduction

Topiramate, a sulfamate-substituted monosaccharide (Etopira, Topirva, Topmate) primarily used in the management of migraine, Seizure disorders and bipolar disease andmay produce acute bilateral angleclosure.[1–10]

The mechanism consists of ciliochoroidal effusion with anterior rotation of the ciliary body anddisplacement of the lens-iris diaphragm.[11] Topiramate mayalso disrupt the blood-brain barrier, leading to increased protein content in cerebrospinal fluid and simultaneous blood ocular barrier breakdown, suggesting a common inflammatory mechanism.[12] Initial treatment consisted of discontinuing topiramate and administering ocular hypotensive medications.

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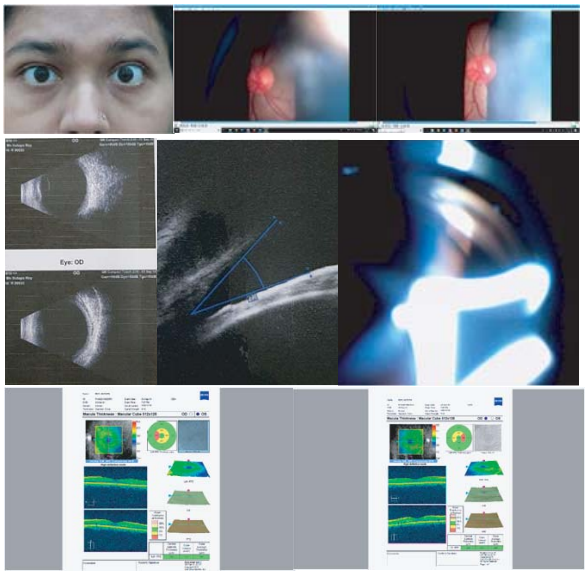
Use of topical cycloplegia, systemichigh-dose steroids, mannitol, peripheral iridotomy, andchoroidal drainage have been described.[13–16]

In this article three case reports of topiramate induced bilateral acute angle closure and their treatment will be described.

Case report - 1

A 25year-old woman reffered from a medical college hospital with complaints of acute bilateral vision loss and eye pain. The past medical history and review of systems were unremarkable. The ocular history was unremarkable and she was previously emmetropic. Her medications included diabetic and migraine related medication containing topiramate 50 mg, which had been started 1week prior, as prescribed by her endocrinologist. On examination, the height was 163cm and weight was 62.1 kg (body mass index 23.4). Initial examination revealed vision of finger counting at 1 footin the right eye and 6/60 in the left eye, which corrected to 6/9 B/E with a correction of -3.50D B/E. Pupils were 5 mm, round and minimally reactive B/E, with no afferent pupillary defect. Intraocular pressures (IOP) with Goldmann applanation tonometry were 48 mm Hg in the right eye and 50 mm Hg in the left eye. Anterior segment examination was remarkable for mild conjunctival chemosis and markedly shallow anterior chambers B/E. There was no significant corneal edema. On gonioscopy there was 360 degrees of angle closure B/E (Shaffer Grade 0). Fundoscopic examination demonstrated subtle retinal folds. Anterior segment optical coherence tomography (OCT) and Pentacam Scheimpflug Imaging revealed markedly shallow anterior chambers and narrow anterior chamber angles. Ultrasound biomicroscopy confirmed ciliary body edema and choroidal effusions B/E were visualized on Bscan ultrasonography. Enhanced depth imaging OCT showed a markedly thickened choroid and distortion of the innerretinal contour due to the suprachoroidal effusion and choroidal. Slit-lamp

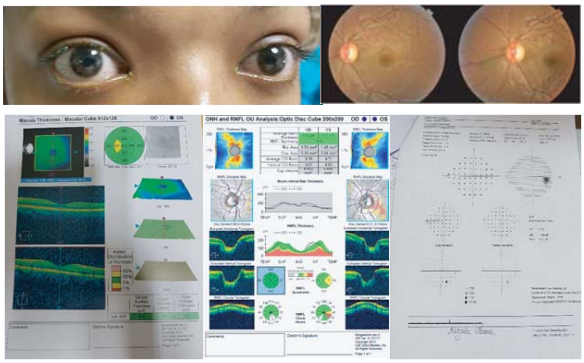
photograph at presentation showing a shallow anterior chamber in the left eye, markedly so peripheralto the slit photograph.



Case report – 2

A medical student, 22-year-old healthy girl with a history of migraine presented with severe pain in both eyes with headache and vomiting. She gave history of taking oral topiramate 50 mg twice daily 10 days before. She was already treated by two different ophthalmologist and diagnosed as Primary Acute Angle Closure Glaucoma. Her primary IOP was 48 and 53 mmHg in right and left eye respectively and Vision was counting finger in both the eyes. She was given maximum anti glaucoma therapy. She was also advised for YAG LPI in her both eyes. On examination after 10 days I found her visual acuity improved to 6/36 R/E and 6/60 in L/E with myopic correction. Intraocular pressures (IOPs) were 24 mm Hg R/E and 28 mm Hg L/E. Slit-lamp examination showed mild conjunctival hyperemia, mild corneal edema, shallowed anterior chamber, slightly reactive pupils in both eyes. Gonioscopy revealed angle closure stage 0but with indentation grade 1 for 360, domed iris convexity, and prominent ciliary processes visible through the lens, suggestive of anterior displacement of the iris-lens diaphragm in both eye. The optic discs was normal. The C:D was 0.45 in both eyes. B-scan ultrasonography and ultrasound biomicroscopy showed circumferential ciliochoroidal effusions, which induced the anterior displacement of the iris-lens diaphragm seen gonioscopically. Anterir Segment optical coherence tomography revealed a shallow anterior chamber and 360-degree iridocorneal contact in B/E

The topiramate was discontinued and oral acetazolamide, Steroid and topical Beta blocker and Brimonidine were given. On 2nd day, her IOPs were >20 butiridocorneal contact persisted. With continuous treatment after 3 days IOP came to normal range <20 mmHg in both eyes. Repeat AS OCT confirmed the opening of angle B/E with persistent shallow anterior chamber. The visual acuity 1 week later was 6/9 R/E, 6/12 L/E and IOP was 10mm Hg B/E. She had clear corneas, deep anterior chambers and complete resolution of the choroidal effusions, induced myopia resolved and refraction became normal for distance vision.



Case report - 3

A 52-year-old healthy woman with a history of migraine presented with bilateral acute onset of pain, blurred vision and redeye. She gave history of taking oral topiramate 50mg twice daily 7days before presentation. Her visual acuity was 6/36 R/E and CF 4 ftin L/E. Intraocular pressures (IOPs) were 34 mm Hg R/E and 48 mm Hg L/E. Slit-lamp examination showed conjunctival injection, mild corneal edema, markedly shallow anterior chamber, and mid-dilated slightly reactive pupilsin both eyes. Gonioscopy revealed angle closure stage 0 for 360 and prominent ciliary processes visible through the lens, suggestive of anterior displacement of the iris-lens diaphragm in both eye. The optic discs and maculas were unremarkable. B-scan ultrasonography and ultrasound biomicroscopy showed circumferential ciliochoroidal effusions, which induced the anterior displacement of the iris-lens diaphragmseen gonioscopically. Anterir Segment optical coherence tomography revealed a shallow anterior chamber and 360-degree iridocorneal

contact in B/E

The topiramate was discontinued and oral acetazolamide, Steroid and topical Beta blocker and Brimonidine were given. On 2nd day, her IOPs were in the 30s but iridocorneal contact persisted. With continuous treatment after 3 days IOP came to normal range <20 mmHg in both eyes. Repeat ASOCT confirmed the opening of angle B/E with persistent shallow anterior chamber. The visual acuity 1 week later was 6/12 R/E, 6/18L/E and IOP was 10mm Hg B/E. She had clear corneas, deep anterior chambers and almost complete resolution of the choroidal effusions. There was very mild induced myopia and with -.25 Sph power refraction became normal for distance vision and presbyopic plus power now needed for near vision.

Discussion

Almost 3% of all patients taking topiramate may have acute angle closure, myopic shift, idiosyncratic ocular adverse reactions such as ciliochoroidal detachment, ciliary body edema, anterior displacement of the lens-iris diaphragm, increased lens thickening etc.

Other less frequent reactions include periorbital edema, nystagmus, diplopia, and scleritis. Bilateral blurred vision is frequently the presenting symptom and in 85% of the cases IOP elevation occurs within 1- 2 weeks of starting topiramate. Doubling the recommended dose has caused IOP elevations within hours.

Treatment consists of discontinuing topiramate and lowering IOP. Topical miotics exacerbate the condition and peripheral laser or surgical iridotomy are usually not helpful, as pupillary block is absent. Choroidal drainage has been reported effective in normalizing IOP and deepening the anterior chamber, but this is invasive and supraciliary effusions can recur. It is better to try to open the angle to provide time for the effusion to resorb and the anterior rotation of the lens-iris diaphragm to resolve spontaneously.

Banta et al1 first reported a case of uveal effusion andsecondary angle closure associated with topiramate usein July 2001. The clinical findings of topiramate-associated angle closure have been well described. This report aimsto alert clinicians regarding the possibility of topiramate induced bilateral angle closure developing secondary tothe use of topiramate

Rhee et a described a 43-year-old patient with topiramate-associated angle closure with high-frequency ultrasound evidence of ciliary process swelling and forward displacement of the lens-iris diaphragm. In a review of 86 cases of topiramate-induced angle closure, 6 the onset was reported to occur a mean of 7 days from the initiation of therapy with 85% of cases occurringwithin the first 2 weeks of treatment. Twelve subjects were treated medically with full recovery. Twenty-three patients stopped the medication and had complete. Although controversy exists regarding the exact mechanism of acute myopia and angle closure after sulfonamide use, most authors have attributed this to ciliary body swelling. Thepresence of choroidal effusion suggests an association between topiramate-induced forward rotation of the ciliary process and forward displacement of the lens-iris diaphragm that contributes to the myopic shift, anterior chamber shallowing, and resultant angle closure. Although the exact mechanism is notclear, the fluid movement in choroidal effusion is potentially related to drug-induced changes in membrane potential. This adverse event is theorized to be an idiosyncratic reaction, on the basis of similar complications seen with other sulfa-derivativedrugs. Sulfonamide-derived medications, such as topiramate, are well known to induce transient myopia throughciliary body edema, causing relaxation of the zonules, lensthickening and forward movement of the lens-iris diaphragm.

A mechanism of lens osmotic disturbance and subsequents welling causing anterior chamber shallowing was previously postulated. Ultrasound lens thickness measurements, however, demonstrated that changes in lens thickness accounted for only a small amount of the observed anterior chamber depth decrease. It is now believed that the anterior chamber shallowing is predominantly due to ciliochoroidal effusions. Our cases clearly demonstrates a massively thickened choroid on enhanced depth OCT imaging. Quigley et al proposed a mechanism of topiramate associated acute secondary angle closure and posited that a developing choroidal effusion exerted pressure on the vitreous, resulting in resistance to flow through the vitreousbody. The vitreous in turn compresses the lens-iris diaphragm, displacing it anteriorly. They suggested that fluid passing through the vitreous can

only gain access to the anterior chamber through the area between the vitreous base and the central adherence of the anterior hyaloid faceto the posterior lens. Topical cycloplegic agents help lower IOP by increasing the diffusional area for fluid to leave the vitreous cavity by retracting the ciliary processes thus widening the ciliary body diameter and posteriorly rotating the lens-iris diaphragm. As the mechanism of angle closure does not involve pupillary block, peripheral iridectomy and topical parasym-pathomimetics are not useful in the treatment of this type of secondary angle closure and could worsen the condition by causing further forward rotation of the lens-iris diaphragm as well as further breakdown of the blood aqueous barrier. In addition, we advise caution on treating patients with systemic carbonic anhydraseinhibitors, as there have been reports of acetazolamide causing an idiosyncratic reaction characterized by transientmyopia, ciliary body edema, uveal effusions, anterior rotation of the lens-iris diaphragm and acute angle-closure glaucoma. Management in refractory cases with argon laser iridoplasty has been reported. Rhee et alfirst reported the successful use of mannitoland intravenous methylprednisone to treat patients with this condition, and suggested that the response tosteroids supported the theory that ciliochoroidal effusion issecondary to an inflammatory etiology.

This case reports highlights the need for physiciansprescribing migraine and weight loss medications containing topiramate to be aware of the potentially sight-threatening adverse effect of bilateral acute angle closure. Patients starting therapy with these agents should be advised to seek ophthalmologic care immediately should they develop decreased vision or eyepain. Our report also supports that immediate withdrawl of topiramate, use of a combination of aggressive and prompt oral steroid treatment as well as topical therapy is useful for the treatment of topiramate-induced secondary acute angle closure glaucoma.

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Case Presentation: Bilateral Angle-Closure Glaucoma

S M Noman[1], N Sultana[2]

Abstract

Purpose : To present an atypical and rare case of bilateral angle closure glaucoma.

Case Report : After detailed history taking and meticulous examination, the patient was diagnosed as a case of bilateral angle closure glaucoma due to microspherophakia. After laser Iridectomy and with medication IOP was not controlled. So filtration surgery was done in both eyes to control IOP.

Conclusion : Bilateral angle closure glaucoma can occur in the microspherophakia cases. Meticulous examination with proper attention and management in time can solve the problem.

Key words : Angel closure glaucoma, iridectomy, intraocular pressure

Introduction

Angle closure glaucoma is a common problem faced in the glaucoma clinic. Microspherophakia with swollen lens complicate the case. Those cases do not respond well with antiglaucoma medications and laser iridectomy. Pupil block component and angle closure both make a complicated result.

Case History

A 35-year-old woman presented to the glaucoma clinic with complaints of ocular pain that had persisted for 1 day. She reported a sensation of pressure and blurry vision in her left eye with no apparent exacerbating factors. Her UCVA measured 6/9 OD and 6/36 OS. A slit-lamp examination of her left eye revealed moderate conjunctival injection, corneal edema, and pigmentary deposits on the corneal endothelium. The anterior chambers of both eyes were shallow centrally and flat peripherally (Figure 1), but these findings were more pronounced in her left eye.

The crystalline lenses were clear, and the IOP measured 46 mm Hg OD and 52 mm Hg OS. Both pupils were nonreactive and dilated midway, and they demonstrated posterior synechiae presumed to be from chronic iridolenticular contact. Fundoscopy revealed pink optic nerves with sharp margins, bilateral vertical cup-to-disc ratios of 0.25, intact neuroretinal rims, and retinal nerve fiber layers without para-papillary atrophy. Darkroom gonioscopy revealed a convex iris approach and intermittent peripheral anterior synechiae in both eyes for 360º.

The patient was diagnosed with bilateral acute angle-closure glaucoma (ACG) and was started on topical ocular hypotensive treatment. No systemic medications or cycloplegic agents were prescribed. When we evaluated the patient 2 days after her initial presentation to the clinic, the eye drops had adequately reduced and stabilized her IOP, and the corneal edema in her left eye had resolved. The patient subsequently underwent bilateral peripheral Iridotomies. Postoperatively, the patient's UCVA returned to 6/6 OU, and her IOP was initially controlled with topical therapy in both eyes.

Her anterior chambers remained shallow however, and she continued to demonstrate appositional closure on gonioscopy that was not relieved with compression. A subsequent fundoscopic and stereophotographic evaluation of the optic nerves showed cup-to-disc ratios of 0.4 OD and 0.7 OS. We also noted pathological changes in the retinal nerve fiber layer bilaterally (Figure 2) and an inferior notch in the left optic nerve that correlated with superior visual field defects (Figure 3) (mean deviation, -10.99 dB OD and -17.75 dB OS) on Humphrey visual field testing (Carl Zeiss Meditec, Inc., Dublin, CA).

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Figure 1. A slit-lamp photograph of the patient's right eye showed a fixed, middilated pupil (A) and shallowing of the central and peripheral anterior chamber (B).

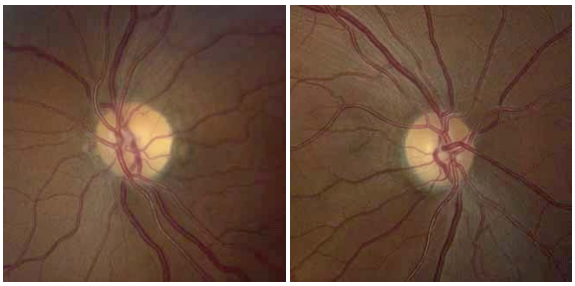

Figure 2. Fundus photographs of the patient's right (A) and left (B) eyes showed pathological changes in the retinal nerve fiber layer secondary to acute ACG.

Two months after the patient initially presented to the clinic, she was re-evaluated in our office, at which time her IOP were elevated despite self-reported adherence to a regimen of three topical ocular hypotensive medications. She did not report experiencing ocular pain, headache, blurred vision, or erythema despite IOPs of 48 mm Hg OS and 50 mm Hg OD. The examination did not reveal any corneal edema or intraocular inflammation in either eye.

After pupillary dilation round swollen clear lens with small diameter was revealed. We decided to do Trabeulectomy in both eyes. Right Trabeculectomy was done under hyperosmotic agent. Post operative IOP was 10 mm Hg in right eye and 25 mm Hg in left eye. We did Trabeculectomy of left eye after 14 days. Post operatively IOP was 12 mm Hg in both eyes. Normal IOP was maintained later on.

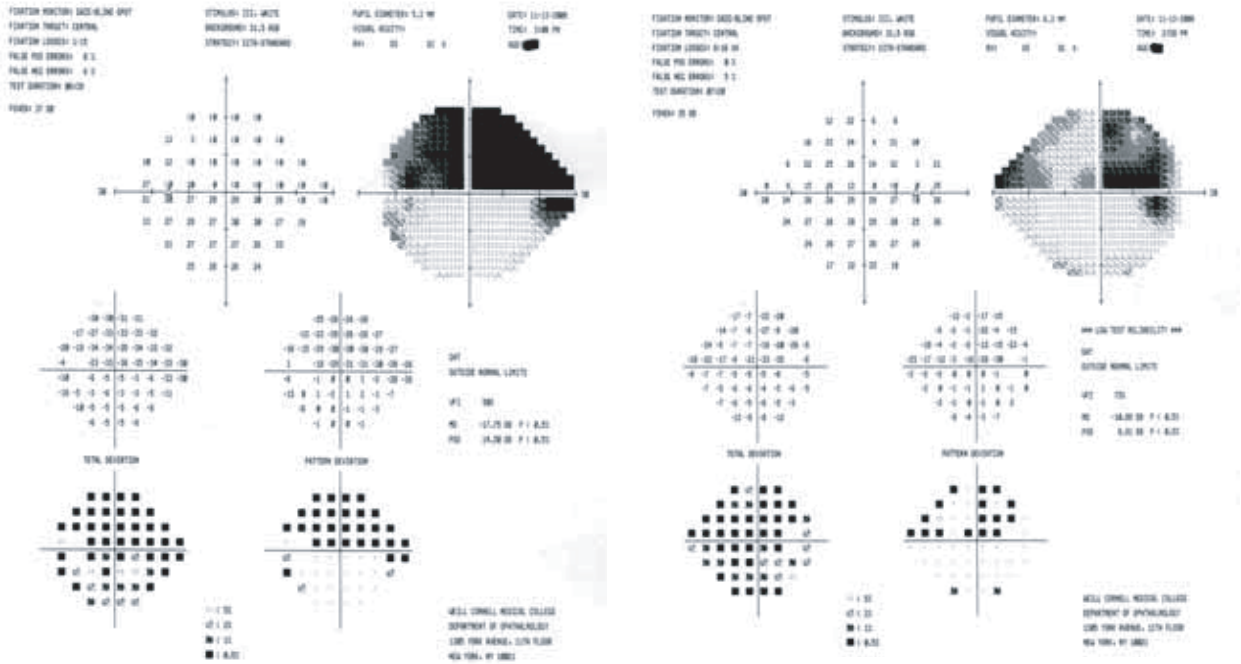

Figure 3. Perimetry showed superior visual field defects in the patient's right (A) and left (B) eyes. The defect in the left eye cor- related with a notch in the left optic nerve.

Outcome

Postoperatively, the patient achieved an acuity of 6/60 OD with mild myopic correction. We also noted significant postoperative deepening of the anterior chamber, and gonioscopy revealed an essentially open angle with some residual peripheral anterior synechiae nasally. We continue to monitor her closely.

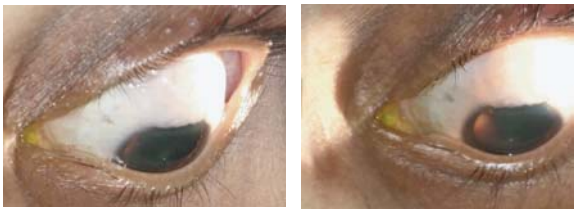

Figure 4. Post Operative trabeculectomy filtration bleb in the both eyes.Discussion

Bilateral ACG is an unusual and uncommon occurrence. The differential diagnoses for bilateral ACG are limited and include uveal effusion related to medication, general anesthesia, snake venom, and lenticular/zonular abnormalities.[1-5] In this case, we ruled out uveal effusions.

Based on the size and shape of the patient's crystalline lenses, we instead considered a diagnosis of spherophakia. Spherophakia is a rare congenital abnormality in which the crystalline lens' unusually large anteroposterior diameter causes it to take on a spherical formation. Clues to the diagnosis of spherophakia include a shallow anterior chamber, ACG, and high lenticular myopia (diameters of 4.5 to 4.9 mm).[6] Increased curvature of the lens is associated with weak, elongated zonules that can lead to ACG with pupillary block and the formation of peripheral anterior synechiae, subluxation of the lens into the anterior chamber, inflammation of the ciliary body, or progressive narrowing of the angle by the lens' anterior movement.[7,8]

Microspherophakia (the presence of a spherical lens with a reduced equatorial diameter) is associated with systemic diseases such as Weill-Marchesani syndrome, Marfan syndrome, homocysteinuria, Klinefelter syndrome, Alport syndrome, and Meyer-Schwickerath-Weyers syndrome.[8-12] Investigators have hypothesized that spherophakia occurs when an incompletely developed ciliary body and its loose elongated zonules do not exert sufficient pressure to flatten the developing lens.

The lenses of patients with spherophakia therefore retain a fetal spherical conformation.[13] Pupillary block in spherophakia is exacerbated by treatment with miotic drugs, because the relaxation of the ciliary body allows the lens to move forward and obstruct the pupillary aperture. On the other hand, mydriatic agents can sometimes relieve pupillary block by increasing tension on the zonules and pulling the lens complex posteriorly.[14]

Surgeons can attempt to break an attack of ACG with laser peripheral iridotomy or lensectomy. The latter option may not be effective for eyes that have peripheral anterior synechiae or a damaged trabecular meshwork. Surgeons have advocated that patients who have peripheral anterior synechiae over more than 270º of their angle undergo goniosynechiolysis to help open the angle.[15-17]

If damage to the trabecular meshwork is advanced, the previously described surgical interventions may not prevent glaucomatous progression. In these cases, patients required filtering surgery to lower their IOP. However, because filtering surgery tends to flatten the chambers of eyes with ACG and may contribute to the development of malignant glaucoma.[15]

Conclusion

Bilateral angle closure glaucoma can occur in the microspherophakia cases. Meticulous examination with proper attention and management in time can solve the problem.

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4th Asia-Pacific Glaucoma Congress

The 4th Asia Pacific Glaucoma Congress (APGS) 2018 took place at Busan Exhibition and Convention Centre(BEXCO) from April 13-15,2018 in Korea.

The 4th Asia Pacific Glaucoma Congress (APGS) was the largest glaucoma meeting in Asia Pacific region.

The 4th Asia Pacific Glaucoma Congress (APGS) participants were 729. The overall feedback on the educational content was very positive.A team of 38 ophthalmologists from Bangladesh Glaucoma Society (BGS) attended there. 6 speakers and 11 presented their papers in the symposium.

The 4th Asia Pacific Glaucoma Congress (APGS) was a huge gathering for exchange our knowledge, views and ideas specially to update the latest glaucoma diagnostic and surgical procedure.

8th World Glaucoma Congress

The 8th World Glaucoma Congress 2019 will be held at the Melbourne Convention & Exhibition Centre (MCEC) Australia from March 27-30, 2019. Melbourne Convention & Exhibition Centre (MCEC) is recognized as Australia's Leading Meetings and Convention Centre. It is set on the banks of the Yarra River. The MCEC is situated within 20 minutes from the airport and within 15 minutes all Melbourne landmarks are reached.

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