Journal Bangladesh Glaucoma Society

July 2015 Volume 03 Number 02

JBGS

Contents

Journal
Bangladesh Glaucoma Society

Volume - 03 Number - 02 July 2015

Journal
Bangladesh Glaucoma Society (JBGS)

Volume - 03, Number-02, July 2015

Published by :
Dr. Zakia Sultana Shahid
Scientific Secretary, BGS
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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(Introduction, Materials & Methods, results, Discussion, conclusion).

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Epidemiology and Genetics of Primary Angle Closure Glaucoma (PACG)

N. Khan[1], M. N. Islam[2]

Epidemiological concepts addresses :

How common is the disease ?
Who is most affected ?
What is the underlying cause ?
What can be done to prevent it ?

Recent world-wide study showed:[1]
PACG - 59%. World-wide.
Globally Bilateral Blindness by PACG - 3.9 million in 2010 and it will increase 5.3 million by 2020.
In Asia[1]
China alone have PACG - 47.5% (10 million)

Japan	5%
Thailand	3.8%
Singapore	3.2%
Aravind, India	2.6%
Dhaka, Bangladesh	0.4%

In Bangladesh, Hospital & clinic based study in 6-districts in 2010-2011(Dhaka, Sylhet, Chittagong, Bogra, khulna & Dinajpur, showed PACG (48.7%) and Female are more than Male.[2]

According to M.M.Rahman at al in Banhladesh among people aged 40 yrs and older, the prevalence of PACG was less 0.4%. Females were more than male. Blindness by PACG was 57%. 90% people were unaware of their diseases.[3]

In India, Hospital based study showed : PACG, New Delhi-AIIMS-55%, Chandigarh and Aruvind eye hospital – 47%[4]

In Karachi a clinic based study showed that among 40 years or older people had 30.67% chronic angle closure glaucoma (CACG).[5]

Cause of PACG :

PACG is strongly hereditary. More common in Asians than the Western populations.[1]
Females are at high risk of PACG .about – 70 %[1]
Because of genetically

* Shallow A/C
* Small eye with Hyperopia

&

*Reduced estrogen level in old age (which is protective of optic nerve)

Genetics of Primary Angle Closure Glaucoma:[6]

The size of the anterior chamber was suggested that genetic factors were involved in its pathology and the action of a large number of inherited genes along with environmental factors result in anatomical abnormalities of PACG. But the genetic basis is not well understood.

Recent research shows ,although more than 15 genetic loci have been reported in association with POAG on chromosome 8q. But the genes underlying PACG are still unknown.

In Southern Chinese population, MMP-9 gene might be associated with PACG.

4 in Pakistani population, heat-shock protein 70 (HSP70) genes have been reported.

In Napalese population, hepatocyte growth factor (HGF) gene may be involved in the pathogenesis of PACG.These genes are related to the regulation of axial length and structural remodeling of connective tissue.

Prevention of advanced PACG - needs :

1. Early diagnosis & Treatment.
2. Expert medical team for diagnosis.

3. Regular screening program.
4. Laser facilities.
5. Regular follow-up.
7. Change of life style.
8. Proper counseling.
9. Compliance.

Conclusion

The prevalence of PACG is particularly high among older Asians and the number of people worldwide with the condition will increase dramatically in the coming decades. So, all health care providers need to increase their awareness of this condition .

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Primary Angle Closure Glaucoma in Chittagong, Bangladesh – Modes of Presentation and Management Patterns at a Tertiary Eye Care Centre

M. A. Karim[1]

Purpose : The purpose of the current study is to describe clinical manifestations, management and its outcome of patients who were diagnosed with Primary Angle Closure Glaucoma at the Glaucoma Department of the Chittagong Eye Infirmary and Training Complex, Bangladesh.

Method : A hospital based observational case series study. Study period was from 1st January 2011 to 30th June 2011. All cases were diagnosed by a single consultant and were diagnosed based on clinical presentations, ophthalmic examination (including gonioscopy). Detail history taking and ocular examinations were done that included stit lamp biomicroscopy, applanation tonometry, gonioscopy and fundoscopy. Management detail was recorded. Patients were followed up after one week, 1 month, 3 months of initial visit. Examination and investigation findings were documented as much as possible.

Result : A total number of 84 patients with PACG were included. Majority of patients (93%) were between ages of 30 to 70 years. Females predominated with a total of 75%. Symptoms were experienced by 73% of patients whilst the remaining 27% did not have any complaints. Majority of patients were from rural areas (71%). 79% of patients had an acute presentation with symptoms appearing within the week of presentation. 49% of patients had a visual acuity of < 6/60. 24% were hypermetropic and 13% myopic in the affected eye. 82% of patients had closed angles in the affected eye. 73% were given both medical and laser treatment whilst, 6% required surgical treatment.

Conclusion : PACG is a leading cause of blindness in East Asian countries and due to the high prevalence in this region there is great interest in the natural history of the disease. In Bangladesh, the trends are similar to other Asian countries and hence sharing and integrating of information can help us better manage this disease. There is now considerable optimism that screening and prophylactic treatment for PAC and PACG may be a viable method of preventing blindness in very large numbers of people in Asia.

Key Words : PACG (Primary angle closure glaucoma), closed angle, laser peripheral iridectomy, trabeculectomy

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Introduction :

Glaucoma is the leading cause of irreversible blindness worldwide.[1] Primary angle closure/glaucoma (PAC/G) is more predominant in China and Mongolia than among Caucasians and Africans. Primary angle-closure glaucoma (PACG) is a major form of glaucoma in Asia, compared to Africa and Europe, especially in populations of Chinese and Mongoloid descent.[2-4] Prevalence studies in southern India found that the prevalence of PACG in Indians is also high[5,6] almost similar to that reported in Mongolians. Primary angle-closure glaucoma is sight-threatening in a short time period versus vision loss in POAG. Almost half of PACG patients were blind in one or both eyes in Andhra Pradesh,[5] and PACG accounted for most of the glaucoma blindness in Singapore.[4]

A significant proportion at risk is the adult population and since PACG is a more blinding disease than POAG[7,8] It has significant implications in causing a burden to the society. This study was done in Chittagong, Bangladesh to present some of the patterns of this blinding disease which can be treated and prevented from causing severe visual consequences.

Method :

This is a hospital based observational case series study. Study period: 1st January 2011 to 30th June 2011. In this period, all newly diagnosed cases of PACG were included in the study. All cases were diagnosed by a single consultant and were diagnosed based on clinical presentations, ophthalmic examination (including gonioscopy).

Detailed history taking and ocular examinations were done that included stit lamp biomicroscopy, applanation tonometry, gonioscopy and fundoscopy. Data about medications, glaucoma surgery if needed and post operative follow up.

Management detail was recorded. Patients were followed up after one week, 1 month, 3 months of initial visit. Examination and investigation findings were documented as much as possible.

Data analysis was done by SPSS V-13.

Results:

A total number of 84 patients with PACG were included. Majority of patients (93%) were between ages of 30 to 70 years. Females predominated with a total of 75%.

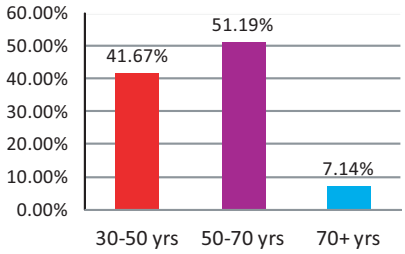


Figure-1 : Age group distribution

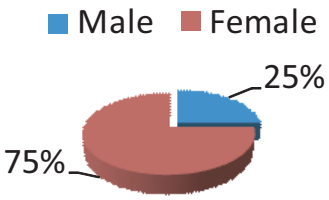


Figure-2 : Gender distribution

Symptoms were experienced by 73% of patients whilst the remaining 27% did not have any complaints. 79% of patients had an acute presentation with symptoms appearing within the week of presentation.

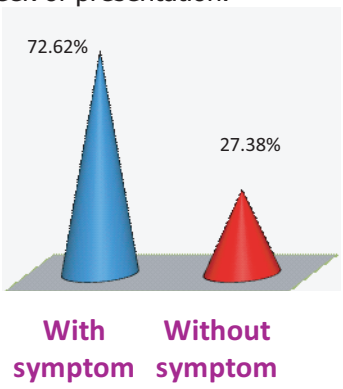


Figure-3 : Presence of Symptoms

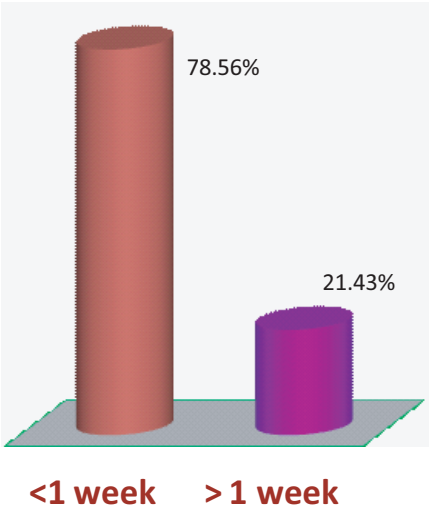


Figure-4 : Duration of symptoms before presentation

Majority of patients were from rural areas (71%) and majority were of a low socioeconomic status (69%). Almost 80% of patients had never had treatment previously.

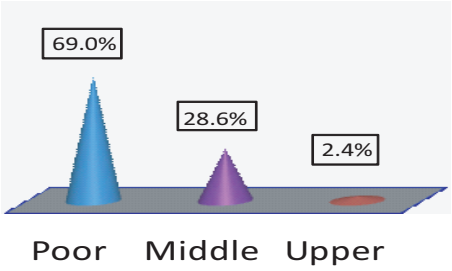


Figure-5 : Socio economic Status

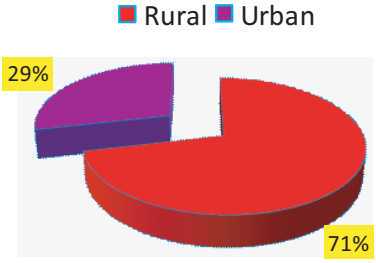


Figure-6 : Residential Status

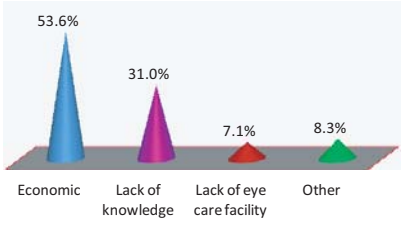
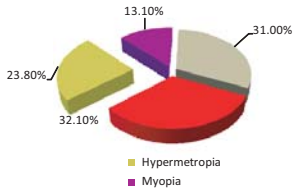


Figure-7 Reasons for delayed presentation

24% cases were hypermetropic, 13% myopic and 30% were emetropic in the affected eye. In 32% cases in the affected eye refraction was not possible due to hazy media. In the fellow eye 12% cases were myopic, 30% cases were hypermetropic, 2% cases were emetropic and in 56% cases refraction was not possible due to hazy media.



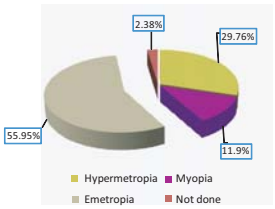
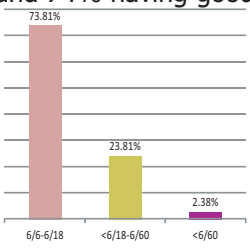


Figure-8 : Refraction of the Affected Eye

Figure-9 : Refraction of the Fellow Eye

49% of patients presented with significant decrease in vision (<6/60) in the affected eye and 74% having good vision in the fellow eye.



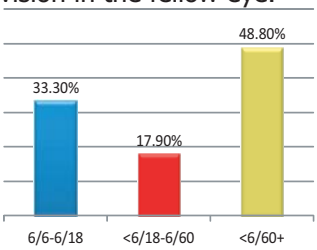
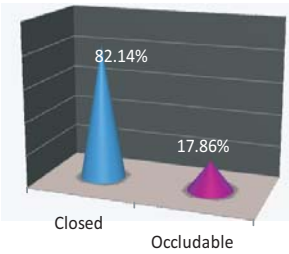


Figure-10: BCVA of the Affected Eye at presentation

Figure-11: BCVA of the Fellow Eye at presentation

In the affected eye 82% of patients have closed angles and 18% cases have narrow angle. In the fellow eyes 68% cases having narrow angles and 32% cases with closed angle.



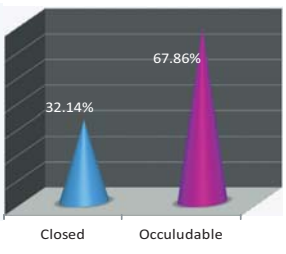
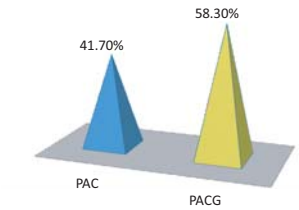


Figure-12: Gonioscopy of the Affected Eye

Figure-13: Gonioscopy of the Fellow Eye

58% of patients were diagnosed with PACG in the affected eye and 16% in the fellow eye. 85% of the fellow eyes had primary angle closure.



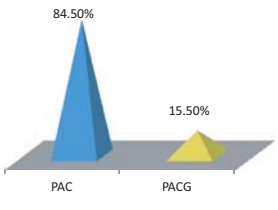
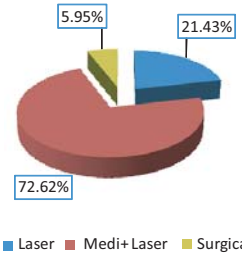


Figure-14 : Diagnosis of the Affected Eye

Figure-15: Diagnosis of the Fellow Eye

Majority of patients were effectively treated in both the fellow and affected eyes with medications and surgery. Only 6% needed initial surgery in the affected eye.



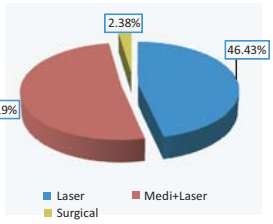
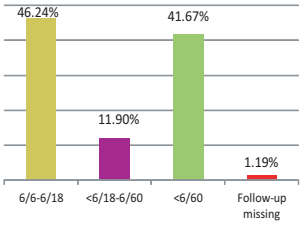


Figure-16: Treatment of the Affected Eye

Figure-17: Treatment of the Fellow Eye

Improvement of visual acuity was seen in those who had moderate loss of vision in the affected eye. There was an increase in vision in the fellow eye.



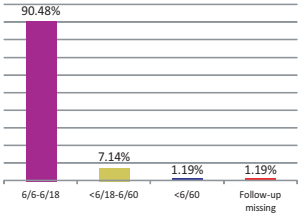


Figure-18: BCVA of the Affected Eye after Treatment

Figure-19: BCVA of the Fellow Eye after Treatment

Discussion

Asians account for almost half of the glaucoma population in the world, with the number of people suffering with glaucoma nearing 70 million.[9,10] Primary angle-closure glaucoma (PACG) is significant in this region especially in populations of Chinese and Mongoloid descent.[3,4,11] The high prevalence shown from Indian studies[5,12] gives an indication of the numbers in Bangladesh since there is similar ethnicity and origins.

Ethnic background is one of the major factors determining susceptibility to primary angle-closure (PAC). Among people aged 40 years and over, the prevalence of PAC (the number of cases present at one point in time) ranges from 0.1% in Europeans,[13] through 1.4% in East Asians[3,14] and up to 5% in Greenland Inuit.[15]

Visual compromise due to PACG usually increases after age 40.[1,2] 93% of our patients were between 30-70 years; hence we do have some cases less than 30yrs which should be noted. Female gender is recognised as a major predisposing factor toward development of PAC. The prevalence of occludable drainage angles, PAC and PACG, all tend to be higher in women than men.[3,16] This trend is also seen in our study which shows 75% of our patients being of the female gender. Also in Bangladesh, marginalization of women and their access to health care, poverty issues and low literacy rates of women further compounds the problem if PACG in women.

Shallow anterior chamber is a predisposing factor for angle-closure. With increasing age, the depth of the anterior chamber reduces and trends show that it is shallower in women than men.[17] Ethnic groups where there is high prevalence of PAC tend to have shallower anterior chambers.[17] Anterior chamber depth is determined by the position of the lens within the globe. Diagnosis of PACG is verified by gonioscopy, but this is not routinely available in many parts of Asia. So this poses a problem.

Refractive status, axial length, anterior chamber depth and lens thickness are usually associated. Hypermetropes have shallower anterior chambers than myopes and hence angle-closure is typically associated with hypermetropia. Our study shows a predominance of hypermetropia over myopia.

By alleviating pupillary block, laser peripheral iridotomy (LPI) is an effective treatment of PACG. It is safe and effective as prophylaxis against acute attacks[18-20] and widens the angle.[21] LPI is useful in the early stages of the disease. As our study shows, most of the patients we encounter in our clinic live in rural areas; are of low socioeconomic status and have barriers in accessing health care. When they arrive at us for treatment they already have established PACG with glaucomatous optic neuropathy and visual field damage, hence LPI is not satisfactory as long-term therapy.[21,22] In a Singapore study, as many as 90% of PACG eyes treated by LPI required further medication to control intraocular pressure, and 50% eventually required surgery.[22] LPI alone may be dangerous for patients and they risk the chance of having further glaucomatous visual damage. In Bangladesh, also follow up is a problem with patients. Hence, surgery is usually a better form of long-term treatment of PACG. Selecting which patients need conservative treatment versus surgical treatment is a challenge since various factors need to be taken into consideration. The ability to decide and allocate the specific management protocols to our patients will allow us to follow up patients appropriately and to advocate early and appropriate treatment in selected individuals at risk for further visual loss. In our study majority of our patients were managed medically and with laser. 6% of patients had to have primary surgery done in the affected eye and 2.4% of patients needed to have primary surgery in the fellow eye.

In many parts of Asia and including Bangladesh, where there is limited access to lasers, many ophthalmologists resort to surgery as the definitive form of treatment of PACG. There is a lack of consensus on the best approach to the surgical management of PACG, as well as a paucity of information on the long-term results of surgery in this condition. The surgical options are diverse, including surgical peripheral iridectomy, filtering surgery, lens extraction, combined lens extraction and filtering surgery, angle-widening procedures such as goniosynechialysis, and angle-widening procedures combined with lens extraction. In our study our surgical procedures included trabeculectomy with mitomycin-c and also combined cataract extraction with trabeculectomy.

Conclusion

Over the last decade, the understanding of the epidemiology and management of primary angle-closure has advanced considerably. PACG is a leading cause of blindness in East Asian countries and due to the high prevalence in this region there is great interest in the natural history of the disease. In Bangladesh, the trends are similar to other Asian countries and hence sharing and integrating of information can help us better manage this disease. There is now considerable optimism that screening and prophylactic treatment for PAC and PACG may be a viable method of preventing blindness in very large numbers of people in Asia.

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Hypotony after trabeculectomy – risk, cause and management in a referral hospital: 2 years Retrospective Study

I. Anwar[1], **Z.S. Shahid**[2]

Abstract

Importance : Trabeculectomy is the surgical standard of care for patients with medically refractory glaucoma. The use of anti metabolite agents, such as mitomycin, has increased the success rate in long run but also the rate of complications after trabeculectomy especially low intra ocular pressure.

Objective : To determine the cause, risk factors, out come and complications of post trabeculectomy hypotony.

Design, Setting, And Participants : For this retrospective, population-based study, I enrolled the patients with post trabeculectomy hypotony whether the primary trabeculectomy surgery was done at Bangladesh Eye Hospital or operated elsewhere from October 2012 to July 2015.

Exposures : After reviewing the patient records, we determined the risk factors, occurrence of Complications and steps needed to combat hypotony. I reviewed relevant publications in Ovid, MEDLINE, and PubMed to identify studies representative of the reported trabeculectomy complication rate.

Results : In 56 eyes of 56 patients, deveoped post trabeculectomy hypotony 11 eys had angle open and 45 eyes had angle closure disease. 43 eyes had superficial scleral frap traingular and closed with single suture and 13 eyes had either traingular or quadrangular flap with two or more sutures.10 eyes developed choroidal effusion and 6 eyes developed hypotony maculopathy

Conclusions and Relevance : Risk factorsfor development of Post trabeculectomy hyoptony included use of MMC, angle closure disease, high myopia, initial high Intra Ocular Pressure (IOP)which suggests that low target IOP was set before trabelectomy and single suture to close the superficial scleral flap.

Trabeculectomy is still the surgical standard of care for patients with glaucoma that not achieving the target intra ocular pressure (IOP) with most tolerated medical treatment. The introduction of adjunctive anti metabolite agents, such as fluorouracil and mitomycin, has increased the success rate of trabeculectomy by reducing scarring at the filtration site, although these agents have also been reported to increase the rate of post trabeculectomy complications.[1-5] The number of trabeculectomies performed during the past 15 years has declined. The explanation for this decline is likely multi factorial, including an increase in medical management with additional topical agents, concern about trabeculectomy-related complications, and an increase in the use of minimally invasive Glaucoma procedures and glaucoma drainage devices. Studies from 1995 through 2004 indicated a 53% decrease in the number of trabeculectomies and a concomitant 184% increase in tube-shunt procedures.[6]

This downward trend of trabeculectomy was also supported by a survey of members of the American Glaucoma Society,[7,8] which showed an increasing preference toward tubes, shunts and a decline in trabeculectomy combined with mitomyc in treatment because of trabeculectomy-associated complications, including early hypotony, blebitis and endophthalmitis.

At Bangladesh Eye Hospital like most of the centers in Bangladesh, the initial surgical intervention for uncontrolled open-angle glaucoma is still trabeculectomy. Unless contraindicated by other risk factors, the use of mitomycin was standardized at 0.2mg/mL but unfortunately we could not extract the concentration of MMC used from Operation Notes of patients in those patients who were operated elsewhere and developed hypotony and was either referred to our hospital or came to our hospital by themselves. So in this study, we could not determine the rate of hypotony

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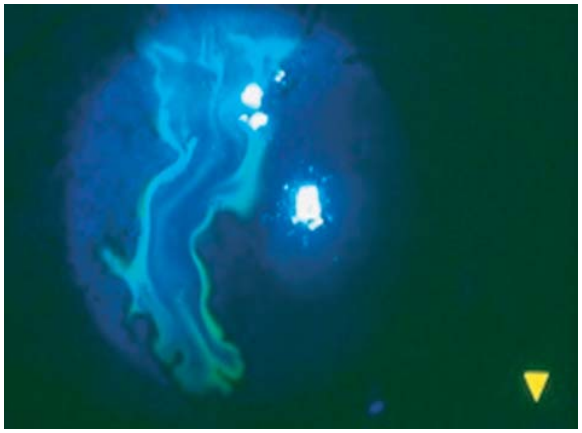
after trabeculectomy related to MMC concentration.

Methods

The present study was approved by the institutional review board of Bangladesh Eye Hospital. Informed consent was waived, and the data were de identified. The study primarily included primary open or closed angle glaucoma who had trabeculectomy surgery either with cataract surgery or alone. From the records, we recorded whether there was use of mitomycin or not applied during surgery, risk factors, measures needed to treat hyoptony and complications. Hypotony was defined as IOP of 6 or less by Applanation Tonometry. The complications included choidal effusion and hypotony maculopathy detected by indirect ophtalmoscopy and B Scan, OCT and recorded by color fundus photograph. All available follow-up information for each patient was recorded; all subsequent procedures to address complications were noted. All surgeries were done with a limbal-based approach. The associations between hypotony, complications and patient age, sex and prior laser peripheral iridotomy were explored through Cox proportional hazards regression analysis.

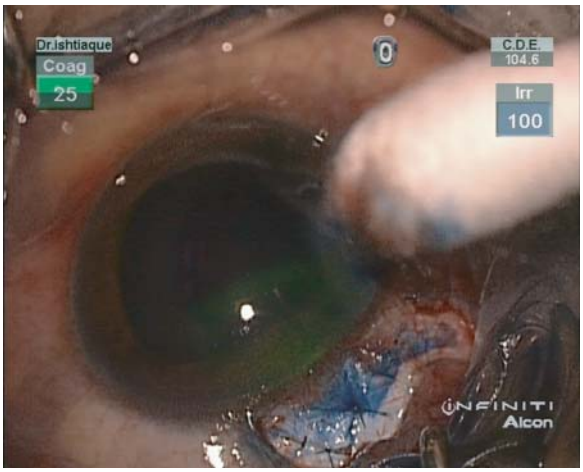
Results

I identified 56 patients at Bangladesh Eye Hospital who developed hypotony after trabeculectomy or combined operation during the study period; 56 eyes were included in the study.

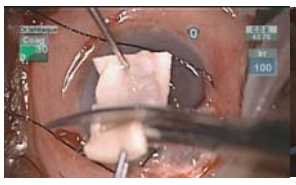


Siedel test was performed to see whether there was any leak from conjuntival closure wound or from button holing of conjunctiva. When Siedel test was positive leaking was found from conjuctival wound gap patient was taken to operation theater and wound was closed.

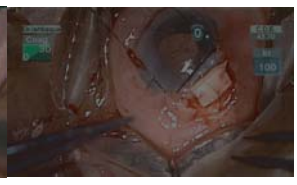
When Siedel test was positive due to conjuntival button holing large diameter bandage contact lens were applied and eyes were closed with pad and bandage for 24 hours and then siedel test was performed to see if the leak was sealed. When Siedel test was negative pad bandage was applied for 24 hours and checked wheather anterior chamber was formed and IOP came to normal range.



If there was no improvement in the chamber depth patient was taken to Operation Theater to check any over filtration with trypan blue applied over the sclera flap to see the wash out of the dye indicating over filtration in a specific area and additional suture was placed necessary to form the anterior chamber.



Slceral flap excised to cover the exposed area



The scleral flap was sututed to act as superficial flap

The predominant type of glaucoma in my study that developed hypotony was closed-angle. All cases of secondary glaucoma were excluded

because I use a primary glaucoma drainage device for these high-risk patients. I evaluated the records from 56 eyes of these 56 patients.

Causes of hypotony, anterior chamber depth and Siedel test result of the 56 hypotony eye is listed in tale 1.

Table 1 - Cause of Hypotony in relation to chamber depth and Siedel test

Anterior Chamber	
Formed	5
Shallow/ Flat	51
Siedel Test Positive	4
Leak from Conjunctival wound	3
Leak from Conjunctival Button Hole	1
Siedel Test Negetive	47
Excess drainage	40
Leak from Sutute track of scleral flap	2
No scleral flap	2
Choroidal effusion	3

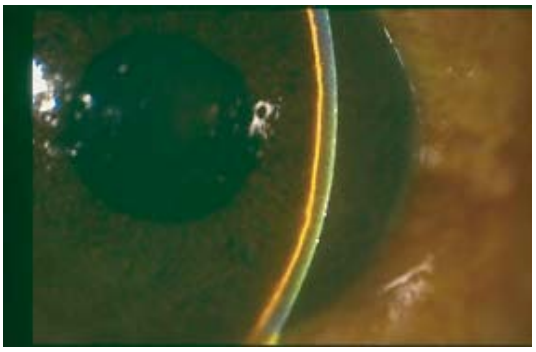
Sclera flap configuration and suture number used to close the flap was assessed through examination and operation notes. Out of 56 trabeculectomy 30 had triangular and 26 had quadrangular shape flap. And this difference was not statistically not significant ($p<0.05$). Number of suture used to close the superficial flap was noted and it was found that 43 eyes had single suture and 13 eyes had two or more sutures to close the superficial flap. So, single suture was a risk factor in developing hypotony after trabeculectomy.

The use of Mitomycin C has attributed to the long term success of trabeculectomy but this is also increased the incidence of post operative hypotony. In my study Mitomycin C was used in 40 eyes and 16 eyes did not receive any Mitomycin. But the concentration and the stage of application of MMC could not be assessed. This is a limitation of the study.

Discussion

When a patient with post trabeculectomy hypotony was recruited in my study at first the anterior chamber depth was assessed.

If the depth was normal it was due to i) over filtration or ii) choroidal effusion and it was treated conservatively with regular follow up. Systemic steroids was given to choroidal effusion eyes. Choroidal effusin was cause of hypotony in some eyes and in some eyes it was complication of prolong hypotony.



When the anterior chamber depth was shallow or flat Siedel's test was perfomed. It was positive from two settings – i) leaking from conjuntival wound ii) leaking from conjuctival button hole. The patient was taken to operation theater and suture was applied to seal the leak. If the Siedel test was negative the management depended on iris lens diaphragm relation to conreal endothelium. When iris or Crystallin lens or IOL was touching the cornea immediate surgical measure was taken – the conjunctival suture was cut to expose the sclera site. Trypan blue was applied over the sclera flap to find out if the cause of flat anterior whether it was over filtration or leaking from button holing of the superficial sclera flap.

Pad and bandage applied to eyes with hypotony with considerable gap between iris and cornea

If there was considerable gap between iris and cornea pad bandage was applied, steroids dosing was lowered to facilitate sub conjunctival fibrosis and eye was again assessed 24 hours later. And if chamber depth was shown to increase then eyes was observed. Most of the

eyes responded with this management. But if there was no improvement in the chamber depth scleral flap resutured.

Complications

Choroidal effusion developed in 3 eyes as a result of prolong hypotony and in 3 eye hypotony maculopathy. As stated earlier choroidal effusion was cause of hypotony after trabeculectomy and complication for prolong hypotony. It was treated with systemic sterod. But in 1 eye there was no improvement with this treatment and it was referred to retina department. Hypotony maculopathy was observed in young patient (mean age 41 years).

Conclusion

As post trabeculectomy hypotony occurred more in Angle closed eyes, eyes with high pre operative IOP, myopic eyes and when Mitomycin was used. So we should be cautious using Mitomycin in this type of eyes. Number of suture used to close the superficial sclera flap was related to post trabeculectomy hypotony. So we should consider using multiple or releasable suture or if needed we can LASER suturelysis if IOP is high after trabeculectomy.

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Original Article

Filtration Surgery – Trabeculectomy versus trabeculectomy with Ologen for the treatment of glaucoma : a pilot study

S. M. Noman[1]

ABSTRACT

Background : To present the result of our study that compares the out-comes of trabeculectomy with collagen implant versus conventional trabeculectomy for uncontrolled intraocular pressure (IOP).

Methods : 60 eyes of 60 patients were randomly selected for trabeculectomy either with Ologen implant (study group) or without implant (control group). Preoperative history taking & examinations were done. Data included age, gender, glaucoma type, IOP and number of postoperative glaucoma medications were collected. Post operative IOP, number of post operative glaucoma medications & post operative complications were recorded. Each patient was followed up for at least 6 months.

Result : No significant differences were observed between the groups like preoperative IOP and number of pre operative anti glaucoma medications. Post operative IOP in both groups were significantly lower than preoperative level at all follow up. The number of glaucoma medications were reduced from a preoperative mean of 3.5 ± 0.5 to a 6-month postoperative mean of 0.2 ± 0.5 ($P < 0.001$) in the study group and from 3.5 ± 0.7 to 0.4 ± .1 ($P < 0.001$) in the control group. Collagen group had statistically significant less complications were observed in this study.

Conclusion : Trabeculectomy with OloGen does not show any significant advantages over the trabeculectomy alone in terms of Intraocular pressure but immediate post operative complications are less with Ologen implantation, that indicates Ologens safety profile. Large sample size & prolong follow up are needed to confirm the safety & long term out come of trabeculectomy with OloGen.

Key Words : Trabeculectomy, Ologen, Glaucoma, Bleb, Intraocular pressure

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Introduction

Cairns 1968 introduced trabeculectomy for the treatment of glaucoma. Trabeculectomy bleb can be failed by wound healing & brosis resulting obstruction of drainage fistula. Failure can be precrented by inhibition of fibrosis. Fibroblast growth beneath the conjunctiva (between 3rd - 5th post operative day) plays an important role in bleb failure.[1] Adjunctive antimetabolites like 5-fluoro- uracil (5-FU) and mitomycin-C may enhance the success rate by preverting fibrosis.[2]

Antimetabolites increases the risk of post operative wound leak, hypotony & endophthalmitis [1.3] Studies in animal models show, the uses biodegredable collagen matrix implant beneath the conjunctiva helps in controlling wound healing process & maintain space for drainage with out post operative complications those are common with antimetablites use. [4] the back ground of this study is to compare outcomes of trabeculectomy with ologen implant with the trabeculectomy without implant.

Materials & Method

This is a prospective randomized clinical trial that was done in the glaucoma department of Chittagong Eye Infirmary and Training Complex, Bangladesh. Randomizely patients are divided into trabeculectomy (control group) and trabeculectomy & collagen (study group) 6x1 mm biodegradable, porous collagen matrix (atelocollagen plus glycosaminoglycans) were used for the implant.

Fig-1 : A Piece of Collagen

CEITC hospital review board approved the study following Helsinki declaration Informed consent was taken after detail explanation about the implant and operation. Explanation was done to Muslim people as Ologen is not halal origin.

2 Patients refused due to religious binding. No patient lost for follow up. Young age grow up (<18 yrs), neovasular glaucoma, history of previous ocular surgery or laser & absolute complicated glaucoma were excluded form the study. Preoperative following data were collected age, gender, diagnosis, level of intraocular pressure (day before surgery), numbers of medications. All patients were under went single future trabeculectomy. Post operative IOP, bleb condition & number of glaucoma medications, were also recorded in each follow up. IOP measurement was done with Goldmann applanation tonometry.

Success was defined with some criteria : (1) complete success when IOP of 21mmHg or less with out antiglaucoma mediations & (2) qualified success when IOP of 21 mmHg with the use of antiglaucoma medication. Hyptony was defined as an IOP <6mmHg. Flat anterior chamber can be defined as peripheral iridocorneal touch with central at least I corneal thickness depth.

Surgical Technique

Surgeries were done by single surgeon (SMN). After peribulbar anesthesia & proper drapping, fornix based incision was given around 12'O Clock. Triangular, superficial scleral flap (4x4mm) was produced facing apex towards 12 O' clock position. 2x1 mm deep sclerectomy & a peripheral iridectomy was performed thereafter. Scleral flap was closed with a single suture (10/O nylon). Ologen was placed over the apex of the triangular flap beneath the conjunctiva after making the operation area dry. Conjunctiva was closed like a wing with 2 sutures by 10/O nylon. Post operatively all patients were treated with Atropin 1%, 3 times daily for 2 weeks, Moxifloxacin 4 times daily for 1 week & Prednisoln acetate eye drop 6 times daily for 3 weeks then tapered gradually.

Statistical analysis was done with windows SPSS. Pre operative & demographic data & IOP comparison were analyzed with students T test. Surgical failure success & complications were analyzed with the c^2 test. With long rank test, Kaplan-Meier survival analysis for surgical success were calculated.

P values < 0.05 were taken as statistically significant.

Fig-2 : Collagen Implantation under Conjunctiva

Results

60 eyes were enrolled in the study & randomly divided into two groups of Trabeculectomy wth or without ologen implant.

Table-1 describes demographic & diagnostic data before operation. There were no significant differences between the groups in terms of age, gender, eye laterality, diagnosis, pre operative

IOP and number of topical & systemic anti glaucoma medications.

8 patients from the study group and 9 patients from control group needed systemic carbonic anhydrase inhibitors.

Operations were uncomplicated in both groups.

Table-1 :

	Study group	Control group	P-value
No. of eyes	30	30	
Age (years)			
Mean (±SD)	61.3 (±18.5)	70.9 (±12.9)	0.188
Range	20–80	32–86	
Median	65	74.5	
Gender			
Male	17 (55%)	18 (60%)	
Female	13 (45%)	12 (40%)	0.759
Eye laterality			
Right	12 (40%)	17 (55%)	
Left	18 (60%)	13 (45%)	0.17
Diagnosis			
POAG	15 (50%)	15 (50%)	
PXG	6 (20%)	6 (20%)	
PACG	3 (10%)	6 (20%)	
IG	6 (20%)	3 (10%)	0.838
Preoperative IOP (mmHg)			
Mean (±SD)	27.5 (±4.3)	34 (±10.6)	0.289
Range	20–35	21–51	
No. of preoperative medications			
Mean (±SD)	3 (±0.5)	3.5 (±0.7)	0.613
Range	2–4	1–4	
Time of preoperative medications (months)			
Mean (±SD)	45.15 (±37.34)	43.35 (±35.96)	0.180

** SD, standard deviation; IOP, intraocular pressure; POAG, primary open-angle glaucoma; PXG, pseudoexfoliative glaucoma; PACG, primary angle closure glaucoma; IG, in ammatory glaucoma.

Mean IOPs for both groups are listed in Table-2 No difference is observed in IOP measurement 6 month after operation between two groups. Post operative IOP levels in both groups is significantly lower than preoperative one. ($P<0.05$) None of the eyes in the study group developed high IOP>21mmHg post operatively where 2 eyes of control group developed so at the six month visit.

Post operatively in control group mean number of antiglaucoma medications was dropped from (3.5 ± 0.7) to (0.4 ± 0.1) ($P < 0.001$). Where in the study group from (3.5 ± 0.5) to (0.2 ± 0.5) ($P < 0.001$). There is no significant difference in their reduction between & groups.

Table-2 :

	Study group	Control group	P-value
Preoperative	27.5 (±4.3)	34 (±10.6)	0.269
Range	20–35	21–51	
Postoperative visits			
6 months	16 (±4)	15.5 (±3)	0.950
Range	11–21	10–21	

Fig-3 shows Kaplan-Meier survival analysis for both groups using complete success definition. No statistical differences observed between survival curves.

At the 6 month 27 (90%) eyes in both groups showed complete success. All eyes in the study grops 100% & 29 (97%) of 30 eyes in the control group showed qualified success. ($P > 0.66$)

No statistical significance difference was observed in post operative patients in terms of post operative complication (Table-3).

In the first post operative day 2 eyes in the control group & 2 eyes in the study group developed hypotony those were improved within one month.

Two eyes in the control group & two eyes in the study group developed flat anterior chamber with out positive Seidel test those were resolved spontaneously after giving patching for two days. 1 Patient from the control group & 1 patient from the study group developed encapsulated bleb. No patient developed cataract in the study group where as 1 patient developed cataract in the control group at the end of the 5^{th} month. No patient develop endophthalmitis in either groups.

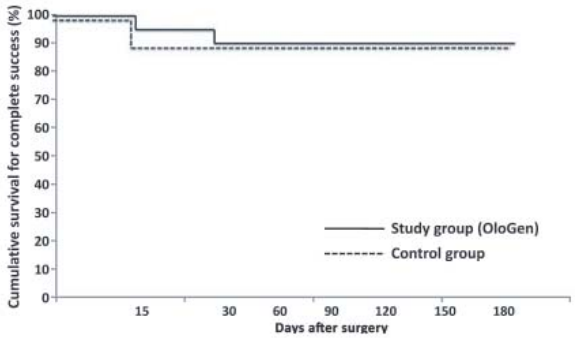

Fig-3

Table-3

	Study group (%)	Control group (%)	P-value
Hypotony	2 (5)	2 (5)	1
Flat anterior chamber	2 (10)	2 (5)	0.548
Hyphaemas	4 (5)	3 (15)	0.179
Encapsulated bleb	1 (10)	1 (25)	0.211
Cataract	0	1	0.311

Discussion

Penitrating glaucoma surgical procedures allow a powerful reduction of IOP. The pressure reducing effect of penetrating surgery is probably still higher than that of non peneterating strategies, particularly in the long run. [5, 6, 7].

Trabeculectomy as the standard procedure in penetrating anti glaucoma to us surgery was introduced by Cairns in 1968 [8].

The method was developed further over subsequent decades to address various problems. In 1990, MMC was applied as an anti metabolite during trabeculectomy [9]. Various studies demonstrated significant enhancement of success rates and post operative IOP through intra operative use of MMC [10] This is associated with an increase in adverse effect such as cataract formation, avascular blebs, thinning of the conjunctiva, subsequent blebitis and end ophthalmities. [11-13]

The current focus is on the development of less toxic agents & implants to inhibit cicatrisation with out adverse effects.

One approach in the development of biodegradable implants to serve as a place holder and prevent conjunctiva and scleral adhesion.

A few different biodegradable implants are due to be tested in animal models. With a poly (L-lactide-co-epsilon-caprolactone) film, designed to work as an adhesion barrier in filtration surgery, a significantly lower postoperative IOP was found in relation to control eyes and no signficant difference to outcome in MMC-treated eyes was detected.[14] A solid hyaluronic acid-carboxymethyl cellulose film significantly inhibited sub conjunctival scar formation and prevented adhesions of conjunctiva and sclera.[15] The use of seprafilm (sodium hyaluronate and carboxymethylcellulose) reduced postoperative conjunctiva-sclera adhesion. A porous collagen-glycosaminoglycan matrix (ologen implant) was tested in animal models. This implant was designed to prevent collapse of the subconjunctival space, for example, the conjunctive-sclera adhesion. It led to a randomsed collagen deposition and microcyst formation after penetrating anti-glaucomatous surgery in contrast to the negative control and decreased early postoperative scarring [16, 17]. Moreover, the ologen implant will also be adjuvant in repairing postoperative bleb leaks [17]. In human subjects, the ologen implant was tested non augmentation in deep sclerectomy. This study revealed that deep sclerectomy with ologen implantation is an effective and well-tolerated method for reduction of IOP. [18] A further pilot study revealed non-significant differences in postoperative IOP after trabeculectomy with ologen and sole trabeculectomy. [19] In summary of the previous studies, the use of the ologen implant promises comparable IOP reduction after trabeculectomy and a lower risk profile in comparison with the use of anti-metabolites, for example, MMC and 5-fluorouracil, although the use of ologen implant does not seem to offer a significant advantage compared with trabeculectomy alone in a pilot study. [19]

Recent studies in animal models reported that, the use of a bio-engineered biodegradable, porous collagen implant offers the potential for a new method of providing controlled resistance seteveen the anterior chamber and the sub conjunctional space in the early post operative period, as well as maintaining long term IOP control by avoiding loosely structured filtering bleb [4]. According to the manufacturer, the Ologen implant used in our study may normalize sub conjunctival wound healing and maintain good filtration & biodegrade within 30-90days.

Fig-4

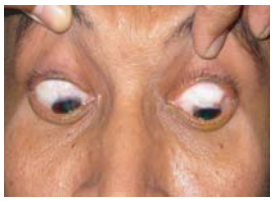

Fig-5

Fig-6

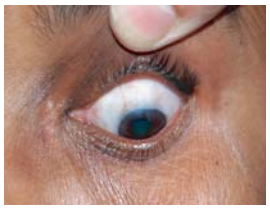

Fig-7

Fig 4-7 : Some Implanted Collagen in filtration surgery

Our study reveals that trabeculectomy with implanation of an ologen implant is a safe method for penetrating anti-glaucomatous surgery. We did not detect any ologen-specific side effects, such as translocation of the implant or erosion of the conjunctive. No allergy was detected and corkscrew vessel scores were comparable in the two interventional groups.

In the early post operative period, excessive aqueous filtration could cause low IOP. Severe hypotony could result in severe complications such as choroidal detachment, gradual bleb failure, cataract & corneal edema & can be associated with maculopathy and loss of visual acuity. [19]

In our study, there was no vision threatening complications were observed except temporary hypotony & shallow anterior chamber (same number of patients in both groups) those were improved with conservative management gradually.

Post operative IOP levels were significantly lower than pre operative levels with both groups at 6 months after operation.

The mean number of antiglaucoma medications used in both groups was significantly reduced after surgery, there was no significant difference between the two groups in terms of either the mean post operative IOP with the mean number of antiglaucoma medications used. This result is very much similar with the study of Papaconstantinou et al. [19] There were non statistically significant differences between the two groups in terms of post operative complications. That is very similar with other study [19].

Papconstantenour et al [19] experienced one case of endopthalkmitis with 2 cases of positive Seidel test in the study group. In our study we did not face such complications in either groups.

One patient from the control group developed cataract at the end of 5th month after filtration surgery. He underwent cataract surgery with implantation. Still bleb Morophology & IOP are normal limit in that patient.

4 eyes from the control groups & 2 eyes from the study groups developed hyphema at immediate post operative period (1st to 3rd).

This is probably due to leaking of blood from the scleral flap angle to the anterior chamber. In the study group probably it is less due to relative tight sealing due to pressure of the ologen.

Even though there were no statistically significant differences between the two groups in terms of post operative complications, there may be clinical significance in the fact that 4 eyes from the control group developed hyphaema & one eye developed cataract which needed cataract surgery.

Ologen did not show any allergy to anybody in our study. But biodegradation is slower than the mentioned period of 60-90 days. Even in all eyes

of the ologen group the implant degraded partially even at the end of 6 month after filtration surgery.

In conclusion of their study we can say that, trabeculectmy with ologen implantation have not significant advantage over trabeculactomy only. Additionally there were no statistically significant differences between the two groups in terms of complications.

Large sample sizes, prolong follow up are needed to confirm those outcomes with safely as well as efficacy of ologen in filtration surgery.

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Evaluation of Causes and Management of Steroid Induced Glaucoma in Vernal Keratoconjunctivitis

M.S.I. Prodhan[1]

Abstract

Purpose : To evaluate the causes and management of steroid induced glaucoma in patients with vernal keratoconjunctivitis (VKC).

Materials and Methods : This prospective study was done in glaucoma department of National Institute of Ophthalmology (NIO&H), Dhaka during the period of July/13 to June/14.Total 18 patients of VKC with steroid induced glaucoma were selected for this study. Clinical signs and symptoms, management and outcome of these patients were observed for upto six months.

Results : Among 18 patients males and females were 15 and three respectively. Ages of the patients were between 9-17 years. With steroid induced glaucoma the pressure elevation is gradual. Therefore, like primary open angle glaucoma very few symptoms exist. Cases, in which intraocular pressure did not normalize upon cessation of steroids, needed medical and surgical intervention.

Conclusion : In VKC patient, most effective drugs, steroid should be carefully administered and used only for a brief period of time, to avoid secondary development of glaucoma. Improper counseling, self medication and injudicious use of steroid for prolong period are the main cause of steroid induced glaucoma. All patients who use chronic corticsteroid medication in any capacity should have a full ophthalmologic evaluation during the course of treatment.

Introduction

Vernal keratoconjunctivitis (VKC) is a bilateral chronic inflammation of the conjunctiva. It is a variety of allergic conjunctivitis. It affects children between three to sixteen years of age, though it may appear earlier than that and continue into adulthood. In the majority of cases, symptoms resolve at puberty. Although the name vernal suggests a seasonal spring time occurrence, frequently the disease persists throughout the year[1].

Intense itching, irritation, burning, photophobia are the main symptoms of the disease.Among the signs flat topped papillae, cobble stone papillae of upper tarsus, conjunctival hyperemia, trantas dots are diagnostic. Some time corneal lesion like shield ulcer may be present. The diagnosis is generally based on signs and symptoms of the disease. Anti-allergic drops, steroids and ocular hygeine are the main stay of treatment for vernal catarrh patients. Steroid induced intraocular pressure (IOP) elevation typically occurs within a few weeks of beginning of topical steroid therapy in steroid responder.In general population five to six percent peoples are steroid responder. In the majority of cases, the IOP lowers spontaneously to the baseline within a few weeks to months upon stopping the steroid. In rare instances the IOP remains elevated.The study of steroid induced glaucoma in vernal keratoconjunctivitis patients is important for two reasons. Many patients who receive topical steroid therapy are susceptable to the development of ocular hyper tension, which if unrecognized can lead to glaucomatous optic neuropathy and the irreversible loss of vision. In addition, it helps us in evaluating the efficacy of different treatment options available for the management of steroid induced glaucoma[2].

Materials and methods : This study was prospective in nature and carried out in patients of vernal keratoconjunctivitis (VKC) with steroid induced glaucoma. Total 18 patients of VKC with steroid induced glaucoma were selected for this study presented to glaucoma department of

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National Institute of Ophthalmology (NIO&H), Dhaka during the period of July/13 to June/14. All patients were examined by same ophthalmologist. Evaluation of patient included detailed history, visual acuity, introcular pressure measurement, slit lamp examination of the anterior segment, gonioscopy, dilated fundus examination for optic disk evaluation and Humprey visual field analysis (30-2) of both eyes.

Diagnosis of vernal keratoconjunctivitis was based on the typical history and characterstic signs and symptoms. Associated glaucoma was labelled when the patient had elevated intraocular pressure with typical optic cupping and corresponding visual field deffects. Patients with persistently elevated intraocular pressure and ongoing disk damage were treated first medically. Beta blockers, Alpha-2 agonist, carbonic anhydrase inhibitors (topical and systemic) were used variably in patients depending upon the target intraocular pressure and the affordability by the patient. Clinical signs, symptoms, management and outcome of these patients were observed for upto six months.

Results : Among 18 patients males were 15 (83.33%) and females were three (16.67%) (Table-I). Ages of the patients were between 9-18 years (Table-II).With steroid induced glaucoma the pressure elevation is gradual. Therefore, like primary open angle glaucoma very few symotoms exist. Out of 18 patients only two (11.11%) patients presented in early stage and 16 (88.89%) at very advanced stage (Table-III). Five (27.78%) patients were using topical corticosteroid for less than one year period and 13 (72.22%) patients for more than one year (Table-IV). Cases, in which intraocular pressure did not normalize upon cessation of steroids, needed medical and surgical intervention. Eleven (61.11%) patients treated with anti-glaucoma medication and 07 (38.89%) patients required surgical (trabeculectomy with MMC) intervention (Table-V).

Table-I : Sex ditrbution of patients

Sex	No. of patients	Percentage (%)
Male	15	83.33
Female	03	16.67

Table-II: Age distribution of patients

Age in years	Male	Female	Total	Percentage (%)
9-12	03	00	03	16.67
13-15	05	01	06	33.33
16-18	07	02	09	50.00

Table-III : Severity of disease

Severity of disease	Number of patients	Percentage (%)
Early stage	02	11.11
Advanced stage	16	88.89

IV : Duration of steroid use

Duration of steroid use	Number of patients	Percentage (%)
<One year	05	27.78
> One year	13	72.22

Table-V : Treatment option

Treatment option	Number of patients	Percentage (%)
Medical with anti-glaucoma drugs	11	61.11
Surgical (trabeculectomy with MMC)	07	38.89

Discussion

Vernal keratoconjunctivitis (VKC) is a recurrent and chronic atopic disease typically affecting young boys in tropical climates. Ocular symptoms include mild to moderate itching, photophobia, foreign body sensation, pain and thick mucous discharge. These may be exacerbated by non-specific triger factors such as wind, dust, bright light and heat. Ocular signs include conjunctival injection, tarsal and/or limbal papillae, punctate epithelal keratitis, shield ulcers. The clinical management of VKC requires an early diagnosis, correct therapy and evaluation of prognosis. The diagnosis generally

based on the signs and symptoms of the disease, in difficult cases can be aided by conjunctival scrapings, demonstrating the presence of infiltrating eosinophils[3].

During this study we examined and counseled the patients. Patiens visited different eye clinics during the course of the disease. Steroids were used without counselling of the disease. Patients continued to use the steroid, because of the immediate relief in symptomps[4]. They had no awarness of the steroid associated complications. While on topical steroids they were visited clinicians, but steroid related rise in intraocular pressure was missed. Patients that came to us with advanced steroid induced glaucoma (irreversible) and had been on this treatment for many years. Hence, injudicious use of steroid and improper examination lead to steroid induced glaucoma.

In vernal keratoconjuntivitis patients, therapeutic options are many. In most cases topical therapy should be chosen on the basis of seveity of the disease. The most effectve drug steroid, should however be carefully administered, and only for a brief period, to avoid secondary development of glaucoma. A 2% solusion of cyclosprine should be considered as an alternative[5]. The long term prognosis of patients is generally good; however 6% patients develop corneal damage, cataract or glaucoma[3].

Glucorticoid therapy can cause elevation of intraocular pressure in many susceptable individuals, who are often refered to as `steroid responders.' Approximately 40% of general population can develop IOP elevation (>5 mmHg) after topical ocular administration of a potent glucocorticoid for 6 weeks. A smaller percentage of these individuals (4-6%) experiece a large increase in intraocular pressure (>15mmHg). They are called high responder. This IOP elevation usually progresses over the course of weeks to month of therapy and generally reverses after discontinuation of the drugs, although there are reports of irreversible IOP elevation[6]. Most of the patients of the study group did not complain headeache or haloes around light with rise in intraocular pressure. One likely explanation could be the gradual rise in intraocular pressure that occurs in steroid induced glaucoma; therefore, like primary open angle glaucoma very few symptoms exist[6]. Few of them were aware of the decrease in vision, but they and the clinician they visited mistook it to be due to the corneal involvement in vernal catarh. Clark reported many features of steroid induced glaucoma that mimic primary open angle glaucoma (POAG) [1].

Exact pathophisiology of steroid induced glaucoma is unknown. It is known that steroid induced IOP elevation is secondary to increased resistance to aqueous outflow. Some evidence indicates that the defect could be increasd accuumulation of glycosamine glycane (GAG) or trabecular meshwork- inducible glucocorticoid response (TIGR) protein, which could mechanically obstruct the the outflow. Main mechanism is membrane stabilising action – normally GAGS are present in TM causing "biologic oedema" & degraded by hyaluronidase present in lysosomes of goniocytes. Steroid stabilizes lysosomal membrane. So, concentration of GAGs increases and outflow of aqueous is obstructed. Steroid also increase expression of collagen, elastin and fibronectin within the TM and induce expression of sialoglycoprotein. Steroid inhibits phagocytosis by the endothelial cells lining the TM, leading to an accumulation of debris within the meshwork. It also decreases expression of extracellular proteinases including fibrinolytic enzymes stromolysin. Steroid decreases synthesis of prostaglandin that regulate aqueous outflow. Glucocorticoid induced ocular hypertention can occur with a wide variety of routes of administration including topical, oral, intravitreal, periocular, nasal or inhalation. The propensity to induce ocular hypertension is dependent on potency of the steroid, frequency of administration, dose and duration of the treatment. If unrecognized, this steroid induced ocular hypertension can lead to secondary open angle glaucoma that in many ways mimics POAG[5]. Although the steroid induced ocular

hypertension is generally reversible upon discontinuation of steroid therapy, the glaucomatous optic neuropathy is irreversible. It was found that when glaucoma did not reverse on cessation of steroid treatment, medical management was not sufficient in most cases to achieve the target pressures and surgical treatment was generally needed.

Trabeculectomy with antimetabolites (MMC) was chosen as the standard treatment. Indications for the use of anti-metabolite included younger age and conjunctival congestion in most of the patients. It remained as an effective method of treatment in advanced steroid induced glaucoma. Other surgical options are trabeculectomy with ologen, glaucuma drainage implant such as Ahmed glaucoma valve, Molteno implant, mini shunt such as Express mini shunt, iStent

Long term follow up proved trabeculectomy with MMC as an effective method for control of intraocular pressure in VKC with steroid induced glaucoma. Improvement of vision postoperatively in seven patients was due to the resolution of corneal oedema seen on presentation due to raised intraocular pressure.

Conclusion : In VKC patient, most effective drug, steroid should be carefully administered and only for a brief period of time, to avoid secondary development of glaucoma. Improper counseling, self medication and injudicious use of steroid for prolong period are the main cause of steroid induced glaucoma. All patients who use chronic corticsteroid medication in any capacity should have a full ophthalmologic evaluation during the course of treatment. Careful monitoring of the patients on corticosteroids (especially those with a family history of glaucoma) is very very essential. Self medication and injudicious use of steroid should be avoided. If necessary, steroid therapy must be used with intermittent drug holidays and never on a continuous basis. Counseling of the patient receiving steroid regarding side effects of steroid is very very important. Patients on topical corticosteroid therapy should receive follow up care at regular intervals by an ophthalmologist to monitor the ocular condition and intraocular pressure. If steroid is needed, weak steroid can be used and IOP must be monitored closely. Untreated patient may lead to permanent visual damage and blindness.

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Iridocorneal endothelial syndrome-A case report

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Abstract : A 45 years old female presented to the glaucoma clinic of Ispahani Islamia Eye with a history of pain & reduced vision in her left eye for one month. It was accompanied with headache. Ocular examination LE revealed thinning of iris, polycoria, localized iris atrophy & ectropion uveae . Intraocular pressure was 14 mmHg in the right eye(RE) and 30 mmHg in the left eye(LE). Gonioscopy of RE revealed open angle & LE 360 degree closed angle with multiple broad based peripheral anterior synechiae. The optic disc of the left eye had a vertical cup of 0.6. with thinning of inferior rim, RE cup was 0.4wth healthy neurorerinal rim(NRR). Specular microscopy of LE showed unilateral abnormal endothelium with irregular cells of variable shape and size. Visual field of LE revealed central scotoma. We treated this patient as a case of progressive iris atrophy ((variety of iridocorneal endothelial syndrome) with secondary angle closure glaucoma.

Introduction : Iridocorneal endothelial syndrome (ICE-S) is a peculiar ocular disorder. The term was proposed by Yanoff to describe three rare overlapping conditions – essential (progressive) iris atrophy, iris naevus (Cogan-Reesesyndrome) and Chandler's syndrome[1]. The iridocorneal endothelial (ICE) syndrome is characterized by abnormalities of the corneal endothelium, iris, and anterior segment leading to secondary angle closure glaucoma. ICE syndrome is usually unilateral, nonheritable and most common in white women in the third to fifth decades[2]. Dysfunctional endothelial cells migrate over the trabecular meshwork and anterior portion of the iris, resulting in peripheral anterior synechiae, ectropion uvea, correctopia, and iris hole formation[3]. Although associated with distinctive clinical features, 68% cases are misdiagnosed initially.[4]

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Prevalence of glaucoma associated with ICE ranges from 46-82%.[5] We are presenting this rare cause of glaucoma because of its diagnostic and therapeutic challenges.

Case History : A 45 years old previously healthy woman presented with one month history of blurring of vision and pain in LE. There was no history of colored haloes, photophobia or trauma. At the time of presentation her best corrected visual acuity(BCVA) RE 6/6 & LE 6/24. Intraocular pressure of RE 14 mm of Hg and LE 30 mmHg. Slit lamp examination of RE revealed normal (Fig-1) & LE showed localized thinning of iris, polycoria, ectropion uveae, corectopia (Fig-2a) & hammerd silver appearance of corneal endothelium (Fig-2b), Gonioscopy on RE was 360 degree open angle & LE 360 degree closed angle with multiple broad PAS (Fig-2c). Fundus examination showed RE C:D ratio 0.4, healthy NRR, LE C:D ratio 0.6,thin inferior rim. Central corneal thickness 537 mm RE and 557 mmLE. Endothelial cell counts 2552 cells/mm2 in RE and 1410 cells/mm in LE. Specular microscopy LE showed decreased cell count, polymegathism & dark cells with light central spot and light periphery. Margins of the cells were not clear(Fig-3). VF of RE was within normal limit, LE showed few central scotoma (Fig-5)

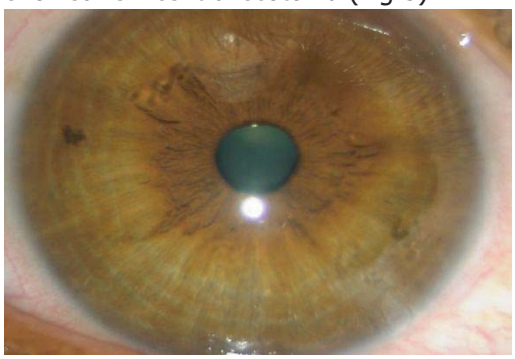


Fig- 01 : Slit lamp exam-RE

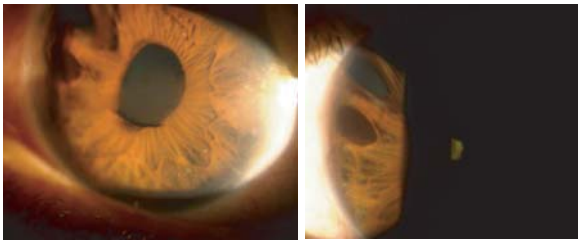


Fig- 2a : Slit lamp exam-LE

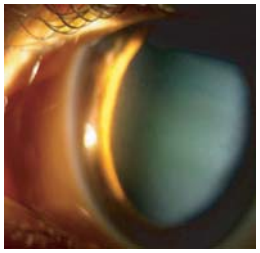
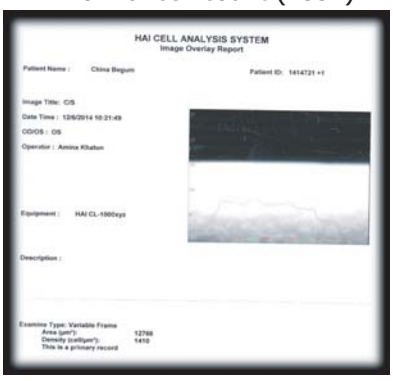


Fig 2b: Slit lamp exam LE

Fig 2c: Gonioscopy exam LE

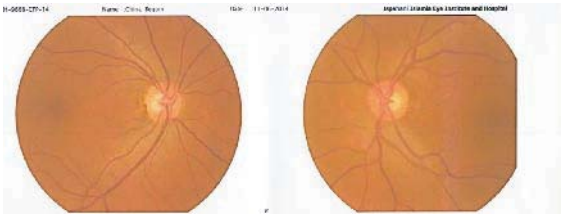
Right eye
Normal, hexagonal shaped cells.
Normal cell count (2552)



Left eye
Abnormal endothelium with large spaces between cells & low cell count(1410)

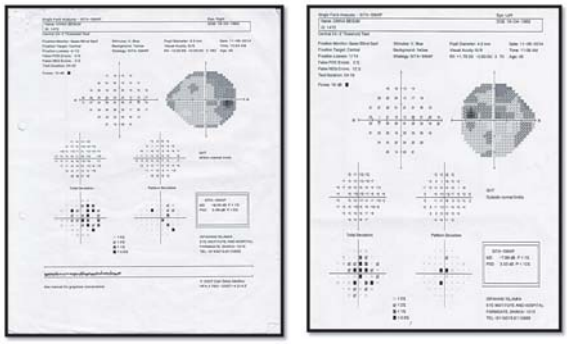
Fig-3 : Specular Microscopy

With the above clinical presentation and investigations, we came to a diagnosis of iridocorneal endothelial syndrome (progressive iris atrophy) with secondary angle closure glaucoma LE.



Right Eye Left Eye

Fig-4 : Color Fundus Photography



RE LE

Fig-5 : Humphery Visual Field Analysis

Topical anti glaucoma medications- timolol and brimonidine & brinzolamide were given. Even with topical and systemic medications IOP remained high. So augmented trabeculectomy (trabeculectomy with mytomycin-C) was done. IOP returned to 14 mmof Hg, cornea became relatively clear & BCVA was 6/12 on 1st POD (Fig-6).

Her subsequent follow up till date was satisfactory.

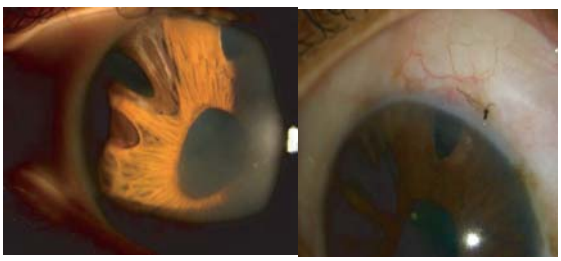


Fig-6 : LE-Status post Trab with Mitomycin-C

Discussion

ICE is a spectrum of disease characterized by primary corneal endothelial abnormality. Typically a unilateral condition although sub clinical abnormalities may be seen in other eye.[6] Usually it is seen in middle aged adult and have a female predilection. There is no systemic or genetic association. Three main variants are progressive iris atrophy, Cogan Reese syndrome and Chandler's. Distinction can be made between the three clinical variants based on clinical exam:

Chandler Syndrome : This is the most common of the three sub-types, representing approximately 50% of all cases if ICE syndrome. Of the three, Chandler Syndrome typically presents with a greater degree of corneal pathology with associated corneal edema. The edema can be microcystic, even with a normal intraocular pressure (this is common of all three variants). The iris findings are less common, and a majority of patients have no iris changes at all, making the diagnosis a challenge.

Essential / Progressive Iris Atrophy : The iris findings of this variant can be quite robust and progressive over time. Polycoria, corectopia, iris hole formation, ectropion uveae, and iris atrophy are all common findings at the time of examination.

Iris Nevus / Cogan-Reese Syndrome : This variant of ICE syndrome is distinguised by its unique iris findings. The anterior surface of iris has tan pedunculated nodules or diffuse pigmented lesions. However, iris atrophy is uncommon with these particular patients.

Corneal endothelial changes are typical having a hammered silver appearance. Specular microscopy will show characteristic ICE cells. [Dark cells except for a light central spot and light peripheral zone]. Clear hexagonal margins will be lost. Associated features are pleomorphism and decreased cell count.

The true etiology of ICE syndrome is not well understood.[7] It has been theorized that an underlying viral infection with Herpes simplex virus (HSV) or Epstein-Barr virus (EBV) leads to a low grade inflammation at the level of the corneal endothelium, resulting in its unusual epithelial-like activity.[8] Polymerase chain reaction (PCR) testing of corneal endothelial cells from ICE syndrome patients has been found to have high percentages of HSV DNA in comparison to normal controls.[9]

Glaucoma and corneal decompensation are serious sequelae of ICE. Glaucoma can be due to extensive PAS or obstruction of anterior chamber angle by membrane. Prevalence of glaucoma is 46-82%.[10] Younger patients are more affected.

Primary aim of the treatment is to reduce corneal oedema and glaucoma. Control of corneal oedema can be achieved by lowering IOP and by using hypertonic saline and soft contact lens. Persistence of corneal oedema can lead to corneal decompensation. If visually significant corneal oedema persists then keratoplasty is indicated. One report states that all cases of penetrating keratoplasty for ICE-S fail within 2 years.[11] A better surgical option is deep lamellar endothelial keratoplasty (DLEK). It provides patients rapid visual outcome with minimal refractive changes. Additionally, replacement of the dysfunctional endothelium through descemet stripping with endothelial keratoplasty (DSEK) can successfully treat corneal oedema and associated visual loss.[12]

Glaucoma in early stages can be controlled by aqueous suppressants. Miotics are not effective due to obstruction of trabecular meshwork. Long term medical management is usually ineffective. When no longer controlled medically, surgery is indicated, of which better options are augmented trabeculectomy and shunt surgeries. Surgical procedures have variable success rate. Late failure can occur due to obstruction of fistula by synechiae or by endothelialization. Failure can occur due to inflammatory response also. Failure rate is high in young individuals. If a membrane is identified gonioscopically or presumed to be occluding the ostium, Nd:YAG laser treatment may be effective, but recurrent obstruction of the surgical ostium is likely.

Long-term surgical outcomes have been reported to be slightly better with glaucoma drainage implants (survival of 71% at 1 year, 71% at 3 years, and 53% at 5 years) versus trabeculectomy with antifibrotic agents (survival of 73% at 1 year, 44% at 3 years, and 29% at 5 years).[13]

Regardless of the procedure, it has been noted that these patients typically require multiple surgeries to maintain stable IOP control. If surgical success is not obtained with a trabeculectomy or glaucoma drainage device, it may be necessary to treat patients with a ciliary body destruction procedure. Typically this is done with diode laser cyclophotocoagulation (diode CPC), and is reserved for intractable cases of glaucoma.

Aqueous shunt surgery is a common first-line surgical approach for glaucoma associated with ICE syndrome. Common complications in this setting include (1) proliferation of the ICE membrane onto the tube with occlusion of the lumen tip and (2) the formation of iridocorneal adhesions in the area of the tube with forward migration of the tube and obstruction of the lumen tip. Treatment with an Nd:YAG laser may be effective in restoring patency of the tube lumen obstructed by iris tissue or the ICE membrane. The tube frequently must be repositioned.

Conclusion

Though uncommon in routine practice, ICE syndrome has attracted much attention both for its pathogenesis and challenges in its diagnosis and treatment. Management of eyes with ICE syndrome with glaucoma is always a challenge as there is no way to stop progression of ICE cells. Treatment is usually focused on managing glaucoma either through medication or possibly surgery to reduce IOP. Failure rate of medical treatment is more than 70%. So it is better to intervene surgically as early as possible. But there is a high chance of failure even after surgery. In patients in whom trabeculectomy fails, early consideration should be given to a GDI procedure. These patients typically require multiple surgeries to maintain stable IOP control. Medication might also be prescribed to reduce corneal oedema. If visually significant corneal oedema persists then keratoplasty is indicated. A meticulous anterior segment exam is indispensable in any patient presenting with monocular glaucoma. This is a challenging disease process to manage and requires close collaboration between the glaucoma and cornea specialists.

As physicians' understanding of the underlying cellular and molecular mechanisms of this disease spectrum improves, new therapies may become available that inhibit further proliferation of the transformed endothelial cell population. Such advances may improve the outcome of the incisional surgical treatment of the associated glaucoma and extend the survival of corneal transplants.

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Original Article

Non contact Tonometery and Direct Ophthalmoscopy: Ideal method for mass glaucoma (POAG) screening.

M.S.R Chowdhury[1], S.M.B. Islam[2], M.A. Salek[3], S.A. Faysal[4], M.N. Islam[5]

Abstract

Purpose : Easy and quick diagnosis of POAG by measuring IOP with Non contact tonometer and fundus examination with direct ophthalmoscope.

Methods : A multicentric study was conducted from 2nd to 10th January and 8-12th March 2015.Inclusion criteria was age 35-65 yrs ,Doctors, Lawyers and employers of ministry of social welfare. Exclusion criteria was any ocular inflammatory condition, persons below the age of 35 yrs. Observation parameters included recording of IOP with non contact tonometer, optic disc cupping by direct ophthalmoscope and visual field examination in suspected cases .

Result : Total 1005 subjects were examined, among them 825 male (82%) and rest were female 180(18%). Normal(12-18mmof hg) intra ocular pressure with normal optic disc found 946(94.12%), raised (18 & above) intra ocular presure but optic disc are normal was 30(2.99%) and 29(2.89%) subjects with both raired intra ocular presure and cupping (.5-1.0) of the optic disc was found in male 20(68.96%) and female 09(31.04). Visual acuity of all the patient recorded ranging 6/6 to NPL. Two of them were 6/6 with cupping. 9. Glaucomatus visual field change found in 29 subjects. Awaererness about glaucoma in this study group was markedly high.

Conclusions : Non contact tonometry and direct ophthalmoscopy is easy and quick method for mass screening of primary open angle glaucoma.

Key words : non contact tonometer, ophthalmoscope, glaucoma, mass screening.

Introduction

Glaucoma causes irreversible blindness[1]. Global glaucoma blindness is 6.7 million[2], which is second leading cause of blindness (cataract is the first cause 19.3 million[3]). It shares 15% of world blindness[4].

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Glaucoma is a chronic, progressive optic neuropathy caused by a group of ocular conditions which lead to damage of the optic nerve with loss of visual function. The most common risk factor known is a raised intraocular pressure.[5]

Open angle glaucoma is the most common form and occurs when intra ocular pressure rises and progressively damage optic nerve, resulting permanent and irreversible vision loss. Primary open angle glaucoma mostly asymptomatic, only intelligent subject can notice otherwise patients do not notice until disease is advanced and accompanied by severe vision loss. Even in developed countries 50% of glaucoma sufferers remain undetected[6].

Triad of primary open angle glaucoma includes raised intra ocular pressure,cupping of the optic disc and specific visual field changes. we measure intra ocular pressure by Non contact tonometer.To examin the optic disc cupping direct ophthalmoscope was used.

As the disease is silent killer of vision, early detection and treatment is needed to prevent blindness[7]. Mass screening is needed for early detection of glaucoma and to create awareness among the general people since our people are less aware about glaucoma.

Materials and Methods

Prospective randomized three centre study of a total 1005 subjects were conducted in Samaj Saba Mela, ,organized by Ministry of Social welfare at Director General office, Agargoan, Dhaka(2-10 Jan15),Dhaka National Medical College (8-10 March 2015)) and Dhaka Bar Auditorium 12 March in the occasion of World Glaucoma Week2015 (8 to 14 March) where 675,125 and 205 subjects were examined respectively. The study parameter included

measurement of IOP by non contact tonometer, observe optic disc cupping with direct ophthalmoscope as wel as visual field analysis.The study was arranged by Dristy Unnayan Sangstha(DUS), Dhaka.

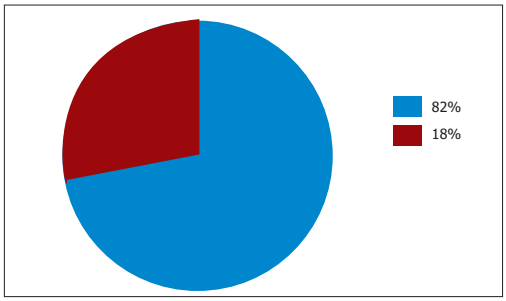
Results

Age and gender

Total 1005 subject examined among them 825 male (82%) and female are 180(18%).Age range was 35 65 years.Most of the patients are 35-45 years.Mean age of the study group was 45.57.

Table-I : Gender and Age

No. of Pt.	Male%	Female %	Age 35-45	45-65
1005	825(82%)	180(18%)	605(60.20%)	400(39.80%)

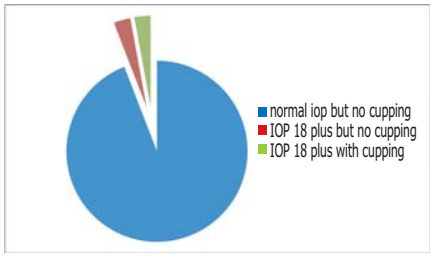


Intra ocular pressure and optic disc cupping:

Normal intra ocular pressure(12-18) mm hg with normal optic disc found in 946(94.12%), raised(18 & above) intra ocular presure but normal optic disc 30(2.98%) and 29(2.88%) had both raired intra ocular presure and cupping(.5-1.0) of the optic disc.

Table : II

No. of Pt.	IOP (12-18) & no cupping	IOP 18 & above but no cupping)	IOP 18 & above with cupping
1005	946((94.12%)	30(2.99%)	29(2.89%)

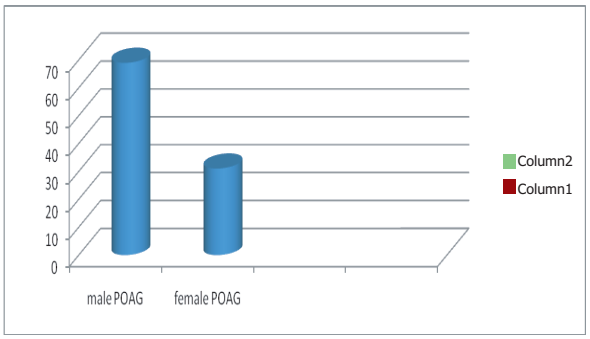


Gender ratio in POAG:

Total 29 patients were diagnosed.Among them 20(68.96%) were male and 09(31.04%) were female.

Table : III

Site	Glaucoma	Male	Female
Total	29(100%)	20(68.96%)	09(31.04%)

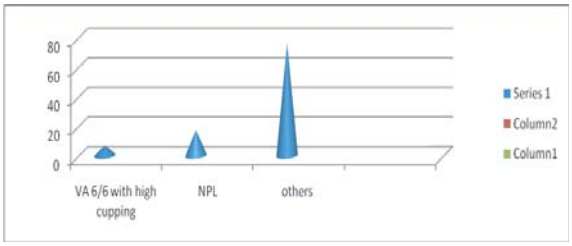


Visual acuity in glaucoma patients :

Visual acuity of all patients are recorded by Snellen chart.Among the normal 6/6 visual acuity 02(6.89%) were suffering from primary open angle glaucoma(29/2).

Table : IV

Site	Glaucoma	Visual acuity 6/6 with high cupping	NPL	6/12-PL
Total	29(100%)	02(6.90%)	05(17.24%)	22(75.86%)



Cup disc ratio:

By direct ophthalmoscope grading of cup : disc ratio was done.976(97.13%) had normal C:D ratio and 05(.49%) had C:D ratia 1.0 .Cupping show severity of damage of optic disc.

Table : V

Site	No. of Pt.	C:D .3-.4	C:D-.5-.6	C:D .7-.9	C:D 1.0
Total	1005	976(97.13%)	11(1.09%)	13(1.29%)	05(.49%)

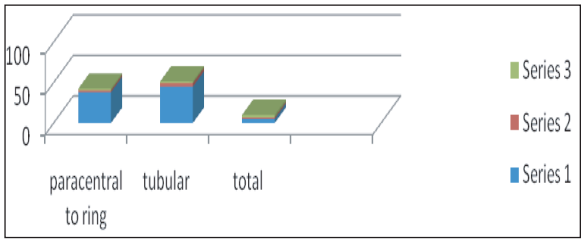
Glaucomatus visual field defect :

Visual field analysis by Humphery show 976(97.11%) are normal and 29(2.89%) glaucomatus change of different grade

Visual defect are 05(17.24%) total scotoma,13(44.83%) tubular scotoma and others are 11(37.93%).

Table : VI

Glaumatus field defect	No of subject	%
Paracentral to ring scotoma	11	37.93
Tubular scotoma	13	44.83
Total scotoma	05	17.24
Total	29	100%



Pattern of glaucoma awareness :

Most of the subjects were literate and in active service..Among them never heardof glaucoma 99(9.85%),heard but no idea 700(69.65%), inadequate idea105(10.45%) and knowledge of glaucoma 101(10.05).They knew it from electronic and print media, Rallies and ophthalmologist chamber display.

Table : VII

Status	No of pts	Percentage(%)
Never heard to glaucoma	99	9.85
Heard to glaucoma but no idea	700	69.65
Inadequate idea	105	10.45
Knowledge(Understanding glaucoma)	101	10.05
Total	1005	100

Discussion

Glaucoma is not curable. Its progression and damage of optic nerve can be delayed and prevent blindness if detected early and continued treatment as per doctors advice.It is a life long process like diabetes and hypertension.

Male 20(68.96%) suffer more than female 9(31.04) and age group is mostly 45 yrs.This finding is similar to the of Rahman MM,[8] Regarding the extent of damage ,our observation is that due to late reporting to doctors patients loss their useful vision before first visit and subsequently total blind 5(17.24%). This finding is similar to Rahman M et al[8].Late presentation, threatening central vision is an important risk factor for glaucoma blindness. this indicate low awareness in general people.

We have seen never heard of glaucoma 99(9.85%), Heard of glaucoma but no idea 700(69.65%), Inadequate idea 105(10.45%) and knowledge about 101(10.05%). This finding is dismilar with study by Saw SM et al[9] in Southern India who shows that 22.9% patient had heard of glaucoma and knowledge of glau (2.3%) was very poor. This finding also does not correlate with Mansouri K,et al,[10] who showed 70% of average Swiss population never heard glaucoma and 24.7 % can only describe glaucoma is a disease of eye related to raised IOP.The variation may be due to the educational level of the study group.

Intra ocular pressure measure by non contact tonometer is accurate more then (90%) subject.This is similar with the study of Woo jeong choi,MD et al[11].

Conclusions

Blindness is a devastating physical condition which affect emotion and economic status. Of the indivisual, family, community as wel as country. Noncontact tonometry is an fast and

easy procedure, well-excepted by patients,As it is non contact, there is ,no chance of transmission of infectiont. Non contact tonometry and direct ophthalmoscope is the best to detect primary open angle glaucoma in mass screening.

Acknowledgement

Dr. Saiyid Ahmed Memorial Foundation, Anwar Hossain Foundation.

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Original Article

Role of Central Corneal Thickness in the Management and Severity of Glaucoma

M. A. Karim[1]

Abstract

Purpose : To study the role of central corneal thickness in the management and severity of Glaucoma.

Methods : After informed consent, 200 eyes of 100 patients those who were suffering from primary open angle glaucoma and normal tension glaucoma fulfilled the criteria for the study and 200 eyes of 100 normal people as a control group were included in the study. Each patient's age, sex, family history of glaucoma, visual acuity, intraocular pressure, central corneal thickness, vertical cup- disc ratio, notching and thinning of the neuroretinal rim, mean deviation of visual field and number of glaucoma medications were analyzed.

Results : Two hundred eyes of 100 patients met the inclusion and exclusion criteria. Central corneal thickness was significantly lower in Primary Open Angle Glaucoma and Normal Tension Glaucoma (Mean: 513.94 μm) than in control (Mean: 545.065 μm) [p = 000, t-test]. Thin central corneal thickness was significantly associated with an increase in mean deviation of visual field (p=0.032), increased vertical cup-disc ratio (p=0.008), notching and thinning of the neuroretinal rim (p=0.000) and number of glaucoma medications used (p=0.025).

Conclusion : Central corneal thickness (CCT) is a powerful clinical tool in determining the severity of glaucoma. Measuring central corneal thickness may aid the ophthalmologist in identification of high risk glaucoma patients and thus can help determine the mode of management of glaucoma patients.

Introduction

Glaucoma is the single most frequent irreversible cause of blindness and it is the second leading cause of blindness worldwide behind cataract.[1] In Bangladesh, the national blindness prevalence survey found the overwhelming majority of cases were blind from cataract (80%), while glaucoma was responsible for 1.2% of the total.[2,3] The prevalence of definite and probable glaucoma was found to be about 3.1%.[4] Glaucoma is therefore an important public health problem.

Glaucoma is the leading cause of irreversible blindness in the world.[15] The aim of glaucoma management is to lower intraocular pressure to a safe level at which there will be no progression of the disease. Several studies have showed the influence of central corneal thickness in the measurement of intraocular pressure, which is the only modifiable factor in the management of glaucoma i.e. the only factor we can treat or manipulate. A thin central cornea is not only associated with underestimation of intraocular pressure but also associated with advanced glaucomatous optic neuropathy that needs aggressive treatment.

In Bangladesh, as a developing country with fewer resources, glaucoma patients usually visit the doctors very late when they have already developed an advanced stage of the disease. Most people are unaware of the damaging effects of glaucoma on their vision. During this study with glaucoma patients it has been seen that most of the patients with advanced stages of the disease have thinner corneas. This observation inspired the investigators of this study to correlate the central corneal thickness with the severity of glaucoma in an effort to gain support in the management of glaucoma patients.

Objectives

General Objective

To study the role of central corneal thickness in the management of glaucoma patients and to determine if central corneal thickness measurement can be correlated

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with the degree of severity of glaucoma.

Materials and Methods

Study Design :

A hospital-based observational study.

Sample Size :

In this study 200 eyes of 100 patients having POAG and NTG, who met the inclusion and exclusion criteria for this study, and also 200 eyes of 100 persons from a control group were included for statistical analysis.

Method of Sample Collection :

During the study period, all patients suffering from primary open angle glaucoma and normal tension glaucoma and who fulfilled the various criteria were enrolled in the study. The control subjects were collected from the outpatient department who visited the hospital for refractive errors without having any eye diseases.

Criteria for Selection of Subjects:
Inclusion Criteria:

1. Patients who are 30 years or older and suffering from primary open angle glaucoma and normal tension glaucoma.
2. The diagnosis of POAG was based on patients having an IOP of 22 mmHg or higher at the initial visit or a history of recorded IOP more than 22 mmHg, having characteristic glaucomatous optic neuropathy with diffuse or focal optic rim thinning, hemorrhage, cupping, or nerve fiber layer defects indicative of glaucoma, and corresponding visual field loss.
3. NTG was based on patients having an IOP of less than 22 mmHg at the initial visit, along with characteristic glaucomatous optic neuropathy with diffuse or focal optic rim thinning, hemorrhage, cupping, or nerve fiber layer defects indicative of glaucoma, and corresponding visual field loss.
4. The control group, of comparable age and gender, was included from the outpatient department who came for refractive problems without having other ocular pathology that may influence the corneal thickness, intraocular pressure and visual field defects.

Exclusion Criteria :

1. Patients having any ocular and systemic disease that may influence the corneal thickness.
2. Patients with retinal disease or severe refractive errors that may affect visual field analysis.
3. Patients having a secondary cause of glaucoma, including pseudoexfoliation and pigment dispersion syndromes.
4. Patients who had undergone any ocular surgery (especially LASIK procedures) or retinal laser procedures, including pan retinal photocoagulation.

Methodology

All the patients were included in the study after informed written consent. After enrollment in the study all patients underwent a thorough history taking including age, sex, occupation and family history of glaucoma in a first-degree relative. Snellen's visual acuity test was done and best corrected visual acuity was measured with appropriate correction in all patients. Slit lamp biomicroscopy was done in all patients to exclude any abnormality in the anterior segment. Intraocular pressure measurement was done by Goldmann applanation tonometer and intraocular pressure was adjusted for the central corneal thickness. Gonioscopy was performed using two mirror goniolens to detect any abnormality in the anterior chamber angle. Stereoscopic dilated detailed fundus examination was done by Ocular 60D lens and features for glaucomatous optic neuropathy were noted and also exclusion was done for any pathology that can cause visual field defects. Visual field testing was done by Humphrey visual field analyzer using 24-2 Swedish interactive threshold algorithm (SITA) standard program. The number of glaucoma medications

prescribed was noted. Advance Glaucoma Intervention Study (AGIS) score of mean deviation of visual fields was graded on the basis of the degree of damage on the total deviation printout. A score of 0 represents a normal visual field, 1 to 5 represents mild disease, 6 to 11 moderate disease, 12 to 17 severe disease, 18 and above end-stage glaucoma.

The patients who had reliable Humphrey automated 24-2 SITA standard perimetry were included in the statistical analysis. Reliable Humphrey automated perimetry was defined by the following characteristics: fixation losses less than 20%, false-positive responses less than 33%, or false- negative responses less than 33%.

Each patient's CCT was measured using an ultrasonic pachymeter (OcuScan^{TM} RxP, Alcon, USA). Initially all the patients were informed regarding the procedure. Topical anesthetic drop was applied in the conjunctival sac and then the ultrasound pachymetry probe was placed on the center of the cornea. After measuring the central corneal thickness the result was displayed on the monitor. The average of 5 CCT readings was recorded for statistical analysis. Patients then underwent visual field testing by Humphrey automated visual field testing 24-2 SITA standard perimetry protocol (Humphrey Instrument Inc, San Leandro, CA). To achieve a reliable visual field, the test needed to be repeated for few patients.

Results

The study included 200 eyes of 100 patients having POAG and NTG those who met the inclusion and exclusion criteria for this study and also 200 eyes of 100 control populations. The mean age of the 100 patients in the study group was 55.72 years (Range: 32-85 years) and in the control group the age ranged from 35 to 58 years with a mean of 45.91 years.

The patients from both groups were of comparable gender. Patients were 53% male and 47% female in the study group and 55% male and 45% female in the control group. All study subjects were Bangladeshi and from same ethnic origin. Sixty-six percent (66%) of patients had a diagnosis of NTG and thirty-four percent (34%) had a diagnosis of POAG.

The occupation of the patients were 33.7% service holder, 19.8% retired service holder, 25.26 % housewife, 17.4% businessman and 3.5% farmer. The best corrected visual acuity (BCVA) of the study population in the right eye was 6/6 - 6/18 in 87% cases and 6/24 – 6/60 in 13% cases. In the left eye, the BCVA was 6/6 - 6/18 in 90% cases, 6/24–6/60 in 9% cases and worse than 6/60 in 1% cases.

Anterior segment examination was unremarkable in all cases except RAPD in 56% cases and mild cataract in 9% of cases. Gonioscopy showed open anterior chamber angle in all cases. Posterior segment examination showed remarkable glaucomatous change in the optic nerve head in both eyes. The mean vertical cup disc ratio in the right eye was 0.798 ± 0.11 (median 0.80) and in the left eye was 0.779 ±0.118 (median 0.80) (Figure 3A and 3B). Among all the patients vertical cup disc ratio was equal or more than 0.70 was seen in 91% cases in the right eye and 89% in the left eye.

In the right eye, the notching of the neuroretinal rim was seen in 68% cases and thinning of the neuroretinal rim was seen in 24% cases. In the left eye notching of the neuroretinal rim was seen in the 65% cases and thinning was neuroretinal rim was seen in the 26% cases (Figure-4). Notching and thinning in the neuroretinal rim was seen in 92% cases in the right eye and 91% cases in the left eye. There were no diabetic and hypertensive retinopathy seen in the study group and also no other abnormality in the macula and peripheral fundus.

Both eyes of one hundred study patients and one hundred control population were included for statistical analysis. The mean intraocular pressure in the right eye of patients group of primary open angle glaucoma (POAG) was 28.53 (SD±10.67) mmHg with the range from

14 to 54 mmHg and in the left eye was 25.55 (SD± 8.49) mmHg with the range from 14 to 46 mmHg.

In the normal tension glaucoma (NTG) group the mean intraocular pressure in the right eye was 14.30 (SD ± 2.63) mmHg with the range from 10 to 21 mmHg and in the left eye was 14.47 (SD ± 2.75) mmHg with the range from 10 to 22 mmHg.

The intraocular pressure in the right eye of all patients (POAG & NTG) was 19.14 (SD ± 9.39) mmHg and in the left eye was 18.24 (SD±7.74) mmHg. In the control population the mean intraocular pressure in the right eye was 14.68 (SD±2.06) mmHg and in the left eye the mean intraocular pressure was 14.56 (SD±2.15) mmHg. The mean intraocular pressure in the study population was 18.69 (SD+8.56) and in the control population was 14.62 (SD±2.01) mmHg.

The central corneal thickness in the right eye of primary open angle glaucoma (POAG) patients ranged from 448 to 582 µm with the mean of 521.088 µm (SD ±35.65) and in the left eye ranged from 444 to 588 µm with the mean of 520.705 µm (SD ±37.06). In the normal tension glaucoma groups the central corneal thickness in the right eye ranges from 398 to 624 micron with the mean of 510.40 micron (SD ±40.99) and in the left eye ranges from 405 to 599 micron with the mean of 510.30 micron (SD ±37.61).

The mean central corneal thickness in the patients with POAG was 520.896 µm and in the NTG group the mean thickness was 510.443 µm. There was a statistically significant difference in the central corneal thickness between the primary open angle glaucoma and the normal tension glaucoma ($p < 0.000$, t-test). So the NTG patients have significantly thinner corneas than the POAG patients. Among all the patients in the study group (POAG & NTG) the central corneal thickness in the right eye was 514.04 (SD±39.40) µm and in the left eye was 513.84 (SD±37.56) µm.

In the control population the central corneal thickness in the right eye was 544.72 (SD ±23.17) µm and in the left eye was 545.41 (SD ± 23.00) µm. The mean central corneal thickness in the study population was 513.94 (SD ± 38.48) µm and in the control population was 545.06 (SD±23.08) µm. The study revealed a statistically significant difference in the central corneal thickness between the study group and the control population ($p<0.000$, t-test).

Table 1 : Descriptive Statistics of Variables in Study Group

Variables	Right Eye	Left Eye
Vertical cup disc ratio		
Mean ± SD	.798 ± .110	.779 ± .118
Median (range)	.80 (.50-1.0)	.80 (.40-.90)
Visual field (MD)		
Mean ± SD	-13.91 ± 8.82	-13.20 ± 9.29
Median (range)	-12.31 (1.0-31.8)	-10.47(1.0-35.60)
Visual field (PSD)		
Mean ± SD	7.15 ± 3.40	7.33 ± 3.73
Median (range)	7.32 (1.02-15.30)	6.88 (1.68-16.03)
IOP (mmHg)		
Mean ± SD	19.14 ± 9.40	18.24 ± 7.54
Median (range)	16 (10-54)	16 (10-46)
CCT (µm)		
Mean ± SD	514.04 ± 39.40	513.84 ± 37.57
Median (range)	513.50 (398-628)	511 (405-599)

The mean CCT was 514.04 µm in right eye and 513.84 µm in left eye. The central corneal thickness in the male patients was 514.68 µm and in the female patients the central corneal thickness was 512.27 µm. There was no statistically significant difference in the central corneal thickness between the male and female patients ($p = 0.775$). The patients with a positive family history of glaucoma had a mean CCT of 533.85 µm, while those without a family history had a mean CCT of 511.72 µm. There was also no statistically significant difference in CCT between patients with and without a family history of glaucoma ($p = 0.372$, t-test).

Table 2 : Descriptive Statistics of Variables in Control Group

Variables	Right Eye	Left Eye
IOP (mmHg)		
Mean ± SD	14.68 ± 2.064	14.56 ± 2.157
Median (range)	15 (10-18)	14.50 (10-18)
CCT (µm)		
Mean ± SD	544.72 ± 23.17	545.41 ± 23.00
Median (range)	550 (480-585)	550.50 (478-587)

Humphrey visual field examination (HVF 24-2 S/S) was done in all cases of the study group and moderate to end stage visual field loss was seen in 77% cases in the right eye and 74% cases in the left eye.

The patients (POAG & NTG) enrolled in the study received medical treatment initially to control intraocular pressure. Among them 23% patients received one medication, 68% patients received two medication and 9% patients received more than two medications to control intraocular pressure. Thus, 77% patients received more than one medication to control intraocular pressure in the study group.

Table 3 : Distribution of Patients According to Corneal Thickness

	Study Group	Control Group
Thin Corneas (CCT < 545 µm)	159	81
Normal/Thick (CCT 545 µm)	41	119
corneas	200	200

Odds Ratio: 5.7

Using 545 µm as a normal CCT value, the control group and study group (glaucoma patients) were each sub-grouped into thin corneas and normal/thick corneas. All patients with a CCT value of < 545 µm were grouped in the thin cornea group, and patients with a CCT 545 µm were grouped in the normal/thick cornea group. A calculated Odds Ratio of 5.7 shows that patients with thin corneas are significantly associated with glaucoma.

Analysis using the ANOVA test, showed that a thin central corneal thickness is a predictor for a worse glaucomatous optic neuropathy (that is assessed by mean deviation of visual field test, vertical cup-disc ratios, notching and thinning of neuroretinal rim and number of medications used for glaucoma).

Table 4 : p values for Tests of Significance of CCT as a Predictor of Glaucomatous Damage

	Mean Deviation	Pattern Std. Deviation	Vertical Cup Disc Ratio	Neuroretinal Rim Status	Number of Medications
CCT	*0.032	0.099	*0.008	*0.000	*0.025

*p values < 0.05 considered as significant

Discussion

Applanation tonometry has been the gold standard for measuring intraocular pressure replacing indentation tonometry (readings of which were influenced by more parameters e.g. scleral rigidity).[40] For applanation tonometry, central corneal thickness remains a factor impacting on the intraocular pressure measurements as initially acknowledged by Goldmann and Schmidt.[14] As later studies showed, it was evident that central corneal thickness and intraocular pressure readings are firmly connected with each other.[18,41] Subsequently, studies have demonstrated that the IOP of patients with thicker corneas may be overestimated with applanation tonometry[18,39,41,42] and that eyes with normal-tension glaucoma have abnormally thin corneas.[16,19-22, 42-44] Since one third of glaucoma patients still have progression despite good control of IOP, these findings stimulated clinicians and investigators to ponder and speculate on the relationship between CCT and glaucoma severity and glaucoma progression.[45-47]

This study compared the central corneal thickness between the primary open angle glaucoma and normal tension glaucoma and also the average thickness of POAG and NTG with the normal control subjects. In control population the mean CCT of 545.06 ± 23.08 µm, which was similar to the representative value of 544 ± 34 µm which has been shown to be the average CCT value of a normal human from a conglomerate of previously published worldwide studies on ultra-

sound measured CCT.[42] The mean CCT of control population in this study was slightly lower (within approximately 10 µm) than the

range of values for studies done on Caucasian patients (UK 554 µm; Switzerland 552 µm; USA 556 µm, 552.6 µm; Canada 556.7 µm, Germany 555.9 µm).[19,22,24,26,28,44,48] Compared with results from studies done in other Asian countries, the results in this study were in some cases similar or approximately within 10-15 µm of their mean CCT values: 541 µm (Singapore); 556 µm (China); 552 µm (Japan); 545.5 µm, 537 µm (India) and 530 µm (Nepal).[49-54] Pachymetric measurements from the Barbados Eye Study found that black participants (n=1064) tended to have thin corneas (Mean CCT: 529.8 µm).[27] Herndon et al (Mean

CCT: 537 µm), Rosa et al (Mean CCT: 530 µm), Shimmyo et al (Mean CCT: 535.5 µm) and the OHTS also showed that CCT was thinner in black participants.[24,26,28,55] From these values, it was seen that the mean CCT of the Bangladeshi controls are slightly lower than Caucasians and higher than those of Blacks.

Central corneal thickness has recently been recognized as a significant risk factor for progression of ocular hypertensive patients to primary open angle glaucoma (POAG) in the Ocular Hypertension Treatment Study.[12] Approximately 8% of ocular hypertensive developed POAG. This study was the first to prospectively demonstrate that a decreased CCT predicts the development of POAG from OHT. Participants with a CCT of 555 µm or less had a three–fold greater risk of developing POAG compared with participants who had a CCT of more than 588 µm. Apart from decreased CCT, they also found from multivariate analyses that baseline factors that predicted the development of POAG included older age, larger vertical or horizontal cup- disc ratio, higher intraocular pressure and greater pattern standard deviation.

A few other studies have also looked at CCT as a factor for determining the severity of glaucoma. Herndon et al retrospectively examined the initial visit of consecutive POAG patients over a 5-year period in North Carolina, U.S.A.[24] they examined a total of 350 eyes of 190 patients (Mean Age: 65.6 years) of which 55% were black and 42% white. They found that a lower CCT was a powerful clinical indicator for a high Advanced Glaucoma Intervention Study (AGIS) score, increased mean deviation of visual field, increased vertical and horizontal cup-disc ratios and increased number of glaucoma medications.

Another study was done in Germany on 622 eyes of 333 white patients (Mean Age: 46.8 years) with OHT and glaucoma.[56] Apart from analyzing whether glaucomatous optic nerve damage at initial presentation was related to CCT, but they also examined if the rate of progression of glaucoma during follow-up was related to CCT (Mean Follow-Up Time = 62.7 ± 33.2 months).

In this study, analyses have showed that mean CCT was significantly related to a worse glaucomatous optic neuropathy (that is assessed by mean deviation of visual field test, vertical cup-disc ratios, notching and thinning of neuroretinal rim and number of medications used for glaucoma). (See Table 7) These are in keeping with the findings of OHTS, Meideiros et al and Herndon et al.[12,23,24] In this study it was shown that, in glaucoma patients, the mean CCT was significantly related to a worse glaucomatous optic neuropathy but we won't be able to comment on whether CCT has an affect on the rate of progression of glaucomatous changes.

Limitations of the Study

There are some limitations in this study. This study initially planned on following the patients' progression of glaucomatous damage but due to factors such as irregular follow-up, insufficient follow-up data and drop-out of patients it weren't able to assess this statistically. Rather, this data reflect the findings at the initial examination by a glaucoma specialist. Also, there may be some referral bias in the patient population. Patients referred to a glaucoma specialist at a tertiary care institution may have more advanced, intractable glaucoma than those in the general population and therefore may not represent the majority of early glaucoma patients. The number of the study population was less in this study and a single

center was involved for patient collection. A multi- center study with a large population would reveal better results to draw conclusions. Finally, the AGIS grading system was designed for full-threshold strategies, but all of the patients in this study had SITA standard analysis. However, there were consistent with all interpretations, which should quantify the results in a uniform fashion.

Conclusion

This study concludes that the mean central corneal thickness (CCT) in study group was 513.96 µm and in the control group was 545.06 µm. The study subjects had significantly less CCT in comparison to the control group (OR=5.7), as seen in other previous published studies. This study proved that, in glaucoma patients, a thin mean central corneal thickness was associated with an advanced glaucomatous optic neuropathy. Central corneal thickness is a significant predictor of glaucomatous damage as measured by variables such as mean deviation of visual field, vertical cup-disc ratio, notching and thinning of the neuroretinal rim and number of glaucoma medications in patients with primary open angle glaucoma and normal tension glaucoma. Measuring CCT in glaucoma patients may help identify those patients who are at a higher risk for having severe glaucomatous damage, thus enabling the ophthalmologist to treat their disease more aggressively.

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Glaucoma : Are we always on the right track!

M. N. Kamal[1], M.N. Kamal[2], M. Imtiaz[3]

Abstract:

Purpose : To find misdiagnosed patients of glaucoma being treated on the basis of intraocular pressure measurement only.

Material and Methods : Total 102 patients, 68 males and 34 females were included in the study. All patients were selected from eye OPD with labeled or suspected glaucoma over 40 years of age. To reach a final diagnosis other risk factors in addition to intraocular pressure were considered. Majority of the patients were in close follow up for one year.

Results : 52 out of 60 patients were misdiagnosed as glaucomatous and 20 out of 42 patients were misdiagnosed nonglaucomatous based on single reading of intraocular pressure.

Conclusion : the examining doctor should take pain in diagnosing the devastating ocular condition by taking in to consideration all the risk factors. When in doubt he/she should not hesitate to seek the opinion of other colleagues. He/she should not misguide the patients.

Introduction

Primary open angle glaucoma is a chronic, painless, progressive ocular disorder which leads to irreversible visual loss by damaging the optic nerve fibers. It is very common cause of silent visual loss and is responsible for 90% of all cases of glaucoma[1]. Intraocular pressure is the only risk factor which can be modified, but diagnosis based on intraocular pressure alone usually leads to misdiagnosis.

Material & Methods

All the patients were selected from eye outpatient department with diagnosed or suspected glaucoma. Most cases were already diagnosed by ophthalmologist in the periphery and were on antiglaucoma therapy. Some patients specifically attended the OPD to exclude glaucoma. They were on no treatment. Main complaint was nonspecific headache. Some presented with progressive dimness of vision, which did not improved with glasses. Those patients were thoroughly investigated on subsequent examination, correlating the other risk factors with optic disc and visual field observations. Total 102 patients were included in the study, 68 males, and 34 females, all were over 40 years of age. Those already on treatment were asked to revisit one week after cessation of therapy. All the patients were examined by consultant ophthalmologist. Both schiotz and applanation tonometer were used.

First group of 60 patients were already on antiglaucoma therapy. 40 male and 20 females. On first examination complete history regarding myopia, diabetes, trauma, family history and steroid drops were taken. History of previous refraction and ocular treatment was noted. All medications were stopped and they were asked to revisit after one weak. Next week IOP was checked with applanation and schiotz by consultant ophthalmologist. Optic disc was examines with direct ophthalmoscope and Volk 90D lens. In doubtful cases patients were admitted for phasing for 24 hours. At least 6 readings were taken. In few cases where optic cup enlarged >0.5 CD or bilateral disparity was observed, water drinking test was performed. Second group included 42 males and 14 females. On first examination they were diagnosed non glaucomatous but revisited again for reconfirmation. They were investigated and examined by same protocol as mentioned above.

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Table-1: comparison between group-1 and group-2:

Group	Total patients	Status of presentation	Misdiagnosed (%)	Diagnosed (%)
Group1	60	Labeled or suspected glaucomatous	52(87%)	8(13%)
Group2	42	Labeled nonglaucomatous	20(48%)	22(52%)

Table-2 : sex ratio in group -1

Total patients	Diagnosis	Male(%)	Female(%)
60	52(misdiagnosed)	40(77%)	12(23%)
60	08(diagnosed)	6(75%)	02(25%)

Table-3 : sex ratio in group-2

Total patients	Diagnosis	Male(%)	Female(%)
42	20(misdiagnosed)	15(75%)	5(25%)
42	22(diagnosed)	15(68%)	7(32%)

Results

52 out of 60 patients (86.7%) were misdiagnosed as glaucomatous. Out of these 40 were male(76.9%) and 12 were females(23%). 20 out of 42 patients were misdiagnosed non glaucomatous (47.6%) out of these 16 were male (80%) and 04 were female(20%) (Table 1,2,3).

Discussion

Intraocular pressure is the most important and manageable risk factor for glaucoma management. However decision to treat or not to treat glaucoma only on intraocular pressure reading may be misleading. There are many ways in which pressure reading becomes erroneous. Usually the patients are not cooperative and move eyeball during recording. Palpebral fissure may be too small due to previous trauma or scarring. The tonometer scale may move poorly and pressing the base against cornea usually gives false reading. Scleral rigidity may create a problem. The tonometer reading may be slanted, placed on sclera instead of cornea or lids and cornea may not be in horizontal position.

The current definition of glaucoma precludes intraocular pressure (IOP) as a defining feature.[2] One can diagnose glaucoma even when the IOP is "normal." Unfortunately, in day-to-day practice, excessive cupping of the optic disc is considered to be pathognomonic of chronic glaucoma. One tends to diagnose "normal tension glaucoma" (NTG) if the IOP falls within the acceptable range often without investigating nonglaucomatous causes. In fact, in many cases, long-standing optic neuropathy can result in optic disc cupping. Causes of nonglaucomatous optic disc cupping include methanol poisoning,[3] arteritic anterior ischemic optic neuropathy,[4] and rarely chronic compressive lesions of the optic nerve. [5,6,7,8]

Doctors in busy clinic usually ignore the history of myopia, diabetes, steroid intake, family history and medication used for glaucoma[9]. Furthermore fundi are not examines at the same time. So correlation is lacking. The fear of losing vision because of glaucoma compels them to undergo unnecessary prolonged medical treatment or surgery. Since increased IOP is considered to be the primary risk factor for development of glaucoma, are we over treating a lot of patients who would turn out to be normotensive or ocular hypertensive. In such circumstance it is the batter to stop all medications for one to two weeks to recheck IOP and to do provocative test to confirm glaucoma[10]. on the other hand, if we set a target pressure of 15 mm of Hg for a patient who would turnout (later) to be suffering from normal tension glaucoma, we are being in to a false sense of security while the patient continues to lose visual field[4]. These are the questions which need to be addressed before making a final diagnosis. Another two types of mistakes by the general ophthalmologists have been noticed quite frequently while managing glaucoma[11]. The first one is the misdiagnosis of neuro ophthalmological cases as glaucoma or failure to recognize the coexistence of neuro-ophthalmological cases and glaucoma. The second error occurs is in prescription writing which is of paramount importance. The purpose of the present write up is to discuss these commonly occurring two mistakes while managing glaucoma patients.

In the current scenario of medical practice, there is a lot of consumer protection and incidents of patients suing the doctors for medical negligence. It is very important for the doctors to be well aware of the very common avoidable medical errors in management and should always be vigilant to make sure that such errors are not committed by them while managing their patients. Also making a correct diagnosis and giving the correct and most effective treatment to a patient is the responsibility of a clinician.

Medical error does take place in our clinical practice, which sometimes, may lead to medico legal cases. The term "medical error" itself, suggests that it should be prevented from occurring. Medical error is any deviation from the process of care, which may or may not cause harm to the patient. It could be an error of execution (failure of a planned action to be completed as intended), or an error of planning (application of inappropriate plan) or an unintended outcome resulting from an act of omission (forgetting a task or sub-task leading to injuries from the patients' underlying disease, which could have been prevented by optimal care) or an act of commission (doing a task incorrectly leading to injuries caused by a medical intervention).

Medical negligence, in law, is defined as failure to exercise the degree of care expected of a person of ordinary prudence in protecting others from a risk of harm. It may render one civilly and sometimes criminally liable for resulting injuries. The doctrine of negligence does not require the elimination of all risk, but rather only foreseeable and unreasonable risk.[12]

Limitation

As this study was carried out at Ad-din Sakina Medical college, Jessore, Bangladesh , we had to work with the conventional glaucoma diagnostic tools. We do not have modern tools like visual field analyzer, OCT etc.

Conclusion

Misdiagnosed glaucomatous patients are more common than misdiagnosed non glaucomatous patients. Patients more than 50 years of age were more misdiagnosed than younger ones. One should not depend on one parameter that is intraocular pressure. History, fundus examination, and visual field analysis are sufficient in most cases. When in doubt other investigations like OCT can be done. One should not hesitate to seek opinion of his seniors. When in doubt IOP should be compared between two schiotz tonometer or schiotz and applanation tonometer and suspected patients may be examined many times to put them into either category and treating physician should make an effort to diagnose the disease.

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Management of Lens Induced Glaucoma

Z. S. Shahid[1], M.H. Rahman[2]

Abstract

Lens Induced glaucoma is common occurrence in Bangladesh especially in rural area. Late appearance of a patient to a doctor, late surgery leads to a poor visual outcome. So for this season early diagnosis and treatment of cataract to give a good vision to the patient is necessary. The article highlights the importance and way to early diagnose and management of visually disabling cataract in right time.

Introduction

In 1900, lens induced glaucoma was first described by Gifford (1) & Row Reuss (2) independently. Gifford described it as a glaucoma due to hyper mature cataract But Reuss described it glaucoma due to absorption of lens matter though the intact lens capsule. Subsequently by different ophthalmologist (3-5) it has been named in different way eg. phacotoxic glaucoma, phaco anaphylactic glaucoma, phacolytic glaucoma etc. but in common we use the term lens induced glaucoma (LIG). It can be either due to immature cataract, mature cataract, hyper mature cataract or traumatic cataract. It can present either as open angle or closed angle.

Idea is to control the intraocular pressure immediately followed by cataract extraction and intra ocular lens implantation.

Late reporting of the patient, delay treatment lead to reversible blindness into irreversible loss of vision. Especially in rural area of developing country.

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The pathophysiology, diagnosis and treatment of different lens-induced glaucoma will be reviewed in this article [6-9]

Classification

A. Lens Induced open-angle glaucoma

1. Phacolytic glaucoma
2. Lens particle glaucoma
3. Phaco antigenic glaucoma (Phaco anaphylaxis glaucoma).

B. Lens induced secondary angle closure glaucoma

1. Phacomorphic glaucoma
2. Glaucoma induced by lens dislocation

LENS INDUCED OPEN-ANGLE GLAUCOMA

1) Phacolytic Glaucoma

Pathophysiology : Phacolytic glaucoma is an inflammatory process caused by the leakage of lens material through the capsule of a hypermature cataract. The released lens material is composed of altered lens protein, macrophages, and other inflammatory cells that lead to trabecular meshwork obstruction and precipitate glaucoma (6-9, 13, 18-20)

Diagnosis : The typical presentation of Phacolytic glaucoma is a painful eye with photophobia, decreased vision, and severe conjunctival hyperemia. Diagnosis is usually made by the presence of prominent cell or white material/particles in the anterior chamber, flare, reaction without keratic precipitates, corneal edema, increased intraocular pressure, and evidence of pseudohypopyon may also be present (lens protein deposits layering in the

inferior angle). Gonioscopy reveals an open anterior chamber angle. (6-9, 13, 18)

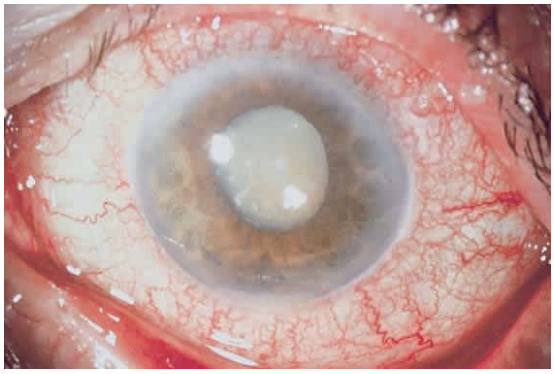

Fig: Phacolytic glaucoma with hypopyon

Management of Patients with phacolytic glaucoma should be treated initially with optical cycloplegia, topical steroids, and aqueous suppressants. The immediate goal of therapy is to reduce the inflammation and to reduce the intraocular pressure. Cataract extraction is the definitive treatment for phacolytic glaucoma (6-9, 13, 18)

2) Lens-Particle Glaucoma

Pathophysiology : Lens particle glaucoma, in contrast to phacolytic glaucoma, is secondary to a disruption of the lens capsule, which may occur after cataract surgery, penetrating lens injury, or laser posterior capsulotomy. The disrupted lens releases lens particle material in the anterior chamber leading to the obstruction of the aqueous outflow (6-8, 13)

Diagnosis : The presentation is usually delayed for a few weeks after the precipitating event, but it may occur months or years later. A history of surgery or trauma is an important element in making an accurate diagnosis. Clinical findings include elevated intraocular pressure and evidence of cortical lens material in the anterior chamber. Other possible signs are corneal edema, synechiae , and cell/flare reaction in the anterior chamber. (7, 8, 13, 21-23)

Management Medical therapy is initially aimed to control the intraocular pressure, topical steroids may be given to reduce inflammation, and mydriatics to prevent synechiae formation. If the lens particle/material does not reabsorb, there will be a large amount of lens material in the anterior chamber and the intraocular pressure cannot be controlled, surgical removal of the lens is undertaken (6-8, 21-23)

3) Phacoantigenic Glaucoma (formerly known as Phacoanaphylaxis)

Pathophysiology: Phacoantigenic glaucoma is a granulomatous inflammatory reaction directed against own lens antigens leading to obstruction of the trabecular meshwork and increased intraocular pressure. It is important to mention that phacoanaphylaxis is not the correct name of this condition since it is not an allergy. The mechanism causing the reaction seems to be an Arthus type immune complex reaction mediated by IgG and the complement system (6-8, 13)

Diagnosis: Phacoantigenic glaucoma usually occurs between one and fourteen days after cataract surgery or trauma. Clinical findings include "keratic precipitates", anterior chamber cell/flare reaction, synechiae and residual lens material. Less common is the presence of Glaucomatous optic neuropathy (6-8, 13)

Management : Initial therapy is to control the intraocular pressure with lOP-lowering medications and to reduce the inflammation with topical steroids. If medical treatment is unsuccessful, surgical removal of residual lens material is indicated (6-8, 13)

LENS INDUCED SECONDARY ANGLE-CLOSURE GLAUCOMA

1) Phacomorphic Glaucoma

Pathophysiology : Phacomorphic glaucoma is a lens-induced secondary angle-closure glaucoma that may occur as a result of mature cataract formation. Narrowing of the angle can occur slowly with formation of the cataract by pushing the iris forward or acutely precipitated by an intumescent Catarectous lens leading to obstruction of aqueous flow between the border of the pupil and the anterior capsule of the lens (pupillary block) (6, 9, 10)

Diagnosis : Phacomorphic Glaucoma is diagnosed by the presence of eye ache of decreased vision, evidence of mature cataract formation (slit lamp exam), angle closure

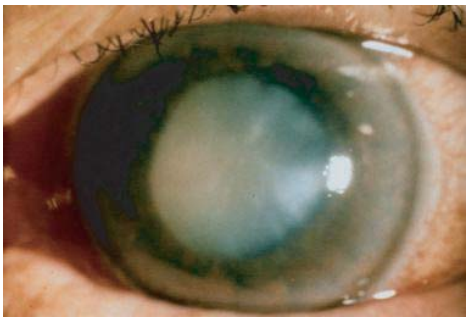

Fig : Phacomorphic glaucoma with shallow anterior chamber

(gonioscopy), and elevated intraocular pressure (applanation tonometry) in the affected eye. Distinguishing factors between primary angle-closure and phacomorphic angle-closure are presence of an intumescent cataractous lens and presence of cell and flare. Axial length measurement (usually short in phacomorphic) and records of the refraction may also be helpful in distinguishing between the two conditions (7, 9, 10, 13)

Management : Initial treatment of phacomorphic glaucoma is to lower the intraocular pressure with medical therapy such as topical beta blockers, carbonic anhydrase inhibitors and hyperosmotic agents such as oral glycerin. Parasympathomimetic agents should be used with caution since they can precipitate pupillary block. If the intraocular pressure is not controlled with medical therapy alone, a laser iridotomy is usually performed, other options include corneal depression with a Zeiss 4-mirror lens. If the fellow eye is also predispose to angle closure glaucoma, prophylactic laser iridotomy should be performed. Definitive treatment consists of cataract extraction (9-13)

2) Glaucoma induced by Lens-dislocation

Secondary angle-closure glaucoma may occur if the lens is displaced from its normal anatomical position (Ectopia lentis). Forward displacement of the lens may result in narrowing of the anterior chamber angle and pupillary block causing secondary angle-closure. Ectopia lentis may be present as an isolated clinical entity secondary to trauma or associated with systemic disorders such as Weill-Marchesani syndrome, Marfan's syndrome, homocystinuria, among others. The biochemical defects in these conditions result in defective lens fibers (zonular fibers that suspend the lens in position), often causing subluxation/dislocation of the lens, thus increasing the risk of secondary angle-closure (6, 13-17)

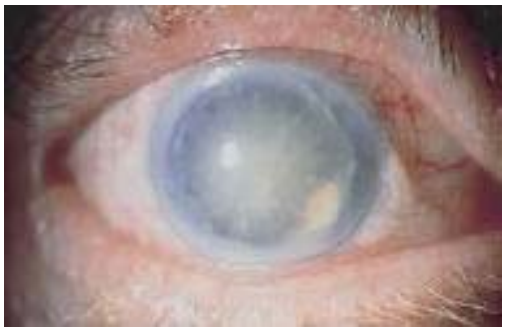

Fig : Anterior dislocation of lens. Leading to secondary angle closure glaucoma

Diagnosis : The clinical presentation of Ectopia lentis varies according to the individual state of the lens but when dislocation of the lens results in angle-closure and pupillary block, patients usually present with a painful eye, decreased visual acuity, and history of difficulties with accomodation and near vision. Diagnosis is made by the presence of a dislocated/ subluxated lens, angle-closure, and intraocular pressure elevation in the affected eye (7, 13, 14)

Management : The Management of glaucoma induced by ectopial lentis depends on the degree of lens dislocation and the presence of pupillary block. In cases of partial subluxation without pupillary block, conservative treatment with intraocular pressure monitoring could be followed. If pupillary block is present, a laser iridectomy is usually indicated. When total anterior dislocation occurs, removal of the lens is the definitive treatment (7, 13-14)

Conclusion

Lens induced glaucoma comprises a number of different glaucomatous process where human crystalline lens. There a common role in raising IOP. Early diagnosis and treatment of cataract is very important as delayed treatment result poor visual outcome. Even irreversible loss of vision.

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Medical Management of Uveitic Glaucoma

M.A. Mian[1]

Abstract

Raised intraocular pressure is a common and frequently serious complication of anterior uveitis. The milieu of inflammatory cells, the mediators they release, and the corticosteroid therapy used to treat the uveitis can participate in the pathogenesis of uveitic glaucoma. These factors alter the normal anatomic structure of the anterior chamber and angle, influencing aqueous production and outflow. These changes act to disrupt the homeostatic mechanisms of intraocular pressure control. Structural changes in the angle can be acute, such as in secondary angle closure with pupillary block glaucoma, or chronic, such as combined steroid-induced and secondary open angle glaucoma.

Management of uveitic glaucoma may be difficult because of the numerous mechanisms involved in its pathogenesis. Diagnostic and therapeutic decisions are guided by careful delineation of the pathophysiology of each individual case. The goal of treatment is to minimize permanent structural alteration of aqueous outflow and to prevent damage to the optic nerve head. This article reviews the pathogenesis of uveitic glaucoma, with specific attention to etiology and medical management, with emphasis on the more recent developments in each category.

Key words : ocular hypertension, trabeculitis, heterochromiccyclitis, herpetic uveitis, steroid responder.

Introduction

Uveitic glaucoma is diagnosed when uveitis is associated with elevated intraocular pressure causing glaucomatous field loss and/or glaucomatous field damage. If unrecognized or untreated this condition may lead rapidly to blindness. It was first reported by Joseph Beer in 1813,subsequently by Desmans in 1821 and Mackenzie in 1830.

Glaucoma may occur in 20% of all uveitis patient. It may affect patients of any age group. In Asian people,the usual causes are Fuch'sheterochromic uveitis, herpetic eye disease and glaucomatocycliticcrisis. It is rare for HLA B27 associated uveitis. It is a condition with self-limited recurrent episodes of markedly elevated intraocular pressure (IOP) with mild idiopathic anterior chamber inflammation. It is most often classified as secondary inflammatory glaucoma. Fluctuation of IOP, ciliary body shutdown, multiple mechanism of intraocular pressure (IOP) elevation, assessment of glaucomatous damage hampered due to miotic pupil or hazy media-these are the diagnostic dilemmas of uveitic glaucoma. Posterior uveitis is less likely to affect the aqueous outflow pathway and consequently less likely to lead to secondary glaucoma.

Successful management of uveitic glaucoma depends on recognition of uveitic syndrome and clarification of the mechanism that contributing to glaucoma.Both inflammation and raised intraocular pressure require treatment. Uveitic ocular hypertension is common and must be differentiated from Uveitic glaucoma.

Uveitic Glaucoma Vs Ocular Hypertension

Uveitic ocular hypertension refers to intraocular pressure (IOP) 20 mmHg or greater above baseline without evidence of glaucomatous optic nerve damage. Uveitic glaucoma is defined as elevated intraocular pressure (IOP) resulting in progressive neuroretinal rim loss and/or development of typical, perimetric, glaucomatous field defects.

Pathology and Pathogenesis of Uveitic Glaucoma

Uveitic glaucoma maybe closed angle with or without pupillary block. It may also be open angle, due to acute anterior uveitis caused by

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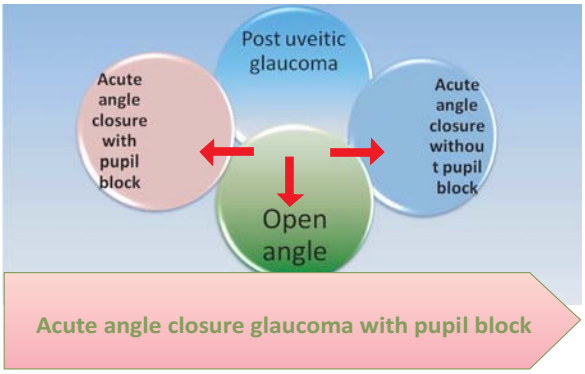
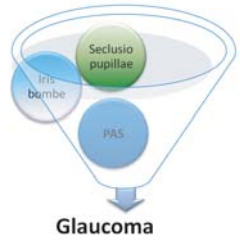
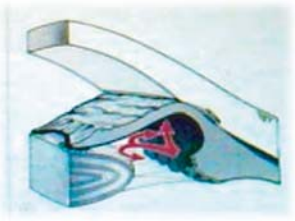
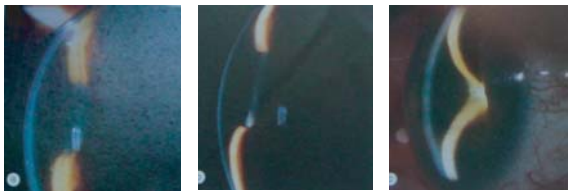
acute trabeculitis or trabecular obstruction. In chronic anterior uveitis these secondary glaucoma is due to trabecular scarring or sclerosis secondary to chronic trabeculitis.

Chronicity, age, corticosteroids, Juvenile Idiopathic arthritis, and ANA positive uveitis without evidence of arthritis – are the main reasons for uveitic glaucoma. Seven reasons increase intraocular pressure (IOP) in uveitis, these are- Clogged Trabecular meshwork, Inflammed Trabecular meshwork, Damaged Trabecular meshwork from chronic uveitis, Peripheral Anterior Synechiae, Posterior Synechiae and Steroid therapy. Clogged trabecular meshwork is due to white blood cells entering into spaces of Trabecular meshwork. It is diagnosed when there is an increase in intraocular pressure (IOP) in very red, hot uveitis without synechia. Inflammed trabecular meshwork is due to trabeculitis causing decrease in outflow capability. It is the most likely mechanism for Posner-Schlossman Syndrome. It is diagnosed when uveitis is very mild, no synechia, no treatment yet, and quite high intraocular pressure (IOP). Damaged Trabecular meshwork from chronic uveitis, is the type of increased intraocular pressure (IOP) seen in Fuch'sHeterochromiccyclitis, diagnosed in mild chronic uveitis without synechia. Peripheral anterior synechiaeCan cause chronic increase in intraocular pressure (IOP). It has also potential to close angle which can be diagnosed with gonioscopy. It will need chronic treatment of intraocular pressure (IOP) reduction. Posterior synechiae can cause an attack of acute glaucoma which needs peripheral iridectomy or iridotomy. intraocular pressure (IOP) can be increased due to steroid therapy specially in individuals who are corticosteroid responder.









DIAGNOSIS : Before going to actual management, detailed history and symptoms, visual function specially perimetry, Slit-lamp biomicroscopy, Etiology clues, Tonometry, Gonioscopy, Fundus biomicroscopy, UBM, anterior segment OCT can be done for proper evaluation and appropriate management of the patient. For accurate diagnosis of uveitis, details uveitis workup including Complete Blood Count and differential count, HLA typing, Erythrocyte Sedimentation Rate and C reacting protein, Acetylcholine Converting Enzyme, lysozyme, Chest X-Ray, Mantoux test, FTA ABS, VDRL, Titers for toxocariasis and toxoplasmosis should be done.

To confirm aetiology, during trabeculectomy and peripheral iridectomy the excised trabecular block should be submitted for histopathology,

immunohistochemical and microbiological study.

Treatment Goals in Uveitic Glaucoma : Treatment of uveitic glaucoma is a challenging job. The aim is to reduce pain, redness, photophobia, prevent sequelae, i.e. synechiae, Cystoid Macularedema, cataract as well as to control intraocular pressure and halt the progression of optic neuropathy.

Medical treatment

Intensive corticosteroid therapy is the main stay to treat acute uveitis. Strong cycloplegics is also essential for treatment. Steroid therapy may increase intraocular pressure (IOP), either by reducing inflammation and improving aqueous production or by decreasing outflow. Miotic agents should be avoided because they may aggravate the inflammation and cause posterior synechiae. Prostaglandin analogs may exacerbate inflammation in poorly controlled uveitis. Steroid-induced ocular hypertention rarely occurs before 3 weeks after initiation of cortecosteroid therapy. Early intraocular pressure (IOP) elevation with inflammation are almost always due to inflammation that require aggressive treatment. When the inflammation becomes quiet but intraocular pressure (IOP) is 30mmHg or higher, topical steroids may be slowly tapered and aqueous suppressants may be added. When steroid is contra indicated immunosuppressives should be taken into consideration with prior consultation with a medical specialist.

Undertreating uveitis with corticosteroids to minimize intraocular pressure (IOP) elevation at the expense of good control of inflammation is a false economy. Medical therapy is given with-Beta-blockers, Carbonic Anhydrase inhibitors and Adrenergic agonists. In this situation beta blockers are the first line of drug. Prostaglandin analogues and Miotics should be avoided in uveitic glaucoma. Medical control is achieved if angle is completely open, no peripheral anterior synechiaeor pigment deposit is present in the angle. Medical therapy should be initiated to reduce intraocular pressure and to control uveitis to make the patient suitable for the appropriate surgery.

Laser treatment, Laser peripheral iridotomycan be done in case of pupillary block angle closure glaucoma. Incisional glaucoma surgery eg. Trabeculectomy with MMC and in refractory cases glaucoma drainage device (GDD) may be considered with satisfactory result.

Conclusion

Elevation of intraocular pressure secondary to intraocular inflammation frequently presents a diagnostic and therapeutic challenge. In uveitic glaucoma the patient first develops uveitis, either due to trauma, systemic disease or idiopathically. The ensuing inflammation results in a rise in intraocular pressure (IOP) through several mechanisms. Untreated, the patient will eventually experience glaucomatous optic atrophy, or possibly central retinal artery occlusion. Successful management of uveitic glaucoma involves prompt and aggressive measures. Pilocarpine and other miotics are contraindicated in inflammatory glaucoma. Miotics will induce ciliary spasm and increase inflammation, fostering both posterior and peripheral anterior synechiae. Medical control of intraocular pressure (IOP) is more likely to be achieved if the angle is completely open. Intraocular pressure lowering effect of ocular hypotensive drug is less predictable in uveitic glaucoma.

Steroids are absolutely necessary to manage inflammatory glaucoma. Withholding steroids in

inflammatory glaucoma is extreme mis-management. Strong cycloplegia is necessary in managing uveitic glaucoma. We should avoid miotics and the prostaglandin analogue. We should keep in mind to treat the inflammation first and the intraocular pressure (IOP) secondarily.

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Enthusiastic use of Ahmed Glaucoma Valve – A case report

Z. S. Shahid[1], H. Rahman[2], S A Nahid[3], M. Shirajul Islam[4]

Abstract :

Background : To report a case in which glaucoma drainage device had been implanted without proper indication.

Method : A man of 60 yrs. old came to OPD of a tertiary hospital for follow up. He had undergone phacoemulsication with Ahmed Glaucoma Valve Implantation in his left eye for cataract & Glaucoma. In his right eye he has cataract with Yag-Laser peripheral irridotomy for angle closure glaucoma. We examined his eyes meticulously and tried to justify valve implantation as a primary surgery.

Introduction

Glaucoma drainage devices are designed to divert aqueous humor from the anterior chamber to an external reservoir, where a fibrous capsule forms about 4-6 weeks after surgery and regulates flow. These devices have shown success in controlling intraocular pressure (IOP) in eyes with previously failed trabeculectomy and in eyes with insufficient conjunctiva because of scarring from prior surgical procedures or injuries. They also have demonstrated success in complicated glaucomas, such as uveitic glaucoma, neovascular glaucoma, and pediatric and developmental glaucomas, among others.

Since the introduction of the first glaucoma drainage device, Molteno implant, various modifications of the original design and improvements in surgical techniques over the past 40 years have led to greater success and lower complication rates. In addition, other glaucoma drainage devices have been introduced and offer unique features designed to facilitate implantation, improve IOP control, and reduce acute postoperative hypotony.

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Types

Professor Anthony Molteno developed the first glaucoma drainage implant, in Cape Town in 1966. [2] Following on the success of the Molteno implant, severl varieties of device have been developed from the original, the Baerveldt tube shunt, or the valved implants. These are indicated for glaucoma patients not responding to maximal medical therapy, with previous failed guarded filtering surgery (trabeculectomy). The flow tube is inserted into the anterior chamber of the eye and the plate is implanted underneath the conjunctiva to allow flow of aqueous fluid out of the eye into a chamber called a bleb.

The Express Mini shunt is a newer, non-valved device that was originally designed to provide a direct conduit from the anterior chamber to the sub-conjunctival space or bleb. In this position it was unstable and tended to erode through the conjunctiva. Now the more common use is as a modification of the trabeculectomy procedure, placed under a scleral flap, replacing the sclerostomy step (see trabeculeculectomy).

In comparison to the glaucoma drainage devices that use an ab externo procedure, ab interno implants, such as the Xen Gel Stent, are transscleral implants to channel aqueous homor into the non-dissected Tenon's space, creating a subconjunctival drainge area similar to a bleb.3,4 The implants are transscleral and different from more other ab interno implants that do not create a transscleral drainage, such as iStent, CyPass, or Hydrus.5

Indications

The glaucoma valve implant is indicated for glaucoma patients not responding to maximal medical therapy with previous faild guarded filtering surgery (trabeculectomy) or in cases where conventional drainage surgery in unlikely to succeed. Common situations where the of a glaucoma implant as a primary procedure in indicated include

1. Neovascular glaucoma - glaucoma associated with vascular disease of the eye (often diabetes).
2. Inflammatory Glaucoma
3. Traumatic glaucoma - glaucoma associated with injury to the eye.
4. ICE syndrome
5. Open angle glaucoma with failed trabeculectomy
6. Silicone glaucoma - glaucoma due to Silicone used to repair a detached retina.
7. Refractory Congenital Glaucoma-
8. PKP with glaucoma –
9. Aphakic glaucoma

Contra indications

There are some relative contraindications-

1. Patient unable to comply with self care in the post operative period.

2. Borderline Corneal endothelial function

Surgical technique

The flow tube is inserted into the anterior chamber of the eye and the plate is implanted the conjunctiva to allow flow of aqueous fluid out of the eye.

The first-generation Molteno and other non-valved implants sometimes require the ligation of the tube until the bleb fromed is mildly fibrosed and water-tight.7 This is done to reduce postoperative hypotony-sudden drops in postoperative intraocular pressure (IOP).

Valved implants valve. Studies show that in severy cases of glaucoma, double plate Molteno implants are associated with lower mean IOP in the long term compated to the Ahmed glaucoma valve.8

Second and third generation Molteno implants incorporate a biological valve and studies show considerable improvement in postoperative outcome over the older style Ahmed and Molteno implants.

Baervedle shunts (non-valved) have been shown to have lower rate of surgical failure than Ahmed shunts (valved), 9-10 this have may be related to the high rate of valve failure, or to the larger plate surface of the Baerveldt shunt.

Complications

The majority of complication occur shortly after surgery. These are generally related to high pressure (due to inflammation following surgery) or low pressure (too much aqueous flow through the tube). Periods of low pressure which are more associated with non-valved shunts, can cause retinal detachments, maculopathy of haemorrhages. Periods of high pressures, which are more association with valved shunts, are detrimental to the optic nerve.

Long term complications of this surgery include diplopia and corneal oedema.

There are also device related complications, which will require surgical revision e.g. erosion, where the conjuctiva dissipates over the shunt leaving it exposed, the condition of which may be revised or prevented in advance by the use of amniotic membrane,1 donor patch grafts,11 or collagen matrix implant; 11-12 blockage, where a particle becomes logged in the tube line blocking flow, retraction, where the tube line slip out of correct position such that flow is inhibited or halted, valve failure, where the valve stops working blocking flow completely-this will require the device to be replaced.

Surgical failure due to the ongoing scarring over the conjunctival dissipation segment of the shunt may become too thick for the aqueous humor to filter through. This may require preventive measures using antifibrotic

medication like 5-fluorouracil (5FU) or Mitomycin-C (during the procedure) or creating a necessity for revision surgery with the sole or combilative use of biodegradable spacer of collagen matrix implant. [13]

Comparative studies of glaucoma drainage devices and trabeculectomy

Wilson et al. compared short and intermediate results of trabeculectomy and AGV in a randomized clinical trial and reported statistically lower mean IOP with trabeculectomy than with AGV at week 6-15 and months 11-13. The cumulative probability of success was 83.6% for the trabeculectomy group and 88.1% for the AGV (P=0.43). There was no significant difference in complication rates between the two groups, but the AGV group required more glaucoma medications postoperatively. The same investigators subsequently reported the long-term results of these two procedures (64). The cumulative probabilities of success at months 41-52 were 68.1% for the trabeculectomy group and 69.8% for the AGV group (P=0.86). Adjunctive medication requirement was also comparable in both groups with longer follow-up.

Our Case

Patient named Mustafa Kamal age 60 yrs. Came to a tertiary care hospital for check up with no ocular complain on 8/7/2011.

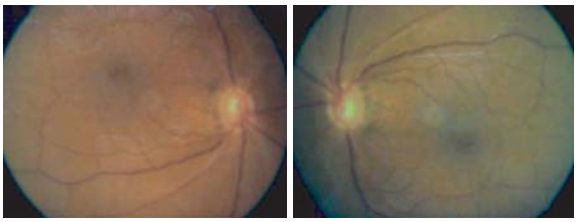
He had a past history of Yag PI both eye at Shankara Netraloya, India one year back. He had undergone phaco emulsification and PCIOL implant with AGV implant in his left eye six month back in Singapore. He also give the history of stay in the hospital for one and half month after operation. He has positive family history of glaucoma (father)

He was using lumigae Eye drop once daily in his right eye & predfort eye drop thrice daily in left eye.

On examination his visual acuity is right eye 6/36 with pinhole 6/9. Left eye 6/24 with pinhole 6/9. IOP was 13 mm Hg in his right eye and 14 mm Hg left eye, conjunctiva was healthy in right eye and in left eye there was a huge bleb in supero temporal quadrant.

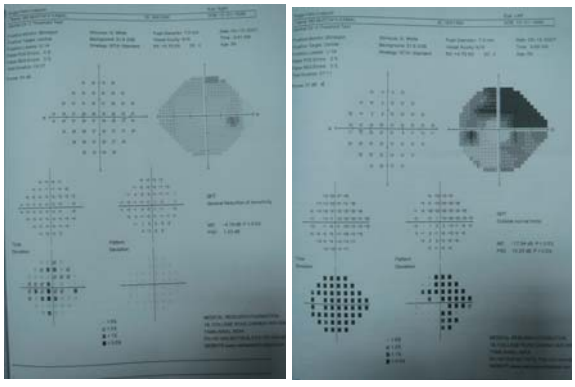
Right eye **Left eye**

Cornea was clear in both eyes. AC was slightly shallow in right eye and deep with containing tip of AGV in left eye. Yag PI patent in both eyes. Pupil round regular and reacting in right eye and slightly larger left eye with RAPD. Lens showed nuclear sclerosis in right eye and preudophakia left eye.



Right eye **Left eye**

C/D ratio 0.6 in right eye and in left eye 0.75 with inferior notching. HVF, R/E normal and L/E showed presence of glaucomatous field defect. OCT, R/E Normal and left eye showed thinning of RNFL.



Right eye **Left eye**

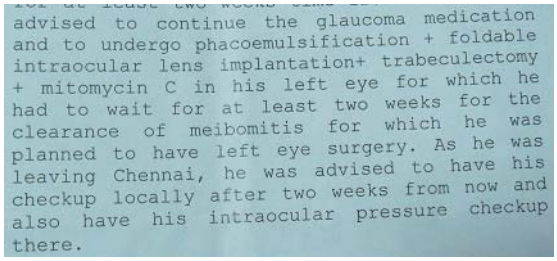
Our diagnosis was PACG with S/P PI in both eyes with nuclear sclerosis in right eye & pseudophakia with S/P AGV implantation in left eye. The surgical options of these patients were-

1. Trab + phaco + PCIOL ± Antimetabolites

2. Trab + ECCE/SICS+PCIOL ± Antimetabolites

3. Phaco + PCIOL

The patient was seemed in Sankara Netralaya, India. They advised-


advised to continue the glaucoma medication and to undergo phacoemulsification + foldable intraocular lens implantation+ trabeculectomy + mitomycin C in his left eye for which he had to wait for at least two weeks for the clearance of meibomitis for which he was planned to have left eye surgery. As he was leaving Chennai, he was advised to have his checkup locally after two weeks from now and also have his intraocular pressure checkup there.

Glaucoma Valve implantation is becoming popular overtime but it should be reserved for second surgery. First choice of valve implantation should be restricted for neuvascular Glaucoma, inflammatory glaucoma, refractory congenital glaucoma, post PK glaucoma, failed trabeculectomy etc. In this case the surgeon of this patient enthusiastically used AGV that should not be- we think.

Conclusion

Glaucoma drainage devices have been successful in controlling IOP in eyes with previouslyfailed trabeculectomy and difficult glaucomas, Since their introduction over 40 years ago, numerous modifications in design and improvements in surgical technique have enhanced clinical outcomes and minimized complications. The decision to choose a particular type of drainage device depends on a patients underlying characteristics in terms of preoperative IOP and optic nerve status, desired long-term IOP control, and the surgeon's comfort and preference. Careful preoperative screening and planning along with meticulous surgical technique helpminimize postoperative complications. There are many guideline from EGS, PPP, APGIS for us to make a justified decision.

We must be careful while choosing the patient with proper indication of implant as this is related with patients finance also. Any work we do it should be justified.

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