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Anti VEGF in Glaucoma!

M. N. Islam[2]

It has been well accepted that Neovascular Glaucoma needs treatment by Anti VEGF (vascular en-dothelial growth factor). This class of anti-angiogenic agents not only arrests the vascular endothelial cell proliferation and prevents vessel growth, but also induces regression of existing vessels by increasing endothelial cell death.

Bevacizumab, marketed as Avastin, is a humanized monoclonal antibody that inhibits vascular endothelial growth factor, is one of the most closely watched drugs in the world today. Ophthalmologists are both enthused to have it in their armamentarium and concerned that its "miracle drug" reputation is too optimistic.

In patients who present with neovascular glaucoma, the immediate injection of 1.25 mg of bevacizumab into the vitreous before pan-retinal photocoagulation (PRP), results in a significant regression of iris new vessels within two days. In the early stages of the disease, many patients have concomitant decrease in intraocular pressure (IOP) lasting several months. If no permanent PAS, new vessel regress in the angle and IOP usually decreases.

In February 2004, the Food and Drug Ad-mi-ni-stration in USA ap-proved bevacizumab, for intravenous administration in patients with colon cancer, in combination with 5-fluorouracil-based che-motherapy regimens. Bevacizumab is a recombinant humanized monoclonal immunoglobulin G1 antibody directed against VEGF. Intravitreal bevacizumab injection has shown encouraging results in the treatment of wet type of age-related macular degeneration, Branch and Central Retinal Venous Occlusions, Severe Proliferative Diabetic Retinopathy, Diabetic Macular oedema and iris neovascularisation. Topical or sub conjunctival application of bevacizumab was found to be effective for inhibiting corneal neovascularisation. A recent pathological study demonstrated that neutralization of VEGF reduced vascularity and decreased scar formation during wound healing, showing that VEGF strongly influenced scar tissue formation.

In this volume of JBGS, Zakia Wadud, Bibekananda Biswas & Maliha Sharmin has written on "Evaluation of Efficacy of Sub Conjunctival Bevacizumab in Augmenting Trabeculectomy for Glaucoma". They have studied sixteen individuals (9 females; 7males) with a diagnosis POAG or PACG. Several studies have shown that trabeculectomy with sub conjunctival bevacizumab injection resulted in successful IOP control.

As we know, Trabeculectomy remains the main surgical technique of all types of Glaucoma surgery. However, long term surgical success of trabeculectomy is often limited by scar formation and fibrosis in the process of wound healing. Unlike most surgical procedures, surgical success of glaucoma filtering surgery is achieved through the inhibition of wound healing. Modulating the wound healing process to reduce scar formation around the scleral flap and thereby inhibiting obstruction of filtration tract, plays a crucial role in the success of glaucoma filtering surgery.

The initial steps in wound healing are inflammation and coagulation, leading to a cascade of biological events including cellular, hormonal, and growth factor release. These events finally lead to scar tissue formation. Antimetabolites such as mitomycin C (MMC) or 5-fluorouracil (5-FU) are widely used to reduce wound healing by inhibiting fibroblast proliferation in trabeculectomy. The use of antimetabolites results in relatively avascular filtration blebs with less fibrovascular scarring

and an increased success rate. However, because of their non-specific mechanism of action, these agents can cause widespread cell death and apoptosis, resulting in potentially sight-threatening complications such as severe postoperative hypotony, bleb leaks and endophthalmitis. Thus, alternative antifibrotic agents that are more efficacious and safer are needed to optimize the healing response. Glaucoma filtering surgery entails fashioning an external filter for aqueous drainage, and a prerequisite to its optimum functioning is a patent filtering bleb. Since fibroblast function and growth of new vessels is a component of healing of the bleb, there have been attempts to retard this healing by the use of bevacizumab.

When glaucoma surgery-Trabeculectomy is planned, the adjunctive use of bevacizumab may be beneficial in NVG which has been published in different studies. Chen et al demonstrated that, in patients with NVG who underwent trabeculectomy, adjunctive IVB improved final visual acuity and decreased the rates of intra- and postoperative complications, including hyphema. Another retrospective study by Ma et al evaluated the adjunctive use of IVB during the implantation of an Ahmed Glaucoma Valve in NVG. The investigators found similar success and complication rates as with those patients who did not receive bevacizumab. The ideal dose of injections for NVG is unknown, although Gupta et al found no difference when 1.25 versus 2.5 mg bevacizumab was injected intracamerally during trabeculectomy with mitomycin C for NVG.

Dr. Kahook and his team at the University of Pittsburgh first reported treatment of neovascular glaucoma with Avastin, which is now, perhaps ironically, the standard of care for that problem. The same team was also first to use it as a wound modulator after trabeculectomy surgery, in lieu of 5-FU and mitomycin.

In my study of more than 100 cases of Trabeculectomy I used Sub Conj Bivacizumab around the bleb immediately after operation. My cases are still under study, but I found many cases with succulent bleb with long time decrease of IOP and very few post op complications comparative to per op use of 0.3% MMC sponge for one minute.

Recent advances in understanding the mechanisms of angiogenesis have facilitated the development of new treatment options for neovascular ocular diseases and anti fibrotic agents in trabeculectomy surgery. I do hope this majic drug will be available even in drop forms so that very comfortably our patients will get these benefits and help to prevent blindness .

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Effect of Nd Yag Laser Iridotomy to Reduce Intraocular Pressure in Iris Bombe

M.S.I. Prodhan[1], Z.S. Shahid[2]

Abstract

Purpose : The aim is to determine the efficacy of Nd YAG LASER iridotomy on intraocular pressure decrease in secondary glaucoma caused by iris bombe in patients with chronic uveitis.

Methods : A prospective study conducted at National institute of ophthalmology & hospital, Dhaka during the period of July 2012 to July 2015. Total 56 patients were enrolled with iris bombee due to ring synechia and increased intraocular pressure. Patients with active anterior uveitis were excluded from the study. Patients were examined meticulosly with slit lamp. Intraocular pressure was measured with Goldman applanation tonometer before and after iridotomy. Gonioscopy was done with 3-mirror Gonioscope.

Results : The study group consisted of 56 patients, 36 male and 20 female, mean age 32.5 years. Fifty six Nd YAG LASER iridotomy (Nidek ophthalmic laser) were performed in 56 patients. We used multiple hits, power 8 mj/pulse in 1-2 pulses per shot in 5 attempts. Iridotomies were performed at the periphery of the iris in the range from 11 o'clock to 1 o'clock position preferably at maximum bulging. Intraocular pressure was measured before and after Nd YAG LASER iridotomy by applanation tonometer. The average value of intraocular pressure before and after Nd YAG LASER iridotomy was 32.5 mmHg and 17.5 mmHg respectively. The result was statistically significant.There were 11 repititions of the iridotomies due to frequent recurrences of uveitis leading to blockage of prepared Nd YAG LASER iridotomy with fibrin material.

Conclusions : Nd YAG LASER iridotomy reduces intraocular pressure significantly in patients with secondary glaucoma with chronic uveitis. It is the method of choice in the treatment of increased intraocular pressure in iris bombe.

Key words : YAG laser iridotomy, iris bombe, IOP.

Introduction

Elevation of intraocular pressure secodary to intraocular inflammation frequently presents a diagnostic and therapeutic challenge. The elevation of intraocular pressure may be transient and innocuous, or persistent and severely damaging. The prevalence of secodary glaucoma increases with chronicity and sverity of disease. Secodary angle closure glaucoma is caused by posterior synechiae extending for 360 (seclusio pupillae) which obstract aqueous flow from posterior to the anterior chamber. The resultant increased pressure in the posterior chamber produces anterior bowing of the peripheral iris (iris bombe) resulting in shallowing of the anterior chamber and apposition of the iris to the trabeculum and peripheral cornea[1,2,3]. Laser iridotomy is performed to re-establish communication between the posterior and anterior chambers in eyes with pupillary-block angle-closure glaucoma. In 1956, Meyer-Schwickerath first reported the use of light energy to create a hole in the iris[4]. With the introduction of lasers in the 1960s, investigation of this modality continued, primarily with ruby lens. After the advent of argon laser technology in 1970, several reports of successful argon laser iridotomy appeared in the literature. During the 1980s, Nd: YAG laser is introduced for laser iridotomy. Now Nd: YAG laser is popular for doing peripheral iridotomy in primary angle closure glaucoma and iris bombe in secondary glaucoma[5, 6, 7].

Methods

A prospective study conducted in National institute of ophthalmology & hospital, Dhaka during the period of July 2012 to July 2015. Total 56 patients of iris bombee with increased intraocular pressure due to ring synechia were enrolled. Age of the patient was between 15 to 50 years. Among 56 patients 36 were male and 20 were female. Patients with active anterior

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uveitis were excluded from thestudy. Careful history and meticulous examination with slitlamp was done. Intraocular pressure was measured with Goldman applanation tonometer. Gonioscopy was done with 3- mirror Goniolens.

Results

The study group consisted of 56 patients, 36 male and 20 female, mean age 32.5 years. Fifty six YAG LASER iridotomy (Nidek ophthalmic laser) were performed in 56 patients. We used multiple hits, power 8 mj/pulse in 1-2 pulses per shot in 5 attempts. Iridotomies were done at the perphery of the iris in the range from 11 o'clock to 1 o'clock position preferably at maximum bulging. Intraocular pressure was measured before and after YAG LASER iridotomy by applanation tonometer. The average value of intraocular pressure before and after YAG LASER iridotomy was 32.5 mmHg and 17.5 mmHg respectively. There were 11 repititions of the iridotomies due to frequent recurrences of uveitis leading to blockage of prepared YAG LASER iridotomy with fibrin material.

Fig. I. Iris bombe

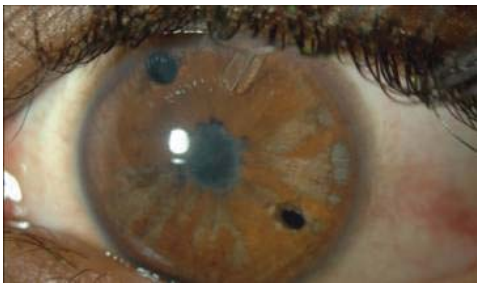

Fig. II. After LPI

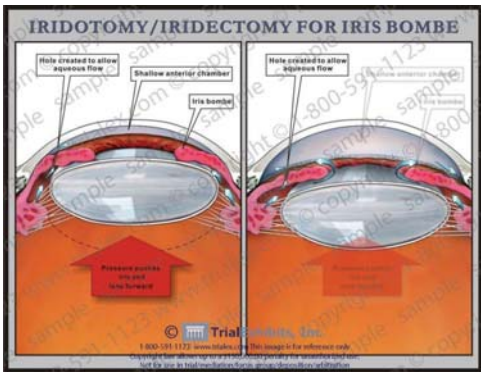

Fig. III. How LPI reduces IOP

Table-I : Sex ditrbution of patients

Sex	No. of patients	Percentage (%)
Male	36	64.29
Female	20	35.71
Total		100

Table-II : Age distribution of patients

Age in years	Male	Female	Total	Percentage (%)
15-25	09	04	13	23.22
26-35	16	09	25	44.64
36-50	11	07	18	32.14

Table-III : IOP before iridotomy

Age in years	Male	Female	Total	Percentage (%)
IOP 20-25 mmHg	08	05	13	23.22
IOP 26-35 mmHg	18	11	29	51.78
IOP 36-45	10	04	14	25

Table-IV : IOP after iridotomy

Age in years	Male	Female	Total	Percentage (%)
IOP 10-15 mmHg	10	06	16	28.57
IOP 16-20 mmHg	19	10	29	51.79
IOP 21-25 mmHg	07	04	11	19.64

Table-V: IOP reduction rate

	IOP	Reduction Percentage%
Average IOP before iridotomy	32.5 mmHg	53.84
Average IOP after iridotomy	17.5 mmHg	

Discussion

Chronic anterior uveitis with ring synechiae results in iris bombe. Iris bombe causes peripheral anterior synechiae results in uveitic glaucoma. Raised IOP due to iris bombe endangers the optic nerve function. This results in reduced visual acuity. Iris bombe is a condition in which there is apposition of iris to the lens or anterior vitreous, preventing aquous humour flow from posterior to anterior chamber. The pressure in the posterior chamber rises, resulting in anterior bowing of the peripheral iris and obstruction of the trabecular meshwork. This may result in an acute attack of secondary angle cosure glaocoma. Nseudophakia Soemmering ring sometime causes pupillary block resulting in iris bombe[8]. Iris bombe causes ocular pain and mild to severe decrease of vision. Diagnosis of iris bombe was done by slitlamp examination. Peripheral iris bulge forward and occlude anterior chamber angle. Gonioscopy was done and found angle closure in different grade. All these patients were treated previously with maximum tolerable antiglaucoma and antiinflammatory drugs. But IOP was not reduced signifiifantly. Visual acuity was gradually decreasing due to raised IOP. After peripheral iridotomy IOP was reduced significantly (53.84%) and visual acuity improved. This result simulates with the result of Micic N Eur J ophthal[9].

Conclusions

YAG LASER iridotomy reduces intraocular pressure significantly in patients of iris bombe with secondary glaucoma. It is the method of choice in the treatment of increased intraocular pressure in iris bombe.

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Juvenile open-angle glaucoma- Management and outcome at a tertiary ophthalmic center

S. M. Noman[1], N. C. Dey[2]

Abstract

Background : For the documentation and describe clinical manifestations management and outcome of management of the patients diagnosed as Juvenile open-angle glaucoma at the glaucoma department, CEITC, Chittagong, Bangladesh.

Method : This is a hospital based prospective observational case series review. 20 patients who were diagnosed as Juvenile open-angle glaucoma from November 2008 to December 2010 were included in this study.

Patient particulars, history with main causes of hospital presentations were recorded. Ophthalmic examinations and management given were documented. Similar relevant details were recorded for different follow-up periods.

Results : 40 eyes of 20 patients were included in this study. There were 16 male and 4 female. All cases were bilateral. Age more than 18yrs. (18-35) in 16 patients and below 18yrs. (5-18) in 4 patients. 15 patients came from rural area and 5 patients from urban. Pretreatment average IOP in the both eyes was 32±3mmhg, which was 15±1mmhg after treatment. 24 of 40 eyes were presented with advance field defects. 85% (17 patients) had myopic refractive error. In 18eyes pre treatment presenting visual acuity was $<$6/60 and $>$6/60 in the rest of the eyes. Visual acuity was improved after treatment. In 21 patients (53%) IOP was controlled with 2-3 medications. In 19 eyes (48%) IOP was controlled with filtration surgery.

Conclusion : As Juvenile open-angle glaucoma presented with high IOP and advance field defect, early diagnosis, appropriate investigations and medical or surgical management is mandatory to stabilize IOP and to prevent progression of field defects.

Introduction

Juvenile open-angle glaucoma (JOAG) which has an age at onset of (5-35) years tends to be more aggressive. It is usually resistant to medical therapy and is associated with more severe visual impairment than primary open angle glaucoma[1].

Identifying risk factor are important because this information may lead to development of strategies for disease screening and prevention and may be useful in identifying persons for whom close medical supervision is indicated. Thick compact tissue in the angle represents an immature development of the trabecular meshwork and may be one of the primary cause of increase intraocular pressure in Juvenile glaucoma[2] the more extensive the immaturity, the earlier the glaucoma will become manifest. GLCIA, the first open angle glaucoma gene, was initially mapped in a large Juvenile glaucoma family that localized to chromosme[1] the mutation in the gene, which are suspected to be responsible for open angle glaucoma, produce a protein, myocilin that is induced in trabecular meshwork.

Method

This was hospital based combined non concurrent and concurrent prospective cohort study of all cases presenting to the glaucoma clinic with a diagnosis of Juvenile open-angle glaucoma. Cases were identified throughout a two years period from November 1st 2008 to December 1st 2010.

All patients were reviewed by a single consultant. Details of history included the biographical details of patients (age, gender, address etc) and history of presentation.

Ophthalmic examination was done on patients and examination details included visual acuity (VA); intra-ocular pressure (IOP) measurement by Goldmann Applanation Tonometer; gonioscopic findings by Goldmann 2-mirror

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contact goniolens; fundoscopic findings and any other notable ocular findings. The method of management was recorded.

For previously diagnosed patients, their medical records were retrieved and relevant data were extracted and asked to come for follow-up as necessary. Newly diagnosed patients were duly processed and asked to return for future follow-up visits. At least three follow-up data were recorded, 1 month after diagnosis of joag, then 3 months and 6 months. On all visits ophthalmic examination was done by the same consultant.

After collection of data, they were then tabulated and analyzed. Outcomes of management were assessed mainly with regards to IOP control. Statistical analysis was done using SPSS v.13.

Results

A total number of 40 eyes of 20 patients were encountered during the study period. All of the cases were bilateral affected of these 10 were newly diagnosed and 10 were previously diagnosed. The ages of 16 patients (80%) were between 18-30 yrs. And same of 4 patients were (5-18) yrs. Mean age (23±7.13) years. 16 patients (80%) were male and 4 were female. 50% of the patients are student. Remaining 50% were either service holder or businessman or daily laborers. 15 patients (70%) came from rural area. 65% of them were from middle class family and 35% of them from poor family. (90%) 18 patients came with gradual decrease vision in the both eyes and 2 patients (10%) came with only headache.

Mean duration of symptom was 2yrs. 8 patients (40%) had a strong family history of glaucoma. 50% of them were previously treated by local ophthalmologist. Reasons of delayed presentation were the lack of knowledge (60%), lack of eye care facilities (20%) and poor economy (20%).

17 patients (85%) had myopic refractive error, 1patient (5%) had hyperopia and the rest 2 patients had no refractive error. Average IOP at presentation was around 35mmhg which was reduced to around 15mmhg after treatment (P=1.440).

24 eyes had advance field defects like total field loss (5.26%), biarcuate scotoma (21.05%) and tubular field (31.59%). IOP was controlled with either 2 or 3 medications in 21 eyes (52.5%), those patients (47.5%) resistant to medical treatment needed filtration surgery to control IOP (table-5).

Presenting visual acuity was <6/60 in 18 eyes (45%) and 6/9-6/60 in 22 eyes. Post management visual acuity was improved (table-6).

Table I: Demographic features and presentation of the patients:

	N	Percent
Age group		
5-18	4	20
18+	16	80
Mean age	23 years SD ± 7.13 years.	
Gender		
Male	16	80
Female	4	20
Occupation		
Student	10	50
Service	3	15
Business	4	20
Daily labor	3	15
Patient's residence		
Rural	15	75
Urban	5	25
Socio economic condition		
Poor	7	35
Middle	13	65
Rich	0	0
Presenting complain		
Decreased Vision	18	90
Eye ache	2	10
Duration of symptom (mean time) –	02 year	
Family History		
No	12	60
Yes	8	40
Reason of delayed presentation		
Economic	4	20
Lack of eye care facilities	4	20
Lack of Knowledge	12	60
Previous eye treatment		
Yes	10	50
No	10	50

Table II : Status of Refractive Error

	N	Percent
Myopia	17	85.0
Hyperopia	1	5.0
No refractive error	2	10.0
Total	**20**	**100.0**

Table III : Management of Intraocular Pressure

IOP	Right	Left
Before treatment	33	35
After treatment	15	16
	P = 0.440	

Table IV : Visual Field test of patients

	N= 38	Percent
Nasal step	5	13.15
Bearcuate Scotoma	8	21.05
Tubular	12	31.58
Inferior Actuate Scotoma	2	5.26
Superior Actuate Scotoma	9	23.68
Total field loss	2	5.26
Total	38	100.0

Table V : Treatment of the patients

Medical treatment	N	Percent
Two Medications	15	37.50
Three medications	6	15.00
Surgical treatment		
Trabeculectomy	11	27.50
Trabeculectomy with MMC	8	20.00
Total	**40**	**100**

Table VI : Visual acuity of the patients

VA	Presenting VA	Post management VA
6/6-6/18	19 (47.50%)	25 (65.0%)
6/24-6/60	3 (7.50%)	3 (5.0%)
6/60+	18 (45.0%)	12 (30.0%)
Total	40 (100.0%)	40 (100.0%)

Discussion

Primary glaucoma represents a significant public health problem. Although rare, untreated Juvenile glaucoma patients are ultimately diagnosed as primary open angle glaucoma after 35yrs. It is an important cause of blindness in the western countries and in blacks[3]. It is also not uncommon in this subcontinent.

Kass and Becker were among the first to observe a strong correlation between family history and glaucoma[4,5]. Based on their observation, the researchers suggested that the most effective method of glaucoma detection would be to check family members. 40% of our patient had a strong family history of glaucoma. The percentage may be more as the rest of the patient did not know the cause of their relative's blindness.

Polanasky hypothesized that, mutations of the trabecular meshwork glucorticoids genes could cause elevated IOP. This is called TIGR protein or myocilin was identified in Juvenile open-angle glaucoma families. We did not do any genetic analysis in our patient.

Juvenile open-angle glaucoma terminology often used when open-angle glaucoma diagnosed at young age (typically 10-30yrs.)[3]. Mean age of our study populations is (23±7.13)yrs. So it is strongly similar to other studies.

Although primary glaucoma's are more common in female, male are predominant in our study populations. Sensitive patients whose visual perfection is a factor usually present in the clinic due to their visual problems. 50% of our patients are student who presented earlier than others.

Most of our patients are from rural middle or poor class families. This may be due to lack of awareness and lack of health care facilities at rural area. Poor economy and remoteness may also play role. In our study, main reason of delayed presentation is lack of knowledge about the disease.

Electron microscopy specimens of anterior chamber angle reveal thick compact tissue consisting of cells with fine processes and extra cellular substances. Thick compact tissue represents immature development[2]. We did not do any histopathology but gonioscopic examination shows abnormal processes over trabecular meshwork, concave iris insertion suggestive of immature development. Angle was open 360^0 areas in all patient.

Presentation of JOAG is aggressive. In this study all patients presented with high intraocular pressure (30-35)mmhg, increased C:D ratio) >.8:1 and with advance field defects.

Juvenile open-angle glaucoma is associated with more severe visual impairment than primary open angle glaucoma[8]. 45% of our case (eyes) presented with <6/60 vision. As 50% of our patients are students, they are visually sensitive and their presentation was quite earlier.

Aggressive Juvenile open-angle glaucoma is more resistant to medical therapy[8]. 47% (19eyes) of our cases were resistant to medical therapy; they were treated with 2-3 medications. Those patient who's IOP was not controlled even with two medications, filtration surgery was advised.

Trabeculectomy, a penetrating filtration procedure, is the treatment of choice in treating medically uncontrolled open angle glaucoma. However, intra operative and postoperative complications are not uncommon[9-11]. In our series, no intraoperative or post-operative complications occured.

The success rate of filtration surgery in young patient is believed to be lower than POAG.[12] To decrease the fibrovascular proliferation, we did 8 filtration surgeries with mitomycin c in relatively more advance cases.

Primary trabeculectomy in young adults may have a favorable outcome despite no antimetabolite therapy.[13] We also did 11 filtration surgery without antimetabolite which are still doing well.

Conclusion

Juvenile open angle glaucoma presents usually at an advance stage. Those patient who have strong family history of glaucoma, should do a routine periodic eye checkup. Even at an advance stage appropriate medical or surgical treatment can stop further progression of the diseases. Filtration surgery with mitomycin c is recommended for very advance cases to assure longtime functioning bleb.

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Evaluation of Efficacy of Sub Conjunctival Bevacizumab in Augmenting Trabeculectomy for Glaucoma

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Abstract

Purpose : To evaluate the efficacy of intraoperative sub conjunctival injection of Bevacizumab in augmenting trabeculectomy for glaucoma.

Design : Nonrandomised, prospective, interventional case series.

Participants : Sixteen individuals (9 females; 7males) with a diagnosis POAG or PACG, a recorded intraocular pressure (IOP) of more than 21 mmHg, glaucomatous damage on visual field or optic disc, and taking a maximum tolerated dose of IOP-lowering medication.

Intervention : Unilateral trabeculectomy with sub conjunctival bevacizumab (0.05 ml, 1.25 mg) adjacent to the bleb using a 30-gauge needle and tuberculin syringe administered immediately after trabeculectomy.

Main Outcome Measures : Intraocular pressure (IOP) was the primary outcome and secondary outcomes included number of antiglaucoma medications, complications and number of eyes achieving successful outcomes at 6 month follow-up.

Results : Mean age was 46.56 ± 5.58 years. The mean preoperative IOP was 28.5 ± 5.14 mmHg (range 18-34) and the patients were taking on an average of 2.81 ± 0.75 IOP-lowering medications (range 2-4). The mean postoperative IOP was 9.5 ± 1.9mmHg (range, 7–14 mmHg) on day 1, 10 ± 1.9 mmHg (range, 8–14 mmHg) on day 7, 11.5 ± 1.96 mmHg (range, 8–16 mmHg) at 1 month, 12.4 ± 1.31 mmHg (range, 10 –14 mmHg) at 3 months, and 12.9 ± 1.34 mmHg (range, 10 –14 mmHg) at the 6-month follow-up with no IOP-lowering medications. Preoperative best corrected visual acuity was 0.73 ± 0.34, whereas at 6 months after trabeculectomy, it was 0.74 ± 0.47.

Conclusion : Trabeculectomy with intraoperative sub conjunctival injection of Bevacizumab may offer a useful option for improving the outcome of filtering bleb.

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Introduction

Glaucoma is the second leading cause of blindness world wide.[1] Trabeculectomy remains the main surgical technique.[2] However, long term surgical success of trabeculectomy is often limited by scar formation and fibrosis in the process of wound healing.[3,4] Unlike most surgical procedures, surgical success of glaucoma filtering surgery is achieved through the inhibition of wound healing.[5] Modulating the wound healing process to reduce scar formation around the scleral flap and thereby inhibiting obstruction of filtration tract, plays a crucial role in the success of glaucoma filtering surgery.

The initial steps in wound healing are inflammation and coagulation, leading to a cascade of biological events including cellular, hormonal, and growth factor release. These events finally lead to scar tissue formation.[6] Antimetabolites such as mitomycin C (MMC) or 5-fluorouracil (5-FU) are widely used to reduce wound healing by inhibiting fibroblast proliferation in trabeculectomy. The use of antimetabolites results in relatively avascular filtration blebs with less fibrovascular scarring and an increased success rate.[7] However, because of their non-specific mechanism of action, these agents can cause widespread cell death and apoptosis, resulting in potentially sight-threatening complications such as severe postoperative hypotony, bleb leaks, and endophthalmitis.[8,9]Thus, alternative antifibrotic agents that are more efficacious and safer are needed to optimize the healing response.

Recent advances in understanding the mechanisms of angiogenesis have facilitated the development of new treatment options for neovascular ocular diseases. Neovascularisation (NV) occurs as a result of angiogenic stimuli,

including the vascular endothelial growth factor (VEGF).[10] Bevacizumab (Avastin®; Genentech, South San Francisco, CA, USA) is a recombinant humanized monoclonal immunoglobulin G1 antibody directed against VEGF.[11]Intravitreal bevacizumab injection has shown encouraging results in the treatment of age-related macular degeneration, severe proliferative diabetic retinopathy, and iris neovascularisation.[12] Topical or sub conjunctival application of bevacizumab was found to be effective for inhibiting corneal neovascularisation.[13]A recent pathological study demonstrated that neutralization of VEGF reduced vascularity and decreased scar formation during wound healing, showing that VEGF strongly influenced scar tissue formation.[14] Several studies have shown that trabeculectomy with sub conjunctival bevacizumab injection resulted in successful IOP control.

We conducted this study to evaluate the efficacy of sub conjunctival injection bevacizumab in trabeculectomy.

Patients and Methods

Study Design: This study was a prospective, nonrandomized, interventional case series conducted between January 2015 to January 2016 at Lions Eye Institute & Hospital, Agargaon, Dhaka (LEI&H).

Study Subjects: Sixteen individuals with a diagnosis of primary open angle glaucoma or primary angle closure glaucoma with a recorded intraocular pressure (IOP) of more than 21 mmHg, visual field or optic disc changes characteristic of glaucoma, and taking the maximum tolerated dose of medication were recruited from the Glaucoma Clinic, LEI&H.

Thirty patients of either gender were assessed for eligibility, of which sixteen patients satisfied the inclusion and exclusion criteria that are summarized in Table 1.

Indications for surgery were based on: (1) IOP associated with high probability of glaucoma progression and (2) glaucomatous visual field loss or changes of the optic disc indicative of progressive glaucoma damage.

Signed informed consent was obtained from all patients.

Preoperative Assessment : Before surgery, all participants underwent a comprehensive ophthalmic examination, including best-corrected visual acuity (BCVA),slit-lamp biomicroscopy, IOP measurement by a calibrated Goldmann applanation tonometer, funduscopy with a 78 D lens, gonioscopy and visual field perimetry using Humphrey Field Analyzer wherever visual status of the patient permitted.

Preoperative data recorded separately for study analysis for each patient included age, gender, type of glaucoma, number of anti-glaucoma medications, IOP and BCVA.

At the time of surgery, date of surgery, type and site of conjunctival flap, site and dose of sub-conjunctival bevacizumab injection, and intra-operative complications were recorded.

Surgical Technique: All surgeries were performed under local anaesthesia using the same technique. A 6-0 silk corneal traction suture was used. A fornix-based conjunctival flap was dissected either at the supero-nasal or supero-temporal quadrant. Meticulous hemostasis was performed with bipolar cautery. Using a 15-degree knife to delineate and a crescent knife to dissect, a half-thickness 4 x 4 mm trapezoidal scleral flap was fashioned. Two diagonal scleral flap sutures were preplaced using 10-0 nylon. A corneal paracentesis was made using the 15-degree knife. Sclerectomy was performed with a Kelly-Descemet's punch, and peripheral iridectomy was performed with Vannus scissors. The scleral flap was closed with 2 10-0 nylon sutures. The conjunctiva was closed with 2 interrupted 10-0 nylon wing sutures. After suturing the conjunctiva, 1.25 mg/0.05ml of reconstituted bevacizumab was injected sub conjunctivally over the sclera flap area, with a 30-G needle and a tuberculin syringe. The needle entrance was at least 8 mm away from the bleb to prevent any needle track leakage.

Postoperative treatment included topical 0.5% moxifloxacin hydrochloride 4 times per day for 1 month and 1% prednisolone acetate every 2 hours for 2 weeks and then tapered off slowly over 10 weeks. Antiglaucoma medications were discontinued on the day of surgery. After surgery, antiglaucoma medication was added if necessary.

Follow-up Evaluations and Outcome Measures: Patients returned for 7 postoperative follow-up visits within 6 months: days 1, 7, 14, 30, 60, 90 and 180. A window of ± 7 days was allowed for the 30-, 60- and 90- day visits and one of ± 14 days was allowed for the 180- day visit. After month 6, follow-up visits were continued every 3 months.

At each postoperative visit, the examination included measurement of BCVA, IOP, slit-lamp biomicroscopy, Seidel testing, funduscopy, number of anti-glaucoma medications used, presence of complications, postoperative interventions and morphologic characteristics of the filtering bleb.

The IOP was considered the primary outcome measure. Complete success was defined as an IOP of 21 mmHg or less and/or at least 20% reduction in preoperative pressure without any anti-glaucoma medications. Qualified success was defined as the following: (1) IOP of 21 mmHg or less and at least 20% reduction in the preoperative IOP with anti-glaucoma medication, where the number of anti-glaucoma medications was less than that used before surgery; or (2) in eyes with preoperative IOP of 21 mmHg or less, postoperative IOP equal to or less than preoperative IOP and a reduction of at least 2 medications.

Failure of the treatment was defined as IOP < 6mmHg or IOP > target IOP despite medication, or needing further glaucoma surgery, or development of adverse events that needed treatment in the operating room, such as suturing bleb leakage and anterior chamber reformation.

Table 1 : Summary of Incusion and Exclusion Criteria for Determining Patient Eligibility

Inclusion Criteria	Exclusion Criteria
Age between 30 to 60 years	Secondary glaucoma such as traumatic glaucoma, neovascular glaucoma, uveitic glaucoma, aphakic glaucoma, steroid induced glaucoma, pseudoexfoliation, and pigment dispersion glaucoma
Diagnosis of POAG or PACG on maximally tolerated medical treatment with uncontrolled IOP or the target IOP is not reached, or intolerance for antiglaucoma medications	Coexistent visually disabling cataract and glaucoma requiring combined phacotrabeculectomy
Patient willing to comply with the study protocol	History of previous conjunctival incision surgery
	History of any previous anti-VEGF therapy
	Any ocular condition that would prevent accurate IOP measurement
	Active ocular infection or inflammation
	Patient with uncontrolled systemic hypertension, congestive heart disease, and renal failure or history of systemic thromboembolic disease

Results

The average age was 46.56±5.58 years (range, 38–56 years). There were 7 males and 9females. Eight patients had primary open angle glaucoma and eight patients had primary angle closure glaucoma.

The preoperative IOP was 28.5±5.14 mmHg (range, 18–34 mmHg) with 2.8±0.75 IOP-lowering medications (range, 2–4).

The preoperative best-corrected visual acuity was 0.73±0.34, and that at the 6-month follow-up was 0.74± 0.47.

The postoperative IOP was 9.5±1.9mmHg (range,7–14 mmHg) on day 1, 10±1.9 mmHg (range, 8–14 mmHg) on day 7, 11.5±1.96 mmHg (range, 8–16 mmHg) at 1 month,12.4±1.31 mmHg (range, 10 –14 mmHg) at 3 months, and 12.9±1.34mmHg (range, 10 –14 mmHg) at the 6-month follow-up with no IOP-lowering medications. At 6 month follow-up there was a mean IOP reduction by 54.7%.

On a detailed slit-lamp examination, none of the patients had any ocular complications, including intraocular inflammation, postoperative hyphema, IOP increase, and increase in cataract or endophthalmitis. Two patients showed Seidel test positive on first postoperative day which sealed spontaneously within 1 week with application of bandage contact lens. There was sub conjunctival bleed in four patients at the site of injection.

None of the patients had thin, elevated, cystic blebs, and vascularisation was restricted to the periphery of the bleb. Systemic complications related to sub conjunctival bevacizumab were not found in any of the patients.

Discussion

The success of trabeculectomy mainly is related to the control of postoperative scar formation at the site of bleb formation. Fibroblast proliferation and neovascularisation are the 2 main and related components of wound healing. Angiogenesis is a key element of the proliferative phase of healing, supplying oxygen and nutrients to support the rapid growth of the cells mediating repair.[15] Wound angiogenesis is controlled to a large extent by levels of the proangiogenic factor VEGF that serves as a unique mitogen specific to vascular endothelial cells.[15] Bleb failure is defined as the appearance of a flat, vascularised and scarred bleb. Bleb failure occurs because of increased vascularisation of the conjunctiva with associated migration of fibroblast secondary to cytokines toward the wound healing causing scarring and closure of the fistula tract leading to flap fibrosis.[16] Angiogenesis often is an unwanted process in the postoperative period after glaucoma filtration surgery.[17,18] Important angiogenic factors in ocular neovascularisation are VEGF, basic fibroblast growth factor (FGF), insulin-like growth factor, and epithelial growth factor (EGF).[19-21] VEGF is important during the proliferative phase of wound healing.[22] Previous studies found that the VEGF levels were elevated in patients who had a trabeculectomy. Moreover there are also evidences that show that VEGF has a direct effect on fibroblasts.[23]Inhibiting the neovascular signal cascade with anti-VEGF agents may decrease fibroblast proliferation.[24]

Because angiogenesis forms an integral part of wound healing, bevacizumab can have a role to play in improving the success of surgery by the combined inhibitory effect on fibroblast activity and angiogenesis.

Kahook and associates previously reported that bevacizumab prevents excessive scar formation after needle bleb revision in patients with a failing bleb after trabeculectomy.[21] In the study by Grewal and associates, 12 patients underwent trabeculectomy with bevacizumab (1.25 mg), and the mean IOP decreased from 24.4 mm Hg to 11.6 mm Hg (52%), with no medications at 6 months after surgery.[24] In a study including 6 eyes that underwent trabeculectomy with adjunctive bevacizumab, Choi and associates indicated a 62% reduction in the IOP from 37.5 ± 14.4 mm Hg before surgery to 12.2 ± 3.3 mm Hg 6 months after surgery with no medications. However, that study included high-risk patients with neovascular, uveitic and postvitrectomy glaucoma with a higher baseline IOP.[25]

In our study comprising 16 glaucomatous eyes, the mean reduction of IOP was 54.7% from preoperative IOP at 6 month, which is comparable to the previous studies. Moreover, the short-term viability of bevacizumab-augmented trabeculectomy was represented clinically by a functioning filtering bleb and a reduced IOP. The clinical safety was represented by absence of signs of local toxicity or intraocular inflammation and by the lack of any adverse effects for the patient.

Some of the limitations of this interventional case series are the small sample size, the short-term of follow-up of 6 months and an uncontrolled study design. Moreover, due to the relatively stringent criteria for inclusion,

exclusion, and success, the study design may not be applicable to all glaucoma patients. A larger cohort of patients and a longer follow-up period are warranted to determine the possible long-term complications of sub conjunctival bevacizumab and its effect on the outcome of trabeculectomy. Further controlled studies, comparing the results of trabeculectomy augmented with bevacizumab with those of trabeculectomy with antifibrotics are needed. In future we plan to study, a comparison with bleb characteristics after trabeculectomy augmented with antifibrotics involving a larger sample size and longer follow-up period.

Conclusion

A single intraoperative sub conjunctival injection of bevacizumab in association with trabeculectomy is safe and effective in terms of IOP control.

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Original Article

Relationship of IOP and Blood Glucose Level in POAG Patients with Type 2 Diabetes : A Tertiary Center Study

F. Hossain[1], R. Mannan[2]

Abstract

The aim of this study was to evaluate the relationship between blood glucose levels and intraocular pressure (IOP) fluctuation in Primary Open Angle Glaucoma patients having type 2 diabetes. 165 glaucoma patients with type 2 diabetes underwent a complete ophthalmic examination,fasting and post prandial venous blood glucose testing, pachymetry and applanation tonometry. Postprandial IOP was significantly higher than baseline IOP($P < 0.001$). Postprandial blood glucose levels were significantly higher than baseline measurements ($P < 0.001$). There is also a significant association between the baseline glucose levels and IOP change ($P = 0.001$). The extent of glucose level change remained significantly associated with IOP despite consideration of gender, age and baseline IOP ($P < 0.001$). In summary there is a significant relationship between blood glucose level and IOP in type 2 diabetic patients with glaucoma.

Introduction

Glaucoma is a progressive optic neuropathy characterized by degeneration of retinal ganglion cells (RGCs) and their axons, resulting in changes in the optic disc and progressive visual field loss.

Although glaucoma is a multifactorial disease, the intraocular pressure (IOP) still remains its major modifiable risk factor. Several large randomized clinical trials described the relationship between IOP and glaucoma development and progression. Therefore, adequate determination of an individual's CCT and IOP value is very important in the management of glaucoma.

The IOP can be influenced by different systemic factors such as hypertension, atherosclerotic diseases, body mass index, and diabetes. For instance, Lee and colleagues studying the relationship between IOP and systemic disorders found that increased mean blood pressure is strongly correlated with risk of increased IOP.

Diabetes is found to be associated with higher IOP values in most population studies, but the exact underlying mechanisms are still unclear. Few studies have proposed that changes in corneal biomechanics (increased corneal hysteresis) in diabetic eyes would lead to overestimation of IOP. However, it is still unclear whether variations in glucose levels could lead to IOP changes in diabetic individuals.

As diabetes and open angle glaucoma or ocular hypertension may coexist in many patients, a better understanding about variations in blood glucose levels and its affect CCT and IOP changes would give additional information to reliable and accurate IOP measurement.

Methods

This is a prospective observational study. We have strictly followed to the Declaration of Helsinki and a written informed consent was obtained from all participants.

Patients

We prospectively enrolled diabetic patients with POAG. All participants underwent a complete ophthalmological examination including review of medical history, best-corrected visual acuity, slit-lamp biomicroscopy, IOP and CCT measurement, gonioscopy, dilated funduscopic examination, and refraction. Exclusion criteria were diagnosis of type 1 diabetes, ocular hypertension, corneal opacity or irregularities that could alter the ophthalmological examination, refractive error greater than ±5 D spherical or cylindrical greater than ±3 D, and

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central corneal thickness (based on ultrasound pachymetry) above 600 microns or below 450 microns. Diabetes was defined according to self-reported physician diagnosis and all diabetic patients were under medical treatment for diabetes .

Capillary Glucose Testing

All participants underwent capillary glucose testing in two distinct situations: first, baseline measurements (fasting for exactly 8 hours, i.e., after overnight fasting) and, second, postprandial measurements (exactly 2 hours after the meal, i.e., after lunch time). The same examiner performed all measurements in a masked fashion. The measurement of capillary glucose was performed by collecting blood from the patient's finger, pierced through the skin by a lancet and checked with an automated device (One Touch Life Scan, Johnson & Johnson, CA, USA).

Intraocular Pressure Assessment

Immediately after the capillary glucose testing, IOP was measured in both eyes (i.e., fasting for exactly 8 hours and exactly 2 hours after lunch time) of each patient by Goldmann tonometry applanation (Haag-Streit, Köniz, Switzerland). The calibration of each instrument was checked at the beginning of each session, according to the manufacturers' instructions. All measurements were taken with the patient in a sitting position. The same examiner performed all IOP measurements in a masked fashion and a different examiner performed the glucose levels measurements. Whenever both eyes were eligible, the right eye was arbitrarily chosen for analysis.

Statistical Analysis

Descriptive statistics included mean and standard deviation values for normally distributed variables. Paired -test was used for comparison of IOP values between each time point (baseline and postprandial).

The association between changes in glucose level and IOP variation was investigated using univariable and multivariable regression analysis. Whenever both eyes were eligible, the right eye was arbitrarily chosen for this analysis.

All statistical analyses were performed with commercially available software.

Results

A total of 165 patients type 2 diabetic patients with POAG were included. Diabetic patients had been followed for an average of 6 months . Age and IOP did not differ significantly between the groups. Baseline glucose levels were higher in the patients.

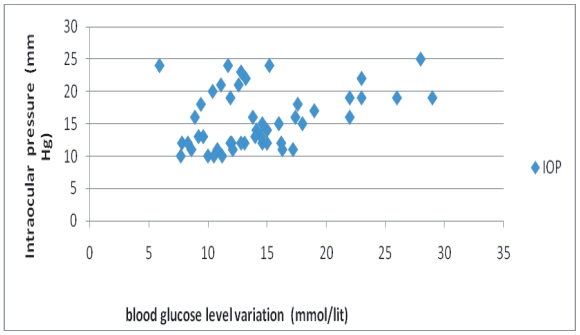
Table-I

Demographic and clinical variables in eyes of diabetic patients with POAG.

	No of diabetic patients (n= 165)
Age (±SD) (years)	58.0 ± 8.9
Gender (%)	
Male	95 (58.0%)
Female	70 (42.0%)
CCT(m)	529.2 ± 22.2

CCT = central cornea thickness; SD = standard deviation.

Table-II : Relationship of IOP with changes in blood glucose level



Postprandial IOP was significantly higher than baseline IOP. Figure : 2 shows the IOP distribution at each time point. Postprandial glucose levels were significantly higher than

baseline measurements (mean increase of 3.4 mmol/lit).

In the univariable analysis, there is a significant (positive) association between glucose levels variation and IOP change ($P<0.001$).

In the multivariable analysis, the magnitude of glucose level change remained significantly associated with IOP variation, even including age, baseline IOP, and gender as confounding factors.

Discussion

Till now it was not much clear whether blood glucose levels could influence CCT and IOP variation altogether. We found a significant increase in postprandial IOP values in diabetic patients with POAG.

The relationship between diabetes and IOP has been underscored indivisually in previous publications and their results reveal a positive association between diabetes IOP as in The Barbados Eye Study or the Blue Mountain Eye Study.

However, our data are consistent with these two studies, as we found a mean IOP increase of 2.3 mm Hg in diabetic patients during the postprandial period. Several hypotheses have been created to explain the association between high glucose levels and IOP. Some researchers believe that there are genetic factors associated. Others agree with the idea that a diabetic person could have an autonomic dysfunction which would lead to an IOP increase. However some believe that elevated blood glucose results in the induction of an osmotic gradient which leads to fluid shifts into the intraocular space .

Regarding the clinical implications of our study, we, the Ophthalmologists often see diabetic patients on daily practice. Many of these patients already might have glaucoma or ocular hypertension or are glaucoma suspects. Usually much attention is given to each mmHg variation in IOP, the glycemic control is rarely taken into account. Based on our findings, glycemic levels variation may influence IOP change and is therefore relevant for diagnosis, treatment and management, especially in diabetic patients, whose average IOP variation (between baseline and postprandial measurements) was approximately 15% (for an average glycemic variation of 40%). Clinicians should consider the patient's glycemic status and glucose level variations with IOP values assessment in diabetic patients.

It is also important to discuss some limitations of the study as well. First, its small sample size. Second, glucose levels were assessed only twice (baseline and postprandial). Multiple measurements might have provided additional data and possibly allowed a more detailed analysis. Third, the investigation of other systemic co-morbidities may have been insufficient. Fourth, we did not correlate the duration of diabetes and IOP. So, further studies should be done to evaluate the causative relationship between glucose levels and IOP variation.

Conclusion

The study suggests that there may be an important relationship between blood glucose levels and IOP values in type 2 diabetic patients with POAG. Postprandial IOP seems to be significantly higher in these patients compared to that of baseline values, revealing a strong association with the extent of glucose level increase. This fact should be considered while measurement of IOP in POAG patients.

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Angle Recession Glaucoma - a clinical review

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Abstract

Traumatic glaucoma refers to a heterogeneous group of posttraumatic ocular disorders with different underlying mechanisms that lead to the common pathway of abnormal elevation of intraocular pressure (IOP) and increased risk of optic neuropathy.

Angle-recession glaucoma is classified as a type of traumatic secondary open-angle glaucoma.[1] This condition may be underdiagnosed because onset is often delayed and because a history of eye injury may be distant or forgotten.

Angle recession, with or without glaucoma, is a common sequelae of blunt ocular trauma and one characterized by a variable degree of cleavage between the circular and the longitudinal fibers of the ciliary muscle. Traumatic microhyphema and gross hyphema are both equally associated with a high risk of angle recession[2] This article will give us the clinical review of Angle Recession Glaucoma.

Introduction

Angle recession glaucoma (ARG) is a secondary open angle glaucoma that is associated with ocular trauma. Recession of the anterior chamber angle is a common slit lamp and gonioscopic finding following concussive ocular trauma. A small percentage of these people go on to develop glaucomatous optic neuropathy and vision loss days, months or even years later[1]. There are reports of glaucoma developing up to 50 years after the injury[2].

Historical Perspective

Angle recession was first described by Collins in 1892[3]. The association between trauma and unilateral glaucoma was made by D'Ombrain in 1949[4]. In 1962, Wolff and Zimmerman astutely connected the pathologic entity of the angle's recession with the clinical phenomenon of unilateral chronic glaucoma following trauma. 2 Physicians must consider the possible development of glaucoma after any ocular trauma, which may damage the trabecular meshwork and/or other ocular structures relating to aqueous outflow. The consequent transient or prolonged elevation of IOP may lead to glaucomatous optic neuropathy. Ophthalmologists must also be cognizant that the treatment for the ocular injury, such as the use of steroid therapy, can further complicate the management of elevated IOP. Ocular trauma may be classified as either blunt or penetrating. This article focuses on the former—namely, angle-recession glaucoma (ARG). It can be further subdivided into two stages of injury: early and delayed onset. In cases of early-onset ARG, a clinical examination may reveal iritis with or without hyphema. Clues such as hyphema with or without an iridodialysis and/or cyclodialysis cleft should alert physicians that the trabecular meshwork has sustained damage. The delayed onset of angle recession will cause a permanent elevation in IOP months to years after the initial blunt injury.

Risk Factors

The main risk factor for angle recession is trauma. Girkin, et al. used the US Eye Injury Registry to demonstrate that 3.39% of people go on to develop ARG at 6 months following blunt ocular trauma[8]. A 10 year prospective study of 31 eyes by Kaufmin and Tolpin reported that 6% with angle recession will go on to

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develop glaucoma[1]. Since it appears that angle recession per se does not always lead to glaucoma, there may be other factors at play that have not yet been discovered.

It has been reported that up 60% of eyes with non-penetrating or concussive trauma will develop some degree of angle recession[5]. Angle recession is also strongly associated with traumatic hyphema with studies reporting a 60-100% incidence[6]. The most commonly reported forms of trauma associated with ARG are recreational activities and assault[7]. However, a portion of patients never recall a specific event despite other clinical signs of trauma such as iris sphincter tears or traumatic cataract.

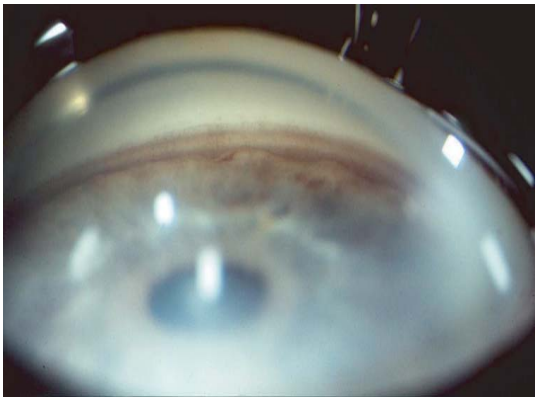

Figure 1 : Irregular widening of the visible ciliary body in a quadrant with angle recession

Several publications have demonstrated that the greater the number of clock hours of angle recession, the greater the likelihood of developing elevated pressures and glaucoma. Authors have reported varying amounts ranging from 180 to 240 degrees as "at high risk," but most studies confirm that greater than 180 degrees of recession makes glaucoma more likely[9,6,10]. Interestingly, one study reported 50% of ARG patients will go on to develop open angle glaucoma in the contralateral eye[11]. This has led to the hypothesis that angle recession does not directly cause elevated IOP, but may accelerate the process in an already at risk eye.

More recently, Sihota, et al. found the presence of increased pigmentation at the angle, elevated baseline IOP, hyphema, lens displacement, and angle recession of more than 180° were significantly associated with the occurrence of chronic glaucoma after closed globe injury[12].

Mechanism and Pathophysiology

The mechanism of glaucoma associated with angle recession appears to involve 5 processes.

First, blunt force delivered to the globe initiates an anterior to posterior axial compression with equatorial expansion. Sudden indentation of the cornea may be a key factor in angle trauma, creating a hydrodynamic effect by which aqueous is rapidly forced laterally, deepening the peripheral anterior chamber and increasing the diameter of the corneoscleral limbal ring.

Second, this transient anatomic deformity results in a shearing force applied to the angle structures, causing disruption at the weakest points if the force applied exceeds the elasticity of the tissues.

Third, although multiple anterior segment structures can be damaged by the above mechanism, a common site of avulsion involves the ciliary muscle. In angle recession, the ciliary body is torn in a manner such that the longitudinal muscle remains attached to its insertion at the scleral spur, while the circular muscle, with the pars plicata and the iris root, is displaced posteriorly. During this process, shearing of the anastomotic branches of the anterior ciliary arteries can occur, resulting in a hyphema. The anterior chamber typically becomes abnormally deep in the meridians of recessed angle due to posterior deviation of the relaxed iris-lens diaphragm. Subsequently, a fissure representing the separation of the longitudinal and circular fibers may be visible by gonioscopy or by histologic examination.

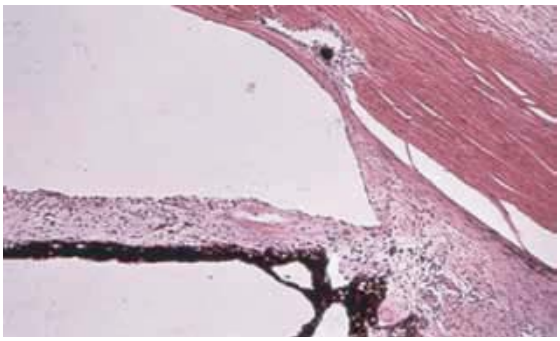

Figure 2: A histological section of angle recession illustrates the tear between longitudinal and circular fibers, with longitudinal fibers still attached to the scleral spur. There is retrodisplacement of the iris root.

Fourth, in some cases, angle recession progresses to glaucoma. The contusional deformity, when extensive, may result in trabecular dysfunction, which may lead to early or delayed loss of outflow facility and elevation of IOP. The mechanism is not well understood, but evidence suggests an increased incidence of primary open-angle glaucoma (POAG) in the other eye of affected patients. One theory suggests that patients with angle-recession glaucoma have an independent, perhaps genetic, predisposition to chronically diminishing trabecular function in both eyes. A finite portion of the trabecular meshwork in eyes with angle recession is initially rendered dysfunctional by the injury and/or the healing process. With time, the outflow capacity of the remaining meshwork is gradually reduced because of preexisting innate factors; the ultimate result is elevated IOP.

Fifth, chronic elevation of IOP leads to optic neuropathy characterized by progressive optic cupping and visual field loss.

Clinical Approach

Physical examination

Patients who suffer blunt trauma to the eye will require a thorough slit-lamp examination and a careful, detailed characterization of the angle structures via gonioscopic examination. Initially, gonioscopy may not be possible, owing to iritis or pain. This examination may be delayed until the patient can cooperate. Clinical clues such as hyphema, iridodialysis of the iris root, and/or cyclodialysis will guide the clinician to pursue gonioscopy sooner than later to confirm angle recession.

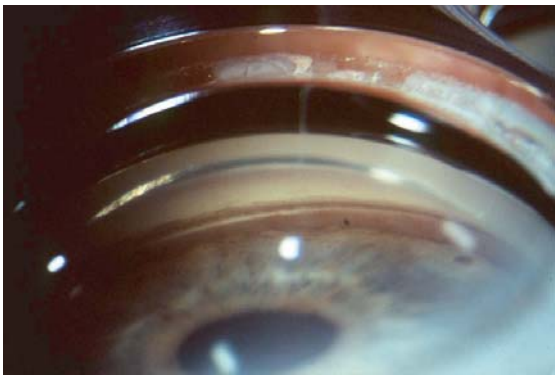

Figure-3: Gonioscopic view of patient with angle recession. Note the marked posterior displacement of the iris, with a wide ciliary body band posterior to the scleral spur.

1. Gonioscopy- The key exam finding in angle recession is widening of the ciliary body band which is seen on gonioscopy. This exam finding, in the presence of elevated pressure and nerve damage, leads to the diagnosis of angle recession glaucoma. Some normal eyes have a broad ciliary body band thus it is important to compare suspected areas of recession to the opposite eye to see what the "normal" ciliary body band looks like to avoid mistaking physiologic or even 360-degree recession as normal.

2. Slit Lamp Exam- Other evidence of ocular trauma should clue the clinician to look for angle recession. Such findings might include sphincter tears, corneal scars, Vossius ring, iridodialysis, iridodenesis, phacodenesis and hyphema.

3. Angle recession should be differentiated from cyclodialysis, which is the disinsertion of the ciliary body from its attachment to the scleral spur.

A number of anterior segment abnormalities

often accompany angle recession:

Cyclodialysis, Iridodialysis, Iridoschisis, Anterior synechia, Iris sphincter tears, Mydriasis, Iris atrophy, Transillumination defects, Iritis, Zonular breaks, Phacodonesis, Subluxated lens and Cataract

4. A strong association exists between hyphema and angle recession, but the ciliary body also can be severely damaged from blunt trauma, without the appearance of a hyphema. Posterior segment abnormalities, which may signify prior episodes of trauma, include the following:

Vitreous opacities, Chorioretinal scars, Macular hole, Retinal breaks, Retinal detachment and optic atrophy.

5. An uncontrolled and sustained elevation in IOP in angle-recession glaucoma, as in other forms of glaucoma, ultimately leads to progressive cupping of the optic nerve and loss of the visual field.

6. Snellen visual acuity is typically uninvolved until the late stages of glaucoma.

7. Formal visual field testing is of paramount importance in diagnosing and monitoring the disorder.

Diagnostic Considerations

Angle recession refers to a tear between the circular and longitudinal fibers of the ciliary body. Cyclodialysis is defined as a detachment of the ciliary body from its insertion at the scleral spur. Iridodialysis is separation of the iris root from its attachment to the anterior ciliary body. By comparison, iridoschisis refers to splitting of layers of iris stroma. All of these conditions are sequelae of blunt ocular trauma, and any of these conditions may coexist. Laboratory studies have indicated that iridodialysis and cyclodialysis occur at higher blunt impact energies compared with the relatively lower thresholds resulting in angle recession.[5]

Investigations

1. Visual field Analysis-Because progressive loss of visual field is a potential outcome, formal visual field testing is the most important adjunctive diagnostic modality in detecting and following up the disorder.
2. Optic nerve photography is also important for documenting and monitoring glaucoma.
3. OCT/HRT/GDx-Computerized disk analysis and analysis of nerve-fiber layers has been gaining acceptance in the diagnosis and management of all forms of glaucoma.
4. Additional Imaging-
    - o The diagnosis of angle recession is confirmed during office examination.
    - o Usually, imaging is necessary only to evaluate comorbidities due to trauma.
    - o On occasion, gonioscopy is difficult or impossible in traumatized eyes because of corneal edema, corneal scarring, hyphema, synechia, or other opacity. In such cases, high-frequency ultrasound biomicroscopy (as a supplemental tool to standard office examination) is effective for evaluating abnormalities of the angle in the anterior chamber.[32, 28]

■ Ultrasound biomicroscopy (UBM) produces high-resolution axial images of the anterior globe, providing cross-sectional views of the angle in vivo similar to those of a histologic section. This noninvasive procedure is readily performed in a clinical setting in an intact globe, and it provides information otherwise unavailable from convention examination.

- o High-resolution images of angle recession, zonular deficiency, iridodialysis, and cyclodialysis have been described. Zonular deficiency and angle recession are the most

common UBM findings in a closed-globe injury.28

- o Ultrasound biomicroscopy findings of a wider angle and absence of cyclodialysis have been reported to be significant predictors for the development of traumatic glaucoma in eyes with closed-globe injury.6

- Slit-lamp optical coherence tomography (SL-OCT) has also been described as a method of imaging angle recession, but it may be less reliable than UBM.[29]

Management

The necessity of initiating treatment of angle-recession glaucoma depends on the severity of the initial injury and the somewhat variable clinical course as healing progresses. Normotensive eyes with angle recession of more than 180° should be routinely reexamined for an indefinite period to monitor for the development of late glaucoma.

Medical Care

- In patients with an abnormal elevation of IOP, the decision to begin therapy is based on the clinician's overall assessment of the risk of vision loss.
  - o The severity of IOP elevation, optic nerve appearance, and visual field findings contribute to the decision-making process. Glaucoma medications should be implemented in the early stage of the condition.[34]
  - o Treatment almost always is indicated when the IOP is greater than an arbitrary range of 25-28 mm Hg and/or when glaucomatous optic nerve or visual field changes are documented over time.
- After the diagnosis of angle recession is established, its management is similar to that of POAG, with a few special considerations.
  - o Use of topical aqueous suppressants in the initial medical treatment is preferred; these include beta-antagonists, alpha-agonists, and carbonic anhydrase inhibitors.
  - o Prostaglandin analogs, which increase uveoscleral outflow, have a theoretical benefit in angle recession because the trabecular meshwork is thought to be dysfunctional in such cases.
  - o Use caution in administering miotic agents because pilocarpine has been reported to cause a paradoxical elevation of IOP in angle recession, presumably due to a reduction of uveoscleral outflow.
  - o Atropine has been reported to reduce IOP in angle-recession glaucoma; therefore, cycloplegic agents may have a role in treatment.
  - o A trial of a cycloplegic agent should be reserved either for cases involving failure of conventional glaucoma therapy or for cases with other indications for cycloplegia (eg, inflammation).
- Of course, the judicious use of steroid therapy with fast tapering is required to address the concomitant iritis from the blunt injury.

Surgical Care

Surgical intervention in angle-recession glaucoma is usually indicated when maximally tolerated medical treatment has failed[34] and when the risk of progressive visual loss outweighs the estimated risk of the planned surgical management. In general, outcomes of surgical treatment are less favorable than those of POAG.

Laser trabeculoplasty

Laser trabeculoplasty has been associated with short-term success, though the procedure has been reported to have poor long-term effectiveness, particularly in eyes with more than 180° of angle recession. IOP elevation may become worse in response to ALT.

Filtration surgery

Filtration surgery has a success rate lower than that of POAG.

Trabeculectomy in eyes with angle recession is associated with decreased postoperative reduction in IOP, increased rates of bleb fibrosis and bleb failure, and increased dependence on postoperative medical treatment of glaucoma.[35]

The adjunctive use of antimetabolites, particularly mitomycin C, can improve the success of trabeculectomy. This finding suggests that an antimetabolite should be used during the initial filtering procedure. A 2001 report described effective results with an acceptable complication rate in such cases.[33]

In the management of severe blunt trauma cases involving angle recession with dense vitreous hemorrhage and/or retinal detachment, combined trabeculectomy and pars plana vitrectomy has been reported with some successful outcomes.[34]

Tube shunt devices

Benefits with the implantation of tube shunt devices have been demonstrated, but outcomes are reportedly less successful in angle recession than in other types of refractory glaucoma.

A 1993 study showed the superior results of trabeculectomy with antimetabolite over Molteno implantation in cases of posttraumatic angle-recession glaucoma.

Conclusion

Glaucomatous optic neuropathy can be a devastating consequence of angle-recession blunt injury. Early diagnosis and aggressive intervention to lower the IOP are of the utmost importance. Once the sequela of the injury (eg, hyphema) resolves, appropriate counseling is the next crucial step. Physicians must educate patients on their injury so that they understand their lifetime risk of developing glaucoma. Careful lifelong monitoring of their IOP and examinations of their optic nerves is recommended for patients who experience angle recession, because glaucoma is usually an asymptomatic disease.

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Topiramate Induced Ocular Complication—A Case Report

M.A. Rahman[1], Z.S. Shahid[2], M.H. Rahman[3]

Abstract

Topiramate is an anti epileptic drug, also used for treatment of migraine, is reported to cause various ocular adverse effect. Among them Acute Angle Closure Glaucoma (AACG) and Acute Myopia are significant.

A case is presented with Acute Myopia with Impending AACG after short term use of Topiramate for treatment of Migraine.

Possible mechanisms are discussed.

Treatment is the withdrawal of medication and supportive treatments as indicated.

Usually prognosis is satisfactory

Introduction

Topiramate is a drug used to treat epilepsy in children and adults, and it was originally used as an anticonvulsant. The drug is also used to treat migraine, due to the effect it has on the blood vessels in the brain. It has been found to be increasingly effective for migraine sufferers with limited side effects.

A case report is documented with ocular complications after short term use of topiramate prescribed for migraine.

Case Report

A 17 years old female patient presented to ophthalmology clinic, Apollo Hospital, Dhaka, with sudden loss of vision in both eyes. Patient also complained of eyeache, headache, redness and swelling of eye lids since last 03 days. She had no history of dimness of vision in either eyes nor used any spectacles. Earlier the patient was treated by neurologist for Migraine. Patient taken tablet Topiramate for treatment of migraine as prescribed by neurologist for 07 days (25 mg. one tab daily at night). She was also prescribed Tab. paracetamol, Tab. Amitryptaline, On examination her visual acuity was 2/60 in both eyes. Marked conjunctival congestion and chemosis. Clear Cornea and Normal pupillary reaction, lens-clear. Fundus shows mild macular oedema, otherwise normal. Color vision normal. Perimetry within normal limit. IOP Rt.23mmHg, Lt 22 mm Hg. Refraction : Rt.-7.00 DSph with--0.75 DCx105 =6/9 Lt. –6.00 DSph with –0.75 DCx100= 6/12. Gonioscopy—angle narrow gr. 2. B-scan—choroidal thikness1.0 mm, Retina on, no choroidal effusion.

Patient was diagnosed as Topiramate induced Acute Myopia with impending Acute angle closure glaucoma, both eyes.

Patient advised to stop taking Topiramate and kept without medication for follow up.

She was reviewed after 02 days, Lid swelling reduced, mild conjunctival congestion, visual acuity was 6/6 with --4.00 DSph. Lens. IOP 22/20 mm Hg, Gonioscopy as before, Fundus-WNL, OCT (Macula)—WNL

Again reviewed after 06 days. Visual Acuity 6/6 (Un Aided), Anterior segment-Un remarkable , WNL. IOP 16 MMHg both eyes. Others Nothing Significant.

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Discussion

It is well documented that topiramate primarily causes Ocular side effect. So far my knowledge concern and after limited search of the literature I didnt find any case report on to piramate induced ocular complication in Bangladesh, This may be the first case report presented.

Chemically, topiramate is a sulfamate-substituted monosaccharide, related to fructose, a rather unusual chemical structure for an anticonvulsant.[1]

Recent clinical reports indicate that it may have mood stabilizing properties.[2] Other off-label and investigational uses of topiramate include the treatment of essential tremor, bulimia nervosa, obsessive-compulsive disorder, idiopathic intracranial hypertension and cluster headache.

Topiramate is quickly absorbed after oral use.[3] Most of the drug (70%) is excreted in the urine unchanged. The remainder is extensively metabolized by hydroxylation, hydrolysis, and glucuronidation. Six metabolites have been identified in humans, none of which constitutes more than 5% of an administered dose.

Several cellular targets have been proposed to be relevant to the therapeutic activity of topiramate. These include (1) voltage-gated sodium channels; (2) high-voltage-activated calcium channels; (3) GABA-A receptors; (4) AMPA/kainate receptors; and (5) carbonic anhydrase isoenzymes. There is evidence that topiramate may alter the activity of its targets by modifying their phosphorylation state instead of by a direct action.[4] The effect on sodium channels could be of particular relevance for seizure protection. Although topiramate does inhibit high-voltage-activated calcium channels, the relevance to clinical activity is uncertain. Effects on specific GABA-A receptor isoforms could also contribute to the anti seizure activity of the drug. Topiramate selectively inhibits cytosolic (type II) and membrane associated (type IV) forms of carbonic anhydrase.[5] The action on carbonic anhydrase isoenzymes may contribute to the drug's side-effects, including its propensity to cause metabolic acidosis and calcium phosphate kidney stone.

Data collected from spontaneous reporting systems have identified one hundred and fifteen cases of ocular side effects of Topiramate which include acute-onset angle closure glaucoma, acute myopia, supra-choriodal effusions, peri-orbital oedema, scleritis, blepharospasm, oculogyric crisis, nystagmus and diplopia.

Acute myopia between 2 to 8.75 dioptres, presents in adults and children with sudden bilateral blurring of vision.

As topiramate is a sulphamatedpreparation, the mechanism of acute myopia is similar to that reported with sulphonamides and acetazolamide. The severity of ciliary body oedema, cilio-choroidal detachment and forward movement of the iris lens diaphragm causes myopia. Myopia may precede and persist after resolution AACG. Myopia on its own resolves following discontinuation of the drug

The mechanism of development of AACG

Ciliary body oedema or cilio-choroidal detachments due to cilio-choroidal effusion causes a forward rotation of the ciliary body which displaces the iris forward to close the anterior chamber angle precipitating an attack of secondary AACG.[6]

The Cilio-choroidal effusion caused by Sulfonamides is an idiosyncratic response in the uveal tissue and is dose independent.[7] The hapten hypothesis postulates that reactive drug metabolites binds to proteins, forming altered proteins which are recognized as foreign substances and incite immune reactions.

Swelling of the lens may also contribute to the shallow anterior chamber. In patients on topiramate this was demonstrated byhigh frequency or standard ultrasound.[8] Though the configuration of the anterior chamber has not been mentioned it is possible that they may have contributed to the precipitation of an attack of AACG secondary to topiramate use.

Treatment

Topiramate should be discontinued and an alternative medication may be prescribed in discussion with the primary physician. The initial treatment should include cycloplegia, in an attempt to displace the iris- lens plane posteriorly, topical and systemic ocular hypotensives and topical steroids. Caution has been suggested with the use of acetazolamide, a sulfamated drug, concurrently with the continued use of topiramate for fear of inducing renal calculi and further ciliary body oedema. Laser peripheral iridotomy used in 23% of reported cases has not been uniformly effective in relieving the secondary angle closure and should be reserved for cases where the above treatment fails. Rapid resolution of an attack has been reported with the use of intravenous methylprednisolone and mannitol.

Conclusion

Ocular complications from topiramate therapy is not very common. Increasing use and growing popularity of the medication for multiple diseases, it is important to create awareness among patients and healthcare providers of the possible drug-induced ocular sequelae and the importance of taking care for appropriate treatment and follow-up. Proper patient. Communication and teamwork within the interdisciplinary team are important to facilitate timely care for patients. Appropriate counseling to the patient during prescription of the drug regarding the ocular symptoms is essential in the management and eventual resolution of this patient's acute episode of bilateral secondary angle-closure glaucoma and acute myopia.

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Juvenile Xanthogranuloma- A Case Report

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Abstract

Purpose : To report a rare case of xanthogranuloma of the eye and its management.

Method : A 14 years old girl presented with headache, proptosis and loss of vision in left eye. She had a history of recurrent spontaneous hyphema and was treated conservatively. Routine blood examination including peripheral blood film was normal. CT scan of the orbit showed left intracranial extension of orbital mass with bony erosion. Craniotomy and removal of the mass was done by a neurosurgeon and was sent for histopathology.

Result : Histopathology revealed xanthogranuloma with tuton giant cells, foamy histiocytes, fibrosis and areas of inflammation. Aspiration and biopsy of bone narrow showed normal cellularity. Few months after operation, her left upper lid become swollen due to recurrence of the xanthogranuloma and globe become enophthalmos due to chronic inflammatory orbital disease.

Conclusion : Although juvenile xanthogranuloma is an uncommon disease, it is one of the most important causes of spontaneous hyphema in children. Iris and lid involvement are common but orbital involvement with intracranial extension is rare. In this case we reported such rare case and its management.

Introduction

Juvenile xanthogranuloma is a self limiting dermatological condition of infancy that usually appears in the head neck region and intraocular involvement has been well documented during last 15 years. It is a benign histocytic disease of uncertain pathogenesis[1-2].

Diagnosis is based on histological examination that shows a nodular, dense infiltrate of polygonal or spindled mononuclear cells with moderate amount of cytoplasm and scattered tuton giant cells[2].

Correct diagnosis especially important because of the possibility of successful eradication of the lesion and control of its complication.

Case Report

Miss. Amena, age 10 yrs went to an ophthalmologist on January 2002 with the complaints of mild pain, redness and reduced vision in her left eye for last few days. She was diagnosed as a case of left hyphaema and treated conservatively. She had no history of bleeding disorders or trauma.

After 4 months she developed same complaints with severe pain in her left eye due to recurrent hyphaema and was treated conservatively. After 6 months, she developed gradual proptosis of left eyeball with occasional headache and reduced vision in the left eye (Fig 1).

Fig 1: Left Mild Proptosis

After 3 months she was admitted in a tertiary hospital with severe proptosis and loss of vision in left eye. There was no abnormality in routine blood examination including peripheral blood film.

CT scan of orbit and brain revealed left intracranial extension of orbital mass with severe

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bony erosion.(Fig 2).

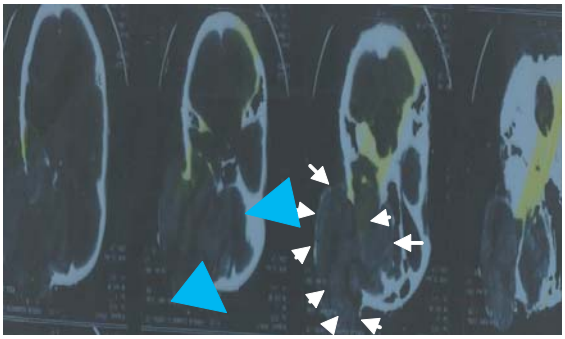

Fig 2 : CT scan shows intracranial extension of orbital JXG.

Suddenly she developed severe headache and vomiting due to raised intracranial pressure. Craniotomy and removal of mass was done as much as possible in the neurosurgery department and tissue sent for histopathology.

Histopathologist found a granulomatous mass with abundant histiocytes and presence of Tuton Giant cell (xanthogranuloma) (Fig 3).

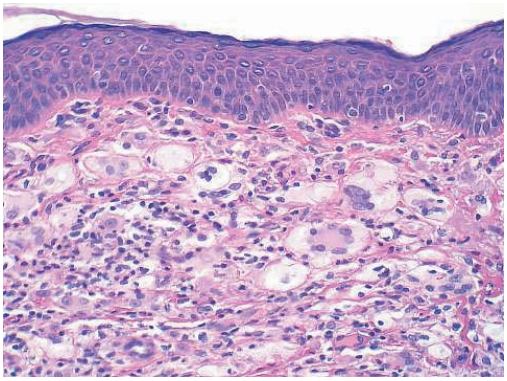

Fig 3 : Histopathology shows histiocytes and tuton giant cells

On 2004 she came to Chittagong Eye Infirmary and Training Complex (CEITC) with reduced vision in right eye (6/36) and pale disc. Left eye was phthisical, bony deformity in the left superior and lateral orbital wall and craniotomy scar overhead (Fig 4). She had no complaints of eyeache or headache in the subsequent follow up and visual acuity of the right eye was stable.

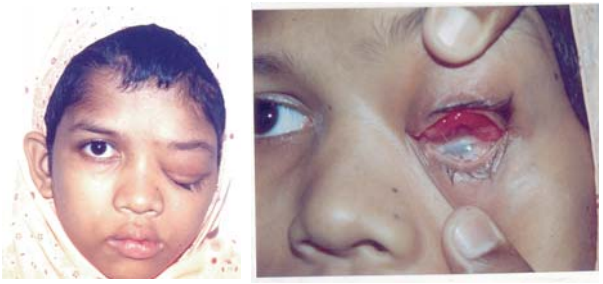

Fig 4: Postoperative enophthalmos

Discussion

Xanthogranulomatous disease, including Erdheim-Chester disease, adult periocular xanthogranuloma and necrobiotic xanthogranuloma, may involve the orbit and ocular adnexa. The disease are linked by common histopathology, specifically by the tutongaint cells, which is usually found in the background of lymphocytes, plasma cells and histocytes and they may manifest clinically[3]. Alpar et al, first described orbital involvement in 1983.

Histopathology of this case showed lymphocytes, histiocytes and characteristic tuton giant cells suggestive of xanthogranuloma.

In Erdheim-Chester disease, xanthogranuloma cells infiltrate the bone, heart, lungs retroperitoneum, perinephric and other tissues (Valmaggia et al. 1997). Xanthogranulomas, which are firmar, bulkier, and more indurated than xanthelasmas, may be present on the eyelied (Jakbiec at al, 1993). When deeper tissues of the orbit are involved, proptosis may be the presenting symptom (Alper et al.1993). In adult periocular xanthogranuloma, the extraocular muscles, adenexal tissue and lacrimal gland may be affected. Additionally, the patient may manifest, symptoms of asthma.[3]

Necrobiotic xanthogranuloma is frequently associated with a systemic paraproteinemia Pathologically, necrobiotic foci are seen, and nonpruritic, waxy, yellowish or flesh-coloured plaques, frequently with ulceration, may be present in the periocular area[3]. In juvenile

xanthogranuloma, which usually occurs only in children (Egan et al, 1999). The orbit, eyelid and conjunctiva may be involved and involvement of the iris may present as spontaneous hypheama[3].

As the 10 years old child had no visceral involvement, no paraproteinemia and no histopathological evidence of necrotic tissue in the specimen, we can excluded Erdheim-Chester disease, Adult periocular xanthogranuloma and Necrobiotic xanthogranuloma.

Age of onset, recurrent hyphema, orbital involvement and histopathologcal evidence confirms the case as a Juvenile xanthogranuloma.

Ocular involvement occurs in 10% of cases of juvenile xanthogranuloma[4]. The iris is the most common location, followed by the eyelid, orbit, choroids, optic nerve retina, conjunctiva, limbus and cornea[4].

In this case recurrent hyphaema was due to iris involvement that was not confirmed

by detail examination in a remote area. Due to orbital involvement, this patient developed proptosis in the left eye. Due to intracranial extension of the orbital lesion, patient developed headache and vomiting due to raised intracranial pressure. Decrease vision in the right eye may be due to secondary optic atrophy and phthisical left eye may be due to post operative orbital degeneration.

The etiology of juvenile xanthogranuloma is not fully known. The papules and nodules of the skin in this disease represent collection of langerhan cell histiocytes. Cells of origin are dermal dendrocytes. Some author reported the granuloma reaction due to physical or infectious etiology[5-6]. Inhibition of cellular apoptosis appears to play a minor role in the growth of xanthogranuloma[7].

The appearance of giant cells and foamy lipid –laden histiocytes generally occurs later and apparently produced by histiocytes.

Serum lipid levels are normal and remain normal. 50% patients with ocular involvement have skin lesion that is totally absent in this case.

Ideal pharamacotherapy in a early case is steroid that can be given topical subconjunctival, intralesional or through systemic route.

But in this case with orbital involvement with intracranial extension, surgical normal was done to safe the patient's life.

Radiotherapy is also effective in uveal lesion with secondary glaucoma. Management of orbital xanthogranuloma by methotrexate had also been reported.

Conclusion

Juvenile xanthogranuloma is the most frequent cause of recurrent hyphaema in children.. The risk of morbidity is high with ocular involvement. Although cutaneous lesions are self limiting, extensive ocular involvement may create vision threatening and even life threatening condition. So, early diagnosis with appropriate examination and investigation is mandatory.

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A case of secondary glaucoma due to posterior chamber intraocular lens in anterior chamber

J U Mahmud[1], ASM Moinuddin[2], M J Alam[3], A. Khanam[4]

Introduction

Glaucoma is a chronic progressive optic neuropathy characterized by optic nerve head damage and corresponding visual field defect in which intraocular pressure is a major risk factor.[1]

It is the second leading cause of blindness after cataract.[2]

In post traumatic glaucoma[3], glaucoma develops after various types of injuries like concussion, contusion, penetrating injury and chemical burn. Prompt and meticulous management can prevent development of glaucoma.

A blunt non-perforating injury may cause lens opacification that is traumatic cataract either as an acute event or as a late sequel. Rupture of the lens capsule generally leads to rapid hydration of the lens cortex causes milky white cataract to form. Lens protein may leak in the aqueous and vitreous and may cause uveitis and /or vitreitis leading to secondary glaucoma. Secondary glaucoma may also be developed after ACIOL implantation.

We mention a case of secondary glaucoma due to posterior chamber intraocular lens in anterior chamber.

Case report

A 25 years old male patient by profession agricultural worker named Abdur Rahim came from sirajgonj on 10-03-11 with the complaints of painful red right eye. His vision was grossly diminished. He gave his history of cataract operation 6 months back in a local hospital following a trauma about 1 year back. On examination his vision was found hand movement, ciliary congestion, pseudophakia (anterior chamber intraocular lens), there were cells and flare in anterior chamber and raised intraocular pressure (34 mm of Hg). Patient was so anxious and worried that he was willing to enucleate the painful eye. He was admitted into eye ward, East-West Medical College Hospital for urther management.

On conservative treatment by topical corticosteroid and moxifloxacin, and a short course of Tab. acetazolamide supplemented by tab. Potassium, his intraocular pressure reduced to 24mm of Hg and anterior chamber reaction reduced. After remission of acute stage, it was found that a posterior chamber intraocular lens (PCIOL) was implanted in anterior chamber.

On this conservative treatment when eye become quite, he was released from hospital. But he came back after 2 weeks with the same condition i.e. painful red eye with raised Intraocular pressure and mild anterior chamber reaction. So he was readmitted and after a conservative treatment when again eye become quite, he was operated for extraction of posterior chamber intraocular lens from anterior chamber. Implantation of ACIOL and Trabeculectomy also done in right eye under local anaesthesia at the same sitting. After 6 weeks post operative treatment, his eye become quite and intraocular pressure reduced to 17.3 mm of Hg. He was followed up for 2 years and found happy with workable corrected vision 6/12 and N6.

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Discussion

Secondary glaucoma is common in ophthalmic practice because of high incidence of ocular infection, inflammation, complicated cataract surgery and trauma. Secondary glaucoma may be open angle or angle closure type. Lens induced glaucoma may be due to phacomorphic, phacolytic, sublaxation or dislocation of lens. Pseudophakic and aphakic glaucoma are among the commonest form of a secondary glaucoma.[4] Secondary glaucoma may be due to secondary angle closure by pupillary block or secondary open angle due to presence of retained cortical matters, pigment dispersion, vitreous in anterior chamber or late trabecular meshwork fibrosis.[5] In our case patient was suffering from painful red eye with reduced vision and raised intraocular pressure. On conservative treatment inflammation subsided, even that intraocular pressure was raised and was not well controlled by drugs, patient was also unwilling to use long term drugs due to poor economic condition and poor result of drugs. Here c-loops of posterior chamber intraocular lens blocked the maximum area of anterior chamber angle mechanically and also due to anterior chamber inflammatory reactions leading to trabeculitis may cause trabecular blockage which may had impaired aqueous drainage. So there was chronically raised intraocular pressure. It was managed by a combined intraocular surgery done under local anaesthesia, extraction of posterior chamber intraocular lens from anterior chamber and implantation of anterior chamber intraocular lens in anterior chamber with a drainage operation–trabeculectomy. All the procedures and post operative managements were quite smooth and uneventful.

Conclusion

Pseudophakic patients with ACIOL should follow up properly for long term. Posterior chamber intraocular lens is not only unacceptable for anterior chamber implantation, also harmful for patients eye health.

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Bilateral nanophthalmos, angle closure glaucoma and pigmentary retinal degeneration –a new syndrome?- A case report

S. Begum[1], Z. Wadud[2], S.J. Kabir[3], S. Alam[4], S.N. Hassan[5]

Abstract

Nanophthalmos is a relatively rare genetic abnormality characterized by bilateral small eyes associated with shortened axial length & high hyperopia without other ocular or systemic malformations. Patients with nanophthalmos are prone to primary angle closure glaucoma in adulthood. Management of glaucoma in these patients is challenging. Here we are reporting a case of nanophthalmos with associated angle closure glaucoma & pigmentary retinal degeneration-a rare combination. Patient presented to us with profound loss of vision in both eyes. He had trabeculectomy in right eye & laser peripheral iridotomy in left eye done elsewhere. Ocular examination revealed characteristic finding of nanophthalmos with poor vision in both eyes(right > left) & raised intraocular pressure(IOP) in left eye. We performed trabeculectomy with mitomycin-C & cataract extraction in left eye & kept it aphakic. Patient had a relatively good vision & well controlled IOP till date. We are keeping a good follow up of the patient as it represents a distinct combination of rare features, probably a new syndrome .

Introduction

Nanophthalmos is a rare genetic disease, characterized by a small eye secondary to compromised growth[1]. Nanophthalmos is derived from the Greek word nano, meaning dwarf, and nanophthalmic eyes typically present very high to extreme axial hyperopia without other obvious structural defects[2]. Nanophthalmos is usually characterized by bilateral symmetrical small eyes, associated with: shortened axial length (20mm or less; at least two standard deviations below age-matched controls), high corneal curvature, high lens/eye volume ratio with narrow iridocorneal angle, high hyperopia (ranging from +8.00 to +25.00) and scleral thickening3. Patients with nanophthalmos are prone to primary angle closure glaucoma in adulthood, result of the disproportion between the normal increase in lens volume and a smaller axial length. Diagnosis and follow up are rather difficult due to a small disc, where even a small cup may be a sign of glaucoma, along with inaccurate visual field testing, because many patients have high plus lenses and reduced BCVA4. Management of glaucoma in these patients is challenging. Glaucoma filtration surgery is considered as the last choice because of the high incidence of intraoperative and postoperative complications5,6

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Case History

A 48 years old male born of a non consanguineous marriage presented to the glaucoma clinic of Ispahani Islamia Eye Institute & Hospital with the complaints of decreased vision in left eye for 4 -5 yrs & profound loss of vision in right eye for the last 10-12 yrs. He also gave H/O of glaucoma filtration surgery RE in 1992 & laser peripheral iridotomy LE in 1992. Pateint had used high hypermetropic glasses since the age of 10 yrs. Regarding family history one of his brothers has high hypermetropia.

On ocular examination his BCVA RE was PL PR (inaccurate) & LE vision was HM & PR present in all quadrant. He was an average built male and general examination showed no systemic

abnormality. She had deep-set eyeballs, narrow palpebral apertures & mild ptosis. Intraocular pressure of RE was 10 mm of Hg, slit lamp examination RE showed thin cystic bleb, non patent peripheral iridotomy, pupil was constricted, irregular ,non reacting, 360 degree posterior synechiae with complicated cataract & gonioscopy revealed 360 degree closed angle with multiple broad peripheral anterior synechiae., IOP of LE was 28 mm of Hg, slit lamp examination revealed non patent peripheral iridotomy, pupil was constricted, non reacting, 360 degree posterior synechiae, complicated cataract &

gonioscopy revealed 360 closed angle e PAS at supero-nasal part. His axial length RE was 17.49mm & LE was 15.84mm, intraocular lens power RE was 34.23 & LE was 38.50.Ultra sound B-scan both eyes showed choroidal thickening with short axial length. We treated this case as angle closure glaucoma with complicated cataract with nanophthalmos both eyes with status post trabeculectomy RE & laser peripheral iridotomy LE.

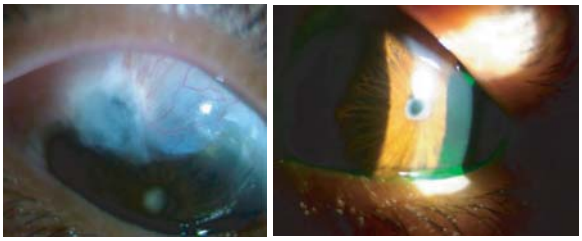

Fig. I : Slit lamp photographs of the right and left eyes

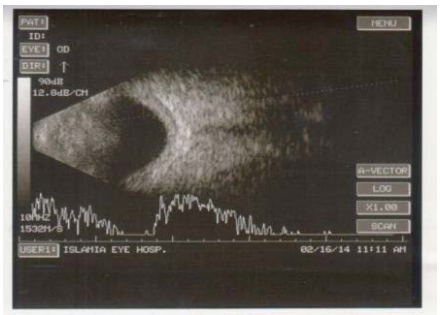

Fig. II : B-Scan ultrasonography- echogenic vitreous cavity with thick choroid & short AL

For managing this case we first started both systemic & topical antiglaucoma medications ,topical steroid & atropine eye drop & planned for trabeculectomy with mitomycin-C with synechiolysis + SICS with PCIOL LE. We did trabeculectomy with mitomycin -C+ SICS e anterior vitrectomy,IOL not given as there was zonular dehiscence at 9o' to12 o' clock with vit. prolapse . on 1st POD BCVA was 6/60 with

+20D & IOP was 15 mm of Hg,formed bleb & fundoscopic examination revealed hazy view, small hyperemic disc & pigmentary changes along blood vessels.

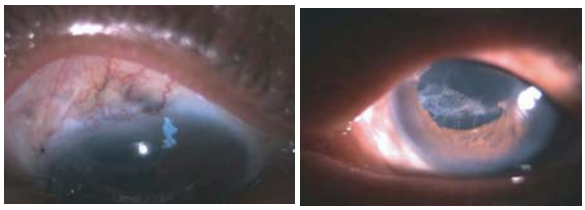

Fig. III : Slit lamp examination of LE (1st POD)

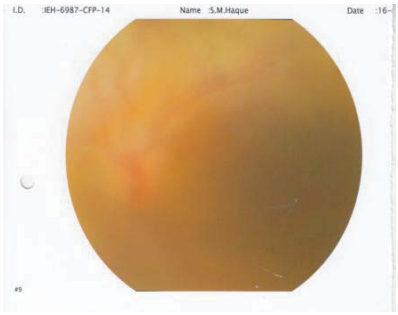

Fig. IV : Colour fundus photography of LE

With the above findings a final diagnosis of angle closure glaucoma in nanophthalmos associated with pigmentary retinal degeneration was established.

Discussion

Nanophthalmic eyes are frequently associated with angle closure glaucoma7.The association of angle closure glaucoma with pigmentary retinal dystrophy is also not infrequent 8.The simultaneous occurrence of nanophthalmos,

angle closure glaucoma and pigmentary retinal dystrophy has been conclusively documented by Ghose et al9, who concluded that the triad could be a new syndrome. Subsequenly in 1987, MacKay et al10 reported a pedigree of a family with seven related patients who had the combination of pigmentary retinopathy, cystic macular degeneration, high hyperopia, nanophthalmos and angle closure glaucoma. They concluded that these associations represent a distinct recessively inherited syndrome. Interestingly enough, this syndrome was reported many years ago by Hermann11 In 1958, he described a pedigree with 13 affected members in four generations and the inheritance was autosomal-dominant. Nanophthalmos is often associated with varying degrees of angle-closure glaucoma. A relative pupillary block etiology secondary to posterior 'pushing' mechanism is the most common cause of angle closure glaucoma, which eventually leads to peripheral anterior synechia (PAS) formation. The anterior chamber angle can also be closed by physical displacement of the peripheral iris by anteriorly rotated ciliary processes when nanophthalmos presents with annular ciliochoroidal effusion and ciliary body detachment 12. Response to medical treatment is poor and miotics may even make the condition worse by relaxing the lens zonules in these patients. Laser iridotomy is very beneficial in the early stage of glaucoma to eliminate the pupillary block component before the occurrence of PAS formation. ALPI is another laser treatment of choice if the anterior chamber remains appositionally closed after iridectomy13.When PAS have developed, intraocular surgery may be required. Surgery is considered as a last resort because it is often followed by a considerable number of complications. Sudden decompression of the globe during surgery may trigger the development of massive uveal effusion, which may lead to secondary retinal detachment, intraocular hemorrhage and malignant glaucoma and loss of vision. We modified our classical trabeculectomy technique with preplaced scleral flap sutures, tight suture closure and leaving viscoelastic material in the anterior chamber to overcome intraoperative choroidal effusion.

Cataract surgery in nanophthalmic eye presents an array of challenges even to seasoned cataract surgeons. Pre-op, short eyes raise uncertainty with IOL power calculation. Intraoperatively, a shallow anterior chamber, small corneal diameter, and positive posterior pressure can conspire to make maneuvers within the anterior segment difficult and hazardous to the endothelium, iris, and posterior capsule. Extreme axial hyperopia, with high dioptric IOL requirements, may complicate lens implantation. Phacoemulsification in an eye with a shallow anterior chamber can be challenging due to limited working space. Often, a cohesive viscoelastic such as Healon5 can be used to deepen the anterior chamber. In extreme cases, when this measure is inadequate, a pars plana vitrectomy tap and the removal of a small amount of vitreous can help to create space and deepen the anterior chamber14.

The high refractive errors leading to ametropic amblyopia and macular changes are the leading cause of reduced vision in childhood nanophthalmos. In contrast, the leading causes of visual loss in adults with nanophthalmos are glaucoma and the high rate of complications following intraocular surgery.

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