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FREE PAPERS

From the desk of Editor in Chief

Editorial

Undergraduate Ophthalmology Curriculum: Deserves Need Based Development to Address Vision-2020: The Right to Sight

Bangladesh, a thickly populated country over 160 million inhabitants of which 72% are rural, farming is main occupation, 40% live below National poverty level and 44% are illiterate[1]. Rural people are poor, illiterate and under privileged and they are backbone of agri-based economy of Bangladesh and produce food for us. There are approximately 905 Ophthalmologist in Bangladesh, 45% resides in Dhaka,1 to 3 at district level and virtually none at upazilla level for 3.5 lac population[2] whereas WHO recommends one for 50,000 in developing countries [3]. The right to sight is same for urban and rural people but reality is there is virtually no Ophthalmologist for rural 72% people. Last 10-15 years trend of producing Ophthalmologists and their distribution nationwide is poor. At this situation graduate physician working in rural area can play a great role at least to give primary eye care for unreached people, but do they really?

General physicians' (GP) view their undergraduate ophthalmic medical education as inadequate and is reflected in their confidence and understanding[4] Inadequate clinical exposure and lecture at the undergraduate level is important reason why many GP do not treat the eye conditions but refer them to eye specialist[5]. Medical education in South East Asia Region (SEAR) follows legacy of colonial past and perceived to be divorced from the real needs of the people[6], also true for ophthalmology.90% common eye conditions are avoidable but emphasis on Primary Eye Care (PEC) is inadequate[5].

Study conducted by us on Health Care Seeking Behavior of Ocular Injury Patients (n 520) at Dristisheba O Gobeshona, Jamalpur, 2010 (January to December) reveals, patients sought first treatment 63.2% from Allopath & Haemeo village quack, medicine shop, Kobiraz and tried self-treatmen, 30.4% from Ophthalmologist and only 6.4% from Graduate Doctor (GP), indicates patients with ocular conditions are not going to GP as expected[7]. So our product (MBBS doctors) not serving purpose in the field of eye care in Bangladesh. Main stay is Ophthalmic Medical Education and its backbone, curriculum.

Ophthalmic education is the cornerstone to improve eye care globally. The curriculum represents the expression of educational ideas in practice. It includes all the planned learning experiences of a school or educational institution .Curriculum development is a continuous process and always open for critique[8]. Expert realizes the wide variability of educational standards, pattern and prevalence of diseases, social structures for provision of eye care in geographical regions. Therefore encourages continuous modification of curriculum according to the needs of different global communities[8]. Studies review on ophthalmological curriculum of South Asia and far east countries identify inadequacies in terms of duration, contents, KAS ratio, clinical and community posting, etc[5]. In Bangladesh ophthalmology training is a part of surgery and evaluation is done with surgery during professional examination[9].There is no alternative of skilled human resource[10]. We need efficient medical graduates to address the growing

local eye care needs for fulfillment of global commitment of elimination of avoidable blindness and Vision-2020, The Right to Sight. For which we need an effective training tool-the curriculum. A new ophthalmology curriculum was developed with the participation of ophthalmologists randomly selected (n 400) from entire Bangladesh using Delphi technique, study title: *Development of community based ophthalmology curriculum for undergraduate medical course in Bangladesh*[11].

WHO recommends that, the curriculum for ophthalmology at undergraduate level needs to be reconstructed urgently, community eye care needs more attention, community oriented and community based teaching should be an integrated part of curricula, community ophthalmology should have designated teaching hours, training of faculty and faculty student ratio should draw attention & prioritization of learning topics should be done[5].

Curriculum is product of consensus of all concerned. Through community need based development of ophthalmology curriculum and it's implementation maintaining a standard throughout nation to improve capability of graduate physicians during undergraduate education can ensure PEC and fulfill the obligation to serve unreached rural people to address the success of Vision-2020, The Right to Sight.

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Editorial

The Target IOP in glaucoma: How much 'low' is low?

Glaucoma ,affecting about 2% of the population, is the first , second or third leading cause of blindness in every country of the world[1]. In Bangladesh its prevalence over the age of forty years is stated to be 3.1%. Intraocular pressure is a continuous positive risk factor for optic nerve damage, and is the only factor that we can modify with treatment. Lowering IOP slows down the rate of glaucomatous loss of vision. This so called "Target" IOP determination is a notion that has gained widespread popularity globally. I would like to focus on the goal and treatment as well as the role of IOP in glaucoma progression.

Before we try to determine the target pressure in individual patients, rather individual eyes, let us ask ourselves, what is the goal of our treatment? Is it to stop retinal nerve fibre loss? or to prevent the loss of visual field? More importantly, do we want to retain the quality of life of our patients, and at what cost?[2]

With ageing, the thickness of the retinal nerve fibre layer decreases. So loss of RNFL is a normal phenomenon - which may not be stopped by lessening a single risk factor like IOP. So, let us say, the target is to halt overly rapid loss of RNFL. We have seen marked visual field defects to progress relentlessly downhill to extinction despite of an ideal IOP.

The steady progression of visual field defects may be due to IOP's that are still too high. Progression of visual field defects over many years is common, despite supposedly adequate treatment. This observation has led us to set target IOP's to increasingly lower levels. Now let us think of quality of life.

Experienced clinicians have observed that subjective well-being of a patient may greatly differ from the physician's judgment of the severity of a disease. People with grossly affected optic discs and poor visual acuities may manage remarkably well and quite a lot of patient with advanced visual loss may be little incapacitated, managing their day to day chores pretty well[2].

Thus, it appears that the real target of our treatment should be to find the best balance between the highest possible quality of life at the lowest possible cost, especially in countries like ours. Here 'cost' entails both financial burden and the side effects from the treatment itself.

For example, lifelong use of pilocarpine may be economically acceptable but it may reduce the quality of life by severely limiting the visual function of an individual. Again, glaucoma filtering blebs are uncomfortable to many patients. Also, to many of our patients, the price of a prostaglandin analogue, like latanoprost, Bimatoprost or Travaprost may well be beyond the budget.

A clinician may consider two different target IOP's in two eyes of the same individual. For example a patient may have the same high IOP in both eyes at presentation. If he has a unilateral pseudoexfoliation or unilateral traumatic angle recession, the target IOP for the more susceptible eye should be lower than the fellow eye.

People keen about definition's may get some additional satisfaction from the definitions provided by different authorities on glaucoma. In 1989 American Academy of Ophthalmology defined target IOP as the upper limit of stable range of pressures deemed unlikely to cause further optic nerve damage in a particular eye. The European Glaucoma Society developed their own definition in 1998: "The mean IOP obtained with treatment that prevents further glaucomatous damage" Brubaker settled on this piece of working definition: The IOP at which the rate of retinal ganglion cell loss is no greater than the age related loss.

In practice, we should set a target pressure for individual eyes of every patent at presentation depending on the IOP, visual field & the optic disc appearance. On subsequent follow ups, the target pressure should be readjusted after re-evaluation of effect, cost, compliance & quality of life of the patient.

If the IOP is 25 mmHg or above with normal visual field and optic disc, set the target IOP at 25 mmHg. But if either the field or the discs are glaucomatous, bring down the IOP by 30% or more. The same rule of thumb applies to IOP levels between 15 to 25 mmHg at first presentation. If the VF defects threaten fixation, set the target pressure at 15 mmHg or lower, whatever be the initial IOP.

If the IOP is already below 15 mmHg with progression of the disease, bring down the IOP even further, medically and surgically or combined. Some are reluctant to perform surgery for fear of inducing hypotonic maculopathy. Occasionally one has to take this risk even and surgically reduce the IOP to single digit values.

In a nutshell, IOP lowering needs to be individualized with the goal of preventing any decrease in the quality of life during the patients lifetime.

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

Dr Shams Mohammed Noman

Abstract

Purpose : *To document 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 (5-40) yrs. 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

. FCPS, ICO, FELLOWSHIP IN GLAUCOMA

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 contact goniolens; fundoscopic findings and any other notable ocular findings. The method of management was recorded. Diagnosis of JOAG was done based on a combination of history, gonioscopic findings, fundoscopic findings and IOP readings.

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

Table 1: 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 2: 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 3: Management of Intraocular Pressure

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

Table 4: 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 5: 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 6: 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)mm hg, enlarged C:D ration >.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 were arrived.

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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Clinical Features and Visual Outcome in Vogt Koyanagi Harada Syndrome (VKH)

Tariq Reza Ali, Mostafizur Rahman,M. Saiful Islam,
M. Arifuzzaman, Nazmun Nahar, Masud Hashmee

Abstract

Introduction: *Vogt-Koyanagi-Harada disease (VKH) is a systemic autoimmune disorder directed against antigens most likely associated with melanocytes present in such tissues as the choroid, the meninges, the inner ear, and the skin. It is characterized by bilateral, chronic, diffuse granulomatous uveitis, accompanied by characteristic neurological, auditory and integumentary features. The eye is typically the most involved organ, and visual sequelae are the most frequent and debilitating consequences of the disease.*

The first description of a whitening of eyelashes, eyebrows, and hair associated with ocular inflammation dates from 10th century Persia. However, it was not until 1932, when Babel appreciated that the features of anecdotal cases reported years earlier by Vogt (1906), Harada (1926) and Koyanagi (1929) overlapped and were in reality manifestations of the same disorder, which he termed Vogt-Koyanagi-Harada disease.

The incidence of VKH is highly variable worldwide. In Japan, VKH accounts for over 8% of uveitis. It is also a frequent cause of uveitis in certain Latin American countries, especially Brazil, where VKH is the main cause of autoimmune noninfectious uveitis. VKH is the most common uveitis diagnosis made in Saudi Arabia.4 In contrast, VKHis seen in only approximately 1% to 4% of all uveitis referrals in the United States. In South India VKH comprises 2.2% of all uveitis cases.

The disease usually affects patients between ages of 20 and 50 years; however, there have been reports of VKH in children, with one report of VKH in a 4-year-old child. Most series indicate that women are affected more often than men, although this sex predilection does not seem to hold true for Japanese patients.

There is no study in Bangladesh regarding demography of VKH in this part of the world. We wanted to report the presentation, initial findings, investigations, treatment modalities, results and complications in 25 consecutive cases of VKH syndrome attended in the retina services of a tertiary eye care centre of Bangladesh. We had the intention to share our experiences and also to compare our cases with other centers of the world. We wanted to propose a unified treatment protocol for VKH patients.

Introduction

Vogt-Koyanagi-Harada disease (VKH) is a systemic autoimmune disorder directed against antigens most likely associated with melanocytes present in such tissues as the choroid, the meninges, the inner ear, and the skin. It is characterized by bilateral, chronic, diffuse granulomatous uveitis, accompanied by characteristic neurological, auditory and

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Method

25 consecutive patients of VKH disease diagnosed at the retina center of Islamia eye Hospital were evaluated retrospectively. Diagnosis was made according to the new diagnostic criteria accepted in the first International Workshop on Vogt-Koyanagi- Harada disease, 2001.[13]

Complete Vogt-Koyanagi-Harada Disease

1. No history of penetrating ocular trauma
2. No evidence of other ocular or systemic disease.
3. Bilateral ocular disease (either a. or b. below):

a) Early manifestations

i. Diffuse choroiditis manifested with either

1. Focal areas of subretinal fluid, or
2. Bullous serous subretinal detachments.

ii. If equivocal fundus findings, then both:

1. FA showing focal delayed choroidal perfusion, pinpoint leakage, pooling of luorescein within subretinal fluid, and optic nerve staining
2. Ultrasonography showing diffuse choroidal thickening without evidence of posterior scleritis

b) Late manifestations

i. History suggestive of findings from 3a, or both ii and iii, or multiple signs from iii

ii. Ocular depigmentation

1. Sunset glow fundus, or
2. Sugiura's sign

iii. Other ocular signs

1. Nummular chorioretinal depigm entation scars, or
2. RPE clumping and/or migration, or
3. Recurrent or chronic anterior uveitis.

4. Neurological/auditory findings:

a) Meningismus
b) Tinnitus
c) Cerebrospinal fluid pleocytosis.

5. Integumentary findings (not preceding CNS/ocular disease)

a) Alopecia
b) Poliosis
c) Vitiligo

Incomplete Vogt-Koyanagi-Harada Disease
Criteria 1 to 3 and either 4 or 5 from above.
Probable Vogt-Koyanagi-Harada Disease
Isolated ocular disease (only criteria 1 to 3 from above).

Patients from January 2009 to October 2009 were included with a minimum six months of follow up.

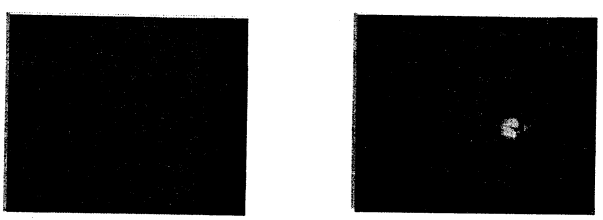


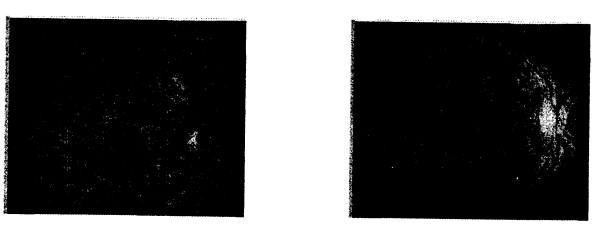
Fig 1: Posterior segment findings in VKH patients

The medical records were reviewed for demographic data, clinical features, investigations, treatment and follow up, initial and final visual acuity etc. The complications noted in the sheet also sorted out.

Results

There were 18 female (72%) patients with 37 years of mean age (SD ± 14.23). The median follow up was 11 months (6 to 16 months).13 of 25 (52%) patients came to our institute within 1 month of symptoms.12/25 (48%) presented after 3 or more months. The median duration of symptoms was 30 days (range 1 day to 1 year). 14(56%) patients presented with anterior uveitis. Posterior segment findings were vitritis in 12 (48%) patients, disc oedema in 17 (68%) patients, focal SRF in 13 (52%) patients, bullous serous RD in 14 (56%) patients, choroidal detachment in 4 (16%) patients. Delayed posterior segment findings were : sunset glow fundus in 5/25 920%) patients, nummular scar 2/25 (8%) patients and RPE pigmentary changes in 6/25 (24%) patients. 4 patients showed extraocular manifestations: tinnitus 2/25 (8%), meningismus 1 (4%) and poliosis 1/25 (4%). 20 of 25 patients underwent B-scan ultrasonography, 18 (90%) had choroidal thickening, localized serous retinal detachment was present in 15 (75%) patients and choroidal detachment was present in 5 (25%) patients. Fluorescein angiography was performed in 13 of 25 patients. Multiple areas of hyperfluorescence with leaks was present in 9 (69.23%) patients, large hyperfluorescent areas was present in 6 (46.15%) patients, pooling of dye was present in 8 (61.5%) patients and disc staining was present in 9 (69.23%) patients.

Patients were treated with oral prednisolone and intravenous methyl prednisolone. Oral steroid (1 mg/kg) was given in 24 (96%) patients. Median duration of treatment was 9 months (range 3 to 17 months). Three pulses of 500 mg to 1000 mg intravenous methyl prednisolone was given in 11 (44%) patients. Additional immunomodulator (azathioprine) was given in 12 (48%) patients. Median duration of immunomodulator was 14



months (range 1-48 months). No patient got cyclophosphamide or methotrexate and obviously no patient received two immunomodulators along with oral steroid.

The presenting visual acuity was ?20/400 in 33 eyes, 20/100 to 20/200 in 9 eyes, 20/50 to 20/80 in 6 eyes and ? 20/40 in 2 eyes. Visual outcome at final follow up was ? 20/40 in 12 eyes, 20/50 to 20/80 in 16 eyes, 20/100 to 20/200 in 13 eyes and

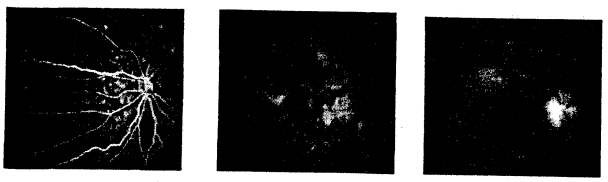


Fig 3: FFA findings of a VKH patient

?20/400 in 9 eyes. Notable complication were cataract in 12 eyes, glaucoma in 4 eyes, macular scar in 4 eyes, retinal detachment and phthisis bulbi in 1 eye.

Discussion

VKH is not uncommon in our country. Proper diagnosis is essential to start prompt treatment. No matter the form of the disease the patient manifests, three conditions must be present for the diagnosis of VKH: (1) no previous history of penetrating ocular trauma (either surgical or accidental); (2) no clinical or laboratory evidence suggestive of other ocular disease; and (3) bilateral ocular involvement. These criteria reinforce the requisite of thorough history, physical

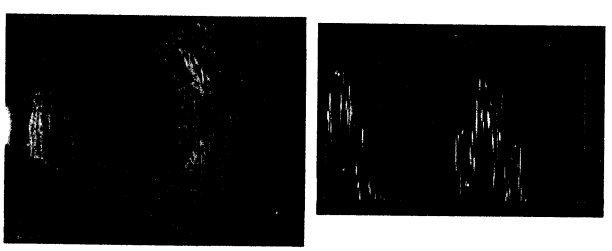


Fig 4: B-scan impression of a VKH patient

examination, and appropriate ancillary testing in making the appropriate diagnosis.

The standard initial therapy for VKH is prompt and aggressive use of systemic corticosteroids, to which the disease is especially responsive, particularly in the early stages (Figure 6). Typical dosing regimens range from 1.0 to 2.0 mg/kg/day of oral prednisone or 1000 mg of intravenous methylprednisolone for 3 days, followed by high-dose of oral corticosteroids. Early therapy with a slow taper tailored to the clinical response, usually over a minimum of 6 months, has been shown to improve the prognosis by reducing the length of disease, increasing the incidence of a convalescent phase, and decreasing the extraocular manifestations.[14] A recent multicenter international study on the treatment of VKH demonstrated that high-dose oral corticosteroids and intravenous corticosteroids were equally effective, with similar outcomes in both treatment groups.[15]

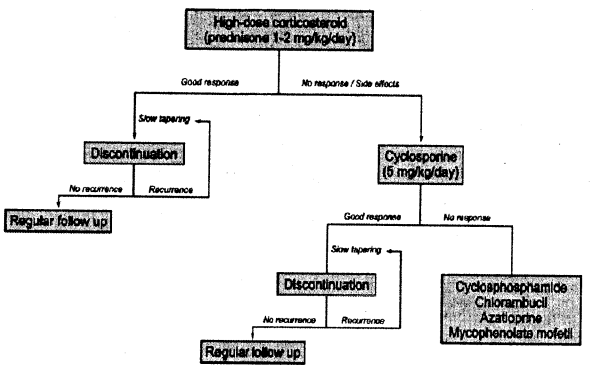


Fig. 6: Standard treatment protocol

The standard treatment protocol is given below:

We prescribed Tablet prednisolone in almost all patients. Intravenous methyl prednisolone was not given in all patients. We decided to infuse methyl prednisolone in patients with poor vision (<20/400) at presentation, but we couldn't follow it in all patients as there were more than one treating physician. We found good results if the patients got prompt treatment.

Recommendations

We recommend B-scan ultra sonogram and FFA in all patients along with dilated indirect ophthalmoscopy to diagnose the case of VKH. Also our recommendation is to admit the patient for pulse therapy if the presenting vision is <20/400 and to continue systemic steroid after 3 days. If the vision does not improve after 2 weeks and if the patient is intolerant to systemic steroid, we should add other immunomodulator and to taper systemic steroid. We recommend not stopping the immunosuppressant at least for 1 year as the disease can flare up in any time. Periodic follow up and systemic evaluation is also essential to combat this devastating disease.

Limitations of the study

This is a retrospective analysis. All cases were not possible to include in this study. There were also different treating ophthalmologists. Proper management protocol was not prepared and was not followed in all patients. We recommend a prospective study in our country to enrich our knowledge and skill and also to share our experiences with other parts of the world.

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Relationship of central corneal thickness (CCT) with refractive error

1. Dr. Md. Ruhul Amin Khan

Abstract

The purpose of this study was to determine the variation of the central corneal thickness (CCT) with spherical equivalent refractive error. A total of sixty (N=60) subjects of which male 42(n=42) and female 18(n=18) within 15-66 years with mean age of 43.6 ±13.2 years were taken for this study. Among the subjects, 30 had myopia and 30 had hypermetropia. The central corneal thickness was assessed with ultrasonic pachymeter and refraction done by Topcon autorefractometer and trial lens set.The mean CCT was 513.30 micrometer,with a standard deviation of 45.15 (range 497-529) micrometer in right eyes and 514.47 micrometer with a standard deviation of 45.74 (range 498-531) micrometer in left eyes in myopia & in hypermetropia that was 523.70 micrometer,with a standard dediation of 35.82 (range 510-536) micrometer in right eyes and 529.90 micrometer with a standard deviation of 39.17 (range 516-544) micrometer in left eyes. The mean myopic refractive error was -3.55D,with a standard deviation of -2.24D (range -2.75 to -4.35) diopter in right eyes and -3.43 D with a standard deviation of -2.46 (range -2.55 to -4.31) diopter in left eyes in myopia & in hypermetropia that was +150 D ,with a standard dediation of +0.89 (range +1.18 to +1.82) micrometer in right eyes and +1.44 D with a standard deviation of +0.30 (range +1.33 to +1.55) diopter in left eyes.Result reveals that there were no linear correlation between CCT and spherical equivalent refractive errors(r=0.28,p=0.03 in myopia & r=0.004 ,p=0.96 although the association between CCT & myopia was slightly significant. The central corneal thickness was weakly correlated with age (p=0.48); with increasing age the central corneal thickness decreases.The central corneal thickness was not affected by gender.

Introduction

A thin CCT has been reported to be a risk factor for developing POAG among ocular hypertensive eyes. A thin scleral bed of lamina cribrosa seen in deeply excavated optic nerves in glaucomatous eyes is a quite common finding in advance glaucomatous eyes. Association between thin cornea and weak sclera contributing to vulnerability of lamina cribrosa has been postulated. (Eghosasere et al, 2007) Corneal thickness is important and consider for two reasons. First, corneal thickness affects the measurements of IOP so that the measured IOP may be inaccurate if the corneal thickness is not average. The actual average central corneal thickness (CCT) is approximately 544 µm. IOP about[5] mmHg lower than measured for each 100 µm if the cornea is thinner than normal (Brand et al, 2001). The thin central cornea itself is associated with more severe glaucoma. The ocular hypertension treatment study (OHTS) identified reduced central corneal thickness as a risk factor for glaucoma in patients with IOP between 24 mmHg and 32 mmHg (Ehlers et al,1975).

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Primary open angle glaucoma represents a significant public health problem.. Blindness prevalence for all types of glaucoma was estimated at more than 8 million people; with 4 million cases caused by POAG. The different types of glaucoma were theoretically calculated to be responsible for 15 % of blindness, placing glaucoma as the third leading cause of blindness worldwide following cataract. (Glaucoma; AAO: 2004-2005)

Several risk factors and not all risk factors are known; increase the likelihood of the development of POAG. Besides increased IOP, factors known to be associated with an increased risk for the development of glaucoma include advanced age, decreased corneal thickness, racial background and a positive family history.

The importance of decreased corneal thickness has only recently been appreciated and may be more than just an artifact that causes under reading of true IOP. Thinner cornea may be correlated with underlying factors(Kanski, 2007: Clinical ophthalmology, 6th edition). An unexpected finding of the Ocular Hypertension Treatment Study (OHTS) was that CCT proved to be a most potent predictor factor for the development of POAG-patients with CCTs only 40 micrometer thinner than the OHTS average had a 71 % increased incidence of POAG end points. This risk relationship was independent of IOP or base line cup to disc ratio. Most strikingly in the multivariate model, the increased incidence of POAG among African-American OHTS subjects was explained by thinner CCT and increased CD ratio at baseline.

Corneal thickness affects the measurement of IOP. Thicker corneas resist the indentation inherent to nearly all methods of IOP measurement including applanation, air-puff, tonopen and pneumotonometry. Average corneal thickness is approximately 534 micrometer (optical measurement) or 544 micrometer (US measurement) in normal eyes. It has been found to be thicker in groups of patients with ocular hypertension and thinner in patients with normal tension glaucoma. The exact effect of CCT has on IOP is not known, but IOP has been estimated to increase to 2-7 mmHg at 100 micrometer of increased corneal thickness (Bruce, Text Book of Glaucoma). Doughty and Zaman reported that the mean CCT in normal eyes was 534 micrometer in optical pachymetry while for ultrasonic pachymetry, the mean CCT was 544 micrometer (Doughty et al ,1998) . The CCT has been considered as a masking factor hiding elevated IOP rather than independent risk factor.

Myopia was not predictive of the development of POAG in the OHTS.In contrast,several well-performed case-control and population-based studies have reported an association between myopia and POAG.(Perkins ES et al,1982)Nemesure et al found that CCT was directly related to refractive error, although no systemic alteration in CCT was found in myopia (Nemesure et al,2003) . Similarly, Lene and Neils aimed that the process by which myopia progresses does not to a measurable degree influence the CCT(Lene et al, 2005). Over time, it has been shown that myopic refractive errors are associated with thin CCT. Duch et al posited that high ametropia may bias the measurement of CCT(Duch et al ,2001). Studies that have attempted to investigate the effect of refractive errors on CCT have reported conflicting results. One report showed no correlation between corneal thickness and the level of myopia, whereas another study found the cornea to be thinner in more myopic eyes (Journal of Glaucoma ,June 2006). High myopia is a moderate risk factor of ocular hypertension because myopic discs are more susceptible to glaucomatous damage at a lower IOP than emmetropic discs.. But in another study, there are no linear co-relation between CCT and spherical equivalent errors (Cho et al ,1994)

Measuring CCT may aid the ophthalmologist in identification of glaucoma patients at high risk of

progression. (Archives of Ophthalmology,January 2004) Another study suggests higher 54 suptibility to glaucoma of highly myopic eyes versus non-highly myopic eyes (Investigative Ophthalmology and Visual Sciences ,2000).

Since CCT may inversely influence the amount and rate of progression of glaucomatous optic nerve damage ; it is the purpose of present study to evaluate the variation of CCT with spherical equivalent refractive error.

Rationale of the study

A thinner CCT measurement predicted the development of POAG.. Case-control and population based studies have reported an association between myopia and POAG. But there are conflicting report regarding association of CCT and refractive error.We can identify patient for developing POAG who are more likely to benefit from early medical treatment and regular follow up. Measuring CCT may aid the ophthalmologist in identification of glaucoma patient at high risk of progression and also to estimate target pressure.

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

Objective of the study :

General objective

- To investigate the association of CCT with refractive error.

Specific objectives

- To examine relationship between CCT and myopia
- To compare relationship of CCT with hypermetropia
- To estimate association of CCT with age and gender

Ultimate Objectives

- To determine risk factors for developing POAG by measuring CCT
- To estimate target IOP
- The information will be used for refractive surgery

MATERIALS AND METHODS

Type of study: Prospective observational study

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

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

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

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

Inclusion criteria

- Age between 15 -66 years
- Healthy patients except refractive errors .

Exclusion criteria

- Previous ocular surgery
- Ocular pathology such as keratoconus
- Recent contact lens wearer in previous 2 weeks
- Patients with significant astigmatism

Methods

- The ocular parameters of 60 consecutive eyes were measured:
- All the subjects were first refracted to determine their refractive status. Measurements of refractive status were taken with the autorefractor after adequate preparation of subjects.
- The central corneal thickness was assessed with the pachymeter . Measured

CCT for the subject was taken as the average of ten different readings and recorded in microns (μm)

- Analysis was carried out to examine the relationship:

- CCT and myopic eyes
- CCT and hypermetropic eyes
- CCT between male and female
- CCT among different ages

Data management analysis

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

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

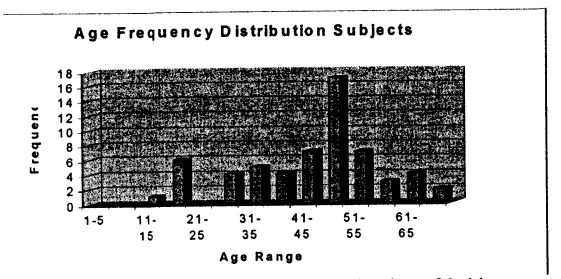

Figure 1 : Age Frequency Distribution of Subject

Results

A total of sixty (N=60) subjects consisting of males (n=42) and females (n=18) within 15-66 years with mean age of 43.6 ± 13.3 years were used for this study. Of all the subjects, 30 had hyperopia and 30 had myopia.The mean hyperopia was +1.50 ± 0.89D (OD) and +1.44± 0.30D (OS) while the mean myopia was -3.55 ± 2.24D (OD) and -3.43 ± 2.46 D (OS) [Table 1].

Although readings were obtained for both eyes, to

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

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

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

Myopia:OD	513.30 ± 45.15	497-529
OS	514.47 ±45.74	498-531
Hyperopia:OD	523.70 ±35.82	510-536
OS	529.90 ±39.17	516-544

avoid duplication of results only the readings of the right eye (OD) were used for analysis.

Table 2 shows the mean, confidence interval of CCT of hyperopes and myopes respectively. Pearson correlation coefficient showed that although the association between CCT and MSEM (r=0.28;p=0.03) was significant but CCT and MSEH (r=0.002) was not significant; the linear regression was however not significant in both cases.) The correlation between CCT and age was equally weak (r=0.09;p=0.48).

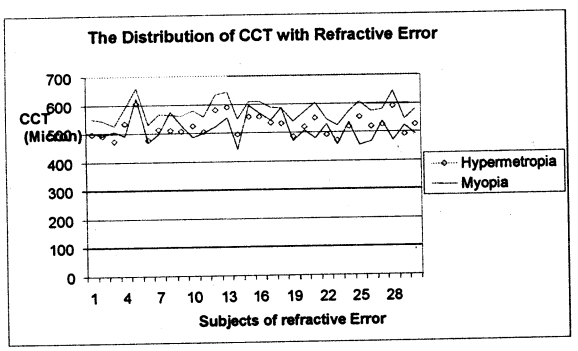

Figure 2 : Distribution of CCT with Refractive Error

Figs 2-3 show the scatter plot of CCT vs. Refractive Error, CCT vs. Age respectively.

There was no significant difference in mean CCT between males and females (Unpaired t-test: t=2.01, df=58, p=0.049). Summarily, CCT was not affected by gender.

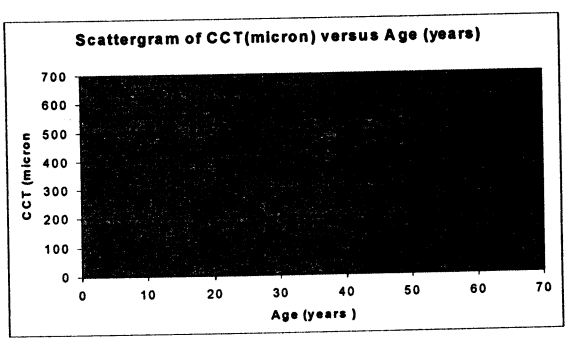

Figure 3 : Scattergram of CCT versus Age

DISCUSSION

.This study reveals that there was a slight association between CCT and MSEM (r=0.28, p<0.05) but the linear regression was not statistically significant. Similarly, there was a slight association between CCT and MSEH (r=0.004, p<0.05) and the linear regression was equally not significant. Summarily, no linear prediction can be made about CCT and mean spherical equivalent refraction. This was consistent with findings of Lene et al who reported that CCT was not systemically altered in myopia. Similarly, Ehlers et al and Price and colleagues claimed that CCT does not appear to be correlated with refraction (Ehler et al,1975). On the contrary, Nemesure et al posited that CCT was directly related to refractive error, but high ametropia may bias the measurement of CCT(Nemesure et al,2003) . In this study it was shown that the difference in mean CCT between males (499.5 ±36.2?m) and females (534 ± 47.2?m) was not significant (Unpaired t-test: t=2.01, p>0.05) and the 95% confidence intervals were 495.5 – 524 ?m and 515 – 553 ?m respectively.. Summarily, CCT was not affected by gender. A slight association between IOP and CCT was found in the myopic group but the linear regression was not statistically significant (r=0.02). However, the association between IOP and CCT in the hyperopic group (r=0.07, p>0.05), no linearity can be predicted between IOP and CCT. There was a slight association between CCT and age (r=0.22, p<0.05), although the linear regression was not statistically significant (ANOVA: F=1.14, p=0.3). This was in line with the study of Lleo and colleagues who reported a non-linear correlation between CCT and age (r=0.083, p=0.065). Nemesure et al reported an inverse relationship between CCT and age. The effect of age suggests age-related corneal biomechanical changes (Nemesure et al,2003) .

Conclusion

In conclusion, this study has shown that there is no linear correlation between CCT and MSEM and MSEH. This means that CCT is neither affected by MSEM nor MSEH although there was negative association between CCT and Myopia. The slight association between CCT and age indicated a reduction of CCT with increasing age. It was also shown that CCT was not influenced by gender.ccv

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Problems to convince the patients for cataract surgery presenting to a hospital

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Dr.Md.Towhid Anwar

Abstract

Purpose: *To assess the barriers for the acceptance of surgery among patients with cataract and visual disability.* ***Materials and Methods:*** *A short-term descriptive study was conducted in patients with cataract presenting to a hospital. Socio-demographic data were entered in a proforma. An interviewer-assisted questionnaire, surveying knowledge about cataract and barriers to cataract surgery, was administered in Bengali.* ***Results:*** *There were 100 patients (53 men and 47 women); 14 were bilaterally blind (vision <3/60 in the better eye). Attitudinal barriers included: could manage daily work (71%), cataract not mature (68%), could see clearly with the other eye (64%), too busy (57%), female gender (37%), fear of surgery (34%), fear of surgery causing blindness (33%) or death (13%), old age (33%), it is God's will (29%) and worry about cost of surgery (27%). The barriers relating to service delivery, cost, and affordability included: insufficient family income (76%), not knowing another person who had undergone cataract surgery (26%), no one to accompany (20%), distance from hospital (20%) or from a main road (9%) and lack of transport (7%).* ***Conclusions:*** *Attitudinal barriers were reported more often, rather than issues of accessibility or cost. Eye care providers should address the identified barriers for increasing acceptance of surgery in the study area.* ***Keywords:*** *Barriers, cataract, healthcare utilization.*

Introduction

Age-related cataract is responsible for 48% of world blindness, which represents about 18 million people, according to the World Health Organization (WHO) and 60% in Bangladesh. By 2020, the elderly population is expected to double, further increasing the number of blind people. Strategies for reducing cataract backlog include increasing the number of cataract surgeries performed. However, despite rapid increase in the availability of quality services, surgical acceptance is still low in some segments of society. A few studies, mostly from other developing countries, have addressed barriers to accepting surgery for cataract. However, there are few studies in the Bangladeshi scenario, addressing regional issues. Determining barriers to use of eye care services is critical for planning strategies to prevent blindness. Ours is a secondary level, district hospital that caters mainly to low socio-economical patients of Jessore and surrounding districts. The hospital is government-funded and provides free service to patients. There are non-government hospitals and private ophthalmic centers in the area. Yet, we see many patients with advanced unilateral or bilateral cataract. The aim of this study was to determine possible reasons for the delay in acceptance of cataract surgery in such a set up.

Materials and Methods

This was a hospital-based, descriptive, short-term (3-months) clinical study. Inclusion criteria:

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consecutive patients, aged 45 years or above, with vision <3/60, the principal cause was cataract.

Patients were briefed in appropriate local language about the purpose and procedure of the study. Socio-demographic data were noted on a proforma and included age, gender, rural or urban residence, literacy, and employment. A questionnaire surveying knowledge about cataract and barriers to acceptance of cataract surgery was administered after written informed consent, in Bengali. The questions on barriers were devised from the existing literature and required yes/no responses only. Seventeen barriers relating to patient attitude, service delivery, cost, and affordability were investigated. The English-language version of the proforma and questionnaire is appended. To maintain uniformity and reliability of data collection, all questionnaires were interviewer-assisted by the same interviewer, The interviews were conducted in a separate room away from relatives and other patients.

The diagnosis of cataract was based on torchlight and distant direct ophthalmoscopy. Vision was assessed by Snellen's chart and the World Health Organization definitions of normal vision (best corrected visual acuity (BCVA) ?20/60 in the better eye), visual impairment (BCVA <20/60 but /10/200 in the better eye), and blindness (BCVA <10/200 in the better eye) were used.

Data were entered into an excel worksheet and each barrier that was reported by a patient was given a score of one point. Mean and median age of patients, mean duration of cataract and mean of total barriers per patient was calculated using the statistical package SPSS.

Results

The study included 100 patients (53 men and 47 women), with age ranging from 45-86 years (median 62 years). All the women in our study were exclusively homemakers. Distance from

Table 1 : characteristics of patients included in study

Gender	Male	Female	
	53	47	
Age	Range	Mean (SD)	
		63(9.74)	
Residence	Rural	Urban	
	51	49	
Literacy	Literate	Illiterate	
	44	56	
Occupation	Employed	Unemployed	
	21	79	
Cataract	Unilateral	Bilateral	
	49	51	
Stage of more advanced cataract	Immature	Mature	Hyper mature
	22	25	4
Vision in the better eye	? 20/60	<20/60to ? 10/200	<10/ 200
	33	53	14

hospital varied from 1 to 100 km. The socio-demographic and other characteristics of the patients are given in [Table - 1]. Patients had known that they had cataract for periods varying from 4 days to15 years (mean 11.6 SD 21.4 months). Ninety-seven patients knew that cataract was curable and 87 knew that surgery was mandatory for its cure.

Total barriers per patient ranged from one to 13; mean 6.2 SD 2.57. The frequency of reported barriers relating to patient attitude, service delivery, cost, and affordability are given in [Table - 2]

Table 2

Barrier	No. of patients who reported it as a barrier
Could manage to do daily work	71
Cataract was not mature	68
Could see clearly with other eye	64
Busy with work	57
*Being female	37
Fear of surgery	34
Old age	33
Fear that surgery could lead to loss of eye sight	33
Worry about cost of surgery	27
Fear that surgery could lead to death	13

*Reported by female only.

Discussion

The socio-demographic data show that the study included comparable numbers of men and women. The urban-rural representation was also equal.

Table 3

Barrier	No. of patients who reported it as barrier
Insufficient family income	76
Not knowing another person who had undergone cataract surgery	26
No one to accompany	20
Distance from hospital	20
Distance of home from a main road	9
Lack of transport	7

However, more than half the patients were illiterate and over 3/4^{ths} were unemployed reflecting the type of patients catered to by this tertiary-level teaching hospital.

In the present era of microsurgery, it is no longer necessary to wait for cataract maturity before operating; many phacoemulsification surgeons prefer operating on immature cataracts. In this study, patients had known about their cataracts for periods ranging from 4 days to 15 years. Moreover, more than half the patients with bilateral cataract had mature or hypermature cataracts. Fifteen years represents a disturbing delay in seeking intervention and gives impetus to the need to study barriers specific to a region in relation to its socio-cultural practices.

Fourteen patients with cataract delayed surgery even though they were bilaterally blind. This is surprising. However, other authors have reported that bilaterally blind patients often do not feel the need for cataract surgery. They tend to wait till complete dependency and lack of functional mobility ensues. Though coping mechanisms prompt these patients to deny their visual handicap, they nevertheless have considerable levels of anxiety and other psychological

problems.By delaying intervention, patients only make matters worse for themselves and their relatives. Getting cataract surgery done while there is still useful vision in the other eye allows the patient to travel unaccompanied and look after himself in the hospital, thus reducing the dependency and burden on relatives. Ophthalmologists have an important role in this regard. If patients who present for treatment are told to wait because cataract is not mature or advanced enough, other barriers may make it difficult for the patient to present again. Studies have shown that financial limitations rank high as reasons for not having cataract surgery. However, in our study, the major barriers (operative in >55% patients) were more often related to patient attitude (ability to manage routine work, cataract not mature, could see clearly with the other eye, busy with work), than to issues of service delivery or cost and affordability (insufficient family income). Although 76% of the patients reported insufficient family income as a barrier, only 27% were worried about the cost of surgery. Possibly, they were more concerned with indirect costs. Other studies have shown that indirect costs relating to loss of a day's income, delegating household responsibilities and transportation for both the patient and his attendant are important barriers to intervention. Efforts to reduce indirect costs include conducting operations in the patients' own villages or facilitating transport to and from the surgical facility.Illiterate and unemployed patients could also be benefited since it would take some of the pressure off relatives. Authors have suggested that training of health care workers or community members in recognition and referral of such patients might help them to seek intervention earlier, rather than later. Further studies in the Bangladeshi scenario are required to gauge the effect of such interventions on attendance for cataract surgery.

Barriers operative in 25-55% patients included fear of surgery or of surgery causing blindness, old age and not knowing anyone who had undergone cataract surgery. Thus, although 87% of the patients interviewed knew that cataract was curable by surgery, patients with cataract hesitated to use the services. Probably, fear and other factors were operative. Studies have shown that interaction with a patient who has undergone the operation could help in reducing fear and abolishing fatalistic beliefs that blindness was an inevitable part of old age or God's will.

In this study, women were greatly concerned that their gender prevented them from seeking intervention earlier. This suggests that they were aware of and unhappy with, the lack of importance given by the family to their health. Other studies have also shown that women avail of surgery at a later date than men. Authors report that due to poor self-esteem and low expectations, women try to carry on with their routine work even when severely visually handicapped.

Compared to attitudinal barriers, issues of accessibility were relatively unimportant in this study. Thus, distance from the hospital and of residence from a main road was cited infrequently as a barrier. Other authors have reported this as well. However, one of the limitations of this study is that the patients represent a hospital-based population. Thus, the barriers reported by them may be different from those of community members who never present at all. Moreover, the barriers are likely to be inter-dependent. Running multivariate regressions to predict all the barriers would have entailed lots of tests and were not done. The other limitation is the small sample size. Since this was a short term (3-months) project, the sample size was fixed at 100 patients. To reduce chances of systematic error in our study, the same interviewer interviewed all patients. By interviewing the patient away from relatives and other patients, we tried to reduce bias resulting from social pressures. This is likely to make the results of our study reliable.

This study of barriers to intervention in patients with cataract shows that while intervention may have become more accessible, there is a need to modify patient attitudes to make surgery more acceptable. Thus, attempts to enhance acceptance of surgery must be sensitive to regional attitudinal barriers.

Acknowledgement

Salauddin ahmed, superintendent, 250 bed general hospital,Jessore,Bangladesh.

Appendix [Figure-1]

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APPENDIX

S. no:	Name	Age/gender

Area of residence: Urban/rural

Distance from Delhi/this hospital:

Education level: None/primary/intermediate/high school/college

Occupation: None/homemaker/labor/skilled labor/private business/professional

Vision and cataract	Right eye	Left eye
Best corrected vision		
Stage of cataract	Immature	Immature
(by torch light and distant direct ophthalmoscopy)	Mature	Mature
	Hypermature	Hypermature

Assessment of patient's knowledge of cataract

1.	Do you know which disease you are suffering from?	Yes/no
2.	Is cataract curable?	Yes/No
3.	Is operation mandatory for curing this disease?	Yes/No
4.	When did you first come to know about this disease?	

Barriers relating to patient attitude: Did you delay treatment because

1.	You were afraid of undergoing an operation?	Yes/No
2.	You were worried about the cost of the operation?	Yes/no
3.	You feared that operation would lead to loss of eyesight?	Yes/No
4.	You feared that operation would lead to death?	Yes/No
5.	You could see clearly with the other eye?	Yes/No
6.	You could manage to do your daily routine work?	Yes/No
7.	Your cataract was not mature?	Yes/No
8.	You were busy with work?	Yes/No
9.	You thought blindness was God's will?	Yes/No
10.	You are a female?	Yes/No
11.	You are very old?	Yes/No

Barriers relating to service delivery, cost and affordability: Did you delay treatment because:

1.	You were living very far from the hospital?	Yes/No
2.	Your residence in the village/town is not linked to a main road?	Yes/No
3.	There is no transport from your residence to this hospital?	Yes/No
4.	You don't know any other person who has undergone this surgery in your village/town?	Yes/No
5.	No one could come along with you?	Yes/No
6.	your family income is not sufficient?	Yes/No

Variability of Ocular Manifestations of Intracranial Tumours present in different location of brain : A case series study

Dr. Md. Sibgatullah

Purpose: *The study was done to assess the ocular manifestations and diagnostic features of brain tumours, and to see the variation of clinical features occur in accordance with their location, among the patients presented at Chittagong Eye Infirmary and Training Complex (CEITC).*

Methods: *This is a prospective study done from November 2003 to June 2008 (56 months) at CEITC. 186 cases of primary brain tumours were diagnosed on the basis of history, clinical features and neuroimaging of brain.*

Results: *186 cases of primary brain tumors were detected during the 56 months of study. 37(20%) of tumours were situated in the anterior cranial fossa, which includes the cerebral hemisphere and diencephalons (thalamus and hypothalamus). 113(61%) of tumours were situated in the middle cranial fossa including the sella and parasellar region. 36(19%) cases of tumours were present in the posterior cranial fossa including the brainstem and cerebellum. Loss of vision was the main presenting complaint in 28(76%) of anterior, 103(91%) of middle and 32(89%) of posterior cranial fossa tumour patients. Good vision (BCVA 6/6 to 6/18 in both eyes) was present in 7 (46%) of anterior, 14 (12%) of middle and 9(25%) cases of posterior cranial fossa tumours. At presentation 12 (32%) of anterior, 26 (23%) of middle and 10 (28%) cases of posterior fossa tumour patients were blind, as their best corrected visual acuity (BCVA) was less than 3/60 in the better eye. Cranial nerve palsies were seen in 5 (13.5%) of anterior, 13(11.5%) of middle and 22(61%) cases of posterior cranial fossa tumours. Atrophic changes of the optic nerves were observed in 7 (9%) of anterior, 83 (73%) of middle and 5 (14%) cases of posterior cranial fossa tumours. Papillodema was seen in 28 (76%) of anterior, 12 (11%) of middle and 28(78%) of posterior fossa tumours. Nystagmus was seen in 2(5%) of anterior, 3(3%) of middle and 14(39%) of posterior cranial fossa tumour patients.*

Conclusion: *Intracranial tumours which produce adverse effect on vision, morbidity and mortality, should be diagnosed at early stage. Ophthalmologists may be able to help and direct neurologists and neurosurgeons to localize and identify the brain tumours correctly.*

Introduction

Ophthalmic findings frequently constitute an important fraction of the clinical expression of central nerves system disorders, particularly tumours. About one half of the patients who harbour a space occupying lesions have some ophthalmic symptoms or findings that are directly related to the presence of such a lesion[1]. If headache is persistently restricted to one region its localization value is increased in the absence of papilloedema[1]. The patient may first see an ophthalmologist about headache, failing vision and or double vision. It is the responsibility of the ophthalmologist to suspect brain tumours.

Raised intacranial pressure is responsible for most non-localizing symptoms and signs. Headache is among the initial symptoms in 20 % patients of brain tumours and 70% of patients during the course of the illness [1]

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The signs that arise when the function of a specific area of the brain is interrupted are defined as focal or localizing sign. Because focal sings often precede generalized signs, it is crucial to recognize them in order to reach an early diagnosis. Tumours are usually progressive in nature and seldom involve a single lobe but spread to the adjacent regions. If the area occupied by the tumour subserves a specific visual-sensory or visual-motor function, a specific ophthalmic sign will emerge that will allow an accurate localization of the tumour in many cases[1]

Visual loss may be an initial complaint in brain tumours. Slowly growing neoplasm produce a gradual painless visual loss that may progress for months to years. Decreased visual acuity with visual pathway compression usually prompts the patient to seek medical care. Axonal loss resulting from compression of pregeniculate visual pathways, eventually leads to disc pallor and optic atrophy. In papilloedema there is passive oedema of the optic disc that is caused by raised intracranial pressure. The transient loss of vision with preservation of central vision occurs in the early stage of papilloedema. However visual loss with central scotoma is significant in fully developed chronic atrophic papilloedema[1].

Expanding tumours displace the cerebral tissues either across the midline or in caudal direction to compress the diencephalic and brainstem structures. The nerves innervating the extraocular muscles are stretched, displaced and compressed, and their blood supply is interrupted, leading to ocular motor nerve palsy which has no localizing value. The best known example is unilateral or bilateral palsy of the abducens nerve. In the absence of increased intacranial pressure cranial nerve palsies have significant localizing value. Intrinsic brainstem tumours destroy the nuclei and intramedullary portions of cranial nerves. Extrinsic tumours stretch and displace the nerves in the posterior and middle cranial fossa.

Visual field loss may occur in brain tumour if it interrupts the visual pathway. Field defect may permit fairly accurate localization of tumours.

Pathological nystagmus may develop in intracranial lesions due to three established mechanisms;[2,3] a) Visual loss in one or both eyes in the first decade of life when the lesion lie anterior to the lateral geniculate body. Neural transmission of a clearly focused image during this period is necessary to keep the eye still. b) Vestibular disfunction which occurs due to lesion in the vestibular nerve or labyrinth or results from on imbalance in vestibular signals in the brain stem lesion. c) Neural integrators dysfunction which results from lesion in the medullary nucleus prepositus hypoglossi or medial vetibular nucleus responsible for horizontal eccentric gaze and lesions of mesencephalic interstitial nucleus of cajal responsible for vertical gaze. The cerebeller flocculus contributes to both horizontal and vertical signals.

Ophthalmologists can diagnose brain tumours after meticulous history taking and careful ocular neurological and systemic examination. They can also sometimes localize the tumour by observing the different ocular manifestation present in different area of brain.

Materials and Methods

It is a prospective study done from November 2003 to June 2008 (56 months) at Neuro-ophthalmology clinic at Chittagong Eye Infirmary and Training Complex.

Patients were selected on the basis of:

Loss of vision with or without other visual (e.g. Diplopia, Nystagmus, Optic atrophy & Papilloedema) and non- visual (e.g. Headache, Vomiting, Hearing deficit, Ataxia) features, and MRI or CT scan of brain suggestive of brain tumours.

Patients with previously diagnosed brain tumours, metastatic tumours, surrounding, tumours extended to the brain, aneurysms and other vascular anomalies and where neuroimaging could not be done were excluded from this study.

Clinical assessment was done by detailed history taking, recording age and sex, visual and non-

visual symptoms, and duration of symptoms. Examination included Snellen visual acuity test, ocular motility test and corneal and facial sensitivity test. Hearing was tested in each ear in turn by whispering to patient whose eyes were closed and whose other ear was occluded by finger pressure on the tragus. Fundus examination was done by direct ophthalmoscopy and indirect slit lamp biomicroscopy.

MRI or CT scan of brain (with contrast) was done in all the cases.

Results

A total number of 186 cases of primary brain tumours were diagnosed in the 56 months of study. Mean age was 32.45yeras, SD ± 1.50. Among the patients 91(49%) cases were male and 95(51%) cases were female. About 96 (52%) of patients are between 21years to 40 years of age. (Table-1).

About 37(20%) tumours are located in the anterior cranial fossa which includes total cerebral hemisphere and diencephalons (Fig-1), 113(61%) tumours are in the middle cranial fossa which includes the sella and parasellar region (Fig-2) and 36(19%) in the posterior cranial fossa including the brainstem of cerebellum (Fig-3).

Commonest visual complaint was dimness of vision in one or both eyes present in 163(88%) cases and non-visual complaint is headache present in 35(46%) of cases. (Table-4)

Good vision (BCVA 6/6 to 6/18 in both eyes) present in 17(46%) of anterior, 14(12%) of middle

Table: 1. Age and Gender distribution of brain tumors patients

Age group	Male	Female	Total
0-10	10	9	19
11-20	15	14	29
21-30	25	26	
31-40	17	28	96(52%)
41-50	12	12	24
50+	18	6	24
Total	91	95	186

Mean 32.45, Min 31.0 SD 1.50

Table: 2. Location of tumours

Location	N	%
Anterior cranial fossa	37	20
Middle cranial fossa	113	61
Posterior cranial fossa	36	19
Total	186	100

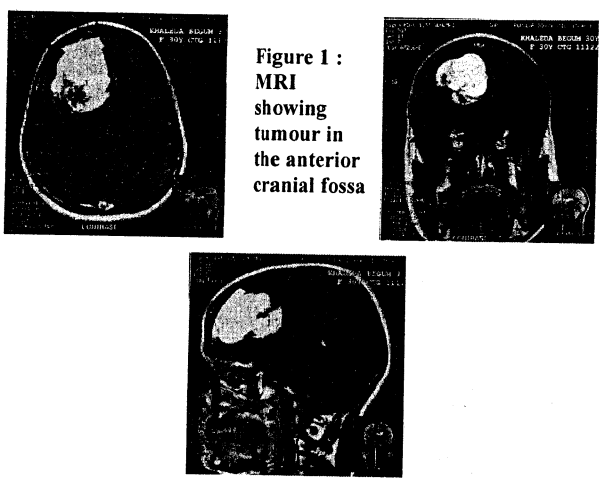


Figure 1 : MRI showing tumour in the anterior cranial fossa

In this study it is observed that posterior cranial fossa tumours are nearly double in female patients than male.

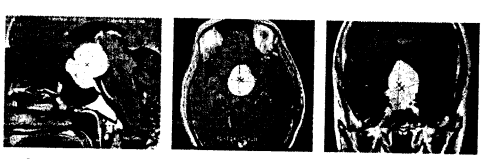


Figure 2 : MRI showing tumour in the middle cranial fossa (Pituitary macroadenoma)

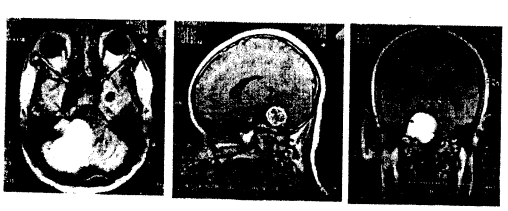


Figure 3: MRI showing tumour in the posterior cranial fossa (cerebellopontine angle tumour)

Table: 3. Gender distribution of tumours in different location of brain

Gender	Anterior cranial fossa		Middle cranial fossa		Posterior cranial fossa		Total
	No.	%	No.	%	No.	%	
Male	16	43	63	56	12	33	91
Female	21	57	50	44	24	67	95
Total	37	100	113	100	36	100	186

Table: 4. Major Complaints

Complaint	Location of tumor						Total	
	Anterior cranial fossa		Middle cranial fossa		Posterior cranial fossa			
ual	No.	%	No.	%	No.	%	No.	%
• Dimness of Vision	28	76	103		32	89	163	88
• Double Vision -Visual	3	8	7		7	19	17	9
• Headache	19	51	44	39	22	61	85	46
• Vomiting	8	22	6	5	9	25	23	12
• Shortness of Hearing	0	0	0	0	14	39	14	8

and 9(25%) cases of posterior fossa tumours. 12(32%) of anterior, 26(23%) of middle and 10(28%) of posterior fossa tumours patients were blind (BCVA <3/60 to NPL in both eyes) at presentation. (Table-5)

Normal optic disc on both eyes are seen in 2(5%) of anterior, 18(16%) of middle and 3(8%) cases of posterior cranial fossa tumours. Papilloedema is found in 28(76%) of anterior and 28(78%) of posterior fossa tumoiurs and only 12(10%) cases of middle cranial fossa tumours. On the other hand atrophic change of the optic nerve is present in 83 (73%) cases of middle cranial fossa tumours but only 7 (19%) of anterior and 5 (14%) of posterior fossa tumours. (Table-6).

Table: 5. Visual acuity (BCVA) at presentation

Comments			Location						Total	
			Anterior cranial fossa		Middle cranial fossa		Posterior cranial fossa			
	One Eye	Other Eye	No.	%	No.	%	No.	%	No.	%
Good vision	6/6 to 6/18	6/6 to 6/18	17	46	14	12	9	25	40	22
Blind	<3/60 to NPL	<3/60 to NPL	12	33	26	23	10	28	48	26
Monocular Blind	6/6 to 6/18	6/24 to 3/60	3	8	53	47	8	22	64	34
Low Vision Both eyes	6/24 to 3/60	6/24 to 3/60	2	5	13	12	6	17	21	11
Monocular Low Vision	6/24 to 3/60	<3/60 to NPL	3	8	7	6	3	8	13	7
		Total	**37**	**100**	**113**	**100**	**36**	**100**	**186**	**100**

Table: 6 Optic Disc Features

	Location						Total	
	Anterior cranial fossa		Middle cranial fossa		Posterior cranial fossa			
	No.	%	No.	%	No.	%	No.	%
Normal	2	5	18	16	3	8	23	12
Papilloedema	28	76	12	10	28	78	68	37
Pale (Partial atrophy)	1	3	38	34	0	0	39	21
Atrophy	6	16	45	40	5	14	56	30
Total	**37**	**100**	**113**	**100**	**36**	**100**	186	100

Discussion

This was an eye hospital based study where patients usually presented with visual problems and headache. The patients were not aware that

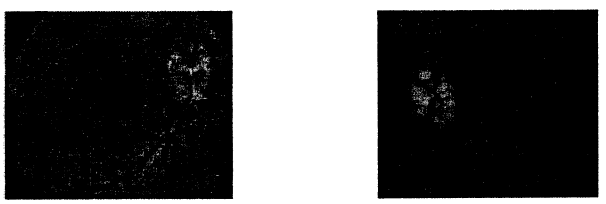


Figure 4 : shows papiloedema in posterior fossa tumour

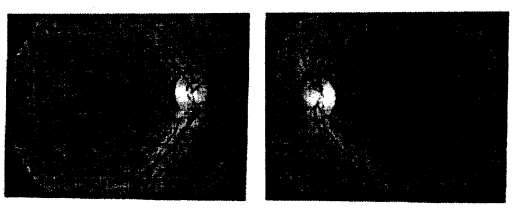


Figure 5 : shows 'Bow tie' shaped optic disc atrophy in pituitary tumour

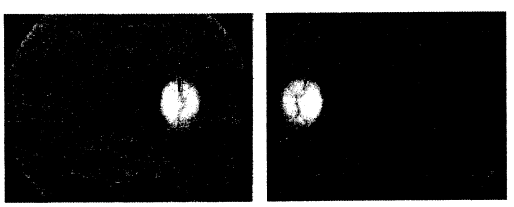
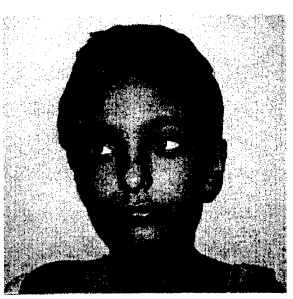


Figure 6: shows secondary optic atrophy in anterior cranial fossa tumour

Single or multiple cranial nerve palsy present in only 5(14%) of anterior and 13(12%) cases of middle cranial fossa tumours (Fig-7), but present in 22(61%) cases of posterior fossa tumours (Fig-8). Nystagmus is more common in posterior fossa tumours (14,39%). (Table-7)

Table: 7. Neurological association

	Anterior cranial fossa		Middle cranial fossa		Posterior cranial fossa	
	No.	%	No.	%	No.	%
Cranial nerve Palsy	5	13.5	13	11	22	61
Nystagmus	2	5	3	8	14	39



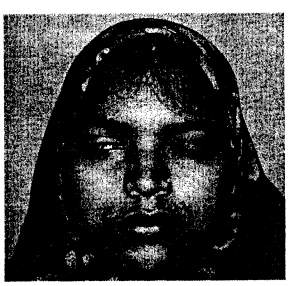


Figure 7 : Right 6th Nerve palsy in middle cranial fossa tumour
Figure 8 : Right 7th nerve palsy in posterior cranial fossa tumour

Table: 8 Neuro Imaging of patients

Neuro Imaging	No.
MRI	**64**
CT	**120**
MRI with CT	**2**

they had intracranial tumours. The diagnosis of brain tumours were made by the ophthalmologists by careful observation and examination of ocular and others clinical manifestations and with the help of neuroimaging of brain.

Tumours are usually progressive in nature and seldom involve a single lobe but spread to the opposite hemisphere. In this study for simplicity, the locations of intracranial tumours were arbitrarily divided into three areas: 1) anterior cranial fossa tumours, which include cerebral hemisphese and diencephalon, 2) middle caranial fossa tumours, which include the sella and parasellar region, and 3) posterior cranial fossa tumours which include cerebellum and the brainstem. Temporal lobe is a part of cerebral hemisphere and included in the anterior cranial fossa, though it is extended to middle cranial fossa.

Ocular manifestations of dimness of vision, double vision, nystagmus papilloedema, optic atrophy or field defect, and non-ocular manifestations like headache, vomiting, deafness, and ataxia make the suspicious of brain tumours. Tumours of the brain may produce symptoms that arise from their location, size, malignancy or non-specific mechanical effects.

Ocular manifestations produced by brain tumours, depend upon location of tumours. In this study commonest tumours presented to ophthalmologists are middle cranial fossa tumours, which produce more effect on visual acuity. Papilloedema is found in most of the cases of anterior and posterior cranial fossa tumours. Optic atrophy is very common in middle fossa tumours. Cranial nerve palsy and nystagmus are more common in posterior fossa tumours

This 56 months study showed that, 20% cases of tumours are in the anterior cranial fossa, 61% in the middle cranial fossa and 19% in the posterior cranial fossa. But study done by Faraque and associates in neuro-surgery department of Bangabandhu Sheikh Mujib Medical University (BSMMU) hospital, Dhaka., It showed that 22% in anterior, 39% in middle and also 39% in posterior cranial fossa are involved by intracranial space occupying lesions (ICSOL)[4] The difference is probably due to the fact that, BSSMU study included the subarchnoid hemorrhage, giant aneurysm, AVM, brain abscess tuberculoma and hydatid cysts. These are excluded from our study. Besides the study was done in neurosurgery department where the presenting complaints are different from patient coming to the eye hospital for ocular complaints.

Our study showed the number of male and female patients are almost same (91 and 95 respectively) but in the posterior fossa tumours the male and female ratio is 1:2 which needs further study to prove the fact.

The study showed that 88% patients had complaints of dimness of vision and 9% had double vision, which is in consistence with BSSMU study, which showed that 92% had visual impairment and 16% had double vision.

This study showed that 26% patients were blind at presentation as their BCVA was less than 3/60 in the better eye and 34% cases were monocular blind. This suggests that patients usually present late and diagnosis is also made at late stage.

Our study showed that 37% patients had developed papilloedema and 51% cases had atrophic change in the optic disc, which is consistent with the BSSMU study showing 37% had papilloedema and 37% had atrophic changes in the optic disc.

Our study further showed in middle cranial fossa tumours atrophic changes found in 73% of cases and papilloedema was found in only 10% cases. On the other hand in the other area of brain tumours more than 75% of cases had papilloedema and less than 20% cases had atrophic disc changes. This is because the commonest tumours in the middle cranial fossa are pituitary tumour [5] and papilloedema is extremely rare in pituitary tumours [6]

This study also showed that 61% of posterior fossa tumours are associated with cranial nerve palsy. This is because most of the cranial nerve nuclei are

situated in the brainstem. Besides nystagmus was found in 39% cases of posterior fossa tumour as nystagmus is the most constant sign of cerebellopontine angle and also in cerebellar lesion which are situated in the posterior cranial fossa.

Limitation of this study is that only ocular manifestations are described in this study. Humphrey visual field could not be done in most of the cases because of extremes of age, very poor vision, poor health and non-compliance of the patients, is not shown in this article. Though Magnetic resonance imaging (MRI) with gadolinium is the most sensitive and specific method used to detect brain tumours, because of poor economic condition of the patients, CT scan of brain was done in most of the cases instead of MRI in our study.

Conclusion

Brain tumours are diagnosed at an advanced stage of visual loss, when patients are blind or nearly blind. Patients with gradual loss of vision, headache, optic disc changes, papilloedema, cranial nerve palsy, nystagmus and visual field defect should have an MRI or CT scan brain with contrast done to diagnose the case early.

Intracranial tumours, which produce adverse effect on vision, morbidity and mortality, should be diagnosed at early stage. Ophthalmologists may be able to help and direct neurologists and neurosurgeons to localize and identify the brain tumours correctly.

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Theraputic Response of 100 cases of Migraine to Flunarizine

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Abstract: *Current anti-migraine agent fail to achieve optimum therapeutic response in many migraine patients. We evaluated the efficacy of flunarizine in 100 patients of migraine. One hundred and twenty patients were enrolled but twenty patients were reluctant regarding followup. These twenty patients were excluded from the study. Out of 100 patients 57 (57%) were female and 43 (43%) male showing an overall ratio between female:male 1.33:1. Age of the patients ranged from 15 to 50 years. Out of 100 cases 54 (54%) had common migraine 8 (8%) had classical migraine with visual hallucination and 28 (28%) suffered from both types of attack. Tablets flunarizine was given once a day on consecutive five days in a week for 6 months as prophylactic anti-migraine agent. Sixty eight (68%) patients were free from the disease recurrence at six months follow up while 11 (11%) patients experienced decrease in both frequency and severity of the migraine attack, five (5%) patients claimed to have reduced severity and another seven (7%) patients had reduced frequency of migraine. Nine (9%) patients claimed to have no effect to flunarizine.*

Introduction

Migraine is often a familial disorder, more common in females, characterized by recurrent attacks of headache widely variable in intensity, duration and frequency. The headache is commonly unilateral, associated with nausea and vomiting and may be preceded by or associated with neurological and mood disturbances. However, all these characteristics are not necessarily present during each attack or in every patient.

Materials and Methods

One Hundred patients with migraine aged between 15 to 50 years were selected from outpatient department of Ophthalmology at Uttara adhunik Medical college Hospital from June 2009 to June 2010. Detailed clinical history was taken in all cases. Blood pressure recording and Ophthalmoscopy were done routinely in all the cases. Patients with hypertension, Sinusitis, Refractive error and tension headache were excluded from the study. Relevant investigations were done in all cases to exclude any secondary cause of headache. Individuals with Eye and ENT problems detected for the first time were not included in the series. CT scan of brain was done in 6 cases and MRI in 3 cases. All the cases were treated with Flunarizine (10 mg daily) consecutively for five days in a week for 6 months along with avoidance of triggering factors to prevent further attack of migraine headache. For early abortion of an episode and during an episode of headache, oral Zolmitriptan (2.5 mg daily), oral Indomethacin or Diclofenac was given along with anti-emetic Metoclopramide (10 mg thrice daily).

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Results

In this series, out of 100 cases 57 were female and 43 male. Ratio between female: male is 1.33:1. Highest occurrence (65; 65%) of Migraine was found in 3rd decade. Out of 100 cases 54(54%) had common migraine, 8(8%) had classical migraine with visual hallucination and 28(28%) suffered from both types of attack. During acute attack, 30(30%) patients of this study responded to treatment Zolmitriptan with or without Metoclopramide and the remaining 70 patients were treated with non-steroid anti-inflammatory drugs like Indomethacin and Diclofenac with or without Metoclopramide.

All of the 100 patient were given prophylactic treatment with Flunarizine.

Clinical response was assured at follow up after six months. Table-1 shows that 68(68%) patient remained free from recurrence, 11(11%) patients noticed decreased in both frequency and severity of the migraine attack, 5(5%) patients claimed reduced severity only and another 7(7%) had reduced frequency of migraine. Nine (9%) patients did not show any response.

Table-1.Response to prophylaxis with Flunarizine. (N-100)

Response	No	%
No recurrence	68	68%
Decreased in both frequency and severity of attack	11	11%
Decreased severity of episode only	5	5%
Decreased frequency of attack	7	7%
No response to prophylaxis	9	9%
Total	100	100%

Discussion

Due to various complexity of modern life incidence of Migraine is increasing. In our study highest occurrence (65; 65%) of Migraine was found in 3rd decade. Incidence was higher in female than male giving the ratio of (1.33:1). Preventive treatment of migraine is designed to decrease headache frequency and severity. It should be used if headaches are too frequent. Disabling or associated with worrisome neurological features or if acute treatment is over used or ineffective. A drug should be chosen on the basis of its efficacy, cost effectiveness, adverse event profile and the presence of coexistent conditions. In our study we used Flunarizine in the prophylaxis of migraine and the result was promising. Out of 100 patients 68(68%) patients remained free of recurrence at six months follow up. Considering the impact of migraine on time loss, monetary loss and the impairment of familial, social and leisure activity migraine should not be left untreated as a non-curable disease. In this study Flunarizine showed an excellent result in migraine prophylaxis which is very cost effective and showed excellent tolerability profile.

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Spectrum of fungus in culture positive fungal keratitis at a tertiary eye hospital of Bangladesh

Dr. Md. Shafi Khan

Abstract:

PURPOSE: *To report the species of fungus in culture positive fungal keratitis and their age and sex correlation at Islamia Eye Hospital and MAI Institute of Ophthalmology, Dhaka, Bangladesh.*
METHODS: *We reviewed the records of 310 cases of culture-positive fungal keratitis treated from 01.01.2010 to 30.06.2010 at Islamia Eye Hospital and MAI Institute of Ophthalmology, Dhaka, Bangladesh. The data of age, sex and species of fungus were collected and analyzed.*
RESULTS: *The study included 310 eyes of 310 patients. 203 (65.48%) patients were male and 107 patients (34.51%) were female. The mean age was 42.11±16 years (range 7-85 years). Early identification of fungal elements was achieved by 10% KOH wet mount of corneal scrapings in all the cases and only culture positive cases are taken for the study. 132 cases had corneal infections caused by Fusarium sp. (42.58%), 117 cases by Aspergilous sp.(37.74%), 22 cases by Acromonium sp.(7.09%), 8 cases by alternaria sp.(2.58%), 3 cases by candida sp.(0.96%), and 28 cases by unidentified hyaline fungal sp. (9.03%).*
CONCLUSION: *In contrast to other studies from our subcontinent and other countries, we found Fusarium sp. to be the most common fungal isolate, followed by Aspergillous sp. in culture-proven cases of fungal keratitis. The predominance of fungal keratitis in males was most pronounced in their middle years of age.*
KEY WORDS: *Species of fungus, fungal keratitis, Fusarium sp.*

Corneal blindness is a major public health problem worldwide and infectious keratitis is one of the predominant causes[1].Corneal infection of fungal aetiology is very common[2-5] and represents 30% to 40% of all cases of culture-positive infectious keratitis in South India[6-7] Fungal keratitis is also a major ophthalmic problem in Bangladesh.

Filamentous fungi are responsible for a larger proportion of these corneal infections in tropical climates than in temperate climates, particularly following trauma with vegetative matter. In tropical climatic conditions as in South Florida,[2] Bangladesh,[4] South India[6] and Nepal[8] the incidence of fungal keratitis is reported to be from 17% to 40%. In temperate climates such as Britain[9] and Northern United States[10] the proportion of fungi causing suppurative keratitis is very low. Similarly, at the high altitude of Johannesburg, South Africa, the incidence is not more than 2.3%.[11,12]

At least 70 genera of fungi have been associated with fungal keratitis·[13] Of these, Fusarium species and Aspergillus species are responsible for 70 % of cases.[14-16] The aetiological and epidemiological pattern of corneal ulceration varies significantly with patient population, health of the cornea, geographic region, climate and also tends to vary

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over time. Hence, an understanding of the current status of the regional, epidemiological features, risk factors, the presence of ocular and/ or systemic co-morbidities, occupational status and knowledge of region-wise aetiological agents is important in the prevention and appropriate management of fungal keratitis. But there is a paucity of data on the spectrum of fungal keratitis in Bangladesh.

So the purpose of this study was to identify the species of fungus and study the age and sex variations of fungal keratitis presenting at a tertiary referral centre in Bangladesh and to make a platform for further study with large patient population and more variables like risk factors, epidemiological features, response to treatment etc.

Materials and Methods

Patients

A restrospective analysis was performed of all patients with culture-proven fungal keratitis seen over a period of 6 months, January to June, 2010, at a tertiary eye care referral centre in Bangladesh. A total of 310 consecutive patients with culture positive fungal keratitis were analyzed.

Clinical and laboratory Procedures

All patients received a slitlamp biomicroscopic examination by an ophthalmologist.

After a detailed ocular examination using standard techniques, an ophthalmologist or a trained laboratory technologist took corneal scrapings under aseptic conditions from each ulcer using a sterile Bard- Parker blade (No. 15).

The scraping material obtained from the leading edge and the base of each ulcer was spread onto labeled slides in a thin, even manner for 10%, KOH wet mount. The scraping material was initially inoculated directly onto Sabouraud's dextrose agar (SDA). Meticulous care was taken in collection of material and its aseptic transfer to the appropriate culture media. All inoculated media were incubated aerobically.The inoculated Sabourauds destrose agar were incubated at 27°C, examined daily and discarded after 1 week if no growth was seen.

All laboratory methods followed standard protocol. Microbial cultures were considered significant if there was confluent growth at the site of inoculation on one solid medium, and/or if growth of one medium was consistent with direct microscopy findings.

Results

During the study period of 6 months, from January to June,2010 , 310 patients of culture positive fungal keratitis were identified and included in the study. Among them 203 (65.48%) were male and 107 (34.51%) were female with an overall ratio of male to female patients of 1.89 to 1.

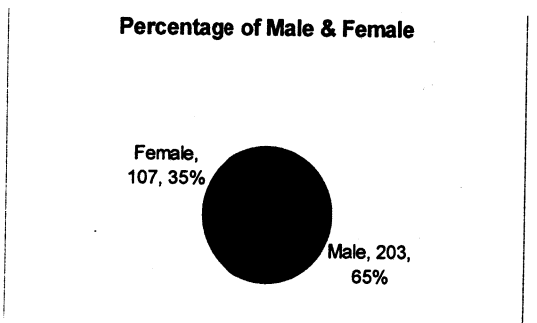

Fig.1 : Ratio of male to female patients

The predominance of fungal keratitis was most pronounced in the middle years of life. Most of the patients were between the ages of 21 to 60 years (253 patients,81.61%). The mean age of the patients were 42.11±16 years ranging from 7 to 85 years.

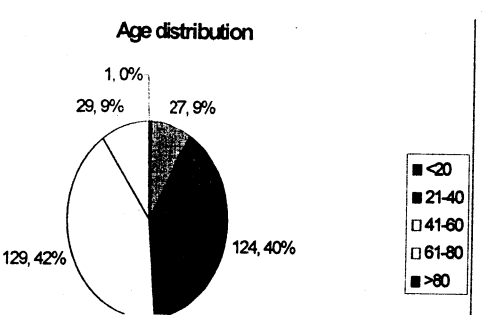

Fig.2 : Age distribution

Table 1 : Distribution of different species of fungus in different age group

Age	Total (n %)	Filamentous (Fus +Asp +Acremonium) (n %)	Dematatious (Alternaria) (n %)	Non filamentous (Candida) (n %)	Unidentified (n %)
<20	27(8.70)	25(8.06)	0(0)	0(0)	2(0.64)
21-40	124(40)	115(37.09)	2(0.64)	1(0.32)	6(1.93)
41-60	129(41.60)	106(34.19)	5(1.61)	1(0.32)	17(5.48)
61-80	29(9.35)	24(7.74)	1(0.32)	1(0.32)	3(0.96)
>80	1(0.32)	1(0.32)	0(0)	0(0)	0(0)

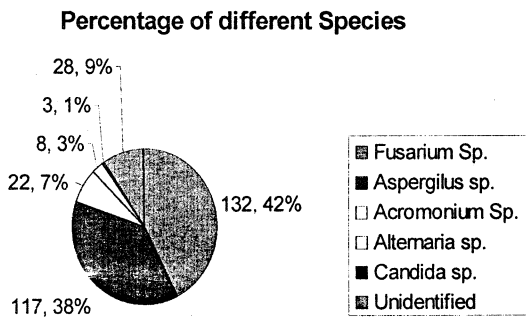

Fig.3 : Percentage of different species

Among the 310 patients, most of the isolated fungus were filamentous (271, 87.41%) that is Fusarium sp.132 (42.58%), Aspergillus sp. 117 (37.74%) and Acremonium sp. 22 (7.09%). Dematatious (Alternarie sp.) or pigmented fungus were 8 (2.58%). Nonfilamantous fungus or yeast (Candida sp.) were very few in number (3, 0.96%) and Unidentified sp. were 28 (9.03%).

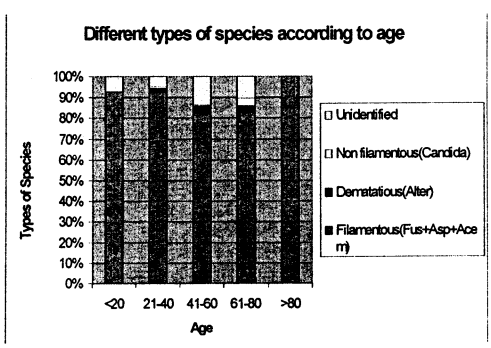

Fig.4 : Different types of species according to age.

Discussion:

Fungi are ubiquitous eukaryotic microorganisms. Fungi that infect the cornea are broadly classified as yeast or molds. Yeasts are unicellular fungi characterised by an oval or round structure, the blastoconidium. Molds are Organisms with filamentous structure (hyphae) and a tangled mass of hyphae which constitutes the mycelium. Filamentous fungi may be classified as septate or non-septate. Fungi reproduce sexually by the formation of spores and asexually by forming conidia or sporangiospores. The disease-causing fungi in the cornea usually are in an asexual phase of their life cycle when they are cultured from infected cornea.[16, 17]

In this study we identified different species of fungus in culture positive fungal keratitis and analyzed their age and sex correlation.

Fusarium sp. was isolated as the predominant species in this study, similar to the reports from South Florida[2], Ghana[3] and South India [24]. This is in contrast to most reports of Aspergillus sp. from other parts of India.[13, 18, 19] and Candida sp. in other parts of the World [20-22].The diatomaceous fungi are frequently reported as causes of keratitis in many tropical and subtropical regions[23] though in my study I found the pigmented fungus were very few in numbers. In this study among the 310 patients, most of the isolated fungus were filamentous (271, 87.41%) that is Fusarium sp.132 (42.58%), followed by Aspergillus sp. 117 (37.74%) and Acremonium sp. 22 (7.09%). Dematatious (Alternarie sp.) or pigmented fungus were 8 (2.58%). Nonfilamantous fungus or yeast (Candida sp.) was very few in number, only 3

(0.96%) which was in contrast to reports from Australia and USA (about 40%)[2,25]. The unidentified sp. was 28 (9.03%).

The incidence of fungal keratitis was significantly higher in males as 203 (65.48%) were male and 107 (34.51%) were female with an overall ratio of male to female patients of 1.89 to 1 which is similar to a report from South India [24] and many other studies.[3,6,7]

We observed that people aged 21 to 60 years that is the age of physically active persons were more often affected by fungal keratitis (253, 82%), which was similar to the report from Tamil Nadu, South India.[24]

Conclusion

This study presents predominant species of fungus in culture positive fungal keratitis patients at a tertiary eye care centre of Bangladesh and correlate it with age and sex distribution. Though the study population and the variables are small in number but we hope it will help the upcoming researchers to study on a large population and with more epidemiologic factors. Fungal keratitis continues to be a cause of concern to the ophthalmologists in Bangladesh. So more elaborate study should be done in future.

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Randomised Controlled Trial Of Topical Cyclosporine In Steroid Dependent Allergic Conjunctivitis

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Dr.Md.Towhid Anwar, Dr.Zahidul Ahsan

Abstract

Aim: *To evaluate the efficacy, safety, and therapeutic effect of topical cyclosporine 0.05% as a steroid sparing agent in steroid dependent allergic conjunctivitis.*

Methods: *Prospective, randomised, placebo controlled trial comparing signs, symptoms, and the ability to reduce or stop concurrent steroid in steroid dependent atopic keratoconjunctivitis and vernal keratoconjunctivitis using 0.05% topical cyclosporine compared to placebo. Steroid drop usage per week (drug score), symptoms, and clinical signs scores were the main outcome measures.*

Results: *The study included an enrolment of 40 patients, 18 with atopic keratoconjunctivitis and 22 with vernal keratoconjunctivitis. There was no statistical significant difference in drug score, symptoms, or clinical signs scores between the placebo and cyclosporine group at the end of the treatment period. No adverse reactions to any of the study formulations were encountered.*

Conclusions: *Topical cyclosporine 0.05% was not shown to be of any benefit over placebo as a steroid sparing agent in steroid dependent allergic eye disease.*

Introduction: Allergic eye disease is a common debilitating ocular surface disease that is highly variable in severity and duration. Milder disease is more common and does not affect the cornea, but the most serious forms of ocular allergic disease—atopic keratoconjunctivitis (AKC) and vernal keratoconjunctivitis (VKC)—may involve the cornea and can be sight threatening.[1–3] Treatment is directed towards the allergic response with antihistamines and mast cell stabilisers are the mainstay of therapy. Severe or resistant diseases are well recognised and steroids are frequently required. Steroids can be highly effective, but may cause unwanted elevation of intraocular pressure in steroid responders and increase the risk of corneal infection through local immunosuppression. In addition, induction of cataract and delayed wound healing can be problematic.

Cyclosporine is a non-steroidal immuno modulator that inhibits antigen dependent T cell activation. Cyclosporine also has a direct inhibitory effect on eosinophil and mast cell activation and release of mediators, which is likely to be important in allergic inflammation.[4–7]

Topical cyclosporine has been used in several formulations in an effort to reduce steroid dependence. A placebo controlled trial using topical cyclosporine 2% in maize oil showed it to

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be an effective and safe steroid sparing agent, but its use was limited by frequent intense stinging in the patients.[8] In a large series of patients with AKC, significant keratopathy developed in two thirds of patients managed with the usual regimen of oral antihistamines, topical mast cell stabilisers, and topical steroids.[9] A randomised controlled trial (RCT) using a novel emulsion of ciclosporin 0.05% in topical steroid resistant AKC, showed a much better tolerated treatment that had some effect in alleviating signs and symptoms of AKC.[3] We undertook a prospective randomised double masked study to assess, the therapeutic effect of topical ciclosporin 0.05% as a steroid sparing agent in steroid dependent allergic conjunctivitis.

MATERIALS AND METHODS

Study design

This was a prospective, RCT, comparing signs, symptoms, and the ability to reduce or stop concurrent steroid in steroid dependent AKC or VKC using 0.05% topical ciclosporin compared to placebo.

Patients

Patients of either sex with steroid dependent AKC or VKC, who were willing to comply with the protocol and who provided informed consent, were recruited from the National Institute Of Ophthalmology, Bangladesh and 250 beded General Hospital, Jessore. Written informed consent was obtained from all patients before the initiation of any study medication or study related procedure.

Patients were excluded from the study if they where using systemic steroid or immunosuppressive drugs or non-steroid anti-inflammatory medication or had associated ocular infections or coexisting ocular diseases such as corneal diseases, glaucoma, and optic atrophy. If patients were using topical or systemic ciclosporin, this medication was discontinued 2 weeks before the start of the trial.

Other exclusion criteria included history of periocular injections of steroids within a period of 6 months, ocular surgery within the previous 6 months, and using steroid eye drops for reasons other than allergic diseases (that is, uveitis)

Pregnant or lactating mothers were also excluded. Patients could be discontinued before the completion of the study owing to adverse events, protocol violations, lack of efficacy, or personal reasons.

Study protocol

Patients were assigned randomly based on a predetermined randomisation list generated by computer to receive either 0.05% topical ciclosporin (Cyporin, Aristopharma Ltd, Bangladesh) or placebo (artificial tear). Allocation coding was undisclosed until all patients had completed the study. Both patients and physicians were masked to the identity of the drops used for the duration of the trial. Identical unit dose vials were used to hold the study treatments.

During the treatment phase, all patients were instructed to instil one drop of the study medication (0.05% ciclosporin or placebo eye drops) four times daily to both eyes, in addition to their usual treatment. The clinical response was used to reduce and stop topical steroids when possible. During the treatment phase, patients returned for evaluation after 1 week, 1, 2, and 3 months of treatment.

Outcome measures

Parameters used to assess treatment outcomes included symptoms, signs, and reduction or cessation of steroids. Symptoms of itch, redness, tearing, soreness (burning, discomfort, foreign body sensation) discharge, and photophobia were graded and recorded at each visit by the clinician on questioning. Clinical signs were graded by the physicians for the lids (ptosis, lid skin dermatitis, lid margin thickening, lid margin thickening, lid margin distortion, and lid margin hyperaemia) for

the conjunctiva (hyperaemia and oedema for the bulbar conjunctiva, hyperaemia, infiltration and papillae for inferior conjunctiva, and subepithelial conjunctival scarring, cicatrisation, hyperaemia, infiltration, and papillae for the superior tarsal conjunctiva) and for the cornea (tear film deficiency, epithelial disease, opacity, stromal thinning, neovascularisation, lipid deposition).

Topical steroid drops were modulated according to the clinical response. Steroid drop usage per week was calculated as the "drug score." Allowances were made for the relative potency of different topical steroid preparations by using a multiplication factor (that is, number of drops per week multiplied by five for prednisolone acetate 1%, by four for dexamethasone 0.1%, by three for fluoromethalone 0.1%, by two for prednisolone phosphate 0.3%, and by one for prednisolone phosphate 0.1%).

Statistical methods

All statistical analyses were conducted with Microsoft excel. Scores were derived separately for symptoms, signs, and steroid drug usage. We used the t tests to compare between the placebo and treatment groups the scores at a given time point or the reduction of scores from the initial to the final time point. We need to use *t* tests, as the number of observations in each group was fewer than 25. We also considered the binary outcomes that are indicators of the presence of a symptom or score, and used the logistic regressions to compare between the treatment or placebo groups the proportions of the presence of a symptom or sign at a time point. To examine the change over time for an outcome we used the generalised linear models with an exchangeable working correlation that allows dependency between observations from the same patient.[10] The generalised linear models were used with a normal distribution and log link for the drug, symptom, and sign scores, and the generalised models with a binomial distribution and log link for binary outcomes. The log link for the scores showed that the scores decreased exponentially with time. A test with a p value less than 0.05 was considered to be significant.

RESULT

Of the 40 patients with steroid dependent allergic eye disease 20 patients were assigned to receive 0.05% topical ciclosporin and 20 to receive placebo. Five patients were discontinued from the study. Of the five that discontinued, three were in the Ciclosporin treatment group (one was lost to follow up after enrolment and two withdrew for personal reasons) and two of these were in the placebo group (one was found not to meet the study criteria after randomisation and one withdrew for personal reasons). Thus, for description and analysis 35 patients were used (17 in the treatment group and 18 in the placebo group) (fig 1). At the time of enrolment 15 patients were diagnosed with steroid dependent AKC (eight patients in the treatment group) and 20 patients with steroid dependent VKC (nine in the treatment group). No adverse reactions to any of the study formulations were encountered.

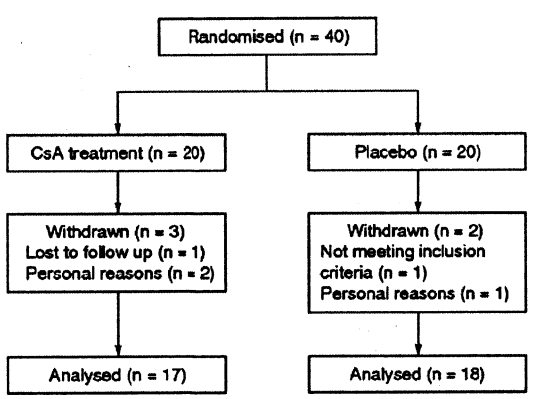

Figure 1 Randomisation of participants. (CsA, ciclosporin A.)

Of 17 patients in the Ciclosporin treatment group, 15 (88%) were males and their age mean at presentation was 26.2 years (SD 18). Of 18 patients in the placebo group, 13 (72%) were male and the age mean at presentation was 26.2 years (SD 16.3). There was no significant difference between the two groups in terms of sex or age.

The initial weekly steroid drop usage score was 99.3 (SD 45.1) in the placebo group, and 66.5 (SD 45.9) for the Ciclosporin treatment group (fig 2). There was a marginally significant difference between the two groups in the initial weekly steroid drop usage score (p?=?0.05). The final steroid drop usage score was 39.9 (SD 45.8) for the placebo group, and 42 (SD 44.7) for the treatment group. However, there was no significant difference in the final steroid drop usage score between the two groups (p=0.9). Also the reduction in steroid drop usage score in the placebo group was not significantly different from that in the treatment group (p=0.6).

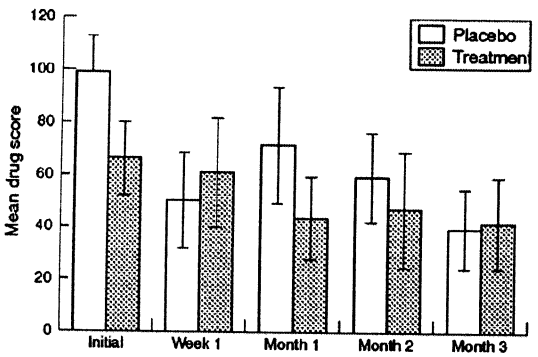

Figure 2

Mean (SEM) steroid drop usage per week (drug score) before and during the trial.

The initial symptom score was 5.9 (SD 4.3) for the placebo group, and 6.1 (SD 4.0) for the treatment group while the final score was 2.1 (SD 2.9) for the placebo group and 2.8 (SD 3.1) for the treatment group (fig 3). There was no significant difference in either initial symptom score (p?=?0.9) or final symptom score (p=0.5). However, there were significant reductions over time in itching (p=0.04), and redness (p=0.01) for the CsA treatment group. The placebo group also experienced significant reduction over time in redness (p=0.01) and white discharge (p=0.01). All the other symptoms did not attain statistical significance of change over time in either placebo or treatment CsA group.

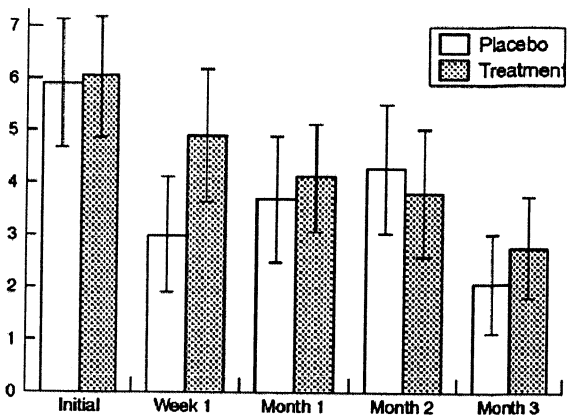

Figure 3

Mean (SEM) symptom score before and during the trial.

Finally, there was no significant difference between the placebo and treatment groups in the initial clinical sign score (p=0.7) or the final clinical score (p=0.6) (fig 4). For specific signs, patients in the treated group showed significantly greater improvement over time in the lid margin thickening (p=0.02), inferior and superior conjunctiva hyperaemia (p=0.01 and p=0.01 respectively), inferior conjunctiva papillae (p=0.03), and corneal tear film deficiency (p?=?0.05). The placebo group showed significant reduction over time in bulbar conjunctiva hyperaemia (p=0.02). The other signs did not attain statistical significance of change over time in either of the groups.

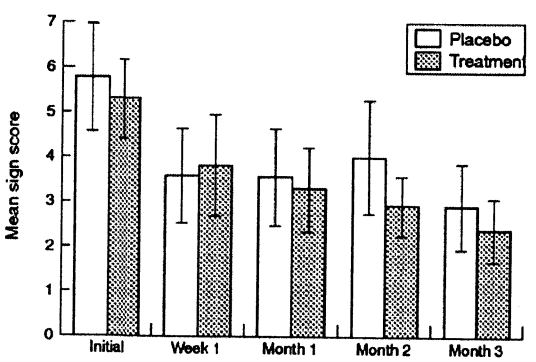

Figure 4

Mean (SEM) sign score before and during the trial.

DISCUSSION

The results of our trial failed to show a beneficial effect from the addition of topical ciclosporin 0.05% in steroid dependent allergic eye disease. In various parameters including symptom score, sign score, and drug score, there was no statistically significant difference between treatment and placebo groups over the period studied.

Our study population included patients with steroid dependent AKC and VKC and examined the effect of topical ciclosporin A (0.05%) on symptoms and signs or a steroid sparing effect. It was masked, randomised, and prospective and used vehicle as the placebo control. Although there was a difference in composite drug scores between the groups at baseline, other baseline parameters were equivalent. No significant difference in reduction of steroid was noted over the study period. The trial instruction was to use the clinical response to guide the reduction in topical steroid drops and to reduce as possible. The individual decision to reduce steroids was made by the treating clinician.

The symptom score used is a well validated tool and similar to those used in previous studies.[3,8] Similarly, the signs score has been used previously.[3,8] In addition to the primary analysis using the aggregated scores we also examined each feature individually and found no statistically significant pattern.

A negative result of this study is in contrast with a recent multicentre trial using the same preparation as in our study. Topical ciclosporin 0.05% in a novel emulsion (Restasis) in 22 patients with AKC refractory to steroid treatment, showed some beneficial effect on pooled symptoms and signs scores over the treatment period without adverse effects.[3] However, that trial compared ciclosporin with a placebo (artificial tears) non-vehicle and was limited by a small sample size (22 patients) and a relatively short duration. Steroid use was maintained at pre-enrolment levels.

In a smaller prospective RCT in similar patient groups, the study by Hingorani *et al* used topical ciclosporin A 2% in maize oil, which had been manufactured in the pharmacy at Moorfields Hospital. Twenty one patients with steroid dependent AKC were studied and ciclosporin was shown to have a greater steroid sparing effect than that of vehicle albeit at the cost of poor patient tolerance of the adjunctive therapy.[8]

With 40 patients, our study had an 80% study power to detect a 0.45 difference in the proportion of patients stopping steroid in either group. To detect a smaller difference of 0.20, we would have needed 82 patients in each group.

The lack of response in this study may indicate that there is no benefit from the topical ciclosporin drops. In acute allergy, there is an immunoglobulin E (IgE) mediated type 1 hypersensitivity reaction, but in chronic disease, this changes to a mixed cell response.[11,12] In VKC and AKC, there is a TH-2 cell mediated chronic inflammation with an increase in CD4+ cells.[7] Topical ciclosporin is an anti-CD4+ cell agent, but acts more specifically on the TH-1 cells and so theoretically may be of less benefit in allergic TH-2 cell mediated hypersensitivity.[12,13]

Alternatively, the dosage of ciclosporin may have been insufficient. The use of steroid may mask any benefit from ciclosporin drops, although this was not seen in the earlier trials.[3,8] Similarly, either the concentration or frequency of treatment may have been too low, although the protocol was similar to previously reported studies.[3,8]

This study highlights the strengths of a RCT. Our perception and the perception of our patients, together with reports in the literature gave support to the use of adjunctive therapy in steroid dependent allergic eye disease. As no definite benefit has been shown, we have no option but to temper our enthusiasm. One possibility is to repeat a larger and more powerful study. The other option

is not to recommend Restasis eye drops (ciclosporin 0.05%) in steroid dependent allergic conjunctivitis.

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The spectrum of Vogt-Koyanagi-Harada disease in Bangladesh

Dr Jasmin Ahmad

AIM: *The aim of this report was to provide a detailed description of the clinical profile, management, and outcome of Vogt-Koyanagi-Harada (**VKH**) disease in patients of Bangladesh (a homogenious population of patients).* ***METHODS:*** *Retrospective review of all the patients of VKH disease presented to our institute between January 2001 and December, 2005 was carried out. A standard data acquisition form was used for the analysis of demographic and clinical features. Their history, ocular findings at initial and final visit, investigations, treatment with response and ocular complications were noted from the record.* ***Result :*** *The study cohort consisted of 31 VKH patients. Among them 19 (61%) were women and 12 (39%) were men, with a mean age of 37.58 years, SD = ± 13.49 years (range 18 to 66 years). All cases were bilateral. Visual outcomes were good, with final visual acuity of better than 6/9 in 33 (55%) of 60 eyes and worse than 6/60 in only 10 (16.67%) of 60 eyes. Visual acuity was significantly improooved (P= 0.0001) after getting treatment. All patients were treated with systemic corticosteroid on an averaged for 6 months, but was prolonged for years in patients who developed chronic uveitis. 4 patients needed systemic corticosteroids in the form of intravenous injection for their exaggerated presentation of disease. We did not get any complication in 14 (25.81%) eyes,46 (74.19%) eyes showed at least one complication. 28 eyes developed cataract, 14 eyes showed glaucoma, 2 eye showed sub retinal fibrosis, and 01 eye became soft.*

Key Words: *Vogt-Koyanagi-Harada disease, corticosteroid therapy, complications.*

Introduction

Vogt-Koyanagi-Harada disease (VKH) is a multi-system autoimmune disorder directed against self-antigen most likely associated with melanocytes, present in such tissues as the eye(mainly uvea), the skin, the meninges and the inner ear [1,2].

It was10th century Persia, the first description of a whitening of eyelashes, eyebrows, and hair associated with ocular inflammation. In 1932, Babel appreciated that the features of cases reported by Vogt (1906), Harada (1926) and Koyanagi (1929) were overlapped.

Vogt in1906, first described a case with bilateral idiopathic uveitis, poliosis and alopecia [3]; Harada in1926, reported five cases of bilateral posterior uveitis and retinal detachment [4]; Koyanagi in 1929, defined the main characteristics of the syndrome in 16 cases with headache, fever, dysacusis, vitiligo, poliosis, alopecia and bilateral pan-uveitis with occasional exudative detachment [5]. VKH disease more commonly affect pigmented individuals (races) of both sexes [1, 2, 6]. Females are more commonly affected[2, 6, 15] .The age of onset of the disease is reported mainly between 10 and 52 years , with a peak frequency in the third and fourth decades [1]. But it can happen in children also[7, 8, and 9].

Depending on revised diagnostic criteria, the disease is classified as complete, incomplete or probable based on the presence of extra-ocular findings (neurological, auditory and integumentary)[2]. The clinical course of VKH is divided into four phases: prodromal (mimics a

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viral infection), uveitic (bilateral diffuse uveitis with papillitis and exudative retinal detachment), convalescent (tissue depigmentation), and chronic recurrent (recurrent uveitis and ocular complications)[1]

The exact cause of VKH disease remains unknown[2], but evidence suggests that the pathogenesis is related to T cell (T helper type1)-mediated autoimmune response[1,2,6,7]. According to different studies, tyrosinase family proteins are the antigens specific to VKH disease[1,2, 7].The mechanism that triggers this autoimmune response is unknown, but viral infection may be a triggering agent[1,2] .

Its frequent association with other autoimmune disorders such as Hashimoto's disease, thyrotoxicosis contributes further to hypothesize an autoimmune origin [1,·] VKH has been linked to human leukocyte antigen DR4 (HLA-DR4) and HLA-Dw53, LD-Wa, even though the majority of authors deny any familial inheritance [1].

Melanocytes, in the body affected by the syndrome, are responsible for the typical features of VKH.

Clinical symptoms correlate with destruction of melanocytes in the affected areas such as skin, eyes, ears and CNS. The eye is the most involved organ, and visual impairment are the most frequent and debilitating consequences of the disease[1,].In eyes, VKH is characterized by bilateral, chronic, diffuse granulomatous panuveitis[2], accompanied by characteristic neurological and auditory features[1].

The diagnosis of VKH is based on a combination of clinical findings[1,2]. The first attempt to establish the diagnostic criteria for VKH disease occurred in October,1978 at the annual meeting of the American Uveitis society[2,13].The revised diagnostic criteria for VKH disease was reported by International Committe in an Work-shop on VKH disease on October 19-21,1999 at Los Angeles[2].

The differential diagnosis includes sympathetic ophthalmia, sarcoidosis, primary intraocular B-cell lymphoma, posterior scleritis, and uveal effusion syndrome[1].

The effective therapies for VKH disease are still remain unknown[2]. The principles of therapy are to suppress the intraocular inflammation with early and aggressive use of systemic corticosteroids, followed by slow tapering[1,2,7,12,15,16,17]. Other immuno-modulatory agents (most often cyclosporine) may be needed for non-responsive patients or when corticosteroid side-effects are not tolerated[1,2,12,15,16,17,18]. Visual prognosis is generally good with prompt diagnosis and aggressive treatment[1].

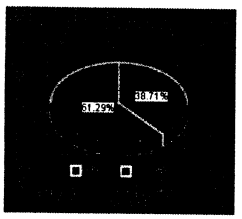
This study was done to describe the clinical profile of VKH disease patients in a homogenious population like Bangladesh.As far we know, there is no such study in bangladeshi population.

METHODS

We retrospectively reviewed the clinical charts of all patients diagnosed with VKH disease seen at Chittagong Eye Infirmary & Training complex between January, 2001 and December, 2005.

All patients had typical ocular findings compatible with VKH disease[2] in the acute, chronic & recurrent stage. Other uveitic conditions had been excluded by medical history, clinical findings, laboratory examination, and ancillary testing. Diagnosis of VKH disease was based on the revised criteria described recently[2]. The following laboratory and ancillary tests were performed: complete blood count with differential, erythrocyte sedimentation rate, blood sugar, blood chemistry, urine analysis, X-ray chest, tuberculin skin test, Venereal Disease Research Laboratory test, Fluorescent Treponemal Antibody Absorption test. In addition, patients were examined with, intravenous Fundus Fluorescein Angiography (FFA), posterior segment ultrasound and Optical Coherence Tomography (OCT) whenever necessary.

A standard data acquisition form was used which included age, gender, initial and final visual acuities, clinical findings (slit-lamp examination, dilated fundus examination) at presentation and at final visit, treatment received (details of corticosteroid therapy)& ocular complications; which were noted from the record.



Statistical analysis

The association between two categorical variables was investigated using Fisher's exact test as appropriate. Fisher's exact test, a P-value less than 0.05 indicated statistical significance. All analytical procedures were done with the help of SPSS 13v.

RESULT

The study cohort consisted of 31 VKH patients. Among them 19 (61%) were women and 12 (39%) were men, the difference between the two percentages was not significant (*P= 0.209*). The age range of our cohort is18 years to 66 years with a mean age 37.58 years (SD = ± 13.49). All cases

TABLE 1. Summary of demography, types of disease with treatment & complication of 31Patients with Vogt-Koyanagi-Harada disease

Age (yrs)	Gender	Types	Stage of Disease	Initial Treatment	Complication	
					RT	LT
18.00	Female	Complete	Acute	Early high dose	None	None
20.00	Female	Incomplete	Acute	Early high dose	Glaucoma	Glaucoma
22.00	Female	Complete	Chronic	Early high dose	Cataract	Cataract
23.00	Male	Complete	Recurrent	Early high dose	Glaucoma	Glaucoma
24.00	Male	Complete	Chronic	Early high dose	None	None
25.00	Male	Complete	Chronic	Low dose	Glaucoma	Glaucoma
25.00	Male	Complete	Recurrent	Early high dose	Glaucoma	Glaucoma
26.00	Female	Incomplete	Recurrent	Early high dose	Cataract	Cataract
27.00	Male	Incomplete	Acute	Early high dose	None	None
30.00	Male	Incomplete	Chronic	Early high dose	None	None
30.00	Female	Complete	Chronic	Early high dose	Glaucoma	Glaucoma
30.00	Female	Incomplete	Chronic	Late high dose	None	None
30.00	Female	Incomplete	Chronic	Low dose	Macular degeneration	
32.00	Male	Complete	Acute	Early high dose	Macular degeneration	
34.00	Female	Incomplete	Chronic	Early high dose	Soft eye	Soft eye
36.00	Male	Complete	Acute	Low dose	Cataract	Glaucoma
36.00	Male	Incomplete	Recurrent	Early high dose	Cataract	Cataract
36.00	Female	Incomplete	Acute	Early high dose	None	None
40.00	Female	Complete	Acute	Early high dose	Glaucoma	Glaucoma
41.00	Female	Complete	Chronic	Low dose	None	None
42.00	Male	Complete	Chronic	Early high dose	Cataract	Cataract
42.00	Female	Complete	Acute	Early high dose	None	None
45.00	Female	Incomplete	Chronic	Early high dose	Cataract	Cataract
47.00	Female	Incomplete	Chronic	Early high dose	Cataract	Cataract
50.00	Female	Complete	Chronic	Low dose	Glaucoma	Glaucoma
50.00	Female	Complete	Acute	Early high dose	Cataract	Glaucoma
55.00	Female	Complete	Chronic	Late high dose	Cataract	Cataract
57.00	Female	Complete	Chronic	Early high dose	Cataract	Cataract
63.00	Male	Complete	Chronic	Early high dose	Cataract	Cataract
63.00	Male	Incomplete	Chronic	Low dose	Cataract	Cataract
66.00	Female	Complete	Chronic	Low dose	Cataract	Cataract

were bilateral. 62 eyes of 31 patients were analyzed.

According to the revised diagnostic criteria[2], at onset 19 (61.29%) patients had the complete form of the disease, 12 (38.71%) had the incomplete form (Table: 2). the difference between the two percentages was not clinically significant (*P= 0.209*).From patient's record we could not find any patient without neurological along with continuous sign (Probable VKH) [2].

9 (29.3%) patients presented in the acute stage, 18 (58.06%) in the chronic stage and 4 (12.90%) with the history of recurrent (Table: 2).

At presentation we got visual acuity better than 6/9 in 13 (20.97%) eyes, within 6/12- 6/60 in 12 (19.35%) eyes and less than 6/60 in 37 (59.68%) eyes (Table:3). After treatment visual outcomes were good, visual acuity 6/9 or better in 33 eyes (55%), within 6/12- 6/60 in 17 (28.33%) eyes and less than 6/60 in 10 (16.67%) eyes (Table: 3).Vision after treatment of one patient (2 eyes) were missing from record.

The difference between the two percentages (before & after treatment) was significant at 6/9 or better level (P =0.0001*)* as well as at 6/60 or worse level (*P* = 0.00001) (Table: 3). So the visual improvements were significantly good in this study.

All eyes had mild anterior chamber (AC) reaction of 1+ to 2+ cells except for 3 (4.54%) eyes that had 3+ cells. One eye showed coagulam in AC. Mutton fat KPs were found in 4 eyes (Table: 4)

In 07 (7.6%) eyes, fundus could not be seen at presentation (due to media opacity) & in 2 eyes fundus could make out through hazy media, in 18 (14.6%) eyes fundus examination showed hyperaemia and swelling of the optic disc, 13 (10.6%) eyes showed exudative retinal detachment , 11 (8.9%) eyes showed macular edema. Dalen Fuchs nodules were observed in 25 (20.3%) eyes, Sunset glow fundus was noted in case of 44 (35.8%) eyes during first visit (Table: 5).

FFA was performed in all cases with clear media. Multiple pinpoint hyperfluorescent leaks at the level of the retinal pigment epithelium, staining of the disc, and late accumulation of fluorescein in the subretinal exudates were observed in our patients. But in chronic cases with sunset glow we

Table. 2: Distribution of Types and Stage of disease:

Visual acuity	Before treatment		After treatment	
	N	%	N	%
6/6-6/9	13	20.97	33	55.00
6/12-6/60	12	19.35	17	28.33
<6/60	37	59.68	10	16.67
Total	62	100	60	100

Table: 3. Visual acuity distribution before and after treatment of VKH patients.

Types	Stages				P value
	Acute	Chronic	Recurrent	Total	
Complete	5	11	3	19 (61.29%)	0.802
Incomplete	4	7	1	12 (38.71%)	
Total	**9 (29.04%)**	**18 (58.06%)**	**4 (12.90%)**	**31 (100%)**	

Table 4: Anterior chamber reaction before and after treatment reaction

	N	% of findings
1+	13	19.70
2+	14	21.22
3+	3	4.54
Coagulam	1	1.51
Mutton Fat KP	4	6.06
Normal	31	46.97

get generalized distribution of hyper and hypo fluorescent patches.

Fig 2: Clinical features of VKH disease. (A) Vitiligo (B) Papilitis (C) FFA - Multiple pinpoint hyperfluorescent leaks at the level of the retinal pigment epithelium, staining of the disc, and late accumulation of fluorescein in the subretinal exudates. (D) Exudative RD (E) FFA - accumulation of fluorescein in the subretinal exudates (F) Sunset glow fundus with Dalen-Fuchs nodules (G) Generalized distribution of hyper and hypo fluorescent patches.

Table5: Fundus before and after treatment

Fundus Findings	Before treatment N	(%)
No view	7	7.6
Papillitis	18	14.6
Exudative Retinal Detachment	13	10.6
Hazy	2	1.6
Dalen-Fuchs nodules	25	20.3
Macular edema	11	8.9
Sunset glow fundus	44	35.8
Total	**120**	**100.0**

After getting treatment, fundus still could not be seen in 04 (6.3%) eyes & 2(3.1%) had hazy media. Papillitis, macular edema and exudative retinal detachment resolved in all cases except one had mild papillitis. Depigmentation of the fundus was evident in 41(64.1%) eyes resulting in the characteristic appearance known as 'sunset glow' fundus. In addition, multiple small, yellow, well-circumscribed nodule (Dalen-Fuchs nodules) developed in the midperiphery of the fundus in 16 (25%) eyes (Table: 5).

All patients with acute stage & some patient with chronic stage were treated with systemic corticosteroids therapy for averaged 6 months, but were prolonged in patients who developed chronic uveitis. Most of the patients treated with Oral prednisolone (1 mg/ kg body wt/day) for a week then tapered by 10 mg weekly. Tapering to the dose of 10 mg per day within a 2-months period. The duration of systemic corticosteroid therapy ranged from 3 to 18 months. 4 (13.32%) patients got high dose of initial systemic prednisolone as intravenous Inj. Methyl prednisolone 1gm daily for 3 days followed by oral prednisolone (1 mg/ kg of body weight per day) (Table 6). 8 (26.68%) patients did not get any systemic prednisolone, they were only treated with topical corticosteroids and cycloplegic agents (Table 6).

Anterior segment inflammation was treated with topical corticosteroids and cycloplegic agents in all cases. No patients required immunomodulator

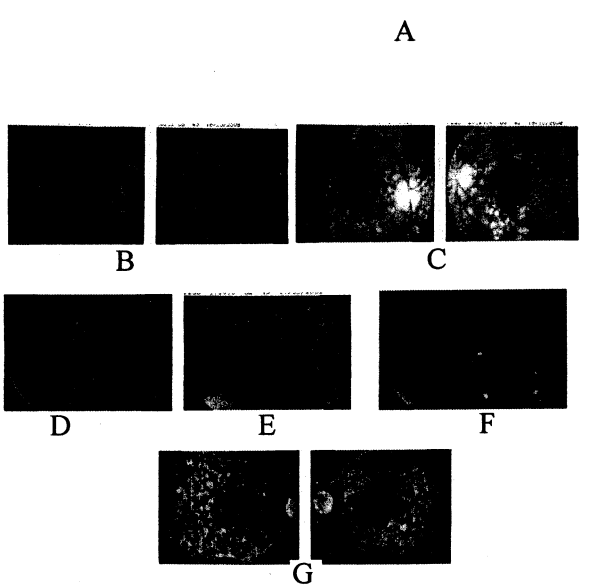


Table 6: Initial corticosteroid therapy

Systemic prednisolone	N	Percent
Inj. Methyl prednisolone 1gm daily for 3 days followed by oral prednisolone (1 mg/ kg body wt/day)	04	13.32
Only oral prednisolone (1 mg/ kg body wt/day)	18	60.00
None	08	26.68
Total	30	100.00

Table 7: Maintaining dose of oral prednisolone.

Dose	N	Percent
10 mg daily	11	36.63
7mg daily	02	6.70
5 mg daily	09	29.97
No maintaining doses	08	26.70
Total	**30**	**100.00%**

in addition to corticosteroids during our study period. Data were missing in one patient.

11 (36.63%) patients got maintaining dose of 10 mg of oral prednisolone daily, 2 (6.70%) got 7 mg oral prednisolone & 9 (29.97%) got 5 mg of oral prednisolone daily (Table7). 8 (26.7%) patients did not have any maintaining oral prednisolone.

We did not get any complications in 16 (25.81%) eyes. 46 (74.19%) eyes showed at least one complication. 28 (45.16%) eyes developed cataract, 14 (22.58%) glaucoma, 2 (3.23%) eyes showed sub retinal fibrosis, and 2 (3.23%) eyes became soft eye (Fig:2).

Disscussion

The VKH is one of the major causes of posterior uveitis. The disease usually affects patients between ages of 20 and 50 years[1, 10, 11]; however, there have been reports of VKH in children[7,8,9]. The age range of our cohort is18 years to 66 years which is consistent with the references of literatures. The mean age of our series is 37.58 years (SD = ± 13.49), nearly similar to the series of Yumiko Yamaguchi et al[10] & Tugal-Tutkun I's [11] (mean age at presentation is 31 years in both studies). Study population of Snyder DA[13] with the mean age of 39.7 years. The mean age was 34 years in study population of Read RW, Rechodouni A et al[16] .Sachdev N [18] included VKH patients with a mean age of 33.7 years.

Most series indicate that women are affected more often than men, [1, 2, 6,7,11,13,16,18] like our series in Bangladeshis 61% women and 39% men. Abu El-Asrar1 et al[7] get 87% girls and 13% boys in their paediatric group. Tugal-Tutkun I's [11] series consist of 32 female, 13 male. Snyder DA[13] had 60% of female in his cohort. Read RW, Rechodouni A et al[16] get 67% female in their series. . Sachdev N [18] included 5 women and 2 men in his study.

According to the revised diagnostic criteria[2], at onset 19 (61.29%) patients had the complete & 12 (38.71%) had the incomplete form of disease,none could be categorise as probable VKH[2]. The difference between the percentages of two categorise was not clinically significant (*P= 0.209*).But the findings in Turkish patients by Tugal-Tutkun I's [11] is different, he got 20% of patients had the complete form, 51% had the incomplete form of the disease, and 29% had probable VKH disease

Of our series 9 (29.3%) patients presented in the acute stage, 18 (58.06%) in the chronic stage and 4 (12.90%) with the history of recurrence. In Tugal-Tutkun I's [11] series 42% patients presented in the acute or subacute stage and 58% in the chronic stage, we also get 58.06% in the chronic stage.

Visual outcomes were good in our series, 33 (55%) eyes got 6/9 or better vision, 17 (28.33%) eyes achieved vision within 6/12-6/60 & only 10 (16.67%) eyes had vision less than 6/60.This finding is consistant with that of other studies in literature. Rubsamen PE, Gass JD et al[12] get good visual outcomes , with final visual acuity of better than 20/30 in 66% of eyes and of worse than 20/400 in only 7% of eyes. Visual acuity of 20/40 or better was achieved in 82.6%eyes of paediatric series of Abu El-Asrar1 et al[7] by final follow -up. Tugal -Tutkun I's [11] get final visual acuity was better than 0.5 in 59%, between 0.1 and 0.5 in 21%, and less than 0.1 in 20%. Read RW, Rechodouni A et al[16] showed final visual acuity of better than 20/40 in 49% of eyes,22% obtained 20/50 to 20/100 and only 29 % of eyes with final visual acuity of worse than 20/200.

In Tutkun I's [11] series Sunset-glow fundus was observed in 89% of eyes. We observed Sunset-glow fundus in 35.8% at initial visit & in 64.1% at final visit.

Most of our patients (73.32%) were treated with Oral prednisolone (1 mg/ kg body wt/day). (Table 6).This treatment is similar to other authors. All patients of acute VKH in Tugal-Tutkun I's [11] series received systemic corticosteroid therapy. All patients of Rubsamen PE, Gass JD et al[12] series were treated with systemic corticosteroids for

averaged 6 months, but was prolonged (48 months) in patients who developed chronic uveitis

4 of our patients got intravenous Injection Methyl prednisolone 1gm daily for 3 days followed by oral prednisolone because they had a worse initial visual acuity and bullous exudative RD at presentation. Intravenous corticosteroids were used more frequently in eyes that had a worse initial visual acuity and bullous exudative RD at presentation[7].

Literatures suggest early and aggressive use of systemic corticosteroids, followed by slow tapering for VKH treatment[7, 12, 15, and 17]. Such treatment may shorten the duration of the disease, may prevent progression into the chronic stage and may reduce the incidence of extra ocular manifestations as well[7, 12, 15, and 17].

16 (25.81%) eyes of our cohort did not show any complication. 46 (74.19%) eyes showed at least one form of complication. We got cataract in 28 (45.16%) eyes, glaucoma in 14 (22.58%) eyes, sub retinal fibrosis in 02 (3.23%)eyes , and phthysis bulbi in 02 (3.23%) eyes (Fig.2).It is nearly similar to the findings by Tugal-Tutkun I[11],his series showed cataract in 53%, glaucoma in 29%, subretinal fibrosis in 22%, choroidal neovascular membranes in 7%, and phthisis in 4%[11]. Read RW, Rechodouni A et al[16] showed 53% get at least one complication, their findings are cataract in 42%, glaucoma in 27 %, subretinal fibrosis in 6 % and choroidal neovascular membranes in 11% .In Abu El-Asrar1 et al[7] series 11 eyes developed at least 1 complication, including cataract in 8 eyes, glaucoma in 8 eyes, subretinal neovascular membranes in 2 eyes, and subretinal fibrosis in 1 eye[7].

CONCLUSIONS

VKH disease is not rare in Bangladesh. Based on our results, most patients with **VKH** seem to be late referrals. Ocular complications were common among these patients. VKH disease may involve predominantly the posterior segment, and respond well to the corticisteroid therapy. Early detection and prompt management may prevent many visual disabilities in VKH cases.

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Advanced Stage of Secondary Glaucoma in a Patient of Weill-Marchesani Syndrome: A Case Report

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Abstract

A young adult male patient presented to glaucoma clinic of national institute of ophthalmology & hospital, Dhaka with the complints of gradual dimness of vision in both eyes for five years. Typical features were bilateral subluxated lens (infero-temporal), micospherophakia, shallow anterior chamber with 360^0 angle close, short stature (brachymorphy), short and stubby fingers (brachydactyly). Before attending to NIO&H the patient was diagnosed as primary angle closure glaucoma and treated accordingly. In glaucoma clinic of NIO&H we examined the patient with slit lamp after dilatation of pupil; intraocular pressure was measured with applanation tonometer, anterior chamber angle was examined with gonioscope. The patient was diagnosed as Weill-Marchesani syndrome with advanced secondary angle closure glaucoma. In our centre the patient was managed initially with medical treatment then laser peripheral iridotomy and finally by extraction of crystalline lens with AC IOL implant. During subsequent follow up it is noted that intraocular pressure was normal and preexisting vision was maintained well.

It is concluded that ophthalmologist can play a crucial role for meticulous examination of patient with glaucoma. Thorough examination of ocular and systemic conditions of patient with narrow angle glaucoma is essential for early diagnosis and proper management of Weill-Marchesani syndrome. ***Key:*** *Glaucoma, surgery, Weill-Marchesani syndrome.*

Introduction

Weill-Marchesani syndrome is a rare systemic connective tissue disease characterized by dystrophia mesodermalis hyperplasia. Inheritence is autosomal recessive. Clinically it is opposite to the patients with Marfan syndrome[1]. Systemic features include short stature (brachymorphy), short stubby fingers (brachydactyly) and mental handicap. Ocular features include microspherophakia, lens subluxation (inferior and bilateral), angle anomaly associated with mesodermal dysgenesis. Causes of secondary glaucoma are pupil block by small and spherical lens and angle anomaly. Treatment include mydriatics and timolol eye drop, laser iridotomy, lens extraction and trabeculectomy according to necessity[2].

Case report

Md. Gazi of 34 years attended to glaucoma clinic of National institute of ophthalmology and hospital with the complaints of gradual dimness of vision in both eyes for five years. On ocular examination visual acuity was found cf. 5 ft. in right eye and 3 ft. in left eye and no improvement with pinhole. Slit lamp examination revealed microspherophakia, subluxated lens inferiorly, shallow anterior chamber with unequal AC depth in both eyes (Fig.1). On fundoscopic examination C:D ratio was ·9:1 in right eye and ·95:1 in left eye

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(Fig.2). Intraocular pressure was 56 mm Hg in both eyes. Gonioscopically anterior chamber angle was close 360^0. On systemic examination mental health was normal, short stature (Fig3), short and stubby fingers in both limbs (fig.4). On family history there was no history of consanguinous marriage and parents were normal in appearance. He had one sister and two brothers who were normal. Before attending to NIO&H the patient was diagnosed as chronic primary angle closure glaucoma and treated with pilocarpine and timolol eye drop. In our centre we diagnosed the patient as Weill-Marchesani syndrome with secondary angle closure glaucoma. Initially we treated the the patient medically with tropicamide, timolol, brimonidine eye drop and oral acetazolamide. After treatment intraocular pressure was reduced from 56 to 26 mm Hg. Then we followup the patient for two month. After that we did laser peripheral iridotomy in both eyes in two sittings. Again we follow up the patient for three month and intraocular pressure was found 19 mm Hg. Then we did extraction of lens with AC IOL implant in both eyes one after another. The patient was followed up regularly and intraocular pressure was found 12 mm Hg in both eyes. Now the patient is mainting his daily life with existing vision.

Discussion

Angle closure glaucoma is classified as primary and secondary. Again primary and secondary angle closure glaucoma are classified as pupil block and without pupil block. Every patient with angle closure glaucoma should be examined carefully to exclude any secondary causes of angle closure. Ectopia lentis is one of them. Common causes of ectopia lentis are trauma, exfolliation, Marfan syndrome, homocystinuria, microspherophakia and Weill-Marchesani syndrome[3]. In ectopia lentis pupillary block may occur which results in iris bombee, shallow anterior chamber and secondary angle closure. Other association of Weill-Marchesani syndrome is myopia.

The current study addresses the association of secondary glaucoma in a patient of Weill-Marchesani syndrome. This patient had short stature, short and stubby fingers, microspherophakia, subluxated lens. Subluxation of lens is the most important manifestation of Weill-Marchesani syndrome. Both microsph erophakia and subluxation of lens can be missed easily in early stage of disease if careful examination is not done under full mydriasis.

At present the patient is close to blind due to advanced stage of glaucoma. Early proper diagnosis and treatment could prevent the present situation of the patient. Lack of awarness of the patient is also a factor for this condition. In case of failure of medical regime and laser iridotomy early extraction of lens perhaps could save the patient from such a bad prognosis. Byoung sun chu and A.Ryo et.al. found the simillar bad prognosis of advanced glaucoma in a patient of Weill-Marchesani syndrome[4].

Conclusion

Like myopia secondary glaucoma is an important association of Weill-Marchesani syndrome. So our message is that ophthalmologist can play a crucial role in examining the patient of glaucoma for early diagnosis and proper management of secondary glaucoma in a patient of Weill-Marchesani syndrome to prevent the threatened complication like blindness.

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Congenital Nasolacrimal duct obstruction: Treatment modalities and their out come

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Abstracts

This prospective study was conducted in Sadar hospital, Narail during the period of January 2006 to December 2008. Total 224 epiphora cases aged from 0- 4 years were included in this study, out of which 178 (19.20%) were diagnosed as congenital nasolacrimal duct obstruction (CNDO). 152 (85.39%) cases were cured by conservative treatment (Lacrimal massage and antibiotic eye drop. Out of remaining 26 (14.61%) cases: 20 (76.92%) cases were symptom free on 1st probing, 04 (66.67%) on 2nd probing and 02(33.33%) did not improved even after subsequent probing, which needed surgical intervention. The purpose of our study was a) to find out of incidence of CNDO and b) to find out the out come of digital massage and probing in CNDO.

Majority of patient (85.39%) respond well to digital massage. After one year of age the efficacy of digital massage decreases. So early diagnosis and counseling of parents are very much important to manage congenital nasolacrimal duct obstruction.

Introduction

Epiphora is a common problem in children. This is mostly due to failure of canalization of distal end of nasolacrimal duct (NLD), accounting for about 20% cases[1] . Digital massage over the sac is the initial management of this condition. Probing is the second choice of management if the digital massage fails to make the patient symptom free. Most of the patients become symptom free by digital massage. Probing within first 1-2 years has a very high success rate but there after efficacy decreases[2]. This study was conducted to evaluate the efficacy of digital massage and probing in the management of congenital nasolacrimal duct (CNLD) obstruction in the age group of 0-4 years.

Methodology

This prospective study was carried out in the outpatient department (OPD) of Narail Sadar Hospital during the period of January 2006 to December 2008. During this period 927 children aged 0- 4 years attended OPD, out of which 224 had epiphora and 178 were diagnosed as CNLD obstruction (19.20%).Diagnosis was made on the basis of history of watering in one or both eyes from the child's parents and by physical examination, particularly regurgitation test. In 44 cases (4.75%) watering was due to conjunctivitis, foreign body in the eye and others. Initially all the cases were treated by digital massage and topical antibiotic eye drops at out-door. The parents were meticulously trained about how to give massage over the sac, how to instill eye drop and how to maintain lid hygiene. After 6 months first probing were done in those who didn't become symptom free and second probing were done after another 6 months in those who were not cured by first probing. These patients were admitted in the hospital and probing was done under general anesthesia. Symptoms free conditions were assessed, recorded and analyzed.

Results

The efficacy of digital massage and probing were recorded, analyzed and expressed in tables as follows:-

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Table- I: 0 - 4 years child attended with complain of watering at out patient department of Narail Sadar Hospital in the year 2006 – 2008

Events.	0 – 6 Months	6months -1 year	1-2 years	2-4 years	Total (%)
Total patient.	255	203	154	315	927 (100%)
Patient of watering	93	51	37	43	224 (24.16%)
Patient of watering due to CNLD obstruction	86	39	24	29	178 (19.20%)
Patient of watering due to other than CNLD obstruction.	6	12	13	14	44 (4.96%)

Table – II: Age-distribution of CNLD obstruction (n = 178)

Age	No. of patient	Percentages %
0-6 months	86	48.31%
6months-1 yr.	39	21.91%
1-2 yrs.	24	13.48%
2-4 yrs.	29	16.30%
Total	178	100.00%

Table - III: Conservative management -digital massage (n = 178)

Age	No. of patient	Symptom free(%)	Symptomatic (%)
0-6 months	86	84 (97.67%)	02 (2.33%)
6months-1 yr	39	36 (92.30)	03 (7.70%)
1-2 yrs.	24	20 (83.33%)	04 (16.67%)
2-4 yrs.	29	12 (41.13%)	17 (58.87%)
Total	178	152 (85.39%)	26 (14.61%)

Table -IV: 1st probing : after 6 months of conservative management: (n = 26)

Age	No. of patient	Symptom free (%)	Symptomatic (%)
0-6 months	02	02 (100%)	00
6months-1 yr	03	03 (100%)	00
1-2 yrs.	04	03 (75.00%)	01 (25.00%)
2-4 yrs.	17	12 (70.58%)	05 (29.42%)
Total	26	20 (76.92%)	06 (23.08%)

Table- V: 2nd probing : after 6 months of 1st probing : (n = 06)

Age	No. of patient	Symptom free (%)	Symptomatic (%)
0-6 months	00	-	-
6 months-1 yr	00	-	-
1-2 yrs.	01	01 (100%)	
2-4 yrs.	05	03 (60%)	02 (40%)
Total	06	04(66.67%)	02(33.33%)

Discussion

The development of NLD begins with the formation of a solid cord of ectoderm between the maxillary and lateral swellings & canalization of this cord .The lower end of the NLD is the last part to canalize, complete canalization usually occurring soon after birth. The canalization may be delayed leading to epiphora in about 20% new-born[3,4].

This study showed 19.20% of children aged 0- 4 years have NLD obstruction (Table—I) and majority of them present within 6 months (Table-II). Kushner BJ recorded in his study that 30% termed infants had NLD obstruction at birth[5].

It was revealed from this study that 85.39 % were symptoms free by digital massage and antibiotic eye drop (Table- III) .The success rate was much higher, 96% in 0 – 1 years group (Table-III).

Others studies showed that epiphora disappear by conservative treatment in 96% - 98% cases[6] , 72% cases[7,8], 85% cases[9,10] & 95% cases[11].

In the age group of 0- 2 years, 08 (88.88%) cases out of 09 became symptom free after 1st probing (Table-IV) and remainingg 01 was symptom free(Table-V)after 2nd probing. Dr. Md. Forhad Hossain in his study showed that 95.83% success rate in age group of 1-2 years[12].

Conclusion

Epiphora is common problem in children and majority of the cases particularly in the age group of 0-1 year (96%) became cured by digital massage. After one years of age the efficacy of digital massage decreases. So early diagnosis and counseling of parents are very much important to manage congenital NLD obstruction.

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Prevalence of Refractive Errors in School Children of Rural Areas of Bangladesh

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Abstracts

The aim of this study is to determine the prevalence and type of refractive errors and its consequences in school going children. Primary data was collected by random sampling from 300 students of class 1X and X of three schools in the rural areas of Narail district. Visual acuity was tested by Snell en's chart. Retinoscopic and Ophthalmoscopic findings, findings related to refractive errors , unaided and corrected visual acuity were noted. Out of 300 students 47 had refractive error (15.66%), out of which myopia- 12.00%, hypermetropia - 2.33% and astigmatism -1.33%. One children developed amblyopia. School children form a major part of our population . So, to prevent blindness, to reduce ophthalmic morbidity every educational institution should provide vision screening facility & all the student should be examined for acuity of vision.

Introduction

Visual handicaps are burdens not only for themselves but also for their families and society. Refractive error is the commonest cause of defective vision. If it is not detected and treated in time it may result in permanent visual disability like amblyopia, strabismus, nystagmus etc. So, it is vital to detect and correct the errors timely.

The aim of this study was to determine the prevalence and type of refractive errors and its consequences in school going children.

Methodology

Primary data was collected by random sampling from 300 students of class 1X and X of three schools in the rural areas of Narail district. The questionnaire was prepared and recorded the information, particularly complaints related to eye and vision. It was followed by Snellen's chart, Retinoscopy and Ophthalmoscopy. Collected data was revised in accordance with the objective of our study.

Results

Out of 300 students 47 had refractive error (15.66%).Results are shown in tables.

Discussion

The prevalence of refractive error was 15.65% in this study. Similar findings were observed by Islam[1], where prevalence rate was 11.67%. In this study it was found that myopia -76.59% ,hypermetropia - 14.90% and astigmatism -

Table -I : Presentation (N=47)

Presentation	Number	Percentage
Defective vision	19	40.43%
Headache	15	31.95%
Incidental	13	27.69%
Total	47	100%

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Table -II : Types of refractive errors: (N = 47)

Types	Number	Percentage
Myopia	36	76.59%
Hypermetropia	07	14.90%
Astigmatism	04	8.51%
Total	47	100%

Table -III: Severity of refractive errors: (N = 47)

Severity (visual equity)	Number	Percentage
Less than 6/60	02	4.26%
6/60	03	6.38%
6/36	09	19.15%
6/24	12	25.53%
6/18	03	6.38%
6/12	18	38.30%
Total	47	100%

Table -IV: Visual acuity after correction: (N = 47)

V/A	Number	Percentage
6/6	44	93.62%
6/9	02	4.26%
6/18	01	2.13%
Total	47	100%

8.51%(table-II). Yeamli Khan et al[2] found myopia - 48.83%,hypermetropia - 27.91% and astigmatism - 23.26%. Among the students with refractive error, 40.43% (19)complained visual disturbances, 31.95% (15) headache and 27.69%(13) were detected incidentally (table-I).

This is comparable with a similar study of Md. shariful islam bhuiya[3].The severity of refractive error in terms of unaided visual acuity (table-III) was less than 6/60 in 2 cases(4.26%), 6/60 in 3 cases(6.38%), 6/36 in 9 cases(19.15%), 6/24 in12 cases(25.53%), 6/18 in 3 cases(6.38%) and 6/12 in 18cases(38.30%). After correction (table-IV) visual acuity was 6/6 in 44 cases (93.62%), 6/9 in 02 cases (4.26%), and 6/18 in 01 cases (2.13%). Ophthalmoscopy revealed normal findings in all cases. That is one children developed amblyopia.

Conclusion

Refractive error is one of the most important causes of treatable blindness.

The prevention of blindness in children is considered as one of the main priorities of the World Health Organization's VISION 2020 – the Right to Sight programme .Almost half of children in the world become blind from avoidable causes of which 15% are treatable and 28% preventable. In this study 15.65% of school children in a rural area were found to have refractive error and one children developed amblyopia. So knowledge about refractive error should be incorporated with National Eye care program that is primary health care. Every educational institution should provide vision screening facility and all the student should be examined for acuity of vision . Strenthening of school health programme with vision test at an regular interval should be provided.

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Small Incision Cataract Surgery with clear corneal incision and with scleral tunnel incision : A Comparative study

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Abstracts

The increasing number of cataract blindness is due to low surgical output and high rate of poor surgical out come. Phaco surgery is the modern technique of extra capsular cataract extraction but difficult to master. Alternately Manual Small incision cataract surgery (SICS) is cost effective and easy to learn. So it may help to reduce the burden of cataract blindness and achieving the target of vision 2020 would be possible. In our country most of the surgeon's practice SICS with scleral tunnel incision, but it can also be performed by clear corneal incision. This prospective study was conducted on 100 eyes of 80 patients in Khulna Medical College Hospital, Khulna. The selected cases were divided into two groups –Group A (50 eyes) under went SICS with clear corneal incision and group B(50 eyes) underwent SICS with scleral tunnel incission.In this study different parameter between two groups were observed and analyzed . It was found that final visual out come was slightly better, mean astigmatism, per and post operative complications were less in group A that is SICS with clear corneal incision. More over this procedure is less time consuming. Considering all this points SICS with clear crneal incision may be better option for cataract surgery.

Introduction

The number of blind people due to cataract increases annually. This cataract crisis is not only due to low surgical output but also due to high rate of poor surgical out comes.

Surgical technique, complications and induced astigmatism are major contributors to poor surgical out comes[1,2,3]. These are common in conventional extracapsular catarct extraction (ECCE) where incisions are secured by sutures. Phacoemulsification with self sealing small incision (up to 3mm) greatly reduces post-operative astigmatism[4] and speeds the visual rehabilitation. But it is very expensive and difficult to master due to complex instrumentation.. Alternately Manual Small incision cataract surgery (SICS) needs simple instrumentation. It is easy to learn and cost effective. In our country most of the surgeon's practice SICS with scleral tunnel incision, but it can also be performed by clear corneal incision. The aim of our study is to explore the merits and demerits of SICS with clear corneal incision over SICS with scleral tunnel incision.

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Methods and Materials

This prospective study was made on 100 eyes of 80 cataract patients admitted in Khulna Medical College Hospital, Khulna during the period of July 2008 to June 2010. Nuclear, cortical, posterior sub capsular and mature cataracts were included in this study.

Traumatic. Complicated, congenital and cataract associated with any complication were excluded. Total cases were divided into two groups – Group A and group B, each containing 50 cases. Group A (50 eyes) under went SICS with clear corneal incision and group B(50 eyes) underwent SICS with scleral tunnel incision.

In group – A (50 cases), an external incision 4.5 to 6.5 millimeter (according to size of the nucleus) was made at the anterior limbus. With crescent knife corneal tunnel was made, 1.5 to 2 millimeter (mm) into clear cornea, anterior chamber (A/C) was entered with a 3.2 mm angled keratome & incision was enlarged with 5.1 mm angled keratome.

A/C was formed with viscoelastic substance, a large capsulorhexis (6mm) was made using a cystitome & capsulorhexis forcef. Hydro-dissection and nuclear dislocation was performed by injecting ringers solution beneath the anterior capsule. Nucleus was prolapsed into A/C by Sisky hook and then expressed by a irrigating vectis.

In group – B (50 cases), after peritomy and cauterization an external incision of same length was made on the sclera 1.5 to 2.0 mm behind the limbus. With crescent knife scleral tunnel was made up to the clear cornea and A/C was entered with a 3.2 mm angle keratome.The subsequent steps were same as those in group – A. In all cases rigid PMMA intra ocular lens with 6 mm optic size were implanted in capsular bag. All operations were done under peribalber anesthesia.

Results: The observations and results are shown in the tables:

Table I. Age distribution.

Sl. No.	Age group	Group -A		Group - B	
		No of eyes	Percentage	No of eyes	Percentage
1.	30-40	04	08%	04	08%
2.	41-50	04	08%	04	08%
3.	51-60	16	32%	16	32%
4.	61-70	18	36%	18	36%
5.	71-85	08	16%	08	16%
Total		50	100%	50	100%

Table – II: Morphology of cataract.

Types of cataract	Group-A		Group - B	
	No. of eyes.	%	No. of eyes.	%
Mature cataract	32	64%	32	64%
Posterior sub capsular catarct.	06	12%	06	12%
Cortical cataract	02	04%	02	04%
Nuclear cataract	10	20%	10	20%
Total.	50	100%	50	100%

Table-III: Preoperative visual status with best correction (BCVA) .

Visual acuity	Group -A		Group - B	
	No. of eyes.	%	No. of eyes	%
Hand movement.	22	44%	24	48%
Less than 6/60	10	20%	08	16%
6/60	12	24%	10	20%
6/36	06	12%	08	16%
Total	50	100%	50	100%

Table-IV : Peroperative complications .

Complications	Group-A		Group - B	
	No.of eyes.	%	No.of eyes.	%
Excessive hemorrhage from the wound	00	00%	03	06%
Descemet membrane detachme nt	01	02%	01	02%
Iridodialysis	00	00%	01	02%
Difficult nuclear prolapse in to anterior chamber	01	02%	02	04%
Posterior capsular rent with vitreous loss	01	02%	02	04%
Total	03	06%	09	18%

Table-V: Post-operative complications.

Complications	Group -A		Group - B	
	No. of eyes.	%	No. of eyes.	%
Moderate to severe striate keratopathy	04	08%	08	16%
Moderate to severe uveal reaction	06	12%	06	12%
Hyphaema	00	00%	03	06%
Wound leakage	01	02%	02	04%
Total	11	23%	19	38%

Table-VI: Unaided visual acuity on 1st post operative day.

Visual acuity	Group -A		Group - B	
	No. of eyes.	%	No. of eyes.	%
6/6.	12	24%	10	20%
6/9	09	18%	06	12%
6/12	09	18%	06	12%
6/18	07	14%	10	20%
6/36-6/60	03	06%	06	12%
Less than 6/60	10	20%	12	24%
Total	50	100%	50	100%

membrane detachment occurred in 02% cases, posterior capsular rent in 04% cases , difficult nuclear prolapses in 04% cases, excessive hemorrhage from the wound in 06% cases & iridodialysis in 02% cases . Postoperative complications(table V) were 23% in group-A (clear corneal incision) and 38% in group-B (scleral tunnel incision) - this is also comparable to other study[1,2]. In group – A moderate to severe striate keratopathy occurred in 08%, moderate to severe uveal reaction in 12% & wound leakage in 02% cases.

In group- B on the other hand moderate to severe striate keratopathy occurred in 16%, moderate to severe uveal reaction in 12%, wound leakage in 04% cases and hyphaema in 06%cases.. Visual outcome was also more satisfactory in group - A than group- B (table VI,VII & VIII). Unaided visual acuity on 1st post operative day was -6/12 or better in 60% in group-A and 44% in group-B. After 6 weeks it was- 6/12 or better in 80% in group-A and 62% in group-B. Best corrected visual acuity (BCVA) after 6 weeks was -6/9 or better in 96% in group-A and 90% in group-B. This result of group-A is compatible to other studies on corneal tunnel incision SICS[5] & better than study on scleral tunnel SICS[6] The amount of astigmatism (table-IX) was slightly less in group-A than group-B . In group –A it was .0.50 to 2.50. D in 60% ceases. In group –B it was .0.50 to 2.50. D in 60% cases & 2.75 to 3.75 in 10% cases. Mean astigmatism was 1.28 D in group-A and 1.57 D in group-B - this comparable to other study[5,6]. . Other studies revealed mean astigmatism 0.75 D[7] after clear corneal phaco emulsification with standard 2.8mm incision and 1.25D[4] after clear corneal phaco emulsification with 5.5mm PMMA lens implantation.

Conclusion

Different parameters of SICS with clear corneal and scleral tunnel incisions were observed and analyzed in this study. It was found that final visual outcome was slightly better, mean astigmatism was less and per & post-operative complications were less in clear corneal incision group. It was revealed that clear corneal incision SICS was cost- effective and less time consuming. Its learning curve is also very short so that a large number of ophthalmic surgeons can be trained up in a short period of time. Current Cataract Surgery Rate (CSR) in Bangladesh is only 900[8] .To eliminate blindness due to cataract, current CSR should be raised to 3000 by 2020[9] . Considering all these points SICS with clear corneal incision may be the better option for cataract surgery.

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Table-VII: Unaided visual acuity after 6 weeks.

Visual acuity	Group -A		Group - B	
	No. of eyes.	%	No. of eyes.	%
6/6.	17	34%	14	28%
6/9	11	22%	09	18%
6/12	12	24%	08	16%
6/18	07	14%	10	16%
6/36-6/60	02	04%	06	12%
Less than 6/60	01	02%	03	06%
Total	50	100%	50	100%

Table-VIII: BCVA visual acuity after 6 weeks.

Visual acuity	Group -A		Group - B	
	No. of eyes.	%	No. of eyes.	%
6/6.	41	82%	35	70%
6/9	07	14%	10	20%
6/12	01	02%	03	06%
6/18	01	02%	02	04%
6/36-6/60	00	00%	00	00%
Less than 6/60	00	00%	00	00%
Total	50	100%	50	100%

Table-IX: Post-operative corneal astigmatism:

Amount of astigmatism	Group -A		Group - B	
	No. of eyes.	%	No. of eyes.	%
0.5-1.00	16	32%	14	28%
1.25-2.50	14	28%	16	32%
2,50-3.25	00	00%	05	10%
Total	30	60%	35	70%
Mean astigmatism	1.28		1.57	

Discussion

This study was done to compare the procedures, complications & visual out come between SICS with clear corneal tunnel incision & SICS with sceral tunnel incision . In this study 100 eyes of 80 catarect patient aged 30 years to 85 years(table-I) were selected and divided in two groups. Group-A (50 cases)— selected for SICS wirh clear corneal incision & Group –B – (50 cases) for scleral tunnel incision. Mature , posterior sub capsular, cortical & nuclear cataract were included (table-II) in this study. Preoper ative best corrected visual equity in group- A were: hand movement in 44% cases, less than 6/60 in 20% cases, 6/60 in 24% cases & 6/36 in 12% cases & in group-B it was hand movement in 48% cases, less than 6/60 in 16% cases, 6/60 in 20% cases & 6/36 in 16% cases(TableIII). Both per & post operative complication were more in group-A than group-B[2,3,4,5,6]. Per-operative complications (table IV) occurred in 06% in group-A and 18% in group- B. This is comparable to a study of dr. Md. Abdul Hannan[1] .

In group-A descemet membrane detachment occurred in 02% cases, posterior capsular rent in 02%cases & difficult nuclear prolapses in 02% cases. On the other hand, in group- B descemet

Two advanced cases of *Pseudomonas* keratitis and their management at tertiary centre

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Abstract

Purpose: *The purpose of this study was to report the management and outcome of two advanced cases of corneal ulcer caused by Pseudomonas aeruginosa with non-responded to topical and systemic medications.*

Method: *Clinical observation and surgical intervention of two patients who developed a near total corneal perforation with pseudo cornea and total slough out corneal ulcer by Pseudomonas aeruginosa .*

Results: The first patient: *A 7years old girl had an 8mm left corneal perforation, prolapsed iris covered with exudative membrane protecting from expulsion, vision only PL, PR and B- scan reveled retina was flat and right eye was normal. She was referred for evisceration. Large tectonic Penetrating Keratoplasty (TPK) and Peripheral iridectomy (PI) was done in spite of evisceration. Microbiology shown heavy growth of Pseudomonas aeruginosa in recipient button and no organism in donor button. She gained vision 6/24 unaided, ocular motility and structural integrity till three years from TPK though there were two occurrence of graft rejection.*

The second patient: *A 45-yearsold house-wife referred with advanced form of right corneal ulcer. At presentation to our cornea clinic, her right cornea was total slough out, vision only PL, PR, profuse purulent discharge with mated lashes and 6/6vision in her left eye. Her general conditions were normal. Corneal scraping found Gram negative bacilli and cultured showed heavy growth of Pseudomonas aeruginosa and patient treated with sensitive fortified drops, oral antibiotic but total slough out with impending corneal perforation in her right eye was managed by Conjuntival hood subsequently optical PK and later on SICS with PCIOL, YAG Laser capsulotomy were done and over one and half year she is doing well.*

Conclusions: *Early diagnosis, topical and systemic medications and frequent follow-up is not enough in many cases of Pseudomonas keratitis to halt the devastating diseases process. Many adjunct secondary surgical procedures may be needed for restoration of anatomy and physiology of vision which is possible by expert cornea surgeons in tertiary centre like these cases.*

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Introduction

The healthy cornea is normally very resistant to infection. When the cornea becomes injured, it loses its transparency and protection.

Corneal perforations can result from a variety of disorders and can lead to devastating visual sequelae. *Pseudomonas* keratitis causes rapid corneal descemetoceles and perforations[1]. Despite, recent advances in the medical management of infections keratitis, there remains a subgroup of bacteria, fungi, parasites that does not respond to antimicrobial therapy. *Pseudomonas aeruginosa* is notorious for its resistance to antibiotics and is, therefore, a particularly dangerous and dreaded pathogen. [1]

Fortified antibiotic solutions produce therapeutic concentrations in the corneal stroma while commercially available antibiotic solutions may result in subtherapeutic concentrations[2].

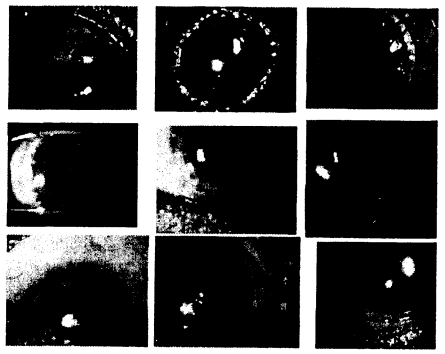
The surgical procedure most commonly used for chronic, infected, or progressive corneal ulcers is a conjunctival hood and or therapeutic penetrating keratoplasty (TPK). Due to scarcity of donor corneas conjuntival flap surgery is the main option in our setting. Conjunctival flaps provide corneal support, fibrovascular tissue to fill corneal defects, and bring blood supply with blood-associated immune components, systemic antibiotics, natural anticollagenases – alpha 2 macroglobulin to the lesion.[3]

When infection and inflammation become subsided and eye is immunologically less reactive then PK is very favorable.

Large- diameter grafts are associated with numerous complications because of the primary problem and the complexity of the procedure. Rejection is a frequent complication despite intensive steroid use.

Case Report-1

In 2007, a 7years old girl student had referred for evisceration with an 8m m corneal perforation,

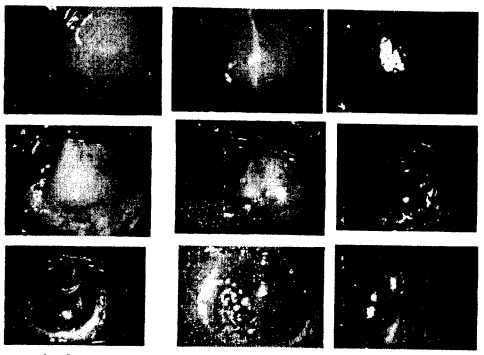


Picture before and after TPK of 1st case 30th month of surgery.

prolapsed iris covered with exudative membrane protecting from expulsion and vision only PL, PR in her left eye and right eye was normal. She had a history nail trauma followed by corneal perforation within 4 days though she received frequent topical and systemic antibiotic. Fortunately that time we got donor cornea and decided to salvage the eye by tectonic PK as there was no retinal/ choroidal detachment. Under general anesthesia large (9.5 and 10 mm) tectonic penetrating keratoplasty (TPK) and peripheral iridectomy (PI) was done. Microbiology showed heavy growth of *Pseudomonas aeruginosa in* recipient button and no organism in donor button. .After TPK anterior chamber was formed, return ocular motility, and gained vision counting finger in 1st post operative day .Subsequently peripheral anterior synechia and secondary glaucoma developed. After synechiolysis, intraocular pressure (IOP) became normal. She gained vision 6/24 unaided with correction 6/18 and structural integrity. She developed endothelial graft rejection at 20th month and 26th month after 1st surgery and managed with intensive steroid therapy.

Case Report-2

A 45-year-old house-wife referred with advanced form of right corneal ulcer in March 2008. At presentation to our cornea clinic, her right cornea was totally sloughed out, vision only PL, PR, profuse purulent discharge with mated lashes in her right eye and 6/6vision in her left eye. Her general conditions were normal. Corneal scraping



Picture before and after conjuntival hood, optical PK, cataract surgery, YAG Laser capsulotomy of 2nd case 15th month of PK

found Gram negative bacilli and cultured showed heavy growth of *Pseudomonas aeruginosa* and patient treated with sensitive fortified drops, oral antibiotic (doxicycline 100mg BD for 7 days) but total slough out with impending corneal perforation in right eye was managed by Conjuntival hood for supportive and tectonic reasons. After conjuntival hood surgery she felt better and urged for cosmetsis and vision. Subsequently after counseling optical PK (8.5 mm/9.00mm) and later on SICS with PCIOL done due to cataract developed. She gained 6/60 vision and good looking eye. After PK, peripheral anterior and posterior synechia and secondary glaucoma developed. After synechiolysis, intraocular pressure (IOP) became normal but synechia recurred and IOP 18mmhg right eye; 12mmhg in left eye. Before cataract surgery her endothelial cell count was1300/mm^2 and after cataract surgery endothelial cell count was 940/mm^2

After 2 month of cataract surgery her vision fell to counting finger due to posterior capsular opacification, so YAG Laser capsulotomy was done and recovered vision 6/36. Itis now Over one and half a year that she is doing well.

Discussion

Corneal ulcers can cause significant loss of vision from scarring and astigmatism, but rapid management can limit the destruction and improve outcome.

Most bacterial infections, when managed expediently, responded to therapy, but due to delay in patient's sleekness or obtaining medical treatment the infections may rapidly progress to corneal perforation.

Despite institution of the proper antibiotics, keratitis due to *Pseudomonas* and other Gram-negative organisms may exhibit increased inflammation and tissue destruction during the first 24 to 48 hours.[5]

Rapid destructive inflammatory response, dense stromal suppuration with copious mucopurulent exudates and ground glass appearance of cornea are characteristic features of *Pseudomonas* keratitis.[1]

Our cases were advanced *Pseudomonas* keratitis where one was big perforated with pseudo- cornea and other one was total slough out with impending perforation needed evisceration. Both of these patient received early medications but condition became uncontrolled indicated infrequent and or resistant medications they used.

Cultured report showed that most of the antibiotic were resistant only polymexcin-B and tobramycin sensitive in 1st & 2nd case.

Pseudomonas aeruginosa is notorious for its resistance to antibiotics and is, therefore, a particularly dangerous and dreaded pathogen. *Pseudomonas aeruginosa* is primarily a nosocomial pathogen. It can bind to molecular receptors exposed on injured epithelial cells. It secretes a number of toxins that disrupt protein synthesis and damage cell membranes of ocular cells and proteases that degrade the corneal stromal extracellular matrix. In our 1st case there was nail trauma and other case no such trauma but she had history of ocular itching.

Descemetoceles and perforations are ophthalmic emergencies that required immediate recognition and intervention. Conjunctival hood/therapeutic penetrating keratoplasty and lamellar grafts have been used to prevent and / or treat perforation[6, 7]

Malik and Singh managed 36*Pseudomonas* corneal ulcers with lamellar and penetrating grafts. All 8 of the lamellar grafts became reinfected and one eye was enucleated. Only 4 of the 28 TPK grafts become reinfected.[8] No reinfection occurred in these two cases as 1st case; large graft removed infected tissue and 2nd graft done in corneal scar after removing conjuntival hood .

Hill in 1986 presented 23 patients with deep, indolent ulceration and / or descemetoceles that were treated with TPK.[9] Early surgery did not jeopardize the final outcome of TPK.

The period of morbidity was shorter, and more rapid visual rehabilitation occurred with early keratoplasty, possible 2ndary to the fact that corneal neovascularization was prevented by earlier surgical management.[10, 11]

In our 1st case TPK restored vision, comfort and ocular integrity though she was referred for evisceration. Our 2nd case; intensive fortified topical ceftriaxone and systemic doxicycline did not halt the diseases process rather became impending perforation urged TPK/ Conjunctival hood.

We did conjunctival hood which prevents perforation and causes vascularized corneal scar. So the patient got time for corneal graft at proper time. Cowden et.al concluded that larger diameter (> 9.5 mm) PKS, can salvage an eye that may have otherwise been lost, with reasonable visual rehabilitation.[4]

In our cases synechia, 2ndary glaucoma developed and in one cases graft rejection occurred and managed properly.

Rejection is a frequent complication despite intensive steroid use. Placement of small diameter penetrating keratoplasty within a rejected large – diameter graft may result in visual improvement in some cases.[4]

Early diagnosis, topical and systemic medications and frequent follow-up is not enough in many cases of *Pseudomonas* keratitis to halt the devastating diseases process. Many adjunct secondary surgical procedures may be needed for restoration of anatomy and physiology of vision which is possible by expert cornea surgeons in tertiary centre .

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Management of Meibomian gland carcinoma at Oculoplasty Department of National Institute of Ophthalmology & Hospital, Dhaka—A case report

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Abstract

Malignant eyelid tumors are not uncommon in ophthalmic practice. Most common eyelid tumor is basal cell carcinoma. Meibomian glands are sebaceous glands. Carcinoma of the sebaceous glands is highly malignant and potentially lethal that arises from Meibomian glands of the tarsal plate. Unlike basal cell or squamous cell carcinoma, sebaceous gland carcinoma occurs more frequently in female and originates twice as often in the upper eyelid reflecting the greater numbers of Meibomian and Zeis glands there. Multicentric origin is common and separate upper and lower eyelid tumors occur in 6%- 8% of patients[1]. It starts as a nodule that initially simulates a chalazion but later causes loss of eyelashes and destruction of the Meibomian gland orifices is characteristic[2].

A case of Meibomian gland carcinoma involving upper eyelid of left eyeball in a 60 years of female was presented at the oculoplasty department of National Institute of Ophthalmology & Hospital. The clinical diagnosis, preoperative findings and histopathologic reports are analyzed.

Key words: *Sebaceous gland, carcinoma, frozen section, histologic features.*

Description of the mass

There is a multiple nodular swelling on erythematous base on the upper eyelid of the left eye. Measuring about 20mm x 14mm containing multiple yellowish colour nodule, ulcerated area over lid margin, loss of eyelash on the medial side of the ulcer and matted lashes on the lateral side. Hard in consistency & non tender, moves with the movement of the lid. The tumour grew as a single nodule on the upper eye lid of left eyeball 1 year back then it grew gradually and ulcerated on lid margin.

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Figure: Preoperative view of the tumour.

Figure: Post operative view on 1st POD.

On examination her visual acuity of right eye 6/12 unaided and with pinhole 6/12 and left eye 6/12 unaided and with pinhole 6/12, there is nuclear sclerosis on lens of both eyes. Palpebral fissure height of right eye is 10mm and left eye is 5mm. There is ulcerated area over the lid margin of left eye over the growth. Loss of eyelashes on medial side and matted lashes on lateral side. Eyelashes are matted on lateral side and loss of lashes on medial side of left upper eyelid. There is no lymphnode enlargement on preauricular or cervical lymphnode. Others are normal in both eyes.

Pre operative findings

The tumour was situated on the upper lid of left eye ball consisting of multiple nodules, color of the nodules are yellowish. At lid margin there was ulcerated area measuring about 6mm x 2mm with loss of eyelash on medial side and matted lashes on lateral side. Hard in consistency but non-tender. Intraocular pressures of both eyes are 10mm of Hg by Applanation Tonometer. Ocular motilities are full in all gages in both eyes. Palpebral fissure height of right eye was 10mm and left eye was 5mm. There were no history watering in both eyes and puncta are normal. There was no exposure keratitis in left eye.

Operative management

As our clinical diagnosis was Meibomian gland carcinoma we decided for operation but our aim is minimal loss of tissue with restoration of normal function of the eye. We choose frozen section biopsy during operation, remove the entire tumour and wound was repaired by lid sharing method. We tried minimal tissue loss and the tumour was sent for histopathological examination.

The report of histopathological examination

Microscopic Description- Section shows a malignant tumour made of atypical cells having hyperchromatic and pleomorphic cells with irregular nuclear margin arranged in nests and sheets. Areas of mitosis are seen.

Diagnos is Poorly differentiated carcinoma or Meibomian gland carcinoma

Discussion

Malignant tumours of the eyelids are not uncommon in the OPD of oculoplasty department at National Institute of Ophthalmology, Sere-Bangla Nagor, Dhaka. Most common is Basal cell carcinoma than others. Squamous cell carcinoma, malignant melanoma, Kaposi sarcoma, masquerading neoplasm are also malignant eyelid tumours. The most accurate diagnosis of malignant tumours of eyelids ensured by biopsy. Surgery is the treatment of choice for all malignant tumours of eyelids. Surgical excision affords the advantages of complete tumour removal with histologic control of the margins. Excision has a lower recurrence rate than any other treatment modality. It also offers superior cosmetic results in most cases[3]. Cryotherapy may be used for pagetoid spread and orbital exenteration may be considered for recurrent or large tumours invading through the orbital septum. These tumours usually metastasize to regional lymph nodes but may also spread hematogenously or through direct extension. It is relatively radioresistant[4]. In our case, the tumour size was enough to causing lid defects but overcome it by lid sharing methods. As we excise the tumour completely, in six months follow up there was no sign of recurrence seen.

Figure: Final out come after surgery.

Conclusion

Management of malignant tumours of eyelids has different options, surgical and nonsurgical. It depends upon the size and type of the tumours. If the tumour involves entire lid then it is very difficult to restore the normal function of the eye. Here, we choose frozen section biopsy for minimal loss of tissue with complete excision of the tumour. Frozen section biopsy is necessary for treating such type of tumours and essential for loss of minimal normal tissue and restoring organic function.

References

1. American Academy of Ophthalmology; Orbit, Eyelids and Lacrimal System; Section-7, Page-180.
2. American Academy of Ophthalmology; Orbit, Eyelids and Lacrimal System; Section-7, Page-181.
3. American Academy of Ophthalmology; Orbit, Eyelids and Lacrimal System; Section-7, Page-177.
4. American Academy of Ophthalmology; Orbit, Eyelids and Lacrimal System; Section-7, Page-182.