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Understanding Secondary Angle Closure Glaucoma: Implications for Clinical Practice

M N Islam[1]

In this issue of the JBGS, Dr Shams Mohammed Noman has published a review article on Management of complicated Secondary Angle Closure Glaucoma. He has discussed the risk factors, signs and symptoms, pathophysiology, imaging and the treatment modalities of secondary angle closure glaucoma. He mentioned Neovascular Glaucoma, Iridocorneal Endothelial Syndromes, Infammatory Glaucoma, Aqueous Misdirection Syndrome, Ciliary Body Cysts, Glaucoma following Scleral Buckling Procedures, Glaucoma after Silicone Oil Injection, Ciliary Body Swelling are the major secondary glaucomas.

The author has concluded the secondary angle closure glaucomas require meticulous history taking, clinical examination, if necessary anterior segment imagings like UBM to identify the specific cause if possible and formulate a treatment plan to avoid ocular morbidity.

In this editorial further discussion has been carried out on other aspects of the Secondary Angle Closure Glaucoma

Secondary Angle Closure Glaucoma (SACG) is a significant subtype of glaucoma that requires nuanced understanding and precise management. Distinct from primary angle closure, SACG arises due to identifiable ocular or systemic factors that contribute to angle closure, including neovascularization, inflammation, and various structural abnormalities. These underlying etiologies add layers of complexity to both diagnosis and treatment, presenting unique challenges in preventing disease progression and vision loss. This editorial explores the mechanisms behind SACG, highlights key diagnostic approaches, and suggests best practices for clinical management to improve patient outcomes.

Mechanisms of Secondary Angle Closure Glaucoma

The pathophysiology of SACG varies considerably, depending on the root cause. Unlike primary angle closure glaucoma (PACG), where the mechanism is generally related to anatomical predispositions such as a shallow anterior chamber, SACG is often linked to exogenous factors like posterior segment neovascularization, uveitis, or trauma. One of the more common causes of SACG is neovascular glaucoma (NVG), which can follow ischemic retinal diseases such as diabetic retinopathy or retinal vein occlusion. Here, ischemia triggers the release of vascular endothelial growth factor (VEGF), leading to neovascularization of the iris and subsequent angle closure. Inflammatory conditions like uveitis can also cause SACG by inducing synechial closure from adhesions or from inflammatory debris obstructing the trabecular meshwork.

This variability in SACG mechanisms demands that clinicians be highly vigilant in identifying the causative factor, as management hinges on treating the underlying disease along with the elevated intraocular pressure (IOP).

Diagnostic Challenges

Diagnosing SACG can be complex due to overlapping symptoms with PACG, such as blurred vision, halos, ocular pain, and even asymptomatic presentations. Gonioscopy remains indispensable for evaluating angle structure and identifying peripheral anterior synechiae (PAS), essential in distinguishing SACG from PACG. Imaging techniques such as ultrasound biomicroscopy (UBM) and anterior segment optical coherence tomography (AS-OCT) also provide valuable insights, especially in cases where angle structures are obscured. These

diagnostic tools allow for a clear assessment of any iris or ciliary body anomalies, lens subluxation, or angle crowding, which may guide subsequent intervention strategies.

Laboratory investigations may also aid diagnosis. For instance, if uveitis is suspected, a comprehensive workup including laboratory tests for infectious or autoimmune etiologies can be useful. In cases where NVG is likely, fluorescein angiography helps to assess retinal ischemia. Differentiating SACG from PACG and identifying its etiology early on can make a pivotal difference in visual prognosis.

Treatment Approaches and Best Practices

The treatment of SACG is two-pronged: it involves addressing both the angle closure mechanism and the elevated IOP. For NVG, anti-VEGF therapy and panretinal photocoagulation are mainstays of treatment, directly addressing the neovascular drive. Topical or systemic IOP-lowering medications may also be necessary, though these alone rarely suffice. In cases of uveitic glaucoma, corticosteroids and other immunosuppressive agents play a central role in controlling inflammation, often alongside IOP-lowering medications.

Laser and surgical interventions become essential when medical therapy alone is insufficient. Peripheral iridotomy (PI) may benefit some cases where angle closure is due to pupillary block, although it is less effective for other mechanisms such as PAS or neovascularization. For these cases, trabeculectomy, glaucoma drainage devices, or cyclodestructive procedures may be required. The choice of intervention depends on several factors, including the underlying pathology, stage of disease, and patient-specific considerations such as age and systemic health. Regular follow-ups with gonioscopy and IOP monitoring are paramount to prevent progression and adjust treatment as needed.

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The Need for a Multidisciplinary Approach

Given that SACG often arises from systemic or other ocular diseases, a multidisciplinary approach is beneficial. Collaboration with specialists in fields such as endocrinology, rheumatology, and vascular medicine can aid in comprehensive management, especially in cases related to diabetes, autoimmune diseases, or vascular pathology. Educating patients about their underlying conditions and the impact of systemic disease control on ocular health is essential for adherence and for reducing recurrence risk.

Conclusion

SACG remains one of the most complex forms of glaucoma, presenting challenges that demand a high degree of clinical insight and a collaborative approach. Through targeted diagnosis and management, tailored to the specific etiology of SACG, we can optimize outcomes and minimize the risk of irreversible vision loss. Advances in diagnostic imaging and a better understanding of SACG's mechanisms provide the tools we need to make more informed decisions, ultimately benefiting our patients. As we continue to deepen our understanding of SACG, ongoing research and case studies will further refine our approach to this challenging condition, enhancing our collective ability to diagnose, treat, and prevent blindness.

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Original Article

Study of Epidemiological Profile and Clinical Outcome of Glaucoma and Changing of IOP Following Blunt Trauma

N L Islam[1], B K D Sarker[2], S J Kabir[3], M I Iqbal[4]

Abstract

Objectives: To understand the epidemiological profile and clinical outcome of glaucoma and changes of IOP after management following blunt trauma patient attending in a tertiary eye hospital.

Methods: This prospective longitudinal study was done in Department of Glaucoma, Ispahani Islamia Eye Institute and Hospital, Dhaka, Bangladesh from July 2023- June 2024. A total of 42 patients with newly diagnosed or referred with post traumatic glaucoma with fluctuating IOP were included in the study. With proper evaluation patient was diagnosed in refractory glaucoma. The main outcome of the study was the VA, IOP, gonioscopic findings, number of anti glaucoma medications, severity of pain and treatment modalities.

Results: The mean age was 38.73 ± 15.44 years with male to female ratio was 2.5:1. The most common history of trauma due to wood stick 7(16.7%) followed by badminton (Cork) 5(11.9%), rod particle 4(9.5%), fall from high 4(9.5%) and hand (slap) 4(9.5%). There were no significant change visual acuity and goniscopic findings from baseline to at 1st week, after 01 month and after 03 months. From baseline mean IOP and mean number of glaucoma medications were significant decreased at 1st week, after 01 month and after 03 months respectively. Post operative pain severity was significantly reduce after 01 months and after 03 months than baseline

Conclusion: The study concluded that wood stick, badminton (Cork), rod particle, fall from high, hand (slap) are common causes of blunt ocular trauma. There was no significant change visual acuity and goniscopic findings from baseline to after treatment. Intraocular pressure (IOP) and anti-glaucoma medications were significantly reducing after treatment.

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Introduciton

Ocular trauma is a significant ophthalmic emergency that is very common. Ocular trauma can lead to the development of secondary glaucoma and can ultimately culminate into blindness if untreated.[1] Glaucoma is defined as a multifactorial anterior optic neuropathy characterized by typical optic nerve head changes and visual field defects for which raised IOP is the key modified factor.[2]

Blunt trauma may be sustained either at work place or during sport activities or it may be a result of street assault. Blunt injuries to eyes are also encountered in the victims of domestic violence.[3] In blunt injury, when an object strikes the eye, the force of the blow transmits hydraulic forces through the aqueous. Immediately after blunt trauma IOP may elevate severely. IOP also may raise due to uveal effusion and vitreous prolapse in anterior chamber. Although the elevation of IOP after blunt trauma is transient, it is important to follow up those patients for long as the patients with angle recession may develop angle recession glaucoma later in life. Elevated IOP may treated medically in early stage. Concomitated disorders such as inflammation, hyphema, lens dislocation should also have to manage.[4,5] In patient with narrow angle and angle closure glaucoma after trauma may treated with laser peripheral iridotomy. When medical and laser treatment failed to control

IOP, surgery was indicated. Through this research we can assess patient's visual outcome, different presentation and long term follow up after blunt trauma.

Methods

This prospective longitudinal study was conducted in the Department of Glaucoma, Ispahani Islamia Eye Institute & Hospital, Farmgate, Dhaka during the period from July 2023 to June 2024. A total of 42 newly diagnosed or referred with post-traumatic glaucoma and patients with fluctuating IOP were included in the study. Patients with raised intra-ocular pressure or evidence of glaucoma above 16 years of age following blunt trauma were enrolled in the study. Uveitis, post-surgical patient and patient with long-term topical steroid use were excluded from the study. After thorough explanation of the procedure, informed consent was obtained from all patients. All patients who undergone a comprehensive ophthalmic examination including visual acuity, applanation tonometry, fundus examination and B-scan ultrasound (if needed) and Humphrey visual field analysis. All those patients are managed medically and surgically (if medical Tx was failed). This patient should be followed up after 1st week then after 1st month then after 3rd month. Statistical analyses were carried out by using the Statistical Package for Social Sciences version 23.0 for Windows. A paired t-test was used for continuous variables and Chi-square test was used for categorical variables. P value<0.05 was considered as statistically significant.

Results

Out of 42 patients, the mean age was 38.73±15.44 years with male and female ratio was 2.5:1 and more than half (54.8%) of patients were affected in the right eye (Table-1). The most common history of trauma due to wood stick 7(16.7%) followed by badminton (Cork) 5(11.9%), rod particle 4(9.5%), fall from high 4(9.5%), hand (slap) 4(9.5%) (Table-2). The majority of 23(54.8%) patients were managed medical treatment (Figure-1). There was no significant change visual acuity from baseline to at 1st week, after 01 month and after 03 months (Table-3). From baseline mean IOP was significant decreased at 1st week, after 01 month and after 03 months (20.12±1.14, 14.84±4.95 and 12.94±3.14 mmHg) respectively. From baseline mean number of glaucoma medications was significant decreased at 1st week, after 01 month, and after 03 months (2.30±0.75, 1.85±0.67, and 1.80±0.77) respectively (Table-4). There was no significant change goniscopic findings from baseline to at 1st week, after 01 month and after 03 months (Table-5). Post operative pain severity was significantly reduce after 01 months and after 03 months than baseline (Table-6).

Table 1 : Socio-demographic characteristics of the study patients (n=42)

Variables	Frequency	Percentage (%)
Age group (years)	15	35.7
31-40	13	31.0
41-50	4	9.5
51-60	5	11.9
>60	5	11.9
Mean±SD	38.73±15.44	
Gender		
Male	30	71.4
Female	12	28.6
Occupational status		
Service	11	26.2
Housewife	11	26.2
Student	9	21.4
Farmer	4	9.5
Business	3	7.1
Driver	2	4.8
Water supply	1	2.4
Garments workers	1	2.4
Laterality of eye		
Right eye	23	54.8
Left eye	19	45.2

Table 2: Distribution of the study patients by history of trauma

History of trauma	Frequency	Percentage (%)
Wood stick	7	16.7
Badminton (Cork)	5	11.9
Rod particle	4	9.5
Fall from high	4	9.5
Hand (slap)	4	9.5
Ball	3	7.1
Bamboo stick	3	7.1
Boxing	2	4.8
Physical assault	2	4.8
Pen	1	2.4
Bomb	1	2.4
Stone	1	2.4
Vegetative	1	2.4
Falling plastic object	1	2.4
Toy	1	2.4
Falling mango	1	2.4
Falling coconut	1	2.4

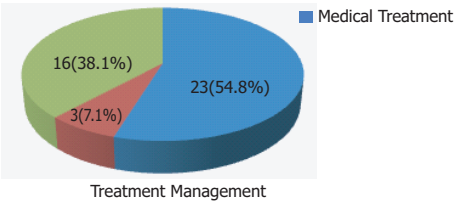

Figure 1: Distribution of the study patients by treatment management

Table 3 : Changes of visual acuity from baseline to study period

Visual acuity	at baseline (n=42)		at 1st week (n=42)		after 1 month #(n=37)		after 3 month #(n=34)	
	n	%	n	%	n	%	n	%
6/60	3	7.1	8	19.0	10	27.0	5	14.7
FC-CF	1	2.4	0	0.0	0	0.0	0	0.0
PLPR	2	4.8	4	9.5	4	10.8	4	11.8
6/12	1	2.4	3	7.1	1	2.7	0	0.0
6/6	4	9.5	2	4.8	3	8.1	3	8.8
FC 2/4	1	2.4	0	0.0	0	0.0	0	0.0
FC 1/4	2	4.8	2	4.8	0	0.0	0	0.0
2/60	2	4.8	0	0.0	2	5.4	2	5.9
FC 3	1	2.4	2	4.8	1	2.7	1	2.9
6/36	5	11.9	3	7.1	2	5.4	4	11.8
6/24	3	7.1	4	9.5	4	10.8	5	14.7
6/18	1	2.4	1	2.4	2	5.4	3	8.8
HM PR	7	16.7	5	11.9	3	8.1	2	5.9
FC ½	2	4.8	2	4.8	1	2.7	0	0.0
3/60	4	9.5	6	14.3	3	8.1	1	2.9
FC 1	2	4.8	0	0.0	0	0.0	1	2.9
FC 2	0	0.0	0	0.0	1	2.7	1	2.9
1.5/60	1	2.4	0	0.0	0	0.0	0	0.0
6/9	0	0.0	0	0.0	0	0.0	2	5.9
*P value			0.634^{ns}		0.420^{ns}		0.443^{ns}	

After 01 months 5 patients and after 03 months 8 patients were dropout due to not continue follow up.

*P value compared between at baseline vs at 1st week, at baseline vs after 01 month and at baseline vs after 03 months

ns=not significant; P value reached from Chi square test

Table 4 : Changes of IOP from baseline to study period

	at baseline (n=42)		at 1st week (n=42)		after 1 month #(n=37)		after 3 month #(n=34)	
	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD
IOP (mmHg)	29.67	±9.10	20.12	±1.14	14.84	±4.95	12.94	±3.14
*P value	-		0.001s		0.001s		0.001s	
Number of anti glaucoma medications	2.76	±0.62	2.30	±0.75	1.85	±0.67	1.80	±0.77
*P value	-		0.003s		0.001s		0.007s	

After 01 months 5 patients and after 03 months 8 patients were dropout due to not continue follow up.

*P value compared between at baseline vs at 1st week, at baseline vs after 01 month and at baseline vs after 03 months

s=significant; P value reached from paired t-test

Table 5: Goniscopic findings from baseline to study period

Goniscopic findings	at baseline (n=42)		at 1st week (n=42)		after 1 month #(n=37)		after 3 month #(n=34)	
	n	%	n	%	n	%	n	%
Angle recession	6	14.3	5	11.9	4	10.8	4	11.8
Open angle	27	64.3	30	71.4	28	75.7	25	73.5
Closed angle	9	21.4	7	16.7	5	13.5	5	14.7
*P value	-		0.779^{ns}		0.535^{ns}		0.675^{ns}	

ns=not significant

Table 6 : Pain severity from baseline to study period

Pain Severity	at baseline (n=42)		at 1st week (n=42)		after 1 month #(n=37)		after 3 month #(n=34)	
	n	%	n	%	n	%	n	%
Mild	1	2.4	5	11.9	1	2.7	0	0.0
Moderate	3	7.1	2	4.8	0	0.0	0	0.0
Severe	5	11.9	1	2.4	0	0.0	0	0.0
No pain	33	78.6	34	81.0	36	97.3	34	100.0
*P value	-		0.138^{ns}		0.049^{s}		0.040^{s}	

s=significant; ns=not significant

Discussion

In the present study most of the patients were belonged to age group 18-30 years 15(35.7%) with mean age was 38.73±15.44 years. Male patients were predominant 30(71.4%) with male to female ratio was 2.5:1. Almost similar studies conducted Maurya et al.[6]; Maurya et al.[7]; Bhagat et al.[8] and Stephan et al.[9]

In this study most common history of trauma due to wood stick 7(16.7%) followed by badminton (Cork) 5(11.9%), rod particle 4(9.5%), fall from high 4(9.5%), hand (slap) 4(9.5%), ball 3(7.1%), bamboo stick 3(7.1%), boxing 2(4.8%) and physical assault 2(4.8%). Maurya et al.[7] obtained that the most common injury was nonoccupational (82.3%) including sports related (23.9%) and road traffic accident (23.6%). Stephan et al.[9] described the most common mode of injury was by stick which constituted about 37.33% followed by tennis ball which constituted about 22.67%. The above mentioned studies finding were almost similar to this study.

In the present study t majority 23(54.8%) patients were managed medical treatment, 3(7.1%) patients required surgery and 16(38.1%) patients required both medical and surgical treatment. Syal et al.[10] documented that the patients were either managed medically or surgically. Out of 200 patients, 42% of the patients required only medical management, while 58% required surgical management.

In this current study there was no significant change visual acuity from baseline to at 1st week, after 01 month and after 03 months. Maurya et al.[6] reported that at the time of initial presentation vision was good (VA > 6/12) in 80 (33.2%) eyes, visual impairment (VA 6/18-6/60) was observed in 69 (28.6%) eyes and blindness (< 6/60 - NOPL) in 86 (35.7%) eyes while visual acuity could not be assessed in 6 (2.5%) eye. After 6 months, vision was good in 138 eyes (57.3%), impaired vision in 48 (19.9%) eyes and blindness in 51 eyes (21.2%). In a study by Gadia et al.[11] fifty per cent of the patients had vision <6/60

In this study from baseline mean IOP was significant decreased at 1st week, after 01 month and after 03 months (20.12±1.14, 14.84±4.95 and 12.94±3.14 mmHg) respectively. In a study done by Maiya[12] reported that the IOP at presentation ranged from 26 to 60 mm Hg with a mean (SD) of 36 (±9.67) mm Hg. At the final follow up, the IOP ranged between 11 and 18 mm Hg with a mean (SD) of 14.63 (2.73) mm Hg. In a retrospective study by Ellong et al.[13] of 1343 files of patients with late post-traumatic glaucoma, the intraocular pressure ranged between 22 and 66 mmHg at the first examination and between 12 and 29 mmHg at the last examination.

In the present study from baseline mean number of glaucoma medications was significant decreased at 1st week, after 01 month and after 03 months (2.30±0.75, 1.85±0.67 and 1.80±0.77) respectively. Sihota et al.[14] described that the mean number of topical medications at the start of therapy in the traumatic glaucoma group was 2.5 (1.2) and was reduced to 1.2 (1.0) at 3 months and 1.1 (0.2) at 12 months. Bhagat et al.[8] also found 20 (26.66%) patients had taken topical antiglaucoma medication and 11 (14.66%) patients had taken topical as well as oral treatment for glaucoma before they presented to us, that are support with my study.

In this study there were no significant change goniscopic findings from baseline to at 1st week, after 01 month and after 03 months. In a study by Maurya et al.[6] demonstrated that open globe injury was more common in the left eye (n = 85, 38.46%) and close globe injury was more common in the right eye (42.79%). Bhagat et al.[8] revealed that gonioscopy was done in 38 patients, 26 (68.42%) had open angles on gonioscopy and 5 patients had closed angles. 17 (44.73%) had angle recession, and 4 patients of these 17 had angle recession 180^0.

Conclusions

The study concluded that wood stick, badminton (Cork), rod particle, fall from high, hand (slap), ball and bamboo stick related injuries are common causes of blunt ocular trauma. Young adult males are more vulnerable and most of the

patients required medical treatment. There was no significant change visual acuity and goniscopic findings from baseline to after treatment. Intraocular pressure (IOP) and anti-glaucoma medications were significantly reducing after treatment. Mass health education and awareness about risk of ocular trauma, morbidity caused by delayed presentation, and need to adopt safety equipment such as goggles, visors, or preventive strategies should be focused, especially during travel, playground, and at workplace.

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Original Article

Types of secondary glaucoma & their management at BIRDEM General Hospital & Bangladesh Eye Hospital & Institute

S Begum[1], S N Hassan[2], S M Noman[3], M K Goswami[4], M N Islam[5]

Purpose : To determine the causes of various types of secondary glaucoma and their management at BIRDEM General Hospital & Bangladesh Eye Hospital & Institute.

Introduction: While the prevalence of morbidity and visual impairment due to primary open angle and angle-closure glaucoma have been well established both in the developing & developed country, the issue of blindness from secondary glaucoma has received little attention from most investigators. This study aims to identify the causes of secondary glaucoma & their appropriate (both ocular & systemic) management and reduce blindness from raised IOP.

Materials & Method: A prospective longitudinal study was done from January 2021 to December 2023 at Ophthalmology Department of BIRDEM General Hospital, Shahbag & Bangladesh Eye Hospital & Institute, Dhanmondi, Dhaka. This study was approved by the Institutional Review Board of BIRDEM General Hospital and Bangladesh Eye Hospital & Institute and adhered to the ethical standards of the Declaration of Helsinki. Total 200 eyes of 165 patients were studied here. Written informed consent of all patients were taken. Complete history regarding ocular & systemic diseases and history of drug was taken. Ocular examination included BCVA and slit lamp examinations. IOP was measured with GAT, gonioscopy & fundoscopy were done for proper evaluation & to find out the exact cause of glaucoma. Investigations like pachymetry (measurement of central corneal thickness), colour fundus photography (for documentation), OCT for glaucoma (ONH+RNFL) & HVFA (24-2,10-2) were done to see the severity of the disease. Underlying causes were evaluated for appropriate management & treatment was started to achieve the target pressure.

Result: Among the 200 eyes of 165 patients the number of male patients (66.7%) was more than the female patients (33.3%). Most common presenting age group was between 51-60 years forming 30%. Neovascular glaucoma was most common (30 %) among all secondary glaucoma. Other secondary glaucoma include post vitrectomized glaucoma (21%), uveitic glaucoma (10%), pseudophakic glaucoma(05%),lens induced glaucoma(10%), traumatic glaucoma (03%), drug induced glaucoma(09%), exfoliation glaucoma(4.0%), pigmentary glaucoma(5.0%) and iridocorneal endothelial syndrome(0.5%).Conservative medical treatment, trabeculectomy with MMC, combined trabeculectomy with phacoemulsification, phacoemulsification with synechialysis, phacoemulsification & Ahmed glaucoma valve surgery were done to control intraocular pressure. YAG laser iridotomies were also done where required. Some cases with painful blind eyes needed cyclocryopexy. In case of NVG using anti

–VEGF & PRP LASER in time with anti -glaucoma medications played a vital role to achieve target IOP. Control of intraocular pressure was quite satisfactory after taking care of the underlying causes & management of raised IOP.

Conclusion: Causes of secondary glaucoma are diverse. In our study neovascular glaucoma is the most common followed by glaucoma after vitreo-retinal surgery. Though secondary glaucoma represents a smaller percentage than the primary glaucoma, it causes significant ocular morbidity and visual impairment if present lately. It is also challenging to manage. Early identification of the primary ocular and systemic diseases that predispose to the secondary glaucoma would play a significant role in managing it and limiting the burden of blindness.

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Introduction

Glaucoma is the leading cause of global irreversible blindness. The global prevalence of glaucoma for population aged 40-80 years is 3.54%. The prevalence of POAG is highest in Africa (4.20%) and the prevalence of PACG is highest in Asia (1.09%). In 2013, the number of people (aged 40-80 years) with glaucoma worldwide was estimated to be 64.3 million,

increasing to 76.0 million in 2020 and 111.8 million in 2040(1). The prevalence of glaucoma in Bangladesh is 3.2% in 35-year-old individuals with older men most at risk. The majority (78%) had primary open-angle glaucoma (POAG), while angle closure was seen in 16%. Of the POAG, 83% (n=251) were normal-tension glaucoma (NTG) (2).

Information on secondary glaucoma in our country is limited & the causes leading to glaucoma are seldom identified. Few studies have described secondary glaucoma as a separate entity but it has been estimated that 6 million people in the world have secondary glaucoma as compared to 67 million who suffer from primary glaucoma. In India, secondary glaucoma represents 6% of total new cases seen annually (3). The causes of secondary glaucoma are diverse but commonly seen entities are inflammatory, lens induced, neovascular and traumatic (4). In our country there is no survey about secondary glaucoma. Surgery is the mainstay for treating secondary glaucoma due to the complications associated with secondary factors (5)

Materials & method

A prospective longitudinal study was done from January 2021 to Dec 2023 at Ophthalmology Department of BIRDEM General Hospital, Shahbag and Bangladesh eye hospital & institute, Dhanmondi, Dhaka. This study was approved by the institutional review board committee of BIRDEM general hospital & Bangladesh eye hospital & institute and adhered to the ethical standards of the Declaration of Helsinki. This study was explained and provided written informed consent were taken from all the patients. Information regarding visual acuity (BCVA), intraocular pressure (with GAT), slit lamp examination (corneal clarity, corneal oedema, PACD, status of the pupil ,anterior and posterior synechia, iris neovascularization and iris atrophy, lens status), gonioscopic findings (excess pigmentation of PTM, NVA, angle recession, iridodialysis, silicon oil blocking the TM, synechial angle closure) and funduscopic findings (condition of optic disc, cup to disc ratio, retinal haemorrhages and neovascularization) were clearly noted. Investigations like pachymetry to find out the actual IOP, colour fundus photography (for documentation), OCT for glaucoma (ONH+RNFL) & HVFA (24-2,10-2) were done to see the severity of disease. FFA & OCT for macula were performed where required (patients with diabetic retinopathy & retinal vascular occlusive diseases). Secondary glaucoma was properly considered to represent eyes in which a second form of ocular pathology caused IOP elevation, leading to optic nerve damage. The secondary ocular pathological process included one of the following: trauma, lens-related issues, uveitis, neovascularization, intraocular surgery and others. Patients that documented a history of primary glaucoma were excluded from the analysis.

Management included treatment of underlying causes (uveitis, diabetic retinopathy, retinal vascular occlusive diseases, removal of silicon oil) & specific treatment (topical & systemic glaucoma medications & YAG Laser iridotomy) to reduce IOP. Surgery was performed in cases of primary disease requiring that (lens induced glaucoma), if progression of glaucomatous damage was demonstrated, or if the IOP was at a level that would cause additional damage.

Result

During the 3 years study period total 200 eyes of 165 patients were studied here. The number of male patients (66.7%) was more than the female patients (33.3%). Most common presenting age group was between 51-60 years forming 31.5% followed by 61-70 years (23%) & 71-80 years (16.36%). The most common type of secondary glaucoma was neovascular glaucoma (30%), followed by post PPV (21%), uveitic glaucoma (10%), lens induced glaucoma (10%), drug induced glaucoma (9%), pigmentary glaucoma (5%), pseudophakic glaucoma (05%), traumatic (4.5%), exfoliation glaucoma (4%), ICE syndrome (0.5%). Traumatic glaucoma was either associated with hyphema or angle recession or traumatic cataract. Among 10 pseudophakic patients 3.5% was with posterior chamber IOL implants

and 1.5% was with anterior chamber IOL implants. Among the lens induced glaucoma, phacomorphic glaucoma was 5.5% & phacolytic glaucoma was 4.5%. Among all neovascular glaucoma 19% was due to advance diabetic eye disease & 11% was due to vascular occlusive diseases (CRVO, BRVO & CRAO). Silicon oil induced glaucoma was 15% (30 eyes) among all post- vitrectomized eye, other causes of glaucoma after vitrectomy was 6%. These incudes post vitrectomy with intravitreal injection of sulphur hexafluoride (SF6), perfluropropane (C3F8), fluid air exchange & scleral buckling procedure. Underlying pathology in cases of vitrectomized eyes were diabetic retinopathy, myopia, retinal vein occlusive diseases, pseudophakia, ocular injury & aphakia. Steroid induced secondary open angle glaucoma was 6% & bilateral angle closure glaucoma after using topiramate, selective serotonin reuptake inhibitors such as fluoxetine was 3% among all drug induced glaucoma.

Table 1 : Gender distribution

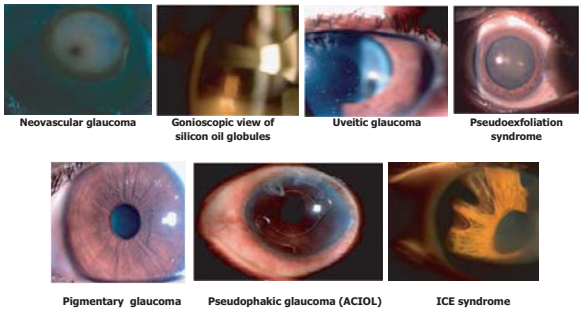
No. of patients	Male	Female
165	110(66.66%)	55(33.33%)

Table 2 : Age distribution

Age group(years)	Patients n (%)
18-30	10 (06%)
31-40	16 (9.7%)
41-50	22 (13.3%)
51-60	52 (31.5%)
61-70	38 (23%)
70-80	27(16.36%)

Table 3 : Types of secondary glaucoma and their percentage

Types of secondary glaucoma	Number of eyes & percentage
Neovascular glaucoma	60(30%)
Post PPV glaucoma	42(21%)
Uveitic glaucoma	20(10%)
Pseudophakic glaucoma	10(05%)
Lens induced glaucoma	
Phacomorphic glaucoma	11(5.5%)
Phacolytic glaucoma	09(4.5%)
Traumatic glaucoma	09(4.5%)
Drug induced glaucoma	18(09%)
Exfoliation glaucoma	08(4.0%)
Pigmentary glaucoma	10(5.0%)
ICE syndrome	03(00.5%)



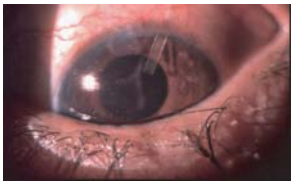
Management of secondary glaucoma: All of the patients were managed conservatively at the initial stages. Among 200 eyes 42% maintained good controlled of IOP with conservative way. Rest 58% needed surgical interventions. The conservative treatment included topical anti glaucoma drugs like topical beta-blockers, alpha adrenergic, prostaglandin analogues, RHO - kinase inhibitors, carbonic anhydrase inhibitors, as well as systemic like oral CAI & intravenous hyperosmotic agents(mannitol). Inflammatory glaucoma was treated with steroids (topial, subtenon's & systemic) and topical cycloplegic drug. Sometimes immunosuppressive agents (azathioprine, methotrexate & mycophenolate, infliximab, cyclophosphamide) were needed to reduced intraocular inflammation suggested by uvea specialist. YAG LASER iridotomy were tried in secondary angle closures due to long standing uveitis. 30% of uveitic glaucoma were managed conservatively, rest 70% needed surgical intervention. Trabeculectomy with antifibrotic agents (Mitomycimn-C), combined surgery (phacoemulsification + PCIOL with trabeculectomy with MMC) & Ahmed glaucoma valve (AGV) surgery were performed where indicated. Cataract surgery with intraocular lens implants was done in lens induced glaucoma along with trabeculectomy where required. Some of them needed topical anti-glaucoma medications to maintain target IOP.30% cases of NVG were managed conservatively. Using intravitreal anti- VEGF & PRP laser in time with

anti -glaucoma medications, topical steroid & cycloplegic drug play a vital role to achieve target IOP. Control of intraocular pressure was quite satisfactory after well controlled of underlying causes (PDR, vascular occlusive diseases) & appropriate management of raised IOP. Surgical interventions were needed in 70% of NVG cases. After regression of new vessels in iris & reduction of inflammation trabeculectomy with MMC & Ahmed glaucoma valve surgery were performed in these cases. Cyclocryopexy were done in few cases of non-seeing painful eye who presented to us in late stage. 71% cases of raised IOP after vitrectomy, were managed with anti- glaucoma medications & removal of silicon oil in time. 29% cases were managed with trabeculectomy with MMC & Ahmed glaucoma valve surgery. Those who underwent trabeculectomy few cases needed further intervention (re-trabeculectomy with MMC, bleb revision with silicon oil wash). Patients who underwent Ahmed glaucoma valve implantation maintain a good IOP & only few cases required topical single combination AGM. In post vitrectomized aphakic eye using silicon oil tamponade inferior peripheral iridectomy was performed to prevent pupillary block glaucoma.56% of drug induced glaucoma was managed conservatively & rest 44% was managed with surgical intervention. Patients taking steroid eye drop for vernal keratoconjunctivitis (VKC) & peripheral ulcerative keratitis, post- operative use (in myopic patients), intravitreal injection of dexamethasone (Ozurdex) for diabetic macular oedema & systemic steroid for pan hypopituitarism are responsible for raised IOP. Cessation of topical steroid, starting anti-glaucoma medications & regular follow up reduce IOP in some cases. As steroid is a lifesaving drug for the patient with pan hypopituitarism, we couldn't discontinue this life saving drug in this particular situation. So, we went for trabeculectomy operation as a permanent solution. Those who were using topical steroid for long time & advanced stage glaucoma we went for trabeculectomy with MMC. Combined phacoemulsification with trabeculectomy was performed in some cases who presented with cataract and advanced glaucomatous damage. Drug induced angle closure glaucoma was bilateral presentation, history reveals using drugs for headache (tab. topiramate) or antidepressant i.e. selective serotonin reuptake inhibitor (SSRI) i.e. tab. fluoxetine. The mechanism of angle closure glaucoma is different in these two situations.1stwe stopped the drug that cause raised IOP in both cases. Addition of cycloplegic drop, topical steroid, AGMs & keeping close observation help to reduce IOP in case of topiramate induced angle closure glaucoma. Patient who was taking tablet fluoxetine presented to us with cataract & pupillary block glaucoma, cessation of drug & adding AGMs(topical+systemic) & latter on Phacoemulsification with PCIOL help to reduce IOP & save the patient's eye. 44% of traumatic glaucoma was managed with conservative way (AGMs+Cycloplegic drop + topical steroid). Paracentesis and irrigation were performed in patients with anterior chamber hyphema. Rest 56% needed trabeculectomy/Combined surgery. Among all exfoliation glaucoma 25 % was treated with AGMs.75% was managed by trabeculectomy with phacoemulsification +PCIOL implantation. 40% of pigmentary glaucoma was managed by topical glaucoma medications, rest 60% needed interventions (trabeculectomy with MMC/combined surgery). Patient suffering from ICE syndrome 33% was managed by topical AGM & hypertonic saline, 66% needed surgical interventions (trabeculectomy with MMC & AGV implantation).

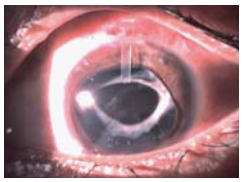
Table 4 : Types of secondary glaucoma & their management

Types of glaucoma	No. of eyes	Management
Neovascular glaucoma	60	Conservative management (Anti-VEGF+PRP Laser)-20(30%) Surgical management-40(70%) Trabeculectomy with MMC, AGV implantation
Post PPV glaucoma	42	Conservative treatment-30(71.42%) Surgical management-12(28.57%) Removal of silicon oil, trabeculectomy with MMC, AGV implantation
Uveitic glaucoma	20	Conservative treatment-06(30%) Surgical treatment-14(70%) Trabeculectomy with MMC, combined surgery, AGV
Pseudophakic glaucoma	10	Conservative treatment-07(70%) Surgical management-03(30%)-trabeculectomy with MMC
Lens induced glaucoma-		Surgical management-
Phacomorphic glaucoma	11	i)Phacoemulsification with PCIOL implantation-13
Phacolytic glaucoma	09	ii) Phacoemulsification with PCIOL implantation + AGM-05
		iii) Phacoemulsification with trabeculectomy-02
Traumatic glaucoma	09	Conservative treatment-04(44.44%) Surgical management-05(55.55%)
Drug induced glaucoma	18	Conservative treatment-10(55.5%) Surgical management-08(44.4%)
Exfoliation glaucoma	08	Conservative management-02(25%) Surgical management-06(75%)
Pigmentary glaucoma	10	Conservative management-04(40%) Surgical management-06(60%)
ICE syndrome	03	Conservative management-01(33.33%) Surgical management-02(66.66%)-Trabeculectomy with MMC

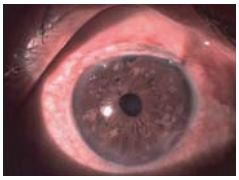
Total No. of eyes	Conservative management	Surgical management
200	84 (42%)	116(58%)



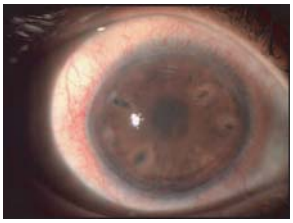
AGV in NVG



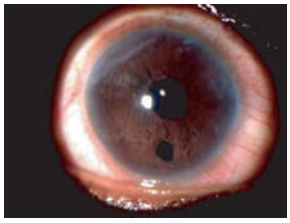
AGV in Post Vitrectomized eye



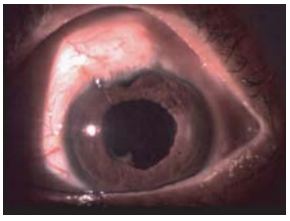
Trabeculectomy in uveitic glaucoma



Multiple PI in uveitic glaucoma



Inferior PI in aphakic Silicon oil filled eye



Combined surgery in pseudoexfoliation glaucoma

Discussion: Glaucoma is a leading cause of irreversible blindness worldwide. Secondary glaucoma is caused by ocular disorders or systemic factors that induce aqueous outflow impairment, resulting in elevated IOP, which then results in glaucomatous optic neuropathy and eventual blindness (6). Irreversible vision loss is more common in underdeveloped and developing countries. In contrast to primary glaucoma, secondary glaucoma can typically be prevented, which is important for the prevention and treatment of blindness, particularly in developing countries and regions. The number of male patients was higher than that of female patients (66.33% vs. 33.33%) in our study, which was similar to previously published results (7,8). The most common presenting age group was between 51-60 years (31.5%) followed by 61-70 years (23%) which is similar to other study (9). Different causes of secondary glaucoma have been observed in different countries and regions. Neovascularization was the most prevalent cause of secondary glaucoma in our study population, accounting for 30% of cases. Previous studies have reported various causes of secondary glaucoma (4,6,8). Similar results were observed in Chinese populations by the Zhongshan Ophthalmic Center (ZOC) in southern China (4). In the ZOC data, trauma and vascular disease were the first and second most common causes of secondary glaucoma, respectively. The ZOC also showed that the proportion of trauma-associated secondary glaucoma had significantly decreased and that the rate of vascular disease-related secondary glaucoma had increased from 2015 to 2019.At BIRDEM general hospital mostly we deal with diabetic patients & most of them presented with advance diabetic eye diseases & these patients also have others comorbidity like hypertension, IHD, hyperlipidaemia, H/O stroke which are responsible for vascular occlusive diseases (CRVO,BRVO,CRAO) those are ultimately responsible for neovascular glaucoma. Population aging and a high incidence of diabetes among adults are associated with neovascular glaucoma in China (10). Similar results were found in North India, where the leading cause of secondary glaucoma was neovascular glaucoma (7). According to our study post PPV glaucoma is the second most common secondary glaucoma (21%). Vitrectomy-related glaucoma also comprises the majority of secondary glaucoma cases in New Delhi, India (11). Similarly, with the increased rate of posterior segment surgery-post vitrectomy glaucoma has increased substantially in the last 2 years in Henan, Central China (10) Conversely, many studies have reported a higher prevalence of syndrome-related secondary glaucoma, which might be due to the higher prevalence of pseudoexfoliation in these countries. In Japan, exfoliating glaucoma caused most cases of secondary glaucoma (13). Our study showed 4% cases suffering from exfoliation glaucoma. Lens-induced issues were the leading cause of secondary glaucoma in neighbouring South Asian countries (14). In our centre, 10% of cases have lens induced glaucoma among all secondary glaucoma. Another study showed that trauma-related glaucoma was the most common type of secondary paediatric glaucoma (15). Study conducted at BIRDEM general hospital showed that traumatic glaucoma is 4.5%. In our study, secondary glaucoma associated with chronic uveitis was 10%. While in another study, uveitic glaucoma was 11.3%(7) & study conducted in Boston USA 9.6% patients had secondary glaucoma due to uveitis and chronic anterior uveitis being the most common entity (16).

Secondary glaucoma occurs with acquired ocular diseases (intraocular inflammation and retinal vascular diseases, pigment dispersion, pseudoexfoliation, blunt trauma, ocular surgery (especially pars plana vitrectomy, corneal grafting and cataract surgery) and drug induced. Management of secondary glaucoma is different from that of primary open angle or angle closure glaucoma and must be tailored for the individual patient. Detection of underlying causes & their early intervention help to reduce development of secondary glaucoma. Glaucoma valve implantation is an effective approach for decreasing and controlling IOP over time (17,18). American Glaucoma Society group

showed that glaucoma valve implantation is being selected as an alternative to trabeculectomy with increasing frequency (19,20). Glaucoma valve implantation has been widely used in refractory glaucoma cases, including neovascular, trauma, and vitrectomy-related secondary glaucoma cases, in many different countries and regions (21,22,23). Here, the most common initial glaucoma intervention for each eye was trabeculectomy. Cataract extraction with PCIOL implantation & combined surgery are increasingly being used at our centre as an effective treatment for glaucoma. Trabeculectomy remains the preferred surgical treatment for secondary glaucoma in rural or undeveloped regions in Bangladesh. The glaucoma valve implantation in most district or hospitals in our country is less due to high cost. At our centre Ahmed glaucoma valve implantation were performed in NVG & post vitrectomized eyes. Trabeculectomy remains the classic surgical method for treating secondary glaucoma in developing or undeveloped countries or districts (4,9). In northern India, trans-scleral cyclophotocoagulation has been extensively adopted as a secondary glaucoma treatment (7). Due to unavailability of this machine, we treated the cases of painful, non-seeing eyes with cyclocryopexy at our centre.

Limitations: This study had several limitations. First, no standardized management or widely established guidelines exist for secondary glaucoma. Patients' comorbidities (ocular and systemic) may have affected the results. The present study was a hospital-based analysis of patients who underwent surgical treatment for secondary glaucoma. Thus, population-based epidemiological studies are needed in the future.

Conclusion: Secondary glaucoma is an important cause of visual morbidity & has increased exponentially in recent times. This is due to the increase in lifestyle related diseases like DM, HTN, IHD causing increase in the prevalence of neovascular glaucoma (NVG). At BIRDEM General Hospital most of our patients are diabetic having with multiple comorbidities (i.e. HTN, IHD, hyperlipidaemia, H/O stroke,) which is also responsible for NVG. On the other hand, complex eye surgery like vitreo-retinal surgery & using different tamponades are big issue for secondary glaucoma in both centres. The other causes of secondary glaucoma are inflammatory, post trauma, lens induced, pigmentary & drug induced glaucoma. As BIRDEM is a tertiary hospital & Bangladesh Eye Hospital is a super specialized centre, the patients with secondary glaucoma are referred with different complaints (headache, loss of vision, redness, ocular pain) from different subspecialty. The management of this complex glaucoma is difficult as they are mostly intractable & do not respond to conventional anti-glaucoma medications. Many patients who could not be managed with multiple anti-glaucoma medications, needed surgical interventions along with vigilant control of underlying pathology. Early detection and good management of conditions associated with the potential for retinal ischaemia and neovascularisation such as good management of DM, hypertension, hyperlipidaemia & IHD helped to prevent NVG. Proper & timely referral system, as well as management of underlying

pathology and early interventions help to prevent blindness due to secondary glaucoma.

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A comparative study between phacoemulsification and laser peripheral Iridotomy in initial management of acute primary angle closure (PAC)

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Abstract:

Purpose: To compare the 6 months efficacy of phacoemulsification and intraocular lens implant (phaco/IOL) with laser peripheral iridotomy (LPI) in the early management of acute primary angle closure (PAC) and co-existing cataract.

Methods: Subjects were randomized to receive either LPI or phaco/IOL in the affected eye within 1 weekof presentation and were examined at fixed intervals over 3 months. Patients underwent a standardized examination that included Goldmannapplanation tonometry, gonioscopy, and CECC measurements. In this prospective, randomized, comparative study, 30 patients of acute primary angle closure glaucoma were selected from glaucoma department. Patients were allocated for management either with phacoemulcification (15 eyes) or with laser peripheral iridotomy (15 eyes).

Results: There were 15 patients randomized to LPI and 15 patients of phaco/IOL. The avarage age of the patients were in group-1 (LPI) 60.47±4.47 and in group-2 (phaco/IOL) was 57.67±4.43 The mean IOP after initial medical treatment was 21.53±2.80 in LPI group and 19.87±2.33 in phaco IOL group. The mean IOP after 3 months in group-1 was 14.53±4.55 and in group-2 13.40±1.30. The mean CECC after intervention in group-1 was 2569.33±33.00 and in group-2 was 2444.67±40.42. The number of antiglaucoma medication used after 3 months, after intervention was more in group-1 than group-2. Post laser complications occurred in group-1 but no detectable complications were found in group-2.The IOP reduction was significant in both groups. The rate of complete success in the phacoemulsification group was similar to that in laser peripheral iridotomy group. The management of PAC in phacoemulsification higher than laser peripheral iridotomy group. There was no significant difference in postoperative complication.

Conclusions: Performed within 1 week in patients with PAC, phaco/IOL resulted in lower rate of IOP failure at 3 months compared with LPI.

Keywords: LPI, Phacoemulsification and IOL, Acute primary angle closure.

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Introduction

Acute primary angle closure (PAC) is a potentially blinding condition and has been reported to occur with high incidence in subjects of East Asian origin.[1] The guidelines and consensus from the International Society of Geographical and Epidemiological Ophthalmology (ISGEO) not only provide principles to define narrow angles, but also postulate the natural process of this disease from primary angle-closure suspect (PACS) to primary angle closure (PAC) to PACG.[2] Risk factors of PAC include race, female gender and older age, anatomical abnormalities (shallow anterior chamber, small corneal diameter, and increased lens thickness).[3,4]

Different options are available in the management of acute PAC. Since long LPI was remaining as a mainstay in the management of acute PAC. The standard initial management of acute PAC is medical lowering of intraocular pressure (IOP) followed by laser peripheral iridotomy (LPI). This is performed to relieve pupil block, the predominant mechanism of acute PAC.

It was reported that phacoemulsification widened the iridocorneal angle, make the anterior chamber deep and attenuated the anterior positioning of the ciliary processes in eyes with acute PAC.[5] Recently, increased evidence has shown that primary lens extraction (phacoemulsification) not only reverses the

acute attack of primary angle closure and raised IOP but also achieves long-term IOP control.[6,7]

In order to compare the Efficacy of laser Iridotomy and phacoemulsification in acute primary angle closure.

Materials and Methods

This study was a prospective randomized interventional comparative study. Patients with primary angle closure glaucoma who fulfill the inclusion and exclusion criteria presenting within the selected study period of Ispahani Islamia Eye Institute and Hospital and initial management of acute primary angle closure glaucoma in the study. After enrollment, patients were randomized into 1 of the 2 groups; laser peripheral iridotomy, group-1 and phacoemulsification and intraocular lens implant, group-2. LPI was performed when the cornea was sufficiently clear after lowering the IOP .After successful LPI, topical IOP lowering medications & steroids were continued for 1 week after procedure.

All operations were performed by consultant surgeons. The panel of surgeons was formed from the group of co-investigators. Technique used was as per surgeon preferance. Phaco/IOL was carried out between 5 & 7 days of IOP control.

Data were collected in a pre-designed data collection sheet. Statistical Analysis was done using SPSS software. Categorical variables were analyzed using the chi-square test and continuous variables were analyzed using the unpaired Student's t-test to detect differences between the groups. Discrete variables were presented as percentages and analyzed using Fisher's exact test. Correlation was considered significant at p value < 0.05. The ethical principle of Helsinki declaration has been followed and informed written consent was taken from all the study patients

Results

30 consecutive patients (who consented for the study) presenting to Ispahani Islamia eye institute and hospital were enrolled for this study. None of these patients were lost to follow up after enrollment visit, so finally 30 patients completed the study.

Group-1: Laser peripheral Iridotomy.

Group-2: Phacoemulsification and intraocular lens implant.

Table 1: Comparison of demographic and clinical features between two groups

	Group-1 N=15	Group-2 N=15	P value
Age	60.47±4.41	57.67±4.43	0.088
Sex (M:F)	6:9	6:9	1.000
Status of IOP-mmHg at presentation	42.27±5.84	36.00±52.00	1.000
Status of IOP after anti glaucoma medication	21.53±2.80	19.87±2.33	0.089

Table 2: Status of IOP reduction from presentation to day 7 after antiglaucoma medication and reduction at day 7 and 3 months after intervention

	Group-1			Group-2		
	Day 7 after Medication	Day 7 after intervention	3 m after intervention	Day 7 after medication	Day 7 after intervention	3 m after intervention
Mean	20.73	24.93	27.73	22.4	26.8	28.87
Percentage	48.91	58.94	65.81	52.41	62.88	67.5

P value (7 days after medication): 0.532, P value (7 days after intervention): 0.491, P value (3 months after intervention):0.868

Table 3: Status of corneal endothelial cell count (CECC).

CECC(cell/sq.mm)	Group-1 N=15	Group-2 N=15	P value
After 7 days of drugs use,	2574.87±34.71	2480.13±38.60	0.0001
At month, 3	2569.33±33.00	2444.67±40.42	0.0001
Decrease at month,3 from initial antiglaucomal medication	5.53	35.47	0.0001

Table 4: Number of drugs used durings 7 days of medical management and over 3 months after intervention.

No. of drugs	Group-1(n-15) No. (%)		Group-2 (n-15) No. (%)		P value
Over 7 days (medical treatment),					1.000
Three	6	40	6	40	
Four	6	40	6	40	
Five	3	20	3	20	
Over 3 months (after intervention)					0.022
None	5	33.3	7	46.7	
One	4	26.7	8	53.3	
Two	6	40	0		

Table 5 : Status of post operative outcome.

Outcome	Group-1(n-15) No. (%)		Group-2 (n-15) No. (%)		P value
At day 7,					0.224
Success (angle open)	12	80	15	100	
Failure (angle close)	3	20	0		
At month, 3					0.224
Success (angle open)	12	80	15	100	
Failure (angle close)	3	20	0		

Discussion

We found that performing phaco/IOL on patients with APAC and visually significant cataract after medical lowering of IOP was superior to LPI in terms of success rates and complications over 2 years, In addition, there was no significant difference in CECC from baseline to 6 months after the procedure for either group, nor between groups.

There were 3 (of 15) failures in the LPI group, which was considerably higher than in the phaco/IOL group (0/15). However, if we compared complete success (i.e., IOP control without additional IOP-lowering medications) between the 2 groups, the superiority of phaco/IOL over LPI was less evident. Laser peripheral iridotomy in the setting of APAC is performed for 2 reasons: to prevent recurrence of APAC and to reduce the risk of chronic rise in IOP. For the first scenario, LPI is very successful, with only 1 subject in our cohort experiencing recurrence of APAC. Other studies have reported similar findings.11 In preventing long-term IOP rise, however, LPI was less successful. We found a failure rate of 20% at 6 months in our study, which is still considerable, although less than the 58.1% failure rate found in the retrospective study by Aung et al2 that spanned 5 years (Singaporean population). Even in Caucasian subjects, IOP rise occurs in a large proportion of patients who have had LPI. In a study of 63 patients who had APAC, 56% required medications or surgery after LPI within 4 years.[8] Another study of a Caucasian population reporte similar findings, with 53% requiring further IOP-lowering medications or procedures among 161 patients.[9] This pressure rise occurs despite strong evidence that LPI does result in opening the drainage angle.[10,11,12] In our study, median Shaffer score at 6 months was higher in the LPI group compared with the phaco/IOL group, although the difference was not significant. It is clear from these findings that increased reduction of IOP in the phaco/IOL group was not purely a function of the degree of angle opening.

There was a significant decrease in the amount of PAS in both groups. The finding that LPI reduces the amount of PAS is surprising because it seems unlikely that the change in pressure differential between the anterior and posterior chamber that occurs after LPI would be sufficient to overcome mechanical closure. However, this finding has been previously reported,18 although there is also some evidence to the contrary.15 The decrease in PAS in the phaco/IOL group may be because our protocol allowed for injection of viscoelastic circumferentially 360° into the anterior chamber, which may have contributed to the breaking of PAS,in much the same way as has been described for viscogoniosynaechialysis.[13,14]

There was no difference in visual acuity between

the 2 groups at 6 months. This is surprising in view of the fact that cataract was removed in 1 group and not the other. However, we showed in a previous publication on this same cohort of subjects that after medical resolution of APAC 50.4% of subjects have visual acuity of _6/12 Snellen.[15] This implies that cataract, although it undoubtedly plays a role in the etiology of APAC, may not always have a significant effect on vision. Therefore, if phaco/IOL is performed in the initial management of APAC, it should be understood by the clinician and patient that it is not necessary only for visual improvement, but also for IOP control.

Our analysis shows that phaco/IOL performed within 1 week of medically controlled APAC does result in superior IOP control at 6 months compared with LPI, the standard treatment, with few complications. Early phaco/IOL should be considered a viable option in the management of APAC, with the caveat that experienced surgeons should carry out the surgery. This latter point is important: Although we found no significant complications in the phaco/IOL group, cataract surgery does have the potential for more devastating complications (such as endophthalmitis and expulsive hemorrhage) than LPI, although our study did not detect such events because of the small number of subjects enrolled. Larger studies would be needed to resolve this issue because of the rarity of such events. Any further studies of this subject would also benefit by examining the impact of these interventions on quality of life.

Conclusion

Primary phacoemulsification plus intraocular lense implantation lowered IOP, reduced the use of antiglaucoma medication and improved symptoms in patients with acute PAC. It is safe and effective method of IOP control and can be considered a first treatment option in managing patient with PAC.

Limitation :

- Short duration of followup.
- Sample size was relatively small.
- Long term results could not be assessed.

Recommendation

- The study should be carried out in larger populations.
- Studies should involve appropriate follow up to determine long term effect, if any.
- Epidemiological studies should be carried out to define the prevalence of the problem in Bangladesh. This will help in subsequent management plans.

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Original Article

Comparison Between Efficacy and Safety of Ab Interno Vs Ab Externo Bleb Needling After Failed Trabeculectomy

Z J Ruma[1], S Rahman[2], Z Farhana[3], M S Mira[4]

Abstract

Purpose: To explore and compare statistically the efficacy and safety of ab interno Vs ab interno bleb needling after failed Trabeculectomy.

Material and method: A prospective comparative study was conducted in Glaucoma department, Vision Eye Hospital over a period from July 2022 to December 2023. Among 20 failed trabeculectomy cases, 10 cases were undergone ab externo bleb needling and 10 cases were undergone ab interno bleb needling. All patients had previously undergone Trabeculectomy and bleb function were failed with increase intraocular pressure. None of the patient had any GDD or other micro invasive glaucoma surgery.

Result : The ab interno comparative study demonstrated a mean IOP reduction of (61.34 ± 17.04) % mmHg in 3 months, compared to a mean reduction of (37.21 ± 31.83) % mmHg (40.1% decrease) in the ab externo group (p =0.049).

Success rate (bleb functioning and IOP reduction) was higher in Ab Interno bleb needling than Ab externo bleb needling but the difference was not statistically significant.

Conclusion: The ab interno approach demonstrated greater outflow resistance and less predictable bleb formation than the ab externo approach. These results have implications for long-term IOP control and success depending on time duration of intervention following bleb failure.

Keywords: IOP, ab interno, ab externo, bleb needling,trabeculectomy, failed bleb

Introduction

Glaucoma is the leading cause of irreversible visual loss worldwide and is associated with raised intraocular pressure

Trabeculectomy has long been recognized as the most effective surgical intervention for reducing intraocular pressure (IOP) in patients with glaucoma.[1–3]Creating a filtering bleb allows for the controlled outflow of aqueous humor from the eye, mitigating the damaging effects of elevated IOP. However, despite its success, sometimes trabeculectomy blebs may fail. Bleb failure refers to the loss of filtration functionality in the trabeculectomy bleb, which leads to inadequate IOP control and potential glaucoma progression. Numerous factors can contribute to bleb failure, including scarring, fibrosis, and inflammation.

The management of bleb failure poses a considerable management challenge, chiefly restoring the function of the failed filtering bleb and reopening the filtration channel's patency. Modalities to deal with this complication include digital massage, releasable sutures removal or laser suture lysis, revision of the filtering site, repeated filtration surgery, or placement of a glaucoma drainage implant. [4–9]

A failed bleb can be make functioning bleb by 2 needling procedures, Abinterno and Ab externo bleb needling approach.

The ab externo approach, although requiring conjunctival dissection, allows the surgeon to direct aqueous humor outflow posteriorly in a more predictable fashion than the ab interno approach. Subconjunctival blebs are, therefore, more likely to be diffuse with a lower risk of long-term. In ab externo procedure, Bleb needling with antimetabolites could be considered an effective and safe procedure after trabeculectomy failure.

Ab interno bleb revision using the Grover and Fellman spatula (Figure 1) offers potential benefits in improved intraocular pressure control

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and spares the conjunctiva for enhanced bleb management. The ab-interno needling approach injection is a novel technique that can be performed and offers the advantage of increasing aqueous outflow through a direct opening of the trabecular meshwork from within the anterior chamber. This technique provides effective management of failed trabeculectomy to achieve intraocular pressure lowering effect and a corresponding decrease in the risk of progressive glaucomatous optic neuropathy.

Figure 1: Grover FellmanSclerostomy Spatula

Criteria of participants

Adult patients (above 35 years of age) who presented with failed trabeculectomy blebs and medically uncontrolled IOP fulfilled the inclusion criteria were included in the study. Eligible participants were collected from Glaucoma clinic of Vision Eye Hospital over a period from July 2022 to December 2023.All patients underwent a comprehensive ophthalmologic examination, including best-corrected visual acuity (BCVA), slit-lamp biomicroscope, Goldmannapplanation tonometry, 4-mirror gonioscopy, and stereoscopic optic nerve head evaluation using the 90-diopter lens. Informed consent was obtained from all participants before their inclusion in the study.

The primary outcome measures were intraocular pressure (IOP) and the usage of IOP-lowering medications 6 month after the procedure. Secondary outcomes included the procedure's failure rate and complications. Complete success was defined as achieving an IOP between 6 to 21 mm Hg without medication, while qualified success required additional medical treatment.

To be eligible for inclusion, patients had to have a patent internal sclerostomy site visualized on gonioscopy and be systemically fit for surgery under local anesthesia. The exclusion criteria were patients unable to complete the required follow-up, those on blood thinners for coronary artery disease or stroke, patients with chronic or recurrent uveitis, anticipated need for combined ocular procedures, or those allergic to xylocaine.

The data of patients completing a minimum follow-up of 6 month were analyzed.

Surgical procedure of ab interno bleb needling:

Following local anesthesia and standard sterile eye preparation, a corneal paracentesis was created inferiorly, approximately 180 degrees away from the sclerostomy site. The anterior chamber was refilled with a dispersive viscoelastic substance injection pilocarpine 1.0% was injected to constrict the pupil. The location of the sclerostomy was confirmed using a sterile intraoperative gonioscope (Mori lens). The Grover Fellman spatula's tip was gently introduced into the bleb through the patent sclerotomy (Figure 3) and used to manipulate and revise the trabeculectomy bleb. The spatula's rounded tip was maneuvered within the bleb to break adhesions, disrupt scar tissue, and create microchannels for improved aqueous outflow. Care was taken to unroof only the posterior aspect of the scleral flap, which allows for a more low, diffuse, and posterior bleb. The dissection was continued as posteriorly as possible to create a large, diffuse, and posterior bleb. The end point of this procedure was the formation of diffuse blebs along with the shallowing of the anterior chamber. Any intraoperative hyphemia was drained using irrigation and aspiration.

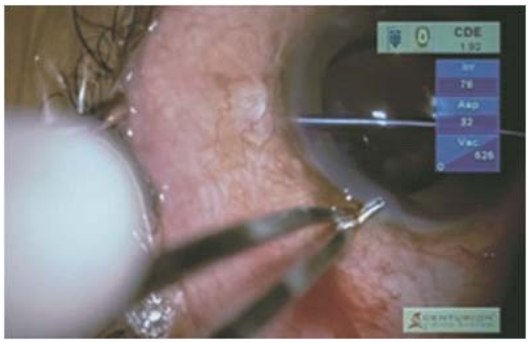

Figure 2: Ab interno needling procedure without gonioscopy lens

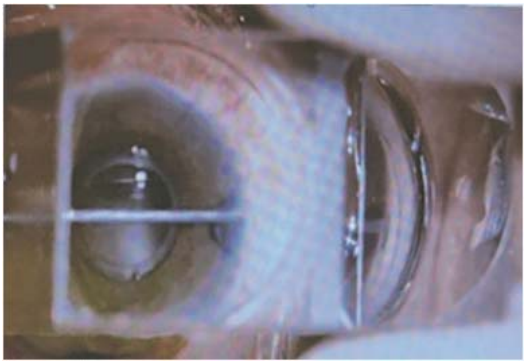

Figure 3: Ab interno approach with gonio lens (Mori Lens)

Surgical procedure of ab externo bleb needling:

At first Betadine and topical Oxibuprocain was applied. A temporal main wound with a keratome was made, either a Lewicki cannula or the irrigation and aspiration handpiece were used to create an infusion into the eye. A bent 25-gauge needle on a tuberculin (TB) syringe was used to enter the conjunctiva superotemporally to the scarred area and use a side-to-side sweeping motion to lyse the scar tissue using the edge of the bevel until visualizing a raised bleb. Having the constant infusion enables the surgeon to see the bleb rise and confirms successful outflow. Injection of 5-fluorouracil (5FU) or mitomycin into the subconjunctiva is helpful to prevent further encapsulation and scar tissue formation.

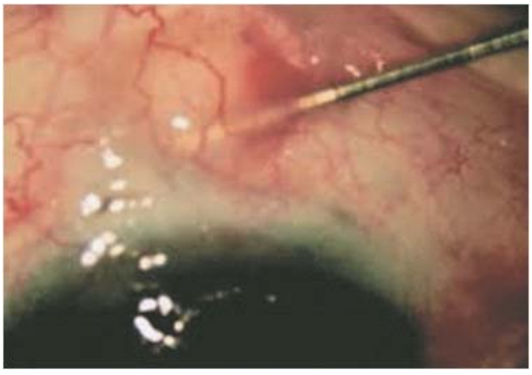

Figure 4: Ab externo bleb revision

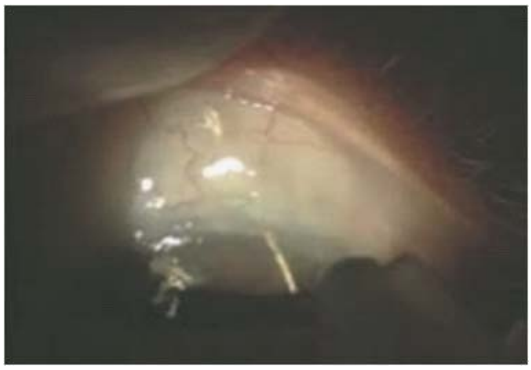

Figure 5: Ab interno bleb revision

Postoperative Care for both bleb needling technique

Follow-up appointments were scheduled to monitor the patient's progress, evaluate IOP control, and assess the success of the bleb revision. The patients were prescribed topical Steroid drops (frequency depending upon anterior chamber reaction and bleb congestion), topical antibiotic Moxifloxacin 0.5% drops with tapering dose, and sometimes topical Cycloplegic eye drops for 1 week.

Follow-up

Enrolled patients were evaluated on follow-up visits at 1 day, 1 week, 4 weeks, 3 months, 6 months, and 1 year after surgery, with intervening visits scheduled as required.

At every visit, IOP, the number of antiglaucoma medications, visual acuity, any complications, and interventions were recorded.

Result

Table I: Preoperative and post-operative IOP (N=20)

	Ab-Interno bleb needling (mean ± SD)	Ab Externobleb needling (mean ± SD)	p-value
Pre-Operative IOP (mmHg)	35.30 ± 7.86	31.50 ± 5.40	0.224
Postoperative IOP (mmHg)	12.80 ± 3.99	20.00 ± 10.67	0.061
Change in IOP (%)	61.34 ± 17.04	37.21 ± 31.83	0.049

Unpaired t test was done

Percent change was significantly higher in Ab Interno bleb needling than Ab externo bleb needling.

The ab interno comparative study demonstrated a mean IOP reduction of (61.34 ± 17.04) % mmHg 3 months, compared to a mean reduction of (37.21 ± 31.83) % mmHg (40.1% decrease) in the ab externo group (p =0.049).

Table II: Comparison of the outcome between the two procedure (N=20)

	Ab-Interno bleb needling(%)	Ab Externo bleb needling (%)	p-value
Successful	9 (90.0%)	5 (50.0%)	
Unsuccessful	1 (10.0%)	5 (50.0%)	0.141

Fisher's Exact test was done Success rate was higher in Ab Interno bleb needling than Ab externo bleb needling but the difference was not statistically significant.

Table III: Percent change was significantly higher in Ab Interno bleb needling than Ab externo bleb needling.)

	Ab-Interno bleb needling(%)	Ab Externo bleb needling (%)	p-value
Successful	9 (90.0%)	5 (50.0%)	
Unsuccessful	1 (10.0%)	5 (50.0%)	0.141

Fisher's Exact test was done

Success rate was higher in Ab Interno bleb needling than Ab externo bleb needling but the difference was not statistically significant

Discussion

Trabeculectomy filtration surgery has a high failure rate in younger patients, as well as old patients due to a high risk of conjunctival scarring. The ab-interno needling approach with or without Mitomycin C injection is a novel technique that can be performed and offers the advantage of increasing aqueous outflow through a direct opening of the trabecular meshwork from within the anterior chamber.

Our study prospectively followed consecutive patients who underwent an ab interno bleb revision and completed a minimum 6 monthsfollow-up. Bleb function could be restored in 90% of patients, and the IOP decreased from a mean of 35.30 mm Hg to 12.80 mm Hg at 6 months. The number of AGM required significantly reduced from a preoperative mean of 3.99 to a mean of 1.25 at 6 months. These results compare favorably with the original case series of AIBR by Grover and Fellman where 10 eyes of 10 patients were retrospectively analyzed. [10] They reported a mean preoperative intraocular pressure of 21.9 mm Hg on 3.7 glaucoma medications, which declined to 12.1 mm Hg on 0.86 drugs at 12 months follow-up. In their series, the procedure failed in 5 eyes, of which 4 underwent a glaucoma drainage implant. Ruparelia et al described their experience of ab interno revision of failed trabeculectomy blebs using a Grover Fellman Spatula or a Troutman Barraquer iris spatula. [11] The report did not separate out the results of the patients done with the GFS, and they used postoperative 5-Fluorouracil instead of preoperative MMC as described in the original description. After 1 year of follow-up, they reported a total success rate of 68% (53% complete and 15% qualified). In our study we did not use any anti metabolites.

Patients who underwent an ab externo bleb revision and completed a minimum 6 months follow-up. Bleb function could be restored in 50% of patients, and the IOP decreased from a mean of (31.50 ± 5.40) mm Hg to (20.00 ± 10.67) mm Hg at 6 months. The procedure failed in 5 eyes (IOP was not so much controlled, but bleb was partially functioning in 2 cases and failed belb was in 3 cases).

A previous systemic review about ab externo bleb needling without antimetabolites found no conclusive evidence that a single needling outstood conservative antiglaucomaTous medication in IOP controlling for encapsulated blebs [12]

We have demonstrated that bleb needling is a useful adjunct as sub conjunctival scarring occurs. In accordance with our findings, bleb needling is a useful method to rescue failing blebs to delay repeat filtration surgery or the need for implanting glaucoma drainage devices.

The mean interval between trabeculectomy and first needling also varies among different studies. Ab Externo bleb needling has been reportedly successful up to 30years after filtration surgery.[13]

We observed during the study that the resulting blebs following the ab interno approach were smaller and more variable. This may, therefore, play a greater role in controlling IOP, and it was observed that the difference in pressure drop across eyes where the tube was inserted via an ab interno approach was greater and more variable between eyes than via an ab externo approach.

Ab interno approach were more successful than ab externo, but according to our observation, after trabeculectomy and failed bleb, greater the intervention time, less the chance of success rate. Those who underwent ab interno bleb revision after 10 months to years, due to dense tissue fibrosis, failure rate was high. But intra operative bleeding is common in ab interno approach.Most of the cases of our study iundergone ab interno approach, the intervention were within less duration following bleb failure and yeildedfavourable result.

Limitation

Our study has several limitations. It is a prospective study without a control group, and it was conducted with a limited sample size over a short duration. Further long-term outcomes and a larger patient cohort would enhance the robustness of our findings.

Conclusion

Ab-Interno bleb Revision (AIBR) is a safe, effective, and minimally invasive intervention for managing failed trabeculectomy blebs. By eliminating the need to reopen the conjunctiva, this technique offers a promising alternative for the treatment of this challenging condition.

On other hand, ab externo bleb needling could be considered an effective and safe procedure after trabeculectomy failure. After the process, patients will gain ideal IOP control and reduce anti glaucomatous medications. Meanwhile, the overall estimates for complications were relatively low in the whole process. An increasing number of needling was the leading risk factor for needling failure.

So we can come to a conclusion that both ab externo and ab interno approach for the treatment of failed bleb are helpful for restoration of bleb function and to reduce IOP. If the patient come within 10 months to 1 year with less fibrosis, then ab introno approach will be more appropriate as its success rate is higher than ab externo approach. But if the patient come after years then ab externo procedure will be more appropriate as in this procedure more surface can be approached.

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Review Article

Ocular menifestations of sturge weber syndrome : a review article

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Abstract

Sturge–Weber syndrome has been included in the group of phakomatoses that is characterized by hamartomas involving the brain, skin, and eyes. The characteristic facial port-wine stain, involving the first branch of the trigeminal nerve and the embryonic vasculature distribution in this area, leads to several ocular complications of the anterior segment and can involve the eyelids and conjunctiva. The posterior segment of the eyes is also affected with diffuse choroidalhemangiomas. However, the most frequent ocular comorbidity is glaucoma with a prevalence rate ranging from 30%–70%. Glaucoma is related to anterior chamber malformations, high episcleral venous pressure (EVP), and changes in ocular hemodynamics. Glaucoma can be diagnosed at birth, but the disease can also develop during childhood and in adults. The management of glaucoma in Sturge–Weber syndrome patients is particularly challenging because of early onset, frequently associated severe visual field impairment at the time of diagnosis, and unresponsiveness to standard treatment. Several surgical approaches have been proposed, but long-term prognosis for both intraocular pressure control and visual function remains unsatisfactory in these patients. Choroidalhemangiomas may also lead to visual impairment thorough exudative retinal detachment and macular edema. Treatment of exudative hemangioma complications is aimed at destructing the tumor or decreasing tumor leakage.

Keywords : Sturge-Weber syndrome, glaucoma, choroidalhemangiomas, port-wine stain, congenital disease, glaucoma surgery.

Introduction

Sturge–Weber syndrome (SWS) has been included in the group of phakomatoses that includes neurofibromatosis, Klippel–Trenaunay syndrome, tuberous sclerosis, and von Hippel–Lindau syndrome.[1] SWS, also known as encephalotrigeminalangiomatosis, is a condition that includes leptomemeningealhemangioma, facial angiomatosis or nevus flammeus (also called port-wine stain [PWS]), and ocular pathological changes.[2–5] The incidence of SWS is ~ 1:50,000 infants, with no significant difference between males and females.[6] No hereditary pattern or predisposition has been shown, and no malignant transformation has been demonstrated.[7,8] Several genes in the 17p1–p13 region, which are known to be involved in SWS, are also known to be linked to rare abnormalities and syndromes, such as retinitis pigmentosa, cerebral astrocytoma, subglottic stenosis, Klippel–Trenaunay–Weber syndrome, and phakomatosis pigmentovascularis.[9–13]

The embryologic basis of SWS has been reported to be related to an impaired development of the cell precursors in the neural crest during the first embryological trimester, leading to the characteristic malformations observed in the central nervous system, skin, and eyes.[3] Happle suggested that somatic mosaic mutations involving the skin cause sporadic or scattered birth defects, and the phase of embryonic development during which this occurs is fundamental.[14] Shirley et al recently published a ground-breaking study where a mutation in the GNAQ gene was identified, which leads to stimulation of cell proliferation and inhibition of apoptosis.[15] The authors found that the mutation is associated with both sporadic PWSs and SWS.[15]

Diagnosis is easily performed when the classical clinical signs of SWS are present, consisting of unilateral facial PWS along the first branch of the trigeminal nerve, hemiatrophy, progressive seizures, contralateral hemiparesis, mental deficiencies, hemianopia, and ipsilateral glaucoma.[16] However, Waelchli et al showed that the PWS distribution may in fact follow the embryonic vasculature distribution of the face, rather than the trigeminal nerve.[17] Neuroim-

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aging techniques help to perform the diagnosis, showing gyriform calcifications that engage the parietal and occipital lobes, leptomeningealangiomatosis, and astrogliosis in the brain.[18] The diagnosis of SWS is based on the presence of at least two of the three manifestations of the classic triad (leptomeningealangioma, PWS, and ocular abnormalities). However, clinical findings of SWS may vary, showing variable neural signs and symptoms with the absence of or varying ocular involvement.[19]

According to the clinical manifestation, SWS is classified into four types: 1) presence of brain and facial angioma, with or without glaucoma, 2) PWS without brain involvement, with or without glaucoma, 3) isolated brain angioma, usually without glaucoma, and 4) type 1 associated with systemic manifestation such as tuberous sclerosis (Table 1).[20]

Table 1 : Classification of Sturge-Weber syndrome

Type	Facial angioma	Leptomenige -alangioma	Glaucoma	Systemic manifestation
Type I	+	+	±	±
Type II	+	-	±	-
Type III	-	+	±	-
Type IV	+	+	±	+

Ocular findings

Approximately 50% of SWS patients show pathologic ocular changes, usually ipsilateral to the PWS, involving the eyelid, anterior chamber, cornea, choroid, and retina.[3–9,13,16,19–21] The presence of PWS may involve the eyelid and trigger pathological alterations in the ocular blood flow. The bulbar conjunctiva (frequently at the limbus) may show diffused or localized area of pinkish discoloration related to increased conjunctival vascularization. Episcleral vessel dilatation can be observed in approximately half of SWS patients.[21]

Several anterior chamber changes have been described in SWS including vascular formations in the trabecular mesh-work near the scleral spur surrounded by large homogeneous extracellular matrix, association of the endothelial layer lining of Schlemm's canal with the basal lamina, and presence of several villi and giant vacuoles within the endothelial cells that appear as transcellular channels.[22]

These anatomical alterations justify the frequent development of glaucoma in 30%–70% of SWS patients.[23,24] Glaucoma in SWS patients shows a bimodal peak of age development: an early-onset (congenital) form affecting ~ 60% of patients and a later-onset form during childhood and adolescence (40% of cases).[23] Glaucoma in SWS is unilateral and is associated with the presence of an ipsilateral PWS,[24] which most frequently involves both the eyelids (72%) and sometimes only the upper eyelid (21%).[13] The most frequent form of glaucoma in SWS patients is open-angle glaucoma leading to progressive visual field loss. However, acute glaucoma attack due to chamber angle closure has also been described.[25–27] Congenital glaucoma is frequently associated with corneal changes (25%), including haze, megalocornea, and buphthalmos.[21] Other anterior segment alterations have been described in SWS patients including iris heterochromia and cataract.[19]

The posterior segment of the eye is also involved with hemangiomas of the choroid (20%–70% of cases).[28] Choroidalhemangiomas can be clinically divided into localized and diffuse forms, but it is the diffuse form that typically occurs in SWS patients.[19,21] Clinically, ophthalmoscopy shows a bright red or red–orange color appearance of the fundus, related to the increase of well-formed choroidal vessels, while hemangiomas appear as diffuse or localized areas with a dark red color and a "tomato ketchup" appearance.[13] The presence of choroidalhemangiomas is frequently asymptomatic; however, the choroid may become significantly thickened,[29] and an increased risk of glaucoma development has been reported in patients with choroidal hemangioma.[30]

Alteration of the choroidal vessels may lead to severe retinal complications and potentially to visual loss. Subretinal hemorrhage, retinal

degeneration, retinal serous detachment, photoreceptor degeneration, cystoid macular edema, macular serous detachment, tortuous retinal vessels, and optic disc coloboma have been described in SWS patients.[21,31]

Ophthalmic management

Routine slit lamp examination is sufficient to detect most of the alterations typically observed in SWS patients; however, if young children cannot cooperate and there is concern for visually threatening diseases such as glaucoma, examination under general anesthesia may be recquired. Anterior segment alterations include eyelids, conjunctival and episcleralhemangiomas, and corneal changes related to congenital glaucoma. Fundus examination by indirect ophthalmoscopy or biomicroscopy shows the typical features of choroidalhemangioma as diffuse or rarely localized areas of flat or elevated dark retina, and frequently highlights the difference in fundus color with respect to the fellow eye. Retinography may be also useful in documenting this color difference.

It is mandatory to strictly monitor glaucoma in SWS patients. Clinicians can rely on more than one parameter for diagnosing and monitoring glaucoma. In very young patients, parameters such as axial length and corneal diameter are important. Gonioscopy is a simple and useful examination to detect iridocorneal angle malformation and to orient the diagnosis toward the type of glaucoma. Intraocular pressure (IOP) measurement, optic nerve evaluation, as well as visual field testing represent the main procedures to diagnose and monitor glaucoma progression. The optic nerve head can be observed with optical coherence tomography, which is a non-invasive and relatively rapid method of evaluation in children who have difficulty in cooperating due to their young age and/or intellectual difficulty linked to diseases or syndromes such as the SWS.[32,33] Visual field analysis is fundamental in evaluating glaucoma progression, but this examination can be challenging to perform in children, although with increasing age and learning more reliable results can be obtained.[34–37]

Several instrumental examinations may also improve the diagnosis and monitoring of choroidalhemangioma and retinal complications, especially in the presence of anterior segment opacity, such as in congenital glaucoma. Ultrasonography allows to detect choroidal alterations and to evaluate their extension, characteristics, and echogenicity. Indocyanine green angiography can add useful information on the extension and intralesional vascularity of the choroid as well as the potential detection of arterovenous shunts. Currently, the gold standard examination to characterize choroidal and retinal morphology is enhanced depth imaging spectral domain optical coherence tomography, which allows the quantification of choroidal thickness as well as the morphology and the caliber of dilated choroidal vessels.[29,38] Magnetic resonance imaging may also be useful to detect broader thickening of the eyes as a consequence of diffuse choroidalhemangioma.

It is crucial to plan complete ophthalmic examinations in the follow-up of SWS patients in order to avoid visual function loss, frequently related to progressive glaucoma, and also to detect the onset of retinal complications such as exudative detachment.

Glaucoma pathogenesis

Several new hypotheses and factors have been formulated on the pathogenic mechanisms leading to the development of glaucoma, and several hypotheses have been formulated to explain the pathogenic mechanisms leading to the development of glaucoma associated with rare conditions.[39–41]

The main theories on the pathogenesis of glaucoma in SWS include the following:

1. A mechanical mechanism related to congenital malformation of the anterior chamber angle leading to increased resistance to aqueous humor outflow: in this case, the iris can also not have the flat anterior insertion characteristic of the congenital form;[24]
2. an increase in episcleral venous pressure (EVP) due to arteriovenous shunts into the

episcleral hemangioma:[29] this theory is based on the observation of a normal angle structure, blood within Schlemm's canal, and more severe glaucoma;[42–44]

3. fluid hypersecretion by either the ciliary body or the choroidal hemangioma;[45] and
4. abnormal hemodynamics of the episclera and the anterior chamber angle due to premature aging of the trabecular meshwork–Schlemm's canal complex, as observed in SWS later-onset glaucoma.[46]

In SWS patients, an increase in EVP, as compared to normal eyes, has been reported in glaucoma-affected eyes.[42,47] However, no significant difference in EVP was observed between glaucoma-affected and normal eyes in SWS patients, suggesting that elevated EVP is only a risk factor for glaucoma onset.[47] Retrobulbar blood flow was also impaired in SWS patients, suggesting an increased risk to develop glaucoma with aging. However, no significant difference in the arterial retrobulbar blood flow was observed between the glaucomatous and the normal fellow normal.[48,49]

Glaucoma pathogenesis in SWS patients is complex, including different interlinked mechanisms that change their role with aging. Indeed, in congenital and early-onset glaucoma forms, angle malformations play a crucial role. In late-onset glaucoma, an increase in EVP, probably related to progressive hypertrophy and dilatation of the episcleral veins as reported for PWS,[50] has a major pathogenic role.

A few cases of acute glaucoma attack due to angle closure have been described in SWS. Maruyama et al reported a case of a patient with SWS who developed acute glaucoma associated with posterior scleritis, edema of the ciliary body, ciliochoroidal effusion, and anterior rotation of the ciliary body.[25] In this case, it was hypothesized that the ciliary body effusion induced angle closure by moving the iridolenticular diaphragm forward, which led to acute glaucoma attack. This mechanism of glaucoma induction has also been described following the use of the drug topiramate, which is used for seizures that can occur in SWS patients with leptomeningeal involvement.[27] Angle-closure glaucoma in SWS has also been described as related to pupil block with or without ectopialentis and pigment dispersion.[51,52] In these cases, ultrasound biomicroscopy may represent a precious tool to identify and characterize the pathogenic mechanism of acute glaucoma attack.[53]

Medical treatment of glaucoma

The main goal of glaucoma treatment is to control IOP and to avoid progressive optic nerve damage and visual field loss. Both medical and surgical approaches have been performed to halt glaucoma progression in SWS patients (Table 2). Due to the rare nature of the disease, only small case series have been published leading to a relatively low level of evidence regarding treatment efficacy. Topical antiglaucoma drugs seem to be less efficacious in SWS patients with congenital glaucoma, while they represent first-line therapy for patients with late-onset glaucoma.[44]

Abbreviation: IOP, intraocular pressure.

Ong et al showed that latanoprost eye drops, as adjunctive therapy, were effective in controlling glaucoma in 50% of 14 patients with SWS at 1 year of follow-up.[54] In line with this, Yang et al demonstrated that latanoprost treatment induced a significant pressure decrease in 33% (two eyes) of SWS glaucoma patients.[55] Latanoprost acts by increasing uveoscleral outflow, theoretically bypassing the obstacle to the passage of aqueous humor due to the increase in EVP. However, long-term use of prostaglandins in SWS patients should be carefully evaluated since one of the potential side effects of these drugs is uveal effusion.

Only a few case reports describe the use of other anti-glaucoma drugs in patients with SWS, suggesting that treatment with beta-blockers and carbonic anhydrase inhibitors is effective in the absence of buphthalmos.[56]

Wygnanski-Jaffe et al used oral propanolol for treating glaucoma in SWS patients.[57] Systemic propranolol is effective in inducing reduction of

Table 2 : Treatment of Sturge-Weber syndrome-associated glaucoma

Author	Year	Number of patients	Type of study	Intervention	Outcome
Basler and Sowka[44]	2011	1	Case report	Travoprost eye drop	Good IOP control
Sharan et al[45]	2009	24	Retrospective	Medical/surgery	Good IOP control: 9 mono- and 9 multi-drug therapy; 10 trabeculectomy and 2 valve implant
Ong et al[54]	2003	14	Retrospective	Latanoprost eye drop	Good IOP control in 50% at 1 year
Yang et al[55]	1998	6	Prospective	Latanoprost eye drop	Good IOP control in 33%
Wygnanski-Jaffe et al[57]	2015	4	Prospective	Oral propranolol	Not effective as single treatment: 75% required additional treatment
Saltzmann et al[60]	2012	7	Retrospective	Trabeculectomy– trabeculotomy	Not effective and high failure rate
Board and Shields[61]	1981	5	Case series	Trabeculectomy	Good IOP control in 20% at 6 months
Agarwal et al[62]	1993	16	Retrospective	Trabeculotomy– trabeculectomy	Good IOP control in 61% at 42 months
Mandal[65]	1999	9	Case series	Trabeculotomy– trabeculectomy	Good IOP control in all patients at 1 year
Audren et al[67]	2006	6	Retrospective	Deep sclerectomy	Not effective at 26 months
Hamush et al[69]	1999	10	Case series	Ahmed valve	Good IOP control in 30% at 60 months
Amini et al[70]	2007	7	Prospective	Molteno tube	Good IOP control in 43% at 36 months
Naranjo-Bonilla et al[71]	2014	1	Case report	Ex-Press® implant	Good IOP control at 10 months
Elgin et al[72]	2007	1	Case report	Ex-Press® implant	Good IOP control with adjunctive trabeculectomy
Van Emelen et al[73]	2000	8	Retrospective	Medical/surgery	Good IOP control in 12% with medical therapy alone

hemangiomas of the skin, orbit, larynx, and eyes.[58,59] However, systemic propanolol was ineffective in three of four children with SWS-related glaucoma.[57]

A hypothesis to justify the low efficacy of antiglaucoma drugs in controlling SWS-related glaucoma is that most of these drugs do not affect EVP, highlighting the need for novel antiglaucoma medications specifically targeting EPV.

Surgical treatment of glaucoma

Frequently, medical treatment in SWS patients is not sufficient to guarantee a good long-term control of glaucoma; therefore, surgical procedures are commonly performed in patients under 2 years of age. Goniotomy and trabeculotomy represent the most appropriate surgical procedures to overcome the malformed anterior chamber angle under 4 years of age. However, Saltzmann et al demonstrated that trabeculotomy fails to control congenital glaucoma in all patients with SWS.[60] These authors suggested to consider alternative surgical procedures and closer postoperative monitoring due to this significant relative risk of failure.[60] One of the possible explanations of surgery failures in SWS children is that goniosurgery does not affect EVP, suggesting that pathogenic mechanisms other than the simple angle malformation influence glaucoma progression.

Other surgical approaches include filtering procedures such as trabeculectomy, posterior lip sclerotomy,[42] and trabeculotomy– trabeculectomy,[61,62] which bypass the alterated trabecular meshwork–Schlemm's canal complex, creating an alternative outflow passage for the aqueous humor, independent to the distal episcleral veins. However, filtering procedures have been associated with severe complications such as expulsive choroidal hemorrhage, bleeding, prolonged flat anterior chamber, and high risk of bleb failure.[46,63] Intraoperative use of antimitotic agents such as mitomycin does not improve the outcome.[64] Therefore, combined procedures such as trabeculotomy–trabeculectomy have been suggested as the first-line approach in infants and children.[61,65]

Alternative surgical procedures include nonpenetratingsclerectomy, valve drainage implants, and ciliodestructive procedures in adults.[66,67] Nonpenetratingsclerotomy has been shown to have an efficacy similar to trabeculectomy in controlling SWS-related

glaucoma, with a lower rate of complications.[67] However, in SWS patients, the presence of episcleralhemangioma and angle malformations make this procedure extremely difficult and increase the rate of failure.

Valve implant is also a successful method for IOP management.[68] In SWS, the Ahmed-type valve was shown to induce long-term decrease of IOP by improving aqueous humor outflow.[69] On the other hand, the use of the Molteno tube showed an unfavorable outcome due to an elevated complication rate in children with SWS.[70]

Two studies reported that Ex-Press® valve was successfully implanted in children with SWS as an elective surgical procedure,[71] and in the other case, 10 days prior to performing trabeculectomy.[72] On the other hand, the use of the Ex-Press® glaucoma implant in another child with SWS was complicated by choroidal detachment, which resolved with medical therapy. Lastly, it is worth mentioning that cyclocryotherapy has been used successfully in patients with SWS and buphthalmos in combination with topical antiglaucoma therapy[73] or trabeculectomy.[74] Nevertheless, cyclocryotherapy can be burdened by severe complications such as hypotony and phthisis bulbi.[74] Unfortunately, sometimes, even with medical and/or surgical treatment glaucoma leads to severe visual field damage, visual acuity loss, and optic nerve atrophy; only rehabilitation methods can help in improving the quality of life of these patients.[75–77]

Treatment of other ocular manifestations

Facial PWSs can be successfully treated by laser. Better esthetic results are achieved when treating the lesions localized on the central forehead rather than the lesions on the central face. To obtain better results, PWS should be treated early since there is a tendency to thickening and nodular transformation with aging.[78]

Laser treatment should be superficial in order to avoid potential complications related to a decrease of brain venous outflow through PWS vessels, potentially leading to cerebral venous deterioration, choroidal vessel dilatation, retinal exudative detachment, and increase in IOP.[7] Therefore, deep photocoagulation and debulking surgery should be avoided in the treatment of PWS.[7]

As previously described, visual loss in SWS patients is mainly related to glaucoma development, but choroidalhemangiomas may also lead to visual impairment thorough exudative retinal detachment and macular edema. The treatment of exudative hemangioma complications is aimed at destructing the tumor or at least at decreasing tumor leakage.[79] Several treatments have been proposed including photocoagulation, photodynamic therapy (PDT), external beam radiotherapy, brachytherapy, and anti-vascular endothelial growth factor (VEGF). The scopes of these methods are to reduce tumor size, induce vessel atrophy, close leaking vessels, and induce inhibition of VEGF to ultimately diminish subretinal fluid, which causes increased retinal and macular thickness as observed with optical coherence tomography.[80–82]

Confluent photocoagulation causing destruction of the tumor has been considered as the most effective treatment in reducing leakage.[83] However, several complications can be observed following intense photocoagulative treatment, leading to the proposal of an alternative, less invasive laser procedure, such as grid treatment, that is safer but has a higher recurrence of subretinal fluid accumulation.[84,85]

PDT is theoretically the most indicated procedure to treat choroidalhemangiomas, inducing vessel atrophy and reducing leakage.[66] However, currently, only a few cases of diffuse choroidalhemangioma have been treated with PDT, probably due to the risk of inducing scarring and pigmentary changes of the fovea.[86]

In case of diffuse choroidalhemangioma complicated by serous retinal detachment, an alternative treatment is external beam radiotherapy.[87,88] Clinical improvement has been observed months following the first application but recurrence frequently occurs. Repeated applications should be carefully evaluated since they can induce several complications such as cataract, neuropathy, and radiation retinopathy.[87] Exudative retinal detachment related to choroidalhemangiomas has also been successfully treated using brachytherapy with cobalt-60 and ruthenium-106.[89–92] Intravitreal injection of pegaptanib (an anti-VEGF) has also been described in one SWS patient with

exudative retinal detachment with satisfactory results.[93,94]

Conclusion

Glaucoma is the most common ocular complication in SWS. The most frequent form is congenital glaucoma, but it can also occur in children and adults, making careful ophthalmic follow-up of SWS patients mandatory. Indeed, glaucoma secondary to SWS is a challenging disease due to its early development and poor response to standard medical treatment.

Surgery is frequently required to obtain long-term control of IOP in order to avoid visual function loss. However, several severe complications related to surgical procedures have been described in these patients, including choroidal effusion, expulsive hemorrhage, and exudative retinal detachment. Moreover, in SWS, the surgical success rate is the lowest among secondary glaucomas, since surgical failure, uncontrolled IOP, and low vision outcomes have been frequently reported.[45,60,61] To date, despite the wide range of available medical and surgical approaches to treat both the congenital and late-onset forms of glaucoma, the development of this ocular complication still represents the worst prognostic factor for vision loss in SWS patients.

Choroidalhemangiomas are present in 20%–70% of cases, and the choroid may become significantly thickened and can be associated with an increased risk of glaucoma development.[29] Choroidalhemangiomas may be asymptomatic; however, severe forms may lead to visual impairment thorough exudative retinal detachment and macular edema. The treatment of exudative complications is aimed at destructing the tumor or at least at decreasing tumor leakage.

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Management of ICE syndrome and secondary glaucoma: a case series

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Abstract

Purpose: To report a case series of Iridocorneal Endothelial Syndrome with secondary glaucoma managed through different way as per need.

Case report:

Case 1:

A 44 years old male came with the complaints of loss of vision in his left eye. On examination, Visual acuity in left eye was PL, PR. Other findings were, cornea; edema, pupil: Irregular D shaped, poycoria, lens pseudophakia, C:D ratio was .8, macular scar and old resolved RD. Intra ocular pressure was 58 in left eye. Right eye findings were within normal limit. After proper clinical examination and investigation, he was diagnosed as ICE syndrome in left eye.

Case 2:

A 29 years female presented with the complaints of blurring of vision in her left eye. On examination: Visual acuity in her left eye was 6/18, pupil: irregular, corectopia, iris: atrophy, iris naevus, lens: transparent. C:D was .8 in her left eye. IOP in left eye was 34. Right eye findings were within normal limit. After proper clinical examination and investigation, he was diagnosed as ICE syndrome with status post trabeculectomy in left eye.

Case 3:

A 52 years male patient presented with the complaints of blurring of vision in his right eye, on examination: Visual acuity was 6/36 in his right eye; cornea: hammered silver appearance, pupil: irregular, iris: atrophy, lens: opaque (NSII, PSC cataract), PAS, C:D ratio was .8 in right eye. left eye findings were within normal limit. After proper clinical examination and investigation, he was diagnosed as ICE syndrome in left eye.

Conclusion: ICE syndrome with secondary glaucoma is a sight threatening serious condition, Proper clinical diagnosis and timely treatment according to sign of the disease is essential for the resolution of symptom and sign. Keywords: ICE syndrome, IOP

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Introduction

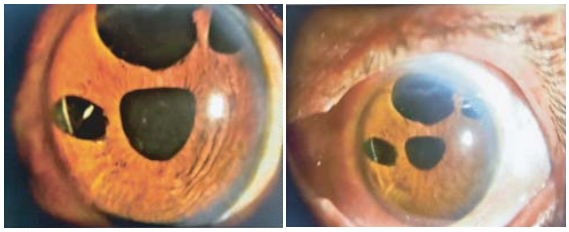
Iridocorneal endothelial (ICE) syndrome is a rare ophthalmic disorder, wherein the basic pathology is an abnormal corneal endothelium that leads to varying degrees of corneal oedema, iris atrophy and secondary angle closure glaucoma.[1] This syndrome typically occurs unilaterally, affects young female without any family history.[2] The true a etiology of ICE syndrome is unclear. Viral cause for the disease has been proposed, based on a history of inflammation in certain cases and on the presence of inflammatory cells on histological analysis.[3] The abnormal endothelial cells may migrate posteriorly, form a membrane like covers over the adjacent structures, iris and trabecular meshwork.[4] The contraction of this membrane leads to characteristic iris changes, iridotrabecular synechiae, and corectopia with the pupil being drawn towards the area where the synechiae are most prevalent and to secondary angle-closure glaucoma.[5]

Case history:

Case 1:

A 44 years old male came with the complaints of loss of vision in his left eye. On examination:

	RIGHT EYE	LEFT EYE
VA (unaided)	6/9	PL, PR+
Cornea	clear	oedema
Anterior chamber	Shallow	shallow
Lens	Transparent	Pseudophakia
Pupil	RRR	Irregular, D shaped, Pserudopolycoria
Iris	Normal	Pseudopolycoria
Fundus	Small temporal lattice, C:D 0.3	Glaucomatous disc, C:D 0.8, macular scar, old resolved RD
IOP	12	58



We did some investigations following all clinical examination.

On Visual field analysis, there was no scotoma in right eye and in left eye there was advanced glaucoma. OCT showed right eye WNL, and left eye showed marked RNFL thinning in all quadrant, and C; D was .88.

With all clinical examinations and investigations patient was diagnosed as Pseudophakia with ICE syndrome left eye.

Then patient was treated with topical and systemic anti glaucoma medications. On his 15 days follow up, IOP was not controlled adequately and patient was underwent Trabeculectomy. After Trabeculectomy, during 1st follow up IOP was 12 mm of Hg.

Case 2 :

A 29 years female presented with the complaints of blurring of vision in her left eye. On examination:

	RIGHT EYE	LEFT EYE
VA (unaided)	6/9	6/18
Cornea	clear	clear
Anterior chamber		Block internal OS of preveous trabeculectomy site.
Lens		Transparent
Pupil	RRR	Irregular, corectopia
Iris	Normal	Iris naevus, atrophy
Fundus	NAD lattice, C:D 0.3	Glaucomatous disc, C:D .8
IOP	12	46

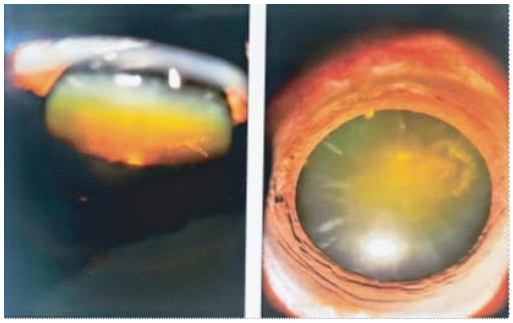
After all clinical examination, some investigation were done. On Visual Field analysis, there was no scotoma in right eye and in left eye there was advanced glaucoma. OCT showed right eye WNL, and left eye showed marked RNFL thinning in all quadrant, and C; D was. 88.With this clinical examination and all investigations we came to a diagnosis of ICE syndrome with status post failed trabeculectomy left eye. Then bleb revision was done under anti glaucoma medications and IOP was controlled as well.

Case 3:

A 52 years male patient presented with the complaints of blurring of vision in his right eye, on examination: Visual acuity was 6/36 in his right eye; cornea: hammered silver appearance,

pupil: irregular, iris: atrophy, lens: opaque (NSII, PSC cataract), PAS, C:D ratio was .8 in right eye. left eye findings were within normal limit. After proper clinical examination and investigation, he was diagnosed as ICE syndrome in left eye.

	RIGHT EYE	LEFT EYE
VA (unaided)	6/9	6/18
Cornea	Hammered silver appearance	oedema
Anterior chamber	Normal	Normal
Lens	NS II + PSC cataract	NS II + PSC cataract
Pupil	Irregular	RRR
Iris	Atrophy, nodule	Normal
Fundus	C:D .8	C:D .4
IOP	26	14



After doing all investigations and clinical examinations, patient was diagnosed as nasal pterygium right eye, ICE syndrome left eye. Then phacoemulsification with PCIOL was done in right eye under anti glaucoma coverage and patients IOP was reduced and VA was improved up to 6/9 with refraction. Patient was doing well with single anti glaucoma medication.

Discussion

Iridocorneal Endothelial Syndrome (ICE) is a unique ophthalmic disorder that involves an abnormal corneal endothelium that leads to varying degrees of corneal edema, iris atrophy, and secondary angle-closure glaucoma[6]. This syndrome, which typically affects young women unilaterally with no family history[7], encompasses three clinical variants: Chandler Syndrome, Essential (Progressive) Iris Atrophy, Cogan-Reese Syndrome (Iris Nevus Syndrome).

The true etiology of ICE syndrome is unclear. Alvarado et al. have proposed a viral cause for the disease, based on a history of inflammation in certain cases and on the presence of inflammatory cells on histological analysis[3]. Further exploring this hypothesis, the same author revealed Herpes Simplex DNA in the pathological corneas by using the PCR (Polymerase Chain Reaction) technique [8].

When medical therapy is unsuccessful at controlling IOP, surgical therapy with a filtering procedure may be necessary. A trabeculectomy with antifibrotic agents (mitomycin-C or 5-fluorouracil) or a glaucoma drainage device (aqueous shunt) have been found to be effective in controlling IOP in ICE syndrome patients

Trabeculectomy is the surgery of choice for ICE syndrome. Shields et al. have reported a 69% success rate in a study conducted in 1978 on 33 eyes[9], while Yanoff reported a 64% success rate 1 year postoperatively and a 36% at 3 years[3]. When the trabeculectomy proves to be ineffective, the reason is usually excessive subconjunctival scarring[10] (a frequent occurrence in patients with ICE syndrome, given their young age). ICE-specific phenomena that lead to failure are bleb and/ or filtering ostium reendothelialization[11] and PAS formation that obstruct the drainage pathway corneal decompensation can similarly be treated with surgery when medical management fails. Penetrating keratoplasty (PKP) or endothelial keratoplasty (commonly DSEK or DSAEK) can be implemented to replace the abnormal corneal endothelial cells and improve corneal function.13 At times, both a filtering and corneal transplant procedure are necessary. Concomitant control of IOP is vital for maintenance of corneal graft

clarit.

Among the three study cases, 2 were underwent trabeculectomy and IOP was controlled without anti glaucoma medications, one case IOP was slightly reduced after cataract surgery. As the IOP spike was not so high and lens was opaque, phacoemulsification was done and after surgery, with single anti glaucoma medication patient is doing well. Further studies are needed to determine the incidence of the disease (previously unknown because of rarity), as well as the possible etiology. The best way of handling and treatment remains under discussion, presenting a limited prognosis until the present day.

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Lens induced glaucoma with traumatic cataract : A case report

S A Nipa[1], S Rahman[2], Z J Ruma[3], S A Nipu[4]

Abstract

Purpose : To report a case of lens induced glaucoma with traumatic cataract.

Case summary : A 42 years old male presented with the complaints of severe pain and dimness of vision in right eye. He had a history of trauma in right eye 6 days back. On examination best corrected visual acuity in right eye was HM and 6/9 in left eye. By slit lamp examination revealed pupil mid dilated, cornea clear, cataract with rupture lens capsule in right eye and NAD in left eye. IOP on his right eye was 42 mm of Hg and left eye was 10 mm of Hg. The patient was diagnosed as Lens induced glaucoma with traumatic cataract. Considering the rise of the IOP, patient received pressure lowering topical and systemic therapy and also with IV mannitol support. The IOP normalized under treatment patient underwent cataract surgery with IOL implantation. His post operative best corrected visual acuity was 6/12 and IOP was 12 mm of Hg in right eye.

Conclusion : Acute reduced vision, redness, eye pain are the main clinical presentation of LIG. Early detection by primary physician is important. Cataract extraction following IOP lowering agent aids in visual recovery and residual IOP control of lens induced glaucoma.

Key words: Lens induced glaucoma, traumatic cataract, rupture capsule.

Introduction

In developing country Lens induced glaucoma is one of the very important causes of secondary glaucoma[1]. LIG may be divided into two categories which presenting as secondary angle closure and secondary open angle glaucoma. SACG relates to blockage of aqueous humour flow from posterior to anterior chamber or another caused by lens swelling (Phacomorphic glaucoma) or lens dislocation (ectopia lentis). SOAG is caused by trabecular meshwork blockage from lens proteins (phacolytic glaucoma), lens trauma, liberation of lens material after capsular disruption during cataract extraction, Nd:YAG posterior capsulotomy (lens particle glaucoma) or any inflammatory reaction occur against own lenticular antigens (phacoantigenic glaucoma)[2].

Case history:

A 42 years old male with history of trauma in right eye 6 days back presented with these clinical finding

Indications	Right Eye	Left Eye
VA	HM	6/9
Cornea	Clear	Clear
Anterior	Remained	Normal
chamber	cortical matter	
Lens	Opaque, Hypermature cataract, ruptured capsule	Clear
Pupil	Mid dilate	RRR
Iris	Normal	Normal
Fundus	NAD	NAD
IOP	42 mm of Hg	10 mm of Hg

No abnormality found in B-scan ultrasound of his right eye. Systemic examination revealed no abnormality involved. Above clinical findings supported the diagnosis Lens induced glaucoma with traumatic cataract. Initially patient was treated with I/v mannitol. Topical brinzolamide, timolol and brimonidine were given to the patient 3 times daily. Additionally he received oral acetazolamide 250 mg and potassium

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chloride 3 times daily. Difluprednate 4 times daily was used as topical anti-inflammatory agent. The IOP normalized with treatment patient went for cataract operation with IOL implantation. During operation cataract was extracted with capsule, also anterior vitrectomy was done and SFIOL was implanted with I/V mannitol support. On 1st post operative day his VA was 6/60 and IOP was 07 mm of Hg in right eye. However,on 7th post operative day his VA was 6/12 and IOP was 12 mm of Hg. On 30th post operative day his VA was 6/12 and IOP was 14 mm of Hg without any anti glaucoma medication.

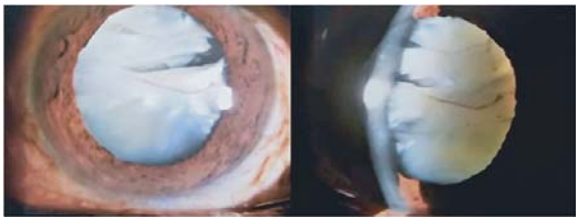

Fig. 1, 2: Dense white cataract with Rupture Capsule

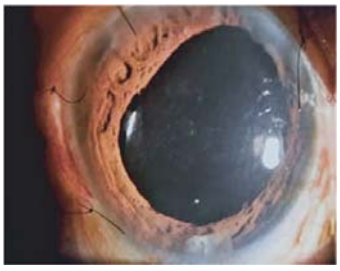

Fig. 3: 7th POD After cataract extraction and SFIOL implantation.

Discussion

Lens-related elevation of intraocular pressure (IOP) directed from variety mechanisms like lens dislocation, inflammation, lens swelling because of phacoanaphylexis or lens particle blocked tubercular meshwork. If increase IOP untreated the optic nerve damages mechanically, that lead to blindness inevitably. Increase IOP lead to compression and bowing lamina cribrosa backward, causes axoplasmic transport of retinal nerve fiber obstruction and ganglion cell death[3].

Slit lamp microscopy shows a hypermature cataract with ruptured capsule and deep anterior chamber. The cellular reaction in AC is variable, from cells and flare to an intense reaction. Large floating white particles consist of protein containing macrophages and lens protein may form a milky appearance to the aqueous. And gonioscopy finding shows an open angle with inflammatory cells and lens derived materials that are most substantial inferiorly[12].

Phacolytic glaucoma is the main complication of hyper mature cataract. Hypermature cataract may also leakage lens protein from intact capsule. Which causes inflammation or blockage tubercular meshwork and elevated IOP.[4] lens particles or proteins were derived from materials formed during early embryonic stage of development of eye. But release of this material at later age from ruptured lens is like as foreign body and form autoimmune granulomatous reaction[5].

Typically, phacolytic glaucoma is handled as an emergency. Elevated IOP medically controlled with topical aqueous suppressants (beta-blockers, carbonic anhydrage inhibitors andalpha-2 agonists), systemic carbonic anhydrage inhibitor and osmotic agent also given, if needed proteinaceous material wash out and cataract is removed[6].

Traumatic cataract or lens dislocations are most common sequel of ocular trauma[7]. Lens removal surgery should be done for abnormal lens position or post traumatic cataracts. Posterior capsular tear or zonular dehiscence are also commonly occurred in ocular trauma patients. Management of ocular trauma with lens dislocation or inadequate posterior capsule is very complicated.

Traumatic rupture of lens capsule is one of the main causes of phacolytic glaucoma and like this traumatic cause one should concern about the zonular state and take special technique during operation like SFIOL, capsular ring etc. Anterior chamber intraocular lens (ACIOL), iris fixed IOL are the alternative option of intraocular lens (IOL) implantation. It may be performed either primary or secondary procedure[8].

The iris-fixed IOL or ACIOL have various type of

complications like cystoids macular edema, endothelial cell decompensation, iris chafing, glaucoma escalation[9]. SFIOL implantation have some benefits like reduce peripheral anterior synechia, risk of corneal decompensation and secondary glaucoma[10,11].

Conclusion

Lens induced glaucoma is the important vision threatening disease which present as painful red eye. Early diagnosis and management mainly cataractous lens removal results in reduction of IOP and favorable visual outcome.

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C3F8 gas induced pupillary block glaucoma -A Case Report

F Yasmin[1], M S Naznin[2], S M Noman[3], B K D Sarker[4]

Abstract

Purpose: To describe management of gas induced pupillary block glaucoma. Method: Case Report.

Case Summary: A 34-year-old woman presented to the IIEI&H with the complaints of painless blurring of vision in her right eye for the last 2 months. Patient had a history of cataract surgery in his right eye one year back. On examination, her visual acuity was 1/60 for distance and N12 for near in right eye and in the left eye was 6/6 and N6. On fundoscopic examination, there was full thickness macular hole which was confirmed by OCT. Pars plana vitrectomy with peeling of the internal limiting membrane was performed under local anesthesia. Internal tamponade was achieved with C3F8 and 7 days face down posturing was instructed. Seven days later the patient came with acute pupillary block glaucoma secondary to gas bubble between iris and crystalline lens causing iridocorneal apposition. The IOP was 42 mm of Hg. We treated the patient with topical and systemic antiglaucoma medication. On the next day, The IOP still remained high and the iridocorneal apposition did not break down. We decided to do an inferior Nd:YAG laser peripheral iridotomy at 6 O' clock position. This peripheral iridotomy results in deepening of the anterior chamber and reduction of IOP to 18 mm of Hg. We stopped all the anti glaucoma medications and follow up the patient after three months, the anterior chamber was well formed with IOP 15 mm of Hg and vision improved to 6/24.

Conclusion: C_3F_8 migration into the anterior chamber, inducing a pupillary block glaucoma is a complication reported in pseudophakic eyes. An early Nd:YAG laser iridotomy is a suitable way to reduce IOP in such cases

Keywords: par plana vitrectomy, laser peripheral iridotomy.

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Introduction

Gas is widely used as vitreous substitutes and surgical adjuncts which is injected into the vitreous cavity to treat retinal surgeries. It is a valuable tool to treat macular hole and severe cases of vitreo retinopathy.[1] It greatly improves the anatomical and functional success rates of vitreoretinal surgery. The three most common types of gas substitutes like SF6, C3F8, C2F6 is widely used due to their innate properties. Gas mediated pupillary block and iridocorneal apposition are known complications in aphakic eyes that under-went vitreoretinal surgery.[2-4] We report a case of a pseudophakic patient with an acute pupillary block glaucoma due to the migration of perfluoropropane (C_3F_8) into the anterior chamber.

Our goal of this case report is to describe a successfully managed case of C3F8 gas induced pupillary block glaucoma.

Case report : A 34-year-old woman presented to the retina department of IIEI&H with complaints of painless, loss of central vision of the right eye for the last 2 months. The patient give history of cataract surgery in his right eye one year back. Her Ophthalmic examination revealed best corrected visual acuity was 1/60 for distance and N12 for near in right eye and 6/6 and N6 in the left eye. Both pupils were reacting normally to light, and intraocular pressure were normal. Dilated fundus examination of the right eye revealed full thickness macular hole(MH). Spectral domain OCT also revealed macular hole [Figure 1]. The patient was advised to undergo surgery for MH under local anaesthesia. Pars plana vitrectomy was performed with ILM peeling. Air fluid exchange done and Internal tamponade was achieved with using C_3F_8 and 7 days face down posturing was instructed.

On the first postoperative day, the anterior chamber was deep and there were no signs of raised IOP. Seven days later the patient came to the retina department with acute pupillary block glaucoma secondary to gas bubble between iris and crystalline lens causing iridocorneal apposition(Fig;2). The IOP was 42 mm of Hg. We treated the patient with topical and systemic antiglaucoma medication. We advised the patient to maintain strict prone position and reviewed on the next day. On the next day, the gas bubble reduced in size because most of the bubble moved behind the crystalline lens(Fig 3).The IOP still remained high and the iridocorneal apposition did not break down. We decided to do an inferior Nd:YAG laser peripheral iridotomy(PI) at 6 O' clock position. This PI results in the deepening of the anterior chamber and reduction of IOP to 18 mm of Hg. We follow up the patient after one month and three months, the anterior chamber was well formed with improved BCVA 6/24 and retina examination showed the macular hole had completely closed which is documented both clinically and by OCT [Figure 4], with IOP 15 mm of Hg without any anti glaucoma medications.

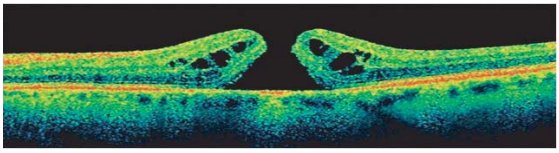

Fig1: Pre operative OCT showing full thickness macular hole.

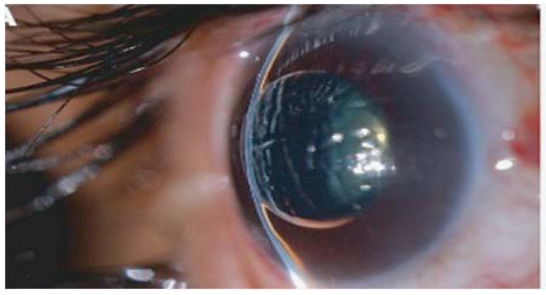

Fig2 : Photograph showing C3F8 gas in AC with complete iridocorneal apposition.

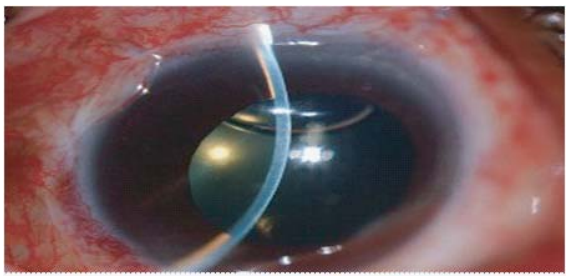

Fig3 : Photograph Showing gas reduced on the next day

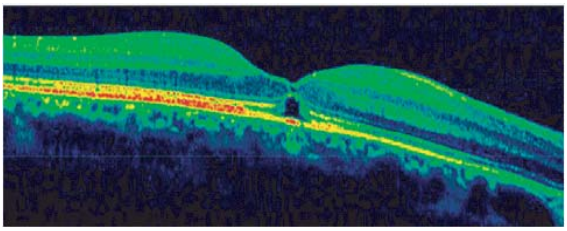

Fig4 : Follow up OCT(after 3 months) showing resolution of macular hole

Discussion

Elevated IOP is a common complication following pars plana vitrectomy with gas tamponade,[1]. Complete iridocorneal apposition with pupillary block in pseudophakic eyes due to expansion of intraocular gas.[2,5] The incidence of this condition was estimated to be 3% in a retrospective study of 336 aphakic eyes.[3] Buoyant forces from the bubble and pupillary block combine to push the iris forward onto the posterior surface of the cornea.

Zonulysis is the pathological change that allows gas migration from the posterior into the anterior chamber. Such changes are usually seen after trauma or surgery. Our patient had history of cataract surgery so, some part of the zonules might have been compromised by previous surgery.

Initially, our patient maintained a good prone positioning and encountered no problems; however, as he did not keep to the position, this probably resulted in the pupillary block by migration of C_3F_8 into the anterior chamber, thus pushing the iris diaphragm anteriorly. In our patient, Nd:YAG laser iridotomy at 6 o'clock was sufficient to treat the pupillary block.

Pars Plana vitrectomy in association with peeling

of the internal limiting membrane appears to provide good outcomes both anatomical and functional in cases of macular hole. However, the patient must be counseled regarding expectations following surgery, and warned about the long duration of recovery, early surgery appears to be a good management alternative for the condition.

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A Case of oculodermalmelanocytosis with secondary open angle glaucoma in a teenaged girl

T R Chhara[1], M Sababa[2], B K D Sarker[3], A R Khan[4], M I Iqbal[5]

Abstract

Oculodermalmelanocytosis (ODM) also known as"the nevus of Ota" and "congenital melanosis bulbi" is a rare condition associated with a number of sight-threatening complications including glaucoma. Open angle glaucoma is the commonest variant in these eyes though presence of angle closure should raise a suspicion of ciliary body or uveal tumor. Wereported an appearance of unilateral secondary open angleglaucoma in a young adolescent female patient with congenital ipsilateral nevus of Ota.

Keywords: glaucoma, hyperpigmentation, oculodermalmelanocytosis, ophthalmic features

Introduction

Oculodermalmelanocytosisis a benign mesodermal melanosis characterized by slate-greyhyperpigmentation or brown-blue discoloration of the facial areasupplied by the ophthalmic and maxillary division of trigeminal nerve as well as some ocular structures.[1] Japanese doctor Mokutaro Kinoshita Ota first described ODM in 1939 as a hamartoma of dermal melanocytes.[2] One hypothesis suggests that it is caused by failure of migration of melanocytes from neural crest cells to their normal location within the basal layer of the epidermis; whereas others emphasize previous radiation exposure, hormonal factors and genetic mutations as additional contributing factors.[3] The hyperpigmentation of the ocular tissues typically involves the conjunctiva, episclera, sclera, cornea, uveal tissues and angle of the anterior chamber. Others include retina, ocular muscles and orbit. Although considered as a sporadic congenital disorder, only half of the patients had skin pigmentation present at birth or shortly after. Sometimes, ODM appears as late as 20 years after birth.[4]

Nevus of Ota occurs predominantly in people of Asian and African descent.[7] The nevus of Ota is most frequent in Asian populations, with an estimated prevalence of 0.014%–0.034%. A study reported the incidence to be 0.038% in a Caucasian population, with a lower incidence in black population (0.014%).[6]

Tanino[5] graded the dermal hyperpigmentation in patients with the following conditions:

1. Type 1 with four subtypes: A (mild orbital type) distribution over the upper and lower eye lids and periocular and temple region; B (mild zygomatic type) pigmentation is found between the infrapalpebral and nasolabial fold and over the zygomatic region; C (mild forehead type), only forehead is affected; and D, alae nasi alone is affected.
2. Type 2 (moderate type): distribution over the upper and lower lids, periocular and zygomatic regions, cheeks and temple.
3. Type 3 (intensive type): the lesion involves the scalp, forehead, eye brow and nose.
4. Type 4: bilateral involvement.

Vishnevskia-Dai et al.[6] recently proposed an ocular classification of the disease based on the anatomical parts (conjunctiva, scleral, iris and choroid) of the eye involved and the extent of its quadrantal involvement. The eye is classified as 1 if only the surface of the eye (conjunctiva and/or sclera) is involved. If another ocular structure is involved with the surface, it is

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designated '+' for the surface involvement (surface and iris (2+), or surface and choroid (3+), or surface and iris and choroid (4+). The '+' designation is only given for surface involvement.

In our work, we report a case of Ota's nevus associated with secondary open-angle glaucoma which was diagnosed fifteen years after the appearance of typical blue-grayhyperpigmentation at birth.

Case report

A 15-year-old Bangladeshi girl presented to the pediatric ophthalmology clinic of our center with complaints of occasional headache and blurring of vision in the left eye. She was referred to our glaucoma department for raised intraocular pressure (IOP) in the same eye. There was no associated history of haloes and no previous ocular trauma or surgery. She also had a pigmented patch in the left side of her face since birth which had been increasing gradually in size over time. Review of systems was not contributory and there was no family history of similar disorder or any ocular illness or blindness.

Examination of the face showed blue-grey hyperpigmentation in the areas of distribution of ophthalmic and maxillary branches of the trigeminal nerve. Furthermore, there were hyperpigmented patches on the conjunctiva, episclera, sclera (Figure 1) however, no iris heterochromia was seen. This corresponds to type 1B as per Tanino's[5] classification and 2D+ according to the ocular classification described by Vishnevskia-Dai et al.[6]

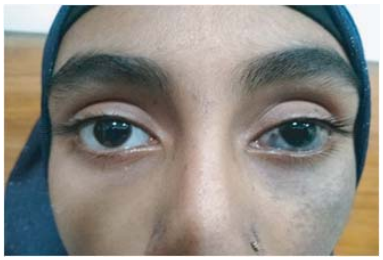

Figure 1: Slate-grey hyperpigmentation in the areas of distribution of ophthalmic and maxillary branches of the trigeminal nerve as well as hyperpigmentation in the leftconjunctiva and sclera.

At presentation, her best-corrected visual acuity was 20/20(Snellen 6/6) in the right eye and 20/32(Snellen 6/9) in the left eye. The IOP was measured in both eyes using a Goldmann applanation tonometer and was recorded 14 and 23 in the right and left eyes, respectively. The anterior segments were normal except the patchy discolorations. Gonioscopy revealed marked hyperpigmentation of the trabecular meshwork in the left eye and the angles were open in both eyes. Dilated fundus ophthalmoscopy demonstrated asymmetric "darker" pigmentation of the choroid layer beneath in the left eye, which was not present in the right eye fundus (Figure 2).Cup-to-disc ratio (C/D) in the right eye was 0.3, C/D was 0.7 in the left eye demonstrating glaucomatous damage to the optic nerve head (Figure 2).

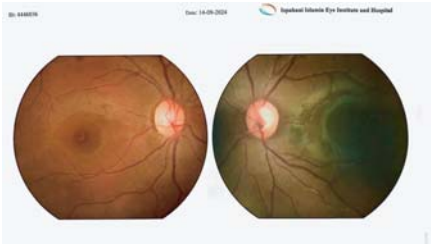

Figure 2 : Fundus photography of the left eye shows "darker fundus" (more pigment in the choroid). The right optic disc shows C/D 0.3 and the left optic disc shows signs of glaucoma (C/D 0.7).

OCT of the left eye reveals glaucomatous excavation of the head of the optic nerve with the relative borderline thinning of RNFL in the temporal part (Figure 3a and 3b).

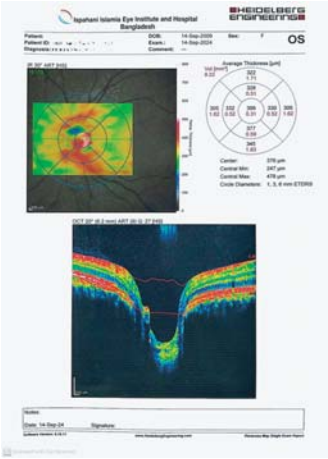

Figure 3a : glaucomatous excavation of the left optic nerve head

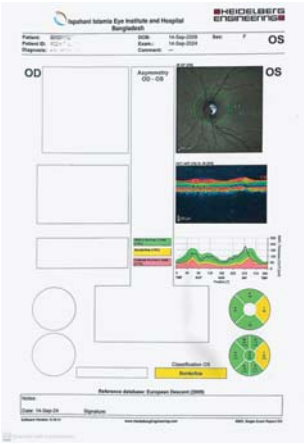

Figure 3b : OCT RNFL of the left eye showing relative borderline thinning in the temporal part.

We could not perform Humphrey visual field analysis due to age constraint. Careful examination showed no systematic changes associated with the nevus of Ota. We also excluded neurological or any other cause of the optic nerve head changes. She was planned with further follow-up scheduled for 6 months.

Discussion

The term Nevus of Ota is used only if dermal involvement is present, and the condition is termed melanosis oculi if only eye is involved. A study done on 194 patients with oculodermalmelanocytosis revealed dermal involvement alone was present in 67 (34.5%) patients, while 12 (6.2%) had only ocular involvement. The remaining 115 (59.3%) patients had both ocular and dermal pigmentation. ODM is associated with glaucoma in 10.3% of cases warranting a regular follow-up examination for glaucoma in these cases.[8] The occurrence of glaucoma in patients with the nevus of Ota may be related to the obstruction of aqueous outflow by accumulated melanocytes, but some authors suggest that their studies have not clearly shown that the Ota's nevus alone can cause the secondary glaucoma.Rutnin[5] reported male-to-female ratio of 1:3, while Shield et al.[9] reported 1:1.3. The female preponderance could be attributed to some genetic or hormonal factors.[4]This case showed appearance of ODM at birth. A previous study found that 87.5% of their patients had pigmentation at birth.[10] Like this case most cases of the nevus of Ota are unilateral (90%), although pigmentation is present bilaterally in 5%–10%. Ocular involvement in our patientincluded conjunctival, episcleral, scleral as well as trabecular meshwork. Previous studies revealed scleral involvement in greater than two-thirds of cases and is associated with an increased risk of developing glaucoma.[11,12] Associated ocular findings included elevated intraocular pressure with or without glaucoma (10.3%), uveitis (2.6%), cataract (1%), asymmetric cupping of the optic nerve head unassociated with glaucoma (9.8%), and orbital melanoma (0.5%).[8] A study on oculodermalmelanocytosis in 194 patients revealed glaucoma in 20 patients. Of the 20 patients, 15 developed ocular hypertension or open-angle glaucoma (70%), 3 patients had congenital or developmental glaucoma (15%), and 3 others had primary angle-closure glaucoma (15%).[8] Glaucoma is a known sequelae of ODM reported in various literatures and may develop at any age, So, patients with ODM and initially normal IOP should be examined at regular intervals.

Conclusion

The most serious complication of oculodermalmelanocytosis is malignant transformation, while glaucoma appears to be the more common one. Patients with oculodermalmelanocytosis and ocular hyperpigmentation should be followed up at regular intervals for the development of either of these complications. When the pigmentation of the chamber angle is intensive there is an increased risk of glaucoma associated with Ota's nevus, and the patient should be referred for periodic measurements of intraocular pressure andshould undergo a comprehensive ocular evaluation. It is however important to state that the true prevalence of ophthalmic sequelae of ODM may be difficult to determine as asymptomatic patients are not likely to present in the hospital. There is sparse information on the pattern of glaucoma presentation of ODM in

adolescent age group in South Asia hence this case report will raise the awareness regarding this oculocutaneous condition and will help in early diagnosis and management to prevent visual functionloss due to secondary glaucoma.

Conflict of Interests

The authors have no financial or proprietary interests related to this paper.

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