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should not exceed 100 characters.

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Novel Direct SLT is becoming a popular method of treatment for the early open angle glaucoma patients

M N Islam[1]

Direct Selective Laser Trabeculoplasty (DSLT) is an automated, transscleral, 1-second procedure that holds promise for increasing accessibility to IOP lowering treatment.

DSLT- (EAGLE BELKIN Laser), is being developed for its potential as a first-line treatment for ocular hypertension (OHT) open-angle glaucoma (OAG) and possibly for angle-closure glaucoma (ACG) that overcomes the limitations of current initial therapeutic options.

The non-invasive, non-contact procedure is performed with automated laser technology that delivers 100 spots to the trabecular meshwork through the limbus in just 1.2 seconds.

In the recent glaucoma meetings in the World Glaucoma Conference in Rome and also in the American Academy of Ophthalmology in San Francisco the DSLT was discussed with much interest and importance.

As we know Selective Laser Trabeculoplasty (SLT) is a laser procedure that is used to treat glaucoma. It is a simple yet effective outpatient laser procedure that is designed to lower intraocular pressure (IOP) associated with glaucoma. The procedure takes five to ten minutes and is performed by Glaucoma specialistsas an OPD procedure [1]. SLT works by applying laser energy to the drainage tissue (trabecular meshwork) located in the front of the eye. This is the natural drain for fluid. SLT stimulates the trabecular meshwork through a chemical and biological change to increase the amount of fluid drained from within the eye. Glaucoma doctors increasingly believe SLT should be the first treatment for glaucoma, replacing medicine and eye drops [1]. SLT lowers eye pressure by about 20 to 30 percent, on average, and is effective in lowering eye pressure in about 80 percent of people who opt for the treatment [1]. Recipients can expect to see noticeable results within three months of treatment[1].

SLT is a good option for people who have primary open-angle, pigmentary, pseudo exfoliation, or juvenile glaucoma[1]. If a patient is intolerant to glaucoma medications or have difficulty taking them as prescribed, they may be a good candidate for SLT[1]. If a patient is currently undergoing glaucoma drug therapy and wish to combine it with SLT, they may also be a good candidate for the procedure[1]. If it is difficult for a patient to commit to regular follow-up treatments due to lack of transportation, finances, or other limitations, SLT may be a good option for them[1].

Many studies have established that SLT is an appropriate first-line option for IOP-lowering, and most recently, results from the Laser in Glaucoma and ocular Hyper Tension (LiGHT) trial[2] showed that SLT was more cost-effective than medication.

SLT, however, is generally performed only by glaucoma specialists because it requires expertise with gonioscopy

According to Michael Belkin, inventor of DSLT, the aim is to provide a solution for addressing the growing worldwide burden of glaucoma-related vision loss.

The reality, however, is that even in areas in developed countries, there are not enough glaucoma specialists to meet the need for services, and the disparity between demand and supply is far worse in developing nations around the world.

DSLT can make laser treatment for IOP-lowering broadly accessible to patients around the world because it is performed without need for a goniolens or slit-lamp delivery system. Not only by Glaucoma specialists, any ophthalmologist or a trained optometrist will be able to do the DSLT laser[3].

Sir Peng T Khaw, professor of glaucoma and ocular healing, University College London and Moorfields Eye Hospital, London, described DSLT as exciting technology because of its convenience and ease of use.

Considering the results of the LiGHT trial and the promise that DSLT has shown in early investigation, DSLT seems poised to create a significant paradigm shift in the treatment of OHT and OAG[4].

Dr. Khaw also mentioned that The LiGHT trial showed that SLT worked better than medications as initial IOP lowering treatment and compared with conventional SLT, DSLT offers a potential method for delivering laser treatment more simply and to more patients.

Its potential impact is significant considering that glaucoma is the second-leading cause of irreversible blindness globally and that the number of people affected by this disease is expected to increase exponentially in the coming years.

Dr. Lindstrom, adjunct professor emeritus, Department of Ophthalmology, University of Minnesota, Minneapolis said that DSLT holds promise as an approach that can provide the benefits of SLT in a procedure that is much easier for both the physician and the patient. Based on its efficacy and safety, SLT is widely accepted as an appropriate alternative to medications as a first-line IOP-lowering treatment.

He also said DSLT brings the opportunity to expand the use of SLT by making it feasible for patients who otherwise might be considered poor candidates because they are uncooperative and in areas lacking the ancillary instrumentation and sophisticated personnel needed to perform traditional SLT[4]

DSLT is marketing by BELKIN Vision and we hope the machine will be available to Bangladesh soon and our glaucoma patients will have a new easy way of treatment.

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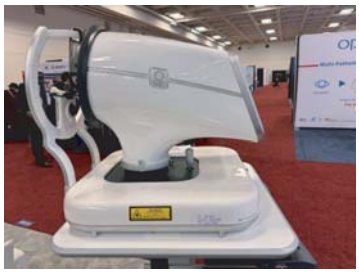

EAGLE DSLT by BELKIN Vision

EAGLE DSLT by BELKIN Vision

References

1. Garg A, Gazzard G.. Selective laser trabeculoplasty: past, present, and future. Eye. 2018; 32(5): 863–876.
2. Gazzard G, Konstantakopoulou E, Garway-Heath D, et al. Selective laser trabeculoplasty versus eye drops for first-line treatment of ocular hypertension and glaucoma (LiGHT): a multicentre randomised controlled trial. Lancet. 2019;393(10180):1505-1516.
3. Goldenfeld M, Belkin M, Dobkin-Bekman M, et al. Automated direct selective laser trabeculoplasty: first prospective clinical trial. Transl Vis Sci Technol. 2021;10(3):5.
4. Mordechai Goldenfeld, [1] Michael Belkin, [2] Masha Dobkin-Bekman, [3] Zachary Sacks, [3] Sharon Blum Meirovitch, [1] Noa Geffen, [4,5] Ari Leshno, [1,4] and Alon Skaat;Automated Direct Selective Laser Trabeculoplasty: First Prospective Clinical Trial; Transl Vis Sci Technol. 2021 Mar; 10(3): 5.

Highlighting Pigment Dispersion Syndrome Misdiagnosed as Acute Bilateral Anterior Uveitis

B K D Sarker[1], N L Islam[2], S Abarar[3], H Nazlee[4], S Rasal[5]

Abstract

Introduction: Clinical features of pigment dispersion syndrome(PDS) may be misdiagnosed with those of acute anterior uveitis. The purpose of this case series is to aid the ophthalmologist in the clinical differentiation between these two disorders.

Discussion: PDS may present with blurred vision, redness, ocular pain, and photophobia,all of which are also symptoms of acute anterior uveitis. These symptoms, plus the fact that pigmentfloating in the aqueous humor can be mistaken for inflammation, make diagnosis challenging. Moreover,the possible co-existence of true anterior uveitis and pigment dispersion makes the diagnosis and treatmentmore difficult.This study presents a series of 5 patients with pigment dispersion who were initially diagnosed as having acute anterior uveitis and treated with anti-inflammatory medication, including corticosteroids. The patients were referred for a second opinion due to poor or no response to therapy and were found to have pigment dispersion instead of uveitis.

Keywords: pigment dispersion syndrome, pigmentary glaucoma, uveitis, masquerading syndrome, acute anterior uveitis.

Introduction

Ocular pigment dispersion may be seen in the general population. During aging process a variable amount of uveal pigment is chronically released and dispersed into the anterior ocular segment[1]. As might be anticipated, pigment dispersion may also be found among individuals with various forms of glaucoma, although pigment in most of these patients is not believed to have a major role in the pathogenesis of the disease[1,2], However, there are several ocular conditions that are associated with an unusual, heavy dispersion of pigment that might significantly increase resistance to aqueous outflow with secondary elevation of the intraocular pressure and glaucomatous neuropathy. This condition is known as pigmentary glaucoma[2,3].

Pigment dispersion syndrome (PDS) is a distinct clinical entity characterized by an acute pigment scattering from the pigment epithelium of the iris and/or the ciliary body. Pigment dispersed in the anterior segment usually deposits on corneal endothelium, trabecular meshwork, iris, and lens.

Patients with PDS may present with a transient elevation of intraocular pressure[1,4,5]. Patients with an acute attack of pigment dispersion usually suffer from bilateral red eye, moderate to severe pain, photophobia, and blurred vision. These symptoms easily resemble those of an acute attack of anterior uveitis (AAU). Moreover, ciliary injection and pigmented particles floating in the anterior chamber may be confused with inflammation and so might easily lead to misdiagnose as AAU.

The purpose of this case series is to aid the general ophthalmologist in the clinical differentiation between a truly acute anterior uveitis episode and that of pigment dispersion syndrome, as well as to aid in the recognition of when these syndromes overlap.

Case Description

Case 1

A 32-year-old female was referred to our clinic with a 1-month history of persistent bilateral anterior uveitis. The patient was first seen by a general ophthalmologist for ocular pain, redness, and severe photophobia in the right eye; 10 days later the same symptoms appeared in the left eye. She was diagnosed as having AAU and was started on dexamethasone eye drop every hour and homatropine 2% eye drop every 6 h with no response to therapy. At that time, diagnostic investigation was negative. She reported that she was told that cells did not

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decrease in spite of topical corticosteroid therapy. When first seen in our clinic, she continued with the same, significant symptoms. On examination, her visual acuity was 6/9 OD and 6/12 OS.

Intraocular pressures were 25 mmHg OD and 31 mmHg OS. Slit-lamp examination disclosed a Krukenberg spindle (Figure 1), scant pigment deposition over the anterior lens capsule, and 1+ pigment floating in the anterior chamber OU. Irides were slightly concave with moderate peripheral stromal atrophy and irregular pupillary contour no transillumination defects were seen OU.Gonioscopy, while on cyclopegics, revealed an open angle with heavy pigment deposition on the trabecular meshwork, especially on the inferior quadrants. Dilated fundus exam was normal OU.

Based on ocular findings, pigment dispersion syndrome with secondary ocular hypertension was diagnosed. She was asked to stop topical steroids and cycloplegics; she was also started on timolol maleate 0.5% twice a day OU, brinzolamide three times a day OU, and acetazolamide 250 mg twice a day. One week later, most symptoms had disappeared. Trace pigment floating in the anterior chamber OU was noted as well as a decrease in intraocular pressures to 15 mmHg OD and 17 mmHg OS. The patient continued on ocular hypotensive medications for 2 months before stopping them. At her last visit, 12 months after initial presentation, no pigment dispersion was noted, and intraocular pressure had remained 15 mmHg OU without medication.

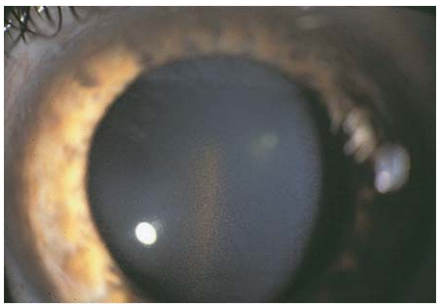

Figure 1: Showing diffuse pigmentation on corneal endothelium with Krukenberg spindle

Case 2

A 33 year-old female came to our clinic with a 2-week history of bilateral blurred vision, extreme photophobia, and redness of abrupt onset. The patient was previously diagnosed as having bilateral acute anterior uveitis (AAU). All laboratory workup performed at that time was unrevealing, and the patient was treated with prednisolone acetate 1% every 3 hour, and tropicamide 1% four times a day with no response or improvement in symptoms.

She was referred to our hospital for diagnosis and treatment of her AAU. On examination, visual acuity was 6/6 in both eyes (OU), and intraocular pressures were 15 mmHg OD, and 11 mmHg OS. The patient had 1+ ciliary injection OU. Slit-lamp examination disclosed 1+ pigment granules in the anterior chamber, papillary contour irregularity, and midperipheral iris transillumination defects OU.

Gonioscopic exam showed an open angle with moderate pigment accumulation on the trabecular meshwork (figure 2), in the inferior quadrant OU.

The patient was diagnosed as having pigment dispersion syndrome, and no additional diagnostic workup was performed. She was asked to stop all medications and advised to continue steroid for 2 weeks 2 times daily. After a 5-month follow-up period, the patient was asymptomatic, intraocular pressures remained normal, and no further pigment dispersion was detected.

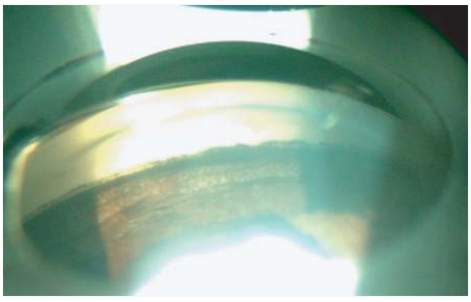

Figure 2: Showing an open angle with pigment accumulation on the trabecular meshwork

Case 3

A 36-year-old male was seen for a 15 day history of redness, ocular pain, photophobia, blurry vision, and tearing, starting in the left eye and followed almost immediately by the right

eye. The patient was diagnosed with bilateral AAU. He was started on prednisolone acetate 1% every hour, his symptoms worsened over time, with development of severe photophobia, intense ocular pain, and decreased vision.

The patient was told that he had persistent inflammation with no improvement to therapy. The patient decided to look for a second opinion. We found visual acuity of 6/9 OD and 6/24 OS. Intraocular pressures were 25 mmHg OD and 31 mmHg OS. At slit-lamp examination, there was 1+ ciliary injection, 2+ Krukenberg spindle, scant pigment deposition over the anterior lens capsule, and 2+ pigment floating in the anterior chamber OU; a slight concavity of the irides was also noted. Gonioscopy showed heavy pigment deposition on the trabecular meshwork in all quadrants OS > OD(Figure 3).

The patient was diagnosed as pigment dispersion syndrome with secondary ocular hypertension OU. All previous medications were stopped and the patient was started on timolol maleate 0.5% twice a day and brinzolamide three times OU, and acetazolamide 250 mg three times a day. Five days after this therapy was commenced the intraocular pressures were 12 mmHg OU. Three weeks after initial visit no pigment dispersion was seen in either eye, visual acuity was 6/9 OU, and IOP was 12 mmHg OU. Irregular pupils and extensive midperipheral transillumination defects OU were noted. After several months the patient achieved a best-corrected visual acuity of 6/6 OU and IOP remained normal; however, the patient still complained of moderate photophobia, especially while exercising.

Figure 3: Gonioscopy showed heavy pigment deposition on the trabecular meshwork in all quadrants

Case 4

A 35-year-old female was referred to our clinic with a 1-month history of ocular pain, photophobia, and redness OU. The patient recalled that symptoms began OS, followed by OD weeks after. She stated that headache and ocular pain were variably present. No other signs or symptoms were positive in the complete clinical history and physical exam. She was previously treated with, brinzolamide hydrochloride 2% twice a day, and dexamethasone 0.1% every 2 h alternating with loteprednol etabonate 0.5% every 2 h.

Best corrected visual acuities were 6/9 OD and 6/6 OS. Intraocular pressure was 14 mmHg OU. Remarkable findings OD were 1+ ciliary injection 1+ cells and 1+ pigment in the anterior chamber, and 2+ flare (figure 4). The left eye showed 1+ ciliary injection, trace cells and pigment in anterior chamber, and 1+ flare. No posterior or peripheral anterior synechiae were found in either eye. Lens and anterior vitreous were clear without relevant findings. Dilated fundus exam in both eyes revealed no abnormalities.

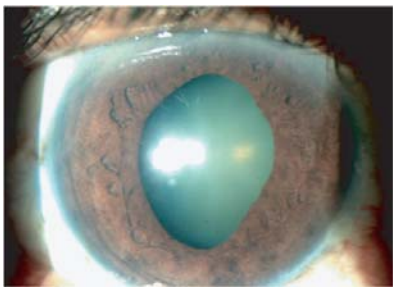

Figure 4: Presenting as uveitis with dilated pupil due to use of cycloplegic

The patient was diagnosed with diffuse anterior scleritis OD and bilateral anterior uveitis. During the following days, cycloplegics were stopped. Two days later, she presented to our clinic with visual acuities of 6/9 OU. IOP was 14 mmHg OD and 13 mmHg OS. Anterior segment examination was remarkable for Krukenberg spindle OU, 1+ pigment clumps in the anterior chamber, trace cells, and no flare in both eyes. Gonioscopy revealed slightly pigmented trabecular meshwork. A diagnosis of overlapping pigment dispersion syndrome and anterior uveitis was made. A very slow tapering of steroids and oral NSAIDs was performed, achieving no inflammatory activity within 3 months. At her last visit, her best-corrected visual acuity was 6/6 in both eyes. At slit-lamp examination no anterior inflammatory activity, no pigment floating, no iris transillumination defects, and no pupillary irregularities were found.

Case 5

A 32-year-old male was presented severe ocular pain, photophobia, redness, and blurred vision, starting in the left eye followed by the right eye 3 days later. The patient was initially diagnosed as having acute bilateral anterior uveitis. He was given atropine 1% once a day OU, and dexamethasone phosphate 0.1% every 3 h. Laboratory workup for uveitis was unrevealing.

During the acute phase, intraocular pressures rose to 32 mmHg OD and 31 mmHg OS.

Because no response to treatment was observed during a period of 3 weeks and anterior uveitis was noted, the patient was referred to our clinic for a second opinion and treatment.On first visit, the patient had a 2+ ciliary injection, 3+ pigment floating in the anterior chambers,and 4+ prominent Krukenberg spindle OU. Gonioscopy showed 360° heavy pigment deposition on the trabecular meshwork of both eyes, especially in the inferior quadrants. Postdilation gonioscopy was not documented. Intraocular pressures were 32mmHg OD and 33 mmHg OS. No optic nerve cupping was observed during dilated fundus examination.

The patient was diagnosed as having pigment dispersion syndrome with secondary ocular hypertension OU.All medications were stopped, and the patient was started on pilocarpine 2% three times a day OU, timolol maleate0.5% twice a day OU, and acethazolamide 250 mg three times a day orally. He was also asked to reduce physical activity to a minimum. After a period of 3 weeks pigment dispersion subsided, IOP dropped to 13 mmHg OD and 14 mmHg OS, and all symptoms disappeared.

Discussion

The PDS is an autosomal dominant disorder with phenotypic onset beginning in most persons in mid-20s[7]. The disorder is characterized by disruption of iris pigment epithelium and deposition of pigment granules on the anterior segment structures. The incidence of PDS is 4-8 per 100,000 population. This condition is more commonly seen in Caucasians and is considered to be rare in Indians [8]. PDS is typically a bilateral disease, although asymmetry may occur. Men and women are equally affected by PDS. Men are significantly younger than women at the time of diagnosis of the disease.

About 60% - 80% of patients are myopes and 20% are emmetropes[5]. The syndrome is characterized by the triad of deposition of pigment on the posterior corneal surface in a vertical line (Krukenberg's spindle), wide open angles on gonioscopy with uniform and heavy pigmentation of trabecular meshwork, slitlike radial transillumination defects in the iris. Other ocular findings commonly observed are: relatively flat cornea, deep anterior chamber and wide open angles.[2] The iris is inserted posteriorly and shows a concave configuration in the mid periphery. This feature is best demonstrated by ultrasound biomicroscopy (UBM) or anterior segment OCT[8,9].

Pigment accumulation on the anterior surface of the iris often appears as concentric rings within the iris furrows. Pigment is also deposited on the posterior surface of the lens in the region of contact between the anterior hyaloids face and posterior lens capsule. Visualization of this circular ring or arc of pigmentation requires pupillary dilatation and is considered pathognomonic of PDS[8]. Patients with PDS and pigmentary glaucoma are at increased risk for retinal detachment and this may occur in 6%-7% of individuals. Retinal breaks and retinal detachment may occur twice frequently in these eyes[9].

One of the main reasons that PDS masquerades as acute anterior uveitis is that pigment granules mimic inflammatory cells floating in the anterior chamber[8]. The wide spectrum of clinical presentation of patients with PDS varies from silent dispersion and ocular hypertension to symptoms resembling those of an acute anterior uveitis attack such as blurred vision, redness, ocular pain, and extreme photophobia[9,10]. In this report of 3 cases of bilateral acute depigmentation of the iris, the main symptoms were a sudden onset of ocular discomfort and red eye, with or without ciliary injection and Krukenberg spindle[11] .

These overlapping symptoms make misdiagnosis even more probable. Persistent anterior inflammatory cells in spite of steroid therapy should prompt one to consider pigment dispersion as a possible diagnosis. The presence of a Krukenberg spindle, pigmentation of the trabecular meshwork, or backward bowing of the iris is highly suggestive of this pathology.

The diagnosis of PDS is even more challenging

when the patient presents on topical steroids, rising the questionable presence of previous inflammation or an overlapping syndrome. However, little or no response to topical, regional, or systemic steroids,associated with no improvement or worsening of ocular symptoms and signs, should raise a suspicion of a masquerade syndrome. Moreover, the rapid response and significant improvement in inflammation after discontinuing anti-inflammatory therapy and initiating PDS

therapy is confirmatory of the diagnosis.

On the other hand, it is remarkable that two clinical entities might be present in the same patient. In the case of patient 5, all clinical features of pigment dispersion syndrome overlapped with a typical presentation of recurrent, alternating iridocyclitis. In this particular case,it is interesting that in some visits the patient presented only with pigment dispersion without ocular hypertension compared to other visits when mild, obvious anterior uveitis with no pigment dispersion was present. Thus, we believe these entities in fact can overlap, making a definite diagnosis even more difficult.

The management of PDS depends on the severity of the pigment dispersion and the association with secondary ocular hypertension or glaucoma. Miotics like pilocarpine have been recommended for limiting pigment dispersion as they are thought to reduce iridolenticular contact and friction by changing iris configuration[12].

In summary, therapy in patients with pigmentary ocular hypertension or glaucoma must be individualized, considering that aggressive therapy might be necessary. If target ocular pressures are not achieved with medical therapy, laser trabeculoplasty and filtering surgery might be of help.

Conclusion

Pigment dispersion syndrome can lead to severe visual field loss and poor quality of life. Therefore, it is very important to consider the diagnosis of PDS in any young patient presenting with symptoms suggestive of acute anterior uveitis. A detailed ophthalmological examination, including gonioscopy, is mandatory to make the correct diagnosis and to avoid the need for extensive laboratory workup that eventually proves to be useless.

It also will eliminate inappropriate use of corticosteroids that, apart from not resolving the condition, further complicate the clinical scenario, either by causing secondary ocular hypertension and glaucoma or by inducing secondary cataract formation. The case series also highlights the importance of looking for subtle signs like phacodonesis and iridodonesis which may be due to increased axial length of the eyeball or may be an associated feature of PDS.

References

1. Scheie HC, Gameron JD. Pigment dispersion syndrome: a clinical study. Br J Ophthalmol. 1981;65:264–269.
2. Speakman JS. Pigmentary dispersion. Br J Ophthalmol 1981;65:249-51.
3. Potash SD, Tello C, Liebmann J, Ritch R. Ultrasound biomicroscopy in pigment dispersion syndrome.Ophthalmology 1994;101:332-9.
4. Chew SJ, Tello C, Wallman J, Ritch R. Blinking indentsthe cornea and reduces anterior chamber volumeas shown by ultrasound biomicroscopy. InvestOphthalmol Vis Sci 1994;35(suppl):1573.
5. Krishnadas R, Ramakrishnan R. Secondary glaucomas: the tasks ahead. Community Eye Health2001;14:40-2.
6. Zentmayer W. Association of an annular band ofpigment on posterior capsule of lens with aKrukenberg spindle. Arch Ophthalmol1938;20:52-7.
7. Liebmann JM, Tello C, Chew SJ, Cohen H, Ritch R.Prevention of blinking alters iris configuration inpigment dispersion syndrome and in normal eyes.Ophthalmology 1995;102:446-55.
8. Campbell DG. Pigmentary dispersion and glaucoma:a new theory. Arch Ophthalmol 1979;97:1667-72.
9. Vitale A, Foster S. Diagnosis and Treatment of Uveitis. Philadelphia: WB Saunders; 2002.
10. Nussenblatt RB, Whitcup SM, Palestine AG. Uveitis: Fundamentals and Clinical Practice, 2nd ed. St Louis, MO: Mosby; 1996:385–396.
11. Gillies WE, Brooks AMV. Clinical features at presentation of anterior segment pigment dispersion syndrome. Clin Exp Ophthalmol. 2001;29:125–127.
12. Campbell DG. Pigmentary dispersion syndrome: a new theory. Arch Ophthalmol. 1979;97: 1667–1672.
13. Karickhoff JR. Reverse pupillary block in pigmentary glaucoma: follow up and new developments. Opthalmic Surg.1993;24(8): 562–563.

Original Article

Atypical Disc. Identified in the glaucoma clinic

M A Karim[1], S M Noman[2], S R Roy[3]

Abstract

Purpose : To document and describe different patterns of non-glaucomatous atypical discs and to identify their other ocular and systemic associations.

Design : A hospital-based prospective observational case series done during the period from 1st November 2009 to 31st October 2011.

Method : Patient particulars, history and complaints were recorded. Ophthalmic examination included visual acuity, refraction, slit-lamp examination, pupillary reaction, tonometry, gonioscopy and funduscopy. The relevant investigations were done and documented. Similar relevant details were recorded on each follow-up.

Results : 30 patients were included in this case series. 13 patients were female, 12 were male. 5 patients were diagnosed as myelinated optic nerve head in both eyes, 5 patients had bilateral titled discs without refractive error; 4 patients had unilateral optic disc pit; 4 patients had bilateral optic disc drusens, 3 patients had bilateral disc coloboma, 2 patients had morning glory disc, 1 patient had optic nerve hypoplasia, 5 patients had macrodisc and 1 patient was diagnosed with post-traumatic optic nerve head avulsion.

Conclusion : Proper ophthalmic examination and investigations are essential for the diagnosis of atypical discs and congenital anomalies of the optic disc. Documentation not only helps clinicians in their clinical practice but also helps in teaching, patient counseling and planning for rehabilitation.

Introduction

The optic nerve begins anatomically at the optic disc but physiologically and functionally from the ganglion cells that cover the entire retina. It is a collection of 1.1–1.2 million ganglion cell axons that traverse the sclera through lamina cribrosa. The optic nerve develops from the neural tube (neuroectoderm). Due to developmental anomalies, infection and trauma there is damage to the ganglionic cells, as well as nerve fibres, which in turn affects the patient's visual acuity and visual field. A normal disc is 1.5 mm in diameter, has a 0.3:1 – 0.5:1 cup disc ratio and a healthy neuroretinal rim.

Traumatic, inflammatory and vascular toxic optic disc abnormalities are not uncommon in this subcontinent but atypical optic disc changes like congenital optic disc anomalies are very rare and usually these patients remain undiagnosed and without rehabilitation. With accurate diagnoses at early age, this can aid us in identifying associated ophthalmic and systemic anomalies so that treatment and rehabilitation can be started.

Methods

This is a hospital-based combined non-concurrent and concurrent prospective case series. Cases were identified throughout a two-years period. All patients were referred from the outpatient department of the Chittagong Eye Infirmary and Training Complex (CEITC). Details of history included maternal and birth history, biographical details and family history. History of trauma was ascertained. Ophthalmic examination was done on all patients and examination details included visual acuity, tonometry, gonioscopy, indirect ophthalmoscopy and optic disc photography. B-scan ultrasonogram was done if necessary.

Results

A total number of 30 patients with atypical optic discs were included in this case series. Amongst them, 29 cases were either congenital or developmental in nature. 1 case was traumatic.

Myelinated Optic Nerve

Of the 5 myelinated optic nerve cases, 3 were

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female and 2 were male. Symmetrical involvement was observed in these cases. No other ocular and systemic associations were present in these cases. In all cases, visual acuity was normal. 2 cases had peripapillary myelination and 3 cases had myelination along the superior arcuate area. On Humphrey's Visual Field (HVF) analysis 4 cases showed corresponding visual field loss such as enlargement of blind spot and arcuate scotomas.

Bilateral Tilted Discs

There were 5 bilateral titled disc cases. 3 were female and 2 were male. They all had normal visual acuity with no refractive errors. They all had small, oval and D-shaped discs that were tilted inferior-nasally.

Optic Disc Pit

4 optic disc pit cases were found; 3 were female and 1 was male with an average age of 40 ± 10 years. Their visual acuity was normal with no refractive errors. HVF analysis revealed caecocentral scotomas in 2 cases. Neither macular oedema nor serous macular detachment was observed in any of these cases. The pit was present in the temporal aspect of the disc.

Optic Disc Drusen

4 patients were diagnosed with optic disc drusen. Of these, there were 3 female and 1 male. All cases were bilateral with minimal disturbance in visual acuity (6/9 – 6/12). 2 cases revealed an enlargement of the blind spot on HVF analysis. All cases had some common features such tortuous blood vessels, increased blood vessels, scalloped disc margin and refractile bodies over the disc. B-scan ultrasonography revealed hyperacoustic shadows in optic nerve head suggestive of calcification.

Optic Disc Coloboma

In the optic disc coloboma cases, all were male with an average age of 50 ± 10 years. 2 of them had isolated disc colobomas and 1 was associated with retino-choroidal coloboma. Visual acuity was diminished in all of the affected eyes (6/60–CF).

Morning Glory anomaly

Of the 2 morning glory cases 1 was male and 1 was female. Visual acuity was impaired in both cases. Typical signs of morning glory seen were seen in both cases (such as large optic discs, funnel-shaped excavation, central glial tissue and spoke-like blood vessel configuration).

Optic Nerve Hypoplasia

There was one patient of optic nerve hypoplasia who was a boy of 6 years. It was a bilateral case in both optic disc and macular hypoplasia. Visual acuity was diminished in both eyes.

Macrodisc

5 macrodisc cases were found (1 female and 4 male) with an average age of 30 ± 10 years. All of them had normal visual acuities without refractive errors. Normal IOP and normal visual fields were observed in all cases. Average cup:disc ratio was 0.6 ± 0.2.

Optic Nerve Avulsion

There was a single case of optic nerve avulsion which had a history of trauma and loss of vision for 2 days.

Discussion

The first portion of the optic nerve contains the nerve fibres of 1 to 1.2 million ganglionic cells that traverse the sclera through the lamina cribrosa. Developmentally, the optic nerve is neural tube derivative. The ganglionic cells of the retina develop axons, to a point where the optic nerve leaves the posterior surface of optic cup. Developmentally this will become the optic disc. In the inferior aspect of the optic stalk, normally there is a groove or fissure which contains mesenchymal tissues and the hyaloid atrery. This fissure becomes the optic canal by fusion at the seventh week of gestation. Failure of the fissure to close completely results in coloboma formation in the retina, choroid and also in the optic nerve. The cells of the optic stalk form neuroglial supporting cells for the axons, and the cavity of the stalk disappears. The stalk together with the ganlionic cell axons, form the optic nerve. The axon of the optic nerve begin to develop their myelin sheaths just

before birth, but the process of myelinetion continues for sometime after birth.

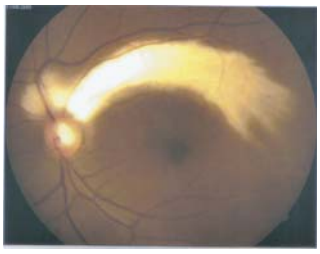

Figure 1: Myelinated optic disc involving superior arcuate area.

In myelinated optic disc, the optic disc is surrounded by bright white streaky and irregular patches. The myelination may extend to the periphery or cover part of the disc. It may be unilateral or bilateral with asymmetrical involvement. Myelinated nerve patches are often seen in the arcuate bundles occasionally abutting the disc. When they are contiguous, these nerve patches may be confused with disc oedema or cotton wool spots[1] (Figure 1).

Of the 5 myelinated optic nerve cases, 3 were female and 2 were male. Symmetrical involvement was observed in these cases. No other ocular and systemic associations were present in any of the cases. In all cases visual acuity was normal. In 2 cases peripapillary meylination was present and in 3 cases meylination was along the superior arcuate area. On HVF analysis, 4 cases showed corresponding visual field loss such enlargement of blind spot and arcuate scotomas. Usually with extensive nerve fibre myelination, myopia, anisometropia and amblyopia are associated. Neurofibromatosis-1 and multiple basal cell naevus are some systemic associations. Neither systemic nor ocular associations were found in our series.

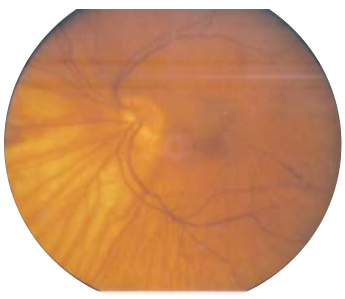

Figure 2: Titled optic disc.

Tilted discs are usually bilateral. They are caused by a developmentally oblique entry of the optic nerve into the globe. For this, one portion of the neuroretinal rim is elevated and the other is depressed. Usually it looks D-shaped and is directed inferonasally. The disc margin may be indistinct and temporal vessels deviate nasally before turning temporally. The visual field may show a superior temporal field defect[2]. Visual acuity and visual field were normal in all of our cases. Titled discs positioned inferonasally was present in all cases. Temporal elevation of the neuroretinal rim with nasal shifting of temporal vessels were observed in all cases (Figure 2).

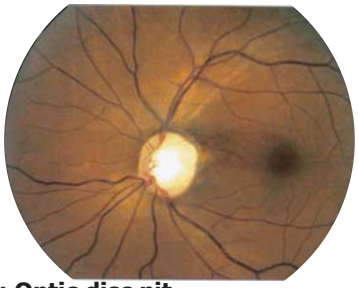

Figure 3 : Optic disc pit.

Optic disc pit is a depressed area within the optic nerve head and usually lies temporally. Visual acuity is normal in the absence of complications. Visual filed defects are common and mimic glaucoma. Serous macular detachment develops in about 45% of eyes with non-central disc pits. The subretinal fluid is thought to be derived from the vitreous. Less likely sources are the subarachnoid space and from abnormal vessels within the base of the pit.[2] Visual acuity was normal in all of our cases. In 2 cases caecocentral scotomas were detected on HVF examination. Discs were normal in shape, no subretinal fluid nor serous macular detachment was observed in our cases (Figure 3).

Optic disc drusen are composed of hyaline-like calcific material within the optic nerve head. They are often bilateral. The buried drusen cannot be seen over the surface. Elevated disc, hyperaemia and early branching of the blood vessels are characteristics of buried drusens. Exposed drusens are usually seen over the surface of the disc. The drusen may be

associated with angioid streak. They may also be associated with retinal bleeding.[3] All of our cases were bilateral with minimal disturbance in visual acuity (6/9-6/12) and with no improvement with pinhole. 2 cases revealed enlargement of blind spot on HVF analysis. In all cases, there were some common features like tortuous blood vessels, increased blood vessels, scalloped disc margin and retractile bodies all over the disc. B-scan ultrasonography revealed hyperacoustic shadows in the optic nerve head suggestive of calcifications (Figures 4a and 4b).

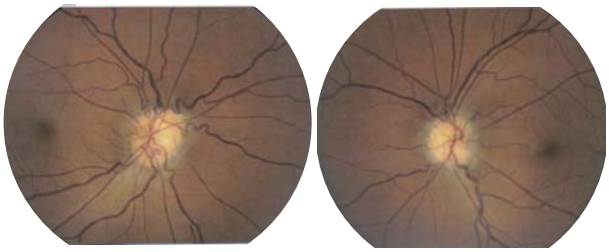

Figure 4a : Optic disc drusen

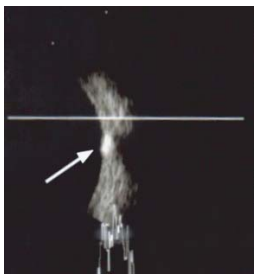

Figure 4b : Hyperacoustic shadow of drusen in optic disc.

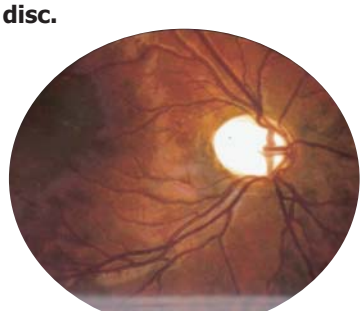

Figure 5: Optic disc coloboma

Optic disc coloboma is a developmental defect due to non-closure of optic fissure at the 7th week of gestation. It may be associated with retinochoroidal coloboma. Sometimes it looks like duplication of the optic disc and is called pseudo duplication[4]. Visual acuity is often decreased. A bowl-shaped excavation (decentered inferiorly) and the remaining superior portion of neuroretinal rim are the clinical features of optic disc coloboma. Perimetry usually shows superior visual field defect. It may be associated with microphthalmos and retinochoroidal coloboma. Complications such as serous retinal detachment and peripapillary choroidal neurovascularization may occur. Some chromosomal anomalies and CHARGE association (heart defects, choanal atresia, retarded growth and development, genital and ear anomalies) are associated systemically. In our 3 optic disc coloboma cases, 2 were isolated disc colobomas and 1 was associated with retinochoroidal coloboma. Visual acuity was diminished in all affected eyes (6/60–CF). The coloboma involved almost the entire disc except for a tiny superior neuroretinal rim. There were no systemic associations in any of the cases. HVF examination revaeled a caecocentral scotoma in 2 cases and an arcuate scotoma-like defect in 1 case which was associated with retinochoroidal coloboma (Figure 5).

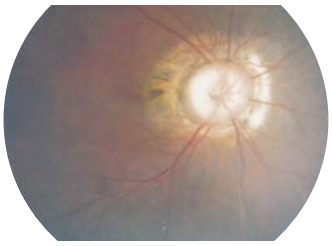

Figure 6: Morning glory anomaly

Morning glory is a unilateral and rare case which affects women more than men. Patients may present with poor vision or strabismus. A funnel-shaped excavation of the optic disc surrounded by a depigmented area, centrally elevated hyperplastic glial tissue and normal straight blood vessels radiating from disc margin are the characteristics of the morning glory anomaly. Non-rhegmatogenous retinal detachment may be associated as a complication with this disease.

Frontonasal displasia that includes mid-facial abnormalities with hypertelorism, basal encephalocele and the absence of corpus callosum are the systemic associations.[5] In our cases one was male and one was female. Both of them presented with severely depressed visual acuity (CF). Both of them were unilateral

and all of the features of morning glory anomalies (such as large optic disc, funnel-shaped excavation, central glial tissue and spoke-like blood vessels were present). No other ocular and systemic associations were found in our cases (Figure 6).

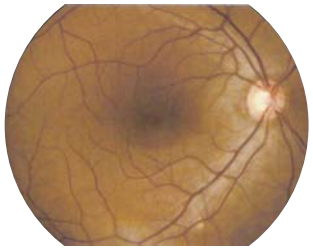

Figure 7: Optic nerve hypoplasia

Optic nerve hypoplasia is a condition that results in under development of the optic nerve. The optic disc appears abnormally small due to immature and decreased optic nerve axons. It may be unilateral or bilateral. Maternal use of alcohol, quinine, protamin zinc insulin, steroids, diuretics, cold remedies and anti-convulsants may cause such a defect. Blindness in early infancy in bilateral cases and squint in unilateral cases are also associations.[2] In our single case, visual acuity was very poor since birth and later esotropia developed gradually. Macular hypoplasia was diagnosed in both eyes. No other ocular nor systemic associations were found. No history of maternal adverse drug use during pregnancy was observed. (Figure 7).

Developmentally bilateral large discs (more than 1.5mm) is not very uncommon. There is an increased cup:disc ratio that resembles glaucomatous optic disc. Optic neuropathy

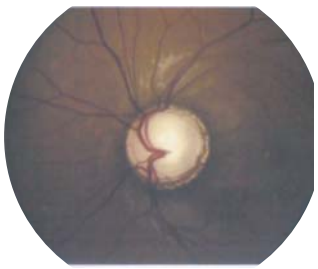

Figure 8: Congenital macrodisc

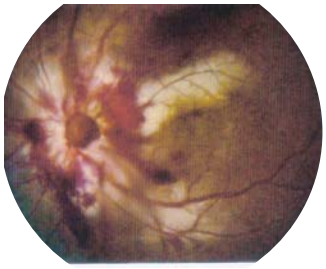

Figure 9: Optic nerve avulsion

looks like normal pressure glaucoma in teenagers with congenital macrodisc.[6] In our macrodisc cases, all of them had normal visual acuity without refractive errors. Intraocular pressure and visual field analysis were normal. The average cup:disc ratio was 0.6 ± 0.2 (Figure 8).

Optic nerve avulsion is caused by severe blunt trauma which is associated with loss of vision, optic disc and retinal hemorrhage and total afferent pupillary defect. Our patient had a history of severe blunt trauma with loss of vision, intra-retinal hemorrhage and restricted ocular motility due to entrapment of muscle within a blow-out fracture (Figure 9).

Conclusion

Different types of optic nerve abnormalities like developmental, inflammatory, traumatic, idiopathic and vascular can be found in clinics. Proper history taking and appropriate examination (like pupillary reaction, visual field analysis, direct or indirect ophthalmoscopy) are very important for the diagnosis of atypical discs. After diagnosis, proper evaluation, counseling and rehabilitation are mandatory.

References

1. Antony C. American Journal of Ophthalmology 2002.
2. Jack J Kanski. Clinical Ophthalmology 2007;6: 82-83.
3. Rubin Stein K. Retinal Complications of Optic Disc Drusen. Br. J. Ophthalmol. 1982; Feb 66(2): 83-95.
4. Islam N. Optic Disc Duplication or Coloboma. Br. J Ophthalmol. 2005 ;Jan 89(1): 20-29.
5. Edward S Harkness Eye Institute, Colombia University, USA.
6. Joast B. Optic neuropathy resembling normal pressure glaucoma in congenital glaucoma. Arch Ophhtalmol. 1998; Oct 116(10): 1384-1386.

Original Article

Awareness and Knowledge about Glaucoma among Patients with or without Glaucoma Diagnosis Attending a Tertiary Eye Hospital: A Questionnaire Based Study

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Abstract

Purpose: This cross-sectional study aimed to assess the level of awareness and knowledge about glaucoma among patients attending a tertiary eye hospital in Bangladesh.

Methods: A questionnaire-based survey was conducted on 300 individuals attending Deep Eye Care Foundation, Rangpur, from April 2023 to September 2023. Informed consent was obtained from eligible participants over 18 years. The questionnaire, available in both English and Bengali, collected demographic data and included questions on glaucoma awareness and knowledge. Data were analyzed using SPSS 25, and the Chi-Square test was applied to determine statistical significance.

Results: Among the participants, 34.7% had heard of glaucoma, with doctors/hospitals being the primary source of information. Awareness varied significantly based on age, residency, education, occupation, and income levels. Among those aware of glaucoma, 59.6% had poor knowledge, 33.7% had average knowledge, and only 6.7% had good knowledge. No significant association was found between glaucoma patients and non-glaucoma patients regarding knowledge level.

Conclusion: This study revealed a substantial gap in glaucoma awareness and knowledge in Bangladesh. While a notable portion of the population had heard of glaucoma, their understanding of the condition was limited, emphasizing the need for targeted awareness campaigns and improved access to eye care services. Addressing these gaps is crucial for early diagnosis, treatment, and preventing irreversible blindness while reducing the economic burden associated with glaucoma-related healthcare costs.

Keywords: Awareness, Knowledge, Glaucoma, Cross sectional study.

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Introduction

Glaucoma is a chronic and progressive optic neuropathy characterized by specific changes in the optic disc and visual field defects, often associated with elevated intraocular pressure.[1] It ranks as the second leading cause of blindness globally, following cataracts, and stands as the primary cause of irreversible blindness worldwide. Unfortunately, it is frequently diagnosed at an advanced stage, resulting in significant eye damage.[2] Early detection and treatment can prevent permanent blindness, but a lack of awareness and inadequate screening tools contribute to delayed diagnosis, even among educated individuals.[3] Studies have indicated that a substantial percentage of glaucoma cases, ranging from 50% to 90%, go undiagnosed.[4] The main culprit behind delayed glaucoma diagnosis is often a lack of awareness[2,3], which significantly increases the risk of glaucoma-related blindness.[3] Socioeconomic factors, including education levels, family history, access to entertainment, and exposure to public health education efforts by government or non-governmental organizations (NGOs), influence the level of awareness.[4]

Insufficient awareness not only impacts the timing of diagnosis but also hinders the utilization of eye care services.[5] Glaucoma-related blindness imposes substantial financial burdens on affected individuals and healthcare systems, reducing the overall quality of life and increasing the cost of recovery, which, in turn, affects a nation's economic development.[6] The

most significant risk factors for glaucoma include advanced age, elevated intraocular pressure (IOP), familial predisposition, and ethnicity.[1] Treatment usually involves IOP-lowering medications such as beta-blockers, prostaglandin analogs, and carbonic anhydrase inhibitors. However, the effectiveness of these drugs in halting glaucoma progression is sometimes uncertain.[6] Surgical interventions like laser trabeculoplasty and trabeculectomy become options when medical treatment fails.[7] Unfortunately, glaucoma diagnoses typically occur in advanced stages, leading to substantial visual impairment.[8]

Therefore, assessing awareness levels is essential for developing effective information, education, and communication models and devising new strategies to raise glaucoma awareness. Increased awareness encourages individuals to seek regular eye examinations and helps reduce the economic burden of this disease.[3,5] This study was designed to evaluate the extent of awareness and knowledge regarding glaucoma among patients using a questionnaire, aiming to develop targeted strategies against this vision-threatening condition.

Materials And Methods

This is a prospective cross sectional hospital based study conducted from April 2023 to September 2023 on 300 individuals attending Deep Eye Care Foundation, Rangpur. Informed consent was obtained from all eligible participants. The Institutional review Board of Deep Eye Care Foundation has given the ethical approval. Inclusion criteria were patients over 18 years and who gave informed written consent to participate in the study. Patients, who were severely ill, not willing to participate, age less than 18 years and mentally unstable patients were excluded from the study.

A questionnaire was prepared in English and translated into easily understand local language Bangla. After collecting demographic data, awareness related questions were enquired, which include medical data, history of DM, HTN, history of eye examination ,history or family history of glaucoma, having heard of glaucoma and source of information. Awareness is assessed by asking, 'Have you ever heard of the term 'Glaucoma'?'" Knowledge details were only obtained from the patients who were aware of Glaucoma. Knowledge was measured by asking participants about altered anatomical site, different types, clinical presentation, risk factors, association with high IOP and visual field, treatment option of glaucoma. This survey was conducted by Doctor, Optometrist and Refractionist who were trained about the questionnaire and had optimum knowledge about glaucoma. The obtained data were entered into MS excel sheet and analysed using the SPSS version 25, the Chi Square test was applied. The P - value < 0.05 was considered statistically significant.

Results

In this study, a total of 300 participants were interviewed during the two one month study period. The age of participants ranged between 18 and 85 years with mean age of 43.9±15.4 years (Table 1).

Table 1: Distribution of Age group of the respondents

Age group (Years)	Frequency	Percentage (%)	
18 - 39 Years	120	40.0	Mean± SD= 43.9±15.4 Minimum = 18 Maximum = 85
40 - 49 Years	66	22.0	
50 - 59 Years	54	18.0	
60 Years & Above	60	20.0	
Total	300	100.0	

Regarding gender, the study had almost equal representation, with 50.7% male participants and 49.3% female participants. In terms of residency, 49.0% of the patients lived in urban areas, while 51.0% resided in rural areas. The educational background of the participants varied significantly. Participants had diverse educational backgrounds, with 22.3% having completed secondary education, 19.0% having no formal education, and 15.3% holding graduate degrees. Occupations ranged from day

labor (2.0%) to service holder (18.3%), and the majority (53.7%) had a monthly income below 20,000 Bangladeshi Taka (Table 2). In terms of medical history, 20% (60) of participants had a history of hypertension, 13.7% (41) had a history of diabetes, and 15.3% (46) had a history of glaucoma. Additionally, 8.6% (26) had a positive family history of glaucoma. When asked if they had ever heard of the term "Glaucoma" before, 34.7% (104) responded affirmatively, while 65.3% had not. Among those who had heard of glaucoma, 86.5% knew that it is an eye disease (Table 3).

Table 2: Socio-demographic criteria of the respondents

Sex	**Frequency (N)**	**Percentage (%)**
Male	152	50.7
Female	148	49.3
Area of Resident		
Urban	147	49.0
Rural	153	51.0
Education		
No Formal Education	57	19.0
Primary Education	44	14.7
Secondary Education	67	22.3
Higher Secondary	45	15.0
Graduate	46	15.3
Post-Graduate	41	13.7
Occupation		
Unemployed	25	8.3
Retired	17	5.7
Farmer	52	17.3
Business	50	16.7
Day Labor	6	2.0
Service Holder	55	18.3
Home Maker	54	18.0
Student	28	9.3
Others	13	4.3
Monthly Income		
< 20000	161	53.7
20001 - 30000	52	17.3
30001 - 40000	24	8.0
40001 - 50000	26	8.7
> 50000	37	12.3
Total	**300**	**100**

Table 3 : History of Glaucoma & Other Diseases with Awareness on Glaucoma

Variables	**Yes**	**No**
History of Hypertension	60 (20.0)	240 (80.0)
History of Diabetes	41 (13.7)	259 (86.3)
History of Glaucoma	46 (15.3)	254 (84.7)
Positive history of Glaucoma in family	26 (8.6)	274 (91.4%)
Have you ever heard of the term "Glaucoma" before?	104 (34.6%)	196 (65.4%)
Do you know that Glaucoma is an eye disease? [n=104]	90 (86.5%)	14 (13.5%)

For those who were aware of glaucoma, the primary source of information was doctors/hospitals (56.8%), followed by family members (19.2%), relatives/friends (9.6%), and mass media (14.4%) (Figure 1). Further analysis showed no significant differences in glaucoma awareness based on gender. However, awareness varied significantly across age groups, with the youngest age group (18 - 39 years) having the highest awareness. Urban residents were more aware of glaucoma than rural residents. Educational level was strongly associated with awareness, with higher education levels corresponding to higher awareness (p=<0.001). Occupation also played a significant role in awareness, with service holders having the highest awareness (p=<0.001). Monthly income was associated with awareness, with higher income individuals having greater awareness (p=<0.001) (Table 4). Among those who were aware of glaucoma (N=104), the mean knowledge score was 6.6 out of a maximum of 13, with 59.6% having poor knowledge, 33.7% having average knowledge, and 6.7% having good knowledge about glaucoma (Table 5). Figure 2 shows the association between patients with a history of glaucoma and the knowledge level of the respondents. There was no significant association found in these two groups. Table 6 presents the frequency of responses regarding basic glaucoma knowledge among the participants who were aware of glaucoma.

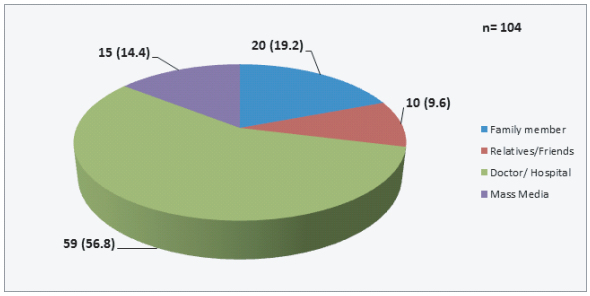

Figure 1: Source of Information who were aware about Glaucoma

Table 4: Association of awareness of glaucoma with socio-demographic Factors

Variables	Aware	Not Aware	Total	P-value
Sex				
Male	53	99	152	0.941
Female	51	97	148	
Age group				
18 - 39 Years	47	73	120	0.05
40 - 49 Years	24	42	66	
50 - 59 Years	10	44	54	
60 Years & Above	23	37	60	
Area of Resident				
Urban	65	82	147	0.001
Rural	39	114	153	
Education				
No Formal Education	6	51	57	< 0.001
Primary Education	12	32	44	
Secondary Education	15	52	67	
Higher Secondary	17	28	45	
Graduate	24	22	46	
Post-Graduate	30	11	41	
Occupation				
Unemployed	11	14	25	< 0.001
Retired	8	9	17	
Farmer	4	48	52	
Business	12	38	50	
Day Labor	1	5	6	
Service Holder	30	25	55	
Home Maker	19	35	54	
Student	11	17	28	
Others	8	5	13	
Monthly Income				
< 20000	38	123	161	< 0.001
20001 - 30000	19	33	52	
30001 - 40000	10	14	24	
40001 - 50000	12	14	26	
> 50000	25	12	37	
Total	104	196	300	

Table 5: Knowledge level among the respondents who were aware of glaucoma

Degree of Level	Knowledge			Mean= 6.6 Std. Deviation= ±3.74 Minimum= 2 Maximum= 13
	Poor <50%	Average 50%-75%	Good >75%	
Number	62	35	7	
%	59.6	33.7	6.7	

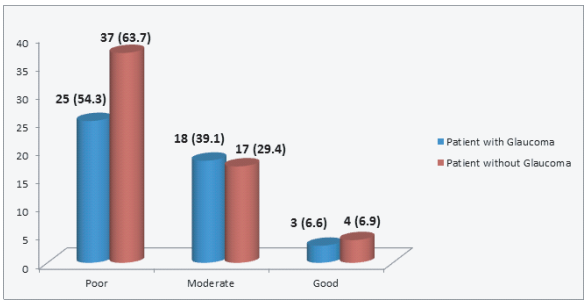

Figure 2 : Association between Patient with or without Glaucoma and knowledge level of the respondents

Table 6 : Frequency of responses regarding basic glaucoma knowledge

Questions	N (%)
Altered anatomical site in glaucoma	
Retina	3 (2.9)
Optic nerve*	34 (32.7)
Cornea	0 (0.0)
I don't know	67 (64.4)
Existence of different types of glaucoma	
Yes*	24 (23.0)
No/ I Don't Know	80 (77.0)
Glaucoma can occur without symptoms	
Yes*	29 (27.9)
No/ I Don't Know	75 (72.1)
Glaucoma is more common in people above 60 years	
Yes*	41 (39.4)
No/ I Don't Know	63 (60.6)
High intraocular pressure, family history and advanced age are glaucoma risk factors	
Yes*	49 (47.1)
No/ I Don't Know	55 (52.9)
Associated with high intraocular pressure	
Yes	55 (52.9)
No	49 (47.1)
Not Always	0 (0.0)
I Don't Know	0 (0.0)
Visual field is affected	
Yes	47 (45.2)
No/ I Don't Know	57 (54.8)
Glaucoma progresses over time	
Yes*	53 (51.0)
No/ I Don't Know	51 (49.0)

Glaucoma causes blindness	
Yes*	57 (51.0)
No/ I Don't Know	47 (49.0)
Glaucoma can be cured	
Yes/ I Don't Know	43 (41.3)
No*	61 (58.7)
Glaucoma has treatment	
Yes*	68 (65.4)
No/ I Don't Know	36 (34.6)
Available treatment options	
Drops, laser and surgery	44 (42.3)
I don't know	41 (39.4)
Drops and surgery	7 (6.7)
Only drops	11 (10.6)
Only surgery	1 (1.0)
Purpose of the treatment of Glaucoma	
Regain vision	24 (23.0)
I don't know	51 (49.0)
Delay progression	17 (16.3)
Stop progression	12 (11.5)
Glaucoma treatment reduces intraocular pressure	
Yes*	52 (50.0)
No/ I Don't Know	52 (50.0)
Glaucoma damage is reversible	
Yes*	27 (26.0)
No/ I Don't Know	77 (74.0)
Glaucoma is inherited	
Yes*	33 (31.7)
No/ I Don't Know	71 (68.3)

***Correct Answer**

Discussion

Glaucoma, a leading cause of irreversible blindness, can be prevented with early diagnosis and treatment. Our cross-sectional study exhibited nearly equal representation, with 50.7% male and 49.3% female participants. Interestingly, we found that 19% of participants had no formal education, a statistic that mirrors findings from a study conducted by S. Lartey et al.[2]

In our study conducted in Bangladesh, we observed that 34.7% of participants had heard of the term "Glaucoma," indicating a notable level of awareness within the population. However, a significant majority, comprising 65.3%, had not previously encountered this term. In stark contrast, a study by Tenkir et al.[8] among Southwestern Ethiopians reported remarkably low glaucoma awareness, with only 2.4% of participants indicating awareness of the condition. This substantial regional disparity underscores the significant influence of varying healthcare infrastructure and public health initiatives.

A study conducted in Bangladesh by Nasrin et al.[9] reported a higher awareness rate, with 50% of the study population being aware of glaucoma. Our study's 34.7% awareness rate aligns reasonably well with their findings, suggesting a certain level of consistency in glaucoma awareness within the country. In contrast, the study by Shahariar Islam[10], also conducted in Bangladesh, revealed lower levels of glaucoma awareness (0.5%). These differences may be attributed to factors such as the urban-rural divide or specific community characteristics. Conversely, the study by Becerril-Ledezma et al.[11] in Mexico reported a significantly higher general awareness proportion of 73.9%, indicating relatively high glaucoma awareness among Mexican patients. In urban Chennai, Ramesh et al.[12] found an awareness level of 13.3%, while Mridula et al.[13], in a tier 2 city of South India, reported an awareness level of 4.8%.

These variations illustrate the wide range of glaucoma awareness levels across different countries and regions, likely influenced by disparities in healthcare systems and public health efforts.

In summary, our study contributes significantly to our understanding of glaucoma awareness and knowledge within the context of Bangladesh. These comparative findings emphasize the necessity of tailored awareness campaigns and improved access to eye care services, accounting for regional variations and unique socioeconomic factors influencing glaucoma awareness. Furthermore, our analysis showed no significant differences in glaucoma awareness based on gender, in contrast to findings from developed countries where males often exhibit lower awareness levels. Urban residents in our study were more aware of glaucoma than rural residents. Education level, occupation, and monthly income were strongly associated with awareness, aligning with the demographic characteristics of the population.[14-17]

Additionally, the primary source of glaucoma

awareness in our study was doctors or hospitals (56.8%), followed by family members with glaucoma and mass media. In contrast, in southern India, the most common source of glaucoma awareness was TV/magazines, followed by family members with glaucoma. Representative German population surveys indicated that friends were the most common source of awareness (44%), surpassing physicians (13%), a trend similarly observed in urban Indian populations.[18-20]

In our study, while we observed a moderate level of glaucoma awareness, we identified a notably low level of knowledge (6.7%) among individuals who were aware of the condition. Comparing our findings to the study by Becerril-Ledezma et al.[11] conducted in Mexico, we observed a similar trend, with both studies reporting a relatively low proportion of participants with good knowledge of glaucoma (15.5%). This suggests that, despite reasonable awareness levels, a significant gap in understanding glaucoma persists among patients in both settings.

Conversely, a study by Atebo et al.[21] in Australia reported a high awareness rate of 93%. However, even with such high awareness, only 29% of participants demonstrated some knowledge of glaucoma. Similarly, the study by S. Lartey et al.[2] found that 74% of participants were aware of glaucoma, but only 27% had some knowledge of its clinical presentation. This finding aligns with our observation that a substantial proportion of individuals may be aware of glaucoma's existence but lack a deeper understanding of its clinical aspects.

Notably, our study did not identify any significant association between glaucoma patients and non-glaucoma patients regarding knowledge level. However, studies by Becerril-Ledezma et al.[11] and Celebi[22] found higher knowledge scores among patients with glaucoma.

In conclusion, our study highlights the importance of region-specific strategies to address the challenges of glaucoma awareness and knowledge. While glaucoma awareness can vary widely between countries and regions, our findings emphasize the need for targeted educational initiatives to bridge the gap between awareness and understanding. These insights underscore the importance of comprehensive efforts to combat this potentially blinding disease effectively.

Conclusions

This study highlights a significant gap in glaucoma awareness and knowledge in Bangladesh. While a notable portion of the population has heard of glaucoma, their understanding of this vision-threatening condition is alarmingly limited, even among those aware of it. This underscores the urgent need for tailored awareness campaigns and improved access to eye care services, especially in regions with lower awareness levels. In summary, addressing glaucoma awareness and knowledge gaps is crucial for early diagnosis, treatment, and preventing irreversible blindness, while also reducing the economic burden associated with glaucoma-related healthcare costs.

References

1. Vakharia, Divya & Gokhale, Suvarna. (2014). Glaucoma Awareness among patients at outpatient department of Ophthalmology in Smt.Kashibai Navale Medical College and General Hospital, Narhe, Pune.. International Journa Of Sciencs & Research. 3. 2319-7064.
2. Nkum G, Lartey S, Frimpong C, Micah F, Nkum B. Awareness and Knowledge of Glaucoma Among Adult Patients at the Eye Clinic of a Teaching Hospital. Ghana Med J. 2015 Sep;49(3):195-9. doi: 10.4314/gmj.v49i3.11. PMID: 26693196; PMCID: PMC4676595.
3. Gupta S, Rishi P, Glaucoma awareness in undiagnosed literate individuals attending routine eye clinic at a tertiary care center. Indian J Clin Exp Ophthalmol 2016;2(4):370-372
4. Sandhya R., Sinha A, Shetty N, A Study of Awareness of Glaucoma among Ophthalmology out patients in a 2-Tier City, Tumkur. Indian J Clin Exp Ophthalmol 2015;1(3):156-158

5. P.Nirmala Jyothi Pamu and Chandana Priyanka.S. (2019); ASSESSMENT OF KNOWLEDGE, AWARENESS ON GLAUCOMA AMONG GLAUCOMA PATIENTS & ITS IMPACT ON DAILY LIFE. Int. J. of Adv. Res. 7 (Feb). 374-378.

6. Priyadarshini, Dr & Rastogi, Priye Suman & Chander, Ashish. (2020). To study glaucoma awareness, knowledge, attitude towards screening in patients of western Uttar Pradesh. International Journal of Medical Ophthalmology. 2. 68-72. 10.33545/26638266.2020.v2.i2b.42.

7. Alqahtani SM, Bakarman MA, Almanjoumi A, Alzahrani SH. Awareness and knowledge about glaucoma among patients visiting the screening clinic in Jeddah Eye Hospital, Saudi Arabia. Int J Ophthalmol. 2021 Jun 18;14(6):887-895. doi: 10.18240/ijo.2021.06.15. PMID: 34150545; PMCID: PMC8165616.

8. Tenkir, A., Solomon, B., Deribew, A.,Glaucoma awareness among people attending ophthalmic services in Southwestern Ethiopia BMC Opthalmology 2010 10:17doi:10: p. 1186/147-2415-10- 17.

9. Nasrin, Nishat & Asaduzzaman, Muhammad & Sharmin, Dilruba & Tithi, Nigar & Hudda, Syed. (2020). A survey-based study on awareness of eye diseases among population of Bangladesh. Asian Journal of Medical and Biological Research. 5. 292-296. 10.3329/ajmbr.v5i4.45267.

10. Shahariar Islam. Knowledge and Awareness of Glaucoma and associated Risk Factors in Urban Community. ScienceOpen Preprints. 2020. DOI: 10.14293/S2199-1006.1.SOR-.PPEIJ79.v1

11. Becerril-Ledezma V, Alvarez-Ascencio D, Del Hierro-Gutiérrez CE, Hernandez-Oteyza A, Jiménez-Román J. Knowledge and awareness of glaucoma in Mexican patients with and without glaucoma diagnosis in an Ophthalmology Referral Center. Int J Ophthalmol. 2022 Jun 18;15(6):990-996. doi: 10.18240/ijo.2022.06.18. PMID: 35814902; PMCID: PMC9203486.

12. Sathyamangalam RV, Paul PG, George R, Baskaran M, Hemamalini M, Madan RV et. al Determinants of glaucoma awareness and knowledge in urban Chennai. Indian j ophthalmol. 2009 Sept.-Oct,57(5); 355-60.

13. Prabhu M, Patil SH, Kangokar PR. Glaucoma awareness and knowledge in a tertiary care hospital in a tier-2 city in South India. J Sci Soc; 2013; Vol.40; Issue-1;3-8.

14. Maharana PK, Rai VG, Pattebahadur R, Singhi S, Chauhan AK. Awareness and knowledge of glaucoma in central India: a hospitalbased study. Asia Pac J Ophthalmol (Phila) 2017;6(3):243-249.

15. Kizor-Akaraiwe NN, Monye HI, Okeke S. Awareness and knowledge about glaucoma and proportion of people with glaucoma in an urban outreach programme in Southeast Nigeria. BMJ Open Ophthalmol 2017;1(1):e000018.

16. Sood D, Sood S, Sood I, Kumar D, Sood R, Sood NN. Glaucoma awareness amongst glaucoma patients seeking a second opinion. Int Ophthalmol 2018;38(4):1441-1449.

17. De-Gaulle VF, Dako-Gyeke P. Glaucoma awareness, knowledge, perception of risk and eye screening behaviour among residents of Abokobi, Ghana. BMC Ophthalmol 2016;16(1):204

18. Krishnaiah S, Kovai V, Srinivas M, Shamanna BR, Rao GN, Thomas R. Awareness of glaucoma in the rural population of Southern India. Indian J Ophthalmol 2005;53:205 8.

19. Pfeiffer N, Krieglstein GK, Wellek S. Knowledge about glaucoma in the unselected population: A German survey. J Glaucoma 2002;11:45863.

20. Dandona R, Dandona L, John RK, McCarty CA, Rao GN. Awareness of eye diseases in an urban population in southern India. Bull World Health Organ 2001;79:96102

21. Attebo, K.,Mitchell , P,. Knowledge and diseased about common eye diseases.Australian and New Zealand Journal of Ophthalmology .1997. 25 (3):p 283-284

22. Celebi ARC. Knowledge and Awareness of Glaucoma in Subjects with Glaucoma and their Normal First-Degree Relatives. Med Hypothesis Discov Innov Ophthalmol. 2018 Spring;7(1):40-47. PMID: 29644244; PMCID: PMC5887606.

Original Article

Clinical characteristics of pigment dispersion syndrome in Bangladeshi population

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Abstract

Purpose: To report the clinical findings and characteristics of pigment dispersion syndrome (PDS) in Bangladeshi population.

Method: PDS suspects with any one of the following signs: corneal endothelial pigmentation, iris trans illumination defects (ITDs), pigment granule dusting on anterior iris surface, posterior iris bowing, trabecular meshwork (TM) pigmentation and lenticular or zonular pigmentation were evaluated for PDS at the Glaucoma specialty clinic at Chittagong Eye Infirmary and Training Complex. Diagnosis of PDS required at least two of the following signs: Krukenberg spindle, moderate-to-heavy TM pigmentation, and any degree of lenticular and/or zonular pigmentation.

Result: Thirty-six patients (24 males and 12 females) were identified as having PDS during a 1-year period, with a mean age of 35.5±7.0 years (range, 22–49). All but two eyes from two patients had myopia of 0.5 D or greater, with the mean spherical equivalent power of 5.20±5.80 D (range, 24.75±0.5). The average IOP at initial diagnosis was 33.7±10.5 mm Hg (range, 16–56). Thirty patients (83.3%) were found to have pigmentary glaucoma at their initial diagnosis. All patients showed homogenous increased TM pigmentation as well as lenticular and/or zonular pigmentation. 61.1% of patients had Krukenberg spindle. None of the patients exhibited spoke-like mid-peripheral transillumination defect except for trace-isolated transillumination in both eyes of the two patients.

Conclusion: The most common clinical findings in Bangladeshi PDS patients include corneal pigment granules, homogeneous TM pigmentation and pigment granule dusting on lens zonules and/or posterior peripheral lens surface. Transillumination defects are uncommon in Bangladeshi patients with PDS.

Keywords: Pigment, Glaucoma, Krukenberg, Pigmentation.

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Introduction

Pigment dispersion syndrome (PDS) is characterized by pigment granule dispersion from the iris pigment epithelium (IPE). The released pigment granules are transmitted by aqueous convection currents and deposit on the corneal endothelium, iris surface, trabecular meshwork (TM), lens surfaces and zonules. Accumulation of pigment in the TM may lead to increased resistance to aqueous outflow and results in the development of pigmentary glaucoma (PG).[1, 2]

The prevalence of PDS among adult whites is as high as 2.45%.[3] Commonest clinical findings in white PDS patients include the triad of corneal krukenberg spindle, mid-peripheral iris transillumination defects, and homogenous TM pigmentation, usually found in young myopic male patients.[4,5,6,7] The prevalence of PDS in blacks >7 years old was calculated to be 0.167%±0.013.[8] Clinical signs in dark PDS patients is narrower than that of typical and classic type in whites. Transillumination defects and anterior iris stromal pigment dusting are, however, not as common in blacks.[8,9,10,11,12,13] To find out the clinical features of PDS in Bangladeshi, we carried out a prospective observational investigation on a series of 36 Bangladeshi patients identified as having PDS according to the identical diagnostic criteria.

Patients and methods

Patient suspected as PDS at Glaucoma clinic at Chittagong Eye Infirmary and Training Complex from May 2015 to April 2018 were evaluated, If the patient had any one of the following signs: corneal endothelial pigmentation, anterior iris stromal pigment dusting, ITDs, posterior iris bowing, increased TM pigmentation, and pigment granule dusting on lens zonules or peripheral posterior surface were included.

Detailed ophthalmic examinations included visual acuity measurement, IOP measurement, refraction, slit-lamp biomicroscopy pre- and post-mydriasis, gonioscopy, funduscopic examination and automated Humphrey SITA standard 24-2 visual field test were done. Systemic and ocular medical history of each subject was also recorded.

TM pigmentation was graded according to Scheie's grading system. Corneal endothelial pigment dusting was described as Krukenberg spindle, diffusive pattern, none. If the patient had received anti-glaucoma medications before the examination, the IOP measured before medication was taken as the baseline IOP. Slit-lamp examination, gonioscopy and funduscopic evaluation of the optic disc of all patients were performed and graded by the same physician to avoid inter-physician differences.

Diagnostic criteria for PDS included at least two of the following three signs: corneal Krukenberg spindle, homogenous moderate-to-heavy TM pigmentation and any degree of zonular and/or lenticular pigment granule dusting. Patients with a history of uveitis, trauma, ocular surgery or anterior segment laser treatment, or any evidence of exfoliation material were excluded. Patients with PDS were diagnosed with secondary glaucoma, if they had two or more of the following findings: initial IOP >21 mm Hg, glaucomatous optic nerve damage (increased cupping or abnormal disc appearance) or glaucomatous visual field loss.

Results

Out of 188 PDS suspects thirty six patients (24 males and 12 females) were identified as having PDS according to criteria. Mean age of the PDS patients was 35±5.0 years (range, 22–49). Male to female ratio was 2:1.

All patients had myopia of 0.5 D or greater except 2. 35 patients had increased baseline IOP of >21 mm Hg in at least one eye at the time of diagnosis with an average of 32±10 mm Hg.

Four patients had family history of glaucoma. Another 8 had family history of PDS. All of them were taking anti-glaucoma medications at the time of evaluation for PDS. Only 12 of the PDS patients had symptoms which were mostly occasional blurring of vision.

Bilateral Krukenberg spindle was found in16 patient and unilateral in 6. (Figure 1a). Of the remaining 14, three had trace diffuse corneal endothelial pigmentation and 8 had no corneal pigment dusting.

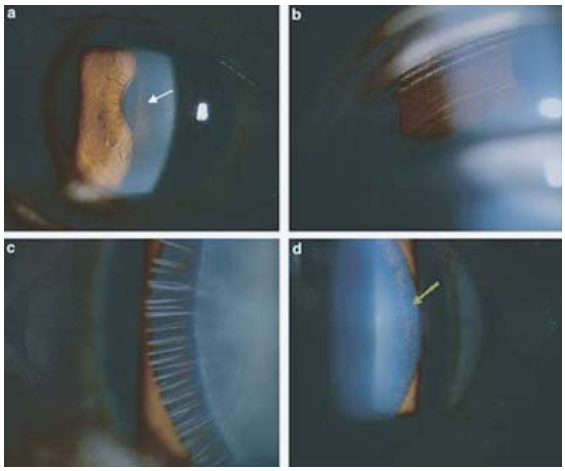

Figure 1 :

(a) Triangular Krukenberg spindle (white arrow) (b) Trabecular meshwork pigmentation. (c) Pigment granules on lens zonules. (d) Pigment accumulated at the zonular attachments to the lens, where it formed a Zentmayer ring, also referred as Scheie's line (green arrow).

Posterior bowing of the mid-peripheral iris was found in all 2 cases. The slit beam was projected vertically through the centre of the pupil at 30–45 degrees.

In 4 patients short slit-like transillumination defects were visualized in iris crypts.

Homogeneous TM pigmentation and pigment granule dusting on lens zonules and/or peripheral posterior lens surface were seen in all patients. On slit lamp exam all patients showed different extent of pigment on lens zonules (Figure 1c) and/or posterior peripheral surface (Zentmayer ring) (Figure 1d).

30 out of 36 patient (83%) developed glaucoma. Out of 30, 20 patients were bilateral and 10 patients were unilateral. 4 patients presented

with blindness at the time of initial presentation. They had severe constriction of visual field in bilateral eyes.

Discussion

Among two phenotypes of PDS, one is the classic type in whites characterized by the triad of Krukenberg spindle, trans illumination defects and homogeneous TM pigmentation. Posterior iris bowing, pigment granule dusting on peripheral posterior lens surface and/or zonules, as well as anterior iris stromal pigment dusting are also common findings in white PDS patients.[3,6,7,14] The other phenotype is the one found in blacks. Black patients may not have mid-peripheral radial ITDs.[8,9,10,11,12,13] The Results of our study have shown that the clinical features of PDS in Bangladeshis are more like that of blacks. Homogeneous TM pigmentation and posterior lenticular and/or zonular pigment granule dusting are the most common findings in both black and Bangladeshi PDS patients. In all, 61% of Bangladeshi PDS patients in our series have Krukenberg spindle. In black patients, this percentage was 57.1%.[8] Nevertheless, there are differences in clinical findings between black and Bangladeshi PDS patients. Posterior iris bowing was very common in Bangladeshi PDS patients seen in 94% of the patients, but is rare in black patients.[8] Trace clusters of pigment granules on inferior surface of the iris were discerned in 6 of the 36 (16%) patients.

The gender and age distribution of these PDS patients was similar to white and black PDS patients. All the patients were detected between 20 and 50 years of age, with a 2:1 male to female ratio.

Black PDS has no ITDs due to dark iris.[8,11,13] There are a greater content of pigment granules in melanocytes and iris stroma in dark irides than in light ones can block an iris transillumination defect. Bangladeshi iris colour is usually brown or dark brown for which there is usual absence of ITDs. Infra-red imaging technique, which has demonstrated to be helpful in detecting ITDs in black PDS patients,[11,13] may pick up iris defects in our patients too.

To our knowledge, there has been no published data regarding clinical features of PDS in Bangladeshi patients. Theoretically it's a rare disease. But the results of this study is so alarming that,this may not be the actual situation. In this study, we found that PDS patients comprised 1.1% (36 out of 3264) of all outpatients in Glaucoma clinic of CEITC. This is much more common than we expected. So it might be underestimated. There is no typical ITDs in Bangladeshi populations. For this, PDS is missed from doctor's attention. 12 of the PDS patients in this study had been diagnosed with primary open angle glaucoma before referral to the authors and this is a common practice so far. Secondly, even though Krukenberg spindle is seen in 61% of our PDS patients, it is not obvious because of the dark background of the highly pigmented iris, especially when the pigmentation was minimal. This pigment does not do any harm of endothelial function like other study. Stromal pigment dusting was absent . Krukenberg spindle, ITDs, and anterior iris stromal pigment granule dusting are all important signs of PDS and in pigmentary glaucoma .But none of them is easily detected in Bangladeshi PDS patients. This may lead to missing of some cases. Lack of early diagnosis, most of them presents with raised IOP and optic nerve damage. In our study 83% of patients had PG and 94% of them had increased IOP at their initial diagnosis of PDS. These percentages are much higher than white PDS patients.[3,6,7,14,15]

Commonest clinical findings in Bangladeshi PDS patients are homogeneous moderate-to-heavy TM pigmentation and pigment granule dusting on peripheral posterior lens surface and/or zonules. Radial mid-peripheral ITDs are rare in this patient population. 61% of the patients had Krukenberg spindle. Among them, 83% had PG at the time of initial diagnosis of PDS. So the clinical findings of PDS in Bangladeshi population is really alarming because they may have glaucoma or may develop glaucoma within a very short time.

References

1. Campbell DG. Pigmentary dispersion and glaucoma: a new theory. Arch Ophthalmol 1979; 97: 1667–1672.
2. Yang JW, Sakiyalak D, Krupin T. Pigmentary glaucoma. J Glaucoma 2001; 10: S30–S32.
3. Ritch R, Steinberger D, Liebmann JM. Prevalence of pigment dispersion syndrome in a population undergoing glaucoma screening. Am J Ophthalmol 1993; 115: 707–710.
4. Sugar HS, Barbour FA. Pigmentary glaucoma: a rare clinical entity. Am J Ophthalmol 1949; 32: 90–92.
5. Scheie HG, Fleischhauer HW. Idiopathic atrophy of the epithelial layers of the iris and ciliary body. Arch Ophthalmol 1958; 59: 216–288.
6. Sheie HG, Cameron JD. Pigment dispersion syndrome: a clinical study. Br J Ophtalmol 1981; 65: 264–269.
7. Gillies WE, Brooks AM. Clinical features at presentation of anterior segment pigment dispersion syndrome. Clin Experiment Ophthalmol 2001; 29: 125–127.
8. Roberts DK, Chaglasian MA, Meetz RE. Clinical signs of the pigment dispersion syndrome in blacks. Optom Vis Sci 1997; 74: 993–1006.
9. Semple HC, Ball SF . Pigmentary glaucoma in the black population. Am J Ophthalmol 1990; 109: 518–522.
10. Roberts DK, Flynn M, Gable EM. Anterior chamber angle anomalies associated with signs of pigment dispersion in a group of black probands and their first-degree relatives. Optom Vis Sci 2001; 78: 133–141.
11. Roberts DK, Wernick MN. Infrared imaging technique may help demonstrate iris transillumination defects in blacks who show other pigment dispersion syndrome clinical signs. J Glaucoma 2007; 16 (5): 440–447.
12. Roberts DK, Winters JE, Castells DD, Teitelbaum BA, Alexander CC. A cross-sectional study of Krukenberg spindles and pigmented lens striae in a predominately black population. J Glaucoma 2005; 14: 57–63.
13. Roberts DK, Chaglasian MA, Meetz RE. Iris transillumination defects in the pigment dispersion syndrome as detected with infrared videography: a comparision between a group of blacks and a group of nonblacks. Optom Vis Sci 1999; 7: 545–549.
14. Siddiqui Y, Ten Hulzen RD, Cameron JD, Hodge DO, Johnson DH. What is the risk of developing pigmentary glaucoma from pigment dispersion syndrome. Am J Ophthalmol 2003; 135: 794–799.
15. Lichter PR, Shaffer RN. Diagnostic and prognostic signs in pigmentary glaucoma. Trans Am Acad Ophthalmol Otolaryngol 1970; 74: 984–998.

Original Article

Comparison of intraocular pressure between air puff tonometer and Goldmann applanation tonometer at a tertiary eye care centre

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Abstract

Purpose: The purpose of this study was to evaluate the role of air puff (AP) tonometer by comparing the measurements of intraocular pressure (IOP) made using this device with those made using a Goldmann applanation tonometer (GAT) at Chittagong Eye Infirmary and Training Complex.

Methods: An observational and comparative study was carried out at Chittagong Eye Infirmary and Training Complex from January 2016 to January 2017. Two techniques for IOP measurements using the standard GAT and the non-contact tonometer (NCT) were compared. A total of 400 eyes from 200 patients were included in the study.

Results: The mean IOP as measured by GAT in the right eye was 14.50±5.59mmHg and in the left eye was 14.87±7.03mmHg while that as measured by NCT in the right eye was 15.97±6.12mmHg and in the left eye was 15.94±6.98mmHg. The mean difference between the two methods of measurement in the right eye was 1.47±0.53mmHg and the left eye was 1.07±0.05mmHg. The readings obtained by NCT were higher than those obtained by GAT. There was no statistically significant difference found in IOP measurements between GAT and NCT according to patient's age, gender or laterality of eyes.

Conclusion: There was a significant difference in the measurement of IOP between GAT and NCT. Goldmann applanation tonometry remains the most suitable and reliable method for measuring IOP.

Keywords: Intraocular pressure, air-puff tonometer, Goldmann applanation tonometer.

Introduction

Glaucoma is the second leading cause of irreversible blindness worldwide.[1] In the developed and developing countries, a significant proportion of glaucoma usually presents to eye care facilities in the advanced stages when the optic nerve is already damaged.[2,3]

Screening for glaucoma based solely on an IOP>21mmHg may miss up to half of the people with glaucoma in the screened population. However, IOP is still seen as a very important risk factor for the development of glaucomatous damage. Although other risk factors affect an individual's susceptibility to glaucomatous damage, IOP is the only risk factor that can be altered at this time.[4]

Applanation tonometry is based on the Imbert–Fick principle, which states that a perfect sphere has its internal pressure equally distributed and that the external force needed to flatten a known area of that sphere is directly proportional to the internal pressure of the sphere.[5]

The Goldmann applanation tonometer (GAT) is currently the most popular tonometer available. It consists of a double prism mounted on a standard slit lamp. The GAT represents the gold standard for IOP measurement. With the GAT, the force required to flatten, or applanate, a constant area of the cornea is measured and related to the IOP using the Imbert–Fick principle. The GAT uses an applanation diameter of 3.06 mm and is performed with the patient seated at the slit lamp.[6]

Air-puff tonometry is an applanation method using a standardized puff of air to flatten the cornea. This method has the advantage that no topical anesthetic or risk of corneal abrasion is involved.[7] The system consists of a central air plenum flanked either side by a light emitter and a light detector. As the pressure of the air pulse directed to the cornea increases to deform the

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cornea, the corneal surface behaves like a plane mirror, reflecting light to the detector. Corneal applanation is measured by collecting light reflected from the central cornea.[8]

The purpose of this study was to compare the IOP with an air puff tonometer and a Goldmann applanation tonometer at Chittagong Eye Infirmary and Training Complex.

Methods

An observational and comparative study was conducted on patients who presented to the Glaucoma clinic of Chittagong Eye infirmary and Training Complex from January 2016 to January 2017.

Exclusion criteria included patients having previous surgical intervention, one-eyed patients, traumatic cases, non-cooperative patients, those with severe visual loss and children less than 8 years of age.

Demographic data were documented and statistical analysis was conducted using SPSS 16.

The research follows the tenets of the Declaration of Helsinki, and each patient gave his or her informed consent to participate in this study. In case of children, informed consent from their guardians was obtained.

The medical devices used in the study were the NCT Huvitz HNT 7000 and the GAT HS Haag-Streit Diagnostics. For the examination, topical anaesthetic eye drops (5 mg/mL proxymetacaine hydrochloride) were used. The medical equipment was calibrated prior to the examination.

The IOP of both eyes of each patient were evaluated, first on the right and then on the left and each eye was used as one isolated data. The NCT measurement was always performed before the GAT measurement to eliminate the effect of ocular massage, which has been described when using the GAT and is absent with the NCT.[9]

Each assessment was conducted by a different optometrist who was completely unaware about the findings by the previous assessment.

Results

The study population comprised of 400 eyes from 200 patients. It included 128 males and 72 females with an age range of 8-85 years, and mean age was 49.75±17.04 years.

The data collected was classified into three groups according to the IOP measurements by GAT and NCT: Group 1, IOP < 12 mmHg: Group 2, IOP 12-24 mmHg; and Group 3, IOP > 25mmHg. The differences in readings were calculated. The data were also classified according to the patient's age, gender and laterality.

The data were analyzed using SPSS 16.

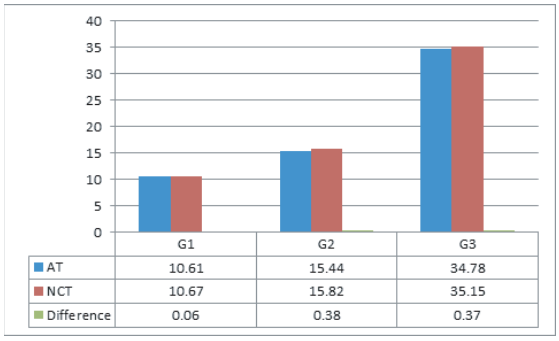
The mean IOP value as measured by GAT in the right eye was 14.50±5.59 mmHg, with a range of 2-46 mmHg and the left eye was 14.87±7.03 mmHg, with a range of 2-54 mmHg.

The mean IOP value as measured by NCT in the right eye was 15.97±6.12 mmHg, with a range of 3-48 mmHg and the left eye was 15.94±6.98 mmHg, with a range of 4-55 mmHg.

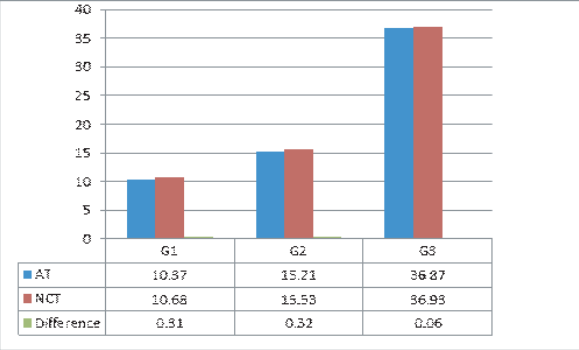
The mean difference of IOP values between GAT and NCT measurements in the right eye was 1.47±0.53mmHg and in the left eye was 1.07±0.05mmHg.

The difference in IOP values between the two devices was statistically significant (P= 0.0001).

This study showed that IOP values measured with NCT were higher than those measured with GAT (Figures 1 and 2).



Right eyes in study : mean IOP as measured by GAT, mean IOP as measured by NCT and the difference between the two readings according to three groups of IOP values (Group 1[G1], <12mmHg; Group 2[G2], 12-24mmHg and Group 3[G3],>24mmHg).



	G1	G2	G3
AT	10.37	15.21	36.87
NCT	10.68	15.53	36.93
Difference	0.31	0.32	0.06

Left eyes in study: mean IOP as measured by GAT, mean IOP as measured by NCT and the difference between the two readings according to three groups of IOP values (Group 1[G1], <12mmHg; Group 2[G2], 12-24mmHg and Group 3[G3],>24mmHg).

Discussion

In the majority of ophthalmic institutes, GAT is the most commonly used and reliable instrument. In addition, it is still considered the gold standard for assessment of IOP.[10]

This study showed there was a significant difference in measurements of IOP between GAT and NCT. The readings obtained by NCT were higher than those obtained by GAT in this study.

Shah et al. study 2012 reported a significant difference between the mean IOP assessed by GAT and air puff tonometry.[11]

Popovich and Shields evaluated 421 eyes and reported that the air puff was a reliable measurement compared to the GAT within the normal range of IOP. This fact is rarely supported in most of the published studies, where the majority of reports suggest that the GAT is the most consistent method of IOP assessment.[12]

Several other studies have compared IOP measurements obtained with GAT and those obtained by non-contact tonometers. Firat et al's study concluded that non-contact tonometer measurements were higher than those obtained by GATs and that this difference was statistically significant.[13]

There was no statistically significant difference found in IOP measurements between GAT and NCT according to patient's age, gender or laterality of eyes.

Conclusion

The GAT remains the most reliable device and is the international gold standard for measuring IOP. Measurements of IOP by NCT are usually higher than those obtained by GAT regardless of the patient's age, gender and laterality of eyes.

Conflict of Interest

The author reports no conflict of interest.

Acknowledgement

Chittagong Eye Infirmary and Training Complex

References

1. Kingman S. Glaucoma is second leading cause of blindness globally. Bull World Health Organ. 2004; 82:887-8.
2. Cook C. Glaucoma in Africa: Size of the problem and possible solutions. J Glaucoma. 2009;18:124-8.
3. Saw SM, Gazzard G, Friedman D, Foster PJ, Devereux JG, Wong ML, et al. Awareness of Glaucoma, and health beliefs of patients suffering primary acute angle closure. Br J Ophthalmol. 2003;87:446-9.
4. American Academy of Ophthalmology. Practicing Ophthalmologists Curriculum: Glaucoma, basic and clinical science course; 2007-2008. San Francisco, CA: American Academy of Ophthalmology; 2007:22-25.
5. Benjamin WJ, editor. Borish's Clinical Refraction, 2nd ed. Butterwoth-Heinemann;2006:501-503.
6. Morrison JC, Pollack IP, editors. Glaucoma Science and Practice. New York, NY: Thieme Medical Publishers; 2003:60-64.
7. Crick RP, Khaw PT. A Textbook of Clinical Ophthalmology: A Practical Guide to Disorders of the Eyes and Their Management, 3rd ed. Singapore: World Scientific; 2003:557.
8. Weinreb RN, Brandt JD, Garway-Heath DF, Medeiros FA, editors. World Glaucoma Association : Intraocular Pressure; Consensus Series 4. The Hague, The Netherlands: Kugler Publications; 2007:22-23.
9. McCafferty SJ, Levine J, Schwiegerling J, Enikov ET. Goldmann applanation tonometry error relative to true intracameral intraocular pressure in vitro and in vivo. BMC Ophthalmol. 2017;17:2015.
10. Essam A. Osman, Priscilla W. Gikandi, Abdul-Rahman A. Al-Jasser, Majeedah Alotaibi, Ahmed Mousa. Comparison of Goldmann Applantion, Noncontact Air Puff and Tono-Pen XL Tonometry in Normal Controls versus Glaucoma patients at a University Hospital in Riyadh, Saudi Arabia. Middle East Afr J Ophthalmol. 2018 Jan-Mar; 25(1): 8-13.
11. Shah MA, Bin Saleem K, Mehmood T. Intraocular pressure measurement: Goldmann applanation tonometer vs. non contact airpuff tonometer. J Ayub Med Coll Abbottabad. 2012;24:21-4.
12. Popovich KS, Shields MB. A comparison of intraocular pressure measurements with the XPERT noncontact tonometer and Goldmann applanation tonometry. J Glaucoma. 1997;6:44-6.
13. Firat PG, Cankaya C, Doganay S, et al. The influence of soft contact lenses on the intraocular pressure measurement. Eye (Lond). 2012;26(2):278-282.

Management of a case of microspherophakia with secondery angle closer glaucoma

S Rahman[1], U Kawsar[2], Z J Ruma[3], Z Farhana[4], M Siddika[5]

Abstract

Purpose: To present an atypical case of secondary glaucoma with non axial myopia and microspherophakia and its management.

Case Summary : A case of an 18-years-old boy presenting to our hospital with blurred vision in both eyes. Ophthalmic examination revealed high refractive error, shallow anterior chamber related to pupillary block caused by small, spherical lenses.

After detailed history taking and meticulous examination, the patient was diagnosed as a case of bilateral non axial myopia with acute angle closure crisis with micro spherophakia. As there were forward buldging of lens iris diaphragm causing flat anterior chamber and angle closure crisis and high myopia, Refractive lens exchange under anti glaucoma coverage were done in both eyes .

Conclusion: Bilateral secondary angle closure glaucoma may occur in a case of microspherophakia, meticulous examination with proper attention and management can restore the vision and limit the complications.

Key words: Non axial myopia, microspherophakia, angle closure glaucoma, IOP, RLE.

Introduction

Microspherophakia is a rare congenital anomaly characterized by the abnormal spherical shape of the crystalline lens. It is characterized by an increased anteroposterior thickness of the lens associated with reduced equatorial diameter.

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Secondary angle closure glaucoma is the most common sight threatening complication of microspherophakia. Glaucoma in microspherophakia may be caused via: Acute pupillary block mechanism caused by forward movement of spherical lens or anterior subluxation of lens due to weak and long zonules. Microspherophakia with swollen lens complicate the case. These type of cases do not respond well with anti glaucoma medications and laser treatment. Angle closure with pupillary block component make this more complicated.

Case report:

A 18 years male, student presented in OPD with blurring of vision of both eyes. His BCVA in R/E was 6/36 with -23.00/ -0.75@100, Left eye 6/60 with -20/ -3.00@ 145. IOP was R/E 30 mm Hg and L/E 28 mm of Hg.

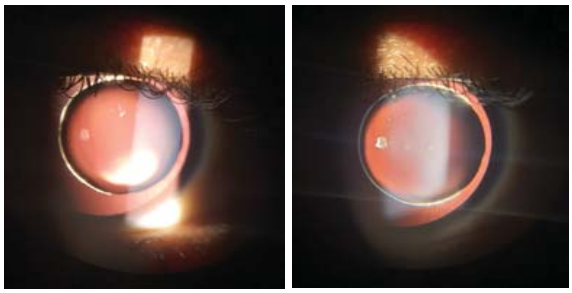

Fig : Dialeted pupil showing microsphrophakia in both eyes. (Slit lamp Examination)

Slit lamp examination revealed shallow anterior chamber, irregular pupil, forward buldging of lens iris diagphram in B/E.

There were glaucomatous damage in optic disc. Patient was diagnosed previously locally as PACG and treated with YAG peripheral iridotomy both eyes.

His glaucoma findings were

	RIGHT EYE	LEFT EYE
VA (unaided)	CF 4Feet	CF 2 Feet
BCVAR	6/36 with -23.OO/-0.75@100	6/60 with -20/-3.00@145
Cornea	clear	Clear
Anterior chamber	Shallow	Shallow
Lens	Microspherophakia forward buldging	Microspherophakia forward buldging
Pupil	Irregular, mid dilated	Irregular, mid dilated
Iris	Forward buldging	Forward buldging
Fundus	C:D- .85, nasal shifting of blood vessels, lamellar dot sign, RNFL thinning	C:D- .9, nasal shifting of blood vessels, lamellar dot sign, RNFL thinning
IOP	30 mm of Hg	28 mm of Hg

With these above clinical presentation we did some investigation including Biometry, Specular microscopy, Anterior segment photography, Anterior segment OCT, UBM, CFP, Visual field analysis, OCT RNFL of both eyes.

Fig : Anterior segment OCT showing narrow angle.

With all investigation and clinical presentation he was diagnosed as non axial myopia with myopic astigmatism with microspherphakia with secondary angle closure glaucoma with status post LPI both eyes.

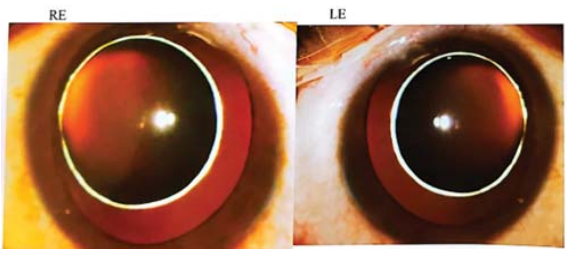

Fig : Slit lemp examination (Retro illumination)

He underwent CLE and SFIOL both eyes under anti glaucoma coverage. After refractive lens exchange with SFIOL BCVR R/E was 6/12with +0.50/-5.50@ 180 degree; L/E was 6/24 with +1.00/-2.50@ 180 degree. IOP right and left eye were 16 and 12 mm of Hg respectively.

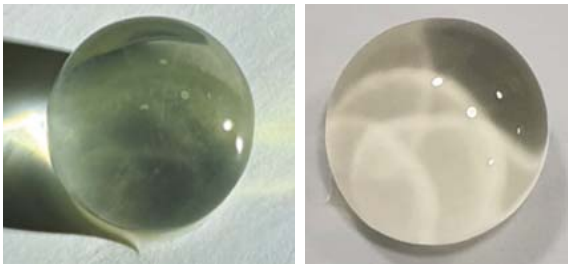

Fig: Crystalline Lens after surgery.

Discussion

Microspherophakia is a rare congenital entity that is characterized by a bi-convex spherical lens with reduced equatorial diameter. This is primarily because of the developmental anomaly of zonules. The clinical manifestations can range from asymptomatic to high lenticular myopia, acute pupillary block, sublocated or dislocated lens, acute angle-closure or chronic angle closure glaucoma [1]

The equatorial diameter of a microsphere lens measures on average between 6.75 to 7 mm (normal, 9.0mm), and the antero-posterior diameter is larger than normal and exceeds 5.0mm (normal: 3, 4 to 4.5 mm) [2].

Several pathophysiological mechanisms have been suggested: A stop in the development or abnormal insertion of the secondary fibers of the lens, which can be linked to a nutritional deficiency due to defects in tunica vasculosa lentis and which can occur in the fifth and sixth months of embryonic life, the lens normally being spherical at this point. Zonular fibers can also be rudimentary due to lack of tension and stopping lens development, so they can remain spherical instead of gradually turning into a normal biconvex shape [1]

Glaucoma is the most common complication of this disease, which threatens vision. Acute angle closure may result from pupillary blockage caused by anterior bowing or subluxation of the lens due to zonular hyperlaxity. [3]

Microspherophakia can lead to chronic glaucoma due to recurrent attacks of pupillary blockage, resulting in synechial closure of the angle [4]. Bilateral angle closure glaucoma is an unusual and uncommon occurrence, the differential for ACG are limited to medication, snake venom, general anesthesia, and zonular or lenticular abnormalities [5]. No standard surgical technique has been adopted due to the variability of the laxity of the zonular fibers [6]. Implantation of intraocular lenses is not always possible due to the absence of a stable capsular support, especially after complicated surgery [7,8]. Also, the implantation of IOL in the capsular bag is questionable due to the post-operative instability of the implant, the unavailability of a small capsular tension ring and the high rate of contraction of the capsular bag. [6]

The main indications for clear lens extraction in patients with microspherophakia are: corneo-lenticular contact, high unilateral myopia, pupillary blockage and secondary glaucoma refractory to medical treatment [7]. Our patient underwent clear lens extraction due to progressive course of angle closure and glaucomatous neuropathy. Scleral fixation intra ocular lens was implanted.

The management of microspherophakia is still under debate. Medical and laser treatment fail in about 60% of the eyes with this condition. Lens extraction remains the first choice if medical treatment and laser iridotomy fail. Kaushik et al [7] described an adult patient with bilateral angle-closure glaucoma with microspherophakia and who's intra-ocular pressure (IOP) was successfully controlled by extraction of the lens and anterior vitrectomy. Kanamori et al. [8] also described a patient with microspherophakia and chronic angle-closure glaucoma who's IOP was well controlled by goniosynechiolysis and lens extraction. Although described by some authors (Khokhar et al, 2012) [9] the use of the capsular tension ring is not the treatment of choice because it is most often a small capsular bag. Scleral fixation represents an alternative (Fan et al, 2003) [10] which have been successfully achieved in some patients.

In our case we were not able to place the intraocular implant in the capsular bag in both of eyes, SFIOL was done. This surgical intervention of RLE allowed the correction of the high myopia in the spherophakia. However, they are only recommended in patients with good anterior chamber depth and compliance with the regular eye exam.

Conclusion

The presence of angle-closure glaucoma associated with high myopia in young subjects should prompt the clinician to diagnose microspherophakia.

The optimal management of glaucoma in microspherophakia is still uncertain. Several factors are responsible for the development of glaucoma in microspherophakia.

Microspherophaka patients should be closely monitored to determine the appropriate method for treating their glaucoma.

References

1.Karmoun S*, Aboulanouar A, Hajji Z, Elmarzouqi B, Elhassan A and Berraho A; Service d'ophtalmologie B, Hôpital des spécialités de Rabat, Morocco.

2. Tindall R, Jensvold B. Microspherophakia In: Roy FH, Fraunfelder FW, Fraunfelder FT (eds.) Roy and Fraunfelder's Current Ocular Th erapy , Sixth Edition. 2008; Chapter 303: 559-560, USA, Elsevier.

3. Chan RT, Collin HB. Microspherophakia. Clin Exp Optom. 2002; 85: 294–299.

4. Nelson LB, Maumenee IH. Ectopia lentis. Surv Ophthalmol. 1982; 27: 143– 160.

5. J T Banta 1, K Hoffman, D L Budenz, E Ceballos, D S Greenfield et al. Presumed topiramte induced bilateral acute angle closure glaucoma.Am J Ophthalmol.2001;132:112-114

6. Al-Haddad C, Khatib L. Vitrectorhexis and lens aspiration with posterior chamber intraocular lens implantation in spherophakia. J Cataract Refract Surg. 2012; 38: 1123-1126.

7. Kaushik S, Sachdev N, Pandav SS, Gupta A, Ram J. Bilateral acute angle closure glaucoma as a presentation of isolated microspherophakia in an adult: case report. BMC Ophthalmol. 2006; 6: 29.

8. Kanamori A, Nakamura M, Matsui N, Tomimori H, Tanase M, Seya R, et al. Goniosynechialysis with lens aspiration and posterior chamber intraocular lens implantation for glaucoma in spherophakia. J Cataract Refract Surg. 2004; 30: 513–516.

9. Behndig A. Phacoaspiration in spherophakia with corneal touch.J Cataract Refract Surg. 2002; 28: 189–191.

10. Fan DS, Young AL, Yu CB, Chiu TY, Chan NR, Lam DS. Isolated microspherophakiawith optic disc colobomata.J Cataract Refract Surg. 2003; 29: 1448-1452.

Original Article

Phacomatosis pigmento vascularis, my experience

S M Noman[1], U S Akbar[2], Z S Shahid[3]

Abstract

Purpose: To observe and describe a rare disease Phacomatosis pigmento vascularis as case series

Method : Detailed history taking and examination was done in the glaucoma clinic of Chittagong Eye Infirmary and Training Complex for the diagnosis of those suspected cases. Detailed systemic examinations of skin, cardiac and nervous system were done. Ocular examinations included visual acuity. intraocular pressure. Gonioscopy (while possible), torch light and slitlamp examination and fundus evaluation were done.

Result : Total of 8 cases were included. Five male and three female. Average age group was 4 +/-2 years. All of them had haemangiomatous lesion in the hand and feet. 5 of them had haemangioma in the both side of the face. 5 cases had bilateral and rest two has unilateral naevus of ota in the eye. Three cases had bilateral and rest five cases had unilateral mild buphthalmos and raised intraocular pressure (iop). There was history of convulsion in two cases and CT brain revealed calcification in the brain. All cases needed trabeculectomy surgery to reduce IOP. Two cases needed Ahmed valve implant.

Conclusion : Phacomatosis pigmento vascularis is a rare entity. Early diagnosis and appropriate management can save their vision as well as life

Key words : Haemangioma.Phacomatosis.Buphthalmos

Introduction

Phacomatosis pigmento vascularis (PPV) is a disorder characterized by the co-existence of vascular and pigmentary birthmarks.[1] Signs and symptoms may include port wine stain, melanocytic nevi (commonly known as moles), epidermal nevi, dermal melanocytosis (areas of blue-gray discoloration), nevus spilus and patches of hyperpigmentation (areas of darker skin). Other skin features may include nevus anemicus (areas of lighter skin) and café au lait spots.[2] About half of people with PPV have systemic involvement which means they have features affecting other areas of the body. People with systemic involvement may have neurologic, ocular (eye) or muscular abnormalities.[2,3] Several subtypes of PPV have been identified which are generally distinguished based on the specific type(s) of skin features present.[1,2]

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Method

This is a observational case series study done for the last 7 years at the glaucoma clinic of ceitc. Patient presented in the paediatric outdoor with vascular malformation in the face were referred to the glaucoma clinic for detail evaluation. In glaucoma clinic history taking like ocular pain photophobia, headeche, convulsion were taken and some examinations like vision refraction, pupilary reaction, CCT IOP measurement by digitally or by applanation tonometry, torchlight and or slit lamp examination and fundoscopy were done. Those patient had vascular malformation in and around the face we checked for oculo culaneous naevus, conjuctival vascular malformation and megalocornea. We did fundoscopy to exclude any glaucomatous change in the disc or haemangioma in the retina and choroid. Fundus photography was done where possible. CT scan of the brain was done while there was a history of convulsion and while it is affordable to the patient. Raised intraocular pressure was managed by trabeculectomy surgery. Tube implantation was done in the failed cases

Result

Among total 8 cases five of them were male and three female. Average age group was 4 +/-2 years. Older one was 6 years and younger was 2 years of age. All of them had haemangiomatous lesion in the hand and feet. But the oldest one had massive haemangioma in the trunk also associated with pachy naevus all over the body.5 of them had haemangioma in the both side of the face Three of them had uper lid swelling in the affected side. 5

cases had bilateral and rest two has unilateral naevus of ota in the eye those were distrubuted following a dermatome of frontal and maxillary division of trigeminal nerve. Three cases had bilateral and rest five cases had unilateral mild buphthalmos and raised IOP. Average IOP was 25=/-5 mmHg where it was possible to be measured. IOP was more where haemangiomatous lesion were more prominent. Two cases there was history of convulsion and CT brain revealed calcification in the brain. All of them were treated with brinzolamide and timolol combination two times daily for reduction of IOP but was not possible to be reduced. Fundus examination revealed c:d of average.8 except two case where it was almost full cupping. All cases needed trabeculectomy surgery to reduce IOP. Two cases needed Ahmed valve implant. Two cases IOP was not controlled where ahmed valve implantation has been planned

fig-1-2 : Haemangiomatous changes in the both side of the face with buphthalmos in the right side

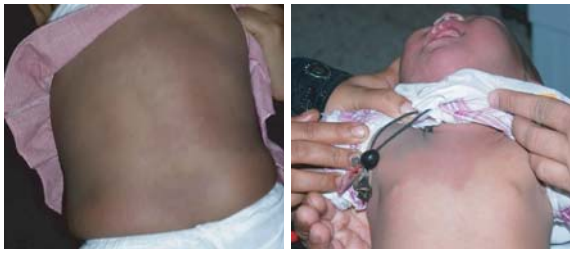

Fig-3-4 : Haemangiomatous lesion in the chest and trunk

Discussion

Isolated PPV is typically a sporadic disorder that occurs for the first time in people with no family history of PPV.[4] Researchers have found that PPV can be caused by a somatic mutation in the GNA11 or GNAQ gene that is present only in the affected tissues of the body. These mutations are not present in the blood or in unaffected tissues, which means the disorder is likely due to non-inherited mutations that are randomly acquired after conception. In some cases of isolated PPV the underlying cause remains unknown.[1]

There is no family history of such disease in our cases and we did not do any genetic analysis.

Treatment and long-term outlook (prognosis) of PPV largely depends whether there is systemic involvement and which body parts or organ systems are affected.[3,4] Isolated PPV without systemic involvement typically does not require treatment. However, large skin lesions may cause problems with body image and self-esteem, so laser treatments may be considered to improve the appearance of skin. In our cases we referred the patient to the skin specialist and they were under observation.

Characteristic signs and symptoms of phacomatosis pigmento vascularis (PPV) involving the skin include port wine stain and various pigmentary lesions (lesions that are brown, black or blue in color). The port wine stain and pigmentary lesions may be extensive, affecting several areas of the body, including the face. Examples of associated pigmentary lesions include:[2,3]

- Melanocytic nevi Epidermal nevi
- Nevus anemicus (areas of lighter skin)
- Hyperpigmentation (areas of darker skin)
- Cafe-au-lait spots
- Dermal melanocytosis (areas of blue-gray discoloration)
- Nevus of Ota

In our series they were presented with vascular malformation in the face and trunk and also in the limb as well as with naevus over face and in the eye(conjunctiva) Around half of people with PPV have systemic involvement (i.e., body systems other than the skin are affected). Eye conditions such as ocular melanosis (also called ocular melanocytosis) are common. Ocular melanosis refers to a blue-gray pigmentation in the white of the eye (the sclerae). This condition often occurs along with nevus of Ota and may affect one or both eyes.[5] In our series 5 cases had bilateral and two cases had unilateral naevus in the face and eye. Naevus follows the dermatom of T1 and T2.

Eye naevus is subconjuctival melanocytosis in nature. Complications of nevus of Ota include glaucoma and melanoma, so people with nevus of

Ota require careful examination and follow-up by an ophthalmologist.[7] Other eye conditions reported in PPV include iris hamartomas, iris mammillations, and iris nodules.[7] When neurologic abnormalities are present they usually become apparent in the first few months of life and may include developmental delay, seizures, intracranial calcifications (calcium deposits within the brain), or cerebral atrophy.[3] Some people with PPV also have Sturge-Weber syndrome or Klippel-Trenaunay syndrome, each of which causes various signs and symptoms.[8]

Two of our cases had cerebral calcification in the CT scan. In almost all of our cases there was secondary glaucoma. The pathogenesis is combined. Developmental anomaly, raised episcleral venous pressure and melanocyte accumulation in the angle were the mechanism. We did goniscopy in two cases where we found angle anomaly and hyperpigmentation of the angle.

A variety of other signs or symptoms have been reported in individual cases of PPV (e.g., primary lymphedema, renal angiomas, moyamoya disease, scoliosis, malignant colonic polyposis, hypoplastic larynx, multiple granular cell tumors, and selective IgA deficiency).[9] In our series we, did not get such association so far

If phacomatosis pigmento vascularis (PPV) is not associated with systemic complications (e.g., Sturge-Weber syndrome, Klippel-Trenaunay syndrome, neurological problems, or eye conditions) it typically does not require treatment. However, because large skin lesions may cause problems with body image and self-esteem, parents of children with PPV, or adults with PPV, may consider laser treatments to improve the appearance of skin lesions.[5,6]

Glaucoma in such cases usually refractory to medicine and also standard trabeculectomy. So trabeculectomy with antimetabolites is comparatively better option. But there is no published report over it. But in the sturge weber syndrome trabeculectomy with mmc yields good result. In our series 4 cases were doing good with trabeculectomy with mmc but in other cases iop was raised again and we did ahmed valve implantation in two cases and two cases we have planned to do so. We are now following those cases for final outcome of the surgery.

Conclusion

Phacomatosis pigmento vascularis is a rare entity. Combined management from dermatologist, neorologist and ophthalmologist are needed to handle those multisystem complex cases. Uncontrolled intraocular pressure may cause blindness. Early diagnosis and appropriate medical and surgical management is mandatory to save the eye from blindness.

References

1. Thomas AC, Zeng Z, Rivière JB, et al. Mosaic Activating Mutations in GNA11 and GNAQ Are Associated with Phakomatosis Pigmentovascularis and Extensive Dermal Melanocytosis. J Invest Dermatol. April, 2016; 136(4):770-778. https://www.ncbi.nlm.nih.gov/pubmed/26778290.
2. Galbraith S. Capillary malformations (port wine stains) and associated syndromes. UpToDate. Waltham, MA: UpToDate; November, 2016; https://www.uptodate.com/contents/capillary-malformations-port-wine-stains-and-associated-syndromes.
3. Diaz LZ. Vascular lesions in the newborn. UpToDate. Waltham, MA: UpToDate; April, 2018; https://www.uptodate.com/contents/vascular-lesions-in-the-newborn.
4. Nanda A, Al-Abdulrazzaq HK, Habeeb YK, Zakkiriah M, Alghadhfan F, Al-Noun R, Al-Ajmi H. Phacomatosis pigmentovascularis: Report of four new cases. Send to Indian J Dermatol Venereol Leprol. May-June, 2016; 82(3):298-303. https://www.ncbi.nlm.nih.gov/pubmed/27088932.
5. Fernández-Guarino M, Boixeda P, de Las Heras E, Aboin S, García-Millán C, Olasolo PJ. Phakomatosis pigmentovascularis: Clinical findings in 15 patients and review of the literature. J Am Acad Dermatol.. 2008 Jan; Epub 2007 Nov 28;
6. Kono T, Erçöçen AR, Chan HH, Kikuchi Y, Hori K, Uezono S, Nozaki M. Treatment of phacomatosis pigmentovascularis: a combined multiple laser approach. Dermatol Surg. June, 2003; 29(6):642-646. https://www.ncbi.nlm.nih.gov/ pubmed/ 12786710.
7. Chekroun-Le Du L et al., Phacomatosis pigmentovascularis type II. European Journal of Dermatology. 1998; http://www.john-libbey-eurotext.fr/en/print/e-docs/00/01/87/6B/article.phtml. Accessed 2/1/2011.
8. Sen S, Bala S, Halder C, Ahar R, Gangopadhyay A. Phakomatosis pigmentovascularis presenting with sturge-weber syndrome and klippel-trenaunay syndrome. Indian J Dermatol. January-February, 2015; 60(1):77-79. https://www.ncbi.nlm.nih.gov/pmc/ articles/PMC4318068/.
9. Narchi H et al.,. Picture of the month. Arch Pediatr Adolesc Med. 2001; http://archpedi.ama-assn.org/cgi/content/full/155/2/191. Accessed 2/1/2011.

Lens induced Glaucoma - Review Article

S M Noman[1], S A Wadud[2], M S I Prodhan[3]

Abstract

Lens–related elevation in Intraocular pressure results from a variety of mechanisms such as lens dislocation, lens swelling (intumescent cataract), inflammation due to phacoanaphylaxis and lens particle blocking the trabecular meshwork. This review was done with the aim of studying the types of lens-induced glaucoma, their clinical features and current management.

Key words: Lens dislocation, lens swelling, lens-induced glaucoma.

Introduction

Lens induced Glaucoma, one of the commonest cause of secondary Glaucoma due to senile cataract mandates an early recognition and management to prevent blindness.[1]

Lens-related elevation in intraocular pressure results from a variety of mechanisms such as lens dislocation, lens swelling, inflammation due to phacoanaphylaxis and lens particle blocking the trabecular meshwork.

A study of these conditions is imperative, since improper management leads to frequent complications and poor visual outcomes.

This review was done with the aim of studying the various types of lens-induced Glaucoma, their clinical features and current management. Apart from the textual literature, online search was conducted using search engines such as Google Scholar, PubMed and Elsevier.

Phacolytic Glaucoma

Phacolytic glaucoma is a condition characterized by leakage of lens contents through an intact capsule.[2,3,4]

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It is a principle complication of hypermature cataract. Hypermature cataract may cause leakage of lens protein from an intact capsule. The lens protein causes intense inflammation and blockage of trabecular meshwork, subsequently responsible for elevation of IOP.[5]

The presentation is with an acute onset of pain and redness. The signs include congestion of the eye, corneal edema, deep AC and a hypermature cataract. The AC shows a significant amount of flare. Clumps of lens material may also be seen in the AC (Fig-1).

The majority of patients with phacolytic glaucoma can be managed through topical cycloplegia, topical steroids and aqueous suppressants. Manual small incision cataract surgery has proven successful in the management of phacolytic glaucoma.

Phacomorphic Glaucoma

This condition is caused by a swollen, cataractous lens causing pupillary or angle block and elevating IOP.[6]

In this type of Glaucoma, the swollen lens may block the anterior flow of the aqueous humour from the posterior chamber pushing the iris forward. Eventually, the trabecular meshwork gets blocked by the iris and leads to a sudden and extreme rise in IOP.[7]

The diagnosis of phacomorphic glaucoma (Fig-2) is made on the presence of pain and redness, shallow anterior chamber, corneal oedema and increased IOP with intumescent lens.

Initially, IOP-lowering medications are used. Topical beta blockers, carbonic anhydrase inhibitors and hyperosmotic agents are the mainstay of medical treatment. After IOP control and establishing intraocular inflammation, we can proceed for cataract extraction (Fig-3).

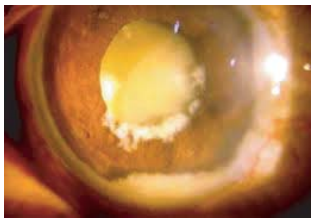

Fig-1:Phacolytic Glaucoma

Fig-2:Phacomorphic Glaucoma

Fig-3:After cataract surgery

Lens – particle Glaucoma

It is associated with a disrupted capsule and the presence of obvious fragments of lens material in the anterior chamber. It may occur after cataract surgery, trauma to the lens, or Nd:YAG posterior capsulotomy. The IOP increase is due to the obstruction of the aqueous outflow by the lens particles.[8]

The patient reports with sudden pain and redness in the affected eye following trauma or surgery. On examination, the cornea is often oedematous, there are fluffy lens particles floating in the AC cataractous lens with ruptured capsule is present.

Initially, a trial of medical anti-glaucoma therapy maybe attempted. Mild to moderate steroid therapy can help to prevent synechiae, pupillary membrane, cystoid macular oedema, etc. If the glaucoma is severe and/ or there is a large amount of lens material in the anterior chamber, its surgical removal should be undertaken.

Phacoanaphylactic Glaucoma

It is a granulomatous, immune-mediated inflammation to lens proteins. The term phacoanaphylaxis is misleading since anaphylaxis requires mast cells, basophils and immunoglobulin E, none of which are present during this process.[9]

Capsule rupture leads to exposure of the eye to lens antigens which are antigenically privileged. When the eye is exposed to these antigens, a severe antibody response involving phacomorphonuclear leukocytes, lymphoid, epithelioid and giant cells occurs. These inflammatory cells may block the trabecular meshwork and elevate IOP.[10]

The clinical signs include lid oedema, chemosis, conjunctival congestion, corneal oedema, AC reaction, posterior synechiae and mutton fat keratic precipitates.

In the past, the differentiation between phacoanaphylaxis and sympathetic ophthalmia was confusing, but once it is understood that the former involves only the anterior segment of the eye, the diagnosis is easy.[11,12]

The first step is to control IOP and inflammation medically. If unsuccessful, then the next step is surgical removal of the remaining lens material.[13]

Epidemiology: prognostic factors-

There are 2 studies from the Indian Journal of ophthalmology and Nepal Journal of Ophthalmology that investigate lens-induced glaucoma, particularly phacolytic and phacomorphic glaucoma.

The first study[14] deals with the frequency and types of lens-induced glaucoma, the reasons for late presentation, and the outcome of current management. The percentage of phacomorphic glaucomas was higher in this study (72%) than the phacolytic (28%). A similar result occurred in relation to gender since the ratio of females: males was 1.7:1. In this study, 38.6% achieved a visual acuity of 6/60 or better, 31.2% less than

6/60, and 30.2% less than 3/60. In the phacolytic group, 65.9% had a poor outcome compared with 59.8% of the phacomorphic group. This poor outcome was associated in the latter group with a longer distance from the hospital, longer duration of pain, and higher IOP at presentation. In the phacolytic group, only the distance was found to influence negatively the visual outcome. In this series, the final visual outcome is worse than in other studies, probably because the majority of the patients reported later than ten days after the onset of pain.

The second study[15]dealt with the age and sex distribution, causes for delayed presentation, immediate post-operative visual outcome and the reasons for poor visual outcome. The percentage of phacomorphic cases (57.5%) was higher than the phacolytic glaucoma (42.5%). Female to male ratio was 2.1:1. The majority of patients (57%) presented after 2 weeks of symptoms and the reason for late presentation in more than half of the patients (52.5%) was financial constraints. Following surgery, 90% had an IOP less than 21 mm Hg at discharge. Visual acuity was either hand-movement or just perception of light in all eyes before surgery. At discharge, 65% of patients achieved a visual acuity of 6/60 or better, 5% had less than 6/60 and 30% less than 3/60. The reasons for poor VA in these patients were optic atrophy, uveitis, macular cause and corneal edema.

Conclusion

This review shows that lens-induced glaucoma can have various modes of mechanism and presentation. It is imperative to identify the correct type of lens-induced glaucoma and manage it accordingly. There is a need to educate both the patient and the cataract surgeon of the dangers of lens-induced glaucoma and of the poor outcome if treatment is delayed.

Conflict of interest

The author reports no conflict of interest in this work.

References

1. Gifford H (1900). The dangers of the spontaneous cure of senile cataract. Am J Ophthalmol; 17:289–293.
2. Shields MB. Glaucomas associated with disorders of the lens. In: Shields MB, editor. Textbook of glaucoma. 4th ed. Baltimore: Williams and Wilkins; 1998.
3. Kothari R, Tathe S, Gogri P, Bhandari A. Lens-induced glaucoma: the need to spread awareness about early management of cataract among rural population. ISRN Ophthalmol 2013; 2013: 581727.
4. Mavrakanas N, Axmann S, Issum CV, Schutz JS, Shaarawy T. Phacolytic glaucoma: are there 2 forms? J Glaucoma 2012; 21: 248-9.
5. Johns KJ, Feder RS, Hamill MB. et al. Lens and cataract. American Academy of Ophthalmology. 2002-2003.
6. Rajkumari V, Kaminibabu KS, Bhabanisana RD, Victor R. Manual small incision cataract surgery in phacomorphic glaucoma: Surgical technique and outcome in North-eastern India. J Curr Glaucoma Pract 2013; 7: 43-8.
7. Jonathan PE, Ellant, Stephen A. et al. Lens induced glaucoma. Documenta Ophthalmia. 1992; (81):317–38.
8. Yanoff M, Scheie HG. Cytology of human lens aspirate. Its relationship to phacolytic glaucoma and phacoanaphylacticendophthalmitis. Arch Ophthalmol. 1968;80:166–170.
9. Skuta GL, Cantor LB, Weiss JS. Basic and clinical science course. In: Intraocular inflammation and uveitis. San Francisco: American Academy of Ophthalmology; 2011.
10. Shields MB. Glaucomas associated with disorders of the lens. In: Shields MB, editor. Textbook of glaucoma. 4th ed. Baltimore: Williams and Wilkins; 1998.
11. Allen JC. Sympathetic uveitis and phacoanaphylaxis. Am J Ophthalmol. 1967;63:280–283.
12. Easom HA, Zimmerman LE. Sympathetic ophthalmia and bilateral phacoanaphylaxis. Arch Ophthalmol. 1964;72:9–15.
13. Riise P. Endophthalmitisphacoanaphylactica. Am J Ophthalmol. 1965;60:911–915.
14. Pradhan D, Hennig A, Kumar J, Foster A. A prospective study of 413 cases of lens-induced glaucoma in Nepal. Indian J Ophthalmol. 2001;49:103–107.
15. Pant Sitoula et al. Lens induced glaucoma in eastern Nepal. Nepal J Ophthalmol 2016; 8(16): 161-166.

Case Presentation: Bilateral Angle-Closure Glaucoma

S M Noman[1], M Z Hassan[2], N Sultana[3], A Siddika[4]

Abstract

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

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

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

Key words: Angel closure glaucoma, iridectomy, intraocular pressure

Introduction

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

Case History

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

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

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

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

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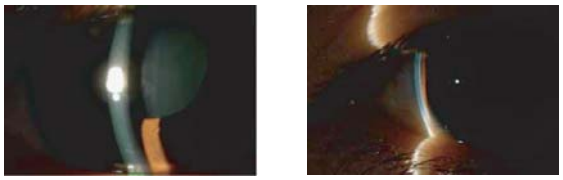

Figure 1. A slit-lamp photograph of the patient's right eye showed a fixed, middilated pupil (A) and shallowing of the central and peripheral anterior chamber (B).

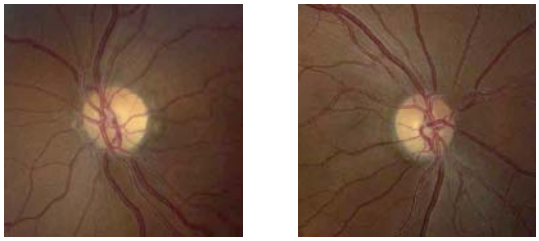

Figure 2. Fundus photographs of the patient's right (A) and left (B) eyes showed pathological changes in the retinal nerve fiber layer secondary to acute ACG.

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

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

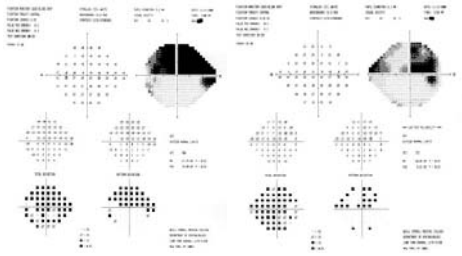

Figure 3. Perimetry showed superior visual field defects in the patient's right (A) and left (B) eyes. The defect in the left eye cor- related with a notch in the left optic nerve.

Outcome

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

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

Discussion

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

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

Microspherophakia (the presence of a spherical lens with a reduced equatorial diameter) is associated with systemic diseases such as Weill-Marchesani syndrome, Marfan syndrome, homocysteinuria, Klinefelter syndrome, Alport syndrome, and Meyer-Schwickerath-Weyers syndrome[8-12]. Investigators have hypothesized

that spherophakia occurs when an incompletely developed ciliary body and its loose elongated zonules do not exert sufficient pressure to flatten the developing lens.

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

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

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

Conclusion

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

References

1. Banta JT, Hoffman K, Budenz DL, et al. Presumed topiramate induced bilateral acute angle closure glaucoma. Am J Ophthalmol. 2001;132:112-114.
2. Craig JE, Ong TJ, Louis DL, Wells JM. Mechanism of topiramate-induced acute onset myopia and angle closure glaucoma. Am J Ophthalmol. 2004; 137(1):193-195.
3. de Guzman MH, Thiagalingam S, Ong PY, Goldberg I. Bilateral acute angle closure caused by supraciliary effusions associated with venlafaxine intake. Med J Aust. 2005;182(3):121-123.
4. Ates H, Kayikcioglu O, Andac K. Bilateral angle closure glaucoma following general anesthesia. Int Ophthalmol. 1999;23:129-130.
5. Srinivasan R, Kaliaperumal S, Dutta TK. Bilateral angle closure glaucoma following snake bite. J Assoc Physicians India. 2005;53:46-48.
6. Willoughby CE, Wishart PK. Lensectomy in the management of glaucoma in spherophakia. J Cataract Refract Surg. 2002;28:1061-1064.
7. Kaushik S, Sachdev N, Singh S, et al. Bilateral acute angle closure glaucoma as a presentation of isolated microspherophakia in an adult: case report. BMC Ophthalmol. 2006;6:29-35.
8. Johnson GJ, Bosanquet RC. Spherophakia in a Newfoundland family: 8 years' experience. Can J Ophthalmol . 1983;18:159-164.
9. Macken PL, Pavlin CJ, Tuli R, Trope GE. Ultrasound biomicroscopic features of spherophakia. Aust N Z J Ophthalmol. 1995;23(3):217-220.
10. Nelson LB, Maumenee IH. Ectopia lentis. Surv Ophthalmol. 1982;27:143-160.
11. Johnson VP, Grayson M, Christian JC. Dominant microspherophakia. Arch Ophthalmol. 1971; 85:534-542.
12. Widder RA, Engels, B, Severin M, et al. A case of angle-closure glaucoma, cataract, nanophthalmos, and spherophakia in oculo-dento-digital syndrome. Graefes Arch Clin Exp Ophthalmol. 2003;241(2):161-163.
13. Dietlein TS, Mietz H, Jacobi PC, Krieglstein GK. Spherophakia, nanophthalmia, hypoplastic ciliary body, and glaucoma in brachydactyly-associated syndromes. Graefes Arch Clin Exp Ophthalmol. 1996;234:187-192.
14. Ritch R, Wand M. Treatment of the Weill-Marchesani syndrome. Ann Ophthalmol. 1981;13:665-667.
15. Kanamori A, Nakamura M, Matsui N, et al. Goniosynechialysis with lens aspiration and posterior chamber intraocular lens implantation for glaucoma in spherophakia. J Cataract Refract Surg. 2004;30:513-551.
16. Campbell DG, Vela A. Modern goniosynechialysis for the treatment of synechial angleclosure glaucoma. Ophthalmology.1984;91:1052-1060.
17. Teekhasaenee C, Ritch R. Combined phaco-emulsification and goniosynechialysis for uncontrolled chronic angle-closure glaucoma after acute angle-closure glaucoma. Ophthalmology. 1999;106:669-674.


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