ILLINOIS COLLEGE OF OPTOMETRY

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Multidisciplinary Care in a Patient with Ablepharon Macrostomia Syndrome

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BACKGROUND

Ablepharon macrostomia syndrome (AMS) is an incredibly rare condition characterized by absent or extremely short eyelids and a large fish-like mouth. The lack of eyelids results in exposure keratopathy and severe visual impairment from a young age[1]. It is caused by a dominant genetic mutation involving mesenchymal tissue development, particularly in the craniofacial region. Systemic clinical features include rudimentary ears with attached lobes, absent or rare body hair, thin and redundant skin, absent nipples, a distended abdomen, and genital ambiguity[1,2]. We present a case of a patient with AMS in which multidisciplinary care, including vision rehabilitation, was used to provide comprehensive evaluation.

CASE SUMMARY

Primary Care Pediatric Exam:

Case History:
- 5 year old African American female presented for kindergarten eye exam
- Birth History: Alcohol use during pregnancy, full term birth (37 weeks), Cesarian section, birth weight in 3rd percentile
- Medical History: Ablepharon macrostomia syndrome (see Photos 1 & 2). Patient was unable to eat solid foods and was on a puree and liquid diet.
- Ocular History: Bilateral penetrating keratoplasties and partial postauricular tarsorrhaphies at 1 month old.
  - o Multiple ocular exams under anesthesia, with last eye exam 2 years ago
  - o Guardian reported an ability to see colors out of OD and minimal vision OS

Visual acuity: see Table 1
Gross oculomotor evaluation: congenital nystagmus OU
Gross anterior segment evaluation:
- Partial tarsorrhaphies OU
- Corneal opacification OU (see Photo 3)
- Ear malformation s/p postauricular tarsorraphies (see Photo 4)

Tactile intraocular pressure: soft and equal OD/OS
Plan: Refer to vision rehabilitation for further visual evaluation and to pediatric ophthalmology (corneal specialist) for an ocular health exam.

Vision Rehabilitation Exam (1 month later):

Further Case History:
- Preparing to attend kindergarten with an individualized education plan (IEP)
- Good mobility, self feeding, and good hearing
- Slight cognitive delay
- Accurate color naming and detection of light/dark changes, per guardian

Visual acuity: see Table 1
Plan: The patient was diagnosed with legal blindness. The following education plan recommendations were made:
- Teacher of the Visually Impaired
- Assistive Technology Specialist
- Orientation & Mobility Specialist
- Explore audio and tactile reading for primary reading mode
- Explore electronic magnification for visual recognition of letters, numbers, and shapes

PHOTO 1 & 2
Ablepharon Macrostomia Syndrome

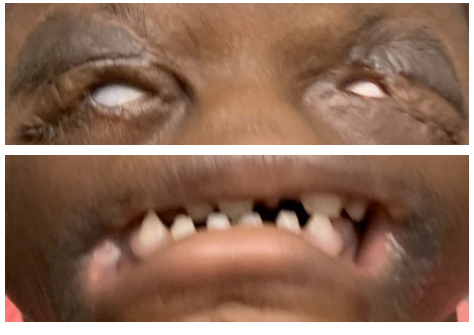

PHOTO 3
Corneal Opacification

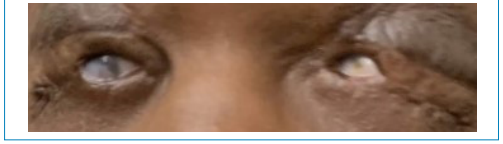

PHOTO 4
Ear Malformation

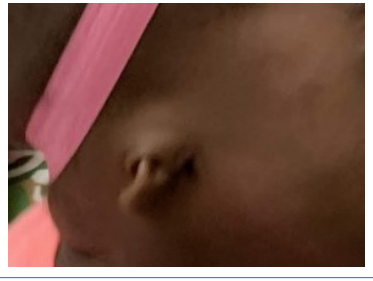

TABLE 1

Primary Care Pediatric Exam	Pediatric Low Vision Exam
Light projection OU (unable to evaluate each eye individually due to resistance to occlusion)	Light projection in all quadrants OU with hand motion ability OD • (-)ability to read numbers or letters • (+)ability to name primary colors • (+)use of relative distance magnification for visual exploration of objects

DISCUSSION

AMS is extremely rare, with only 15 cases being described in the literature[1]. As a result, the current information about ocular management is largely anecdotal. This patient's corneas were treated with an unsuccessful amniotic membrane graft followed by penetrating keratoplasties and partial tarsorraphy[2]. Cruz et al. achieved successful treatment in 3 patients with an alternative treatment in which the eyelids were lengthened using a skin graft. These three patients maintained largely healthy corneas with minimal opacity[1].

The authors were unable to find mention of visual rehabilitation referrals for these patients in the literature, though one article did outline the need to treat AMS holistically[1-4]. De Maria et al. urged physicians to address the psychosocial impacts of AMS[4] as well as the physical impacts. Eye care providers must do the same by addressing the patient's visual behavior as well as ocular health.

CONCLUSION

This case illustrates the importance of multidisciplinary care when evaluating patients with visual impairment. Through a vision rehabilitation exam, the true extent of the patient's visual performance was discovered, and it was ensured that this child receives necessary services in school. This will be essential to her continued visual, cognitive, and academic development.

RESOURCES

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CONTACT INFORMATION

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"50 Shades of Grey": Managing Sibling Cases of Achromatopsia

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BACKGROUND

Achromatopsia is a congenital cause of vision loss due to isolated cone photoreceptor dysfunction. Along managing symptoms of extreme photophobia and functional visual impairment in these patients, it is equally important to address implications on classroom learning and the risk of downstream inheritance.

CASE HISTORY

- A sibling pair (7yo male and 5yo female) of Hispanic descent presented for an initial low vision exam
- Both patients complained of extreme photophobia and difficulty seeing the board during school, with only one sibling (male) possessing transition tinted spectacles.
- Both are enrolled in a regular classroom setting with no current learning modifications.

EXAM RESULTS

Examination found the following for both patients: (See table 1)

TABLE 1

	Patient #1 (7yoM)	Patient #2 (5yoF)
BCVA	OD: 20/125 (Snellen) OS: 20/125 (Snellen)	OD: 20/300 (Lea match) OS: 20/250 (Lea match)
Functional Near Acuity	0.15cm/0.5M	0.15cm/1.5M
EOM, Pupils, Fields	Full, ERRL, (-)APD , FTFC (+)pendular nystagmus	Full, ERRL, (-)APD , FTFC (+)pendular nystagmus
Color (Color Vision Made Easy)	7/9 (CVME), unable to complete HRR diagnostic	6/9 (CVME), unable to complete HRR diagnostic
Refractive Error	OD: +5.00 -0.75 x180 OS: +4.50 -0.75 x180	OD: +5.25 -1.25 x180 OS: +4.75 -1.25 x180
Anterior Segment	Within normal limits	Within normal limits
Posterior Segment	See figures 1-3	See figures 1-3

FIGURE 1A-1B
Fundus Photos OD, OS (7yoM)

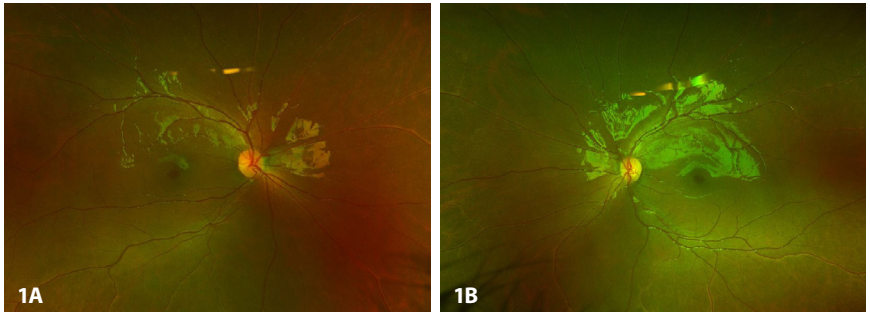


FIGURE 2A-2B
Macula OCT OD, OS (7yoM)

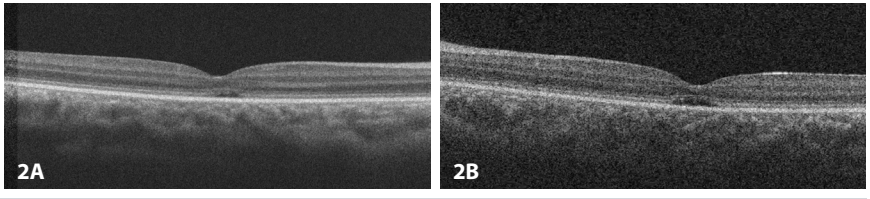
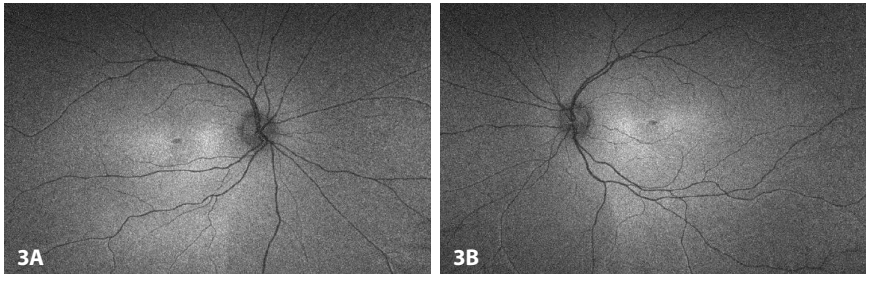


FIGURE 3A-3B
Fundus Auto-fluorescence OD, OS (7yoM)



DIAGNOSIS

Achromatopsia , Category 1 Visual Impairment OD/OS

TREATMENT AND MANAGEMENT

- Visual aids including: Transition tinted spectacles, 4x distance telescope, 65mm dome magnifier, and Cocoon Fitovers (Grey and Amber), Assistive Technology (CCTV); all devise use demonstrated with aid of eldest brother
- Extensive education with the mother using a Spanish interpreter followed to highlight process of establishing an IEP with current educators (including orientation and mobility training) and inform about the increased risks of having more children with achromatopsia downstream due to being a carrier for the gene.

CONCLUSION

Actively engaging family members in the management of the patients' functional visual needs can help better understand the extent of the visual impairment. It is especially important in cases where there is a language barrier to ensure that the parents understand the need to advocate for their children's functional needs inside and outside the classroom. Lastly, genetic testing should be considered to assess future risks of inheritance in families with higher frequency of occurrence of Achromatopsia.

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Exploring a Case of Convergence Insufficiency in a Young Teenager with Limb Girdle Dystrophy

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BACKGROUND

Limb Girdle Muscle Dystrophy (LGMD) is a heterogenous group of diseases that involve the shoulder and hip girdle muscles, often sparing the facial muscles. Unlike myotonic dystrophy where ocular findings are commonly described, only a few cases have reported incidental strabismus, nystagmus, and ptosis in LGMD associated with genetic defects. In this case, we explore the visual symptoms and therapy course of a young patient with limited fine dexterity and mobility secondary to LMGD.

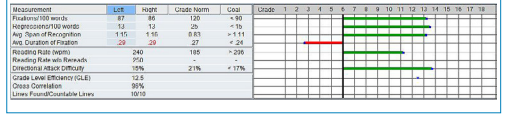
CASE HISTORY

- A 11 year old female presented with symptoms of increased trouble copying tasks from board to paper and overall increased lack of interest in completing homework after school.
- Ocular History: Refractive Amblyopia OD>OS
- Medical History: LMGD, hyperthyroidism, scoliosis, and ganglioneuroblastoma, rendering her paraplegic with limited fine motor dexterity.
- She recently transferred from a homeschool environment to a private school where she is currently integrated into a regular classroom setting.

EXAM FINDINGS

- Initial evaluation revealed vergence and accommodative dysfunction but normal ocular motor function: convergence insufficiency (indicated by a large exophoria at near and insufficient fusional ranges), decreased monocular accommodative amplitudes, age appropriate DEM and Visagraph (see Table 1).

FIGURE 1
Visagraph – Pre vision therapy



Measurement	Left	Right	Grade Norm	Goal
Fixations/100 words	87	86	120	< 90
Regressions/100 words	13	13	25	< 15
Avg. Span of Recognition	1.15	1.15	0.83	> 1.11
Avg. Duration of Fixation	.29	.29	.27	< .24
Reading Rate (wpm)	240		185	> 236
Reading Rate with Retrevals	250			
Directional Attack Difficulty	15%		21%	< 17%
Grade Level Efficiency (GLE)	12.5			
Cross Correlation	98%			
Lines Found/Countable Lines	10/10			

FIGURE 2
Functional Vision Report-Pre Vision Therapy

Analysis	Measurements	Goal
Fixation (Difference between the two eyes)	1%	< 10%
Regression (Difference between the two eyes)	0%	< 20%
Avg. Duration (Difference between the two eyes-mill sec.)	.00	< .01
Cross Correlation	96%	> 90%
Excessive Saccades in Return Sweeps	2	< 6
Anomalies (Uns Eye Unit/ Forward)	1	< 4
Anomalies (Uns Eye Reverse)	0	< 4
Anomalies (Both Eyes Opposite Movement)	0	< 4
Number of Start Time Differences Between Eyes	39	< 10

FIGURE 3A
Vectogram

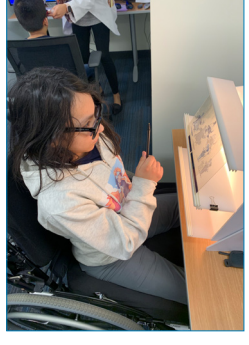


FIGURE 3B
3 Bead Brock String

TREATMENT AND MANAGEMENT

- The patient subsequently underwent a series of vision therapy sessions, with notable improvement in ability to concentrate at near after 7-8 weeks of weekly HVT and OVT.
- Techniques were all performed seated, with less emphasis on high tactile activities (VTS4/SVI/Vivid Vision). At follow up (week 8), she demonstrates increasing accommodative ability and fusional ranges. Her recent neuropsychology report per mother's disclosure reveals age appropriate cognitive function and processing ability.

TABLE 1

	Pre VT	8-10 weeks post VT
BCVA	20/20 OD/OS	20/20 OD/OS
EOM's, Pupils, Fields	ERRL, (-)APD, FTFC	ERRL, (-)APD, FTFC
Cover Test	6XP, 25XP	14XP, 20XP
Stereo	(+)randot, 80 sec Wirt	(+)randot, 20 sec Wirt
Accommodative Amplitudes	OD: 6.5D OS: 8D	OD: 11.0D OS: 10.5D
Accommodative Facility	OD: 9.5cpm OS: 10.5cpm	OD: 13.5cpm OS: 12.5cpm
Fusional Vergence Ranges	BI: X/14/12 BO: X/16/14	BI: X/16/10 BO: X/25/18
Vergence Facility (3BI/12BO)	13cpm	19cpm
DEM	Type I, H/V: 1.22	Type I, H/V: 1.22
Visagraph	See Figure 1	Unable to obtain

CONCLUSION

Vision therapy can still be an excellent strategy to improve sustainability in convergence and accommodative function despite physical handicap in an otherwise cognitively sound individual. As eye care providers, we contribute to the multidisciplinary approach in ensuring the overall wellbeing of our patients both inside and outside the classroom.

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GLOBAL SPECIALTY LENS SYMPOSIUM

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Scleral Lens Curricula in Optometric Education

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INTRODUCTION

As scleral lenses have become more widely available, schools of optometry have incorporated scleral lens education prescription and management into their contact lens curricula.

The purpose of this study is to describe characteristics of scleral lens education in optometric education.

METHODS

Contact lens educators (members of the AOCLE Association of Optometric Contact Lens Educators) were invited to complete an electronic REDCap survey regarding scleral lens curricula at their schools. The survey was available from 6/2019 – 8/2019. Data was summarized using descriptive statistics. This project was approved by the University of Illinois at Chicago IRB.

RESULTS

192 email invitations were sent to members of the AOCLE and 61 individuals completed the survey. Of those, 53 reported being actively involved in scleral lens education at their institution.

Educators reported:

- 9.8 [10.9] years (mean [SD]) experience fitting scleral lenses
- 8.6 [8.5] years teaching scleral lens prescription and management
- Personally completing 23.7 [35.1] clinical scleral lens evaluations/month

Optometric Educators allot 15% of their contact lens curriculum to ***Scleral Lenses*** utilizing 1/3 of their available fitting sets.

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SCOPE
Scleral Lenses in Current Ophthalmic Practice Evaluation

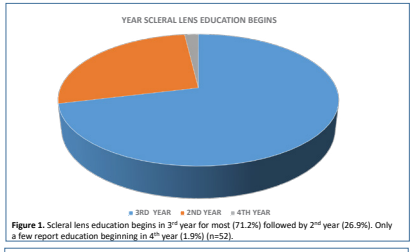

Figure 1. Scleral lens education begins in 3rd year for most (71.2%) followed by 2nd year (26.9%). Only a few report education beginning in 4th year (1.9%) (n=52).

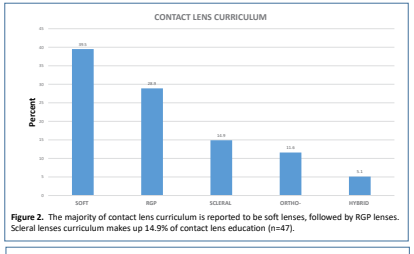

Figure 2. The majority of contact lens curriculum is reported to be soft lenses, followed by RGP lenses. Scleral lenses curriculum makes up 14.9% of contact lens education (n=47).

Educational Sources

Educators rank in-person continuing education (CE) as the most important source of scleral lens info (61%), while 22% consider laboratory consultants most important. Textbooks were the least important source by a majority of respondents (56%) (n=36).

Scleral Lens Fitting Sets

Respondents reported having access to 11.5 [2.2] scleral lens fitting sets in their school clinics (n=38). However, they reported using only 3.8 [2.2] fitting sets at least once per month (n=37).

CONCLUSIONS

- Contact lens educators at colleges of optometry report nearly a decade of experience fitting scleral lenses and rely heavily upon in-person CE to stay current in the field.
- Education on scleral lenses is generally introduced during the 3rd year of most optometry schools curricula.
- Scleral lens education comprises ~15% of the total contact lens curriculum.
- Although educators report having access to over ten scleral lens fitting sets, most actively use about a third of their available sets.

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Optometric educators' guidelines for fitting scleral lenses

SCOPE
Scleral Lenses in Current Ophthalmic Practice Evaluation

Jennifer S. Harthan[1], Cherie B. Nau[2], Amy C. Nau[3], Jennifer S. Fogt[4], Muriel M. Schornack[2], Ellen S. Shorter.[5]

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INTRODUCTION

Scleral lenses are widely available and utilized for the management of corneal irregularity, ocular surface disease and refractive error. However, there is no established standard on ideal fitting guidelines. Scleral lens fitting and evaluation are now a component of optometric education.

The purpose of this study is to describe current education provided for scleral lens evaluation.

METHODS

Contact lens educators (members of the AOCLE Association of Optometric Contact Lens Educators) were invited to complete an electronic REDCap survey regarding scleral lens fitting and evaluation at their schools between June 2019 and August 2019. This project was approved by the University of Illinois at Chicago IRB. Data will be summarized using descriptive statistics.

RESULTS

192 email invitations were sent to members of the AOCLE and 61 surveys were completed.

Of those, 53 reported being actively involved in scleral lens education at their institution.

- **Educators report ideal central scleral lens clearance to be 206 microns ± 44 (mean ± SD, n=40); with ideal limbal scleral lens clearance 62 microns ± 23 (n=36).**

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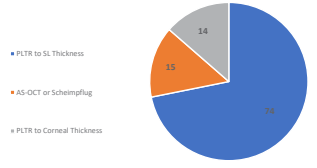
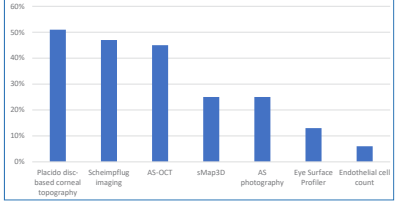
Educators report ideal central scleral lens clearance to be 206 microns ± 44 (mean ± SD, n=40); with ideal limbal scleral lens clearance 62 microns ± 23 (n=36).

Figure 1: Assessment of Scleral Lens Central Clearance



- Educators report teaching students the maximum amount of acceptable conjunctival blanching/compression beneath the haptic of a settled scleral lens to be: one clock dial (30 degrees; 46%), none (39%), followed by one quadrant (15%; n=41).
- The maximum acceptable amount of conjunctival prolapse (entrapment, billowing) with a settled scleral lens was one clock dial (30 degrees; 43%), followed one quadrant (25%), and none (20%, n=40).

Figure 2: Instruments Used During SL Evaluation



- Most educators, 92%, are teaching students to remove lenses to evaluate the anterior segment at each follow up visit.

CONCLUSION

Despite the lack of formal established standards, there is a general consensus among optometric educators on ideal scleral lens central and limbal clearance. Importantly, almost all educators are teaching students to remove lenses to evaluate ocular health at each follow up examination.

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Corneal RGP Management of a Pediatric Patient After Corneal Laceration Repair

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BACKGROUND

Pediatric patients account for about one third of all cases that present after ocular trauma.[1] Additionally, one in five of pediatric patients with ocular trauma result in a ruptured globe.[1]

The initial goal of treatment is to reestablish the integrity of the globe through emergency surgery and to prevent infection. However, repairing the globe can result in significant corneal scarring and irregular astigmatism that can limit visual potential. To avoid a penetrating keratoplasty in a young patient, a rigid gas permeable (RGP) corneal contact lens can be used to mask the irregular ocular surface. Use of corneal RGP in pediatric patients can be complicated by contact lens compliance, difficulty with insertion and removal and contact lens intolerance. [2]

CASE DESCRIPTION

An 8-year-old male presents for a contact lens fitting in the right eye after traumatic corneal laceration repair. Six months prior the patient sustained a full thickness corneal laceration from a tree branch in the right eye. He also developed a traumatic cataract in the right eye secondary to trauma. The patient had undergone corneal laceration repair and cataract extraction with insertion of a posterior chamber intraocular lens.

Entering uncorrected visual acuity in the right eye was 20/100 and pinhole acuity was 20/70. The right eye cornea was remarkable for a paracentral stromal scar extending from 2 o'clock to 6 o'clock (Figure 1). The patient was fit with a diagnostic reverse-geometry corneal RGP lens. This design was utilized due to the peripheral position and elevation of the corneal scar (Figure 2). Best corrected visual acuity with the corneal RGP contact lens was 20/30+.

After two lens remakes this lens design was unsuccessful due the inability to vault over the corneal scar without creating excessive pooling/bubbles in the area surrounding the scar (Figure 3&4) Instead a lens design with a spherical base curve and aspheric peripheral curves was fit. This lens was able to provide adequate clearance over the corneal scar while not creating an excessive amount of clearance elsewhere (Figure 5). Lens comfort was improved and visual acuity remained at 20/30+.

FIGURE 1

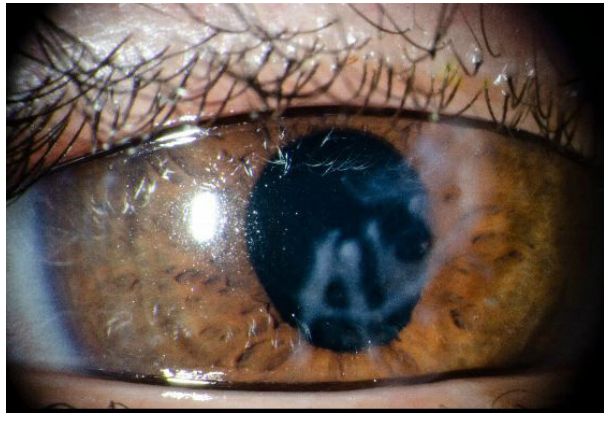


FIGURE 2

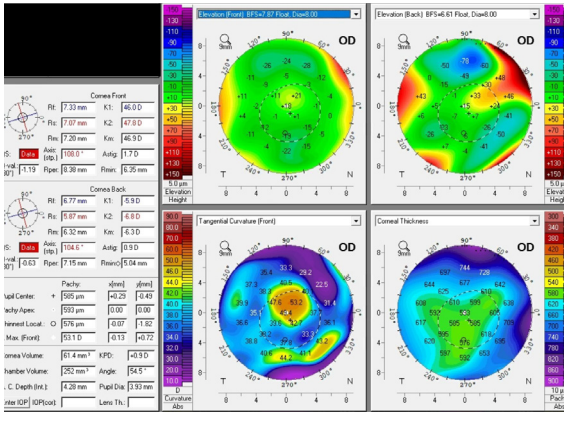


FIGURE 3

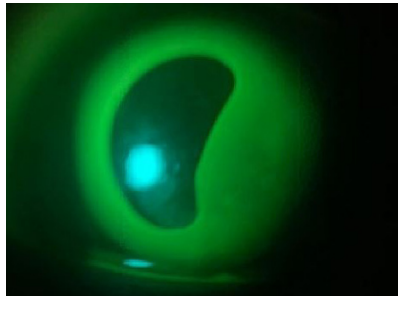


FIGURE 4

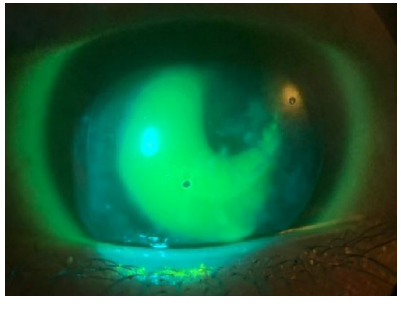
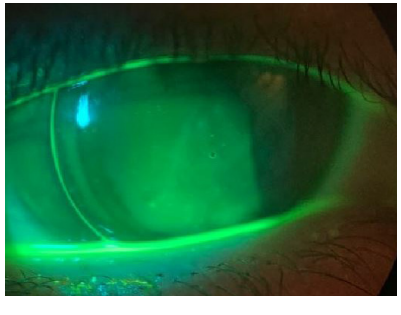


FIGURE 5



CONCLUSION

In patients with irregular astigmatism secondary to corneal laceration repairs, corneal gas permeable lenses often improve best corrected visual acuity from spectacles. [3] RGP contact lenses were found to be well tolerated in a pediatric population, have the advantage of excellent oxygen transmissibility and low bacterial and protein adherence.[4,5] A corneal RGP was chosen over a scleral contact lens because of its smaller diameter for a pediatric patient with a smaller palpebral aperture and for easier handling for the patient's caregivers. Comprehensive education to patient's caregiver should be given to stress the importance of good compliance with lens wear and regular follow-up to monitor contact lens fitting. The patient and caregiver were educated on importance of wearing full-time polycarbonate spectacles as protection from further traumatic events

ACKNOWLEDGMENTS

Andrew Kornaus O.D. and Neil Farbman, M.D. for their involvement in managing this case.

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Successful Scleral Contact Lens Wear in Patient with Topical Steroid Withdrawal/Red Burning Skin Syndrome

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BACKGROUND

Topical Steroid Withdrawal (TSW), or Red Burning Skin Syndrome, is a rare condition seen in some patients who have discontinued long-term use of topical corticosteroids. It is characterized by erythema of the skin that can become thickened with papules, pustules and erosions. This rebound eruption of the skin can extend to areas that have not had previous contact with topical corticosteroids. TSW has not been well-studied and although there is no reported correlation between TSW and ocular manifestations, it can result in rosacea of the face.

CASE DESCRIPTION

A 29-year-old Caucasian female was referred to the Illinois Eye Institute for specialty contact lens fitting. She had a positive systemic history for eczema treated with topical hydrocortisone cream since she was 10 years old. She developed TSW when topical treatment was discontinued at 17 years old (see Image 1.) At the time of presentation to our clinic, her TSW was controlled. Her ocular history was significant for corneal scars OD>OS that developed during her TSW episodes, and dry eye disease, which she was managing with GenTeal gel qhs OU.

Entering BCVA with Manifest Rx

OD: -4.50 sph VA 20/80
OS: -5.00 sph VA 20/25

Pertinent Abnormal Slit Lamp Findings

	OD	OS
Adnexa	Erythema and flaking of skin	Erythema and flaking of skin
Lids/Lashes	Scurf, MGD with telangiectasia	Scurf, MGD with telangiectasia
Cornea	Superior stromal scar with neovascularization*	Faint inferior stromal scar with ghost vessels

*See Image 2

IMAGE 1
Photo of patient displaying active topical steroid withdrawal.

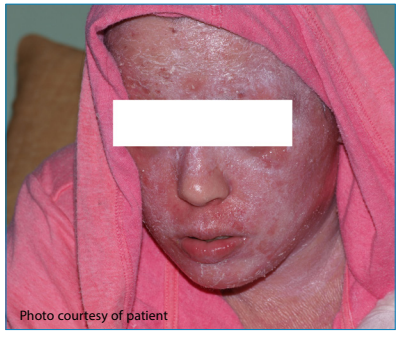
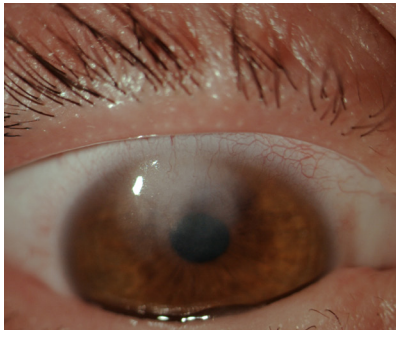
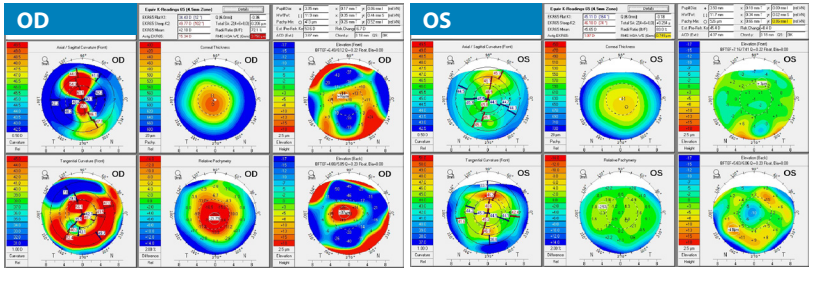


Photo courtesy of patient

IMAGE 2
External slit lamp photo showing corneal scar and neovascularization of the right eye.



Topography
Pentacam testing revealed irregular astigmatism OD>OS, contributing to decreased VA.



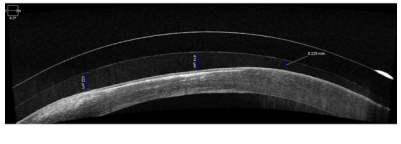
RESULTS

The patient was fit into a Valley Contax Custom Stable scleral contact lens OD (see Image 3) and a daily disposable soft contact lens OS. Her VA improved to 20/25 OD and 20/20 OS at the dispense appointment and remained stable at the one month follow up. The patient was able to maximize daily contact lens wear to ten hours of continuous wear with the addition of preservative-free artificial tears. The scleral lens was ordered with Tangible Hydra-PEG technology due to her dry eye disease and inflammatory condition.

Contact Lens Rx

	OD	OS
Lens	Valley Contax Custom Stable Prime	Acuvue 1-Day Moist
Power	-0.37 sph	-4.75 sph
Base Curve	8.04mm	8.5mm
Diameter	15.8mm	14.2mm
LCZ	-1 steep	
SLZ	-1 steep	
Material	Optimum Extreme with Hydra-PEG	
VA	20/25	20/20

IMAGE 3
AS-OCT of the right eye after wearing scleral lens for 7 hours shows acceptable fit over corneal scar.



CONCLUSION

Although there is no definitive correlation between the patient's ocular findings and her systemic condition of TSW, her presentation showed similarities to patients with other inflammatory conditions such as atopic dermatitis and ocular rosacea, which are associated with keratoconjunctivitis and corneal scarring. Even in cases where etiology is unknown, eyecare providers can use scleral contact lenses as treatment options for patients with dry eye, corneal ectasia and irregular astigmatism to improve comfort and best corrected visual acuities. In this case, the patient was successfully managed with a scleral contact lens to improve her ocular surface disease and ability to continue activities of daily living.

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Corneal Ectasia Following Pseudomonas Infection in a Contact Lens Wearer

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Background

Contact lens related corneal ulcers require prompt and aggressive management due to the risk of permanent vision loss. In contact lens wearers, the most common type of infectious corneal ulcer is due to the microorganism *Pseudomonas* aeruginosa. After treatment, corneal scarring and irregular astigmatism may occur. Glasses and soft lenses may not correct for these abnormalities. For these patients, specialty lenses may provide the best visual outcomes.

Case BD

A 14 yo Caucasion female presents for a GP contact lens fitting following a contact lens related central corneal ulcer OD. She was effectively treated for a *pseudomonas* infection, but the infection resulted in central corneal ectasia and an uncorrected DVA of 8/200, PH to 20/60.

POHx: Myopia OU, Soft CL wear OU
PMHx: Unremarkable
Medications: Pred Acetate 1% taper OD, PFATs qhr OD

Initial Presentation:

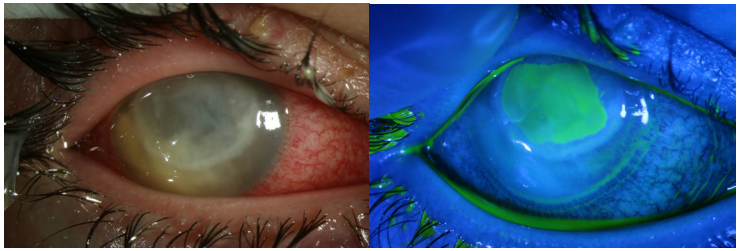

Figure 1. Slit lamp photos of initial ocular presentation of patient BD with and without fluorescein staining. 15% thinning under 5.2 x 6.0 mm superior central corneal ulcer.

	OD
Cornea	No epithelial defect, central edema, mid-peripheral thinning to 50%, moderate diffuse stromal scarring
Keratometry	57.87/45.87@058
Topography	CIM: 8.13 Shape Factor: -0.44

Table 1. Pertinent Findings OD 50 Days after Initial Presentation.

The Fitting Process

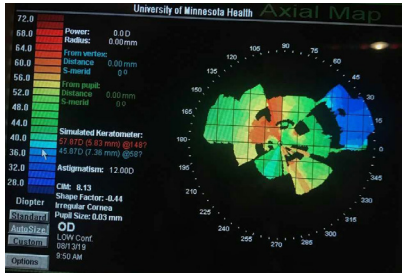

Figure 2. Axial Map OD, summarized findings in Table 1.

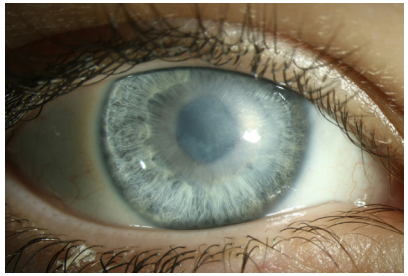

Figure 3. Slit lamp photo OD 50 days after initial presentation. Central corneal ulcer is resolved.

	Brand	BC/Dia.	Power	Fit	OR & VA
Initial Fitting: RGP	Oxyflow	7.2/9.2 mm	-3.00D	Unstable- good centration, central touch, peripheral pooling	-2.75D, 20/40-1

	Brand	BC/Dia.	Power	Sag. Depth	SLZ	Fit	OR & VA
Follow-Up 1: Scleral Fitting	SynergEyes VS	8.4/16.0 mm	Plano	3600 µm	36-42	Good centration, vault > thickness of lens, limbal clearance & landing zone adequate	+1.25D, 20/25-3

	Brand	BC/Dia.	Power	Sag. Depth	SLZ	Fit	OR & VA
Follow-Up 2: Scleral Fitting	SynergEyes VS	8.4/16.0 mm	+1.25D	3500 µm	36-42	Good centration, vault ¾ thickness of lens, limbal clearance & landing zone adequate	Initial: +3.75D 20/20 5 Min. later: 20/40

Conclusion

The patient initially presented for an RGP fitting for corneal ectasia following a contact lens related *pseudomonas* aeruginosa infection. The amount of irregular astigmatism caused instability of the RGP lens. After obtaining topography and keratometry, the patient returned for a trial scleral lens fitting.

Upon follow-up for the scleral lens, near perfect fit and vision were obtained with a small OR. A new lens was ordered with an adjustment to the sagittal depth and power. When the patient returned, lens fit was excellent, but VA was 20/40. After OR, a VA of 20/20 was obtained but within minutes her VA declined to 20/40. Despite re-evaluation of the lens and OR, improved vision could not be obtained. Given the excellent fit and overall improvement in vision, the lens was dispensed. The fit has yet to be re-evaluated.

Long–term RGP wear Induced Limbal Stem Cell Deficiency

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INTRODUCTION

Limbal stem cells reside in the limbal area of the cornea and function to maintain and regenerate the corneal epithelium. Limbal stem cell deficiency (LSCD) is a condition caused by the destruction of stem cell precursors of the corneal epithelium and is typically seen clinically as corneal conjunctivalization.[1] LSCD can be caused by numerous acquired etiologies like chemical injuries, Stevens-Johnson syndrome, or iatrogenic disease.[1]

CASE PRESENTATION

A 61-year-old African American male presents with blurry vision in the left eye (OS). The patient's ocular history is unremarkable except for his long-term history of RGP lens wear for over 30 years. His medical history is remarkable for hypertension which is managed by taking amlodipine and hydrochlorothiazide. The patient's entering corrected visual acuities are 20/20 in the right eye (OD) and 20/40 OS with no improvement on pinhole. Slit-lamp examination reveals 1+ scurf, 2+ Meibomian gland dysfunction, dense 360 arcus and 1+ nuclear sclerosis. Upon follow-up examination, careful staining of the cornea exposed whorl-like staining patterns around the limbus 360 in both of his eyes OS>OD consistent with limbal stem cell deficiency.

DISCUSSION

Our corneal limbal stem cells are crucial in the normal regeneration of the corneal cells and therefore are responsible for maintaining a healthy cornea. Contact lens-induced LSCD is less well-known and often forgotten since its presentation is not as severe as in other cases however the basic pathologic process is the same.[2] Three principal mechanisms associated with long term rigid gas permeable (RGP) lens wear known to damage the ocular surface includes chronic mechanical micro-trauma, hypoxia, and toxic lens solutions.[3] For these reasons, long term RGP lens wear may be responsible for the damage to the limbus destroying the limbal stem cells. Treatment options for CL-induced LSCD begin with conservative management by discontinuing CL wear and maintaining an adequate tear film with aggressive lubrication.[4] This alone can begin to reverse the damage caused by long-term CL wear.

FIGURE 1
Pentacam Holladay report OS showing irregular astigmatism with flattening in the periphery causing reduced BCVA.

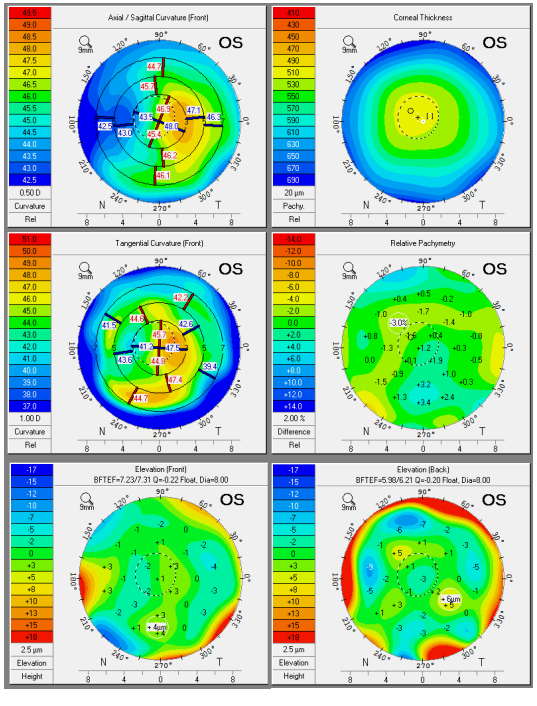


FIGURE 2
Blue light with NaFl demonstrating scalloped staining pattern around the limbus superior>inferior OS.

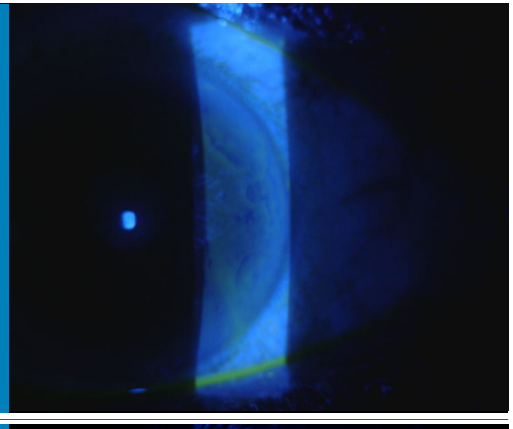
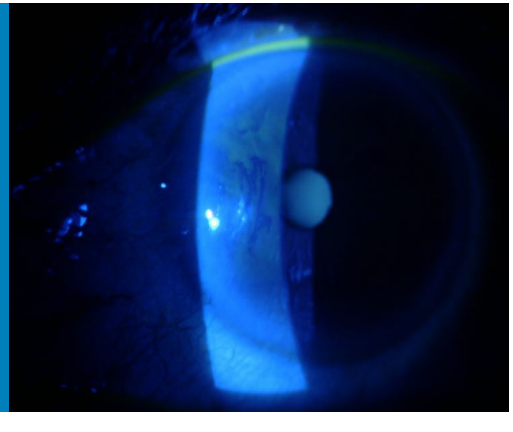


FIGURE 3
Blue light with NaFl showing central whorl-like staining pattern.



CONCLUSION

The long term complications that can arise with prolonged contact lens wear are often forgotten. Although long term RGP wear has been proven to be safe with relatively few complications, keep in mind that a small amount of micro-trauma and hypoxia to the limbus over long periods can build up. This small amount of damage that builds up can lead to LSCD and can present with a range of symptoms including irritation and decreased visual acuity.

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Case Series: Complications of Lagophthalmos Resolved with Scleral Lenses

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INTRODUCTION

Lagophthalmos is a condition characterized by the inability of the eyelid to completely close. Prolonged lagophthalmos causes exposure keratopathy, leading to severe desiccation of the ocular surface. It occurs secondary to numerous conditions including, but not limited to cranial nerve palsies, trauma, infection and iatrogenic diseases.[1]

CASE #1

A 41-year-old Caucasian female presents with severe redness and irritation in both of her eyes (OU). The patient's ocular history reveals Sjogren's disease. Her medical history is remarkable for myotonic dystrophy. Her ocular medications include Restasis two times a day (BID) OU and Lumify BID OU. Slit-lamp examination reveals lagophthalmos, mucous discharge and 2+ diffuse injection OU. Her right eye has a trace posterior subcapsular cataract (PSC) while her left eye has a 2+ PSC. She was scheduled to have cataract surgery OS followed by a scleral fit OU for improved comfort. She was fit in the Onefit Med scleral OU and on subsequent follow-ups, she reported improved comfort with decreased redness and irritation.

CASE #2

A 61-year-old Caucasian female presents with exposure keratopathy OU. The patient's ocular history reveals lagophthalmos secondary to a blepharoplasty OU. Her medical history is remarkable for depression for which she takes Prozac. Her ocular medications include Oasis Tears four times a day OU. Slit-lamp examination revealed lagophthalmos and 1+ diffuse inferior superficial punctate keratitis OU. She has inferior neovascularization and large dense mucoid plaques OS>OD. She was fit in the Zenlens RC scleral OU and reported immediate relief.

FIGURE 1
Case #1: Scleral lens OD completely vaulting the cornea providing a 247um tear fluid reservoir.

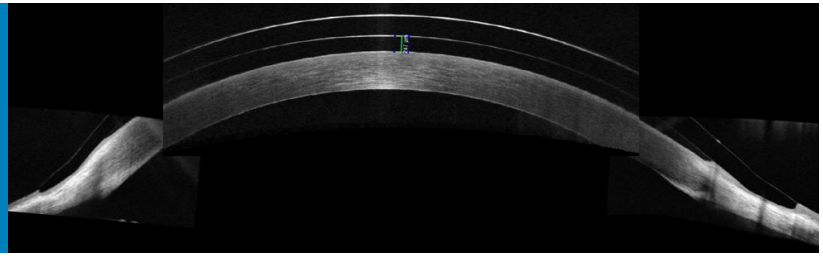
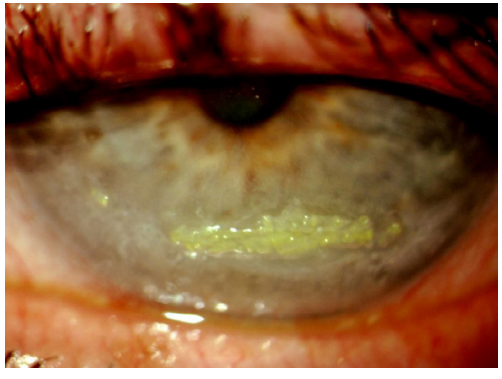


FIGURE 2
Case #2: Incomplete closure of the lid OS causing inferior corneal show.

FIGURE 3
Case #2: Dense linear build-up of mucoid plaques inferior OS where the lids don't fully close.



DISCUSSION

Prolonged lagophthalmous can quickly cause serious ocular surface desiccation that requires intervention.[2] Treatment begins with the least invasive options, but for severe ocular surface disease, patients may ultimately be faced with more involved surgical solutions like tarsorrhaphy.[3] Scleral lenses is an excellent option that provides lasting coverage of the ocular surface and is often underutilized. By completely vaulting the cornea, scleral lenses provide a tear layer that constantly bathes the cornea in fluid preventing dryness and associated symptoms while preventing the need for surgical intervention.[3]

CONCLUSIONS

Although there are many unique presentations of lagophthalmos, associated signs and symptoms are similar. Scleral lenses provided an effective non-invasive management option that is often forgotten.[2] They provide clear vision with all-day comfort that many patients with severe ocular surface disease due to lagophthalmos haven't experienced in years.

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HEART OF AMERICA

8 ICO PRESENTATIONS

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Treating Based on Presumptions: Management of Acute CNVM in POHS

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INTRODUCTION

Subretinal neovascularization is a vision-threatening complication most commonly associated with exudative age-related macular degeneration (AMD). Conditions such as presumed ocular histoplasmosis syndrome (POHS) and polypoidal choroidal vasculopathy (PCV), though less prevalent, can also result in choroidal neovascularization. This case demonstrates a patient presenting with subretinal neovascularization in the absence of a definitive retinal diagnosis, and with risk factors for multiple potential etiologies, including POHS, PCV, and AMD. In cases where the underlying cause is not easily defined, it is important to promptly and effectively treat the threat to vision.

CASE HISTORY

A 74-year-old African American Female presents for retinal consultation for a large peripapillary intra- and subretinal hemorrhage with associated subretinal fluid OD. The patient had complaints of blurred vision OD>OS for several months, with exact onset unknown. She was seen in an urgent care service three days prior, where signs of POHS were noted, with no other pertinent ocular history. Medical history was positive for hypertension and type 2 diabetes, both well controlled.

EXAM FINDINGS

20/60 PHNI	**BCVA**	20/20
PERLLA, no APD	**Pupils**	PERRLA, no APD
FTFC	**CVF**	FTFC
FROM	**EOMs**	FROM
Unremarkable	**Anterior Segment**	Unremarkable
Intra- and subretinal hemorrhage nasal to macula w/ surrounding subretinal fluid, S/N histo spot, PPA, mid-peripheral drusen	**Posterior Segment**	**I/T histo spot, PPA, mid-peripheral drusen**
Distinct PED with adjacent intraretinal fluid	**Macular OCT**	Unremarkable

FIGURE 1A
Fundus photo OD demonstrating peripapillary lesion with resulting intra- and subretinal hemorrhage and associated fluid.

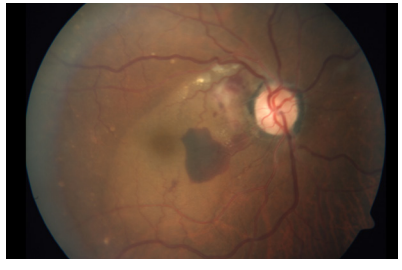


FIGURE 1B
Macular OCT OD showing distinct PED with adjacent intraretinal fluid.

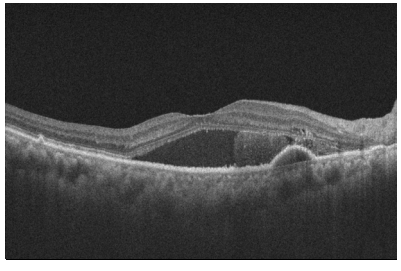


FIGURE 2A
Fundus photo OD representing improvement in retinal appearance 4 weeks s/p bevacizumab injection. Hazy quality contributed to poor tear film at follow-up.

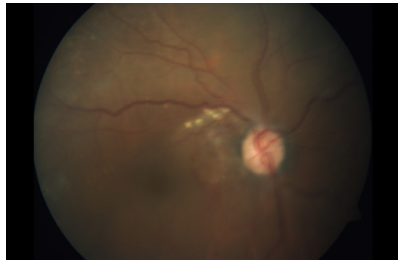
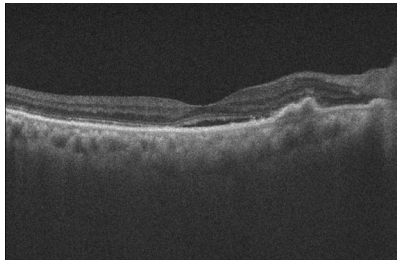


FIGURE 2B
Macular OCT OD demonstrating reduced size of PED and decreased intraretinal fluid.



DIFFERENTIAL DIAGNOSIS

Choroidal neovascularization vs. subretinal neovascularization secondary to:

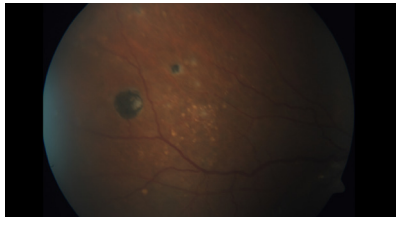
- **Presumed Ocular Histoplasmosis Syndrome**
- **Polypoidal Choroidal Vasculopathy**
- **Exudative Age-Related Macular Degeneration**

DIAGNOSIS AND DISCUSSION

Subretinal and choroidal neovascularization are potential complications of several retinal conditions and can lead to significant vision loss if left untreated. This particular patient shows clinical findings consistent with multiple different possible etiologies, as listed above.

The clinical triad of POHS includes punched-out chorioretinal lesions, peripapillary atrophic changes, and subretinal neovascularization, all of which were present in this patient. However, considering patient demographics and concurrent retinal findings, PCV and exudative AMD are viable differentials. OCT revealed a marked PED with an adjacent associated subretinal hemorrhage, consistent with classic findings of PCV. Additionally, in female patients with PCV, the lesion is more frequently peripapillary in location rather than macular, correlating with the findings of this case. Finally, in the presence of retinal drusen and neovascularization, exudative AMD must be considered. Ultimately, regardless of underlying cause, it is imperative to promptly address vision-threatening neovascularization.

FIGURE 3
Fundus photo OD showing chorioretinal scarring consistent with POHS, as well as scattered retinal drusen.



TREATMENT

Treatment for peripapillary or macular neovascularization can include surgery, photodynamic therapy, or anti-VEGF injections. Studies have suggested that, regardless of location, initial treatment with intravitreal anti-VEGF is a safe and effective approach to regress neovascularization and preserve or improve visual acuity in cases of complications secondary to POHS, PCV, AMD, or idiopathic causes. In this case, bevacizumab injection was initially selected. Four weeks following the injection, the patient demonstrated improvement both in visual acuity and retinal appearance. A second injection was performed at follow-up to resolve the remaining subretinal activity and further improve acuity. The patient is scheduled for further follow-up to determine if resolution has occurred.

CONCLUSION

In cases of visually-significant subretinal neovascularization, it is important to focus on effective treatment, regardless of definitive causation. Studies show that anti-VEGF injections are effective initial treatment options in cases of POHS, PCV, and wet AMD and proved to be beneficial to this patient.

REFERENCES

Available upon request.

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Atypical Anterior Chiasmal Syndrome: Unilateral Optic Atrophy as Presenting Sign of Pituitary Macroadenoma

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INTRODUCTION

A pituitary adenoma is the most common benign tumor of the central nervous system to affect the chiasm and patients present with visual changes 10-30% of the time. Adenomas are classified via size and when the size exceeds 10 mm, they are considered a macroadenoma. Typically, pituitary adenomas cause a bitemporal visual field defect but when the compression is more towards the anterior chiasm and one pre-chiasmatic optic nerve, it can lead to a junctional scotoma. A junctional scotoma is characterized as a central defect in the eye of greater compression and a superior temporal defect in the contralateral eye.

CASE HISTORY

58-year-old African American female patient presented with complaints of painless decreased vision OS for 2 months. No pertinent systemic/ocular health history.

PERTINENT FINDINGS

BCVA: 20/20 OD, 20/80 OS
Pupils: 2+ APD OS
CVF: FTFC OD, superior-temporal and inferior-temporal defects OS
EOM: FROM, (-) pain on eye movement
Posterior segment: Figure 1A, 1B
OCT: Figure 2A, 2B
HVF: Figure 3A, 3B
MRI of brain and orbits: Figure 4A, 4B

DIFFERENTIAL DIAGNOSIS

Pituitary Macroadenoma
Suprasellar Meningioma
NAION

FIGURE 1A
Fundus photo OD displaying ONH with pink and healthy rim, normal vasculature and a flat macula.

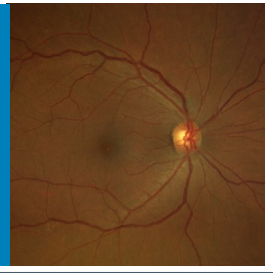


FIGURE 1B
Fundus photo OS displaying diffuse ONH pallor, normal vasculature and a flat macula.

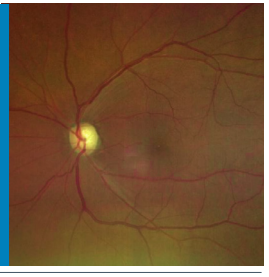


FIGURE 2A
Spectralis OCT of OD ONH showing no signs of RNFL or MRW thinning.

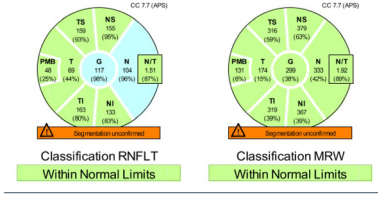


FIGURE 2B
Spectralis OCT of OS ONH showing significant RNFL and MRW thinning.

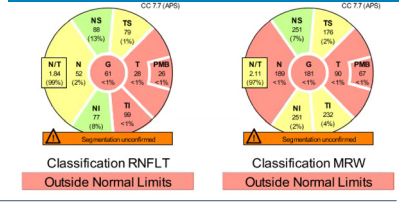


FIGURE 3A
Unreliable 24-2 HVF OD that showed few scattered non-specific depressions.

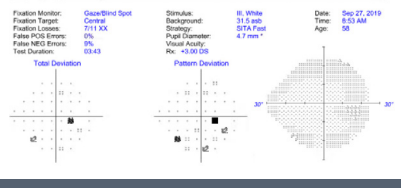


FIGURE 3B
Reliable 24-2 HVF OS that showed a dense temporal and central defect.

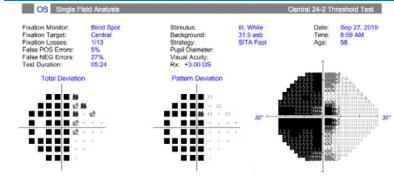
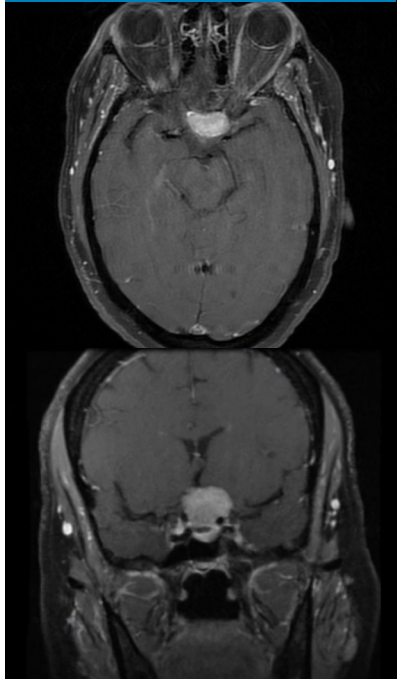


FIGURE 4A,B
Axial and coronal cut of T1 MRI with contrast showing enhancement of a sellar mass with suprasellar extension and mass effect on the anterior optic chiasm and left prechiasmatic optic nerve.



DIAGNOSIS AND DISCUSSION

After confirmation via histology, the patient was ultimately diagnosed with a pituitary macroadenoma with secondary optic atrophy. Lesions compressing the anterior chiasm typically present with junctional scotomas, a central defect on the side of greater compression and a contralateral superior-temporal defect. In this case, the patient had atypical HVF defects; in addition to the expected central defect OS, she had a dense temporal defect OS. This is likely because the lesion was compressing more of the left chiasm and ONH. The HVF OD could not confirm or deny the expected superior-temporal defect because of the unreliable results.

TREATMENT AND MANAGEMENT

Pituitary macroadenomas are removed via trans-sphenoidal adenectomy and visual field loss is a major indicator of the need for surgical intervention. Visual prognosis is better in patients that present earlier, with smaller tumors and with non-central and/or unilateral field defects. The patient underwent surgical resection of the tumor and is scheduled to return for follow up with HVF and OCT to monitor for any visual recovery.

CONCLUSION

This case demonstrates the importance of diagnostic testing and prompt neuroimaging in the presence of unilateral optic nerve pallor. In cases of optic atrophy due pituitary macroadenoma, patients typically have a good visual prognosis.

REFERENCES

Available upon request

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The Use of Imaging in the Diagnosis of Acute Zonal Occult Outer Retinopathy

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INTRODUCTION

Acute zonal occult outer retinopathy (AZOOR) is a rare condition that results in acute loss of one or more zones of the outer retina leading to symptoms of photopsia and visual field loss. AZOOR typically affects young, healthy, myopic females, and the underlying etiology is currently unknown. AZOOR is hypothesized to occur because of an immune response to a viral infection or due to autoimmune disorders. Due to AZOORs occult nature, it is commonly misdiagnosed. Imagining with SD-OCT, FAF, and ICG-A as well as FA, allows for visualization of AZOORs pathognomonic trizonal lesions, thus allowing for rapid diagnosis and appropriate patient management, education, and treatment.

CASE

A 28-year-old Hispanic female presented for vision loss with associated flashes of light that occur throughout the day, every day in the right eye for the past five years. The patient reported a history of treatment with Diamox; however, she developed chest pain and did not notice an improvement in visual symptoms. The patient had previous MRIs, spinal taps, and bloodwork to rule out infectious or inflammatory etiology, all of which were normal.

Pertinent Clinical Findings		
	OD	**OS**
VA CC	20/30, PH NI	20/20
Pupils	PERRL 2+ APD	PERRL, (-) APD
CVF	Abnormal ST, IT, IN	FTFC
EOM	FROM, (-) pain	FROM
Vitreous	(+) mild vitritis	Clear
Lens	Clear	Clear
Optic Nerve	(+) peripapillary atrophy, (+) edema	(+) disc elevation (-) edema (-) PPA
Fundus	Scattered mid-peripheral chorioretinal lesions S,ST, SN, (-)RDs/holes/ tears	Flat x 360 degrees, (-) RDs/holes/tears

FIGURE 1
Posterior pole fundus photo OD, OS

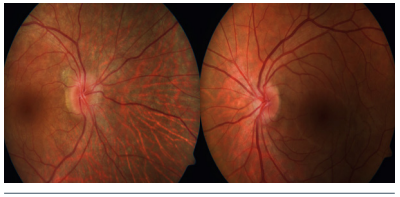


FIGURE 2
Mid-peripheral chorioretinal scars OD

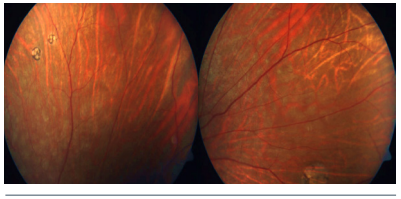


FIGURE 3
SD-OCT of macula OD showing trizonal lesion with ellipsoid zone loss (red arrow), intact sub-foveal ellipsoid zone (yellow arrow)

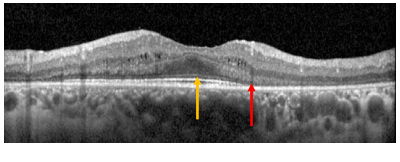


FIGURE 4
Fluorescein Angiography OD showing peripapillary staining without vasculitis, disc or central foveal leakage

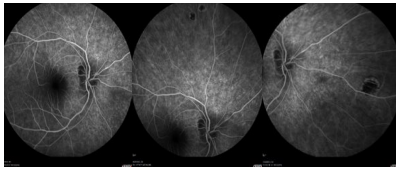


FIGURE 5
FAF with pathognomonic trizonal demarcating lesion OD and normal FAF OS

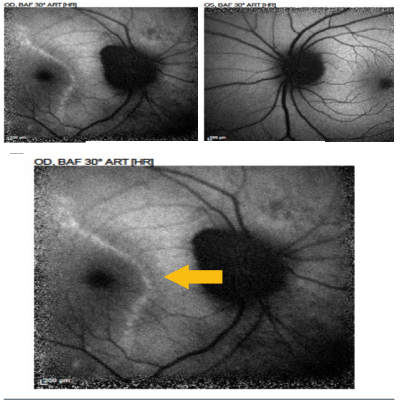


FIGURE 6A
24-2 SITA Faster HVF with unreliable test results OS and hemianopic VF loss OD

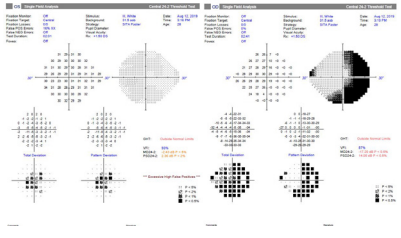
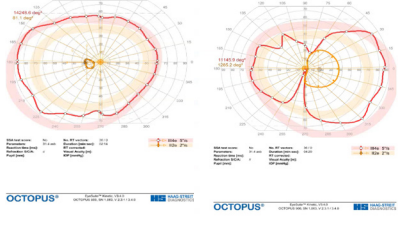


FIGURE 6B
Goldmann VF Perimetry with normal VF OS and enlarged blind spot with constriction OD



DIAGNOSIS AND DISCUSSION

AZOOR has been considered on the spectrum of inflammatory white dot syndromes; however, with the use of SD-OCT, FAF, and ICG-A, AZOOR is now considered an independent pathology and can be identified and diagnosed more rapidly due to its unique trizonal appearance. The trizonal lesion is characterized by three zones. Zone 1 being the delineating line separating the normal fundus from the AZOOR lesion. Zone 2 is characterized by areas of RPE dysfunction within the lesion, and zone 3 is characterized by areas of photoreceptor, RPE, and choroidal atrophy. Younger patients presenting with symptoms of acute or longstanding localized photopsia and visual field loss with minimal fundus changes should be further imaged with SD-OCT, FAF, and ICG-A to supplement and definitively identify AZOOR. Although no gold standard treatment exits for AZOOR, studies have shown that the use of intravitreal Ozurdex has improved visual acuity and improved recovery of the retinal ellipsoid zone. Patients with AZOOR are monitored every six months with visual field testing. Our patient elected to be monitored in 6 months and deferred intravitreal steroids.

CONCLUSION

Acute zonal occult outer retinopathy and its unique trizonal appearance can be diagnosed conclusively with the use of multimodal imagining. Imaging with SD-OCT, FAF, and ICG-A allows for quicker diagnosis of AZOOR and distinguishes AZOOR from other white dot syndromes. A patient's quality of life can be affected by AZOOR due to the visual symptoms and visual field loss; thus, the use of these imaging techniques is essential for improved management, treatment, and patient education.

REFERENCES

Available upon request.

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The Optometrist's Guide to Botany

Steve T Huynh, OD • Leonard V Messner, OD, FAAO - Illinois College of Optometry, Chicago, IL

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INTRODUCTION

Initial referrals for abnormal posterior pole findings in a pediatric patient must rule out whether the underlying condition is congenital or pathologic in nature. Of the congenital nature, one must be aware of other possible systemic association with the condition. This case report showcases the signs of Morning Glory Syndrome, but was referred to the clinic for suspicion of possible ocular malignancy.

CASE HISTORY

5-year old Hispanic female referred for a second opinion to rule out optic nerve glioma in the left eye. The patient denied any visual or ocular symptoms. No pertinent medical or ocular history.

SUMMARY OF PERTINENT FINDINGS

Visit 1
- BCVA 20/20 OD, 20/30 OS
- Excavated optic disc with central glial tuft and radial protrusion of the retinal vasculature, posterior staphyloma, and serous retinal detachment within peripapillary retina OS
- Posterior pole unremarkable OD
- Refer for MRI/MRV of the brain and orbit
- Follow-up in 2 months for repeat dilated fundus exam

Visit 2
- BCVA 20/20 OD, 20/30 OS
- MRI/MRV were non-contributory
- Worsening of serous retinal detachment with all other previous findings stable compared to initial visit OS
- Posterior pole unremarkable OD
- Refer to retina for consultation within 1 month
- Follow-up in 6 months

DIFFERENTIAL DIAGNOSIS

Optic nerve glioma, optic nerve coloboma, peripapillary atrophy

FIGURE 1
Representative fundus photos of the posterior pole for both eyes. The right eye (A) was unremarkable, but the left eye (B) showed excavated optic disc with a central glial tuft and radial protrusion of the retinal vasculature. Additionally, posterior staphyloma and serous retinal detachment was noted within the peripapillary retina.

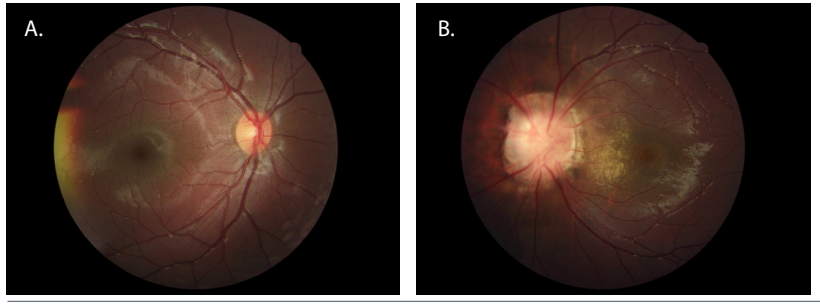


FIGURE 2
Ganglion cell analysis shows irregular thickening nasal to the fovea OS, compared to OD.

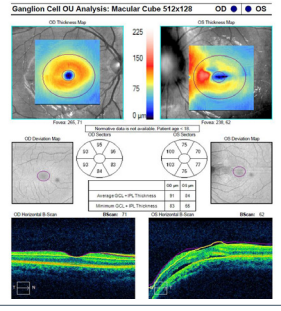
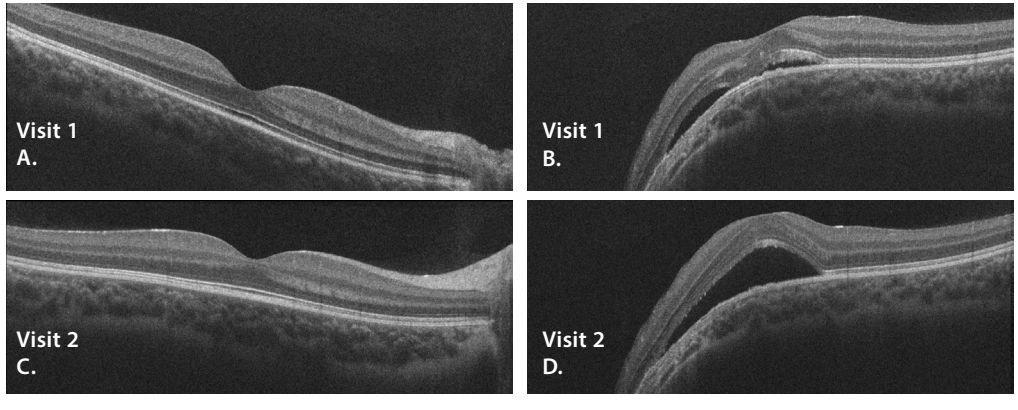


FIGURE 3
Representative HD 5 Line Raster of the Macula showed normal macula findings OD (A, C), but a progressive worsening of serous retinal detachment OS comparing visit 1 (B) and visit 2 (D).



DIAGNOSIS AND DISCUSSION

Morning Glory Syndrome (MGS) is a rare congenital condition with no known specific pattern of genetic inheritance. Predilection tends to occur in females more than males, and presentation is more often unilateral than bilateral. Clinical signs of MGS include an enlarged, funnel-shape excavation of the optic disc, peripapillary chorioretinal pigmentary changes, a central white glial tuft, and radial branching of the retinal vasculature. It is believed that the pathophysiology of MGS relates to an incomplete closure of the fetal fissure.

Visual prognosis is often poor with BCVAs worse than 20/40 in about 70% of cases. Common ocular complications of MGS include amblyopia and strabismus. Up to 30% of cases will also exhibit serous retinal detachments. Patients with MGS may have underlying congenital systemic abnormalities, including basal encephalocele and Moyamoya Syndrome.

TREATMENT AND MANAGEMENT

There are no treatments for MGS. Amblyopia and strabismus should be treated as appropriate to achieve best corrected visual acuity. Serous retinal detachments in MGS are typically monitored closely without treatment every 3-6 months. Lasers may be considered if further retinal complications develop. Diagnostic imaging must be done at the initial presentation of MGS to rule out other congenital systemic conditions.

CONCLUSION

Morning glory syndrome is a rare congenital condition that must be differentiated from other potentially more menacing pathologies through use of diagnostic imaging. The condition is often stable, but may require treatment with progression that endangers the macula.

REFERENCES

References upon request.

Financial Support: No financial disclosure.

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Persistent Red Eye in a Pediatric Patient Diagnosed as Herpes Simplex Virus Keratitis

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INTRODUCTION

About 50-90% of people are seropositive for herpes simplex virus (HSV). Since many people are infected with HSV at a young age, HSV keratitis should be considered as a differential when pediatric patients present with unexplained corneal findings. HSV keratitis manifests in many ways depending on which layer of the cornea is affected. This case demonstrates the clinical course of HSV stromal keratitis in a pediatric patient and improvement in the condition with appropriate treatment.

CASE PRESENTATION

A 12-year-old female presented with a persistent red right eye for several years. She has taken unknown eye drops intermittently throughout this time without relief of symptoms and is currently on ketotifen BID OD and lid scrubs without relief of symptoms.

PERTINENT FINDINGS

	Initial Visit	2 month follow up
BCVA OD	• 20/25^{-}	• 20/20
Conjunctiva OD	• 2-3+ diffuse injection • 1+ papillae	• Trace injection
Cornea OD	• Neovascularization (2.5 mm inferior, 2 mm superior) • Diffuse stromal haze • 3+ diffuse SPK • (-) dendrite/infiltrate	• Neovascularization (stable) • 1+ diffuse SPK • (-) stromal haze • (-) dendrite/ infiltrate

FIGURE 1
Initial presentation showing diffuse bulbar injection OD

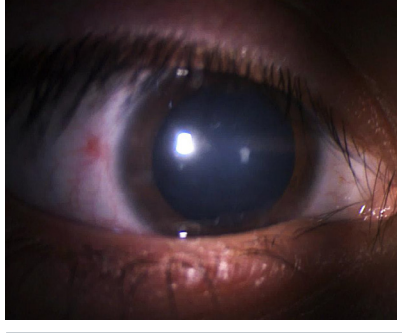


FIGURE 2
Initial presentation OS (unremarkable findings)

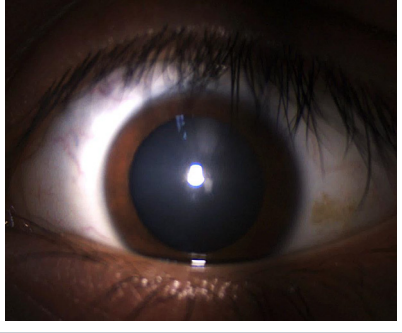


FIGURE 3
Initial presentation showing significant corneal neovascularization with surrounding stromal haze OD

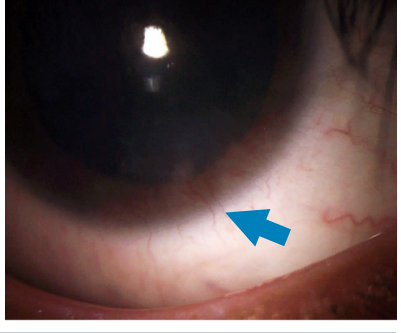


FIGURE 4
Initial presentation showing significant corneal neovascularization OD

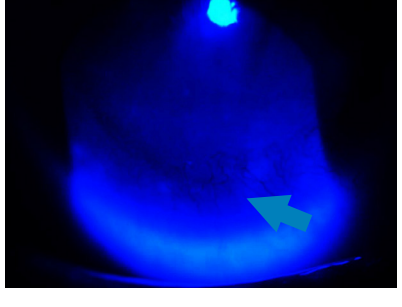


FIGURE 5
2 month follow up showing resolution of stromal haze OD

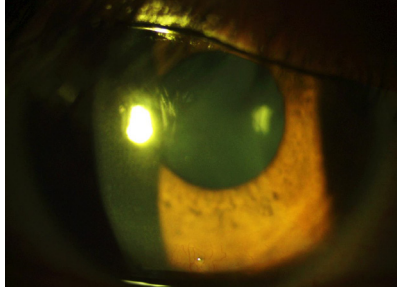
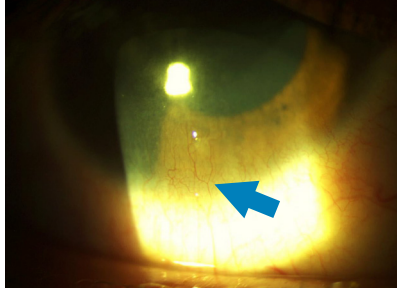


FIGURE 6
2 month follow up showing stability of corneal neovascularization with resolution of surrounding stromal haze OD



DISCUSSION

This patient presented with a non-necrotizing stromal keratitis based on the presence of stromal haze and neovascularization. These findings are a result of an immune response to HSV antigens in the stroma. It is important to note that pediatric patients with HSV keratitis are more likely to be misdiagnosed, as with this case, and commonly have a higher rate of recurrence than adults.

Non-necrotizing stromal keratitis is treated with a topical steroid as well as an oral antiviral. The patient was prescribed prednisolone acetate QID OD and acyclovir oral suspension 7.5 mL QID po (pediatric dosage calculated by her pharmacist). After improvement of signs and symptoms, the topical steroid was tapered to BID OD and the oral acyclovir decreased to a maintenance dosage of 10 mL BID po to prevent recurrence. After treatment, there was significant improvement in the stromal haze and improvement in BCVA to 20/20. The amount of neovascularization remained stable after treatment.

CONCLUSION

HSV keratitis should be considered in pediatric patients with unilateral redness unresponsive to other treatment protocols. It is important to diagnose HSV keratitis in children promptly as the higher recurrence rate in children can lead to corneal scarring and ultimately amblyopia.

REFERENCES

Available upon request.

CONTACT INFORMATION

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Emergent left–sided hemianopia in a patient without cardiovascular risk factors

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CASE HISTORY

- 64-year-old female with history of hyperlipidemia and hypothyroidism presents with complaint of not being able to see out her left eye for the past 5 days.
- She also reports a constant headache for 5 days, but denies scalp tenderness and jaw claudication.
- Her systemic health is well controlled with Crestor and Synthroid.
- The patient is right-hand dominant.

PERTINENT FINDINGS

- Entering acuity was 20/20 in the right eye and 20/20-1 in the left eye.
- Pupils were round and brisk OD and round and sluggish OS and a 2+ RAPD was noted in the left eye.
- Confrontation fields revealed left field constriction in both eyes
- Humphrey Visual Field revealed a homonymous left sided hemianopsia (see Figure 1)
- Slit lamp examination and dilated fundoscopy were unremarkable.
- She was oriented to time, person and place.
- Patient had difficulty with clock drawing test, specifically with spatial planning, requiring multiple attempts (see Figure 2)
  - These deficits are often noted in parietal lesions in the non-dominant cortex.

DIFFERENTIAL DIAGNOSIS

- Right-sided cerebrovascular accident
- Right-sided trauma to the optic tract and/or occipital lobe
- Right-sided neoplasm affecting the optic tract

DIAGNOSIS AND DISCUSSION

- The patient was immediately referred to the emergency department for an immediate stroke workup.
- She underwent neuroimaging and was diagnosed with a 6cm glioma of the right basal ganglia (see figures 3 and 4).

FIGURE 1
Static Humphrey Visual Field showing a left homonymous hemianopia

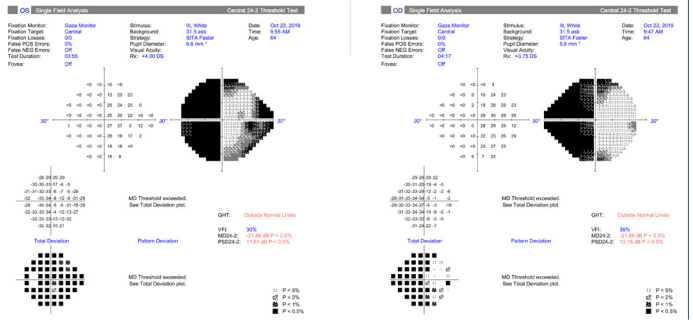


FIGURE 2
Clock Drawing Test showing difficulty with spatial planning

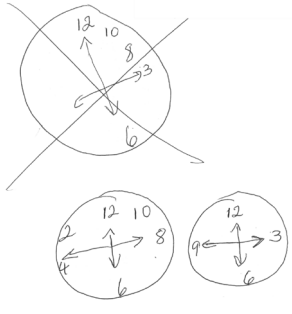


FIGURE 3
T1-weighted sagittal MRI through the lesion

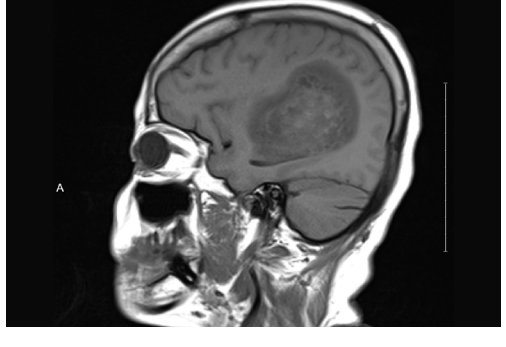
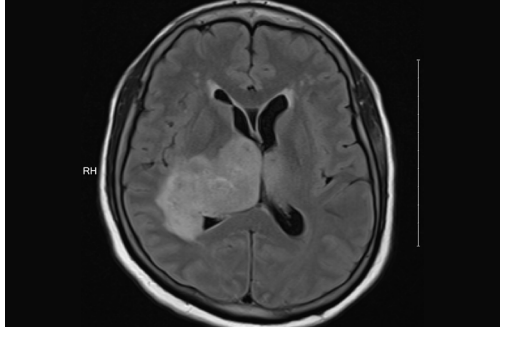


FIGURE 4
T2-FLAIR axial MRI through the lesion



- A hemianopia indicates a lesion in the post-chiasmal visual pathway in the way that an RAPD indicates a lesion in the pupillary pathway.
- The presence of a RAPD in a patient with a homonymous hemianopsia can help localize the lesion to the optic tract contralateral to the visual field defect due to the higher amount of crossed versus uncrossed nerve fibers and the increased sensitivity of melanopsin-containing fibers found in the nasal retina.
- Patients with homonymous hemianopsia can develop difficulties with orientation, mobility and reading.
- Depending on the underlying etiology of the hemianopia, prognosis is typically good, with 55% of patients regaining some visual field within the first month, and complete recovery in about 18% of patients with homonymous hemianopia in the first month.

TREATMENT AND MANAGEMENT

- The patient underwent a right parietal craniotomy biopsy and has begun radiation treatment, chemotherapy and IV Avastin.
- Cranial biopsy resulted in a subarachnoid hemorrhage causing transient left-sided paralysis.
- Pathology reports revealed a "favorable molecular profile"
  - MGMT promoter methylated
  - IDH mutation

CONCLUSIONS

- A new-onset hemianopia warrants an immediate referral for a stroke workup to rule out stroke and stroke mimics.
- Patients with homonymous hemianopsia require a multidisciplinary team of health professionals including optometry, ophthalmology, neurology and occupational therapy.

CONTACT INFORMATION

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Bilateral choroidal neovascular membranes due to pathologic myopia in a developmentally delayed patient

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INTRODUCTION

Choroidal neovascular membrane (CNVM) is one of the most visually threatening complications that can develop from myopic degeneration. Of all patients with myopic degeneration, only 5-10% develop choroidal neovascularization. Of those who do develop CNVM, only 30% develop it bilaterally. Traditional OCT can identify new choroidal net growth by identifying subretinal hemorrhaging, subretinal fluid, or RPE detachment as signs of leakage into the subretinal space. OCT-A can be utilized as a non-invasive tool to better differentiate active and inactive CNVM and direct treatment. This case demonstrates the importance of regularly screening myopic degeneration patients for the development of CNVM, and the use of imaging modalities in diagnosis and management.

CASE PRESENTATION

16 year-old African-American female presents for a routine comprehensive exam. She denies visual or ocular complaints. Patient has developmental delay and is in a special education program.

Ocular History: Myopic degeneration OU
Longstanding macular scar from CNVM OS, s/p Avastin injections
Longstanding constant exotropia OS

Medical History: Developmental delay

EXAM FINDINGS

Visual Acuity: 20/60 OD, reduced from 20/40 the previous year
20/300 OS, stable

Fundus Exam: New macular lesion superior to fovea OD
Stable macular scar OS

OCT: CNVM with overlying subretinal fluid OD
Inactive CNVM OS

OCT-A: Confirmed active CNVM OD

FIGURE 1
Fundus photo OD with new macular lesion, with fibrotic tissue suggesting chronicity

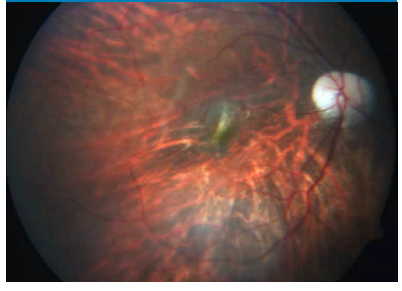


FIGURE 2
Fundus photo OS with large macular fibrotic scar from previous CNVM

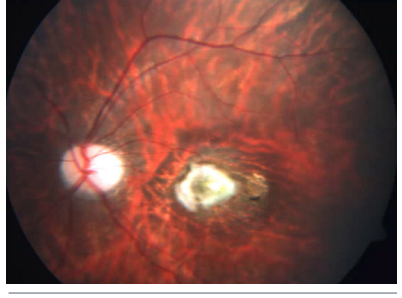


FIGURE 3
OCT OD demonstrating new CNVM with overlying subretinal serous fluid

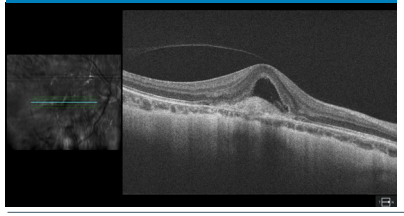


FIGURE 4
OCT OS demonstrating macular scarring from inactive CNVM

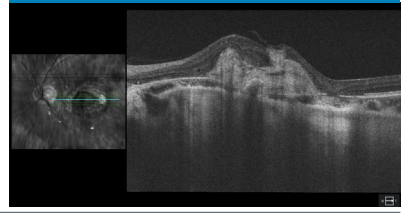


FIGURE 5
OCT Thickness map OD demonstrating macular elevation and loss of foveal contour

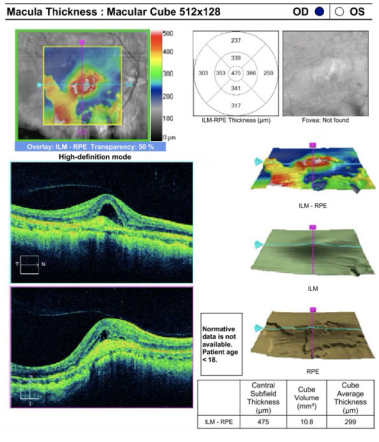
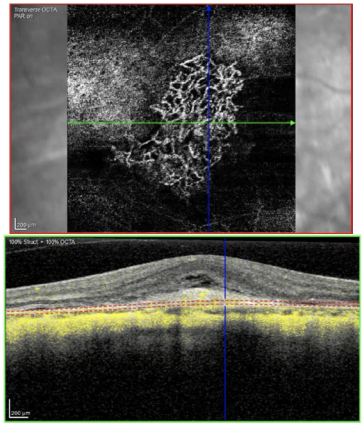


FIGURE 6
OCT-A OD demonstrating hyperreflectivity consistent with active CNVM



DISCUSSION

This patient presented with a new CNVM OD and reduced BCVA, but did not complain of changes to vision. Patients with developmental delay are less likely to report changes in vision. Many patients with myopic degeneration also have amblyopia or other complications that affect BCVA, and may be less sensitive to gradual changes. This increases the benefit of imaging technology for early detection of CNVM.

Though rare, bilateral presentations of CNVM are especially sight threatening. While all CNVMs require prompt treatment, bilateral cases in particular require early detection. New imaging techniques such as OCT-A can be indispensable for diagnosis and directing treatment. The treatment for CNVM is anti-VEGF injections, such as Avastin, to regress the new choroidal blood vessels and preserve as much vision as possible. This patient was referred to a retinal specialist and began a course of Avastin injections OD. Due to her developmental delay, she required anesthesia to undergo the injection. At her one month follow up, the patient's vision OD improved to 20/50. OCT demonstrated improvement in foveal contour and regression of the CNVM; however, there was persistent subretinal fluid requiring a second Avastin injection.

This patient was managed with vision rehabilitation services and Individualized Education Program (IEP). These services can maximize the patient's visual potential and improve her quality of life.

CONCLUSION

Patients with developmental delay or already reduced acuity may not report vision changes. Patients with known risk for CNVM should be followed closely; clinicians should consider advanced multimodal imaging techniques to assist early detection, especially in patients with communication barriers. Low vision services should be utilized to improve quality of life. Coordination of care is imperative for rapid treatment to save vision.

REFERENCES

Available upon request.

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OCT–A and Multimodal Imaging in the Diagnosis of Chronic NAION

Alexa Rowe, O.D. - Captain James A. Lovell FHCC, North Chicago, IL

INTRODUCTION

Non-arteritic ischemic optic neuropathy (NAION) is caused by an infarction in the short posterior ciliary arteries supplying the optic nerve head leading to unilateral painless vision loss. After 6-8 weeks acute ONH edema resolves into atrophy often obscuring the clinical diagnosis. This case reviews the value of diagnostic testing including OCT-Angiography, OCT-RNFL, and HVF in diagnosis of chronic NAION.

CASE PRESENTATION

A 72-year-old Caucasian male presented complaining of a "horn-shaped spot in vision" x 1-2 months OS. Denied any pain, flashes or veil over vision. Medical history was significant for T2DM, HTN, Sleep Apnea (with reported CPAP compliance), Obesity, and Venous Insufficiency. Patient ocular history only remarkable for early dry AMD OU. Pertinent medications included metoprolol and metformin.

EXAM FINDINGS

Entering VAs were 20/20 OD, 20/25+ OS. Pupils were round, equal, and reactive to light with no APD OU. CVF and EOMs were full without abnormalities. Amsler Grid revealed an arcuate scotoma inferior to central fixation OS.

Anterior segment was within normal limits with normal IOP. Posterior segment showed mild ONH pallor superior/superior temporal with collateral vasculature/superficial telangiectasia along the temporal disc margin. No edema or notching was visualized. Vasculature had mild arteriolar attenuation, but no significant plaques or vascular injury noted.

FIGURE 1
Fundus photo OS. Mild ONH pallor superior/superior temporal with collateral vasculature along the temporal disc margin

FIGURE 2
OCT-A OS: decreased retinal perfusion along superior temporal arcade

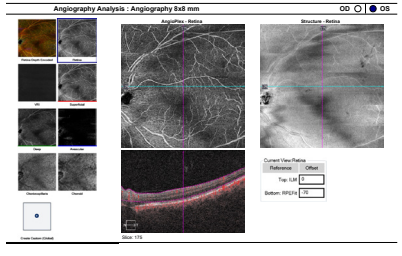


FIGURE 3
OCT RNFL OS: Moderate superior/temporal RNFL thinning

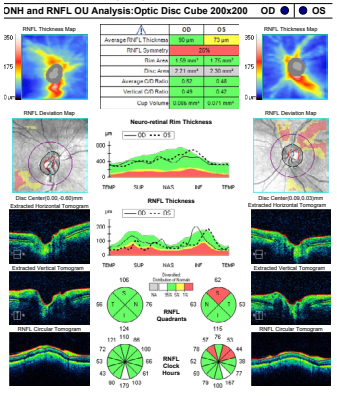


FIGURE 4
HVF 30-2 OS: Inferior defect nearing altitudinal with superior edge point defects

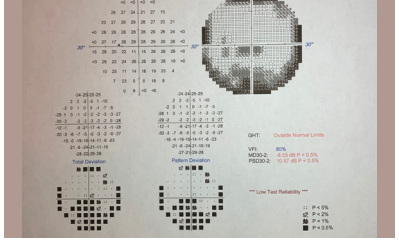
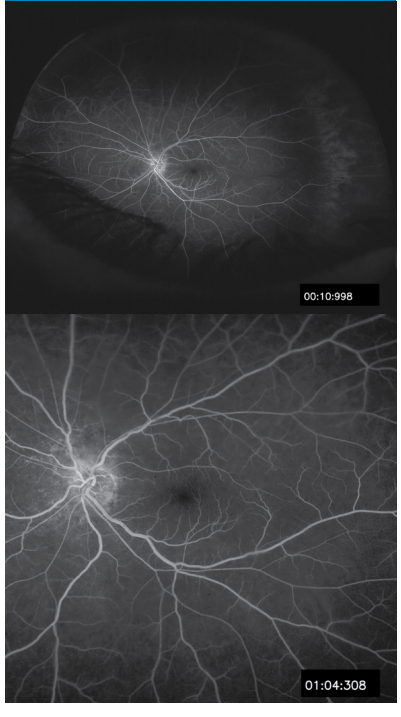


FIGURE 5 & 6
FANG OS: Normal vascular filling. Shunt vessels/superficial telangiectasia along temporal disc margin with significant supratemporal ONH pallor. No NVD or NVE.



DIAGNOSIS & DISCUSSION

NAION is the second most common optic neuropathy and most frequently occurs in patients over 50. Clinical diagnosis is most commonly made in the acute phase with sector/diffuse optic disc edema, but when the edema resolves, as in chronic cases, we're able to rely on imaging techniques to guide to proper diagnosis. OCT angiography has recently been studied as an additional mode of imaging for mapping of retinal perfusion. In our patient's case we can see that the decreased retinal perfusion of the superior temporal retina correlates with RNFL loss and the visual field defect. OCT-A can non-invasively assess retinal perfusion, especially when FANG is unavailable or contraindicated.

Currently there's no quality evidence for effective NAION treatment, especially chronic presentation. VA can improve up to 3 lines in 43% of patients, but VF defects are less likely to improve. Co-management with PCP is important for OSAS CPAP/BPAP compliance, and our patient's multiple vasculopathic risk factors. Management demands follow-up and risk analysis for fellow eye involvement as there is a 15% chance of fellow eye involvement at 5 years.

CONCLUSION

OCT-Angiography can be a valuable tool in assessing retinal perfusion in patients with suspected chronic NAION. It's non-invasive and can be used in conjunction with other imaging techniques when FANG is unavailable or contraindicated. Diagnosis is important to manage risk of fellow eye involvement and catastrophic vision loss.

REFERENCES
Available upon request

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NEUROSCIENCE

1 ICO PRESENTATION

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Risk factors for pinguecula: a narrative review

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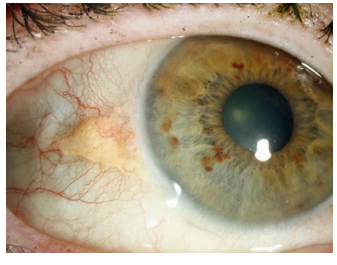
INTRODUCTION



Pinguecula is a round, yellowish, elevated, proliferation of conjunctival connective tissue typically located at the limbus in the palpebral fissure.[1] It will continue to slowly enlarge, sparing the cornea, but is considered to be non-malignant.. Even though pinguecula is quite common, its exact nature and pathogenesis are still unknown.[2-19]

Although pinguecula is usually asymptomatic, it can be the source of an aesthetic concern for the patient and may have the following comorbidities:

- Increased tear breakup time[20-1]
- Pterygium, a fibrovascular overgrowth of conjunctiva onto the cornea[22]
- Conjunctivochalasis[23-4]
- Primary spheroidal degeneration[25]
- Porphyria cutanea tarda[26]
- Tarsal gland dysfunction[27]
- Thyroid orbitopathy[28]
- Kidney transplant[29]
- Diabetes mellitus (possible)[3,30]
- Cataract (possible)[31-3]
- Sjögren syndrome (possible)[34]

While slit lamp examination is preferred for definitive diagnostic purposes, it has been shown that penlight examination can be used to make an accurate diagnosis of pinguecula.[35] Generally, pinguecula requires no treatment, although ocular irritation and cosmetic issues can prompt primary excision of the lesion.[21] The purpose of this study was to determine from the literature what risk factors there are for developing pinguecula, and what, if anything, can be done to manage those risk factors.

METHODS

This was a narrative review, although systematic review protocols were used as much as possible. Use of these protocols was somewhat limited by the nature of the pinguecula literature. In order to find pertinent articles, a Boolean search of PubMed was conducted (from the beginning of its indexing through December 2019) using the following word: pinguecula. From this list of citations, the authors individually reviewed the associated abstracts for information regarding risk factors for pinguecula, and then, after discussion, came to consensus on which of these references had such information. The full articles of these were obtained. The reference sections of newer articles were searched for any other pertinent articles. Once more, consensus was reached among the authors for which articles would be included. Only articles in English were used for this study. Given the paucity of articles and no human clinical trials, no research designs were excluded. There was no quality assessment that was used, since most of the studies were very simple, straightforward basic research designs. This search was focused on a single clinical entity.

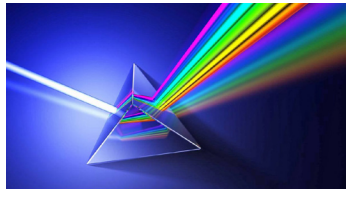
RESULTS

The PubMed search yielded 161 articles. After reviewing the abstracts of all these articles, searching the reference sections of newer citations for further articles, and eliminating duplicates, consensus was reached among the authors on 27 sources that seemed to contain information relating to the risk factors associated with pinguecula.[2-5, 7-13, 15-6, 22, 36-48]

AGE

Pinguecula is considered a senile change following more or less the usual equation of "saturation" at a frequency of 100%, i.e., if one gets old enough, statistically one will develop pinguecula.[22] Multiple clinical studies dating as far back as 1950, have found a correlation between age and pinguecula. [4, 7-10, 15, 16, 22] The low incidence of pinguecula in the pediatric population of a study from Ghana adds further support to this.[2]

UV



Ultraviolet (UV) light can cause eye damage through a variety of mechanisms.[36] There are several reviews about solar radiation and UV light as they relate to eye care and diseases of the eye, including pinguecula.[37-9] All of these reviews summarize that pinguecula has a long history of association with exposure to sunlight and that this association has been broadly accepted, even though the evidence for a correlation between UV radiation exposure and pinguecula is indirect and a direct relationship between UV light and pinguecula has yet to be established. There are multiple studies that were not included in these reviews correlating pinguecula with UV radiation.

Other UV evidence

Evidence	References
Cohorts with increased sunlight exposure had statistically increased rates of pinguecula	3, 5, 7, 9, 13, 16
Increased cumulative occupational sunlight exposure of one year x hour/day, increased risk of pinguecula by 2.1%	7
Pinguecula developed in patient receiving psoralen plus UV A treatment with proper shielding (case report)	40
UV light causes oxidative stress that can lead to oxidative damage to eye tissues	41
Actinically damaged fibroblasts may produce elastotic fibers that are basis of elastoid tissue in pinguecula via a process similar to solar elastosis of skin	40
Decreased antioxidant activity from UV light may cause oxidative damage via glycation with accumulation of advanced glycation end products, which have been immunohistochemically identified in pinguecula	42, 43

OTHER RISK FACTORS

Some authors assert that low latitude, dry, dusty, and hot environments may be risk factors for pinguecula, [7, 15] but there is no clinical evidence of this. On the other hand, there are clinical data relating pinguecula to other potential risk factors, although some of this information is contradictory.

Other risk factors

Risk factor	Yes	No
Welding occupation	X[11]	
Non-use of spectacles	X[3,7]	
Contact lens use	X[44]	X[45]
Smoking	X[13]	X[3, 16]
Ethanol	X[9]	X[3, 16]
Gender (♂>♀)	X[4, 12, 15]	X[3, 8, 16]
Level of education (inverse)	X[13]	X[16]

DISCUSSION

Nothing can be done to manage some of the risk factors associated with pinguecula, e.g., aging and genetic gender. Several risk factors might be able to be addressed, although economic and social realities may be significant hinderances in doing so, e.g., occupation and level of education. Smoking cessation, decreased alcohol consumption, and use of prescribed spectacles may be somewhat easier to accomplish.

Probably the most important factor to address is UV radiation. Educating the patient about the importance of eye protection from the sun and early screening measures are key for any healthcare professional. Physical barriers, such as protective eyewear,[49] are important considerations in decreasing UV light exposure. However, issues deterring their use could be affordability, availability, and lack of education about the importance of ocular actinic radiation protection. There are other strategies to reduce UV light damage that may be employed.

Other strategies of UV management

Strategies	References
Broad brimmed hats	39
Ski goggles	39
UV light filtering hydrogel contact lenses	39
Prescription glasses with UV radiation protection	39
Calophyllum inophyllum oil used as a non-cytotoxic UV filter that might be added to ophthalmic preparations	50 (human conjunctival epithelial cells *in vitro*)
A diet with higher percentages of unsaturated fatty acids could lower oxidative stress to the eye	41
Antioxidant and/or scavenger compounds including, dietary α-lipoic acid, and 4-coumaric acid drops have been shown to decrease corneal and/or conjunctival damage from UV radiation exposure	47, 48, 51 (multiple animal models)
Iodide and manganese superoxide dismutase may reduce UV light damage to ocular tissues and could be added to ophthalmic preparations (e.g., artificial tears)	46 (human conjunctival fibroblasts *in vitro*), 52 (rabbit conjunctival epithelial cells *in vitro*)
Gels containing the natural phenolic compounds: hydrocaffeic and p-coumaric acids decreased UV B radiation damage	53 (human conjunctival cells *in vitro* & rabbit eyes)

CLINICAL PEARLS

1. Pinguecula can be diagnosed with a pen light examination.
2. Pinguecula may be used as a basis for patient education about UV light management and nutritional support.
3. If pinguecula is symptomatic, collaborative care with an ocular clinician is warranted.

References on request

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