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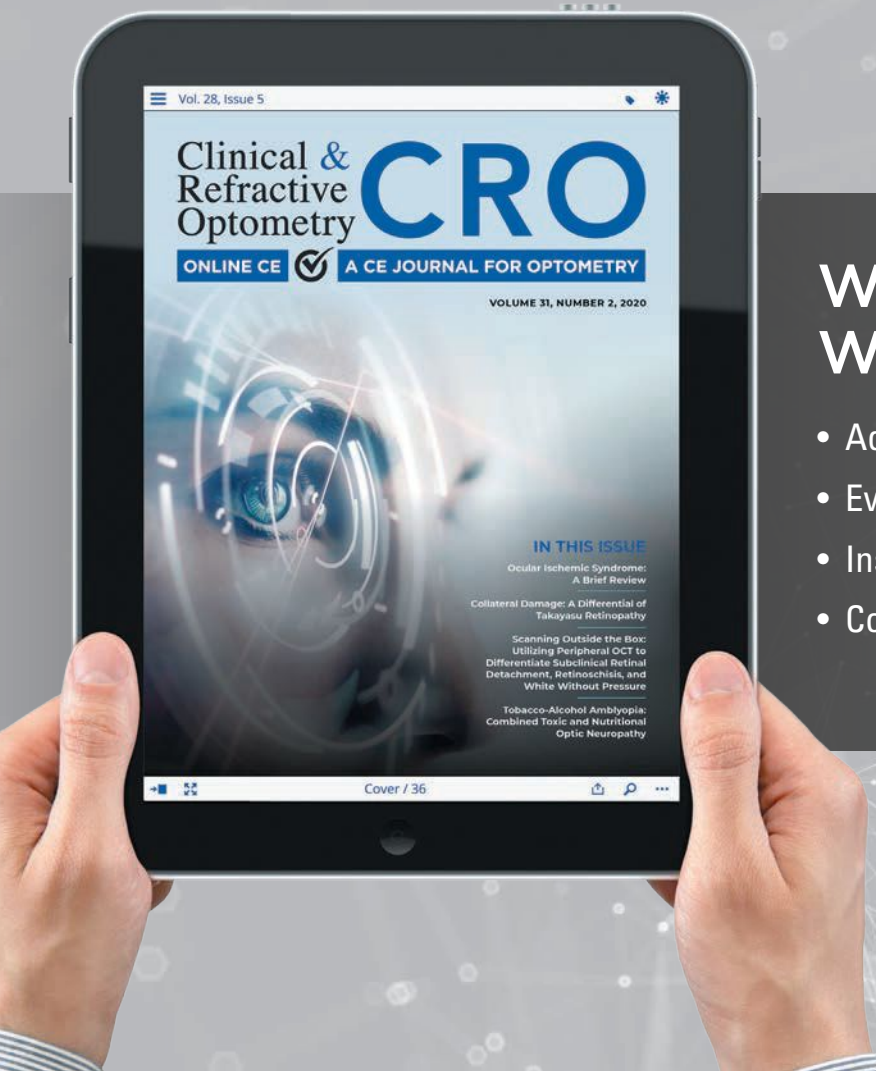

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An Examination of the Race Factor in Clinical Practice

Leslie Wilderson, OD, FAAO, is a graduate of the Nova Southeastern University School of Optometry where she also completed a primary care residency. Dr. Wilderson is currently on the clinical optometry staff of the Northeast Ohio VA system. Additionally, she proudly serves in the United States Air Force Reserve as the Biomedical Sciences Corps Career Field Manager Individual Mobilization Augmentee in the Command Surgeon's Office at Robbins Air Force Base.

During my years of civilian and military practice, I have had the great fortune to care for diverse populations in a range of geographic locations. I have watched the twenty-first century catalyze a medical technology boom that propelled our diagnostic certainty to new levels. And while the technological aspects of health care continuously advance our practice, some ideas have been slower to evolve. Specifically, the idea that put race in the realm of a biological risk factor for disease. The dissemination of information on the perils of using race in isolation have lagged behind the scientific data, and academic institutions are exploring what the Human Genome Project[1] revealed about race to implement reforms. In today's scientific arena, the associations between race and pathology are being challenged to mitigate the effects these generalizations have on clinical outcomes[2], and I have incorporated the principles that negate race as a biological consideration into my practice.

It is known that racial categories are social constructions defined by region specific cultural and historical ideas rather than inherent biological characteristics[3]. Further, the risk of generalizing race-disease associations has a history of facilitating harms that delay diagnoses and cause medical errors[3,4]. An illustration of race's incongruity with medicine is in the misrepresentation of geographic origin when only skin phenotype is considered, which may also ignore multifactorial social and structural determinants of disease[2]. The consideration that genetic differences are far higher within populations grouped by race than between racial groups[5] is an indication that generalizing about disease prevalence by race is dangerous. Since 2003 when the Human Genome Project sequenced the first near complete human genome, it discovered a lack of scientific or genetic basis of race[6]. To that end, new ideas are being infused into clinical thinking yet, because race has been insidiously embedded into clinical practice[2] there are still no clear guidelines.

In a paper that studied multiple subjects with a common disease process, I omitted race references to show that race is not a proxy for pathophysiology. Instead my evaluation used only true biological descriptors. Race is often seen as a shortcut in medical decision making[7] so allowing the structural, physiological, ancestral, and cultural factors to stand on their merits without race as a detractor made a more solid case for my diagnostic conclusions. Reliance on race-disease association algorithms can limit the scope of investigation and miss out on the patient's complex story.

In academic training, race is learned as an independent risk factor for disease rather than a mediator of structural inequalities[3]. In pre-clinical education, we learn that race is relevant to treatment decisions and this generalized approach leads to a host of disparities. Meanwhile, ancestry is more specific than race and can lead to a productive analysis of biological factors. Collectively, advances in diagnostic technology with rigorous science-based applications, and omitting the race factor can mitigate the disparities that cause poor patient outcomes.

In recognizing that racial categories do not provide an accurate picture of human biological variation, a statement from the American Association of Physician Anthropologists (AAPA) on race and racism asserted that the belief in "races" as natural aspects of human biology, and the structures of inequality that emerge from such beliefs, are among the most damaging elements in the human experience both today and in the past[8]. Their statement underscores that race-based medicine should be seriously addressed by the professional societies that set practice standards.

Leslie Wilderson, OD, FAAO

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Refractive Lens Exchange Candidate Selection

Raymond Stein, MD, FRCSC; Rebecca Stein, MD, FRCSC

Refractive lens exchange (RLE) is a popular procedure in presbyopic patients who desire the full range of vision. Advances in IOL designs can allow patients to achieve distance, intermediate, and near vision without the need for glasses. Seeing a smart phone or computer without correction is a significant lifestyle benefit. Patient satisfaction has been very high with both multifocal and extended depth of focus implants.

Candidates for RLE may have any refractive error and are usually presbyopic and desire the full range of vision. Presbyopic low hyperopes are very motivated, as they never wore distance glasses when they were younger, started with readers in their early 40s, and now need glasses for both distance and near activities.

Most hyperopic, myopic, and astigmatic refractive errors can be treated with a RLE. Patients should have a normal cornea, macula, optic nerve, and peripheral retina. Diagnostic testing should demonstrate a normal corneal topography without evidence of irregular astigmatism.

Achieving excellent outcomes is dependent on proper patient selection, advanced IOL calculations, meticulous surgery, and careful postoperative care. This RLE overview reviews important information on patient selection, surgical options, and postoperative care:

1. Indications and contraindications

2. Options for vision correction

3. Surgical techniques and IOL designs

4. Potential complications and treatment

Understanding these important factors allows the clinician to educate patients and provide optimum care. RLE is an exciting area of vision correction that is growing around the world. Clinical outcomes have improved in recent years secondary to many factors.

Eye Condition:

A complete eye examination is necessary to detect any abnormalities of the tear film, lids, cornea, lens, vitreous, macula, optic nerve, and retina. In addition, angle kappa, as defined as the distance between the center of the pupil and line of sight is an important measurement to determine lens implant choice. Patients with a high angle kappa of 0.6 mm or greater have an increased chance of positive dysphotopsias with multifocal lenses. Understanding the indications and contraindications for RLE increases the likelihood of having a successful outcome and a happy patient. (See Table 1)

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Table I

FINDINGS	COMMENTS	INDICATION
Presbyopia[1]	It is unusual for a nonpresbyopic patient to undergo RLE. Possible indications include a noncandidate for hyperopic LASIK or PRK secondary to a high degree of hyperopia, anticipated postop curvature 50 D or greater, and/or too thin a cornea, and does not qualify for an implantable contact lens (AC depth < 3.0 mm).	✓
Refractive Error: No limit as long as the power of the implant is available[2,3]	High myopes with an axial length > 25 mm are at greater risk of retinal tears and retinal detachment. If no significant lenticular changes, to consider an implantable contact lens if AC depth = or > 2.8 mm. High hyperopes are more likely to have a high positive angle kappa, and if = or > 0.60 mm may be better candidates for an extended depth of focus lens or monofocal implant.	✓
Cataract	If visual acuity and/or quality of vision are affected by lenticular changes, then cataract surgery is indicated. If visual acuity and/or quality of vision are normal, then consider RLE. Although RLE and Cataract surgery are the same procedures, government and/or or private insurance programs pay either the full amount or partial payment for cataract surgery depending on lens choice and the type of procedure.	?
Keratoconus, Pellucid Marginal Degeneration, Corneal Scarring and Irregular Astigmatism[4]	Patients with reduced best-corrected spectacle visual acuity (BCSVA) may be candidates for TG-PRK to reduce irregular astigmatism. When there is both refractive and topographic stability this can be followed by vision correction. Presbyopic patients with early lenticular changes may benefit by RLE with a toric or aspheric implant. A multifocal implant with diffractive optics is considered a contraindication.	?
EBMD or Salzmann's Nodular Degeneration[4]	Epithelial basement membrane dystrophy (EBMD) and Salzmann's dystrophy can cause a loss of BCSVA secondary to irregular astigmatism. Treatment is with a superficial keratectomy, which is essentially a debridement. When there is refractive and topographic stability an RLE can be performed. Patients should be counselled that EBMD and Salzmann's can recur and may require a retreatment.	?
Dry Eye[5,6]	Ocular surface disease can impact uncorrected-visual acuity and quality of vision. If there is a superficial punctate keratitis, this should be cleared up prior to surgery. Treatment may consist of management of an aqueous deficiency or meibomian gland dysfunction.	?
Blepharitis[7]	Inflammation of the lid margins can progress to a blepharokeratitis with a decrease in vision. Blepharitis should be treated prior to surgery to optimize the ocular surface and reduce the chance of infection.	?
Pseudoexfoliation[8]	Increased risk of a zonular dialysis at the time of surgery or subluxation of the implant postoperatively. If patient is really motivated, to consider RLE with a monofocal or toric implant. Patients are at higher risk of open angle glaucoma.	?
Age-Related Macular Degeneration[9]	Not a candidate for RLE with a multifocal implant. Surgery should be delayed until there is a visually significant cataract. Consideration at that point should be for a monofocal or toric implant.	✗
Epi-retinal membrane (ERM) or other macular conditions[10]	Not a satisfactory candidate as macular changes can impact both visual acuity and quality of vision.	✗
Angle Kappa > 0.6 mm[11,12]	High risk of halos or glare with a multifocal implant. Difficult for the surgeon to centre the implant on the line of sight. An extended-depth of focus implant can be considered, such as the Vivity, or a monofocal implant, which are not affected by a high-angle kappa.	?
Amblyopia[13]	Patients with essentially one functioning eye are at higher risk of compromised vision if there is an intraoperative or postoperative complication. Encourage glasses or contact lenses. If patient is only interested in a surgical option, then RLE can be performed with proper informed consent.	?
Glaucoma[14]	If intraocular pressures are under control and there is no significant field defect, then RLE can be performed. Patients with uncontrolled glaucoma or a significant field defect are not satisfactory candidates	?

✓ = YES ? = USE CAUTION ✗ = NO

Table 1 continued		
FINDINGS	**COMMENTS**	**INDICATION**
Lattice degeneration and/or retinal holes[15]	Patients are at higher risk of a retinal detachment with intraocular surgery. Consideration can be given to PRK over LASIK because of less pressure on the eye and theoretically a lower chance of a posterior vitreous detachment resulting in a retinal tear.	✗
Fuchs' Corneal Dystrophy[16]	The presence of corneal guttata and an increase in corneal thickness is considered a contraindication to RLE. There is a high risk of progressive corneal edema with loss of vision requiring corneal surgical intervention (DSEK, DMAK).	✗

Personality and Lifestyle:

Knowing both the personality of your patient and their lifestyle is valuable in determining candidacy for RLE and the type of implant. Patients that are hypercritical may not be accepting of any perceived imperfections in their vision. It is also important to understand the patient's occupation, hobbies, and how much time per day they are involved in near activities (computer work, smart phone, reading a book, etc) versus distance activities. Patients that do considerable night driving may not be ideal candidates for multifocal implants. (See Table 2)

Table 2		
FINDINGS	**COMMENTS**	**INDICATION**
Type A or Hypercritical Individuals[17,18]	Usually best to avoid multifocal implants in this group as the visual aberrations, no matter how minimal, may not be well tolerated. Patients may be a candidate for extended-dept of focus or monofocal/toric implant.	?
Drive for a Living[17,18]	As above.	?
Significant Night Driving[17,18]	As above.	?
Pilot, Police Officer, Military[17,18]	As above.	?

Prior Eye Surgery:

A history of previous eye surgery can impact the final uncorrected visual acuity and quality of vision. Patients that have had previous refractive surgery (LASIK, PRK, or RK) can do well with RLE but there is an increased chance of a residual refractive error because of difficulty in determining the ideal implant power of both the sphere and cylinder. Patients should be counselled that a secondary procedure may be required to enhance vision. (See Table 3)

Table 3		
FINDINGS	**COMMENTS**	**INDICATION**
LASIK or PRK[19,20]	Presbyopic patients that have normal corneal topography without evidence of ectasia can be considered for an RLE. Patients often desire the full range of vision and a multifocal or extended depth of focus implant are reasonable options. There is a greater risk of a refractive surprise in this group requiring a touch-up with PRK, a secondary IOL, or lens exchange. Determining the exact power of the sphere and cylinder of the implant is more difficult in the post-refractive group.	✓
RK[21]	RK corneas often create significant higher-order aberrations. Best to avoid a multifocal implant. May be a candidate for extended depth of focus implant and/or a monofocal or toric implant.	✓
Penetrating Keratoplasty[22]	Usually best to proceed with PRK or an implantable contact lens. Increased risk of loss of corneal endothelial cells with a RLE and graft failure.	✗
Retinal Detachment Repair[23]	Best not to proceed with intraocular surgery given the history of a retinal tear and retinal detachment. LASIK or PRK are preferred options. For high degrees of myopia to consider an implantable contact lens, which carries less retinal risk than an RLE.	✗

✓ = YES ? = USE CAUTION ✗ = NO

VISION OPTIONS

Vision Options:

Presbyopic patients typically want the full range of vision. Understanding the indications and contraindications to RLE with various implants allows the clinician to counsel patients with the best options to meet expectations. (See Table 4)

Table 4

FINDINGS	COMMENTS
Full range of vision[24]	Most patients are interested in seeing the full range of vision. There is a great desire to see a smartphone and a computer without the need for glasses. With a multifocal implant there is an increased risk of halos and glare. This typically decreases over time.
Distance, intermediate, and some near[17,18,24]	Multifocal implants are considered a contraindication in type A individuals, anyone who would not tolerate any persistent halos or glare, or ocular conditions that may not allow an excellent level of vision. The extended depth of focus implants like the Vivity and Eyhance can provide an enhanced range of vision compared to a monofocal or toric implant.
Distance only[24]	Prior to the development of the new extended depth of focus implants this was a reasonable option. However, with the new EDOF lenses which have an incidence of halos or glare similar to monofocal lenses there are no downsides to achieving distance, intermediate, and some near.
Monovision[25]	The best candidates for monovision are patients that are successful with monovision contact lenses. Others that may be interested in monovision RLE can do a contact lens trial prior to deciding on the best vision option.

SURGICAL OPTIONS

Traditional vs Laser:

Both traditional and laser RLE can provide excellent outcomes. Patients may be more comfortable with the precision of the femtosecond laser that can create a perfectly round capsulotomy centered on the visual axis and fragmentation of the lens with minimal or no phacoemulsification. In addition, femtosecond laser can make surgery easier to perform and therefore potentially safer in a variety of conditions. (See Table 5)

Table 5

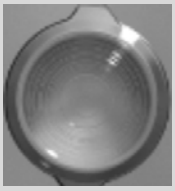
FINDINGS	COMMENTS
Traditional Surgery[26]	In traditional surgery, the surgeon does all steps manually. Outcomes can be very good.
Femtosecond laser[27]	Using the femtosecond laser, many of the steps are automated including limbal relaxing incisions, the capsulorehexis, and fragmentation of the lens. Ultrasound energy is eliminated in over 95% of eyes, which usually results in clearer postoperative corneas and a quicker return of vision. Automated steps are particularly helpful in eyes with shallow or deep anterior chambers, pseudoexfoliation, or zonular laxity.

IMPLANT OPTIONS

Understanding Lens Implants:

Advances in implant designs have lead to the development of aspheric, toric, extended-focus, and multifocal implants. These innovations allow patients to improve uncorrected vision for distance, intermediate, and/or near vision. (See Table 6)

Table 6

FINDINGS	COMMENTS
Aspheric Monofocal Implant[28] 	An aspheric implant attempts to compensate for corneal spherical aberration to improve quality of vision and improve contrast sensitivity under both mesopic and photopic conditions. The average cornea has +0.27 microns of spherical aberration. A lens that reduces this higher-order aberration can enhance quality of vision. Aspheric implants are available from Alcon, J & J, B&L, and others.
Toric Implant[29] 	Correction of astigmatism can be accomplished by the insertion and alignment of a toric implant. When the astigmatism level is 0.7D or greater, a toric implant is typically used. If the astigmatism is less than 0.7 D, then limbal corneal relaxing incisions can be effective. Astigmatism that requires correction is the total corneal astigmatism, which is calculated from both the anterior and posterior cornea. It is important to recognize that this often differs from the refractive astigmatism as well as keratometry.
Trifocal Implant[30,31,32,33]  	Trifocal implants can provide the full range of vision with a very high patient satisfaction rate. Patient selection is important to achieve the best outcomes and satisfaction levels. Some halos and/or glare are common in the early postop period but typically resolve or diminish with time. The most popular trifocal lenses in North America are the PanOptix (Alcon) and Synergy (J & J). These are both diffractive implants that can be visualized postop with concentric rings. The Panoptix lens (Alcon) is labelled as a trifocal but has a quadrifocal design. It has a 6 mm optical zone, a central 4.5 mm region with 15 diffractive rings, and an outer annulus that is refractive. Three step heights give three focal points including 40 cm, 60 cm, 120 cm, in addition to distance. The anterior surface is aspheric and posterior surface is spherical. Diffractive structure allows the lens to transmit 88% of light to the retina at a 3 mm pupil size for high light utilization. The Synergy lens (J & J) is a combination of extended depth of focus lens (Symfony lens) and a multifocal lens (Tecnis Multifocal). The lens filters violet light. Like the Tecnis Multifocal, the Synergy has two distinct focal points at approximately 33 cm (near) and infinity. Like the Symfony IOL, the Synergy provides continuous vision from 33 to 80 cm. Like the Tecnis Multifocal it extends the distance from 60 cm to infinity.
Extended-Depth of Focus (EDOF) Implant[34,35,36,37]  	EDOF lenses provide distance, intermediate, and some near vision. Similar to a monofocal lens, it is rare to have any halos or glare. If one eye is made slightly myopic -0.50 to -.1.00 then reading can be greatly enhanced. The Vivity (Alcon) and Eyhance (J&J) are examples of these lenses. The Vivity lens uses X-wave technology to stretch and shift the wavefront. Has a monofocal-like visual disturbance. It has a one-micron elevation that stretches the wavefront at 2.2 mm. The Eyhance has a continuous change in power from periphery to center. Appears like a standard Tecnis monofocal lens on examination. It has an aspheric anterior surface and a spherical posterior surface. The Eyehance provides improved intermediate vision over a standard monofocal lens but limited near vision.

POTENTIAL COMPLICATIONS & TREATMENTS

Potential Complications & Treatments:

Although complications are rare, all patients must be counselled on potential risks. Patients need to be aware of any acute symptom of concern and report back to their eye doctor. These symptoms include a decrease in vision, wavy vision, hazy vision, field loss, floaters, and flashing lights. Clinicians need to recognize the symptoms and signs of any complication and provide appropriate care or refer back to the surgeon. (See Table 7)

Table 7

COMPLICATION	SYMPTOM(S)	TREATMENT
Positive dysphotopsia[38,39,40]	Halos and/or glare	Usually decreases or resolves over 2 to 6 months secondary to neuroadaptation. Important to rule out a residual refractive error, which can be treated with LASIK or PRK. Ocular surface disease (eg dry eye or epithelial toxicity from medications) may also cause halos and/ or glare and requires treatment. Very rare cases require an IOL exchange.
Negative dysphotopsia[38,41]	Most commonly	Secondary to the capsule overlying the nasal edge of the optic of the implant. Symptoms typically resolve with time, but may take 6 months or longer. Rare case that does not resolve can be managed by elevating the nasal optic above the capsule.
Residual refractive error[42,43]	Sphere and/or cylinder	A residual refractive error especially with a multifocal implant can degrade the quality of vision. Usually best to wait 3 or more months before treatment with LASIK or PRK. At Bochner this is performed at no charge.
Posterior capsule opacification[44,45]	Capsule opacification	If associated with a decrease in visual acuity or there if there is a change in quality of vision, then a YAG capsulotomy should be performed. There is a slight increase in risk of retinal tears and retinal detachment, especially in highly myopic eyes with long axial lengths.
Cystoid macular edema (CME)[46,47]	OCT shows CMS	Usually associated with a decrease in visual acuity or quality of vision. Patients at higher risk are those with diabetes, age-related macular degeneration, or macular traction. Treatment is with a topical steroid four times per day, and a nonsteroidal two times per day. The drops can be discontinued when the OCT shows resolution of the edema.
Posterior Vitreous Detachment (PVD)[47]	Flashing lights and/or floaters	Very common, with or without surgery, is a PVD resulting in flashing lights and/or floaters. Important to rule out a retinal tear that requires treatment. Floaters usually decrease over time and rarely require removal with a vitrectomy.
Epiretinal membrane[48]	OCT shows epiretinal membrane	May be associated with diminished vision. Important to be sure it is not associated with CME that requires topical medication. Rare cases of epiretinal membranes that are visually significant can be managed by an epiretinal membrane peel.

Summary

Refractive Lens Exchange is a popular procedure in presbyopic patients that desire the full range of vision. Innovations in intraocular lens implants can allow enhanced vision without optical aids for distance, intermediate, and near. Clinicians need to understand the indications, contraindications, intraocular lens options, and postoperative risks and management to assure a high probability of success. Freedom from glasses and/or contact lenses after decades of wear can result in high patient satisfaction. It is important to educate all patients that even though their vision may be excellent following surgery they still need to see their eye doctor to maintain the health of their eyes.

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Refractive Lens Exchange Candidate Selection

1. Which of the following patients can benefit from RLE?

- ☐ A presbyope
- ☐ A patient with corneal guttata
- ☐ A patient with age-related macular degeneration
- ☐ A patient with glaucoma and poor IOP control

2. Which of the following patients would not fare well with multifocal implants?

- ☐ Type A or hypercritical patients
- ☐ Patients who drive for a living
- ☐ Patients who are police, military, or pilots
- ☐ All of the above

3. For patients who have a strong desire to have a full range of vision, their RLE should consider:

- ☐ A monofocal lens implant
- ☐ An extended depth of focus lens implant
- ☐ A multifocal lens implant
- ☐ An option outside of RLE

4. All of the following are contraindications for RLE surgery except:

- ☐ Patients with glaucomatous vision loss
- ☐ Patients with a history of multiple retinal tears
- ☐ Patients with severe Fuch's corneal dystrophy
- ☐ Patients who are presbyopic with otherwise normal eye findings

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5. A patient who has been diagnosed with glaucoma may still be eligible for RLE if:

- ☐ The IOP is under control and there is no visual field loss
- ☐ The IOP is out of control and there is no visual field loss
- ☐ The IOP is under control but there has been severe visual field loss
- ☐ Cataracts are also present

6. Which of the following is a lens that enhances the quality of vision by attempting to reduce higher-order aberrations?

- ☐ Aspheric implant
- ☐ Astigmatic implant
- ☐ Panoptix lens
- ☐ Synergy lens

7. Which of the following complications of RLE is categorized by glare and halos?

- ☐ Cystoid Macular Edema
- ☐ Residual refractive error
- ☐ Positive dysphotopsia
- ☐ Posterior capsular opacification

8. If a residual refractive error exists after RLE, LASIK or PRK may be performed after:

- ☐ 1-2 weeks
- ☐ 3 or more months
- ☐ 1 or more months
- ☐ 1 year

9. If posterior capsular opacification occurs after RLE, which should be done?

- ☐ YAG capsulotomy
- ☐ Wait 4-6 months
- ☐ Cataract surgery
- ☐ Nothing, this is a normal post-RLE finding

10. All of the following are potential RLE complications except:

- ☐ Uveitis
- ☐ Cystoid Macular Edema
- ☐ Residual refractive error
- ☐ Posterior capsular opacification

Posterior Scleritis Secondary to Sarcoidosis

Victoria Branca, OD; Amanda Hunter, OD; Fatima Raposo, OD; Paul B Greenberg, MD, MPH; Robert H Janigian, MD; Elina Goman-Baskin, OD

Abstract

Purpose: To report a case of posterior scleritis presumed secondary to sarcoidosis.

Methods: Case presentation.

Results: A 79-year-old male with a history of sarcoidosis presented with asymptomatic conjunctival injection and chemosis of the right eye. The patient presumptively was diagnosed with anterior scleritis of the right eye. At a follow-up visit, examination and diagnostic imaging revealed choroidal folds and increased choroidal thickness. He was diagnosed with posterior scleritis presumed secondary to sarcoidosis in the right eye. The patient was started on topical and oral steroids and the choroidal thickness and folds improved at one-week follow-up.

Conclusion: Posterior scleritis is an unusual manifestation of sarcoidosis, but it should be considered in the differential diagnosis of patients with sarcoidosis who present with a red, painful eye, and choroidal folds and thickening.

Key words: posterior scleritis, choroidal folds, sarcoidosis

Introduction:

Posterior scleritis is a rare but potentially vision-threatening condition that is often misdiagnosed due to its variable clinical presentation.[1] Posterior scleritis represents 2-12% of all cases of scleritis, half of which can be associated with systemic disease, most commonly autoimmune inflammatory conditions.[1-5] Posterior scleritis is uncommonly associated with sarcoidosis.[6-7] Herein we present a case of posterior scleritis in a patient with sarcoidosis and review its diagnosis and management.

Case Presentation:

A 79-year-old male reported to the eye clinic with a complaint of a red and swollen right eye for two days. He denied symptoms of itchiness, pain, or photophobia. Ocular history was remarkable for an isolated episode of anterior scleritis in the left eye one year prior to presentation. His medical history was positive for pulmonary sarcoidosis diagnosed in 2015, hypertension, and hyperlipidemia. His medications included atorvastatin, atenolol, lisinopril, and apixaban. He was not currently being treated for sarcoidosis.

Upon examination, the patient's best-corrected visual acuity was 20/40 for both the right eye and left eye. Finger counting fields revealed a superior field constriction in the right eye with full fields in the left eye. Pupils and extraocular motilities were unremarkable. An anterior segment exam showed upper lid inflammation with a mild ptosis, moderate conjunctival/scleral injection and chemosis, trace corneal microcystic edema adjacent to the nasal limbus, and an anterior chamber reaction of 1+ cells with no flare in the right eye (Figure 1). The left eye was unremarkable. There was a clear and centered PCIOL in the right eye and a mild nuclear sclerotic cataract in the left eye. Intraocular pressures with Goldmann applanation tonometry were 18mmHg in the right eye and 19mmHg in the left eye. Undilated examination of the posterior pole was unremarkable in both eyes.

The patient was diagnosed with non-granulomatous anterior uveitis of the right eye with a suspected anterior scleritis. The gross superior visual field constriction can be attributed to the edematous right upper eyelid. The patient was prescribed topical prednisolone acetate 1% every four hours while awake for the right eye, and to follow-up the next day.

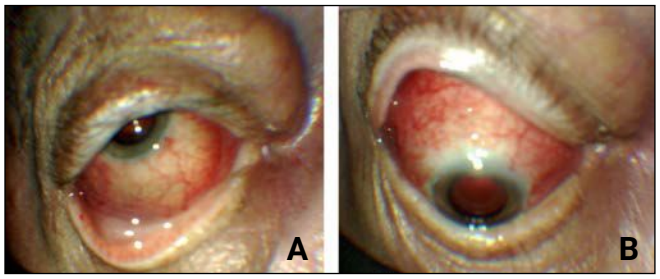

Figure 1A-B. Initial anterior segment presentation of the right eye; moderate conjunctival/scleral injection and chemosis.

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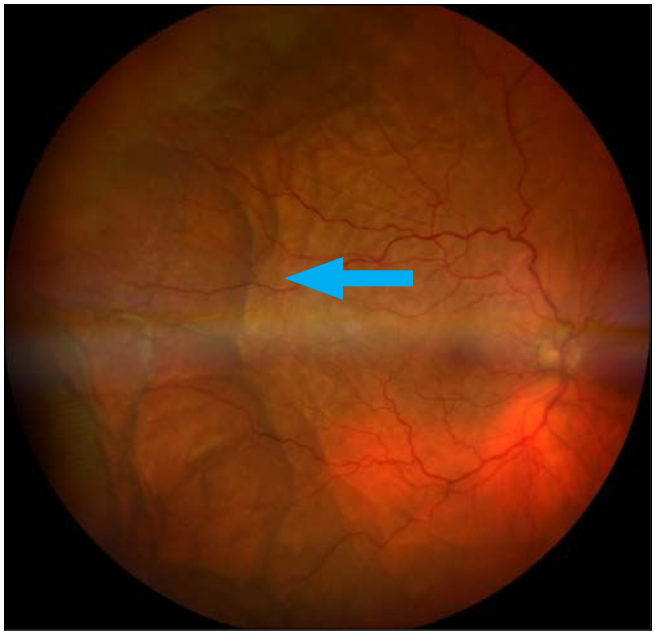

Figure 2. Posterior pole presentation of the right eye with presence of choroidal effusion (blue arrow) in the temporal periphery encroaching the macula.

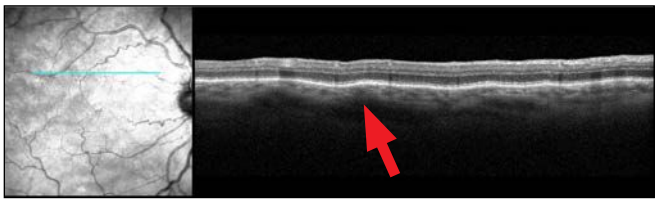

Figure 3. Macular optical coherence tomography (OCT) of the right eye with choroidal folds (red arrow).

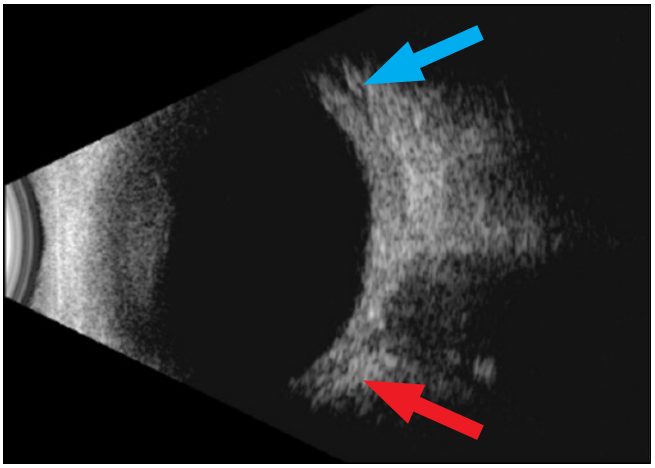

Figure 4. B-scan of the right eye with indicative thickened posterior sclera (blue arrow) and positive T-sign (red arrow).

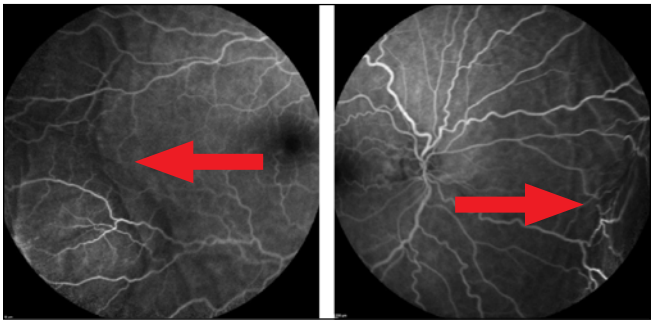

Figure 5A-B. Fluorescein angiography of the right eye indicating choroidal folds (red arrows).

At the one-day follow-up the patient reported mild improvement of the right upper lid inflammation, and minimal improvement in conjunctival/scleral injection and chemosis. The anterior chamber reaction was improved to trace cells. Intraocular pressure was 9mmHg in the right eye. Dilated fundus examination was unremarkable. The patient was instructed to continue topical prednisolone acetate 1% to the right eye every four hours while awake and a referral to ophthalmology service was arranged for the next day.

In the ophthalmology clinic, the patient reported his symptoms had worsened. The right upper lid inflammation had returned and there was moderate conjunctival/scleral injection and chemosis, along with moderate amount of microcystic edema adjacent to the nasal limbus. The anterior chamber was deep and quiet. Topical administration of phenylephrine 2.5% resulted in no blanching of the engorged vessels, and fundus examination remained unremarkable. The patient was diagnosed with diffuse anterior scleritis of the right eye. Oral prednisone 50mg daily, and oral pantoprazole 20mg daily for gastrointestinal prophylaxis. The patient was instructed to discontinue prednisolone acetate 1% and to return to clinic in four days for follow-up.

Laboratory work-up including complete blood count, rheumatoid factor, antinuclear antibody, angiotensin-converting enzyme, antineutrophil cytoplasmic antibodies, rapid plasma regains, fluorescent treponemal antibody absorption, and compliment component 3 and component 4 were unremarkable. Erythrocyte sedimentation rate results were abnormally elevated at 87 mm/hour, and a T-spot testing was ordered but not obtained.

When the patient returned for follow-up four days later, there was noted improvement of the right upper lid inflammation. The conjunctival/scleral injection was mild to moderate, and the corneal microcystic edema was improving. Dilated fundus examination revealed presence of a choroidal effusion in the temporal periphery of the right eye (Figure 2). Macular ocular coherence tomography (OCT) showed RPE undulation consistent with choroidal folds (Figure 3). The posterior sclera and choroid were noted to be thickened with B-scan ultrasonography. A faint "T-sign" was also present consistent with fluid in the subtenon space (Figure 4). Fundus fluorescein angiography (FFA) showed no delays in choroidal, arterial, or venous filling. No signs of optic disc leakage or staining of retinal vasculature was present (Figure 5A-B).

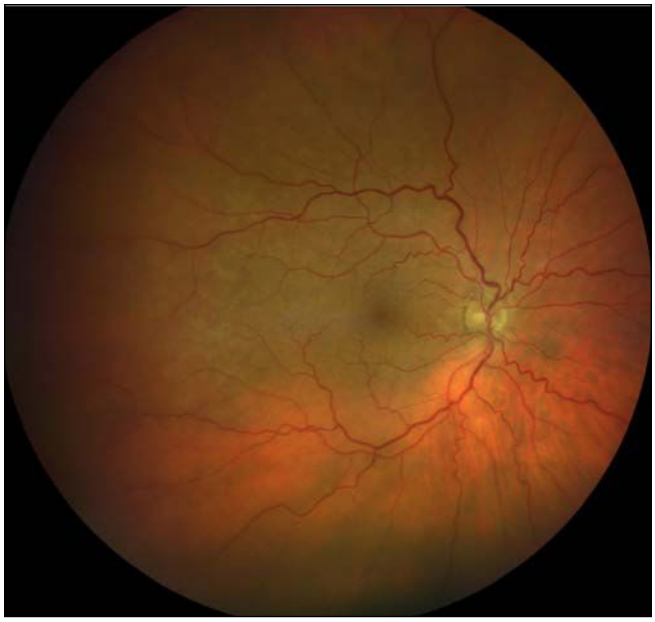

Figure 6. Posterior pole presentation of the right eye with resolution of choroidal folds in the temporal periphery and macula.

The diagnosis was revised to posterior scleritis, likely secondary to sarcoidosis. The patient was instructed to continue the oral steroid and start use of topical difluprednate 0.05% four times daily, and topical atropine sulfate 1% two times daily and to follow-up in one week.

The patient returned for follow-up one week later with improvement in vision of the right eye to 20/25, significant improvement of the anterior segment findings and resolution of the choroidal effusion (Figure 6 & 7). Prednisone was decreased to 40mg daily with a 5mg per week taper. The patient's primary care physician was alerted of the patient's ocular findings and a consultation was placed for further evaluation with rheumatology.

Discussion:

This report highlights a case of posterior scleritis presumed secondary to sarcoidosis. While it presented initially as an anterior scleritis, the subsequent development of a choroidal effusion and folds heralded the development of a posterior scleritis. The patient responded well to a course of oral steroids.

Scleritis

Scleritis is a severe ocular inflammatory condition that can present unilaterally or bilaterally and affects the anterior and/or posterior sclera.[2] Scleritis can be idiopathic, infectious, or non-infectious.[2] The pathophysiology of scleritis is unknown but typically occurs more often in persons with autoimmune inflammatory conditions, most commonly rheumatoid arthritis.[2,8] Scleritis is not often associated with sarcoidosis but should be considered as a differential diagnosis when an episode occurs.[6,7]

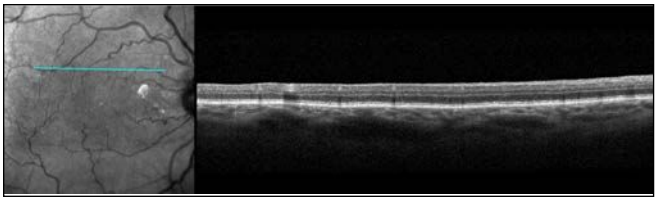

Figure 7. Macular optical coherence tomography (OCT) of the right eye with resolution of choroidal folds.

Ocular involvement occurs in approximately 20-30% of patients with sarcoidosis and may be the only presenting symptom in about 5% of these patients.[6,7,11] Sarcoidosis-related scleritis is an uncommon ocular manifestation, but may present as anterior diffuse, anterior nodular, or posterior scleritis.[6] Diffuse anterior scleritis is generally the most common inflammatory presentation[2]. The less frequent presentation, posterior scleritis, makes up 2-12% of all scleritis cases.[3-5]

The hallmark presenting symptom of posterior scleritis is moderate to severe deep, dull, boring pain.[1,3,9] In addition, there may be associated pain on extraocular motility, proptosis, decreased vision, and an associated anterior scleritis.[1-3] However, it not unusual for a patient to present with absence of any ocular pain, as was the case with our patient.[1-3]

Clinical signs of posterior scleritis are variable and may include eyelid edema, ciliary or conjunctival congestion, anterior uveitis, corneal edema, shallowing of the anterior chamber, limitation of extraocular motility, retinal striae, disc swelling, serous retinal detachment, choroidal folds, and/or choroidal effusions.[1,4,9]

Ocular Diagnostic Testing

Due to the variability of its clinical presentation, diagnostic imaging plays a vital role in diagnosing those with suspected posterior scleritis (Table 2.)[1,2,10]

Systemic Workup

All patients that present with posterior scleritis should have a systemic workup to identify the cause of the condition and any associated metabolic condition (Table 3).[1,2]

Treatment/management of posterior scleritis

Posterior scleritis can be challenging to diagnose and requires immediate therapy.[1,2] The first line of treatment can be oral NSAIDs such as ibuprofen 600-800mg QID, piroxicam 20mg QD, or naproxen 375mg BID.[1,2,6,13] Oral corticosteroids can also be used initially or in cases where treatment with oral NSAIDs are ineffective.[1,2,13] The typical treatment includes prednisone 1mg/kg/day or approximately 60-80mg daily.[1,2,13] If the condition is unresponsive to corticosteroids, the next step involves immunosuppressive/antimetabolite agents such as methotrexate, azathioprine and mycophenolate.[1,2,13] However, these agents are not fast-acting and could take up to six months to fully take effect.[1] In cases where patients require a more rapid solution, biologic tumor necrosis factor (TNF) inhibitors such

Table 2. Diagnostic imaging that can aid in diagnosis of suspected posterior scleritis

Diagnostic Imaging	Findings
B-Scan Ultrasonography	• Choroidal thickening • "T-sign" consistent with scleral thickening and fluid collecting in the posterior episcleral space around the optic nerve
Optical Coherence Tomography (OCT)	• Choroidal thickening • Subretinal fluid
Fundus Fluorescein Angiography (FFA)	• Highlights choroidal folds • Exclusion of other similarly presenting conditions
Indocyanine Green Angiography (ICG)	• Choroidal hyperfluorescence – possibly indicating areas with maximal inflammatory activity
Orbital Computed Tomography (CT)/ Magnetic Resonance Imaging (MRI)	• If B-scan is inconclusive, can identify posterior inflammation and increased choroidal thickness Rules out other conditions, including idiopathic orbital inflammation and myositis

Table 3. Common laboratory testing and diagnostic imaging that should be considered in cases of posterior scleritis

Test	Associated Disease
PPD or QuantiFERON Gold	Tuberculosis
RPR FTA-Abs	Syphilis
Rheumatoid Factor Anti-CCP	Rheumatoid arthritis
ACE Lysozyme Chest X-ray	Sarcoidosis
ANCA	Granulomatous polyangiitis (Wegener granulomatosis); polyarteritis nodose
HLA-B27	Ankylosing spondylitis, reactive arthritis, psoriatic arthritis, ulcerative colitis, Crohn's
CBC ESR CRP	Inflammatory or infectious conditions
ELISA Western blot IgG, IgM	Lyme disease

as rituximab or infliximab are initiated.[1,2,6,13] Prompt diagnosis, appropriate, and aggressive treatment are key to quick recovery and preservation of vision.[1]

Conclusion:

Posterior scleritis is a rare but potentially vision threatening ocular condition. Prompt diagnosis and collaboration with primary care and, as needed, subspecialty physicians are essential to the management of patients with posterior scleritis.

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POST-COURSE TEST

Posterior Scleritis Secondary to Sarcoidosis: A Case Report

1. **Posterior scleritis represents 2-12% of all cases of scleritis, half of which can be associated with which of the following comorbidities?**
- ☐ Obesity
- ☐ Autoimmune inflammatory conditions
- ☐ Sarcoidosis
- ☐ Cancer

2. **What is the hallmark presenting symptom of posterior scleritis?**
- ☐ Severe, deep, dull, boring pain
- ☐ Conjunctival hyperemia
- ☐ Corneal edema
- ☐ Anterior chamber reaction

3. **Which of the following is NOT likely an associated symptom of posterior scleritis?**
- ☐ Associated anterior scleritis
- ☐ Extraocular motility
- ☐ Endophthalmitis
- ☐ Decreased vision

4. **Sarcoidosis-related scleritis is an uncommon ocular manifestation that may present as which of the following?**
- ☐ Posterior scleritis
- ☐ Anterior diffuse scleritis
- ☐ Anterior nodular scleritis
- ☐ All of the above

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5. Using B-Scan Ultrasonoigraphy, what characteristic finding can aid in accurately diagnosing posterior scleritis?

- ☐ Posterior inflammation
- ☐ T-sign consistent with scleral thickening and fluid collection in the posterior episcleral space around the optic nerve
- ☐ Choroidal hyperfluorescence
- ☐ Subretinal fluid

6. Which diagnostic imaging technique can identify 'choroidal hyperfluorescence - possible indicating areas with maximal inflammatory activity'?

- ☐ Indocyanine Green Angiography
- ☐ Optical Coherence Tomography
- ☐ Orbital Computed Tomography/Magnetic Resonance Imaging
- ☐ Fundus Fluorescein Angiography

7. Which of the following systemic work up would determine that Tuberculosis is the underlying condition associated with posterior scleritis?

- ☐ HLA-B27
- ☐ ANCA
- ☐ Chest X-ray
- ☐ PPD or QuantiFERON Gold

8. Which of the following common laboratory tests should be ordered to investigate if Lyme disease was an underlying condition associated with presenting posterior scleritis?

- ☐ ELISA
- ☐ CBC
- ☐ Rheumatoid factor
- ☐ ACE

9. What is the first line of treatment of posterior scleritis?

- ☐ Intravitreal steroids
- ☐ Oral NSAIDs
- ☐ Immunosuppressive/antimetabolite agents
- ☐ Biologic TNF inhibitors

10. In cases where patients require a more rapid solution, which treatment option is most effective?

- ☐ Piroxicam
- ☐ Prednisone
- ☐ Azathioprine
- ☐ Rituximab

A Late Presentation of Unilateral Idiopathic Retinal Vasculitis, Aneurysms, and Neuroretinitis (IRVAN) Syndrome

Zachary G. Walburg, OD; Robert H. Janigian Jr., MD; Joseph Mega, OD; Paul B Greenberg, MD, MPH; Amanda Hunter, OD

Abstract

Although uncommon, idiopathic retinal vasculitis, aneurysms, and neuroretinitis (IRVAN) syndrome can lead to vision-threatening complications if not diagnosed and treated appropriately. This case report describes an atypical presentation of unilateral IRVAN syndrome, which resulted in significant visual loss in an elderly male.

Introduction

Idiopathic retinal vasculitis, aneurysms, and neuroretinitis (IRVAN) syndrome is a rare and usually bilateral condition, which can result in a vision-threatening ischemic and exudative retinopathy.[1,2] The clinical presentation of IRVAN syndrome is variable, which can potentially complicate the accurate diagnosis of the condition.[1,2] Herein, we describe an atypical presentation of unilateral IRVAN syndrome in an elderly male.

Case Presentation

A 76-year-old male with longstanding visual loss in his left eye, presented for a follow-up examination secondary to complications from an unknown exudative retinopathy. The patient had no new complaints and reported stable vision in both eyes.

The retinopathy was noted when the patient initially presented to the eye clinic at 59 years of age. The rest of the patient's past ocular history was notable for pseudophakia in both eyes, with cataract surgery performed three years prior to presentation, and retinal emboli in the left eye resulting from carotid occlusive disease. The patient's medical history included hyperlipidemia, alcoholic cirrhosis with resolved hepatic encephalopathy seven years prior to presentation, peripheral sensory neuropathy, anemia, and bilateral carotid atherosclerotic disease. His medications included folic acid, simvastatin, gabapentin, spironolactone, thiamine, omeprazole, diphenhydramine, and furosemide.

On examination, the patient's best-corrected visual acuity was 6/6 in the right eye and counting fingers at 0.3 meters in the left eye. The left pupil was fixed and dilated. A left relative afferent pupillary defect was noted by reverse testing. Confrontation visual fields were full and extraocular muscle motility testing revealed full range of motion in each eye. Slit-lamp biomicroscopy was remarkable for centered posterior chamber intraocular lenses in both eyes with early posterior capsular opacification centrally in the left eye.

On dilated fundus examination, the right eye was notable only for trace retinal pigment epithelium mottling in the macula. In the left eye, the media was clear, the optic nerve was pale, the retinal vasculature was attenuated with sheathed and sclerosed arterioles, and three incidental retinal emboli were noted: one at the first bifurcation of the superotemporal and superonasal arteries and one at the second bifurcation of the inferotemporal artery. Additionally, subretinal fibrosis and atrophy located at the fovea were noted with associated intraretinal hemorrhages, extensive lipid exudates and fibrotic macroaneurysms superior and nasal to the optic nerve. No neovascularization or cotton wool spots were appreciated (figure 1). The lipid exudates were improved compared to photos taken 20 months prior.

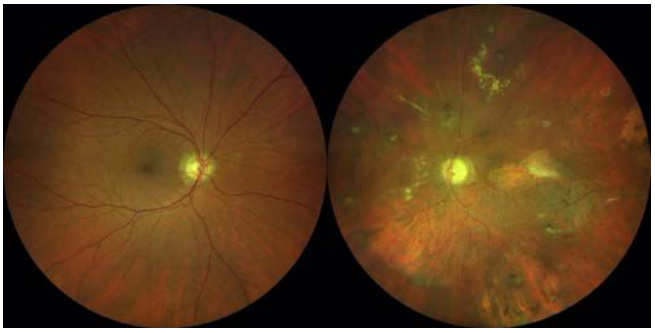

Figure 1. Widefield fundus photographs of both eyes: the right eye was unremarkable; the left eye showed optic nerve pallor, attenuated and sheathed retinal vasculature, scattered retinal hemorrhages and exudates, macular retinal pigment epithelium atrophy, thrombosed arterial macroaneurysms superior, nasal inferotemporal to the nerve, and three emboli in the arterioles.

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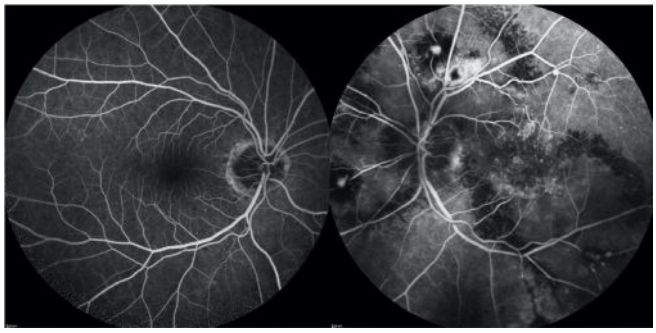

Figure 2. Mid-phase fluorescein angiography (2010): the right eye showed staining of drusen; the left eye showed filling of macroaneurysms, capillary nonperfusion along the inferotemporal, inferonasal and superonasal arcades, occluded retinal vessels and knob-like aneurysmal dilatations along the inferotemporal arcade. The macula showed transmission defects and staining due to severe RPE disease and subretinal fibrosis.

Optical coherence tomography (OCT) of the macula was unremarkable in the right eye. In the left eye, there was a hyperreflective band at the vitreoretinal interface consistent with an epiretinal membrane. There was intraretinal hyper-reflective material consistent with the lipid exudates seen clinically, as well as loss of the outer retinal layers with hyper-reflectivity consistent with RPE atrophy.

Fluorescein angiography (FA) demonstrated early phase occlusion of retinal vessels along the inferotemporal arcade. There was hypofluorescence due to dense lipid deposits and capillary non-perfusion, which was present in the early phase lasting through the late phases. The lipid deposits were appreciated along the superotemporal and inferotemporal arcades, while the capillary non-perfusion was present along the inferotemporal, inferonasal and superonasal arcades. There were scattered bulbous hyperfluorescent lesions consistent with macroaneurysms and microaneurysms. The macroaneurysms were located superior and nasal to the optic nerve and along the inferotemporal arcades, with the microaneurysms scattered throughout the posterior pole concentrated within the arcades. There was hyperfluorescent leakage of the retinal vasculature without disc leakage or leakage consistent with retinal neovascularization (figure 2).

The above findings were consistent with an atypical presentation of IRVAN syndrome. The patient was managed conservatively due to the relatively quiescent disease and the poor prognosis for visual recovery secondary to foveal atrophy and fibrosis.

Discussion

Idiopathic retinal vasculitis, aneurysms and neuroretinitis syndrome is a rare condition of uncertain etiology that can masquerade as a variety of conditions including retinal arterial macroaneurysms,[3] Eale's disease,[4] Behçet's disease,[5] sarcoidosis,[6] and adult-onset Coats' disease.[7] Several characteristics can help distinguish between these conditions and IRVAN syndrome (Table I).

Table 1. Differential diagnoses for IRVAN Syndrome with the characteristics of each condition that differ from IRVAN syndrome

Differential Diagnosis	Key Features
Retinal arterial macroaneurysm[4]	• associated with systemic hypertension • usually diagnosed in the 6th to 7th decade • intraretinal, preretinal and vitreous hemorrhages are present • no neuroretinitis
Eale's Disease[5]	• usually diagnosed in younger males • positive tuberculin test • vestibuloauditory deficits • cerebral infarction • periphlebitis without aneurysms
Behçet's Disease[6]	• occlusive panarteritis • oral aphthous ulcers • genital ulcers • hypopyon
Sarcoidosis[7]	• systemic complications • usually diagnosed in the 3rd to 4th decade • African American race • hypopigmented chorioretinal scars
Adult-onset Coats' Disease[8]	• male predilection • typically does not progress after onset • typically localized to temporal retina

IRVAN syndrome is classically diagnosed in the third to fourth decades of life, with a greater predilection for females.[2] The majority of cases present bilaterally, although there have been reports of unilateral cases, as well as cases that began in one eye and progressed to involve the fellow eye.[8]

This patient's formal diagnosis of IRVAN syndrome was made 24 years after his initial presentation. This discrepancy was most likely related to the patient's atypical presentation of IRVAN syndrome, including late presentation, male sex, and unilateral findings.

In contrast to these atypical findings, the patient's sheathed and sclerosed vasculature from a history of vasculitis, multiple aneurysmal dilations, exudative retinopathy, and capillary non-perfusion as seen on FA were all consistent with IRVAN syndrome. No optic nerve edema was noted, but the presence of optic nerve pallor suggests of a history of chronic optic nerve inflammation. The retinal emboli resulted from the patient's established carotid artery disease and were unrelated to IRVAN syndrome.

As described by Samuel et. al. (2007), IRVAN syndrome can be categorized into five stages. Stage 1 is characterized by macroaneurysms, exudation, neuroretinitis and retinal vasculitis. Stage 2 is characterized by angiographic evidence of capillary non-perfusion resulting from occlusive retinal vasculitis. Stage 3 occurs when there is development of posterior segment neovascularization with or without a vitreous hemorrhage.

Stage 4 is characterized by the presence of anterior segment neovascularization, while stage 5 occurs with the development of neovascular glaucoma.[2] Based on these stages, our patient had stage 2 IRVAN syndrome in his left eye.

The visual prognosis of IRVAN syndrome is related to the severity of the disease, and it correlates with the degree of retinal ischemia and the stage of the condition at the time of initiation of treatment.2 Panretinal photocoagulation (PRP) is recommended if there is retinal ischemia involving two or more quadrants of the retina, as in stages 2 through 5.[11]

In a study by Samuel et. al. (2010), eyes treated at various stages of IRVAN syndrome were evaluated for long-term visual prognosis. They found that eyes treated at stage 2 maintained a best-corrected visual acuity (BCVA) of 20/20, whereas 25% of stage 3 eyes became 20/200 or worse, and treatment initiated in stages 4 or 5 resulted in 50% of eyes progressing to severe vision loss.[2]

Although retinal neovascularization may not impact the likelihood of progression of IRVAN syndrome,2 patients with active neovascularization are at risk for additional vision-threatening conditions, such as vitreous hemorrhage and retinal detachment. Thus, anti-vascular endothelial growth factor (anti-VEGF) agents are indicated in cases of active neovascularization (stages 3 through 5).[2]

As with other retinal vascular diseases, treatment of exudative maculopathy in IRVAN syndrome usually involves focal laser and/or anti-VEGF, but there have been promising results of treatment of macular edema with anti-tumor necrosis factor (anti-TNF) agents, such as infliximab,9 based on their ability to mediate ocular inflammation. Cheema et. al. (2011) reported two cases of macular edema in IRVAN syndrome that responded to treatment with the anti-TNF agent infliximab.

Despite the success of infliximab in treating macular edema caused by IRVAN syndrome, anti-TNF agents should be used with caution due to the risk of activation of opportunistic infections and latent tuberculosis.[9] Additionally, while infliximab can be beneficial in managing macular edema, it does not have any impact on capillary nonperfusion, highlighting the necessity of combination therapy with laser photocoagulation or anti-VEGF agents to manage the risk of development of neovascularization.[10] Direct photocoagulation of arterial macroaneurysms is generally not advised as there is a risk of occlusion of the blood vessel and worsening of retinal ischemia.[10]

Conclusion

Idiopathic retinal vasculitis, aneurysms, and neuroretinitis syndrome can be a highly variable disease with potentially devastating visual complications. Therefore, prompt diagnosis and management of IRVAN syndrome is important to prevent vision-threatening complications.

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POST-COURSE TEST

A Late Presentation of Unilateral Idiopathic Retinal Vasculitis, Aneurysms, and Neuroretinitis (IRVAN) Syndrome

1. What does the acronym IRVAN stand for?

- ☐ Intra-retinal vascular advanced neoplasm
- ☐ Idiopathic retinal vasculitis, aneurysms and neuroretinitis
- ☐ Intracameral retinal vasculitis, aneurysms and neuroretinitis
- ☐ Idiopathic retinal vasculitis and neoplasms

2. Which of the following is a potential masquerade of IRVAN syndrome?

- ☐ Retinal Arterial macroaneurysms
- ☐ Eale's disease
- ☐ Sarcoidosis
- ☐ All of the above

3. In what decade of life is IRVAN syndrome classically diagnosed?

- ☐ First and second
- ☐ Second and third
- ☐ Third and fourth
- ☐ Fourth and fifth

4. Which stage of IRVAN syndrome is best described as 'macroaneurysms, exudation, neuroretinitis and retinal vasculitis'?

- ☐ Stage 1
- ☐ Stage 2
- ☐ Stage 3
- ☐ Stage 4

5. Which stage of IRVAN syndrome is characterised by the development of neovascular glaucoma?

- ☐ Stage 3
- ☐ Stage 4
- ☐ Stage 5
- ☐ None of the above

6. According to Samuel et al. (2010), eyes treated at stage 2 maintained a best-corrected visual acuity of which of the following?

- ☐ Light perception
- ☐ Counting fingers
- ☐ 20/200
- ☐ 20/20

7. According to Samuel et al. (2010) eyes treated at stage 4 and 5 resulted in what percentage of eyes progressing to severe vision loss?

- ☐ 30%
- ☐ 40%
- ☐ 50%
- ☐ 60%

8. What treatment option is indicated in cases with neovascularization (stages 3 through 5)?

- ☐ Anti-VEGF injection
- ☐ Panretinal photocoagulation
- ☐ Anti-TNF injection
- ☐ Direct photocoagulation

9. What novel treatment option has shown promising results of treatment of macula edema in IRVAN syndrome?

- ☐ Anti-VEGF injection
- ☐ Panretinal photocoagulation
- ☐ Anti-TNF injection
- ☐ Direct photocoagulation

10. What treatment option is generally not advised due to the risk of occlusion of blood vessel and worsening of retinal ischemia?

- ☐ Anti-VEGF injection
- ☐ Panretinal photocoagulation
- ☐ Anti-TNF injection
- ☐ Direct photocoagulation

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Animal Eponyms in Eye Care

Alely Hostia, OD; Jeffrey Kyle Wells, OD; Alexandra Vlad, OD; Ruth Hyatt, OD; Darcy L. Eberle, OD; Son Thai Ho, MD

Synopsis

There are many eponyms derived from the animal world that help describe what is observed during an ophthalmological examination. These zoologically-based eponyms tell a story between the animal kingdom and the human eye. Medical nomenclature could be described as suffering from eponymophilia. Eponymous descriptions are ubiquitous in the field of medicine as seen in text in these fields: dermatology,[1] neurology,[2] and trichology.[3] Eponyms, however practical, are profoundly affected by local geography and culture,[4] which can contribute to confusion and misunderstanding, particularly amongst practitioners from different cultures and locations. Deriving meaning from eponyms often necessitates ethnocentric or linguistically exclusive knowledge.5 While the debate surrounding whether to continue the use of eponyms in medicine roars on,4 to the authors' knowledge, a review of animal eponyms used in ophthalmology has been limited, as such, this manuscript will review animal eponyms encountered in ophthalmology and optometry with the aim to clarify meaning and provide a useful reference for practicing and trainee eye physicians. As this work was undertaken to review commonly encountered conditions, no detailed pathophysiology will be described as it is beyond the scope of this article. Of note, not all eponyms reviewed are based on physical appearances (e.g. chicken eyes).

Keywords: animal eponyms, disease, eponyms, eye, ophthalmology, optometry, sign, syndrome

Introduction

The characterization of ophthalmological conditions through pertinent findings and descriptions is continually expanding. Mnemonics, triads, acronyms, acrostics, chunking, and eponyms are commonly utilized in medicine to enhance the understanding of a plethora of medical conditions. Eponyms can aid in identifying and understanding ophthalmological findings. Unfortunately, although some zoological eponyms may have a derogative connotation, causing some authors to advocate their omission,[3] the eponyms reviewed here do not fall under that subgroup. Instead, they allow conditions to become more memorable, and in many cases, help newer clinicians make the connection between certain diseases and their manifestations. Some eponyms (e.g. cat scratch disease) may nudge novel clinicians down the correct systemic work-up path.

Animal Eponyms

1 Bag of worms: the appearance of numerous arteriovenous anastomoses seen as enlarged, tortuous blood vessels, with the artery and vein appearing similar. Treatment is generally not required; however ipsilateral lesions involving the brain can be found in Wyburn-Mason syndrome.[6]

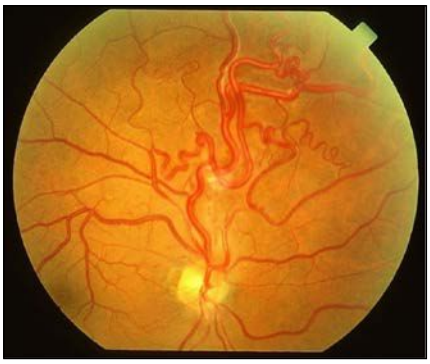

Figure 1. Retinal racemose angioma in Wyburn Mason syndrome. This image was originally published in the Retina Image Bank® website. Henry Kaplan. Wyburn Mason Syndrome. Retina Image Bank. 2013; 6211. ©the American Society of Retina Specialists

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2 Bear tracks: also known as grouped pigmentation of the retina, bear tracks are flat, well-demarcated, hyperpigmented lesions in the retinal pigment epithelium (RPE) and are a subgroup of congenital hypertrophy of the RPE (CHRPE).[7] CHRPE can be associated with familial adenomatous polyposis if bilateral, occurs in multiple quadrants, has a pisciform shape, and irregular borders.[7]

Figure 2. Bear tracks. ©EyeRounds.org

Figure 3. Bear tracks.

3 Birdshot chorioretinopathy: a rare autoimmune posterior uveitis with poorly understood pathogenesis[8] characterized by multiple lightly colored oval choroid lesions (the long axis is radial to the optic disc) with a juxtopapillary predilection.[9] Presentation is most commonly bilateral and symmetric.[8] Although uncommon, a mild anterior uveitis can be present.[8] Cystoid macular edema is common and is the predominant cause of vision loss.[8] The name was coined because the pattern of these lesions mimics the pattern of birdshot from shotgun scatter.[10]

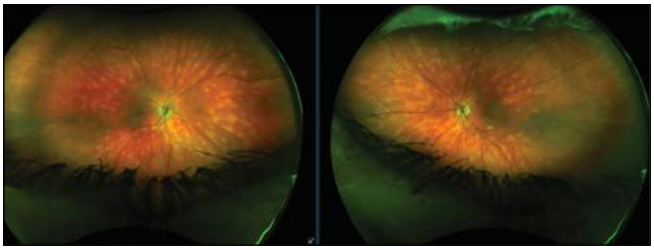

Figure 4. Birdshot chorioretinopathy. ©2020 American Academy of Ophthalmology

Figure 5. Paper shooting target depicting birdshot from shotgun scatter.

4 Bull's eye:
a. Bull's eye maculopathy: the distinct pattern of retinopathy in patients with chloroquine[11] or hydroxycholoroquine[12] toxicity that may appear funduscopically as macular granularity, thinning of parafoveal retinal pigmented epithelium and photoreceptors on optical coherence tomography, or a ring defect on visual field testing.[12] The mechanism of toxicity is not understood.[12] Retinopathy in patients of Asian ancestry may be more peripheral than that of Caucasians.[12]

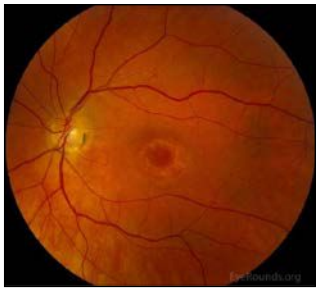

Figure 6. Bull's eye maculopathy. ©EyeRounds.org

Figure 7. Target.

b. Bull's eye rash: also known as erythema migrans, a gradually expanding circular skin lesion that develops at the site of a tick bite containing Borrelia burgdorferi and signifies localized infection.[13-15]

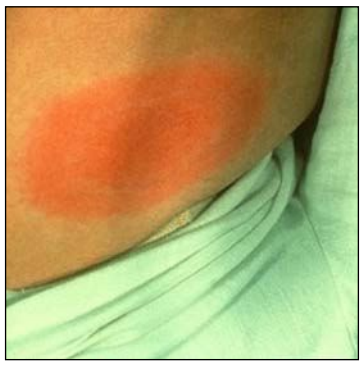

Figure 8. Erythema migrans in Lyme disease. This image was originally published in the Retina Image Bank® website. Henry Kaplan. Uveitis. Retina Image Bank. 2013; 4904. ©the American Society of Retina Specialists

5 Butterfly rash: a raised or flat malar rash that is non-pruritic and occurs over the cheeks and bridge of the nose, with nasolabial folds spared.[16,17] A feature of systemic lupus erythematosus,[16,17] an autoimmune disease of unknown etiology that affects multiple organ systems.[17] Lupus is Latin for wolf.[18] Translated[19] as "a systemic disease in which a wolf turns red". It may also be associated with hydroxychloroquine use.[20]

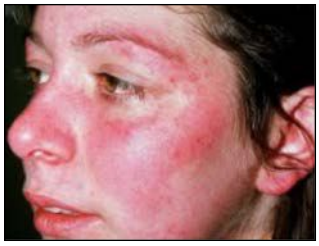

Figure 9. Butterfly rash. DermNet (https://creative commons.org/licenses/by-nc-nd/3.0/nz/legalcode)

Figure 10. Butterfly

6 Butterfly-shaped pattern dystrophy: a bilateral, autosomal dominant condition[21] in which pigmented lipofuscin[22] accumulates in the macular retinal pigment epithelium in a pattern resembling the wings of a butterfly.[23] The condition is due to genetic mutations at the same locus as mutations found in pattern dystrophies, retinitis pigmentosa, and fundus flavimaculatus, resulting in photoreceptor membrane integrity.[22]

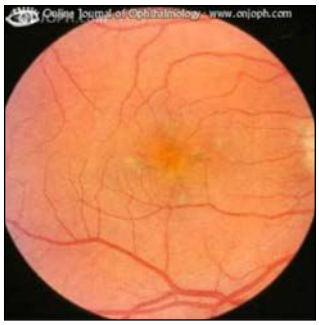

Figure 11. Butterfly-shaped pattern dystrophy.
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Figure 12. Butterfly

7 Camel's sign: also known as camel's second hump sign,[24] the appearance of a second peak on the densitometry graph of a Scheimpflug image[25] due to higher reflection of Descemet's membrane indicative of corneal guttata.[26]

8 Cat eye syndrome: coloboma and anal atresia resulting from an extra acrocentric chromosome, whose expression and transmission varies widely.[27] Ocular features of cat eye syndrome have sizeable variation- and coloboma may be found in the iris or choroid, and may be bilateral or unilateral.[27]

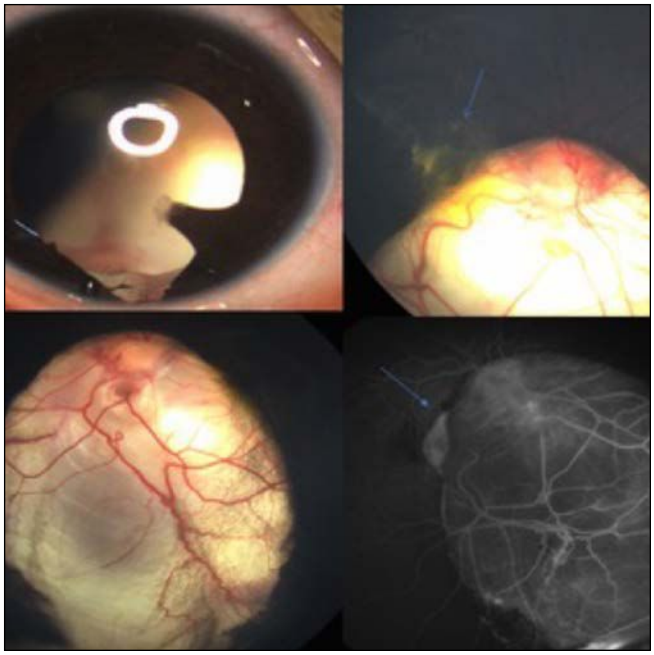

Figure 13. Iris and optic nerve coloboma in cat eye syndrome. Blue arrows depict choroidal neovascularization. This image was originally published in the Retina Image Bank® website. Sophia El Hamichi. Giselle De Oliveira. Cat Eye Syndrome. Retina Image Bank. 2020; 49706. ©the American Society of Retina Specialists

9 Cat scratch disease: a systemic illness contracted through a scratch or bite that transmits *Bartonella henselae*; the classic ocular manifestation is unilateral optic disc edema and exudates that form the pattern of a star at the macula (termed neuroretinitis), and visual acuity can recover after several weeks.[28] Mild inflammation of the anterior chamber and vitreous is common, as is retinal thickening and exudative retinal detachment.[28]

Figure 14. Neuroretinitis with macular star.
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Figure 15. Starburst.

10 Cat's paw retractor: an instrument used in ophthalmic procedures that involve retracting the skin, subcutaneous tissues, and ligaments.[29]

Figure 16. Cat's paw retractor.

Figure 17. Cat's paw.

11 Cattle track sign: also known as cattle trucking or box-carring, a beaded appearance of retinal vessels resulting from interrupted blood flow resulting in "sludge phenomenon", whereby there is a "stagnation of the arterial blood stream without diminution in the size of the arterial column".[30,31] Of note, in one large retrospective study, nearly all patients with total central retinal artery occlusion (CRAO) had a cattle track sign, which may be an important distinction as patients having incomplete CRAO showed a better prognosis.[30]

Figure 19. Cattle car.

Figure 18. Cattle track sign in central retinal artery occlusion. This image was originally published in the Retina Image Bank® website. Hyung-Woo Kwak. Central Retinal Artery Occlusion. Retina Image Bank. 2012; 1683. ©the American Societyof Retina Specialists

12 Chicken eyes: Vitamin A deficiency impedes rhodopsin production, resulting in poor rod function which manifests as nyctalopia. Chickens lack rod photoreceptors, making them night blind.[32]

13 Cotton wool spots: fluffy, white lesions caused by accumulated axoplasmic debris within the ganglion cell axon bundle due to arteriolar occlusion and ischemia.[33]

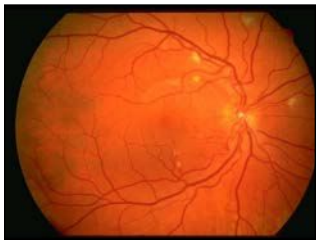

Figure 20. Cotton wool spots. ©2020 American Academy of Ophthalmology

Figure 21. Cotton wool ball.

14 Crab claws: also known as kissing birds or butterfly pattern,[34] the appearance of the corneal topographical pattern in which there is steepening of the inferior peripheral cornea with flattening along the vertical meridian,[35] suggestive of pellucid marginal degeneration (PMD) or keratoconus.[36] In PMD, peripheral corneal thinning in the 4:00 to 8:00 position occurs bilaterally, with epithelium remaining intact.[34]

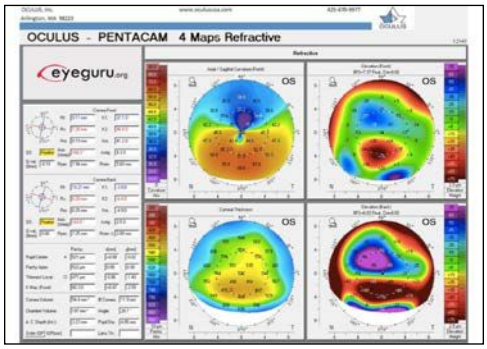

Figure 22. Crab claws or kissing dove pattern on axial curve.

Figure 23. Crab claw.

15 Crocodile shagreen: symmetrical, gray-white polygonal opacities with indistinct edges and clear zones in between (resembling the skin of a crocodile) in the central posterior corneal stroma.[37] Electron microscopy shows vacuoles throughout the corneal stroma.[37]

16 Crocodile tear syndrome: also known as gusto-lacrimation, following traumatic facial paralysis or Bell's palsy, aberrant gustatory nerve fiber regeneration to the lacrimal gland (rather than the salivary gland) causes non-emotional tearing whilst eating.[38]

17 Crow's feet: also known as periorbital rhytids, wrinkles that form at the lateral canthus due to hyperkinesia of the orbicularis oculi,[39] appearing similar to the talons of a crow.

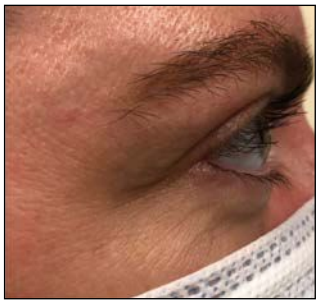

Figure 24. Crow's feet.

Figure 25. Crow's feet.

18 Cutaneous horns of the eyelid: a keratinous mass protruding from the skin, resembling animal horns (unlike animal horns, there is no bony core).[40] Most often occur on sun-exposed skin of elderly men, and lesions are usually asymptomatic.[40]

Figure 26. Cutaneous horn. ©EyeRounds.org, The University of Iowa

19 Elschnig's pearls: the accumulation of equatorial epithelial cells onto the posterior capsule after cataract surgery are clinically visible as "pearls" in posterior capsular opacification.[41]

Figure 27. Elschnig's pearls. This image was originally published in the Retina Image Bank® website. Rene Parada. Elschnig's Pearls. Retina Image Bank. 2015; 25591. ©the American Society of Retina Specialists

20 Fish eye disease: severe corneal opacification resulting from low high density lipoproteins cholesterol.[42]

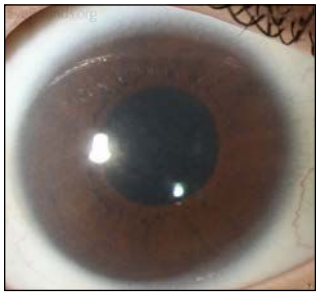

Figure 28. Corneal clouding. ©EyeRounds.org, The University of Iowa

Figure 29. Cloudy fish eye.

21 Halsted mosquito forceps and Hartman mosquito forceps: hemostats to clamp tissues or vessels[43] during ophthalmic surgeries.[44]

Figure 30. Mosquito forceps

22 Horseshoe retinal tear: a U-shaped flap tear which occurs most frequently in the superior temporal peripheral quadrant of the retina, probably due to a combination of gravity acting on the vitreous as well as thinner peripheral retinal tissue.[45]

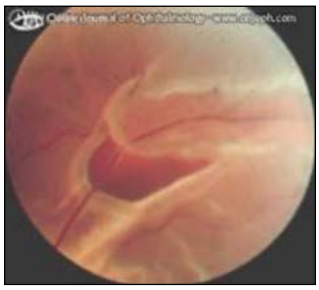

Figure 31. Horseshoe retinal tear. ©Online Journal of Ophthalmology

Figure 32. The heel of a horseshoe oriented with the base of the horseshoe retinal tear; the toe of a horseshoe oriented with the apex of the horseshoe retinal tear.

23 Lobster claw intraocular lens: also known as iris-claw lens[46], an intraocular lens used when there is not adequate capsular support[47] for a traditional intraocular lens.

Figure 33. Claw intraocular lens. ©EyeRounds.org, The University of Iowa

Figure 34. Lobster claw.

24 Leopard spots: also known as giraffe spots, a chorioretinopathy of characteristic hypo- and hyperpigmented spots.[48,49]

Figure 35. Leopard spots. This image was originally published in the Retina Image Bank® website. Mohammad Hossein Jabbarpoor Bonyadi. Miscellaneous. Retina Image Bank. 2019; 27167. ©the American Society of Retina Specialists

Figure 36. Spotted pattern of a leopard's coat.

25 Maddox wing: a dissociated test to control accommodation in pre-presbyopic patients that utilizes a septum to measure heterophoria at near.[50]

Figure 37. Maddox wing. Image courtesy of Neil Handley. The College of Optometrists, London UK. 2021.

26 Molluscum contagiosum: round, centrally umbilicated waxy popular lesions usually occurring in clusters found on the skin and mucous membranes caused by a poxvirus.51 Commonly seen in children, and suspicion of immunodeficiency is raised if found in adults.51

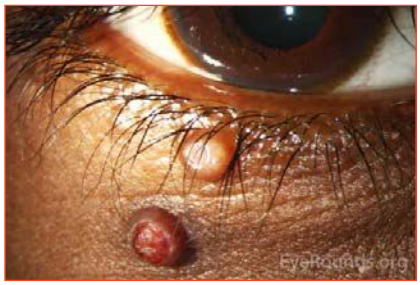

Figure 38. Molluscum contagiosum. ©EyeRounds.org

27 Moth-eaten iris: atrophy of the iris stroma and/or pigment epithelium, most notably in Fuch's heterochromic uveitis,[52] appearing as if eaten by the larvae of moths.[53]

Figure 39. Fuchs' heterochromic cyclitis.©EyeRounds.org

Figure 40. Moth-eaten garments.

28 Mucus fishing syndrome: chronic cycles initiated by ocular irritation leads to conjunctival mucus production which prompts the patient to mechanically remove ("fish") mucus, which in turn causes more irritation and a continuous cycle.[54]

Figure 41. "Fishing" for mucous.

29 Muscae volitantes: collagen fibril accumulation results in light scattering and perceived vitreous floaters which move when the eyes or head is moved,[55] derived from the Latin phrase meaning "flying flies".[56]

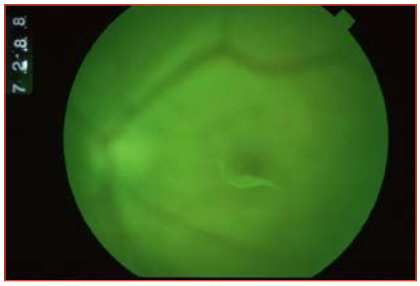

Figure 42. Vitreous floater. This image was originally published in the Retina Image Bank® website. Gary R. Cook. Pre-Macular Floater. Retina Image Bank. 2019; 29867. ©the American Society of Retina Specialists.

30 Mutton-fat keratic precipitates: leukocytes that appear on the corneal endothelium as 'greasy' fat globules,[57,58] similar to the fat of sheep. This can be an indicator of systemic disease such as tuberculosis, syphilis, or sarcoidosis.[57]

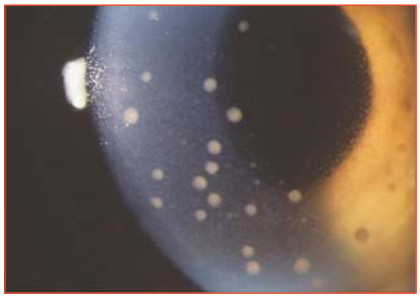

Figure 43. Mutton-fat keratic precipitates. ©2020 American Academy of Ophthalmology.

31 Octopus perimeter: a perimeter designed to be "as fast as an eight-armed technician", which was the original automated perimeter, marking the first time a device of its kind could store results and offer comparison to a normative database.[59]

32 Owl-eye facial rash: also known as "raccoon-like" appearance,[60] a periorbital cutaneous rash characteristically observed in neonatal lupus erythematosus.[61]

Figure 44. Owl-eye facial rash in neonatal lupus erythematosus. DermNet (https://creativecommons. org/licenses/by-nc-nd/3.0/nz/legalcode)

Figure 45. Owl.

33 Panda sign in nevus of Ota: bluish hyperpigmentation that persists after treating periorbital nevus of Ota lesion with laser surgery.[62] The exact mechanism is unknown but could be due to operator over-caution using lower laser intensity or avoidance of areas in close proximity to the eye; or perhaps macrophage clearing of laser irradiated periorbital tissue is less effective than other locations.[62]

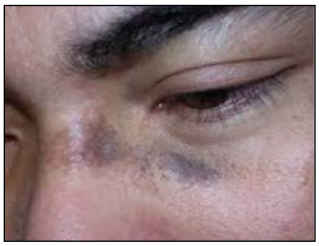

Figure 46. Naevus of Ota. DermNet (https://creativecommons.org/licenses/by-nc-nd/3.0/nz/legalcode)

Figure 47. Panda bear.

34 Periorbital spider angioma: also known as spider nevus or spider telangiectasia, a vascular lesion consisting of a central arteriole body, radiating capillaries like a spider's legs, and surrounding erythema resembling a spider web.[63]

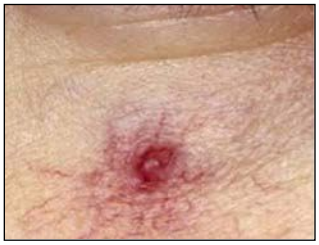

Figure 48. Periorbital spider angioma. DermNet (https://creativecommons.org/licenses/by-nc-nd/3.0/nz/legalcode)

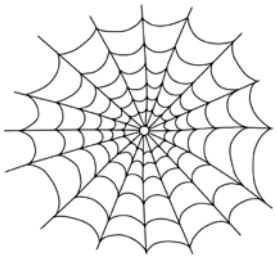

Figure 49: Spider web.

35 Pisciform flecks: yellow fish-shaped[64] lesions which over time atrophy and leave barely perceptible hyperpigmentation in the retinal pigment epithelium characteristic of fundus flavimaculatus.[65]

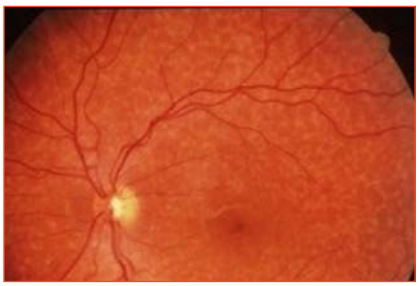

Figure 50. Pisciform flecks in Stargardt's disease. This image was originally published in the Retina Image Bank® website. David Callanan. Fundus Flavimaculata/Stargardt's. Retina Image Bank. 2013; 12774. ©the American Society of Retina Specialists

36 Polar bear tracks: also known as congenital grouped albinotic spots of the retinal pigment epithelium, sharply demarcated chalky lesions occurring in patterns similar to footprints.[66]

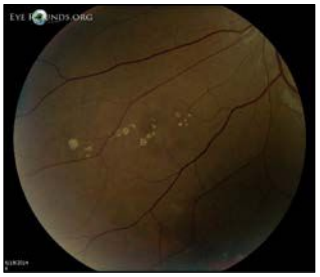

Figure 51. Polar bear tracks. ©EyeRounds.org, The University of Iowa

Figure 52. Polar bear tracks.

37 Pterygium: a triangular-shaped corneal over-growth originating from the interpalpebral bulbar conjunctiva, often with epithelial secretory goblet cells not typically found in corneal epithelial tissue.[67] The term originates from the Greek word pterygion, which is in turn derived from the Greek roots pterón (feather, wing) and ptéryx, ptérigos (wing, fin, bird).[68]

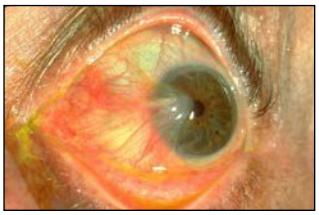

Figure 53. Pterygium. ©2020 American Academy of Ophthalmology

Figure 54. Extended wing of a bird.

38 Racoon eyes: also known as raccoon sign or panda sign,[69] a periorbital hematoma that can be caused by basal skull fracture,[70] particularly if hemorrhaging is absent beyond the tarsal plate.[69,71] In the absence of trauma, it may be associated with neuroblastoma metastasis in and around the orbit,[72,73] or primary amyloidosis from amyloid fibril infiltration into periorbital blood vessels.[74] Anecdotal case reports also attribute etiology to Kaposi's sarcoma,[75] multiple myeloma,[76] and other vascular, infectious, immune-mediated, metabolic, genetic, and malignant etiologies.[69]

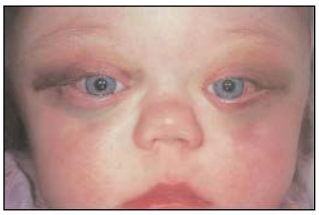

Figure 55. Periorbital hematoma in a 2-year-old with bilateral metastasis of neuroblastoma. ©2020 American Academy of Ophthalmology

Figure 56. Characteristic "black mask" of a raccoon.

39 Retinal operculum: a free-floating flap of detached retinal tissue within the vitreous body.[77] "Operculum" is derived from the Latin term operire, meaning "to cover",[78] and is also the technical term for the gill coverings of many bony fish.[79]

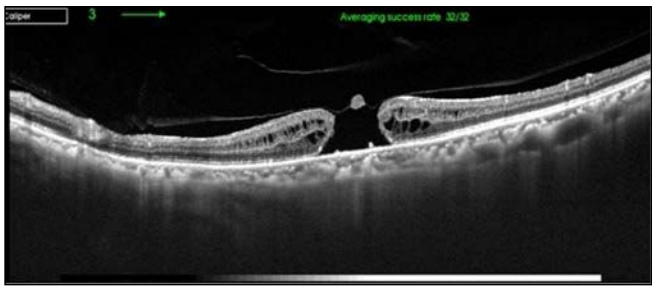

Figure 57. Optical coherence tomography of full thickness macular hole with operculum. This image was originally published in the Retina Image Bank® website. Samarth Mishra. Aditya Birla. Stage 3 Macular Hole With Operculum. Retina Image Bank. 2018; 28613.
©the American Society of Retina Specialists

40 Rhinophyma: Greek for "nose growth"; sebaceous gland hyperplasia in stage IV rosacea.[80]

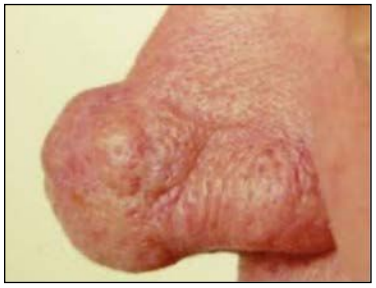

Figure 58. Rhinophyma. DermNet
(https://creativecommons.org/licenses/by-nc-nd/3.0/nz/legalcode)

41 Rodent ulcer: refers to the excavated appearance with elevated pearly margins[81] characteristic of basal cell carcinoma; in Latin, rodere means "to gnaw".[18]

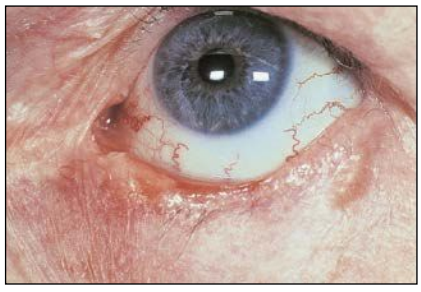

Figure 59. Basal cell carcinoma of the lower eyelid.
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42 Salmon patch:
a. of Hutchinson:82 stromal vascularization and inflammation that appears in syphilitic interstitial keratitis.[83] Most often congenital, although may occur with acquired infection.[83]

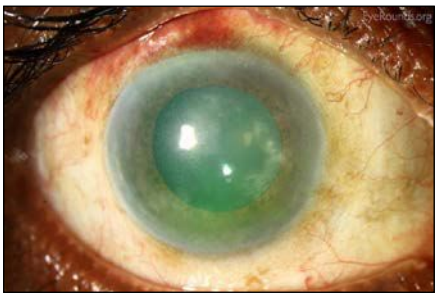


Figure 60. Salmon patch. ©EyeRounds.org, The University of Iowa

b. a painless pink-colored mass on the bulbar conjunctiva or adnexa, it can be associated with lymphoma.[84] Feeder vessels, rapid invasive growth, and ulceration raise suspicion of lymphoma.[84]

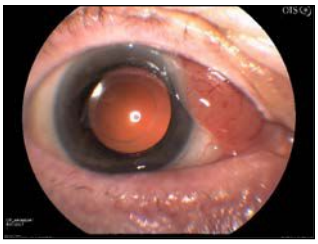


Figure 61. This image was originally published in the Retina Image Bank® website. Nichole Lewis. Salmon Patch Lymphoma. Retina Image Bank. 2017; 27223.
©the American Society of Retina Specialists

Figure 62. Salmon filet.

43 Sea fan neovascularization: similar in appearance to the aquatic invertebrate *Gorgonia flabellum*,[85] retinal neovascularization that proliferates anteriorly from perfused to non-perfused tissue, associated with sickle cell disease.

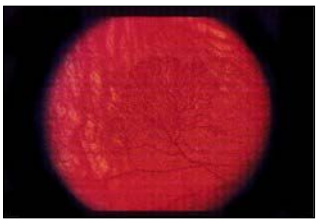


Figure 63. Sea fan neovascularization of the retina. This image was originally published in the Retina Image Bank® website. Maurice Rabb. Macro And Microvascular Sea Fan Changes. Retina Image Bank. 2013; 12289.
©the American Society of Retina Specialists

Figure 64. Sea fan coral.

44 Serpiginous choroiditis: retinal pigment epithelium atrophy relapses and remits leaving behind subretinal areas of swelling that spread equatorially in a creeping, snake like fashion.[86] The etymology of the word serpiginous is derived from the Latin verb sepere meaning to creep or creeping in a similar fashion to a ringworm or snake, hence the root is used in the 13th century English word 'serpent'.[87,88]

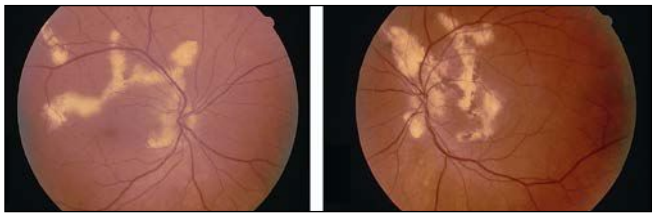


Figure 65. Serpiginous choroiditis.
©2020 American Academy of Ophthalmology

Figure 66. Lateral undulation of a snake.

45 Snail tracks:
a. Posterior polymorphous corneal dystrophy: an asymptomatic dystrophy affecting Descemet's membrane and endothelium that results in horizontal band-like lesions.[89]

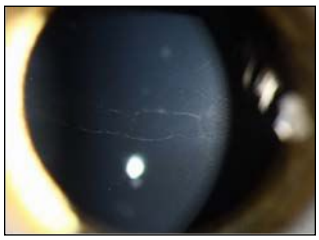


Figure 67. Snail tracks.
©EyeRounds.org, The University of Iowa

Figure 68. A snail track

b. Can be an early stage of retinal lattice degeneration, whereby thinning inner retinal tissue appears opaque or furrowed.[90]

Figure 69. Snail track degeneration of the retina.
©Online Journal of Ophthalmology

Figure 70. A snail track

46 Soft shell technique: a cataract surgery technique using two ophthalmic viscosurgical devices (OVD) within the anterior chamber to create different physical environments improving the success of cataract surgery particularly in difficult cases.[91]

47 Steinert double-ended claw chopper: a surgical instrument whose curvature allows easier engagement with the cataract's nucleus.[92]

48 Tigroid fundus: a striped appearance[93] seen when prominent choroidal pigmentation is interrupted by choroidal vessels, which appear lighter in comparison.[94] Also referred to as tessellated fundus, this physiologic variant is a protective factor in diabetic retinopathy.[94]

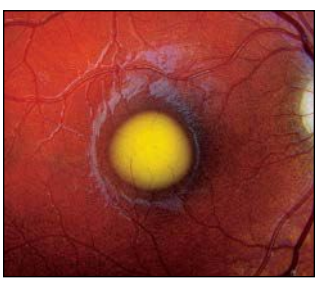
Figure 72. Tiger stripes.

Figure 71. Tigroid fundus with a Drance hemorrhage. This image was originally published in the Retina Image Bank® website. Gregg Kokame. Miscellaneous. Retina Image Bank. 2012; 1354. ©the American Society of Retina Specialists

49 Tri-soft shell technique: a technique used in cataract surgery that specifically uses a dispersive OVD with a viscoadaptive OVD to increase space creation and stability within the anterior chamber.[91]

50 Vermiform movement: refers to the worm-like movement of the iris caused by sectoral constriction of the iris sphincter, most commonly with Adie's tonic pupil.[95] The term 'vermiform' is derived from the Latin word vermis, meaning 'worm'.[96]

51 Vitelliform macular dystrophy: originates from the Latin term vitulus, originally meaning 'calf' and later meaning 'yolk of an egg'.[97] Foveal dome-shaped deposits of lipofuscin and degraded photoreceptor segments that initially appear as an egg cooked "sunny-side up", and later progressing to a "scrambled egg" appearance. Variants of this condition include Best disease and Adult-onset vitelliform macular dystrophy.[98]



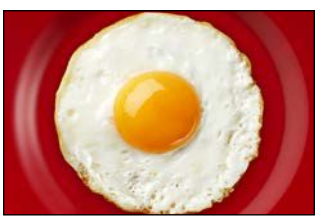


Figure 74. "Sunny-side up" fried egg.

Figure 73. "Egg yolk" stage of Best disease.
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Conclusion

Eponyms can enhance our understanding of medical conditions; however, repetitive eponym use can introduce a degree of confusion for different conditions. Eponymous syndromes that refer to the physician who originally reported the condition can also add a level of complexity: one paper reviewed seventeen eponyms attributed to Sir Jonathan Hutchinson alone.[82] Some of his more notable ophthalmic eponyms include: Hutchinson's disease, Hutchinson's facies, Hutchinson's patch, Hutchinson's pupil, Hutchinson's triad,[82] and Hutchinson's sign.[99]

Whether the eponymous association is obvious or more obscure, there is a diverse array of creatures unknowingly responsible for the names of countless conditions, findings, and instruments in ophthalmology. Without these animals, there would be more eponyms based on scientists' names, providing little to no context about the function, appearance, or etiology of new discoveries. Despite the dimensions of each creature, the animal kingdom plays a sizeable role in expanding the ever-growing glossary of eye-related terms, and it will continue to do so until the cows come home.

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POST-COURSE TEST

Animal Eponyms in Eye Care

1. **Why are zoological based eponyms useful in ophthalmic care?**

- ☐ Allow conditions to become more memorable
- ☐ Help clinicians down the correct systemic workup path
- ☐ Help clinicians become familiar with diseases and their manifestations
- ☐ All of the above

2. **What eponym is commonly used to describe an autoimmune posterior uveitis characterized by multiple lightly colored oval chorioretinal lesions with a juxtopapillary predilection?**

- ☐ Birdshot Chorioretinopathy
- ☐ Bear Tracks
- ☐ Bag of worms
- ☐ Butterfly rash

3. **What eponym is commonly used to describe fluffy, white lesions caused by accumulated axoplasmic debris within the ganglion cell axon bundle due ischemia?**

- ☐ Chicken eyes
- ☐ Crab Claws
- ☐ Cattle Track sign
- ☐ Cotton Wool Spots

4. **What definition is used to describe the eponym 'Mutton-fat keratic precipitates'?**

- ☐ Round, centrally umbilicated lesions found on the skin and mucous membranes caused by a poxvirus
- ☐ Ocular irritation leads to conjunctival mucus production which prompts the patient to mechanically remove "fish" mucus
- ☐ Leukocytes that appear on the posterior cornea as 'greasy' large fat globules, similar to the fat of sheep.
- ☐ Pigmentation that persists after treating periorbital nevus of Ota lesion with laser surgery

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5. **What eponym is commonly used to describe a triangular-shaped growth originating from the interpalpebral bulbar conjunctiva?**

- ☐ Polar bear tracks
- ☐ Pterygium
- ☐ Pisciform
- ☐ Periorbital spider angioma

6. **What eponym is commonly used to describe a periorbital hematoma that can be caused by basal skull fracture?**

- ☐ Raccoon eyes; also known as raccoon sign or panda sign
- ☐ Salmon patch
- ☐ Tri-soft shell technique
- ☐ Steinert double-ended claw chopper

7. **What definition is used to describe the eponym 'rodent ulcer'?**

- ☐ benign swelling and reddening of the nose in stage IV rosacea
- ☐ A free-floating flap of detached retinal tissue
- ☐ Refers to basal cell carcinoma
- ☐ Retinal neovascularization that proliferates anteriorly from perfused to non-perfused tissue, associated with sickle cell disease

8. **What definition is used to describe the eponym 'serpiginous choroiditis'?**

- ☐ The accumulation of epithelial cells that are clinically visible as "pearls" in posterior capsular opacification
- ☐ A striped appearance seen when prominent choroidal pigmentation is interrupted by choroidal vessels, which appear lighter in comparison
- ☐ Also known as giraffe spots, a chorioretinopathy of characteristic hypo- and hyperpigmented spots viewed by funduscopy and with angiography
- ☐ Retinal pigment epithelium atrophy relapses and remits leaving behind subretinal areas of swelling that spread equatorially in a creeping, snake like fashion

9. **What eponym is commonly used to describe foveal dome-shaped deposits of lipofuscin and degraded photoreceptor segments that initially appear as an egg cooked "sunny-side up", and later progressing to a "scrambled egg" appearance?**

- ☐ Vermiform movement
- ☐ Vitelliform macular dystrophy
- ☐ Snail tracks
- ☐ Periorbital spider angioma

10. **Why do animal eponyms provide better understanding to conditions as opposed to using physicians who originally report the condition?**

- ☐ They provide context about the function, appearance or etiology of the condition
- ☐ Non-repetitive use in ophthalmic context
- ☐ Cannot be considered derogatory
- ☐ None of the above