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Epiretinal Membrane Exacerbated by Vitreomacular Traction and Anomalous Posterior Vitreous Detachment: a Teaching Case Report

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Background

Vitreomacular disorders (VMDs) represent a spectrum of vitreomacular interface abnormalities, which includes epiretinal membrane (ERM), vitreomacular traction (VMT) and macular hole (MH). Each VMD encompasses a wide range of severity, from asymptomatic to vision-threatening. The introduction of optical coherence tomography (OCT) has allowed diagnosis of VMDs at their earliest and asymptomatic stages. However, inconsistencies with management exist because there are no reliable prognostic factors to predict which patients with a VMD will need surgery and no consensus on when to refer for surgery. This teaching case report explores a patient with ERM, VMT and anomalous posterior vitreous detachment (PVD) that progressed after more than 20 years of observation. It focuses on the disease process and medical management of ERM and VMT. It is appropriate for practicing clinicians at all levels, from third- and fourth-year optometry students to residents to seasoned professionals.

Case Description

An 89-year-old Caucasian male presented for his annual eye exam with gradually decreasing vision in the left eye in the past year. He denied experiencing photopsia, floaters, trauma or ocular pain or discomfort. He had ERM in both eyes, thicker in the left eye (OS), which had been monitored annually and deemed stable by various providers since a baseline visit 20 years ago (Table 1).

The patient was previously symptomatic for binocular diplopia due to an alternating intermittent exotropia. The diplopia was relieved with 5 diopters of base-in prism. He had undergone cataract surgery in both eyes (OU) 4 years prior to this visit. He had no ocular medications. His medical history was significant for hypertension, benign prostatic hyperplasia, myelodysplastic syndrome, adrenal insufficiency, chronic inflammatory demyelinating polyradiculoneuropathy-monoclonal gammopathy of uncertain significance and gastroesophageal reflux disease. None of these conditions was contributory to his ocular history. His medications were consistent for these conditions. Most notable among them were darbepoetin, hydrocortisone and intravenous immune globulin. He had no significant family ocular or medical history.

Since his routine cataract surgery, the patient’s visual acuity (VA) in the right eye (OD) fluctuated between 20/20 and 20/25. Meanwhile, VA OS had decreased by approximately a line per year, from 20/25 to 20/40. By this visit, his VAs were 20/25 OD and 20/60 OS. The patient reported metamorphopsia that was more prominent OS than OD and denied reports of a scotoma. His pupils were both equal, round and reactive to light in absence of an afferent pupillary defect. His extraocular motility was unrestricted in all gazes. He had full confrontation visual fields OU. The anterior segment exam was unremarkable OU. The dilated fundus exam was significant for PVD OU and a glistening opaque membrane over the macula with retinal striations radiating toward the temporal arcades and optic nerve OS > OD.

OCT macula scan revealed a thick hyper-reflective band with surface retinal wrinkling, confirming the presence of tractional ERM OU. OCT macula scan OD was stable in central macular thickness and appearance compared to previous scans. Of note with the OCT macula scan OS, an area of focal VMT appeared along the inferotemporal arcade. The nasal portion of the vitreoretinal traction appeared to break off just superior to the area of traction (Figure 1). A broad area of vitreoretinal traction temporal to the macula was pulling the temporal portion of the ERM. This portion of the ERM appeared to break off, along with the vitreoretinal traction, just inferior to the superotemporal arcade (Figure 2). In addition, central macular thickness had increased, and an area of retinoschisis arose temporal to the optic nerve, along with a new drusenoid pigment epithelial detachment (PED) temporal to the fovea (Figure 3). OCT
angiography ruled out signs of choroidal neovascular membrane. These unexpected findings prompted a review of all previous OCT macula scans OS, which revealed that the areas of vitreoretinal traction had been present for the past 10 years and the breakage points were first observed 5 years ago. A significant increase in central macular thickness had begun 3 years prior to this visit, while the development of retinoschisis temporal to the optic nerve appeared 1 year prior to this visit.

At this visit, the patient was diagnosed with a tractional ERM complicated by broad VMT and anomalous PVD OS. After consulting with a retina specialist about the retinal changes, observation was recommended. The patient missed his 1-month follow-up but presented 6 months later. At this visit, his VAs were stable and retinoschisis had decreased in size, but central macular thickness and the size of the PED continued to increase. At this point, vitrectomy and membrane peel was recommended. Within a month after surgery, retinoschisis had completely resolved (Figure 4). At a year after surgery, his VA improved by two lines (from 20/60 to 20/40) and he noticed less metamorphopsia. The central macular thickness returned to baseline, but the PED remained stable (Figure 5).

**Figure 1.** OCT macula scan OS in 2020 that prompted review of previous OCT scans. (a) An area of focal vitreoretinal traction (arrow) along the inferotemporal arcade. (b-d) The nasal portion of the vitreoretinal traction appears to break off (arrow) just superior to the area of traction. [Click to enlarge]

**Figure 2.** OCT macula scan OS in 2020 that prompted review of previous OCT scans. (a-c) A broad area of vitreoretinal traction (arrow) along the temporal portion of the epiretinal membrane. (d) This temporal portion, along with the vitreoretinal traction, appears to break off (arrow) just inferior to the superotemporal arcade. (a) Presence of retinoschisis temporal to the optic nerve (star) and a large drusenoid pigment epithelial detachment (triangle) temporal to the fovea. [Click to enlarge]

**Figure 3.** OCT macula scan OS in 2020 that prompted review of previous OCT scans. Blunted foveal contour with the epiretinal membrane across its surface and vitreomacular traction (arrow) at its temporal portion. This scan provides more detail of the retinoschisis (star) temporal to the optic nerve and the drusenoid pigment epithelial detachment (triangle) temporal to the fovea. The area of vitreoretinal traction is within the 3-mm radius of the fovea and measures 5,007 μm. [Click to enlarge]

**Figure 4.** OCT macula scan OS 1 month after vitrectomy shows resolution of vitreoretinal traction and retinoschisis. The drusenoid pigment epithelial detachment (triangle) temporal to the fovea was still present. [Click to enlarge]

**Figure 5.** OCT macula scan OS 1 year after vitrectomy shows continued resolution of vitreoretinal traction and retinoschisis. The drusenoid pigment epithelial detachment (triangle) temporal to the fovea was still present. [Click to enlarge]

**Education Guidelines**

In a didactic setting, students and residents can be presented with this case and OCT images in slideshow format to develop their own primary diagnosis, differential diagnosis and treatment and management plan. Then, the discussion questions can be used to explore a deeper understanding of the vitreous, VMDs and treatment and management considerations. The learning objectives can be assessed by presenting OCT scans of ERMs and VMT at different stages and having the students and residents classify the conditions and develop a management plan based on a hypothetical patient’s symptoms, pertinent clinical findings and OCT scans.

**Learning objectives**

1. Understand the interaction between the vitreous and retina
2. Know the natural PVD process
3. Recognize an anomalous PVD and its resultant VMDs
4. Classify ERMs and VMT syndrome
5. Increase awareness about when to refer patients with ERM/VMT for surgical options
Key concepts

1. The complications that arise from an abnormal interaction between the vitreous and retina
2. The exacerbating inter-relationships between VMDs
3. The significance of OCT in identifying the disease course and aiding management of VMDs
4. The signs and symptoms that may identify a patient with ERM and/or VMT syndrome as a candidate for surgery

Discussion questions

1. What are the components of the vitreoretinal interface?
2. What are the processes of synchysis and syneresis?
3. Which disorders are considered part of the VMD spectrum?
4. How do you classify an anomalous PVD, ERM and VMT?
5. What is the difference between vitreomacular adhesion (VMA) and VMT?
6. What proposed role does anomalous PVD play in ERM and VMT pathogenesis?
7. How does the presence of an ERM exacerbate VMT?
8. When would you observe a patient with an ERM or VMT?
9. When would you refer a patient with an ERM or VMT for surgery?

Literature Review

The vitreous and vitreoretinal interface

The vitreous body is a highly hydrated extracellular matrix within the posterior chamber of the eye. With a high water content of 98-99%, its transparency allows for uninterrupted transmission and refraction of light onto the retina. The interaction between its two principal macromolecules, hyaluronic acid and collagen fibrils, produces its recognized viscoelastic property. The rope-like collagen fibrils are composed of three different collagen types: a core of collagen Type II surrounded by collagen Type IX and a hybrid of types V/XI. The chondroitin sulfate glycosaminoglycan chains of collagen Type IX act like rungs of a ladder to bridge the collagen fibrils together and form an extended structure. The space between the collagen fibrils is filled and maintained by a network of hydrophilic hyaluronic acid. As it attracts water, it swells and "inflates" the collagen scaffold, producing the vitreous' gelatinous consistency. Interestingly, when the collagen fibrils are separated by at least one wavelength of incident light, light-scattering is minimized, which further contributes to the vitreous' transparency and ability to transmit light.

An outer cortex composed of tightly packed collagen fibrils surrounds the vitreous body and is 100-300 µm thick. The posterior vitreous cortex is a lamellar structure that contains a single layer of mononuclear phagocytes, called hyalocytes, approximately 50 µm from the inner limiting membrane (ILM). Studies have shown that hyalocytes are important in stimulating cell proliferation, inducing tangential vitreoretinal contraction, and stimulating collagen gel contraction in response to platelet-derived growth factor and other cytokines. The posterior vitreous cortex runs parallel to the retinal surface and superficially inserts into the ILM, creating the vitreoretinal interface. Adhesion molecules such as fibronectin, laminin and heparan sulfate keep these collagen fibers attached to the retinal surface.

The natural posterior vitreous detachment process

PVD is defined as the complete separation of the posterior vitreous cortex from the retina in all areas posterior to the vitreous base. A natural, innocuous PVD results when two processes work in tandem: vitreous liquefaction (synchysis) and vitreoretinal dehiscence with collapse (syneresis). Synchysis is characterized by the dissolution of the hyaluronic acid-collagen complex. Over time, there is a loss of the limiting Type IX and its chondroitin sulfate chains from the collagen fibrils’ surface, exposing the core of “sticky” collagen Type II. Without chondroitin sulfate chains to space the fibrils apart, the now “sticky” fibrils coalesce and displace the hyaluronic acid that once filled this space. The hyaluronic acid pools water into areas adjacent to the coalesced fibrils, creating pockets of liquified vitreous called lacunae. The vitreous is approximately 12.5% liquid by age 18 and increases to 50-62% liquid by age 80-90.

Concurrent with vitreous liquefaction is vitreoretinal adhesion weakening. The posterior vitreous cortex has strong attachments (from strong to strongest) at the major blood vessels, macula, optic disc and vitreous base. So, the natural detachment course of the posterior vitreous cortex from the retina starts at the major blood vessels, followed by the macula, and finally the optic disc. At the completion of detachment, the posterior vitreous cortex remains attached only to the vitreous base. Though it is unknown what changes occur at the interface to weaken vitreoretinal adhesion, it is hypothesized that the collagen fibrils from the posterior cortex that insert into the ILM slowly degenerate with advanced age.
The liquified vitreous travels through fissures and holes of the posterior cortex and into the retrocortical space.\textsuperscript{1,13} As the volume of liquified vitreous in the retrocortical space increases, it displaces the gelatinous vitreous body from its weakened attachment at the ILM, resulting in syneresis. Evidence from autopsy studies has indicated a 51\% prevalence of PVD in the seventh decade, which further increases to 63\% in the eighth decade.\textsuperscript{2,16} It has been suggested that PVD evolution may occur faster in women older than 60 due to the effect of declining estrogen levels on vitreous collagen in post-menopausal women.\textsuperscript{1}

\textbf{Anomalous posterior vitreous detachment and vitreomacular disorders}

If the liquified vitreous in the retrocortical space displaces the gelatinous vitreous body before vitreoretinal adhesion has sufficiently weakened, a partial PVD with focal and persistent vitreoretinal attachment(s) emerges as a condition termed anomalous PVD.\textsuperscript{4} The sequelae of anomalous PVD depend on the position of the strongest vitreoretinal attachment and greatest liquefaction of the gel.\textsuperscript{7} When this persistent traction occurs at the macula, VMDs transpire, which include vitreoschisis, ERM, VMT, MH, lamellar hole and myopic foveoschisis.\textsuperscript{1} These disorders are characterized by a distortion of macular architecture, such as intraretinal pseudocyst formation, intraretinal schisis, elevation of fovea from retinal pigment epithelium (RPE) and foveal detachment.\textsuperscript{1} They are asymptomatic until the traction is strong enough to distort the outer segments, elevate the fovea from the photoreceptors and/or disturb the spatial arrangement of cones,\textsuperscript{7,14} manifesting into symptoms of reduced central vision, metamorphopsia, micropsia and/or macropsia.\textsuperscript{1}

Anomalous PVD can be classified as either full-thickness or partial-thickness.\textsuperscript{10,15} A full-thickness anomalous PVD occurs when the entire posterior vitreous cortex remains attached to the retina.\textsuperscript{10,17} A partial-thickness anomalous PVD, termed vitreoschisis, occurs when the posterior vitreous cortex lamellae split; the outermost layer of the split remains adherent to the retina while the innermost layer collapses forward with the vitreous body.\textsuperscript{7,10,15} Residual vitreous tissue has been found to be left on the inner retinal surface in nearly 50\% of PVDs.\textsuperscript{14,16}

\textbf{Epidemiology and risk factors}

Since the introduction of OCT in 1993, the ability to visualize and evaluate the vitreoretinal interface has deepened the understanding of its associated disorders. VMDs are underdiagnosed due to their typically asymptomatic nature and subtle clinical appearance. In a study of participants older than 60 with maculae deemed normal by color fundus photography or biomicroscopy, 8.4\% of eyes were found to have a VMD by OCT.\textsuperscript{17} The most common abnormality found was ERM (61 out of 984 eyes).\textsuperscript{17}

\textbf{Epiretinal membrane}

The prevalence of ERM varies across studies, which can be attributed to differences in methods and protocols for grading and how to define ERM.\textsuperscript{18} The prevalence of ERM appears to vary by race, from 2.2\% in a Beijing study in rural China up to 28.9\% among Latinos in a multi-ethnic study conducted in the United States.\textsuperscript{19} However, the role of race and ethnicity remains unclear because the variations are inconsistent across studies. For example, in the United States, Multi-Ethnic Study of Atherosclerosis (MESA) data suggest that the prevalence of any ERM was highest in persons of Chinese ancestry (39.0\%), whereas the data from China suggest ERM prevalence rates were much lower (2.2\% - 3.4\%).\textsuperscript{20}

The original Beaver Dam Eye Study used nonstereoscopic color fundus photographs and found ERM in 11.8\% of the population in at least one eye and in 2.4\% of the population in both eyes.\textsuperscript{21} In the 20-year follow-up study using OCT, a higher prevalence was documented: 34.1\% in at least one eye and 30.3\% in both eyes.\textsuperscript{22} The reported prevalence of early asymptomatic ERM, termed cellophane maculopathy, has varied from 1.8\% and 2.2\% in China to as high as 25.1\% in MESA.\textsuperscript{20} The prevalence of the more severe form of ERM, commonly termed macular pucker, has been more consistent across studies with rates ranging from 0.7\% in rural China to 3.5\% among Asian Indians, 3.8\% in MESA, and 3.9\% in Melbourne, Australia.\textsuperscript{20}

Studies have consistently identified age and PVD as risk factors for ERM. It is estimated that the prevalence of macular ERM is 2\% in patients younger than 60 years, 12\% in patients age 70 or more, and 22.5\% in those age 80 or more.\textsuperscript{7,22,24} In the Beaver Dam Eye Study, the prevalence of ERM increased with age from 28.1\% in those age 63-74 years to 53.2\% in those age 85 years or more.\textsuperscript{22} PVD has been found to be present in 70\% of patients in the earlier stages of ERM\textsuperscript{15,23} and in up to 90\% of patient with advanced ERM.\textsuperscript{24} Other retinal pathologies that have been identified as risk factors for ERM are uveitis, retinal breaks, retinal vein occlusion, proliferative diabetic retinopathy and ocular inflammatory disease.\textsuperscript{26} Cataract surgery has also been identified as a risk factor.\textsuperscript{15,21}

Our patient’s retinal changes and subsequent decreased vision developed approximately a year after cataract surgery, which points to the procedure as the agent for his ERM progression. Even though he had cataract surgery in each eye 1 month apart, the ERM progressed in only the left eye, which was most likely due to the multiple areas of anteroposterior vitreoretinal traction in that eye when none existed in the right eye.
Vitreomacular traction syndrome

Few studies have specifically addressed the epidemiology of idiopathic VMT as it tends to overlap with other retinal diseases, especially MH.\(^5^,\)\(^7^\) The prevalence of isolated idiopathic VMT has been estimated as 22.5 cases per 100,000 of the general population, with an incidence of 0.6/100,000 persons per year.\(^5^,\)\(^2^7^\) In a multi-center prospective study involving 1,950 eyes, VMA and VMT were detected in 38.77% and 1.07% of eyes, respectively.\(^2^8^\) The Beaver Dam Eye Study reported a prevalence of 26% and 1.6%, respectively, for VMA and VMT.\(^2^2^,\)\(^2^8^\)

As it is for ERM, age is the strongest risk factor for VMT. In the Beaver Dam Eye Study, the prevalence of VMT increased from 1% in those age 63-74 years to 5.5% in those age 85 years or more.\(^2^2^\)

**Classification, clinical presentation and symptoms**

Epiretinal membrane

Unlike other VMDs, ERM does not have an OCT-based classification that has reached widespread consensus or mainstream use.\(^1^\) In general, ERM can be classified based on etiology [primary (idiopathic) or secondary] or histology [simple or complex (tractional or contractile)].

Primary or idiopathic ERMs are associated with vitreoretinal traction. Secondary ERMs are associated with retinal pathologies such as retinal breaks, tears or detachments, intraocular inflammation, trauma, retinal vascular diseases and retinal surgery. Interestingly, these pathologies are notable risk factors for ERM development.\(^7^,\)\(^2^0^\) Idiopathic ERM represents approximately 60% of patients with ERM.\(^2^4^\)

Simple ERM grows directly on the ILM and is composed of a monolayer of retinal cells.\(^7^\) It appears as a thin, glistening membrane on the surface of the retina, termed cellophane maculopathy. It is noncontractile with mild to no vision symptoms.\(^7^,\)\(^2^7^\) Complex or tractional ERM is a multi-layer of cells (fibrous astrocytes, myofibroblasts, hyalocytes, macrophages, RPE and glial cells) that is separated from the ILM by a layer of vitreal collagen Type II.\(^7^\) It appears as a thick, opaque membrane that has been proposed to exert a progressive, tangential, inward (centripetal) traction that can pull the underlying retina inward resulting in macular pucker.\(^7^,\)\(^2^0^\) In some cases, this centripetal contraction can lead to the biomicroscopic slit lamp appearance of a hole, historically termed pseudohole. Unlike a true MH, a pseudohole has an intact photoreceptor layer.\(^1^\) If the traction becomes excessive, macular pucker will cause retinal changes, such as irregular wrinkling, nerve fiber layer dragging, ectopic fovea, winding corkscrew vessels surrounding the overlying ERM, or major vessel straightening and crowding.\(^7^,\)\(^2^0^\) It may even cause vitreoretinal traction and/or tractional retinal detachment.\(^2^3^\)

In early stages, ERM is often asymptomatic and detectable only on OCT scans.\(^1^\) Among patients with idiopathic ERM, two-thirds exhibit VA of 20/30 or better, while 85% display VA of 20/70 or better.\(^2^6^\) ERM becomes symptomatic when the traction involves the macula or perimacular regions.\(^7^,\)\(^2^3^,\)\(^2^4^\)

Symptoms of ERM include reduced VA, metamorphopsia, micropsia, macropsia, aniseikonia and/or dragged-fovea diplopia.\(^7^,\)\(^2^3^,\)\(^2^4^,\)\(^3^0^\) In cases where ERM is more severe in one eye, the difference in metamorphopsia, micropsia or macropsia between the eyes can lead to binocular image rivalry.\(^1^\) A patient who has reduced binocular visual quality may close one eye, even in the absence of diplopia or strabismus.\(^3^0^\) With our patient’s history of horizontal diplopia, it was important to perform a full binocular workup to determine proper treatment. The binocular workup for our patient revealed an alternating intermittent exotropia. Therefore, prism correction was the appropriate treatment to relieve his diplopia. If the binocular workup had revealed no strabismus, his symptom of diplopia would have been most likely due to binocular rivalry. If this were the case, a surgical referral for vitreoretinal traction relief would have been most appropriate.

Vitreomacular traction

In 2013, the International Vitreomacular Traction Study (IVTS) proposed OCT-based definitions for VMA, VMT, full-thickness MH, lamellar hole and macular pseudohole, along with an OCT-based classification system for VMA, VMT and MH.\(^1^4^\) Advantages include clinical applicability for the assessment of vitreous state and reproducibility for comparative analysis in clinical studies.\(^1^5^\) Most notably, the classification system provides evidenced-based quantification of the pathology that correlates to treatment outcomes for MH.\(^1^5^\)

VMA is defined as a perifoveal vitreous detachment with macular attachment within a 3-mm radius of the fovea and without associated retinal deformation.\(^1^4^,\)\(^3^3^\) VMA is considered a normal stage in early PVD development. Patients with VMA are usually asymptomatic and detected incidentally.\(^1^\) In contrast, VMT is defined as perifoveal vitreous detachment with macular attachment within a 3-mm radius of the fovea that does exhibit retinal deformation.\(^1^,\)\(^1^4^,\)\(^3^3^\)
Based on the IVTS classification system, VMT is subclassified as focal (≤ 1,500 µm) or broad (> 1,500 µm). This distinction has clinical relevance because the diameter of VMA is inversely related to macular morbidity and foveal deformation. The narrower the area and the higher the angle of adhesion, the greater amount of tractional force exerted and the greater the foveal deformation. Focal VMT is associated with distorted foveal surface, foveal pseudocysts and foveal elevation, and it may lead to MH development. Foveal pseudocysts are associated with diminished VA and vision distortion, such as metamorphopsia. After the release of traction, pseudocysts generally resolve over time with little remaining vision deficit. A broader area of VMA may distribute the tractional force beyond the border of the foveal region, leading to generalized thickening of the macula, intraretinal schisis and associated ERM.

Anteroposterior traction from VMT normally creates a V-shape, with its “point” at the macula and traction on both sides. However, in this case, there was a single area of anteroposterior traction approximately 2,370 µm temporal to the fovea creating a J-shape (Figure 3). This temporal band continued superiorly before breaking off just before the superotemporal arcade with the posterior cortex as a continuous, yet separate layer above the retina. There was another focal area of traction along the inferotemporal arcade, where the nasal portion broke off just superior to this area, leaving a sole area of temporal traction. Because this area of persistent vitreoretinal traction occurred beyond a 3-mm radius of the fovea, this was considered areas of anomalous PVD instead of VMT.

Discussion

The inter-relationship of vitreomacular disorders

While each VMD has its own pathogenesis and disease course, they are often inter-related with anomalous PVD at their incipience.

Vitreoschisis and epiretinal membrane

The effect that vitreoschisis (partial-thickness anomalous PVD) has on the retina depends on the location of the vitreous cortical split in relation to the hyalocyte layer. If the split occurs posterior to the level of hyalocytes, the hyalocytes, as part of the anterior split vitreous cortex, will detach from the retina, leaving behind a relatively thin hypocellular layer of vitreous cortex attached to the macula. If this membrane is also attached at the optic disc, it may cause an outward (centrifugal) tangential contraction, inducing a MH or schitic changes as seen in lamellar holes. Studies conducted at the VMR Institute using OCT combined with scanning laser ophthalmoscopy (SLO) have identified vitreoschisis in 53% of patients with MH and in 43% of patients with macular pucker.

While the exact mechanism of ERM development is still poorly understood, two observations have emerged. First, PVD is characterized as a growth of fibrocellular tissue on the ILM. The type of PVD that has been proposed to induce fibrocellular proliferation is a vitreoschisis with a split anterior to the level of hyalocytes, which leaves the hyalocytes as part the vitreous cortex layer that remains attached to macula. Gandonfer et al. have shown histologically that a thin layer of cortical vitreous is often sandwiched between the fibrocellular ERM and the ILM, confirming the role of residual vitreous material in ERM formation. As mononuclear phagocytes of the reticulo-endothelial cell system, hyalocytes can stimulate the migration of monocytes from the circulation and glial cells from the retina. The proliferation of these cells onto the surface of the retina creates a relatively thick, hypercellular and contractile scaffold that allows uptake of other cells into the membrane. The progression of ERM from cellophane maculopathy to macular pucker is considered a fibrotic process that is sustained by collagen deposition and transdifferentiation of retinal Müller cells, RPE cells and hyalocytes into myofibroblasts. In short, hyalocytes lying on the macular surface proliferate, recruit and stimulate glial cells to proliferate upon an intact ILM. This forms an ERM, a scaffold that allows the uptake of other cells into the membrane and induces contraction.

Full-thickness anomalous posterior vitreous detachment and vitreomacular traction

While vitreoschisis (partial-thickness anomalous PVD) can lead to ERM, a full-thickness anomalous PVD can exert enough persistent anteroposterior traction to produce VMT. The deformation may be evident by changes in foveal contour, distortion of the macular architecture, intraretinal pseudocyst formation, intraretinal schisis or even elevation of the fovea from the RPE, without full-thickness defect of all the retinal layers. When VMT creates outer retinal changes, patients are often symptomatic for reduced vision or metamorphopsia. Progressive and excessive traction at the fovea can result in complete interruption of all neural layers, leading to a MH.

Epiretinal membrane and vitreomacular traction
Since the proposed pathogenesis of both ERM and VMT involves anomalous PVD, it is of little surprise that they often co-exist. In a study of 60 eyes with ERM, a partial PVD with VMA occurred in 57% of the eyes. However, this study was conducted in 1999, before the IVTS Group proposed OCT-based definitions that differentiated VMA (normal PVD stage) from VMT (abnormal consequence). In studies that utilized IVTS Group classification and definitions to study the natural history of VMT, ERM was found in 20-37.5% of eyes with VMT.

Several studies have demonstrated that in eyes with VMT, ERM proliferates onto both the retinal surface and onto the back surface of the detached posterior hyaloid membrane. This reinforces the VMA, preventing its separation and prolonging the tractional stress on the macula. The combined forces of tangential contracture of the ERM and anteroposterior traction of the vitreoretinal attachment magnify any tractional stress on the underlying foveal structure. This sustained vitreoretinal traction limits treatment options for patients with combined ERM and VMT.

**Treatment and management**

The goal for VMD management is to optimize vision and quality of life by preventing vision loss and minimizing metamorphopsia and/or diplopia. As a spectrum, VMDs are either observed or undergo pars plana vitrectomy. A patient with VMT has the additional options of pharmacologic or pneumatic vitreolysis.

As previously noted, the presence of ERM reinforces the adhesion between VMT and the retina, contributing to the low success rate of pharmacological vitreolysis in these patients. In the MIVI-TRUST trial, among the subjects receiving ocriplasmin, 8.7% and 37.4% had resolution of “symptomatic VMA” with and without ERM, respectively. Among patients in the placebo group, 1.5% and 14.3% had resolution of “symptomatic VMA” with and without ERM, respectively. Given the intensifying adhesion between the ERM, VMT and anomalous PVD, our patient would have been considered a poor candidate for vitreolysis. For this reason, this teaching case report focuses on whether a patient with ERM and/or VMT should be observed or referred for vitrectomy.

**Observation**

The current standard of care for early stage, asymptomatic ERM and VMT is observation. The risk to this conservative approach includes MH formation and further vision loss. While spontaneous separation of ERM is uncommon, most ERMs tend to remain stable without need for surgical intervention. Using fundus photography, the Blue Mountains Eye Study found that of patients with ERM, 39% were stable, 26% regressed and 29% progressed over a 5-year period. In a retrospective case series exploring the natural history of idiopathic ERM in 145 eyes over a 10-year period, 14.5% underwent surgical intervention with a mean time to surgery of 6.5 years.

While VMT holds a higher risk for MH formation than ERM, observation is considered a viable option for VMT. Before the commercial use of OCT, the spontaneous resolution of VMT was considered uncommon. Using biomicroscopy to study the natural history of VMT over a median follow-up of 5 years, Hichiki et al. found that the spontaneous resolution of VMT occurred in 11% of eyes at a median duration of 15 months. However, in more recent studies using OCT, the incidence of spontaneous resolution has been 20-43% at a median duration of at least 9 months. Dimopolous et al. studied the natural history of 46 eyes with VMT less than 1,500 µm and without ERM to match the baseline characteristics of an ideal candidate for ocriplasmin. They found that 43.5% of these eyes developed spontaneous resolution of the VMT with a mean duration of 375 days. In a retrospective cohort study of 183 eyes with VMT over an average of 17 months, 60% were stable and 20% resolved with a mean time to resolution of 15 months. In the same study, 12% developed MH and 8% elected to proceed with surgery for worsening symptoms, on average, by 19 and 13 months, respectively.

At this time, no factors have been found to reliably predict which patients will experience spontaneous resolution of VMT and which patients will develop a MH. A few studies have suggested that the following factors may be predictive of spontaneous VMT release: adhesion diameter less than 400 µm, a wide angle (approaching 90°) between the vitreous surface, isolated inner retinal layer distortion, treatment of concurrent retinal diseases with intravitreal injections and VMA surface area less than 101,002 µm as calculated by OCT.

**General recommendations during the observation period**

OCT has been key in identifying the disease course and guiding management of VMD. For cases that require finer detail, such as to differentiate ERM from either a vitreoschisis or a shallow PVD, B-scan ultrasonography and OCT/SLO imaging can be used. While advancement in imaging technology has improved the quality of diagnosis, utility for identifying which patients will regress, remain stable or progress is still lacking. A standard of care for asymptomatic patients with ERM and VMT is observation for 3 months before initiating any treatment to avoid unnecessary surgery. Shorter follow-up intervals with detailed OCT macula scans are recommended for patients with focal VMT or history of MH in the fellow eye. In between
follow-up visits, patients should be educated on the signs and symptoms of progression and advised to perform periodic at-home monocular Amsler grid testing.

Vitrectomy

Vitrectomy with membrane peel is the mainstay treatment for patients with symptomatic ERM and VMT.\textsuperscript{1} Vitrectomy involves removing the vitreous body and mechanically separating the vitreous from the optic disc by inducing a PVD and releasing the traction of the vitreous from its attachment at the fovea.\textsuperscript{1} This facilitates a restoration of the central and outer retinal architecture, resulting in improved neural transmission.\textsuperscript{27} Additional steps include removal of any concurrent ERM and/or peeling the ILM.\textsuperscript{1} Many studies have suggested that ILM peeling is significant for preventing ERM recurrence.\textsuperscript{7,39,51} Common complications of vitrectomy are accelerated cataract progression, ERM development and retinal detachment.\textsuperscript{45,46} Rare adverse events include infection, hemorrhage and MH.\textsuperscript{45}

Indications for vitrectomy

Determining when to refer our patient for vitrectomy was the most ambiguous aspect of this case for two reasons. One, there are no preoperative characteristics that are reliably predictive for patients who would benefit from surgery. Two, it is unclear at what point the damage from either ERM or VMT onto the macula is irreversible. The lack of evidence-based guidelines creates uncertainty about when a referral is considered too early. Do we refer as soon as possible to minimize vision loss, or will surgery be deemed unnecessary? With no consensus on when or which patients with ERM or VMT would benefit from a vitrectomy, most surgical referrals are dictated by progressing symptoms.

Vitrectomy is often indicated for patients who experience a decrease in VA, metamorphopsia, double vision or difficulty using their eyes together.\textsuperscript{1,20,24} A retrospective study reported that 21% of patients required surgery at 4 years if baseline VA was $\geq$ 20/40.\textsuperscript{47} However, there is variance on the level of vision impairment required for intervention, and some patients may be very symptomatic with metamorphopsia despite good Snellen VA.\textsuperscript{1,52} In such cases, the goal of surgery is to reduce metamorphopsia and improve binocularity, thereby improving quality of life.\textsuperscript{52}

Poor prognostic factors for ERM are inner nuclear layer cysts and an associate lamellar hole at baseline.\textsuperscript{1} Metamorphopsia is another poor prognostic factor as it is likely the result of rearranged photoreceptors.\textsuperscript{53} Luu et al. found that ERM with greater central macular thickness and disruption of outer retinal layers were more likely to undergo surgery.\textsuperscript{47} Kakehashi et al. identified ERMs with partial PVD without shrinkage and with VMA as having the worst visual prognosis compared to other types of ERMs, possibly due to the chronic weak vitreous traction exerted on the macula. The presence of ERM and broad-based VMA are associated with persistent VMT. As these are less likely to spontaneously resolve, vitrectomy would be the remaining option to relieve traction.\textsuperscript{5,27} Ultimately, our patient’s decreasing vision, increased macular thickness and outer retinal changes were indicators for vitrectomy.

Possible prognostic factors for favorable surgical outcome

While there are no reliable prognostic factors for a favorable surgical outcome, most surgeons define success in terms of postoperative VA. Prognostic factors associated with higher postoperative VA are younger age, lower central foveal thickness, longer photoreceptor outer segment length [measured from the ellipsoid zone (EZ) to the RPE], intact EZ and duration of symptoms $\geq$ 6 months.\textsuperscript{1,7,24,26,54}

Studied postoperative findings

Hartman et al. reported a restoration of the normal anatomy, on average, 4 months after vitrectomy.\textsuperscript{28} It often takes 4-6 weeks for a patient’s vision to return to preoperative level, and subsequent improvement continues over the following 3-6 months.\textsuperscript{26} It has been reported that 60-90% of patients have VA improvement of two or more lines by 6-12 months after surgery.\textsuperscript{26} Most metamorphopsia and, on average, scores on the National Eye Institute Visual Function Questionnaire - 25 improve postoperatively.\textsuperscript{28} Thus, even in the absence of VA gain, patients report improved quality of life with relief from some or all metamorphopsia.\textsuperscript{26}

For ERM specifically, 70-80% demonstrate improvement in VA of more than two lines following vitrectomy, which continues to improve up to 3 years thereafter.\textsuperscript{1,55} Idiopathic ERM recurs in approximately 10% of cases, and re-operation is required in approximately 3% of cases.\textsuperscript{7} In a recent study, recurrent ERM that required repeat vitrectomy occurred in 5.5% of cases, and all recurrences were at least 5 years after the initial surgery.\textsuperscript{55}

Clinical Pearls
OCT is more accurate than either fundus photography or biomicroscopy for detecting VMD

VMT is defined by persistent vitreous anteroposterior traction within a 3-mm radius from the fovea

Anomalous PVD is when persistent vitreous anteroposterior traction occurs outside a 3-mm radius from the fovea

Persistent anteroposterior and/or tangential traction becomes symptomatic when it affects the macula and perimacular areas

The effect that vitreoschisis has on the retina depends on the location of the vitreous cortical split in relation to the hyalocyte layer

ERM reinforces the adhesion between VMT and macula, preventing vitreomacular separation and prolonging tractional stress on the macula

A binocular workup is important to determine whether binocular diplopia is due to strabismus or binocular rivalry from metamorphopsia, micropsia or macropsia

As a risk factor for ERM, one should consider a patient’s vitreoretinal status before cataract surgery

The current standard of care for early stage, asymptomatic ERM and VMT is observation for 3 months. Shorter follow-up intervals with detailed OCT macula scans are recommended for patients with focal VMT or history of MH in the fellow eye

While there is no consensus on when to refer a patient for vitrectomy, surgery is often indicated for patients who experience a decrease in VA, metamorphopsia, double vision or difficulty using their eyes together

On average, restoration of normal retinal anatomy is observed at 4 months after vitrectomy, and 60-90% of patients show VA improvement of two or more lines by 6-12 months after surgery

Conclusion

The majority of ERMs remain relatively stable. However, when an ERM co-exists with another VMD, any existing vitreo-retinal traction is reinforced and the retinal architecture is further disrupted. Our patient’s previous providers assumed his ERM would remain stable given its 20-year-plus history, his age and lack of associated symptoms. Under this impression, even when his VA began to decline and central macular thickness increased on OCT after cataract surgery, the changes were considered minimal and his management remained the same. Even though he gained two lines of VA (from 20/60 to 20/40) after vitrectomy, his acuity did not return to his baseline of 20/25. Earlier identification of the vitreo-retinal traction, especially when vision began to decrease, would have prompted closer monitoring. Understanding the dynamic role of vitreo-retinal traction with ERM could have led to earlier intervention and a better vision prognosis for our patient.

References

An Atypical Case of Uveitis-Glaucoma-Hyphema Syndrome: a Teaching Case Report
Claire Henry, OD, Deana Lum, OD, FAAO, and Alvaro J. Castillo, OD, FAAO | Optometric Education: Volume 48 Number 2 (Winter-Spring 2023)

Background

Uveitis-glaucoma-hyphema (UGH) syndrome was first described in the late 1970s by F. Thomas Ellingson, MD, as a triad of findings caused by mechanical chafing of the anterior iris by the closed haptic loops of an anterior chamber intraocular lens (ACIOL). Though incidence has greatly decreased with the improved design of ACIOLs and the shift to posterior chamber intraocular lenses (PCIOLs), UGH syndrome remains a concern after cataract extraction. With PCIOLs, mechanical interaction between the intraocular lens (IOL) and the posterior iris or ciliary body causes irritation and erosion of the uveal structures, leading to release of pigment. Ultimately, this breakdown of the blood-aqueous barrier leads to the release of proteins and red blood cells (RBCs) into the anterior chamber. Pigment, blood, and inflammatory cells block aqueous outflow via the trabecular meshwork, which leads to increased intraocular pressure (IOP).

In a single-center retrospective study conducted between 2014 and 2018, iris-sutured IOLs accounted for 50% of the UGH syndrome cases. In-the-bag IOLs accounted for 20%; scleral-sutured IOLs accounted for 13.3%; sulcus-positioned IOLs accounted for 10%; and IOLs having “in-and-out” position accounted for 6.7% of the UGH syndrome cases. Prognosis for UGH syndrome depends on the timing of diagnosis. If IOP remains elevated without treatment for a prolonged period of time, the patient can develop glaucomatous optic neuropathy. Surgical intervention to either replace or reposition the IOL is preferable to medical therapy alone. Surgical IOL repositioning or replacement typically has a good visual prognosis and prevents recurrence.

In this report, we describe the case of a 74-year-old Caucasian male who presented urgently for a red eye and was diagnosed with UGH syndrome. We review differential diagnoses for hyphema, the history, pathogenesis, and clinical signs of UGH syndrome, as well as the treatment and management of hyphema and UGH syndrome. The intended audience is third- and fourth-year optometry students, optometry residents, and practicing optometrists.

Case Description

Initial visit (day 1)

A 74-year-old Caucasian male presented for an urgent exam complaining of a red right eye (OD) for 4 days. He reported mild irritation with foreign body sensation, but denied pain, photophobia, mucus, discharge, and any recent trauma or illness. His ocular history was significant for pseudophakia of both eyes (OU) with PCIOL displacement and subsequent repair OD, radial keratotomy (RK) OU, and two retinal detachments OD, one being macula-off. He was also being managed for severe primary open angle glaucoma OD and suspicion of glaucoma in the left eye (OS) and treated with latanoprost every evening OU. His medical history was significant for hypertension and history of mild stroke, for which he had been prescribed lisinopril and low-dose aspirin. He denied diabetes, sickle cell anemia, and any recent symptoms of a transient ischemic attack. He also denied any drug allergies.

His best-corrected visual acuity OD was light perception, reduced from hand motion at 1 foot 2 months prior, and stable OS at 20/25. Given his reduced vision, he was unable to perform confrontation visual field testing OD, but confrontation visual field was full to finger counting OS. Extraocular motility was grossly full OU. The pupil was difficult to visualize OD at this visit, and the left pupil was round and reactive to light. He appeared to have a stable 3+ relative afferent pupillary defect OD, tested by reverse pupil testing.

Anterior segment examination revealed unremarkable eyelids and eyelashes OU, significant hyperemia of the bulbar conjunctiva OD compared with a white and quiet bulbar conjunctiva OS. The cornea had stable cataract extraction and RK scars OU without Seidel sign, and RBCs on the inferior half of the corneal endothelium OD. The anterior chamber was deep and formed OU. There was a 1.5-mm hyphema OD with 3+ cell (mostly RBCs) and 3+ flare (Figure 1), while OS was quiet. There was poor visibility of the iris and lens OD, while OS had mild nasal iris atrophy and a clear, well-positioned PCIOL.

IOP measured 18 mmHg OD and 16 mmHg OS via Goldmann applanation tonometry. B-scan ultrasound revealed a grossly
intact posterior segment OD, with no indication of vitreous hemorrhage, ocular malignancy, or retinal detachment. Posterior pole OS was unremarkable and grossly stable to previous findings on undilated fundus exam. The patient was diagnosed with hyphema OD of uncertain etiology, primarily due to the inability to visualize the iris and retina.

At this visit, atropine 1% twice a day OD was initiated to stabilize the blood-aqueous barrier, and the patient was instructed to continue latanoprost every evening OU. As the patient was not symptomatic for uveitis, steroid treatment was deferred. The primary care physician was notified of the recommendation to order a carotid ultrasound. Discontinuation of aspirin was considered, but ultimately not advised unless evidence indicated it was causing a re-bleed. Standard hyphema precautions were advised including avoiding physical exertion, remaining at a minimum 45-degree incline, and using a protective eye shield for sleeping. The patient was instructed to return to the clinic the next day, or to go to the emergency department sooner if he experienced pain.

Follow-up care

Though IOP was normotensive on day 1, it increased to 30 mmHg OD at the first follow-up visit. As a result, latanoprost every evening OU was discontinued due to its pro-inflammatory effect and replaced with timolol 0.5%/dorzolamide 2% twice a day OU. Despite combination therapy, IOP remained in the 28-32-mmHg range OD while the hyphema was present. IOP remained stable and normotensive OS.

Over the course of the next 2 weeks, the vertical height of the hyphema decreased while on atropine 1% twice a day OD until it was noted as a microhyphema on day 13. The patient’s vision had returned to baseline at hand motion at 1 foot OD, and 20/20 OS.

By day 13, 3+ cell remained (including RBCs) OD but flare decreased to a grade of 1+, allowing visualization of the iris and appreciable midperipheral circumferential transillumination defects (TIDs) at 1:00-4:00, 6:00-7:00, and 9:00-10:00, and no neovascularization of the iris (Figure 2). The PCIOL was present but flush with the iris. Gonioscopy revealed angles open to ciliary body 360 degrees OU with 1+ pigment in the trabecular meshwork OU, flat iris approach OU, and no evidence of neovascularization of the angle, angle recession, or peripheral anterior synechiae OU. The view to the retina OD remained hazy, but no appreciable vitritis or vitreous hemorrhage was noted. All anterior segment findings OS were within normal limits.

Figure 1. External photo of the right eye at initial presentation: A 1.5-mm hyphema is visible in the inferior anterior chamber, and 3+ flare is obscuring the view of the pupil. The cornea has stable radial keratotomy scars with visible endothelial red blood cell staining on the inferior half. The conjunctiva has significant hyperemia. Eyelids and eyelashes are unremarkable. Click to enlarge

Figure 2. Retro-illumination of the iris of the right eye on day 13: Circumferential transillumination defects 1:00-4:00, 6:00-7:00, and 9:00-10:00 are appreciable. They follow the shape of the IOL optic and haptics. Click to enlarge
The patient was instructed to continue atropine 1% twice a day OD and timolol 0.5%/dorzolamide 2% twice a day OU. He was informed that he no longer needed to maintain a 45-degree incline but was advised to return to the clinic if redness or discomfort returned. The carotid ultrasound was completed and did not reveal any significant or asymmetric stenosis, ruling out ocular ischemic syndrome (OIS) as a diagnosis. Improved visibility of the iris confirmed lack of iris neovascularization, reducing the likelihood of neovascularization due to retinal vascular occlusion. However, because the fundus remained obscured, retinal vascular occlusion could not be definitively eliminated. Retro-illumination of the iris allowed visualization of significant iris TIDs, indicating mechanical interaction between the posterior iris and the PCIOL. Anterior segment optical coherence tomography (OCT) was used to confirm that the PCIOL was displaced anteriorly and interacting excessively with the iris (Figure 3a and 3b), supporting the diagnosis of UGH syndrome. The patient was referred to ophthalmology for evaluation of the IOL.

One month after onset, the patient was evaluated by the general ophthalmologist. At this visit, the hyphema resolved and IOP returned to normotensive with consistent use of timolol 0.5%/dorzolamide 2% twice a day OU. Given that the patient was now asymptomatic and had a long history of complicated and unsuccessful ocular surgeries, he declined surgical intervention. He stated that he was not motivated to preserve vision OD, and he opted to be monitored.

Case history

Given the rarity of in-the-bag PCIOLs causing UGH syndrome, a review of the patient’s ocular history helps to explain why this patient developed such a rare complication. In 2014, he underwent cataract extraction OU. In 2015, he was treated with pneumatic retinopexy for a macula-on retinal detachment OD. Two weeks later, the patient experienced a subluxated PCIOL OD, which the surgeon retrieved and repositioned into the ciliary sulcus. Soon after, the patient experienced a complete macula-off retinal detachment OD, which was subsequently treated with scleral buckle, pneumatic retinopexy, pars plana vitrectomy, endolaser, and silicone oil infusion. When the retina was stable, the retina specialist performed silicone oil removal and fluid-gas exchange.

As a result of this complicated history, the patient developed glaucoma OD, and vision declined to hand motion. The patient’s history of multiple retinal detachments, repeated intraocular surgeries, and IOL dislocation with repositioning into the sulcus likely increased his risk of UGH syndrome compared with non-complicated cataract extraction with PCIOL implantation.

Education Guidelines

Key concepts

1. The blood-aqueous barrier: importance and breakdown
2. Role of inflammation secondary to mechanical chafing on uveal tissue: pathophysiology and consequences
3. IOL malposition: clinical signs, complications, and treatment

Learning objectives

With this case discussion, participants should be able to:

1. Learn about the different etiologies and treatment of hyphema
2. Identify the signs/symptoms of UGH syndrome
3. Learn about the management of UGH syndrome

Discussion questions

1. What role do IOLs play in UGH syndrome, and to what extent do ACIOLs and PCIOLs differ in UGH syndrome? (How does the placement of the IOL in the eye increase/decrease the likelihood of UGH syndrome - ACIOL vs. PCIOL in the sulcus vs.
2. What is the typical pattern of TIDs seen in UGH syndrome cases? Why does this type of pattern occur?
3. What are differential diagnoses for a hyphema?
4. Why should we consider what medications a patient is on when diagnosing a hyphema?
5. What is the treatment for UGH syndrome? How successful is it?

Teaching instructions

The authors recommend reviewing the discussion and going over the discussion questions to ensure a thorough understanding of the topic. Use of figures/diagrams and photos to review the different locations in which an IOL may be implanted would be helpful in understanding the key concepts and pathophysiology of UGH syndrome. Also helpful would be a review of photos of circumferential TIDs and OCT images that highlight the iris/lens interaction.

Discussion

When UGH syndrome was first described in the 1970s, it was exclusively associated with the ACIOLs of the time. With updated lens design, improved lens fabrication, and evolved surgical techniques, the incidence of UGH syndrome has substantially decreased. With the transition to PCIOLs, many thought UGH syndrome would no longer occur as a post-surgical complication of cataract extraction. However, although it happens infrequently, PCIOLs are capable of causing inflammation and breakdown of the blood-aqueous barrier via mechanical chafing on uveal tissue, as is evidenced by this case. While prompt surgical intervention is recommended in most cases, this patient’s ocular history and limited visual prognosis allowed for less aggressive treatment.

Differential diagnoses for hyphema in adults

Hyphema due to trauma

The most common cause of hyphema is trauma. Hyphema-related orbital trauma is most commonly caused by a high-energy blow to the orbit (61-66%), followed by projectile (30.2-36%) and explosion (2.4-3%). There are several possible sources of bleeding in a traumatic hyphema. A direct blow may cause ruptured blood vessels at the anterior ciliary body or iris root, but equatorial expansion and elevated IOP may also cause rupture of ciliary body or iris blood vessels.

Neovascularization of the iris or angle

Ocular neovascularization is the result of ischemia to ocular structures, most often in the form of retinal hypoxia. Ocular ischemia results in release of angiogenic factors, including vascular endothelial growth factor (VEGF) and others, that stimulate new blood vessel growth. Unlike normal ocular blood vessels, these new blood vessels tend to leak fluid and blood and can lead to hyphema when they form in anterior structures. The most common causes of ocular neovascularization are diabetic retinopathy, retinal vein occlusion, and OIS.

a. Diabetic retinopathy: Poor glycemic control in diabetic patients can result in severe retinal ischemia, causing the distressed ocular tissue to release pro-angiogenic factors. This can result in both proliferative diabetic retinopathy and anterior segment neovascularization. Rubeosis iridis may or may not precede angle neovascularization, making gonioscopy a necessity in cases of hyphema or suspected neovascular glaucoma.

b. Retinal vein occlusion: After a retinal vein occlusion, ischemia and capillary nonperfusion lead to an upregulation of inflammatory cytokines and VEGF, resulting in ocular neovascularization. An ischemic central retinal vein occlusion can lead to neovascularization of the iris and neovascular glaucoma anywhere between 2 weeks to 2 years after the occlusion, but it most often occurs around 3 months.

c. OIS: ocular ischemic syndrome is hypoperfusion as a result of stenosis or occlusion of the common or internal carotid artery. Typical retinal manifestations are dilated retinal veins, mid-peripheral retinal hemorrhages, and neovascularization of the disc. Anterior segment involvement may lead to hypotony due to ciliary body hypoperfusion, but if iris and angle neovascularization occurs, this can also result in elevated IOP or hyphema.

Hyphema due to iris melanoma

Though extremely rare, hyphema can be caused by iris melanoma. Though only 2% of iris nevi converted to iris melanoma within 5 years in a Shields study of 1,611 eyes, hyphema was one predictive feature of growth.

Uveitis-glaucoma-hyphema syndrome
UGH syndrome is a condition that occurs after cataract extraction with IOL implantation. This condition is caused by mechanical chafing of the IOL with the iris, causing hemorrhaging, inflammation, and subsequent IOP increase. \(^5\) Though more frequently associated with ACIOLs, UGH syndrome can also occur with poorly positioned PCIOLs. \(^3\)

In the early days of cataract surgery, the anterior chamber was the preferred location for IOL implantation due to frequent dislocation of PCIOLs. The polymethyl methacrylate (PMMA) lenses of the time were too heavy to be supported posteriorly by the zonules and capsular bag, so surgeons viewed the anterior chamber as a more stable location. However, the anterior chamber placement was not without complications. In 1978, Ellingson first published his observation of the triad of uveitis, hyphema, and glaucoma caused by poorly fabricated ACIOLs. \(^1\) Initially coined Ellingson syndrome, uveitis-glaucoma-hyphema syndrome was attributed to poorly finished lenses, primarily warped footplates and poor edge finish. \(^12\) Lenses of similar design, but better manufacture quality, are still used today for ACIOLs with much lower frequency of complications. Other improvements made to ACIOLs over the years include discontinuation of nylon and metal as haptic materials and a switch to flexible semi-rigid loops. Also, lens insertion glides and viscoelastic have allowed for smoother lens insertion. \(^13,14\)

A significant decrease in the incidence of UGH syndrome is attributed to the rise of PCIOLs. In-the-bag placement reduces the risk of direct contact between the PCIOL optics or haptics with uveal tissue. Altogether, these improvements in lens design and surgical technique led to a decrease in annual incidence of UGH syndrome from 2.2-3.0% per year to 0.4-1.2%. \(^3,13\) Case reports of UGH syndrome associated with PCIOLs rarely, if ever, corresponded to uncomplicated cataract surgeries. PCIOL complications that have led to UGH syndrome include haptic migration into the anterior chamber, \(^15\) bag tearing with haptic migration into the sulcus, \(^16,17\) pseudophacodonesis secondary to pseudoxefoliation syndrome, \(^18\) and focal capsular fibrosis leading to mechanical chafing on the posterior iris. \(^19\) In this patient’s case, repositioning of the PCIOL into the sulcus likely led to excessive interaction between the iris and PCIOL.

In this case and many others, TIDs can provide helpful clues in diagnosis of UGH syndrome. TIDs after cataract extraction are common, but pattern and location can differ based on cause. In contrast with pupillary margin TIDs caused by surgical instruments during cataract extraction, circumferential shape and mid-peripheral location of TIDs indicate chafing of the IOL optics and haptic on the posterior iris. This unique pattern is indicative of excessive PCIOL interaction with the iris and is frequently seen in UGH syndrome. \(^5\)

Several treatment options are available for patients with traumatic hyphema. Typically, patients are given an eye shield and told to sleep at an incline to keep the blood in the inferior anterior chamber. Antifibrinolytic agents have been demonstrated to reduce the risk of re-bleeding in traumatic hyphema, but may prolong the duration of hyphema. \(^20,21\) Additionally, many patients reported side effects of nausea and vomiting, and the systemic formulation is contraindicated in patients with renal disease, as in the case with this patient. Also, topical formulations of antifibrinolytic agents must be requested from a compounding pharmacy and are difficult to access. Topical corticosteroids and cycloplegics decrease the risk of developing posterior synechiae, stabilize the blood-aqueous barrier, and reduce symptoms of uveitis. \(^22\) Furthermore, cycloplegics may increase uveoscleral outflow, and corticosteroids reduce the risk of re-bleeding. \(^5,20\) Given the poor visual potential and the initial unknown etiology due to poor visualization of the iris and posterior segment, a conservative management approach was taken with this patient. As the patient was not symptomatic for uveitis, he was treated only with atropine 1% twice a day OD until the hyphema resolved. The prostaglandin analog glucoaemia therapy was also replaced with a combination beta blocker/carbonic anhydrase inhibitor (CAI) due to inadequate IOP control on prostaglandin alone throughout the course of the hyphema.

Currently, there is no consensus regarding what effect aspirin has on the risk of re-bleeding in traumatic hyphema. Some clinical trials have found that use of aspirin made no difference in incidence of re-bleeding. \(^23\) Conversely, other studies found that use of aspirin increased the risk of re-bleed after traumatic hyphema. One small study found that 7 of 12 patients who used aspirin after traumatic hyphema experienced a re-bleed, compared with 1 of 13 patients who did not use aspirin. \(^24\) As a result, the decision to discontinue aspirin or other anticoagulant therapy in the case of hyphema should be made on a case-by-case basis. If a patient uses aspirin purely for its analgesic properties, it would be reasonable to recommend a different analgesic. However, if the patient has been prescribed aspirin or other anticoagulants to reduce the risk of stroke, as was the case with this patient, optometrists should be extremely cautious before recommending discontinuation of anticoagulants. Low-dose aspirin has been proven to reduce risk of ischemic stroke, \(^25\) and discontinuation of aspirin has been shown to lead to increased risk of recurrent stroke from 29% to 51% in moderate-to-high-risk patients. \(^26\) Therefore, discontinuation of aspirin was not recommended in this case, and the patient did not experience a re-bleed. Extreme caution is advised before discontinuing aspirin in cases of hyphema when the patient is at risk of stroke.

Another consideration in this case was the use of prostaglandins for IOP control in the context of active uveitis. The role of prostaglandins as an inflammatory mediator is well-established, but the suggested association between prostaglandins and uveitis remains controversial. In the late 1990s, Warwar and colleagues published a series of case reports indicating the incidence of anterior uveitis was 6% and the incidence of cystoid macular edema was 2% in patients using latanoprost,
suggesting that topical prostaglandins led to intraocular inflammation. Smith et al. found that initiation of latanoprost induced uveitis in 1% of patients without history of uveitis, caused recurrences in 23% of patients with history of uveitis, but did not cause worsening inflammation in patients with active uveitis. In a case series in which four patients experienced resolution of uveitis after discontinuation of latanoprost, all four experienced recurrences when latanoprost was re-challenged. However, a more recent study found no difference in anterior uveitis or cystoid macular edema in patients using prostaglandin analogs vs. other IOP-lowering therapies. Overall, these findings indicate that a very small percentage of people are susceptible to prostaglandin-induced uveitis, and prostaglandins may increase risk of recurrences in patients with history of uveitis.

Because of their slow mechanism of action, prostaglandin analogs are not recommended for treating acute elevated IOP in patients with hyphema or UGH syndrome. However, there is no current recommendation regarding use of prostaglandin analogs for pre-existing glaucoma during the treatment course of hyphema. While it is unknown if the pro-inflammatory properties of prostaglandins alter or prolong the healing course of UGH syndrome, it is unlikely to effectively control further elevated IOP due to hyphema. Therefore, beta blockers and CAIs are recommended during hyphema because they are fast-acting and target aqueous production.

In this case, the patient’s IOP was no longer well-controlled with a prostaglandin analog after the development of hyphema, and he was switched to beta blocker/CAI combination therapy to better control IOP.

With respect to other glaucoma medications, oral CAIs, especially acetazolamide, are contraindicated in patients with sickle cell disease or trait as they can increase sickling of erythrocytes. If an oral CAI must be used, methazolamide would be a better choice.

For hyphema caused by UGH syndrome, surgical repositioning of the IOL is the recommended treatment to prevent re-bleeding and prolonged IOP elevation. In a retrospective study looking at the outcome of 71 cases of UGH syndrome associated with PCIOls, 60 patients underwent surgery. Of these, 46 never had a recurrence of UGH syndrome, seven had only a single recurrence that subsequently resolved, and seven had no recurrences after a second repositioning surgery. In the 11 cases that were not treated surgically, five had a recurrence of UGH syndrome within the first year of follow-up. Of the 60 surgical interventions, 14 had IOL exchange and 46 underwent repositioning of the existing IOL with 71-74% success in preventing recurrences after the initial surgery. Even in cases where surgical intervention did not prevent recurrence, there was significant improvement in best-corrected visual acuity and IOP in the surgical group compared with the non-surgical group.

These findings indicate that the patient in this case report is at high risk of a re-bleed due to declining surgical intervention. However, this patient has profoundly reduced baseline vision and was generally asymptomatic during his bout of UGH syndrome, so there are limited benefits to surgical intervention. The goal of this patient’s ophthalmic care has shifted to comfort rather than vision preservation. Therefore, surgical intervention can continue to be deferred unless ocular comfort is compromised.

**Conclusion**

UGH syndrome is a rare but vision-threatening complication of cataract surgery. Though uncommon, especially in the case of PCIOls, it should remain a differential diagnosis in non-traumatic presentations of hyphema. In this case, the patient’s complicated history of retinal detachment and retinal surgeries led to malpositioning of the IOL, resulting in mechanical chafing and subsequent uveitis and hyphema. Though surgical intervention is recommended in most cases to improve vision and prevent recurrence, this patient declined surgery due to poor visual potential and resolution on conservative topical therapy. Nevertheless, surgical repositioning or replacement of the IOL will result in the best outcome for patients with UGH syndrome and reduce the risk of glaucomatous optic neuropathy. The current recommendation for management of hyphema is bed rest and elevation, topical corticosteroids and/or cycloplegics, avoidance of unnecessary anticoagulants, and control of IOP. These patients must be monitored closely until resolution of the hyphema, and the optometrist must investigate all possible etiologies of hyphema to address the cause and prevent recurrence.

**References**

**Commotio Retinae: a Teaching Case Report**

Raman Bhakhri, OD, FAAO, and Nicole Landry, OD | Optometric Education: Volume 48 Number 2 (Winter-Spring 2023)

**Introduction**

Commotio retinae is acute retinopathy resulting from blunt trauma to the globe. Commonly seen as whitening of the deep sensory retina, it has been recognized histologically as a disruption of the photoreceptor outer segment and damage to the retinal pigment epithelium (RPE).\(^1\)\(^,\)\(^2\) Commotio retinae has also been referred to as “Berlin’s edema” as it was originally theorized that the discoloration was caused by extracellular edema. However, since that time many studies have disproven this hypothesis.\(^2\)\(^,\)\(^3\) Differentials include other traumatic retinal conditions such as choroidal rupture, Purtscher retinopathy, traumatic macular hole, chorioretinitis sclopetaria, and retinal detachment. Non-traumatic differentials include conditions that present with a similar whitening as seen in ischemic retinal conditions such as retinal artery occlusions (RAOs).

Patients typically present with chief complaints of blurry vision, visual field loss, and/or metamorphopsia following trauma. Standard of care when diagnosing commotio retinae includes a thorough eye exam including dilation and scleral depression to aid in diagnosing any accompanying retinal breaks or detachments.\(^4\) Furthermore, additional ocular injuries associated with blunt trauma, such as open globe penetrations, orbital fractures, lens subluxation, and macular holes, must be ruled out upon initial assessment.

With optical coherence tomography (OCT), clinicians can obtain in vivo images of the specific retinal layers affected by commotio retinae. The affected layers can then be monitored for resolution as the condition currently has no treatment and usually resolves without any visual sequelae.\(^5\) This case report aids clinicians in utilizing and interpreting spectral-domain OCT (SD-OCT) findings in their management of commotio retinae.

**Case Description**

A 21-year-old Caucasian male presented to the eye clinic 8 hours after being hit in the right eye with a soccer ball. He had a chief complaint of decreased vision in the right eye since that time. He denied having pain and any flashes or floaters in his vision. His ocular history was significant for myopia but otherwise unremarkable. The patient’s medical history was also unremarkable. He denied taking medications or having any drug or non-drug allergies. There was no family history of ocular disease.

His best-corrected visual acuity was 20/40 in the right eye and 20/20 in the left eye. Testing with pinhole showed no improvement in acuity of the right eye. No significant findings were noted on external examination. The patient’s pupils were equal, round, reactive, without afferent pupillary defect; confrontation visual fields were full in both eyes; ocular motilities demonstrated a full range of motion in both eyes; slit lamp exam was unremarkable. Intraocular pressure measured with Goldmann applanation tonometry was 10 mmHg in each eye. Dilated fundus examination with accompanying scleral depression revealed no significant retinal findings in the right eye (Figure 1). The optic nerve was flat and distinct with a cup-to-disc ratio of 0.4 round. Examination of the left eye was unremarkable. Despite the normal retinal appearance in the right eye, a macula OCT scan was performed due to the reduced visual acuity. The scan was reliable based on signal strength and lack of artifacts. Results revealed disruption of the foveal inner segment/outer segment (IS/OS) junction (Figure 2). Imaging of the left eye was unremarkable. Based on the patient’s history of trauma and OCT findings, he was diagnosed with grade 1 commotio retinae in the right eye.\(^6\)

No treatment was indicated at that time, and the patient was scheduled for a follow-up exam in 4 weeks. He was asked to return to the clinic if he noted any changes in vision. At the follow-up visit, the patient noted a subjective improvement in his vision. The visual acuity in the right eye had improved to 20/20. All other findings, including with dilation, were stable to the previous examination. Scleral depression was unremarkable. A repeat OCT revealed complete resolution of the previous IS/OS disruption in the right eye (Figure 3). The patient was asked to return for a follow-up visit in 4 weeks for repeat dilated fundus examination and to rule out traumatic angle recession with gonioscopy. The patient failed to return and was lost to follow-up.
Education Guidelines

Key concepts

1. The basic anatomy, physiology, and function of the retina and its response to trauma
2. Commotio retinae may have minimal or no clinical signs, which makes additional testing important for accurate diagnosis
3. Clinical signs and symptoms to help differentiate commotio retinae from similar conditions
4. Recognition of OCT findings and long-term implications in patients with commotio retinae
5. Education on proper eye protection in work and sports to prevent ocular trauma

Learning objectives

1. Recognize the clinical presentation of commotio retinae including signs and symptoms
2. List the potential differential diagnoses of commotio retinae
3. Generate an appropriate management plan based on the results of multi-modal testing such as OCT

Discussion questions

1. Knowledge, understanding, and facts about the clinical case and condition presentation
   a. describe the typical appearance and presentation of commotio retinae
   b. discuss complications of trauma that can accompany commotio retinae
2. Differential diagnosis
   a. what other condition(s) should be considered as differential diagnoses for commotio retinae and how can a clinician differentiate between them?
3. Patient management and role of the optometrist
   a. what treatment is indicated for commotio retinae?
   b. what is the prognosis for commotio retinae patients with macular involvement?
   c. what is the prognosis of commotio retinae patients with no macular involvement?
   d. what type of patient education is required for patients who have experienced traumatic injuries to the eye?
4. Critical-thinking concepts
   a. what is the value of OCT in managing patients with commotio retinae, especially macula-involving?
   b. what additional testing should be performed in patients who are diagnosed with commotio retinae?

Assessment of learning objectives

Foundational knowledge of retinal anatomy is vital in understanding the pathophysiology of commotio retinae. Therefore, retinal OCT serves as a perfect teaching tool for educators. Educators could present normal retinal OCT scans and assign the students to properly identify and label anatomical landmarks. Once completed, this could be compared to retinal OCTs of patients with commotio retinae. Students would be asked to compare and contrast the two OCT images and try to arrive at how disruption or damage to specific retinal structures leads to corresponding patient signs and symptoms. This assignment could
be done in small or large-group settings such as in a laboratory or clinic conference room. This could be presented to
optometry students in all class years.

Higher-level concepts can also be assessed. Case studies can be presented to optometry students in their third and fourth year
either in a formal or online classroom. Students can be initially presented the case details and then can be tasked with arriving
at a proper diagnosis and management plan based on the presented findings and ancillary testing. Understanding and
knowledge can be evaluated through open-ended questioning to the class or through formal testing with multiple-choice
questions using a platform such as TurningPoint.

In an online format, the case can be presented along with appropriate adjunct testing and results. Students can then be tested,
through a multiple-choice format, on concepts involving pathophysiology, signs and symptoms, differentials, interpreting
testing, and treatment and management. Before students can move on to the next question in the series, they would be
required to answer the questions correctly. Feedback can be provided after the question is answered correctly. This can allow
students to learn from their mistakes and therefore strengthen their knowledge base.

Discussion

A retinal condition due to ocular trauma, commotio retinae was first described by Berlin in 1873 as Berlin’s edema as he
thought extracellular edema was the cause of the presenting retinal findings. Although the term Berlin’s edema is still used
at times today to describe commotio retinæ at the macula, studies have shown no evidence of extracellular edema but rather
damage to the photoreceptors and the RPE. As trauma is the direct cause, many risk factors can lead to commotio retinæ
either in a contrecoup or coup fashion. Risk factors for commotio retinæ include trauma from high-impact sports, namely ball
sports, but can also include blunt trauma to the face and orbits from violent encounters, car accidents, and falls. One study
noted the presence of commotio retinæ in 30% of patients presenting to a hospital for traumatic eye injuries. Young males
make up the majority of patients affected by commotio retinæ with studies noting the average age of patients to be 20-30
years. Exact epidemiological numbers are likely lacking as many patients tend not to present to eyecare or other providers
when symptoms are lacking, which can be the case with commotio retinæ.

Patient presentation can vary depending on the extent of trauma and
the location of commotio retinæ. Commotio retinæ usually presents as
a gray-white opacification of the retina with possible adjacent
intraretinal hemorrhaging and retinal pigment disruption. If located
at the fovea, patients can present with vision loss accompanied by field
loss. However, some patients may be asymptomatic if the condition
is located beyond the macula. Patients can also present with vision loss
without identifiable retinal whitening. In this case OCT is key in
detecting photoreceptor damage. The location of commotio retinæ in
the retina according to one study favored the temporal retina (inferior
temporal, temporal, or superior temporal) (Figure 4). This aligns with
trauma affecting the overlying sclera as the nasal sclera is somewhat
protected by the normal orbital anatomy. The extent of vision loss
varies and is dependent on the level and amount of photoreceptor
disruption. If the condition is limited to the mid-periphery or periphery
of the retina, visual acuity and visual field loss are unlikely to occur.

As the condition is traumatic in nature, patients can present with
associated findings and complications. These can include angle
recession, traumatic macular hole, retinal tear and detachment,
corneal abrasion, choroidal rupture, lens subluxation, hyphema, orbital
fractures, cataract, and traumatic uveitis.

As previously mentioned, Berlin hypothesized that the retinal findings in commotio retinæ represented intracellular edema;
however, no intracellular edema is present. Other proposed mechanisms of action have included glial cell proliferation and
fragmentation of photoreceptor outer segments. However, in their study, Mansour et al. revealed the retinal findings were due
to traumatic disruption to the outer retina. Specifically, it is thought that concussive forces from trauma damage the outer
retinal layers due to their inelastic structure when compared to the elastic sclera or the elastic layers of the inner retina. This
leads to disruption of the photoreceptor outer segments along with deposition of debris in the subretinal space and RPE
disturbances. With the advent of OCT, further investigations into the pathophysiology of commotio retinæ have shown IS and
OS hyper-reflectivity indicating disruption from the trauma while also correlating to areas of involvement noted on fundus
examination.

Figure 4. (A) An unrelated patient with temporal peripheral
commotio retinæ on initial presentation (yellow box). (B) Repeat
imaging one month later showed complete resolution of the
condition.

Click to enlarge
Ahn et al. further classified the degree of photoreceptor and RPE involvement with a four-step grading scale for commotio retinai, which allowed for predictions in visual and anatomic outcome (Table 1). Four specific bands were identified in the outer retina: the external limiting membrane (ELM), the photoreceptor IS/OS junction, the cone outer segment tips (COST), and the RPE. Using these landmarks, grade 1 represents an increase in IS/OS reflectivity with disappearance of thin hypo-reflective optical space. Grade 2 represents COST defects. Grade 3 represents COST and IS/OS junction defects with grade 4 representing COST defects, IS/OS junction, and ELM defects. It was concluded based on this study that eyes with a higher grade, and therefore more retinal layer involvement, at baseline scan correlated to worse visual and anatomic outcomes.

With advances in technology, such as those seen with OCT angiography (OCT-A) and enhanced depth imaging OCT (EDI-OCT), additional pathophysiological components of commotio retinai have been suggested.

With EDI-OCT, evidence of choroidal involvement has been studied. Burke et al. noted an increase in subfoveal choroidal thickness and increase in choroidal areas in eyes with commotio retinai compared with the normal fellow eye. This also correlated with a decrease in visual acuity in the involved eye. Although the exact pathophysiology was unclear to the authors, they suggested the choroidal findings could be dilation of choroidal blood vessels in response to the trauma. However, a more recent case series noted opposite findings. In this study, 70% of patients had a thinning in central choroidal thickness of the involved eye when compared with the fellow non-involved eye. This was attributed to decreased choroidal blood flow secondary to the trauma. It can be concluded that additional studies are needed to clarify the role of the choroid in commotio retinai.

With OCT-A, researchers have attempted to elucidate possible retinal vascular involvement. Results have varied. Mansour and Shields as well as Wangsathaporn and Tsui did not note any changes in the superficial and deep capillary plexus or the choriocapillaris. However, another more recent study suggested that vascular changes are present, as patients had enlargement of the foveal avascular zone along with decreased vessel density. The authors suggested that their study differs from the previous two in that their patients had more severe blunt injuries resulting in the changes observed. They also stated that their findings correlated to previous studies in which laser speckle flowgraphy showed reduced choroidal blood flow and indocyanine green angiography showed choroidal vascular damage. Although the studies showed possible associations of a hemodynamic nature, one should note that they were all case reports with very small sample sizes. Again, large case-controlled studies are needed to further validate a possible vascular component in commotio retinai.

Novel OCT findings have also been discovered in recent years. One group of researchers noted an original finding they termed micro-elevation of the ellipsoid zone in patients with commotio retinai. Although they noted the finding, they could not pinpoint an exact pathogenesis. Fortunately, the finding was temporary. Another novel finding was a non-reflective dark space in the sclero-choroidal interphase of some patients with commotio retinai, which the study authors speculated may have represented a fluid-filled space secondary to trauma. This finding was also temporary and it resolved during the clinical course of the condition. These findings were not noted in our patient.

Differentials diagnosis

It is important to differentiate commotio retinai from other conditions that may present similarly, namely those of a traumatic nature. Common differential diagnoses include retinal detachment, choroidal rupture, Purtscher retinopathy, traumatic macular hole, and chorioretinitis sclopetaria. One should note that these differentials can present concurrently with commotio retinai due to their similar traumatic etiology. Other differentials can include conditions that can present with a white/chalky retina, namely, ischemic retinal conditions.

- The impact from blunt trauma to the eye may cause abnormal vitreous traction resulting in retinal tears, which can lead to rhegmatogenous retinal detachments (RRD). Patients report an increase in floaters and flashes of light and a decrease in vision. When viewed clinically, a RRD typically has an elevated bullous appearance.
- Traumatic macular hole is a full-thickness defect of the neurosensory retina. Due to this finding, entering visual acuity tends to be much worse than in cases with macula-involving commotio retinai. It may be present after ocular injuries and can be found concurrently with commotio retinai. Traumatic macular holes tend to occur in younger males and are also often associated with sports and work-related accidents.
- Purtscher retinopathy, a rare, occlusive micro-vasculopathy, is characterized by multiple white areas surrounding the optic disc and fovea. Additional features include retinal hemorrhages, cotton wool spots, and Purtscher flecken (polygonal patches...
of intraretinal whitening in the posterior pole). These features are not typically seen with commotio retinæ. Patients commonly present with sudden painless bilateral vision decrease within 48 hours of trauma, which can include direct head trauma, injuries or compression of the chest, and long bone fractures.\textsuperscript{24,25} History, in addition to retinal findings, is key because commotio retinæ tends to be a result of head trauma only.

- **Trauma to the globe can result in a break in the RPE, Bruch’s membrane, and choriocapillaris, and is termed choroidal rupture.\textsuperscript{26,27}** Choroidal rupture appears as white-yellow curvilinear or crescent moon-shaped lesions that eventually lead to retinal scarring. In direct globe injuries, these ruptures are located anteriorly and parallel to the ora serrata, whereas indirect injuries produce ruptures concentric to the optic disc.\textsuperscript{27} This appearance is in stark contrast to the overall and transitory whitening seen with commotio retinæ.

- **Chorioretinitis sclopetaria, also known as traumatic chorioretinal rupture,** is rupture to the choroid and overlying retina caused by forces from a high-velocity projectile (e.g., gunshot, BB gun) to or near the globe. Histopathology shows photoreceptor loss, damage in Bruch’s membrane and the choroid, hyperplasia of the RPE, and multi-layered retinal hemorrhaging. The affected areas of the retina and choroid are subsequently replaced with fibrous tissue, giving the appearance of retinal whitening. This condition often has a poor visual prognosis, especially if the macula is involved.\textsuperscript{28-30}

- **RAOs also present with a white chalky retina; however,** they can be differentiated based on retinal anatomy and etiology. Artery occlusions tend to affect older patients with underlying systemic conditions in an acute fashion, while commotio retinæ patients tend to be young males with a history of trauma.\textsuperscript{31} Artery occlusions also result in more severe vision loss if the fovea is affected compared with commotio retinæ.\textsuperscript{31} Clinicians can also use OCT to differentiate the conditions if needed, as commotio retinæ affects the outer retina while RAos affect the inner retina.\textsuperscript{32}

### Treatment and management

As of now, there is no approved treatment for commotio retinæ. The condition tends to resolve over the course of a few weeks to a few months. Initial restoration of the photoreceptors tends to begin at 1 week post-injury.\textsuperscript{7}

In general, the condition has a favorable prognosis, and most cases resolve completely within 4 weeks with no additional sequelae.\textsuperscript{7} However, in more serious cases, patients may be left with permanent macular damage resulting in vision impairment and paracentral scotomas.\textsuperscript{4,22} As mentioned earlier, eyes with a higher grade of commotio retinæ, as determined by OCT, tend to have worse visual and anatomical outcomes.\textsuperscript{6} Blanch et al. noted that in their study of 53 patients with macula-involving commotio retinæ, 26% had an end acuity of less than or equal to 20/30. According to the authors, if symptomatic paracentral defects were accounted for, the number of patients with visual impairment (acuity loss and/or field loss) would likely have been higher. They also noted the prognosis of extra-macular commotio retinæ to be very favorable with almost all patients recovering pre-trauma levels of visual acuity.\textsuperscript{7}

As with many other ocular conditions affecting the posterior segment, steroids have been considered as a treatment option. Although rarely used, this includes systemic and intravitreal steroids. Mendes et al. in their case report used off-label high-dose intravenous methylprednisolone (550 mg) for 3 days followed by oral prednisolone (30 mg) for 10 days for a patient who presented with commotio retinæ and counting fingers vision that had persisted for 2 months. The vision improved to 20/200 with this treatment regimen. The patient was then given an intravitreal injection of triamcinolone with vision improving and stabilizing at 20/150.\textsuperscript{7} In another case report, oral prednisolone (50 mg) was used for 5 days in a patient with commotio retinæ.\textsuperscript{14} The visual acuity did improve from 20/30 to 20/20 over the course of a week, but one cannot assume the steroid led to this. The improvement may have been due to natural progressive healing. These case reports are of limited sample size. Larger studies are required to validate the use of any medications, including steroids, in the treatment of commotion retinæ. Due to the favorable outcomes without interventional treatment and the lack of case-controlled studies on steroid treatment, steroids are not a viable treatment option currently.

Trauma being the direct cause of commotio retinæ underscores the need for preventive measures that can be taken to avoid visual complications, namely avoidance of high-risk activities and proper use of protective eyewear (polycarbonate lenses, face masks, and protective visors). More than 600,000 sports and recreation-related ocular injuries occur every year, with a small proportion leading to permanent vision loss. Up to 90% of sports-related ocular injuries are preventable by using the correct eye protection.\textsuperscript{33} Eye protection is strongly encouraged for all participants in sports in which there is a risk of injury.\textsuperscript{33} Monocular precautions should be mandatory for those who have lost vision as a result of any traumatic condition, including commotio retinæ. As patients may present with additional traumatic conditions/complications at onset, they should be addressed and treated appropriately.\textsuperscript{8} In addition to performing scleral depression to rule out retinal breaks and detachments, clinicians should perform gonioscopy of the angle to rule out angle recession as an additional complication. One report noted that 60% of eyes developed some degree of angle recession after non-penetrating or concussive trauma.\textsuperscript{34} Clinicians should be aware that angle-recession glaucoma can develop months to years after the initial trauma and therefore glaucoma testing may be warranted in the future.\textsuperscript{35,36}
Conclusion

This case report highlights the importance of SD-OCT for diagnosing and monitoring commotio retinae, especially when there is a lack of obvious retinal whitening. Despite numerous previously documented cases with classic presentations, the condition may not always present with characteristic retinal whitening, as seen in this case.9,37

While there is no approved treatment for commotio retinae, it is advised that patients be monitored frequently post-diagnosis to evaluate for any treatable sequelae. It is also strongly encouraged that any individual participating in sports or other high-risk activities wear proper eye protection to prevent ocular injury.

References


Assessing COVID-19 Sleep Patterns in Optometry Students: Implications for Learning
Matt Valdes, OD, FAAO, Deidre Rios, MS, PhD, AHIP, Sandra Fortenberry, OD, FAAO, Christina Bahn, OD, and Lizette Martinez, OD | Optometric Education: Volume 48 Number 2 (Winter-Spring 2023)

Introduction
As many countries began implementing restrictions aimed at reducing the transmission of COVID-19, there was concern these measures may have unintended consequences on the mental and physical health of affected populations. Disruptions in sleep patterns and mental health issues had previously been linked with restriction of movement, psychological distress, limited social engagement and modified daily routines.¹² Chronic sleep issues can eventually develop into sleeping disorders resulting from the body’s inability to regulate its natural sleep mechanism and negatively impact learning and memory.⁶³ Through this research, we looked to better understand changes in sleep/wake behavior as a result of the COVID-19 lockdowns among a cohort of optometry school students.

Circadian rhythm, the body’s biological clock, is responsible for controlling the sleep/wake cycle corresponding to the 24-hour light/dark phases of the Earth.⁶⁷ In addition, it can be influenced by physiological changes, diet, social/physical activity and artificial lighting.⁷⁸ The COVID-19 lockdowns dramatically altered daily structure and daily routines, leaving no aspect untouched. This led to an abrupt convergence of personal and professional life as “work from home” and remote learning responsibilities intersected with caregiving and self-care needs.¹⁰ This unexpected shift in daily activities requires further analysis to determine whether such changes affect individual sleep behavior and learning potential.

Sleep metrics such as time to bed (TTB), wake time (WAKE) and duration (DUR) have often been utilized to quantify sleep quality and differentiate between sleeping disorders.¹¹ These values are closely connected to societal pressures (e.g., meal times, work responsibilities, social events, etc.) and are generally synchronized with the body’s sleep-wake rhythm.¹²¹³ Earlier sleep research found less WAKE variability during the week among college-aged students, corresponding to the start of the academic day.¹⁴ Would home confinement, growing anxiety and less daily structure lead to the development of abnormal sleep patterns and potentially impact student learning?

Methods

Participants
This longitudinal study was conducted during the summer semester of 2020 during a period of remote learning at University of the Incarnate Word Rosenberg School of Optometry. Seventeen full-time (greater than 16 credit hours) optometry students in their third year (OPT III) were recruited using flyers placed in their school mailboxes and in common-use areas. Participation was voluntary and had no impact on students’ grades. No compensation was provided beyond participants being allowed to keep their activity trackers upon completion of the study. Enrollment required students to complete a pre-study questionnaire and wear a wrist-based accelerometer (WBA) for 30 days (21-day minimum). Exclusion criteria included pregnancy or nursing.

Study approval
This study was approved by the Institutional Review Board and was in compliance with the Declaration of Helsinki. All subjects provided written informed consent.

Data collection
Two sets of passively recorded sleep data, from summer 2019 (n₁₉ = 34) and summer 2020 (n₂₀ = 15), were analyzed and compared. All subject data were de-identified, and unique identification numbers were used to track each participant. No subjects were duplicates in the 2019/2020 cohorts. Data were stored on password-protected cloud systems.

Pre-study questionnaire
Pre-study questions included demographics, perceived sleep patterns, subjective sleep quality and various social behavior metrics (e.g., meal regularity, caffeine consumption and exercise). Only gender and age were considered in this assessment of WBA-based data.
Wrist-based accelerometers

Participants were provided a WBA, commonly referred to as an activity tracker. The WBAs (Xiaomi Mi Band 2, Taipei, TW) in this study utilized movement and heart rate to measure TTB, DUR and WAKE events for all subjects. All participants were encouraged to wear the WBA throughout the day and night for the duration of the study. Passive data collection was then used to create sleep profiles for each participant and cohort (Figure 1).

Statistical analysis

The data were collected and analyzed using Google Sheets (Mountain View, CA) and XLMiner Analysis ToolPak (Incline Village, NV). Jarque-Bera test was applied to confirm the data were normally distributed, and F-test was used to confirm equal variance (p > 0.05 for all data sets). Therefore, an unpaired t-test was utilized for comparison with a similar cohort (OPT III) during the same time frame, 1 year prior (summer 2019). Findings were considered statistically significant if the p-value was less than the pre-specified alpha of 0.05 for the various categories.

Results

Twenty subjects (out of 68) completed the pre-study questionnaire, of which 15 (2 male and 13 female; mean [SD] age, 25.3 [1.6] years) successfully wore the WBA and tracked their sleep for 30 days (minimum: 25 days, average: 28.8 days). The data were then compared to a cohort of 45 (out of 61) students, of which 34 (13 males and 21 females; mean [SD] age, 25.6 [1.9] years) successfully completed the study during summer 2019 (in-person learning), using the same method described above.

Mean sleep duration

Table 1. Summary sleep metrics comparing the 2019 and 2020 cohorts (mean±SD). Click to enlarge

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<thead>
<tr>
<th>Event</th>
<th>2019</th>
<th>2020</th>
<th>Diff</th>
<th>p-value</th>
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<tbody>
<tr>
<td>DUR WKD</td>
<td>9h41m57m</td>
<td>7h37m44m</td>
<td>+56m</td>
<td>&lt;.001*</td>
</tr>
<tr>
<td>DUR WKE</td>
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<td>8h00m40m</td>
<td>-30m</td>
<td>.005*</td>
</tr>
<tr>
<td>DUR Avg</td>
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<td>7h07m40m</td>
<td>+30m</td>
<td>.003*</td>
</tr>
<tr>
<td>TTB WKD</td>
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<td>00:29±5m</td>
<td>-13m</td>
<td>.019*</td>
</tr>
<tr>
<td>TTB WKE</td>
<td>00:19±9m</td>
<td>00:43±5m</td>
<td>-31m</td>
<td>.17</td>
</tr>
<tr>
<td>TTB Avg</td>
<td>00:43±7m</td>
<td>00:31±8m</td>
<td>-12m</td>
<td>.57</td>
</tr>
<tr>
<td>WAKE WKD</td>
<td>07:14±3m</td>
<td>07:51±4m</td>
<td>+38m</td>
<td>.91*</td>
</tr>
<tr>
<td>WAKE WKE</td>
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<td>08:23±5m</td>
<td>-11m</td>
<td>.01*</td>
</tr>
<tr>
<td>WAKE Avg</td>
<td>09:57±5m</td>
<td>09:30±5m</td>
<td>+18m</td>
<td>.28</td>
</tr>
</tbody>
</table>

* indicates a statistically significant change during lockdown

WBA data showed mean [SD] DUR to be 7h37m [40m] during the lockdown (Table 1). The 2020 subjects received significantly more sleep than the 2019 subjects (7:07 [44m] vs. 7:37 [40m], p = 0.03). WBAs allowed for differentiating between weekday (WKD – Sunday, Monday, Tuesday, Wednesday, Thursday) and weekend (WKE – Friday and Saturday) sleep patterns. Each measure consisted of the night they fell asleep (TTB) and their WAKE. For example, Friday sleep data would consist of TTB Friday evening and WAKE Saturday morning. Mean WKD DUR was 56 minutes more than pre-pandemic values (6h41m [57m] vs. 7h37m [44m]). Mean WKE DUR was 30 minutes less than pre-pandemic values (8h05m [58m] vs. 7h35m [40m]). Students were getting significantly more sleep during the week (p < 0.001), significantly less sleep during the weekend (p = 0.05) and significantly more sleep overall (p = 0.03) as compared to 2019 sleep data (Figure 2).

Time to bed

WBA data showed the overall mean [SD] TTB to be 00:31 [60m], which was 12 minutes earlier than in 2019 (00:43 [72m]). WKD TTB was measured to be 00:20 [65m], which was 13 minutes earlier than in 2019 (00:33 [74m]) and not statistically significant (p = 0.53). Subjects went to bed 29 minutes earlier during the WKE with TTB being 01:19 [90m] and 00:48 [57m] in 2019 and 2020, respectively (p = 0.17). TTB metrics were consistent between cohorts with no statistically significant differences for WKD, WKE or overall average as compared to 2019 sleep data (Figure 3).
WBA data showed an overall mean [SD] WAKE of 07:50 [52m] and 08:08 [55m] in 2019 and 2020, respectively (p = 0.28). WKD WAKE was delayed by 43m (07:14 [43m] vs. 07:57 [54m], p = 0.01) in 2020 compared to 2019. Conversely, WKE WAKE was earlier (09:24 [86m] vs. 08:23 [63m], p = 0.01) during the pandemic as compared to 1 year prior. Students' WAKE was significantly later during the week, significantly earlier during the weekend and held steady on average as compared to 2019 sleep data (Figure 4).

**Discussion**

To the best of our knowledge, this is the first study to examine sleep patterns in optometry students during the COVID-19 lockdowns through passive means over an extended period. To better understand how lockdowns affected optometry school students, we observed an increase in DUR, a delay in WAKE and consistent TTB compared to pre-pandemic sleep patterns. This contrasts with prior studies that found declined sleep quality, elevated levels of stress and insomnia among other university student populations during the COVID-19 pandemic.16-17

**Sleep duration**

Overall DUR during the lockdown was longer and more in line with the recommended 7-8 hours of sleep per night.18 The greatest increase was observed in WKD DUR as students increased their average sleep time by 56 minutes compared to pre-pandemic values. This was accompanied by a WKE DUR sleep decrease of 30 minutes. This shift between WKD and WKE DUR was likely due to a lack of sleep debt accrual during the academic week.

**Time to bed vs. wake time**

Studies have also found sleep timing events (TTB and WAKE) to be important metrics in overall sleep quality that correlate with academic performance.13-15 Minimal change was observed in overall TTB from 2019 to 2020, with TTB being slightly more consistent in 2020. Observed increases in DUR were primarily attributed to shifts in WKB and WKE WAKE. The observed delay in WAKE WKD and advanced WAKE WKE were both statistically significant and consistent with prior research looking into the benefits of delayed school start times.19 Watson et al. found no significant change in bedtimes but an increase in overall sleep duration as a result of delayed wake times.19 The American Academy of Sleep Medicine has since stated its support of delayed academic start times for adolescents to reduce sleep deprivation and improve mental health.19-20 These findings could be used to help guide future discussions related to academic start times and best practices for optimal learning among optometry school students.

**Learning and memory processing**

The implications for this sleep study go beyond simple behavioral differences. Prior studies have linked sleep duration and consistency with better academic performance.11-12 Although the mechanism is not well-understood, it is believed the act of sleeping facilitates information restructuring, memory processing and information retrieval.21 Better sleep has also been linked to stress management, which is a common issue in various health professions.22 Future efforts to create a well-rounded approach to academic success should incorporate positive sleep habits.
**Pandemic’s disparate impact**

From the outset, public health officials have had concerns around the unequal challenges facing various populations during the COVID-19 pandemic. Depending on one’s vocation, socio-economic and pre-pandemic sleep quality status, the impact may differ significantly.\(^{20,23-24}\) The first step in managing the potential problems facing optometry students was to evaluate what changes were taking place during this time. Quantitatively, it would appear our subjects improved in some sleep metrics (e.g., TTB and DUR), which have previously been linked to better mental health and a reduction in stress/anxiety during a highly stressful time.\(^{25}\)

**Limitations**

These findings should be considered within the context of the following limitations. As with many sleep studies, reliance on proprietary algorithms that have not been validated introduces a degree of systematic error for all measurements. Sleep onset, duration and wake events are calculated through heart rate and movement, which may not consistently differentiate being “in bed” from being “asleep.” Unequal sample sizes were also considered as it may limit the statistical power. We look to compare more equal cohorts in future studies. Additionally, the WBAs used in our study were also unable to capture naps, which may have played a larger role during the pandemic due to fewer daytime constraints, as one study suggests.\(^{16}\)

**Conclusion**

This study was able to highlight a significant shift in wake times for optometry school students during the COVID-19 lockdowns. The observed increase in sleep duration can be directly correlated with the delayed wake times, most likely due to the flexibility of virtual learning (i.e., asynchronous lectures and increased productivity) and lack of commuting time. Future studies are needed to investigate the relationship between increased sleep duration and academic performance.

**Acknowledgments**

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**References**


Impact of Clinically Centered Microbiology and Immunology Coursework on Student Perceptions and Learning Outcomes

Michelle Demory Beckler, PhD, Beata I. Lewandowska OD, MS, Pranati Movva, MS, and Joshua M Costin, PhD | Optometric Education: Volume 48 Number 2 (Winter-Spring 2023)

Introduction

For learners entering programs in health professions who have limited or no clinical exposure to patient care, the knowledge gained through the basic science courses is difficult to connect to clinical scenarios. This challenge can be overcome by linking basic science concepts to clinical problems, often through patient-based or case-based learning. Medical schools have realized the importance of including clinical tasks early in the curriculum and have termed the mixing of basic and clinical sciences as vertical integration. In a similar fashion, some optometry programs have moved away from the conventional mode of teaching preclinical didactic courses followed by clinical instruction to interlinking the clinical and basic sciences within the curriculum.

Infections by a variety of microorganisms are a common etiology of eye diseases encountered in optometric clinical care. Optometrists must be knowledgeable in basic sciences and the recognition of the symptoms and signs of ocular infections by a variety of pathogens. On occasion, optometrists may need to collect specimens from the ocular surface and prepare them for delivery to a lab for analysis. In addition, optometrists develop care plans to eradicate the offending microorganisms, promote tissue healing by taking into account the patient’s immunological status, educate the patients and their families, and effectively communicate with other members of the healthcare team. Optometric clinics, as outpatient settings, must comply with infection prevention guidelines to minimize the spread of infectious agents. The study of medical microbiology and immunology allows optometry students in their pre-clinical preparation to appreciate the various pathogens responsible for ocular infections as well as to understand the body’s immune responses against the infectious processes.

Two ways microbiology and immunology are taught in optometry programs are as a standalone course, such as the one offered by Nova Southeastern University’s College of Optometry (NOVA), or as topics within an integrated curriculum. Despite the importance of understanding microbiology and immunology as a foundation for other basic sciences and clinical concepts, some programs have reduced the number of hours for microbiology and immunology. NOVA has similarly seen a decrease in the number of hours for didactic lectures, with microbiology reduced from a 3-credit-hour course to a 2-credit-hour course during the 2019-2020 academic year. Considering the reduction in didactic hours or a reduced number of integrated hours of microbiology and immunology within a systems-based curriculum, it is becoming increasingly necessary for basic science course instructors to include vertically integrated, clinically focused course material in a very limited time frame to deliver a high-yield curriculum.

To emphasize the importance of microbiology and immunology to our pre-clinical optometry students, our goal was to integrate clinically correlated concepts and material into the course. We sought to design a clinically rich, standalone medical microbiology and immunology course that integrated clinical cases into didactic lectures and examinations. In addition, we collaborated with clinical optometric faculty to design co-lectures dedicated to the clinical application of microbiology and immunology to demonstrate the significance of the basic sciences to patient encounters. The clinical application of the basic science topics was assessed in summative examinations, and student satisfaction and perception were monitored by surveys.

To the best of the authors’ knowledge, no published studies have examined the impact of vertical integration of clinical application in a medical microbiology and immunology course for optometry students.

Methods

Course structure and exams

Microbiology is a required 2-credit-hour first-year optometry course at NOVA. The course covers the basic immunology of the human body and the biology of microorganisms, incorporating a general medical approach to ocular diseases. In the Fall 2020 administration, the course content was delivered by a total of two microbiology, one immunology and two clinical lecturers. The microbiology and immunology lectures were delivered as didactic PowerPoint presentations with and without polling questions for engagement, depending on the lecturer and the topic. Foundational science material was presented covering
basic concepts in immunology or microbiology with an overall emphasis on its clinical relevance and application of basic science to diseases using case studies and examples. A series of eight clinical co-lectures (CCLs) and case presentations were delivered by the clinical optometric faculty throughout the course and covered two topics each in immunology, bacteriology, virology and mycology (Table 1). These CCLs integrated foundational science information with clinical course and case presentations to highlight the application of microbiology and immunology body-wide, with an emphasis on the ocular region.

Student performance was assessed by three written examinations of 40 multiple-choice questions per assessment, covering lecture presentations, assigned reading material and integrated clinical lectures. These examinations included questions that assessed students’ ability to integrate foundational information with clinical scenarios. The first examination tested immunology material (block 1) and contained six case-based questions, two of which came directly from the CCLs. The second examination tested bacteriology material (block 2) and contained 11 case-based questions, two of which came directly from the CCLs. The third examination tested virology and mycology material (block 3) and contained 12 case-based questions, two of which came directly from the CCLs.

Survey and exam administration

Institutional Review Board approval was granted for this study, which employed quantitative methods using surveys to assess student perception of course material as well as multiple-choice examinations to assess understanding and retention of the learning objectives. While class attendance and examinations were a course requirement, completion of the educational surveys as part of this study was not mandated, and students had the option to decline to answer each of the survey questions without penalty. Recruitment was restricted to the 129 students currently enrolled in the microbiology course in Fall 2020. Announcements to participate were made both during class and via the course page on the Canvas Learning Management System, making it clear that participation was entirely voluntary. No incentives were offered to participate. To minimize any potential feelings of coercion, it was announced that data would not be examined or analyzed in any fashion until the final grades for the course were released. All data were de-identified prior to analysis to protect student privacy.

Between August 2020 and December 2020, 129 students were enrolled in the microbiology course. Of these, 93 were female (72%) and 36 were male (28%). Figure 1 describes the relative timeline of events for the study. Surveys were designed and administered on ExamSoft, a secure testing digital platform. The first survey was administered at the beginning of the course to gauge students’ interest in microbiology and immunology and assess their prior knowledge of the subjects. The following three surveys were given after the delivery of the corresponding clinical correlate lectures and asked students’ perspectives on the course material, delivery of course content and perceived relevance to their careers. The second survey covered the perception of immunology (block 1). The third survey combined topics from bacteriology and virology (blocks 2 and 3), while the fourth survey was a concluding survey containing topics from mycology (block 3) as well as questions pertaining to students’ experiences with the course overall. In an attempt to lessen student survey fatigue, survey questions for block 3 were divided between the final two surveys, thereby eliminating the need for an additional survey to be administered. All surveys ranged from six to nine questions and were used to assess students’ perceptions of the importance and relevance of the survey topics.

Table 1. Click to enlarge

<table>
<thead>
<tr>
<th>Foundational Science Block</th>
<th>Clinically Integrated Topics</th>
<th>Clinical Correlation (Common Signs)</th>
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<tbody>
<tr>
<td>Immunology (Exam 1)</td>
<td>Rheumatic arthritis</td>
<td>Marginal keratitis</td>
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<td></td>
<td>(Type III hypersensitivity)</td>
<td>Periorbital ulcerative keratitis</td>
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<td>Malignant degeneration</td>
<td>Confluent skin infections</td>
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<td></td>
<td>Allergic conjunctivitis</td>
<td>S. aureus infections of eye and</td>
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<td>(Type I hypersensitivity)</td>
<td>alveolar (Pneumonia)</td>
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<td></td>
<td>Viral keratitis</td>
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<td></td>
<td>(Type II hypersensitivity)</td>
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<td>Keratoconjunctivitis</td>
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<td>(Type IV hypersensitivity)</td>
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<td>Oral lesions</td>
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<td>Mucous membrane infectious</td>
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<td>infections</td>
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<tr>
<td></td>
<td>Other infections</td>
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</tbody>
</table>

| Bacteriology (Exam 2)    | Haemophilus influenzae      | Adenovirus conjunctivitis         |
|                         | Bacteriophage                | (Corneal and Conjunctival)       |
|                         | Neisseria                    |                                    |
|                         | Streptococcus                |                                    |
|                         | Mycobacterium                 |                                    |
|                         | Pneumococcus                  |                                    |
|                         | Corynebacterium              |                                    |
|                         | Pasteurella                   |                                    |

| Virology (Exam 3)        | Canine Parvovirus            | Canine parovirus infections       |
|                         | Feline Parvovirus            |                                    |
|                         | Canine Herpes B               |                                    |
|                         | Canine Distemper              |                                    |
|                         | Canine Feline                 |                                    |
|                         | Canine Herpes                |                                    |
|                         | Canine Herpes                |                                    |
|                         | Canine Herpes                |                                    |

| Mycology (Exam 3)        | Candida albicans              | Candida parovirus infections      |
|                         | Saccharomyces cerevisiae      |                                    |
|                         | Aspergillus                   |                                    |
|                         | Histoplasma                   |                                    |
|                         | Cryptococcus                   |                                    |

Figure 1. Relative timing of surveys, exams and clinical co-lectures throughout the three blocks of the course. Black text = introductory and concluding surveys before and after blocks; Blue text = block 1 material and survey; Green text = block 2 material and survey; Orange text = block 3 material and survey. Click to enlarge
of the subject matter and clinical correlation lectures to their future careers. Surveys used either multiple-choice time-frame options (0, 1, 2, 3, 4, or more semesters) or a Likert scale with four options (strongly agree, agree, disagree, and strongly disagree). Responses to four prompts were assessed: (1) How many semesters of microbiology did you take before starting in the College of Optometry? (2) How many semesters of immunology did you take before starting in the College of Optometry? (3) Medical immunology is important to my future career (4) Medical microbiology is important to my career.

Three multiple-choice examinations of 40 questions each were administered using ExamSoft throughout the semester. Exam questions based on the clinical lectures and case-based foundational lectures were administered as part of the regular examinations that assessed student understanding of the course learning objectives. One hundred and twenty total multiple-choice questions were administered over the course of the semester. Of those, six clinical case-style questions assessing the material presented during the case-based and clinical lectures were administered as part of the immunology exam, 11 as part of the bacteriology exam, and 12 as part of the virology and mycology exam. There were eight multiple-choice questions from a total of 120 exam questions based on the material delivered by clinical faculty – two from immunology, two from bacteriology, two from virology, and two from mycology. Table 1 lists the integrated topics covered on each exam.

Data analysis

Data were analyzed and graphed using GraphPad Prism version 9.1.1. Simple linear regression was performed on the raw data. A p value < 0.05 was considered significant. Simple descriptive statistics and tables were constructed using Microsoft Excel version 16.50.

Results

To determine whether the microbiology semester-long course redesign met the objectives of our study, four surveys were administered over the course of the semester (Figure 1). Data reported in this study were drawn from questions present on surveys 1, 2 and 4. Data from all other survey questions were inconclusive (data not shown). Not all students took all four surveys, and out of those who took the surveys, not all answered all questions. At the beginning of the course, 110 students responded to the questions “Medical immunology is important to my future career” and “Medical microbiology is important to my career” using a Likert scale with four options: strongly agree, agree, disagree, and strongly disagree (survey 1; 110 respondents, 85%). Students were then asked the same questions once the immunology (survey 2; 101 respondents, 78%) and microbiology (survey 4; 88 respondents, 68%) subjects were delivered.

Overall student response rate to microbiology course redesign

Anecdotally, there was noticeable excitement and increased engagement from students after each clinical lecture. Students voluntarily returned the surveys in large numbers. Survey 1, the pre-course survey, had 110 respondents (85%), survey 2 had 101 respondents (78%), and survey 4, the post-course survey, had 88 respondents (68%). Additionally, students performed well overall in the course with a class average of 82%.

Prior student experience in microbiology and immunology coursework

Most students reported having some background in microbiology, with a majority of responding students (86%) reporting taking one semester of microbiology prior to matriculating in the College of Optometry. Fewer students had an immunology background, with a majority (66%) having never taken immunology, and 32% having taken only one semester of immunology prior to starting the optometry program (Table 2).

Assessment of students’ perceptions of the importance of microbiology and immunology

The results of the pre- and post-course survey question regarding student perception of the importance of medical immunology to their career indicated most students had a positive association and either agreed or strongly agreed (Figure 2). In total, there were 110
respondents to this question on survey 1 and there were 88
respondents to this question on survey 4. There were no statistically
significant differences between the pre- and post-surveys (paired t
test, p > 0.05) for this question. Perception of medical microbiology as
important to their career also indicated that most students had a
positive association and either agreed or strongly agreed (Figure 3).
In total, there were 110 respondents to this question on survey 1 and
there were 88 respondents to this question on survey 4. There were no
statistically significant differences between pre- and post-surveys for
this question (paired t test, p > 0.05).

Students were assessed at the conclusion of the immunology (block 1) and microbiology sections (blocks 2 and 3) of the course
about their opinions on the usefulness of integrating clinical correlations into the medical microbiology and immunology
material with the questions "I feel incorporating clinical correlation into the medical immunology section of this course is
helpful to my understanding of the course material and how it will apply to my future career as an optometrist" (survey 2) and
"I feel incorporating clinical correlation into the medical microbiology section of this course is helpful to my understanding of
the course material and how it will apply to my future career as an optometrist" (survey 3). Among 104 respondents, 97%
responded positively to the statement that incorporating clinical correlation into the medical immunology section of the course
was helpful to their understanding of the course material, with 69% strongly agreeing (Figure 4). Similar results were
observed regarding the medical microbiology section with 93% of respondents responding positively to the statement and 55%
strongly agreeing out of a total of 100 respondents (Figure 4).

Clinical case-style exam questions based on the clinical lectures and case-based foundational lectures were used to assess
students’ understanding and appreciation of the clinical integration in this course. A total of 29 case-style questions were used.
As students answered more clinically correlated questions throughout the course correctly, their final grades in the course
increased in a linear fashion (Figure 5). Simple linear regression of the relationship is well-represented in a line of best fit
with the equation $Y = 1.975 \times X + 35$ with a goodness of fit ($R^2 = 0.9527$). This slope of the resulting regression line is
significantly different from zero ($p < 0.0001$). Increased understanding of clinical application overall appears to increase
understanding of all the material, including foundational material, indicating an increase in learning outcomes overall.
Further analysis of the questions strictly arising from the CCLs was also predictive of overall success in the course. Eight multiple-choice questions over the semester were based on the clinical faculty co-lecture material. Success on these eight questions was highly predictive of overall success in the course as measured by the exam average (Figure 6). Simple linear regression of the relationship is well-represented in a line of best fit with the equation \( Y = 2.343X + 68 \) with a goodness of fit \( (R^2) = 0.7961 \). This slope of 2.343 is significantly different from zero \( (p < 0.0001) \). Thus, using the number of correct responses to the clinical correlate questions exclusively as a proxy for increased understanding of the material is reasonable and objectively validates student feelings towards the clinical co-lecture material (Figure 4).

Discussion

This study is the first to elucidate the relevance of an integrated microbiology and immunology curriculum adapted to teach optometry students the knowledge necessary for their clinical practice. Previous studies have looked at integrating clinical education in microbiology coursework for medical students\(^{13}\); however, optometry students require clinical education in the classroom that matches their area of specialization. The overarching goal of our course design was to enhance student perception and performance within the course itself, which our data suggest was a success.

By introducing clinical material early in pre-clinical education, we sought to enhance our optometry students’ clinical knowledge. We believe this is particularly important given that with the aging U.S. population, optometric practices are likely to become more medically oriented and will play a larger role in the diagnosis and management of ocular infectious diseases.\(^{14}\) In addition, the knowledge gained through microbiology and immunology coursework is vital in complying with infection prevention guidelines and understanding how to protect optometrists, staff and patients from infections commonly spread in optometry practices.

The most current curriculum comparison of U.S. optometry schools was published in 2003 and discussed a reduction in the number of hours for didactic studies in the 2001-2002 school year when compared with previous years (1991-92 and 1995-96).\(^{15}\) Our program has seen a similar shift. We believe one way to make a standalone microbiology and immunology course successful and meaningful to optometric student education is to mimic the vertical integration seen in programs that use systems-based curricula. Therefore, we used a two-pronged approach in the design of our microbiology and immunology course.

This study incorporated a quantitative approach, which allowed course instructors to evaluate students’ perceptions of the usefulness of the integrated model to learning through survey-based questions and student feedback as well as quantitative evaluation through exam scores. Survey responses gave insight into students’ perceptions of the relevance of the coursework. This metric was important to evaluate because it has been shown that when students perceive what they are learning matches their goals, they are more likely to engage with the material presented to them.\(^{16}\) Students found the clinical integration useful,
and this was reflected in the individual exam scores that increased linearly with increased performance on clinical lecture-based questions. Thus, introducing clinical lectures into microbiology and immunology coursework appears to improve student engagement and performance in the course. It is important to note that this was achieved without any additional resources or budget as clinical optometric faculty are commonly employed by optometric programs. There is a cost associated with faculty time devoted to providing clinical lectures. However, in our program, clinical faculty are given service hours to the college that allowed them to participate in the course.

According to the 2020 Association of Schools and Colleges of Optometry’s “Optometry A Career Guide,” microbiology, but not immunology, is a common prerequisite course for admission to U.S. optometry programs. Therefore, most students enter optometry programs, including ours, with a baseline knowledge of microbiology. In our study, students’ prior knowledge of microbiology was not quantitatively assessed before the start of the course; therefore, it is possible that their previous level of exposure to microbiology could impact their perception of the relevance of the coursework and their performance on assessments in the course. Results of the pre- and post-course survey question regarding students’ perception of the importance of medical immunology to their career in our study were not significantly different from each other (paired t test, \( p > 0.05 \)) in large part due to an overwhelmingly positive agreement to the question. However, it was notable that while overall student perception of the importance of medical immunology to their career was 95% (pre-course) and 96% (post-course) positive, the course increased the number of students who “strongly agreed” by 11% and eliminated all answers of “strongly disagree.”

The perception of medical microbiology as important to the students’ career was also not significantly different before and after the course (paired t test, \( p > 0.05 \)) due in large part to a strong positive response to the question with 88% of students either agreeing or strongly agreeing. However, there was a 10% increase in positive perception to 98% of total respondents who were asked if medical microbiology was important to their career after the course. Among positive respondents, 16% more “strongly agreed” after the course, and there were no longer any students who “strongly disagreed.”

Future studies should address this concern. The relative dearth of research articles regarding optometric curricula also appears to echo this lack of emphasis. At the same time, there is a wealth of research articles regarding microbiology and immunology in other health professions, particularly regarding clinical correlations of the material. In addition, the current study was limited in scope to one class in one optometry program in the United States. It would be illustrative to see the results of a longitudinal study in multiple programs across the country to see whether these results remain consistent and are widely applicable.

Conclusions

At the beginning of our course, most students had prior experience taking microbiology courses, while few had the same previous experience taking immunology courses. Irrespective of that prior experience, most students believed both subjects were important to their future career as optometrists. Vertical integration of clinical content throughout the foundational course in microbiology and immunology was well-received by the students. In addition, students’ course performance was positively impacted by the clinical integration as measured by performance on examinations. While not directly assessed, our results suggest that a clinically focused microbiology and immunology course in an optometric program, and not simply prerequisite microbiology, can positively impact students’ learning experience in their preclinical years. It remains to be seen whether this success carries over into subsequent, more clinically focused courses as well as students’ careers as optometrists.

Given our results, a multi-institutional, longitudinal study looking at the impact of clinically focused microbiology and immunology courses in optometric programs vs. only pre-requisite microbiology on academic and clinical performance is warranted. Future studies may also consider different clinically based integrations, such as case-based, team-based and problem-based learning approaches to promote discussion, invite questioning and encourage self-directed student learning.

References

Call for Papers: Theme Edition to Focus on Global Optometric Education

| Optometric Education: Volume 48 Number 2 (Winter-Spring 2023)

We are pleased to announce a theme edition of the journal that will be dedicated to global optometric education. We welcome manuscript submissions that highlight research, curricula, pedagogy, public health initiatives and other projects that align with the theme edition’s mission of sharing ongoing efforts to advance the profession of optometry worldwide.

You may submit your manuscript in the customary format [https://journal.opted.org/publication-guidelines/] or as an informational report or article. Content-specific reviewers will be assigned to support atypical submissions.

The submission deadline for this theme edition is January 2024. Send your cover letter with an intact and blind copy of your manuscript to submissions@opted.org. Email Optometric Education Editor Aurora Denial, OD, FAAO, DAAO (OE), if you have any questions about the theme edition.
Optic Nerve Compression due to Dolichoectasia: a Teaching Case Report

Payton Yerke Hansen, BS, Katelyn L. Seeley, BS, and Duane L. Tanner, OD | Optometric Education: Volume 48 Number 2 (Winter-Spring 2023)

Background

Dolichoectasia is a progressive vascular disease characterized by the fusiform dilation and elongation of intracranial arteries. This condition is rare and has a poor prognosis when considering 5-year survival rate. Most cases are asymptomatic, increasing the patient’s risk. Accurate diagnosis and timely intervention are crucial for improved outcomes. Dolichoectasia is primarily associated with sustained systemic hypertension. Inherited connective tissue disorders, such as Ehlers-Danlos syndrome, can also lead to weakened vessel walls that contribute to the dilation and distension of the vessel. Chronic increased pressure within the arterial wall interferes with collagen and elastin integrity in the tunica intima. As the connective tissue structure degrades, the vessel begins to enlarge, compressing surrounding structures. The diagnosis of vertebrobasilar dolichoectasia requires the basilar artery, the most affected vessel, to have a minimum diameter of 4.5 mm and a vertical deviation of > 10mm from the expected course.

Less than 10% of dolichoectasia cases present with clinical symptoms. In some cases, the elongated and distended intracranial arteries compress cranial nerves, which inhibits action potential propagation. This compression can cause variable neurologic deficits based on which nerves are compressed. Symptoms that have been associated with dolichoectasia include ischemia, central sleep apnea, cerebellar dysfunction, obstructive hydrocephalus, trigeminal neuralgia and motor defects. Dolichoectasia increases the risk of ischemic or hemorrhagic stroke. The variable symptoms create difficulty with diagnosing and treating dolichoectasia. As a result, many cases are misdiagnosed.

While uncommon, if the dolichoectatic vessel compresses the optic nerve fibers, it disrupts the visual pathway and can lead to vision impairment. We present a case report of dolichoectasia involving the optic nerve in a 79-year-old White male. The case report is intended to serve as a teaching guide to assist optometry students, residents and practitioners in recognizing the clinical presentation of dolichoectasia and using a Humphrey 24-2 full-threshold visual field test to determine whether neurological disease is present.

Case Description

A 79-year-old White male presented at the Salt Lake City Veterans Administration Medical Center optometry clinic for a pigmentary glaucoma follow-up appointment on March 1, 2017 (initially diagnosed by a different provider in 2015). The patient also had a previous diagnosis of cataracts. Other significant medical history did not include a family history of glaucoma. The patient’s blood pressure measured 160/93 mmHg. He had been prescribed medication for systemic hypertension but had self-discontinued it 7 years prior. He also reported that he had white coat syndrome, so he monitored his blood pressure at home. His most recent blood pressure measurement was 132/78 mmHg 2 days before the appointment.

The patient’s medications were acetaminophen/oxycodone, 325/5 mg tablets twice a day as needed for pain; ketotifen ophthalmic drops twice a day for ocular allergies; latanoprost ophthalmic drops once a day for glaucoma; dextran artificial tear drops 4 times a day as needed for dry eyes; and sildenafil citrate for erectile dysfunction prior to sexual activity. Recent lipid profiles, glucose levels, complete blood count and hemoglobin A1c levels were within normal ranges.

The patient had previously been diagnosed with pigment dispersion glaucoma and associated transillumination defects. He had thinner than average corneas with corneal pachymetry measurements of 536 µm in the right eye and 529 µm in the left eye. Eyes with thinner corneas are known to have intraocular pressure (IOP) underestimated by 2-5 mmHg by applanation tonometry. Optical coherence tomography (OCT) showed significant thinning of the retinal nerve fiber layer in the superior-temporal and inferior-temporal sectors of both eyes. Asymmetrical cupping of the optic discs was observed. These tests were repeated as was a Humphrey 24-2 full-threshold visual field test. The results from the visual field test did not correlate with the OCT findings. Instead, there was a consistent yet progressing left incongruous homonymous visual field defect, indicating a possible neurological condition (Figure 1).

The patient also complained of headaches in the morning, but his IOP remained within normal range. He reported significantly clearer vision in the morning after awakening and that his vision worsened throughout the day. The patient was referred for an
MRI of the brain and orbits with and without gadolinium contrast dye concentrating on the right parietal lobe and the orbits. The images showed tortuous and enlarged anterior and middle cerebral arteries that distended and bilaterally exerted a mass effect upon the optic chiasm and optic tracts (Figure 2). The anterior and middle cerebral arteries appeared to be dolichoectatic and resting on the floor of the third ventricle (Figure 3). This confirmed the diagnosis of dolichoectasia.

It was recommended that the patient resume taking medication to lower his blood pressure, but he refused. It remained uncertain whether he had glaucoma in addition to dolichoectasia. Due to this uncertainty, the patient was instructed to continue taking latanoprost and follow-up on his condition within 9 months. The patient underwent cataract extraction surgery. During that surgery, a micro-implant was placed into the trabecular meshwork of each eye to help improve aqueous outflow and control IOP. Once his eyes healed, the patient stopped taking latanoprost. After the diagnosis in 2017, his vision worsened, and debate continued among providers as to whether he had glaucoma in addition to dolichoectasia. In 2018, the patient suffered a non-fatal ischemic stroke.

Education Guidelines

Learning objectives

Students should be able to:

1. describe why the clinical presentation of dolichoectasia can be so varied
2. describe the causes of dolichoectasia
3. recognize and describe how to know when visual field test findings indicate a likely neurological abnormality such as a mass or aneurysm
4. accurately trace the visual pathway through the brain
5. explain the relationship of the cerebral blood supply pathway to the visual pathway and identify areas where compression of visual fibers is most likely to occur
6. acknowledge that a patient may have multiple conditions occurring at the same and create a list of differential diagnoses based on symptoms and examination findings

Key concepts

1. dolichoectasia can cause vision impairment
2. dolichoectasia can present clinical symptoms that are similar to those of other conditions
3. patients can have multiple conditions occurring at the same time; therefore, a holistic approach is necessary for diagnosis
4. dolichoectasia left untreated has a high morbidity and mortality rate

Discussion questions

1. why does dolichoectasia often present with variable symptoms?
2. why are most cases of dolichoectasia asymptomatic?
3. why is dolichoectasia progressive?
4. why does lateralization (field defects respect the vertical midline) on a visual field test indicate a possible neurological
disorder?
5. what is the vascular pathway of the internal carotid artery and the vertebral artery, and how do they relate anatomically to
the cranial nerves?
6. why was the patient’s vision affected in both eyes? How was vision affected? what vision symptoms may the patient have
had?
7. why did the patient’s vision improve after he had been lying down?
8. why was the patient’s visual field defect crossing the vertical midline?
9. how does dolichoectasia increase a patient’s risk for ischemic stroke?

Discussion

Literature review

Dolichoectasia is an uncommon but highly recognized vascular phenomenon that is seen more often in middle-age and elderly
patients. It is also predominant in males. The disease preferentially affects the vertebral and basilar arteries, which was
not observed in the case presented. We observed dolichoectasia in the right anterior and middle cerebral arteries.

Dolichoectasia has a poor prognosis with a 5-year survival rate. More than 90% of dolichoectasia cases are asymptomatic, and
symptomatic cases vary in their clinical expressions. This places an increased burden on doctors to provide a correct
diagnosis in a timely manner. Because blood flow is impeded, ischemia and a higher risk of ischemic and hemorrhagic strokes
are commonly observed. It is not uncommon for intracranial bleeding to occur. The 3- to 5-year survival rate of those
diagnosed with dolichoectasia is 60-70% with asymptomatic patients having a better prognosis.

In some cases, the distended artery may compress cranial nerves, which leads to a variety of symptoms based on what nerve is hindered
and at which position along its pathway the compression occurs (Figure 4). Common symptoms documented are hemifacial spasms
and trigeminal neuralgia caused by the pulsatile compression of the facial nerve root and trigeminal nerve root. Other symptoms that may
be observed are hearing loss, tinnitus and nystagmus caused by the compression of the vestibulocochlear nerve. Horner syndrome and
diplopia may be caused by the compression of the oculomotor, abducens or trochlear nerves, and dysphagia when the medullary
pyramids are constricted. These conditions are uncommon because the cranial vasculature has less access to compress the nerves along the optic
tracts. The potential for these events corresponds to the location, the severity of elongation, and dilation of the arterial wall.

Dolichoectasia was originally regarded as an atherosclerotic disease, similar to aneurysms. Histological studies found that the basis of the
pathology is chronic hypertension, which causes the degeneration of the internal elastic lamina as well as atrophy of vascular smooth
muscle. This deterioration makes the disease highly progressive because as the vessel’s diameter increases, it creates a shift in
pressure based on Poiseuille’s Law. In areas where the diameter is larger, the pressure pushing against the vessel walls increases. The
differences in vessel diameter creates a bidirectional, turbulent blood flow that significantly increases the risk of thrombus formation and
ischemia. Both symptomatic and asymptomatic patients need repeat imaging to monitor the progression of the disease and functional
testing such as blink reflex, vision tests, brainstem auditory-evoked potentials and motor-evoked potentials. The most common
management is controlling hypertension through medication and lifestyle changes. There are also surgical options for severe cases in
which impaired cerebral spinal fluid circulation increases intracranial pressure. Most surgeries involve a ventriculoperitoneal shunt. If the
vessel obstructs the foramen of Monro, a biventricular shunt is used.
In cases similar to what we observed, some vision loss due to dolichoectasia can be regained through the surgical removal of the ectatic area of the vessel, decompressing the optic nerve by separating it from the vessel via interposition of muscle tissue, or de-roofing the optic canal and cutting the dural fold and sheath of the optic nerve. However, this last procedure is highly debated among practitioners.

**Teaching methodology and assessment**

This information could reasonably be covered in one or two 1-hour sessions. Students should spend approximately one-third of the allotted time working in a small group. Another one-third of the time should be devoted to self-study. A larger group wrap-up session should be completed during the last one-third of the time. The facilitator should lead the wrap-up session by choosing students at random to explain different portions of the case.

Participants should think critically about each discussion question using the information provided in the Background and Case Description. At the conclusion of the allotted time for review of the information provided, participants should submit their answers in an essay format. Their responses can be compared to the information below each question.

**Why does dolichoectasia often have variable symptoms?**

The clinical symptoms expressed depend on which cerebral nerves are compressed by dolichoectatic vessels and how it affects the antegrade blood flow in the system. Symptoms are based on the severity of the condition.

**Why are most cases of dolichoectasia asymptomatic?**

Symptoms arise due to the compression of nerves or the disruption of blood flow and are proportionate to the severity of the condition. Dolichoectatic vessels generally do not interfere with the cranial nerves, and they may not hinder the blood flow enough to cause ischemia or stroke.

**Why was lateralization of the patient’s visual field incongruous?**

The patient had multiple dolichoectatic vessels that interfered with his sight depending on the nerves compressed as well as where along the nerve pathway the compression was located. He also may have had glaucoma, which could have caused a portion of the visual field loss.

**Why does lateralization of the field of vision indicate a neurological disorder?**

Lateralized loss respecting the vertical midline in a patient’s field of vision is neurologically significant based on the structure of the optic pathway. Temporal vision fibers travel through the medial portion of the optic nerve to the optic chiasm, where they cross and continue to the occipital lobe. Nasal vision fibers travel through the lateral portion of the optic nerve to the optic chiasm. Lateralization in one or both eyes indicates that the optic pathway is disrupted. In our case, the patient experienced lateralization in both eyes. Because both eyes were affected, we knew there must be a chiasmal or post chiasmal location for the vascular compression. This was confirmed with an MRI scan.

**What is the vascular pathway of the internal carotid artery and the vertebral artery, and how do they relate anatomically to the cranial nerves?**

Blood supply to the brain comes from four main arteries, the two carotid and the two vertebral (Figure 5).

The vertebral arteries travel up the lateral sides of the brainstem and then merge into one larger vessel, the basilar artery. The basilar artery continues up the anterior midline of the pons to the level of the midbrain where it connects into the circle of Willis. The arteries (inferior and superior cerebellar) and arterioles that branch off the vertebral and basilar arteries provide the blood supply to cranial nerves 3 through 12. When the basilar artery reaches the midbrain, it bifurcates into the posterior communicating arteries. These vessels connect with the internal carotid arteries. The carotids give rise to the middle and anterior cerebral arteries. The anterior communicating artery connects the two anterior cerebral arteries to complete the

![Figure 5. Brainstem vasculature and emerging cranial nerves.](image)
formation of the circle of Willis. The ophthalmic arteries are a bifurcation of both internal carotids. They supply the second cranial nerves. The anterior cerebral arteries supply the first cranial nerves.

**Why was the patient’s vision affected in both eyes? How was vision affected? What vision symptoms may the patient have had?**

Vision was affected in both eyes because of the architectural layout of the optic nerve fibers and their distribution throughout the visual pathway. At the optic chiasm, the nerve fibers from both eyes cross and then travel to the appropriate side of the brain and back to the visual cortex. When there is homonymous visual field loss in both eyes that respects the vertical midline, the lesion must be located at or posterior to the optic chiasm, affecting peripheral vision.

**Why did the patient’s vision improve after he had been lying down?**

The forces of gravity played a large part in the patient’s symptoms. In the supine position, gravity moved the distended blood vessel off the impinged visual nerve fibers. In the upright position, gravity moved the vessel back onto the affected visual nerve fibers causing the vision to be worse.

**Why is dolichoectasia progressive?**

As an artery increases in size, it forces the blood to travel from a large-diameter area to a small-diameter area. Based on Poiseuille’s Law, when liquid travels through a system with changing diameter, the velocity increases in the small-diameter area, and the pressure against the walls of the system increases in the area with a larger diameter. The increased shift in pressure against the dolichoectatic area of the vessel creates a larger force against the walls that can cause the intima to further deteriorate. This can lead to a progressive increase in diameter and elongation.

**How does dolichoectasia increase a patient’s risk of ischemic or hemorrhagic stroke?**

The dilated portions of arteries create a bidirectional flow, which causes a net reduction in blood flow leading to ischemia. The bidirectional current causes turbulence in the blood flow, which increases thrombus formation leading to stroke.

**Conclusion**

This teaching case report is especially useful because it requires participants to consider multiple diseases and the anatomy and physiology of the visual system in order to correlate clinical findings to the patient’s actual condition. The clinician must use a variety of testing modalities to effectively accomplish this. The increased morbidity associated with undiagnosed dolichoectasia makes accurate identification and prompt treatment crucial. Multidisciplinary coordination and action are essential in reducing loss of function and extending the patient’s life.

**References**

Horner Syndrome: a Teaching Case Report
Shelly Kim, OD, FAAO, and Sarah Rogers, OD | Optometric Education: Volume 48 Number 2 (Winter-Spring 2023)

Background
This case involves a 74-year-old Caucasian male diagnosed with iatrogenic Horner syndrome (HS) following recent ipsilateral carotid endarterectomy. HS is caused by disruption in the oculosympathetic pathway, which gives rise to the classic triad: ptosis (drooping of the upper eyelid), miosis (constricted pupil), and anhidrosis (absence of sweating).\(^1\) Most cases of HS are acquired due to damage to the sympathetic nerve supply, but congenital HS can occur due to trauma during birth.\(^2\) HS is usually due to a benign condition, but clinicians should always consider a more serious or life-threatening etiology such as neoplasm or carotid dissection.\(^3\) This case report highlights the importance of the optometrist’s role in detection of HS and evaluation and management of an HS patient. It focuses on the importance of accurate diagnosis, as well as radiologic imaging studies needed, to investigate the underlying cause of the condition. This case is appropriate for third- and fourth-year optometry students, optometry residents, and optometry providers.

Case Description
A 74-year-old Caucasian male presented to the eye clinic with a non-specific complaint of right eye irritation. The examiner noticed right upper eyelid ptosis. During case history and questioning, the patient reported this began about 4 days after right-side carotid endarterectomy. This surgery was performed for carotid artery stenosis 1 month prior. The patient had an uneventful left-side carotid endarterectomy approximately 1 year prior. He denied symptoms of amaurosis fugax, head and neck pain, and diplopia. He was also unaware of anhidrosis on the right side of his face.

The patient’s medical history was significant for type 2 diabetes, hypertension, hyperlipidemia, coronary artery disease status post coronary artery bypass graft, and bilateral carotid artery stenosis status post bilateral endarterectomy.

His best-corrected visual acuity was 20/50 in the right eye (OD) and 20/40 in the left eye (OS). The vision was reduced in both eyes due to moderate cataracts. Extraocular movement was smooth and unrestricted in all gazes. Confrontation visual fields were full in each eye with no pain or diplopia. Intraocular pressure (IOP) was 15 mmHg OD and 17 mmHg OS. Pupil assessment revealed anisocoria that was greater in dim illumination, consistent with miosis OD (Figure 1, Table 1). Gross external examination revealed 2-mm upper lid ptosis and reverse lower lid ptosis in the right eye.

One drop of apraclonidine 1% was instilled into each eye at 2:29 p.m., 2:36 p.m., and 2:41 p.m. At 3:24 p.m., 55 minutes after the initial dose of apraclonidine, both pupils were re-evaluated. The right pupil dilated and the left pupil remained unchanged, indicating reversal of anisocoria. The patient was diagnosed with right-side HS with high suspicion of an iatrogenic cause. To rule out other potential causes, non-urgent MRI of the brain and brainstem and magnetic resonance angiography (MRA) of the head and neck were ordered. All studies were unremarkable. The patient’s cardiologist and vascular surgeon were alerted, and the patient was instructed to continue follow-up care with his healthcare team.

Education Guide
The Education Guide includes the necessary information for teaching and discussing the case. The learning objectives, key concepts, and discussion points should guide the teaching objectives.

Key concepts
1. Sympathetic innervation of the eye
2. Hallmark signs and symptoms of HS
3. Critical-thinking in diagnosis, using diagnostic tests in-office to aid in making the diagnosis
4. The importance of the eyecare provider in a team of healthcare professionals

Learning objectives

At the conclusion of this case discussion, participants should be able to:

1. Describe the classic triad seen in HS
2. Try to illicit localizing signs with a thorough case history
3. Understand the different underlying causes of HS
4. Differentiate HS from other pupil and ptosis conditions
5. Assess the urgency of the condition and outline a management plan for the patient

Discussion questions

1. Knowledge, concepts, facts, and information required for critical review of the case:
   a. Describe the classic presentation of a patient with HS
   b. Understand the different topical pharmaceutical agents used in the diagnosis and localization of HS: cocaine, hydroxyamphetamine, and apraclonidine

2. Differential diagnosis
   a. What other conditions are differential diagnoses?
   b. How can HS be differentiated from other pupil and ptosis anomalies?
   c. What in-office pharmaceutical test was performed in this case to diagnose the condition?
   d. Should other differential diagnoses have been considered?

3. Patient management and the role of the optometrist
   a. What are appropriate management options?
   b. What is an appropriate follow-up schedule?
   c. What is the prognosis for a patient with HS?
   d. What is the urgency of additional testing?
   e. Summarize interprofessional team strategies for improving care coordination and communication to improve patient outcomes

4. Critical-thinking
   a. Could the diagnosis be made without topical pharmaceutical agents?
   b. How would the treatment and management plan change if the patient develops late-onset pain?
   c. How would management have changed in a patient with limited insurance coverage?
   d. Considering the high likelihood of iatrogenic HS in this case, did the patient require subsequent radiologic studies?

Discussion

HS is a rare condition that presents with unilateral ptosis, miosis, and facial anhidrosis due to disruption of sympathetic innervation to the eye. It was first described in animals by Francois Pourfour du Petit in 1727. It was investigated further by the French physiologist Claude Bernard, who observed the condition in a soldier who sustained a gunshot injury to his neck in 1854. The condition was formally described and correctly attributed to oculosympathetic paresis by Swiss ophthalmologist Johann Friedrich Horner.

Anatomy

To understand the features of this syndrome, it is critical to understand the sympathetic innervation to the eye. The nerve supply is mediated by three neurons that originate in the posterolateral hypothalamus and end as the long ciliary nerves to supply the iris dilators and superior tarsal muscles (Müller’s muscles).
uncrossed, terminating at the C8-T1 level of the spinal cord in the intermediolateral cell columns (ciliospinal center of Budge and Waller). The pre-ganglionic, second-order, neurons exit from the ciliospinal center of Budge and Waller at the level of C8-T1 and pass across the pulmonary apex. They ascend through the stellate ganglion to synapse in the superior cervical ganglion at the level of C3-C4. The post-ganglionic, third-order, neurons branch off into the sudomotor and vasomotor fibers, which follow the external carotid artery and innervate the sweat glands and blood vessels of the face. The remaining fibers travel in the wall of the internal carotid artery and continue to the cavernous sinus where they join the abducens nerve (CN VI) before joining the ophthalmic division (V1) of the trigeminal nerve (CN V) as the long ciliary nerves.

Etiology

HS is an uncommon condition and occurs with a frequency of approximately 1 in 6,000 people. It can occur at any age and in any ethnicity. It is primarily an acquired condition secondary to systemic or local diseases or iatrogenic causes. However, approximately 5% of cases are congenital.

First-order neurons are mostly affected by intracranial conditions that can include:

- stroke, most commonly lateral medullary infarction (Wallenburg syndrome)
- Arnold-Chiari malformation
- basal meningitis
- intracranial tumors (basal skull or pituitary)
- demyelinating lesions
- spinal cord tumors
- neck/cervical cord trauma
- syringomyelia

Second-order neurons travel across the thoracic region and are affected by:

- malignancies involving the apex of the lung (Pancoast tumor, mesothelioma)
- cervical rib injuries
- mediastinal lymphadenopathies
- lumbar epidural anesthesia
- iatrogenic (thyroidectomy, radial neck dissection, tonsillectomy, coronary artery bypass grafting, chiropractic manipulation, or carotid angiography)

Third-order neurons are near the internal carotid artery and cavernous sinus and are affected by:

- carotid cavernous fistula
- carotid artery dissection (commonly due to trauma) or aneurysm
- other internal carotid artery lesions (thrombosis, endarterectomy, stenting, radial neck surgery, trauma)
- cluster headaches or migraines
- Raeder paratrigeminal syndrome
- herpes zoster infection
- giant cell arteritis
- idiopathic causes

Clinical features

Although HS classically presents with a triad of symptoms — ptosis, miosis, and anhidrosis — the clinical features may vary depending on the location of the lesion and degree of innervation.

Ptosis

The ipsilateral upper lid appears drooped due to paresis of the sympathetically innervated Müller’s muscle. The Müller’s muscle is responsible for approximately 2 mm of elevation of the upper eyelid. The ptosis may be subtle, temporary, and variable. One study found that 12% of HS patients presented without an upper eyelid ptosis. The lower eyelid may appear mildly elevated, a “reverse ptosis,” due to weakening of the retraction of the lower eyelid as well. The resulting narrowed palpebral fissure may induce an appearance of enophthalmos.
The ipsilateral pupil appears miotic due to the disruption of the sympathetic nervous supply. This leaves the parasympathetic supply inhibited and results in pupil constriction. The pupillary reaction to light and accommodation is normal, as these systems are not dependent on the sympathetic nervous supply.

Paresis of the iris dilator muscle impairs pupillary dilation, leading to “dilation lag.” This can be seen clinically by illuminating from below with a hand-held light source and abruptly shutting off the lights. The normal pupil dilates immediately. The Horner pupil dilates several seconds later due to denervation of the parasympathetic tone.

**Anhidrosis**

Ipsilateral anhidrosis is dependent on the level of degeneration. First-order neuron lesions affect the ipsilateral body due to their central origin. Second-order neuron lesions cause ipsilateral facial anhidrosis. Post-ganglionic, third-order, neuron lesions occur after the vasomotor and sudomotor fibers have branched off and show little involvement of the face.

Facial anhidrosis can be evaluated clinically with the sweat friction test. A prism bar and the patient’s forehead are cleaned with alcohol. After allowing for the alcohol to dry, the prism bar is placed flat against one side of the forehead. It is drawn down with mild pressure. The same is done to the other side of the forehead. The side with anhidrosis will provide less resistance compared to the normal side.

Immediately following sympathetic denervation, the temperature of the skin can rise due to the affected vasomotor control. This leads to vasodilation of blood vessels, which can be seen clinically as facial flush, conjunctival injection, and epiphora.

**Iris heterochromia**

Ipsilateral iris heterochromia can occur in congenital HS. The sympathetic activity deficiency can cause interference with pigmentation of the melanocytes in the iris stroma, which can be seen clinically as iris hypopigmentation on the affected side.

**History and examination**

A detailed history and physical examination are crucial in localizing the lesion in HS. One study concluded that the cause of HS will be known at the initial neuro-ophthalmologic consultation in two-thirds of patients.

The following points should be considered when discussing the case history:

- history of exposure to a miotic or mydriatic agent to rule out pharmacologic mimics of HS (common agents that affect the pupil are pilocarpine, brimonidine, belladonna, scopolamine patches)
- balance, hearing, or sensory abnormalities can indicate a first-order neuron lesion
- past trauma or surgeries involving the face, head, neck, shoulder, or back may indicate a second-order neuron lesion
- headache, diplopia, or pain can indicate a third-order neuron lesion (painful HS must be treated urgently as it is associated with carotid artery dissection, which carries an increased risk of ischemic stroke and mortality)
- presence and location of anhidrosis
- consider previous infections, including herpes zoster, tuberculosis, and syphilis
- the value of examining old photographs to investigate the duration should not be underestimated

During the physical examination, consider:

- which may indicate congenital HS
- reactivity of pupils to light and accommodation (light-near dissociation may indicate an etiology separate from HS, such as...
dorsal midbrain syndrome, Argyll-Robertson syndrome, Adie’s tonic pupil)

- measurement of ptosis and consider other causes of ptosis: cranial nerve 3 palsy, levator dehiscence, myasthenia gravis
- vision, including color vision and visual fields
- must differentiate other causes of anisocoria such as physiologic anisocoria, trauma, Adie’s tonic pupil (Figure 2)

**Pharmacological testing in-office**

Any combination of clinical signs of HS should be confirmed with diagnostic pharmaceutical agents in-office. These agents can aid in localizing the lesion responsible for causing the HS.

*Topical cocaine:* Cocaine acts as an indirect sympathomimetic agent, inhibiting the re-uptake of norepinephrine from the synaptic junction of the post-ganglionic fibers and the iris dilator muscle. Cocaine solution (ranging from 2-10%) is instilled into both eyes. Both pupils are evaluated after at least 30 minutes. If the sympathetic innervation is disrupted, norepinephrine cannot be released from the nerve terminal, and topical cocaine produces less pupillary dilation than in the normal eye. Anisocoria of 0.8 mm or more is diagnostic for HS.

Disadvantages of using cocaine include inability to localize the lesion, high cost to compound, detection in urine, limited availability, and strict policy in most medical facilities on regulating controlled substances.

*Topical apraclonidine:* This is the test of choice due to practicality and high sensitivity. It is commercially available as Iopidine 0.5-1% and is indicated to lower IOP in glaucoma patients. It can be used off-label for HS due to its weak alpha 1-adrenergic and strong alpha 2-adrenergic activity. The upregulation of alpha 1-receptors in HS results in denervation supersensitivity such that the affected pupil has greater mydriasis compared to the normal eye. The normal pupil is unaffected or minimally constricts due to the weak alpha-2 adrenergic effect.

When apraclonidine is used prior to glaucoma laser surgery, the protocol is to instill one to two drops prior to the procedure. The effect of lowering IOP can be seen within the hour. However, the use of apraclonidine in confirming HS is an off-label use of the medication. A definitive protocol has not been established. The amount of time required for the maximum mydriatic effect on an affected pupil has not been well-studied. In addition, incomplete lesions of the sympathetic supply can produce mydriasis of the affected pupil without resulting in reversal of anisocoria, making it difficult to establish a definitive diagnosis.

To illicit maximum response of the affected eye while minimally affecting the unaffected eye, practitioners may instill more than one drop of apraclonidine. The most common protocol in clinical practice is:

1. instill 1 drop of apraclonidine 0.5-1% solution into both eyes (or 2 drops 5-10 minutes apart)
2. evaluate both pupils after 30-60 minutes

If the result is reversal of anisocoria where the affected pupil dilates while the normal pupil remains unchanged or constricts, this is confirmatory for HS (Figure 3). In some studies, topical apraclonidine has been shown to be superior to topical cocaine in detecting HS.

Disadvantages to using topical apraclonidine include the inability to localize the lesion and limited effectiveness in acute HS as the denervation sensitivity takes 5-8 days to develop.

*Topical hydroxyamphetamine:* This agent is commercially available as Paremyd. The test is helpful in differentiating between pre- and post-ganglionic lesions. Hydroxyamphetamine causes the release of endogenous norepinephrine from the post-ganglionic axons into the neuromuscular junction of the iris dilator muscle. Two drops of 1% hydroxyamphetamine solution are instilled into each eye and the pupils are evaluated after 1 hour. Dilation of both pupils indicates a lesion of the first neuron or second neuron. If the smaller pupil fails to dilate, it indicates a lesion of the third-order neuron or post-ganglionic neuron.

The disadvantage to using topical hydroxyamphetamine is the need to wait 24-48 hours after cocaine testing.

**Imaging**
Radiological imaging plays an important role in the diagnosis of the underlying cause of HS and ruling out pathologies that carry a substantial risk of morbidity and mortality (Figure 4). The provider should consider the sensitivity and specificity of the imaging technique, its availability, and the patient’s health status (kidney function when considering contrast media, ionizing radiation emitted during the procedure, etc.) when ordering a radiological imaging study. Studies should be ordered based on urgency of the symptoms and localizing signs. Timely diagnostic imaging is crucial to determining the primary cause of HS and directing further management of the underlying condition.

When HS can be localized to the first-order neuron with associated neurological symptoms (ataxic hemiparesis or upper limb paresthesia), magnetic resonance imaging (MRI) of the brain and cervical and upper thoracic spinal cord should be performed. When the HS can be localized to the second- or third-order neuron, the computerized tomography angiogram (CTA) protocol should include the Circle of Willis and the orbits, down to the level of the aortic arch. These images process the head, neck, and lung apices. Patients with third-order neuron or painful HS, with or without localizing signs, should have CTA protocol emergently. HS without localizing signs, pain, trauma, or history of malignancy does not require urgent imaging. CTA protocol should be performed within 6 weeks.

Treatment/management

Treatment options are based on the diagnosis and management of the underlying cause of HS. Timely diagnosis and referral to an appropriate specialist is of critical importance. This requires collaboration with an interprofessional healthcare team which may include:

- optometry
- ophthalmology (neuro-ophthalmology and/or oculoplastics)
- primary care
- pulmonology
- neurology
- neurosurgery
- vascular surgery
- radiology
- oncology

Differential diagnosis

The clinician must consider the extensive number of other potential causes of anisocoria and/or ptosis. Common causes that may present to an optometric practice and how their characteristics differ from that of HS are explained below.

Anisocoria

- Physiologic anisocoria: Anisocoria is seldom greater than 0.8 mm. The difference in pupil size should remain equal in dim and bright illumination. The anisocoria remains stable over time.
- Third-nerve palsy: This condition may cause pupillary dilation, although isolated pupillary dilation is not classically observed in third-nerve palsies. If pupillary dilation is seen in third-nerve palsies, it is usually accompanied by extraocular motility restriction and upper lid ptosis. Extraocular motility restriction is not seen in HS.
- Traumatic mydriasis: The patient may recall a history of trauma or surgery in the eye. Damage to the iris sphincter may be visualized in the slit lamp. The pupil may be irregular in shape due to sectoral damage of the iris sphincter muscle. The pupil in HS is typically round and without iris sphincter damage.
- Pharmacologic mydriasis: The patient should be questioned about the recent use of vasoconstrictors (including antihistamines, decongestants, and stimulants), scopolamine patches, and herbals (such as jimson weed and belladonna). If diluted pilocarpine fails to constrict the pupil, the pupil is pharmacologically dilated.
• Adie’s pupil: The dilated Adie’s pupil demonstrates light-near dissociation, where the pupil response is better to an accommodative stimulus than to light.\textsuperscript{21} Light-near dissociation is not present in HS.

Ptosis\textsuperscript{22}

• Mechanical ptosis: This can be due to mechanical disturbance of the upper eyelid from causes such as tumors, infections, or inflammation. Levator dehiscence due to long-term rigid gas permeable contact lens wear should also be considered.\textsuperscript{22,23}
• Myogenic ptosis: This results from the weakness of the levator palpebrae, which can be caused by various conditions including congenital myogenic ptosis, progressive external ophthalmoplegia, myotonic dystrophy, thyroid myopathy, or inflammation related to orbital myositis.\textsuperscript{22}
• Myasthenia gravis: Variable ptosis, which may or may not include diplopia, can be related to an autoimmune disorder in myasthenia gravis. Ice test can be performed in-office to confirm the diagnosis. A positive result is noted if the eyelid rises 2 mm immediately after ice is applied for 2 minutes. A lab test for antibodies against the acetylcholine receptor can also be performed.\textsuperscript{22}

Critical-thinking concepts

The purpose of this case report is to offer optometry students, residents, and eyecare providers a review of HS and a practical guide to investigate the cause of the condition. A discussion can be led with optometry students and residents in a classroom or clinic setting. Case history and exam findings should be presented in an organized manner (i.e., anterior segment findings to posterior segment findings). Critical-thinking should be encouraged to rule out other causes of anisocoria and work through differential diagnoses. A practical guideline to administer and interpret diagnostic testing, along with guidance on how to order subsequent radiologic studies should be reviewed. The importance of effectively communicating the assessment and plan to the patient should be emphasized.

The patient in this teaching case report presented with a generalized complaint of irritated eyes. While taking the patient’s case history, gross observation revealed an upper lid ptosis of the right eye. Therefore, case history questions were re-directed with the ptosis in consideration. The provider was able to elicit an acute history of right upper lid ptosis, which began 4 days after carotid endarterectomy on the right side about 1 month prior. The carotid endarterectomy procedure was performed 1 year prior on the contralateral side. The case history is ever-evolving and continues throughout the exam.

Due to the patient’s recent surgical history and upper lid ptosis, the pupils were carefully assessed. With the anisocoria being greater in the dark, HS was suspected and ultimately confirmed with topical apraclonidine 1% instilled in-office. The clinician should be able to differentiate other pupil anomalies that may present as anisocoria. The clinician should also be able to evaluate pupillary response in light and dim illuminations and be comfortable assessing light-near pupillary dissociation.

Due to high suspicion of iatrogenic HS, non-emergent radiologic imaging studies were ordered. MRI of the brain and brainstem and MRA of the head and neck were ordered to rule out other potential causes and assess the patient’s risk of stroke. The clinician should be aware of the potentially serious systemic effects of HS. The patient’s cardiologist and vascular surgeon were alerted of the condition, and the patient was instructed to follow-up with his healthcare team.

Conclusion

This teaching case report is intended to provide a comprehensive review of Horner syndrome for optometry students, residents, and eyecare providers. As frontline eyecare providers, it is important for optometrists to recognize the signs of HS and be proficient in using diagnostic pharmaceutical agents to make the diagnosis in-office. Furthermore, it is important to rule out more sinister causes of ptosis and anisocoria, especially if associated with pain. It is critical to assess the urgency with which radiologic imaging should be performed and work as part of a team of healthcare professionals to provide a comprehensive approach for managing the care of the HS patient.

References

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In my tenure as Editor of Optometric Education, I have never been as compelled to pursue a topic in this editorial as I am to pursue the topic of student academic entitlement. Since my previous editorial on the subject,1 I have spoken with numerous faculty and administrators in various graduate and undergraduate settings who overwhelmingly expressed frustration and anger about the issue as well as their perception of a lack of administrative support.

Causes and Effects

My previous editorial touched on several aspects of student entitlement, including how it has been defined, probable causes — a repercussion of narcissism and a shift toward the idea of student as consumer — and consequences.1 To the list of potential causes, Rinsley adds the theory that entitlement is related to the failures of family, schools and government to prepare young people for the responsibilities of being part of a society and a result of growing threats to attaining future goals, diminishing purchasing power and uncertainties related to the environment, world stability or financial success.2 These factors, he suggests, led to a model of instant gratification with a theme of "success without effort and income without productivity."2

As far back as 1986, Dubovsky pointed to the student-as-consumer mindset when he reported what he observed as five features of entitlement in medical education: 1) "Knowledge is a right that should be delivered with minimal exertion and discomfort on the part of the consumer." 2) "A passivity associated with the expectation that others will provide all the education that will be necessary." 3) "Problems in learning are due to the inadequacies of the teacher, the course, the system, rather than the student’s own shortcomings." 4) "One should receive equal recognition and reward regardless of individual effort and ability." 5) "The entitled student is justified in feeling good by making others feel bad, for example by addressing grievances through hostile and disrespectful behavior. Discussion of the student’s behavior is thought to generate stress."3

Others have described the student/consumer concept as a belief held by some students that because they are paying for their education, they deserve to be treated as consumers.4,5 Further, as consumers, they expect to be able to participate in the education process according to their preferences and be catered to.5

The negative effects of student entitlement are many and include grade inflation, disruptive student behavior in the classroom, faculty changing their teaching methods in ways they would rather not, and decreased faculty morale.4 Additionally, students’ expectation of personal excellence may decline, especially if it involves personal sacrifice.5 This is compounded by the avoidance of any situation perceived as difficult or anxiety-producing.3

I have personally encountered student entitlement several times in the past year. On multiple occasions, students who had missed a deadline and consequently lost points emailed me to explain and request the points. While their emails were respectful and polite, and I was sympathetic to their circumstances, I explained why it was important to stick to the criteria outlined in the syllabus. The repeated emails and personal requests made the temptation to concede difficult to resist. Frankly, the situation felt emotionally exhausting.

How Can We Approach the Current Reality?

I found Dubovsky’s 1986 description of student entitlement applicable to today. It is interesting that he reported on student entitlement that long ago, yet most of the recently published papers on the subject associate its characteristics with the Millennial generation (born 1981-1996) and Gen Z (born 1997-2010). In 1986, Millennials were very young or not born. It may be that changes in the world and in academia have empowered students in ways not possible in the 80s, making our experience with entitlement more pronounced. Technology, which enables on-demand communication, information and social connectivity,
has likely contributed to the desire for instant gratification and made the entitlement mindset more common. Also, tuition at colleges and graduate schools has increased, and many institutions are tuition-dependent. In many instances, previous education experiences have set a precedent of supporting student demands, which further enables entitlement.

While it is potentially informative to ponder how the problem rose to this level, the reality is we must deal with the current cohort of students who have a growing sense of academic entitlement. Certainly paying tuition does not buy students the right to dictate to faculty and administrators the course of the education experience and ultimate granting of a degree. Faculty and administrators should be viewed as the experts in both the content and delivery of education. But why do faculty and administrators feel pressure to give way to the demands of entitled students? In addition to the emotional stress, faculty face the threat of unwarranted negative course evaluations from students, and administrators likely fear negative public reviews of their institutions and have concerns about the sustainability of programs and ability to recruit the most qualified students.

Students, most of whom take their responsibilities seriously, are an important source of information regarding their education experience. However, the view that anything that makes learning difficult is an unfair imposition should not be tolerated. Students need to find a way to support and work with students while maintaining a high level of expectations and requirements. Giving in to all student demands is not a responsible approach and sends a message that entitled attitudes and behavior are OK.

The journal welcomes hearing about your experiences with student academic entitlement and any recommendations you have for dealing with this important issue.

References

1. Denial A. Student academic entitlement. Optometric Education. Fall 2022;48(1).