Non-surgical vitreolysis of vitreomacular traction

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Background: Progressive hyaloid liquefaction typically results in posterior vitreal detachment. However, persistent vitreoretinal adhesions may result in pathological traction. In particular, progression of vitreomacular adhesion (VMA) may lead to the development of a macular hole. The standard treatment of VMA is vitrectomy. Recently, non-surgical vitreolysis has been utilized to dissolve persistent vitreoretinal adhesions, induce posterior vitreal detachments and possibly promote non-surgical closure of macular holes.

Case Summary: A 71 year-old male presented to the Optometry Service for examination. Ocular history was significant for retinal photocoagulation of proliferative diabetic retinopathy in the right eye. Upon examination, best-corrected visual acuities were 20/40 and 20/25 for the right and left eyes, respectively. Pupillary function was intact. Both eyes demonstrated non-proliferative diabetic retinopathy. The left eye demonstrated persistent vitreomacular traction resulting in tenting of the fovea. Additionally, the active traction may aggravate the diabetic eye disease. The patient underwent non-surgical vitreolysis with ocriplasmin. The intervention resulted in reduction of vitreoretinal adhesion and partial alleviation of vitreomacular traction.

Conclusion: Vitreomacular adhesion may result in abnormal traction and macular hole formation. Recent investigations suggest that persistent vitreomacular traction may contribute to the development of diabetic macular edema. Ocriplasmin demonstrates protease activity against fibronectin and laminin and promotes lysis of vitreoretinal adhesions. This poster reviews recent investigations examining the contribution of VMA with macular edema. The mechanism of action, clinical indications and safety profile of ocriplasmin are discussed. Moreover, the findings of the Microplasmin for Intravitreous Injection – Traction Release without Surgical Treatment (MIVI-TRUST) trial are reviewed.

“Will I see to drive?” A Case of Goldenhar Syndrome with epibulbar dermoids fit into a hybrid lens

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Background: This case discusses the conjunctival and corneal profile challenges in fitting a hybrid lens for a teenager with Goldenhar Syndrome with limbal dermoids post resection, irregular astigmatism and wanting to see to drive.

Case Summary: A 15 y/o WF is referred for a corneal prosthetic device secondary to irregular astigmatism and remnant epibulbar dermoids at the limbus. Ocular history is DES, irregular astigmatism, limbal dermoid resection surgery OU. Medical History is Goldenhar Syndrome, Choroid plexus papilloma, heart murmur. Surgical History is jaw reconstruction, choroid plexus tumor removal, epibulbar dermoid removal. Medication is gentecal. Previous treatments tried were only spectacles with BCVA being 20/50- OD, 20/40- OS. The cornea had scaring haze inferior and conjunctiva had epibulbar dermoids at the limbus inferior OU and OD superior temporal with cilia; Differential diagnoses were irregular astigmatism, amblyopia. The working diagnosis was irregular astigmatism that varied with
pentacam measurement making Keratometry readings unreliable. A bitoric lens was considered or a soft toric resting over the dermoids. The concern with a corneal prosthetic device to mask the corneal cyl was dermoid tissue elevation impacting the landing area of lens edge. Using a mini scleral would potentially cause impingement or irritation; a hybrid soft skirt resting on it would conform better without impingement. Initial treatment was fitting soft toric lens with good comfort but poor vision. The next treatment option was fitting an RGP bitoric 9.0 diameter which was uncomfortable and did not fit well needed smaller diameter. A ClearKone hybrid vaulted over the cornea and rested nicely on the tissue; VA 20/25-2 OU.

**Conclusion:** It is important to account for the topography of the conjunctiva and cornea when fitting contact lens designs. Conjunctival and corneal tissue irregularities affect lens choice. Prosthetic corneal devices like hybrids can vault the cornea and also land softly on irregular conjunctivas. Hybrid lenses should be prime management for correcting irregular astigmatism with uneven corneal and conjunctival surfaces that a mini scleral lens would impinge.

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**Hemi-central Retinal Vein Occlusion in a Patient with Undiagnosed Systemic Hypertension**

*Erin Mosellen, O.D., Kelly Thompson, O.D., Cincinnati VA Medical Center, Cincinnati, Ohio*

**Background:** A 66-year-old Caucasian male with prostate cancer and gout presents with a sudden decrease in visual acuity and onset of distortion, OD. Fundus exam reveals an inferior hemi-central retinal vein occlusion.

**Case Summary:** The patient had been treated for POAG, OU for 30 years and current ocular medications included Latanaprost QHS OU, Brimonidine BID OU, and Timolol BID OU. BCVA was 20/40 OD (PHNI) and 20/20 OS. Entrance testing was unremarkable with no APD OD, OS. IOPs were 24mmHg and 22mmHg OD, OS. DFE revealed diffuse hemorrhages, venous dilation, and mild edema across the inferior hemi-field with moderate microaneurysms, exudates, and edema at the macula, OD. Fundus photos were taken. Macula OCT testing showed moderate sub-foveal cystic edema, OD. Gonioscopy revealed scleral spur 360°, OU with no NVA, OD. The patient’s last blood pressure reading was 139/70 in office; however, he reported a stable reading of 150/80 with home monitoring and a history of untreated “borderline” hypertension. Bloodwork and lipid panels were ordered that day along with a carotid doppler and cardiac echocardiogram to rule out carotid stenosis and a cardiac source of an embolus, respectively; all testing was within normal limits. Hemi-central retinal vein occlusion (hemi-CRVO) affects the superior or inferior half of the retina and occurs due to an anatomical variation of the central retinal vein (CRV) in which a dual trunked superior and inferior retinal vein exists in the anterior portion of the optic nerve head [1]. This congenital anatomy of the CRV is present in approximately 20% of the population [4]. When either the superior or inferior trunk of the dual-trunked CRV is blocked, a hemi-CRVO occurs. This leads to venous dilation, tortuosity, and hemorrhages in the corresponding half of the retina, along with possible retinal and macular edema [1]. Optic disc edema and disc collaterals may be present upon initial exam as well. It occurs with the greatest frequency in patients over the age of 65 and more commonly in males [2,3,4]. Patients with hypertension, diabetes mellitus, and glaucoma are at a greater risk for developing a hemi-CRVO [2,3]. Hemi-CRVOs are classified as either non-ischemic or ischemic. Non-ischemic hemi-CRVOs occur more frequently and are associated with better visual acuity, absence of an APD, and relatively normal HVF results. Visual prognosis is worse in the ischemic form due to a higher incidence of neovascularization (NVI, NVA,
NVE, NVD), persistent macular edema, capillary non-perfusion, and/or vitreous hemorrhage [1]. There are two goals to treatment: (1) treat the underlying systemic or ocular condition(s) in order to prevent re-occurrences and serious systemic sequeale and (2) treat complications such as persistent macular edema and/or neovascularization. Fluorescein angiography should be used to classify the condition as ischemic or non-ischemic, if not evident based off of the clinical presentation [1]. OCT macular testing is useful to identify and monitor the level of macular edema, and gonioscopy must be performed to monitor for the development of NVA. It is crucial to follow the patient every month for the first six months and to perform DFE, OCT-macula, and gonioscopy testing at each visit. If neovascularization is present, the patient should be referred for PRP; if macular edema is persistent or worsening, anti-VEGF injections or FRP should be done.

Conclusion: Hemi-CRVO is a distinct type of venous occlusion, which occurs only in patients with a congenital dual-trunked CRVO. It is important to communicate with the patient’s PCP to monitor for associated systemic risk factors, to follow-up routinely, and to use appropriate clinical testing. Early detection and treatment is critical to ensure the best possible clinical outcome.

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Evaluation of CLEAN diet on Cholesterol Levels
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Introduction: This pilot study aimed to examine the effects of the CLEAN diet on total cholesterol, HDL, LDL and triglyceride levels. The book CLEAN by Dr Alejandro Junger was published in 2012. CLEAN is a three-week detoxification program that excludes consuming all dairy, glutinous grains, eggs, pork, beef, soy, processed oils and sugar, alcohol, and caffeine. Other foods that are eliminated include those that are more prone to exposure to pesticides, provoke allergic responses or promote inflammation. Approved foods include organic fish, lean chicken, wild game, organic fruits and vegetables, non-gluten grains, olive oil, herbal tea, coconut water, dried fruit, nuts and seeds. (Junger, 2012) Liquid meals are consumed for breakfast and dinner to allow the digestive system more time to detoxify the body of accumulated metabolic waste. There are countless testimonials of participants feeling a greater sense of wellbeing, having clearer skin, less brain fog and bloating. The intention of this study was to show improved blood cholesterol quantifiable to support the subjective participant findings.

Methods: Five yoga studio participants, both male and female, all Caucasian, with ages ranging from 38-53, agreed to complete the CLEAN program. They privately obtained cholesterol testing before starting and after completing three weeks of the CLEAN program and agreed to supply the results. The volunteers supplied demographic information, signed an informed consent and completed a food frequency questionnaire (Fred Hutchinson Cancer Research Center, 2014) at the beginning and end of the program to document food choices. Each participant had a copy of the CLEAN manual that included a list of the approved and excluded foods. Each week all members met to discuss any successes, struggles or asked any questions for community support. The total cholesterol and cholesterol ratio was determined for each participant for the pre and post-program results.

Results: Total cholesterol decreased in three of the five participants. Total cholesterol was determined by adding the LDL+HDL+20% of triglycerides. (American Heart Association, 2014) Triglycerides decreased in two participants. LDL decreased in four participants while HDL increased in all five study members. The cholesterol ratio improved with every participant. The cholesterol ratio was determined by dividing total cholesterol by HDL. (Behrenbeck, 2012) The optimum ratio determined by the
American Heart Association is 3.5 or less. (American Heart Association, 2014)

**Conclusion:** This study was intended to establish quantifiable results of improved blood cholesterol to support the subjective participant findings of wellbeing at the conclusion of the study. Much research has been published correlating a high-fat diet with increased cholesterol that increases the risk for Coronary Heart Disease. The American Heart Association stated that a diet high in saturated and trans fats raises LDL cholesterol. (American Heart Association, 2014) The CLEAN eating plan allowed for mono and polyunsaturated fats which can decrease the risk for coronary heart disease (Willett, 2012) and increase HDL levels. (Mensink, 2003) For this study, not all of the participants total cholesterol decreased. However the HDL ratio increased in all participants, and the cholesterol ratio was improved for all participants. Obtaining a change in cholesterol in just three weeks of altered eating is surprising and does validate participants improved feeling of vitality. While the study of five is small, based on the results, further studies could be warranted with a larger population size with more intervals of cholesterol monitoring.

**Empty Sella Syndrome and Visual Field Defects**

*David Cartwright, O.D., Jacqueline Chang, O.D., Mark Bampton, O.D., Kelly Thomann, OD., VA Hudson Valley Health Care System, Montrose, New York*

**Background:** Empty sella syndrome (ESS) represents a condition associated with partial or complete absence of the pituitary gland. In ESS, the pituitary gland shrinks or becomes flattened and cannot be visualized within the sella turcica when imaged. ESS has historically been divided into primary (with no known antecedent cause) and secondary (due to an identifiable etiology) subtypes. Various visual field defects have been described for both types of ESS, however they are more commonly encountered in secondary ESS. This poster will discuss visual field defects which can be associated with empty sella syndrome, as well as the clinical findings responsible for the visual field defect in this particular case.

**Case Summary:** A 59 year-old male presented for visual field testing following diagnosis of empty sella syndrome. His visual field revealed a repeatable dense inferior quadrant defect in the right eye only, which respected the vertical mid-line. The left visual field was full. The patient had small, crowded optic discs. Optical coherence tomography of the optic nerves revealed significant superior thinning of the retinal nerve fiber, right eye.

**Conclusion:** Empty sella syndrome is an uncommon anatomic condition which is typically diagnosed after neuro-imaging and can lead to neurologic visual field defects. This poster will review imaging findings characteristics of this syndrome. Visual field testing is useful in order to quantify associated visual field loss. Moreover, additional ophthalmic testing that is useful to rule out or diagnose other clinical entities that require medical intervention will be discussed.

**Talc Retinopathy: A Case Report**

*Mary Vivirito, O.D., Paula Matsuno, O.D., Sally Dang, O.D., VA Long Beach Health Care System, Long Beach, California*

**Background:** Substance abuse remains an area of concern for the Veteran population. While prescription medication abuse is high among recent Iraq and Afghanistan veterans, veterans of earlier campaigns are likely to have abused illicit substances such as cocaine. Patients may not always disclose their substance abuse history due to the associated stigma, especially if they have claimed sobriety for several years. Regardless of sobriety length, the effects of drug abuse can have long term effects that
health care professionals must address in order to offer comprehensive care. The following case illustrates the importance of obtaining a complete medical history that includes past and present substance abuse history.

**Case Summary:** 72 year old African American male referred for eye exam by primary care provider. Last eye exam was 2 years ago outside of the VA system. Patient lost his glasses and reports blurry vision without them, otherwise no ocular complaints. Medical history is significant for COPD and hypertension. Family ocular history is unremarkable and patient reports no history of ocular disease, injury or surgery to the eyes. Refractive error reveals hyperopia, astigmatism OS>OD, and presbyopia with BCVA 20/20 OD and 20/40 OS. Patient reports progressive decreasing vision OU but has not noticed difference between the 2 eyes. Anterior segment is unremarkable except for mild cataracts OU. Relevant findings from dilated fundus exam include asymmetric nerve cupping OS>OD, macular mottling, macular atrophy OS only (likely contributing to decreased visual acuity OS) and talc retinopathy OU. Upon further questioning, patient admits to previous recreational drug usage including cocaine and heroin. He reports his last time using was 20 years ago. Duration of drug abuse is unknown. Talc retinopathy falls under the category of crystalline retinopathies. Talc is used as inert filler in methylphenidate hydrochloride tablets (such as Ritalin), which are then crushed for intravenous drug use. Since the drug fillers are not completely metabolized, they remain in the body indefinitely. If intravenously injected it will travel through the venous system into the lungs where the particles can become trapped in the pulmonary capillary beds. Blood flow will then continue around collateral pulmonary vessels and eventually to the eye where the microtalc particles become trapped in the capillaries of the retina. The particles are yellow and refractile in appearance and can be observed intravascularly and extravascularly in the retina. It is possible that the deposits may cause retinal non-perfusion and neovascularization. In some instances macular edema, macular pucker, macular fibrosis, cotton wool spots, and vitreous hemorrhages may occur. It is important to monitor these patients for signs of non-perfusion and neovascularization. Treatment of neovascularization may include pan retinal photocoagulation or anti-VEGF agents. Patient education in regards to changes that have occurred secondary to previous drug abuse were discussed. Fundus photos were taken for documentation and appearance will be monitored annually. Patient will be returning for fluorescein angiography to evaluate possible non-perfusion in the perimacular and paramacular region given reduced acuity OS. He will be continually monitored for evidence of neovascularization, hypoxia and visual changes from our baseline exam. In addition he will return for visual field and glaucoma work up due to asymmetric nerve appearance in several months’ time.

**Conclusion:** Relevant history of drug abuse was not included in patient’s medical history. Only upon retinal exam findings was this information elicited. Although patients may not volunteer this information on their own accord, as eye care providers we may prompt a more complete medical history through our own thorough examination. Visual changes may occur even years after abuse has stopped, therefore careful monitoring of retinal vasculature appearance is indicated at every exam and proper referral ordered as necessary.

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**Treatment Paradigms of Macular Holes**

Meaghan, O.D., Kelly Thompson, O.D., Cincinnati VA Medical Center, Cincinnati, Ohio

**Background:** Macular holes most commonly arise as idiopathic breaks in the retina, located at the fovea. The exact pathogenesis is not fully understood, but is believed to be attributed to the anteroposterior and tangential traction exerted by the posterior vitreous...
cortex at the perifoveal region. Macular holes typically present in individuals over the age of 60 and are more common in females. The most common visual symptoms include metamorphopsia and decreased acuity.

**Case Summary:** A 79 year old male presents for a generally eye exam with complaints of difficulty with near vision over the last month; stable distance vision. Systemic history was remarkable for hypertension, GERD, and osteoarthritis. His ocular history was unremarkable. Corrected visual acuity was 20/400 OD, no improvement with pinhole, and 20/20 OS. Biomicroscopy was unremarkable, OU. Fundus examination revealed a large macular hole, OD with a surrounding fluid cuff and drusen-like deposits at the base of the hole. OCT confirmed a stage 3 macular hole. The patient was examined with a retinal specialist and referred for a vitrectomy.

**Conclusion:** The aim of this poster is to describe the clinical features of a macular hole and modern treatment options. Currently, a vitrectomy is most commonly performed with or without an ERM/ILM peel. This poster will discuss current research on the advantages/disadvantages of performing the ERM/ILM peel and whether vital dyes should be used in this process. Additionally, patient positioning after surgery will be discussed in detail. The standard of care has been to instruct patients to remain face down for one to two weeks post-surgery Current research suggests that limited to no face down positioning maybe be acceptable. The information presented will be useful in determining when to refer, and how to best educate our patients.

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**Atypical Visual Symptom Presentation in Autosomal Dominant Retinitis Pigmentosa: A Case Review**

John Koehler, O.D., Mark Bampton, O.D., Jennifer Kehm, O.D., Jean Jung, O.D., M.S., VA Hudson Valley Health Care System, Montrose, New York

**Background:** I. Case History -46 year old white female-CC: She is clumsy and does not see things in periphery as well as she used to-Ocular History-“Early RP changes” noted in 1997; intermittent mention of RP over comprehensive exams since 1997 without mention of any subjective symptoms or documented visual complaints-Family Hx: macular degeneration (PGM) and retinitis pigmentosa (father, aunt, PGF)- Pt Medical Hx: unremarkable- No active medications - Other salient info: Reports poor night vision and difficulty driving after sundown

II. Pertinent findings -Subjective complaints: Difficulty navigating and difficulty playing basketball-CVF:Slightly reduced in superior fields, OU -DFE/Fundus evaluation: Vessel attenuation and mid-peripheral bone spicules, concentrated in the inferonasal quadrant, OU. -HVF (24-2 SS): Dense ring scotoma in superior quadrant only, respecting the midline, OU. Glaucoma vs RP.

**Case Summary:** II. Pertinent findings -Subjective complaints: Difficulty navigating and difficulty playing basketball -CVF: Slightly reduced in superior fields, OU -DFE/Fundus evaluation: Vessel attenuation and mid-peripheral bone spicules, concentrated in the inferonasal quadrant, OU. -HVF (24-2 SS): Dense ring scotoma in superior quadrant only, respecting the midline, OU. Glaucoma vs RP.

**IV. Diagnosis and discussion -Retinitis Pigmentosa:** Visual symptom presentation generally presents in 2-3rd decade of life, she has no symptoms until mid-4th decade. - Elaborate on the condition and expound on unique features -Associated systemic conditions and evidence of AD inheritance pattern.

**Conclusion:** V. Treatment/ Management -Review treatment options, discuss genetic counseling -Vitamin A therapy: risk vs benefit -Bibliography and literature review VI. Conclusion -Discuss clinical pearls and takeaways with emphasis on pt education, genetic counseling and palliative management of condition. --Pt has AD RP and irregular presentation of being non symptomatic until 4th decade indicates a mild form of the condition. Her VF 24-2 SS defects
are also unique in that they respect the midline, which RP VF defects generally do not.

**CNVMs: When to Suspect Polyploidal Choroidal Vasculopathy**

*Kristen Buskirk, O.D., Kelly Thompson, O.D., Cincinnati VA Medical Center, Cincinnati, Ohio*

**Background:** Compared to other retinal vascular conditions, Polyploidal Choroidal Vasculopathy (PCV) is a newly described and investigated disease process. In 1982 Yannuzzi first described a “peculiar hemorrhagic disorder of the macula involving serosanguinous detachments of the retinal pigment epithelium and neurosensory retina”. It was not until the 1990s that the disorder Yannuzzi identified was given the name Polyploidal Choroidal Vasculopathy and its clinical characteristics were established using dilated fundus exams, indocyanine green angiography (ICGA), and OCT. The fundus exams of patients with this condition revealed subretinal red-orange polyploidal vascular dilations (polyps) of the choroidal vasculature seen most often in the peripapillary region. ICGA and OCT reveal vessel changes and location in the choroid. Since the characteristic polyps are difficult to visualize during a typical fundus exam, the diagnosis of PCV is often misdiagnosed as central serous retinopathy or idiopathic. It is important to correctly diagnose the source of a choroidal neovascular membrane (CNVM) especially in the case of PCV to ensure the most efficacious treatment options are considered.

**Case Summary:** A 62 year old Caucasian male presented complaining of problems with halos, glare, and blur that had progressively worsened over the past two months, OU. The patient’s medical history was positive for Diabetes Mellitus Type 2 without complications, hypercholesterolemia, asthma, and prostate hyperplasia. His ocular history was unremarkable. Best-corrected visual acuity (BCVA) was 20/40 OD, OS. All exam findings were unremarkable besides the DFE that revealed mild drusen, few scattered microaneurysms, and central macular subretinal fluid, OU. OCT demonstrated bilateral subretinal fluid that extended nasally from the fovea. The fluorescein angiogram (FA) showed areas of RPE mottling and window defects temporal to the optic nerve OU, trace late leakage OD, and moderate late leakage OS, suggestive of a CNVM. The patient was diagnosed with bilateral central serous retinopathy with a CNVM OS. Treatment with monthly intravitreal Lucentis injections was initiated OS while OD was to be monitored. Two rounds of monthly intravitreal Lucentis were injected OS until on the third visit when the intraretinal hemorrhages had resolved OU and OCT showed more improvement OD and almost full resolution of subretinal fluid OS. At that point no more treatment was recommended and the patient was scheduled to return in two months for ICGA and OCT. ICGA demonstrated few focal polyps of hypercyanescence inferonasal to the macula and PCV was diagnosed. Exam findings have been stable for twelve months without further treatment. The patient’s visual acuity continued to improve each visit with the most recent BCVA of 20/20 OD and 20/25 OS. The OCT revealed that mild chronic subretinal fluid remained OU but there were no active CNVMs so no treatment was indicated and the patient is to be monitored for changes in four months.

**Conclusion:** This case illustrates how PCV can often be mistaken for other causes of CNVMs such as CSR or ARMD. Epidemiological studies suggest that PCV should be considered in cases of unilateral choroidal neovascular membranes in younger patients of darker pigmented races, however, the patient in this case does not match those demographics. PCV should be considered and ICGA should be performed in cases where choroidal neovascular membranes are located in the peripapillary area. It is important to correctly diagnose the source of a CNVM especially in the case of PCV because additional treatment options other than intravitreal anti-VEGF should be considered. In
this case, treatment with intravitreal Lucentis has stabilized the patient’s PCV choroidal neovascular membrane for almost eighteen months but verteporfin PDT should be considered in patients that do not respond to intravitreal anti-VEGF or are unable to return to the office on a monthly basis for injections.

**Vitreopapillary Traction**

Jennifer Kehm, O.D., David Cartwright, O.D., Mark Bampton, O.D., Becky Forman, O.D., Nancy Wong, O.D., Ph.D., VA Hudson Valley Health Care System, Montrose, New York

**Background:** Vitreopapillary traction represents a recently recognized syndrome characterized by fibrocellular proliferation or incomplete posterior vitreal detachment resulting in traction on the optic disc. Vitreopapillary traction has been proposed to result in damage to the anterior optic nerve through decreased axoplasmic flow and/or mechanical interference of perfusion to the short posterior ciliary arteries. Ocular manifestations of the traction may include intrapapillary and peripapillary hemorrhages, “pseudopapilledema”, perimetric defects and vision loss. Spectral domain optical coherence tomography (OCT) facilitated initial recognition of the clinical entity via enhanced visualization of the vitreal interface at the optic disc. In particular, OCT allows visualization of a hyper-reflective band extending from the vitreal cortex to the optic disc and increased thickness of the peripapillary nerve fiber layer.

**Case Summary:** A 59 year-old male presented to the Optometry Service for examination. Best-corrected visual acuities were 20/20 for the right and left eyes respectively. Pupillary function was intact. For the right eye, dilated fundus examination demonstrated an optic nerve with ill-defined borders and an associated peripapillary hemorrhage. OCT demonstrated a hyper-reflective band representing hyaloid adhesion and attachment to the optic nerve consistent with vitreopapillary traction. Additionally, a partial posterior detachment was observed in both eyes.

**Conclusion:** Vitreopapillary traction represents a recently recognized entity. OCT imaging enhances the diagnosis of vitreopapillary traction. Moreover, OCT facilities differentiating the “pseudopapilledema” associated with vitreopapillary traction versus disease processes associated with true optic nerve edema requiring urgent intervention. This poster examines the pathogenesis and clinical findings of vitreopapillary traction. Additionally, treatment and management protocols are discussed.

**Rethinking The Diagnosis With The Aid of OCT: Adult Onset Vitelliform Macula Dystrophy**

Jacqueline Change, O.D., Mark Bampton, O.D., Jennifer Kehm, O.D., Jean Jung, O.D., M.S., VA Hudson Valley Health Care System, Montrose, New York

**Background:** Vitelliform macular dystrophy is a genetic eye disorder that can cause progressive vision loss. The early onset form typically occurs in childhood and is known as Best disease. It is inherited in an autosomal dominant pattern and degree of vision loss varies. The adult onset form usually begins in middle age, has a less effect on vision and is often misdiagnosed as age related macular degeneration. Five stages of vitelliform macular dystrophy have been described: previtelliform, vitelliform, pseudophypopyon, vitelliruptive and atrophic. Vision and clinical appearance varies with each stage, however, in both the previtelliform and atrophic stage, vitelliform macula dystrophy can easily be confused with other conditions.

**Case Summary:** We present one case of a patient with adult onset vitelliform macular dystrophy who had been previously diagnosed as having resolved central serous choroidopathy fourteen years earlier. In addition, another case of a patient misdiagnosed with
macula cysts is presented. Following examination with optical coherent tomography, the vitelliform lesions were evident and both patients were properly diagnosed. We will include an overview of vitelliform macular dystrophy and discuss how optical coherent tomography has improved the diagnosis of this condition.

**Conclusion:** Adult Onset Vitelliform Macula Dystrophy is an often misdiagnosed condition. The vision and clinical appearance varies with each stage and often appears very similar to other macula conditions. With the advances in technology, specifically OCT, we may find the incidence of vitelliform macula dystrophy to be higher than previously believed.

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**Atypical Optic Neuropathy with abnormal prodrome and visual field defect**

_Alyssa Fasano, O.D., Alexandria VA Medical Center, Pineville, Louisiana; Richard Frick, O.D, White River Junction VA Medical Center, White River Junction, Vermont_

**Background:** The most common acute optic neuropathy for patients over 50 years old is non-arteritic anterior ischemic optic neuropathy (NAION). Often patients awake with sudden, painless, monocular, visual loss accompanied by disc edema and inferior altitudinal visual field defect. Prodromal symptoms present in 25% of patients and can include amaurosis fugax. While the work-up is extensive to rule-out other concerning etiologies, no known scientific proven effective treatment exists. Clinicians should work to aggressively decrease risk factors to avoid another ischemic event.

**Case Summary:** A 69 year-old Caucasian male presented with one night of visual disturbance seen a month ago. History was negative for vascular disease but positive for colon cancer. Best-corrected visual acuity (BCVA) was 20/20 OD and OS. Pupils were normal with no afferent pupillary defect (APD). Dilated fundus exam was normal and revealed a healthy, but crowded, optic nerve head OU. Two years later, the patient presented with a similar complaint of visual disturbances and blurred vision OS. BCVA worsened to 20/25 OS. The patient had developed an APD OS, and the fundus exam revealed optic nerve head edema OS without hemorrhages. Humphrey Visual Field 30-2 (HVF) was performed showing a normal field OD and significant superior and inferior field defects OS. Color Vision was normal OD, OS. Due to the atypical visual field defect, an exhaustive work-up including blood-work, HVF, carotid duplex, fundus photography, Optical Coherence Tomography, and MRI were performed. All testing came back negative. The final diagnosis was NAION with atypical field loss due to anatomic variation of the optic nerve head vasculature.

**Conclusion:** The presence of an atypical prodrome and visual field defects superior and inferior to the horizontal raphe make this presentation unique. This poster will discuss the clinical features, work up, differential diagnoses and management of NAION.

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**Challenges In Active Status Identification of Macular Toxoplasmosis**

_Mike Stevens, O.D., San Antonio Military Medical Center, Fort Sam Houston, Texas_

**Background:** Ocular toxoplasmosis is caused by an infection from Toxoplasma gondii through acquired or congenital routes. Diagnosis of active status is challenging without appropriate tests. This case details management of active toxoplasmosis following advanced testing.

**Case Summary:** I. Case History• Patient demographics - 26 year old Caucasian male Soldier in the Army. • Chief complaint - Decreased visual acuity in left eye x several weeks. • Ocular, medical history - Unremarkable• Medications - None•Other salient information - House cat II. Pertinent findings• Clinical - Monocular chorioretinitis in affected eye•
Physical - None  
Laboratory - Serological tests III  
Differential diagnosis - Primary/leading - Multifocal choroiditis  
Others - Toxocariasis, syphilis IV  
Diagnosis and discussion - Acquired onset x 3-4 weeks; subtle retinal signs upon initial examination  
Questionable active state from prior retinal exams photos
Results: Positive response to treatment.

Conclusions: Good conclusion to an interesting case of active Toxoplasmosis with preservation of vision to remain within the patients area of the military.

Management of a Corneal Foreign Body via Optical Coherence Tomography

Lindsey Ferris, O.D., Southern Arizona VA Health Care System, Tucson, Arizona

Background: Ocular injuries are common; accounting for 37% of all head injuries resulting in lost work production in the US in 2008. Eye injuries often result from lack of eye protection, or improper use of eye protection. These injuries typically involve abrasions of the cornea and/or corneal foreign bodies and are most commonly seen in young males. Patients typically present to the clinic with symptoms of pain, photophobia, and foreign body sensation. Proper treatment involves removal of foreign particles, prevention of infection, and reduction of pain and inflammation. In cases of metallic foreign bodies, who quickly result in rust deposition, efficient removal of the metal particles is key to proper healing of the corneal tissue. Use of anterior segment optical coherence tomography (OCT) can aid practitioners by providing detailed information for both diagnosis and management. This report highlights how the use of anterior segment OCT can aid clinical management and serve as an excellent tool for patient education.

Case Summary: A 23 yr old Caucasian male presented to the clinic w/ moderate foreign body (FB) sensation, photosensitivity, and epiphora, OS, that began a day prior while performing vehicle work. Anterior segment findings revealed a 1.3mm diameter embedded corneal metallic foreign body with surrounding rust ring and mild edema at 11 o’clock mid-peripherally. Anterior segment Optical Coherence Tomography (OCT) was utilized to document the extent of corneal involvement, including depth of penetration, confirming the particle was embedded in the corneal epithelium with mild surrounding epithelial and stromal edema. After instillation of 1% Proparacaine, the foreign body was removed via jewelers forceps, and the rust ring removed to the fullest extent via Alger Brush. A bandage contact lens and Tobradex, 1 gt every 2 hours were prescribed to improve patient comfort, reduce ocular inflammation and prevent secondary infection. The patient returned to the clinic four subsequent times for evaluation, undergoing a second Alger Brush procedure 9 days after the initial presentation to remove residual rust deposits that failed to spontaneously leech from the cornea. At each visit, and after each corneal procedure, anterior segment OCT was conducted to document the state of the cornea. After the second rust removal, the patient was again prescribed a bandage contact lens for comfort, and Vigamox to provide antibiotic coverage. Flurometholone 0.1% (FML) was prescribed one day later to reduce corneal inflammation and scarring. Upon final evaluation 3 weeks post initial presentation, the patient underwent a final anterior segment OCT to document the post-FB status of the cornea, including mild corneal scarring from FB and rust deposit removal.

Conclusion: Advanced technology such as optical coherence tomography can be employed to enhance proper treatment and management of corneal foreign body incidents, improve documentation of ocular tissue pre-and post-medical procedures, and boost patient education. Advanced imaging can provide detailed records of ocular damage, useful for the provider managing the case, future healthcare providers seeing the patient, and the patient him/herself. Foreign body injuries occur commonly and are often due to lack of patient knowledge/use of
protective eyewear. Patients may find typical education on protective eyewear importance/corneal injury risks uninteresting or difficult to understand. Providers can utilize the advanced technology in their practices to enhance proper management and documentation of their patients’ injuries, to establish detailed patient records for future health care, and to serve as dynamic education opportunities for future patients.

LOW VISION RURAL HEALTHCARE with a Low Vision Optometrist utilizing CLINICAL VIDEO TELEHEALTH
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Background: Veterans who live in remote and rural communities have limited access to Low Vision Rehabilitation services with a Low Vision Optometrist. In July 2011, National VA Blind Rehabilitation provided funding to purchase Clinical Video Telehealth equipment to various VHA Low Vision/Blind Rehabilitation Centers. In 2012, Telehealth Clinical Coordinators: 1) facilitated the ordering, set up and training of Telehealth equipment; 2) established and implemented service agreements between various facilities; 3) hired Telehealth clinical technicians at the rural Community Based Outpatient Centers (CBOC); and 4) trained Telehealth clinical technicians on the proper use of Telehealth equipment. A team approach was established and developed with the low vision optometrist at the Buffalo VA VISOR clinic and the local primary care Optometrists/Ophthalmologists at the rural CBOC eye clinic(s) and/or local eye care providers to access the veteran’s ocular health examination and refractive status information. This information is necessary because it cannot be obtained during a low vision clinical video telehealth evaluation. The following case report illustrates one example of concurrent low vision clinical video telehealth services with a low vision optometrist and a blind rehabilitation therapist.

Case Summary: A consult was received on 3/25/14 requesting low vision clinical video telehealth services. The Low Vision Optometrist at the Buffalo VA VISOR clinic, reviewed scanned clinical notes from a local retinal specialist’s 3/6/14 examination and CBOC VA Optometrist’s 4/8/13 examination. Veteran is legally blind secondary to neovascular macular degeneration, mild diabetic retinopathy; pseudophakia OU & injections OU. Visual acuity was recorded on 3/6/14 with current Rx dispensed on 11/22/11: OD -0.75 sphere, 20/150; OS -0.50 -0.25 x 015, 20/200. Veteran was scheduled for a Low Vision Clinical Video Telehealth evaluation with a Low Vision Optometrist and assessment with Blind Rehabilitation specialist on 4/22/14 at a VA 70 miles away. Patient was observed by TCT to have entered the room naturally. Personality was noted as outgoing and was alert per Low Vision Optometrist. Questions and discussion regarding mobility issues, distance vision, illumination and near vision resulted in documenting the patient’s goals were to spot read labels at the store and at home (medicine) and to read his mail. With the use of a near acuity card on the patient side, the TCT was able to hand the chart to the patient as the Low Vision Optometrist instructed patient who read 2.5M OU with his current bifocals; and 0.8M when instructed on the proper use of a +16D illuminated stand magnifier. Discussed in detail the patients functional implications and prognosis of their eye condition. Visual concerns were discussed including safety and general medical care. Instructions were given for contrast, lighting, specific reading techniques, eccentric viewing, principles of magnification, specific active daily living adaptations and the importance of practice in acquiring skills. Psychological factors were discussed including the importance of activity and maintaining independence. Immediately following the low vision clinical video telehealth evaluation, a low vision rehabilitation assessment with a blind rehabilitation specialist (BRS) was conducted via Telehealth and indicated
approximately 66.6% (8/12) of the activities discussed that are reasonable attainable goals were identified as slightly/moderately difficult. These areas were Meal, Home and Financial management, Communications, Personal Care, Leisure time activities, Low Vision Device training and Computer Access training. A home visit was scheduled 6/5/14 to address goals above and instruct and train proper use of adaptive equipment and order equipment as needed per patient goals. Veteran’s care is ongoing.

**Conclusion:** The patient’s positive feedback was appreciation for our low vision clinical video telehealth services as he noted not being able to drive to our Buffalo specialty clinic and would not have been able to receive services.

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**Ocular Decompression Retinopathy: A Case Report**

*Paula Johns, O.D., Zuni Comprehensive Health Center, Zuni, New Mexico*

**Background:** Ocular decompression retinopathy is a complication that can occur after rapid reduction in intraocular pressure. It is characterized by retinal hemorrhages that may be scattered in the mid-periphery or posterior pole. Most documented cases of ocular decompression retinopathy have occurred after glaucoma filtering surgery, but cases have also been seen after paracentesis, iridotomy and other ocular procedures.

**Case Summary:** A 58 year old diabetic, hypertensive female presented to our clinic as a walk in for eye irritation. She had a history of non-compliance with neovascular glaucoma treatment and multiple central retinal vein occlusions in both eyes. On examination, intraocular pressure was measured by Goldmann tonometer at >70mmHg OD, OS. Tonopen measured IOP at 84mmHg OD and 71mmHg OS. Florid neovascularization of the iris was present in both eyes, and there was a central retinal vein occlusion in the left eye with no macular hemorrhages. Maximum medical treatment was initiated and she was referred to a retinal specialist for urgent neovascular glaucoma treatment. The patient was evaluated the next day by the retinal specialist and scheduled for a paracentesis and intravitreal avastin injection OS the following day. At that visit, that patient was on maximum medical therapy with intraocular pressure measured 39mmHg OD and 48mmHg OS prior to avastin injection. After paracentesis and intravitreal injection OS, IOP was measured at 16mmHg before the patient left the office. The morning after paracentesis and avastin injection OS, the patient presented to our clinic with a complaint of sudden decreased vision in the left eye. On examination, visual acuity had decreased from 20/80 OS to counting fingers at 2 feet. Fundus examination revealed large macular hemorrhages in the left eye that were not present at previous examinations.

**Conclusion:** Ocular decompression retinopathy is characterized by retinal hemorrhages that occur after a rapid decrease in intraocular pressure. The mechanism of decompression retinopathy is poorly understood. There are two main theories that seek to explain the pathophysiology: the mechanical theory and the vascular theory. The mechanical theory postulates that a rapid decrease in intraocular pressure causes a change in the shape of the globe, initiating a shearing force on small retinal capillaries that will cause retinal bleeding. The vascular theory hypothesizes that in eyes with high intraocular pressure, the rapid lowering of intraocular pressure causes a change in retinal perfusion that the small blood vessels are unable to autoregulate. It is thought that the vessels would then leak because they are unable to adjust quickly to changes in perfusion pressure. Decompression retinopathy will usually resolve without treatment within 1-2 months. In approximately 14% of cases a vitrectomy is required to clear vision. This is an uncommon complication that patients should be made aware of before rapid lowering of intraocular pressure.