Terrien’s Marginal Degeneration
Kayla Vickers, O.D., M.S., Keller Army Community Hospital, West Point, New York

Background: Terrien’s marginal degeneration (TMD) is an idiopathic thinning of the peripheral cornea possibly secondary to increased histocytes and lysosomes. Usually a bilateral asymptomatic condition with pannus, pseudoepithelialum and circumferential lipid deposits. The prognosis is favorable with corneal astigmatism correction and corneal perforation being rare. The etiology of TMD is poorly understood thereby making treatment and management of these patients difficult. This case study will explore the appropriate therapies that may be utilized in keeping the TMD patient comfortable while protecting corneal integrity.

Case Summary: 24 year old white male presented complaining of red dry and irritated eyes. Patient stated he was previously diagnosed with marginal ulcerative keratitis and dry eyes and treated with Restasis bid OU, Tobradex qid OU, Genteal and lid scrubs over the past three months. Patient stated that he felt condition was “flaring back up.” His uncorrected visual acuities were 20/20 OD, OS at distance and near with an autorefraction of +0.25-0.25x126 OD pl-0.50x028 OS. Automated keratometry readings were 42.75/42.50 OD and 42.75/42.25 OS with distorted mires. Corneas displayed superior and inferior stromal thinning with vascularization and yellow-white lipid deposits OU. Patient was diagnosed with Terrien’s marginal degeneration and blepharoconjunctivitis and treated with Restasis 0.05% and instructed to instill one drop twice a day into both eyes every day for long term therapy; Refresh Plus and instructed to instill one drop into both eyes as needed every day for dry eyes; and Erythromycin ointment and instructed to instill ½ ribbon into inferior cul de sac in both eyes at bedtime every night for long term therapy; Doxycycline 40 mg, take one capsule by mouth with food for three months. Lipid panel and CBC blood work were ordered. Patient educated on long term effects and therapies of condition. Patient instructed to return to clinic in two weeks for instillation of punctual plugs. Differential Diagnosis: Pellucid Marginal Degeneration Marginal Furrow Degeneration Mooren’s Ulcer Keratoconus Arcus Senilis
Conclusion: Corneal dystrophies are many and range from non-inflammatory to sight threatening. Many are idiopathic with no known cause and others are hereditary. No cure exists, treatment is variable and PK the ending result for many rapidly progressive dystrophies. No established treatment protocol for Terrien’s. If inflammation develops, supportive therapy is indicated including corticosteroids, artificial tears and punctal plugs for dryness. However, in more extreme cases with increasing astigmatism, hydrops and corneal perforation, rigid gas permeable lenses, limbal grafts and lamellar keratoplasty may be indicated.

Macular Pigment Optical Density Spatial Distribution Influences on Higher Order RMS Wavefront Error and Intraocular Scatter
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Introduction: A number of investigators have hypothesized a role for macular pigment optical density (MPOD) in higher order root-mean-square (HO RMS) values (Kvansakul et al., 2006) and intraocular scatter (Puell et al, 2008 and Wooten et al., 2002). These hypotheses are based on the established properties of macular pigment (MP)
which include dichroic filtering, polarizing effects, and shortwave light absorption. The current study utilizes a novel device to determine the relationship between the spatial distribution of MPOD across 16 degrees of central retina with both forward scattered intraocular light using the C-Quant device (Oculus, USA) and HO RMS values using the Nidek aberrometer (Nidek, USA) measurements for a 6mm pupil.

**Methods:** A novel customized heterochromatic flicker photometer (cHF\textsubscript{P}) device was used to measure MPOD at 0, 2, 4, 6, and 8 degree retinal eccentricities along the superior, inferior, nasal, and temporal meridians using a 1 degree circular stimulus. MPOD was assessed by 5 repeated measurements at each retinal eccentricity. HO RMS values were determined for a 6mm pupil for 3rd-6th Zernike polynomials over 10 repeated measurements using the Nidek aberrometer. Intraocular scatter was assessed with the C-Quant device using 5 valid, repeatable measures as determined by the ESD and Q parameters. Correlation coefficients were used to determine the relationship between foveal MPOD versus integrated MPOD with intraocular scatter and HO RMS values across a 6mm pupil. A total of 3720 trials were analyzed across 22 subjects with an age range of 21-33 years.

**Results:** Spatial mapping of MPOD across all subjects resulted in a 1st order exponential decay function along all 4 meridians and was best-fit with a Lorentzian distribution function. Integrated MPOD distribution values were calculated using OriginPro 9 software. The MPOD measurements were highly reliable demonstrating a standard error of 0.01 log units at the fovea and 0.03 log units at 6 degrees of retinal eccentricity. Inverse relationships for foveal MPOD with HO RMS values 3rd-4th orders showed approaching significance ($r=-0.351$, $p=0.109$). The inverse relationship of foveal MPOD and intraocular scatter showed near significance ($-0.388$, $p=0.074$). Correlation coefficients for integrated MPOD with HO RMS values and intraocular scatter were non-significant.

**Conclusion:** The findings support that an inverse association exists between foveal MPOD and both HO aberrations and intraocular scatter. This relationship may play an important role in visual performance and retinal protection within a healthy, non-cataractous population. Further study of the cumulative effects of individual MPOD properties is warranted.

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**Red Eye, White Dot: APMPPE Hidden in Plain Sight**

Christopher Caldwell, O.D., Evelyn Reyes-Cabrera, O.D., M.S., Brooke Army Medical Center, San Antonio, Texas

**Background:** “White Dot Syndromes” are inflammatory disorders producing yellow-white retinal lesions differentiated by history, laterality, and FA findings. This case justifies the importance of evaluating the entire ocular system in all patients with red eye signs.

**Case Summary:**

- **I. Optometry**
  - HPI: 18 yo WF; redness, watering, dull pain; photophobia OU; (+) URI
  - Pertinent Findings
    - Meds: BCP; (-) MHx or FMHx
    - BVA 20/25 OD, 20/20 OS; NCT 11mmHg/10mmHg
    - SLE (initial): 1. trace crust, follicles OD>OS, A/C D&Q OU 2. mild anisocoria (longstanding) iii. Post seg (initial): Not evaluated iv. SLE (f/u): clear lids & conj, gr2 KPs , 3+ cells, small KP ant lens OU vi. DFE (f/u): lens & vitr clear; c/d 0.10h/v; (-) FR; (+) 1/2DD diffuse focal white spots posterior pole OU c. DDx / Plan i. Initial: Viral conjunctivitis - Maxitrol QID OU, RTC 3d ii. 2nd Visit: Chorioretinitis with Anterior Uveitis – d/c maxitrol; start PF QID OU, RTC 4d iii. DDx: APMPPE, AZOOR, Birdshot, MCP, MEWDS, PIC, Serpiginous choroiditis ii. Ophthalmology
  - HPI: No change to vision/comfort
  - Pertinent Findings
    - SLE: No change
    - DFE: (-) vitritis; (-) disc pallor; “multiple
yellow creamy retinal lesions” OU iii. FA: early and late hyperfluorescence iv. OCT Macula: RPE disruption OU v. Normal or negative: CXR, CRP, PPD, Lyme, ANA, FTA, RPR, HLA-A29, HLA-B51 vi. Toxo (-) for IgM, (+) for IgG vii. Elevated ESR (23) viii. CBC: elevated neutrophils, monocytes; depressed lymphocytes ix. Ach-E low b. DDx: APMPPE, Serpiginous choroiditis, MEWDS, Birdshot, MCP, PIC c. Diagnosis: APMPPE d. Tx / Management i. Pred 80mg PO QD / nexium 40mg PO QD ii. Increase PF to 1gtt QH OU iii. Follow closely, taper pred/PF over weeks-months

Conclusion: a. Even for common anterior segment pathologies (e.g. viral conjunctivitis), a fundus exam is always appropriate, especially if it is the patient’s first visit to an eye care provider. b. Thorough labs, imaging, and radiography are valuable in accurate diagnosis of uncommon diseases.

Management of Mucosa Associated Lymphoid Tissue Lymphoma
Grace Lea Y Dumayas, O.D., Womack Army Medical Center, Fort Bragg, North Carolina

Background: Lymphoid tissue can be organized as lymph nodes and lymphoid follicles known as mucosa associated lymphoid tissue (MALT). Lymphoid systems include lymphocytes, which are white blood cells that attack bacteria in the blood and are generated from the thymus and the bone marrow, which are the primary or central lymphoid organs. MALT can be further divided into gastric and non-gastrointestinal lymphoma affecting the head, eye, neck and lungs. The etiology of ocular associated MALT lymphomas has been reported to be chlamyphila psittaci.

Case Summary: A chronic, unilateral, follicular, painless conjunctivitis has many possible etiologies. Differential diagnoses range from molluscum contagiosum, chlamydial inclusion conjunctivitis or toxic follicular conjunctivitis. A diagnosis of orbital neoplasm accounts for 10%, specifically, MALT lymphoma, seems unlikely since the prevalence of this non-Hodgkin’s lymphoma occurs at only 8%. It occurs mostly in the 6th decade and females are more affected more than males. This poster discusses two cases with MALT lymphoma on a 36 African American male and a 27Hispanic male who presented with chronic follicular conjunctivitis without improvement after antibiotic, corticosteroid or antihistamine medications. The clinician will have a better understanding on its presentation, associated etiology, tests required, treatment options and its side effects.

Conclusion: Confirmation of MALT lymphoma associated with the ocular adnexa through biopsy is the first step. The neoplasm responds well to application of external beam irradiation of 30.6-45.0 Gray dose radiation therapy with the main side effects of dermatitis, dry eyes and cataract formation. The prognosis or five year life expectancy of MALT lymphoma has been reported at a rate of 91%.

The Macular Cube: Early Detection of Glaucoma Utilizing Zeiss Cirrus OCT Ganglion Cell Analysis
Ann M. Rudick, O.D., Evelyn Reyes-Cabrera, O.D., M.S., Brooke Army Medical Center, San Antonio, Texas

Background: Practitioners strive for early detection and management of glaucoma. This case illustrates the effectiveness of macular spectral domain OCT at detecting ganglion cell loss and advocates for its use as a baseline glaucoma test.

Case Summary: I. Case History 48 y/o WM CC: Blur at near POH/PMH: LASIK OU FOH/FMH: Unremarkable II. Pertinent findings BCVA: 20/20 OD, 20/20 OS IOP-GAT (mmHg): OD 18, OS 17 @ 14:30 SLE: LASIK flap OU CCT (microns): OD 484, OS 478 C/D ratio: OU 0.60/0.60, no notching OU Gonioscopy: Open to CB, 2+ pigment OU 24-2 SS HVF: GHT WNL OU RNFL OCT: Avg RNFL
Thickness (microns) OD 73, OS 74 Ganglion Cell Macular OCT: Avg GCL/IPL Thickness (microns) OD 63, OS 68 III. Differential Diagnosis: POAG High suspicion Ocular Hypertension Physiologic Optic Nerve Cupping Secondary OAG Low suspicion Previous Glaucomatous Damage Optic Atrophy Congenital Optic Nerve Defects IV. Diagnosis and Discussion Pathology of Glaucoma Epidemiology Genetic factors Characteristic findings V. Management Patient educated on presence of glaucoma risk factors, return for baseline tests. A 24-2 SS HVF normal with low reliability OU; CCT<500 microns OU; open angles OU, RNFL showed borderline avg thickness OU; OCT ganglion cell analysis showed thinning OU. Stereophotos of ONH obtained OU. Glaucoma suspect, RTC for 2nd 24-2 SS HVF. The 2nd 24-2 SS HVF was reliable, no defects; POAG based on RNFL thickness, thin CCTs, ganglion cell loss found with macular OCT. Target IOP established. Initiated Xalatan qhs OU. IOP check in 1 month. Good compliance with treatment. Target IOP achieved OU. Repeat 24-2 HVF, OCT, IOP check in 3 months.

Conclusion: OCT macular ganglion cell analysis ought to be used as a standard baseline glaucoma test Importance of comprehensive baseline tests in diagnosing glaucoma Involving patients in the decision making process of glaucoma management

Multi-layer retinal hemorrhages: identification, differentiation, and treatment
Amila Herbert, O.D., Kelly R. Thompson, O.D., Cincinnati VA Medical Center, Cincinnati, Ohio

Background: This case presentation will discuss identification, diagnosis and treatment of retinal artery macroaneurysm (RAMA) as well as important differential diagnosis of multiple retinal vascular diseases with similar clinical appearances.

Case Summary: Initial presentation of a 74-year-old Caucasian male with diabetes and hypertension indicates a chief complaint of continuously decreasing vision, which he reports was unable to be improved by previous eye care provider. Patient reports a vague ocular history including laser treatment, OS as well as an injection OD within the last 6 months. -VA: 20/CF, 20/125 -OD: moderate NPDR, Macular fibrotic scar -OS: moderate NPDR, CSDME, single large retinal heme, PRP scars Flourescein angiography reveals a subretinal, intraretinal, and preretinal heme with an overlying macroaneurysm OS and significant macular edema OU. Discussion RAMA is a rare dilation of a large retinal artery. It most commonly occurs within the first few bifurcations of the arterioles and causes multi-layer hemorrhage within the retina and possibly even within the vitreous. RAMA most commonly occurs in ages over 50 with a history of systemic hypertension and atherosclerotic diseases. Symptoms In the majority of cases RAMA patients are asymptomatic and discovered on routine exams. However, if there is visual disturbance it usually consists of sudden painless vision loss with macular involvement. Clinical Findings Acute hemorrhages within multiple layers of the retina often including subretinal, intraretinal, preretinal and sub-ILM spaces and the possibility of a vitreal heme. It is possible to have associated exudates, edema, and circinate ring. In a flourescein angiography the aneurysm will fill immediately and then continuously leak through the subsequent phases, local surrounding narrowed capillaries, and hypofluorescense in the areas of hemorrhages as they block the underlying fluorescence. Prognosis/Treatment Visual prognosis is often good, even if hemorrhages involve the macula. The majority of patients have spontaneous recovery of vision as the aneurysm and surrounding hemes involute. In cases of persistent macular edema laser photocoagulation is advised, and has been found to improve visual acuity. Studies shows that direct laser to the aneurysm is not advantageous and may even be more harmful. It is important to control underlying systemic diseases. Differential Diagnosis
Primary: Retinal Macular Degeneration, diabetic retinopathy, venous occlusion, Von-Hippel Lindau, IRVAN

**Conclusion:** Even if majority of RAMA cases have little visual involvement and good prognosis it is crucial to identify and diagnosis the proper disease as the possible differential diagnoses may have more serious complications and need for intervention.

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**An Unusual Manifestation of a Minocycline Toxicity**

Carly Rose, O.D., Kelly R. Thompson, O.D., Cincinnati VA Medical Center, Cincinnati, Ohio

**Background:** Minocycline is a tetracycline antibiotic used to treat conditions such as acne vulgaris, MRSA skin infections, and Lyme disease. Ocularly, tetracyclines are also indicated in patients with recurrent phlyctenulosis and marginal keratitis. A known side effect of minocycline is that it can cause skin pigmentation with chronic use. An unusual manifestation of this side effect is conjunctival pigmentation.

**Case Summary:** A 61 year old Caucasian male presented for a general eye exam, with no new complaints. He had a history of diplopia, Type 2 Diabetes without retinopathy, high myopia, and an asymptomatic epiretinal membrane. Systemically, he had COPD, SAPHO, and osteoarthritis. He had been on minocycline for the past 24 years for SAPHO. His best corrected vision was 20/20 (OD) and 20/25 (OS). Biomicroscopy revealed multiple black pigmented, elevated lesions throughout the inferior and superior palpebral conjunctiva. A few isolated black pigmented retinal lesions were evident OD during dilated fundoscopy. Upon further questioning, the patient disclosed that he also had black pigment deposits on his legs and feet, which were being followed by his dermatologist. Anterior and posterior segment photos were taken. The patient was scheduled to be followed every six months to monitor his ocular pigment deposits and every year and a half with dermatology to follow his skin conditions. SAPHO Syndrome is a bone condition that often has skin involvement. It is an acronym that stands for Synovitis, Acne, Pustulosis, Hyperostosis, and Osteitis. The first line of treatment is bisphosphonate therapy for these patients, however some cases respond to antibiotic therapy. This may be because Propionibacterium acnes, an acne bacterium, has been identified through bone biopsies of some SAPHO patients. Minocycline is a tetracycline antibiotic that has been reported to result in marked reduction of inflammation and symptoms in SAPHO patients. A possible side effect of chronic minocycline therapy is pigment deposits in the skin, nails, and teeth. In addition, deposits have been reported in the bones and sclera (resulting in a blue-gray sclera). Conjunctival and retinal pigmentation were much less commonly reported.

**Conclusion:** Chronic minocycline therapy can result in pigment disposition in various parts of the body, including the eye. Ocularly, the sclera is more likely to be involved in this toxicity. It is important to note that the retina and conjunctiva have been anecdotally reported as deposit sites for minocycline. If this occurs, the patient should be monitored and referred to dermatology. Baseline photos should be obtained.

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**Atypical Field Loss in a Patient with a Pituitary Adenoma**

Christopher Caldwell, O.D., Evelyn Reyes-Cabrera, O.D., M.S., Brooke Army Medical Center, San Antonio, Texas

**Background:** Pituitary adenomas are well known to produce bitemporal hemianopia by massing on the optic chiasm. This case documents a patient with a pituitary adenoma and defects limited to the superotemporal fields.

**Case Summary:** a. 74 yo WM, nonsmoker (quit 1997), obese (BMI >30) b. CC: HAs x 3 mo, 1x/day
in PM, 8/10 pain, (+) dizziness, (-) head trauma c. 
FMHx: brother d. 2009 brain tumor; 3 sisters c BC d. 
PHx: HTN, BPH, GERD e. NKDA; standard meds 
for above conditions II. Pertinent findings a. Family 
Prac: i. Vitals: unremarkable (unrem.) ii. ROS: recent 
onset of “rt lower extremity weakness/pain”iii. 
Physical findings: unrem. (gross VF normal OD/OS) 
b. Rad: i. Brain MRI w/o contrast: 6 mm ovoid lesion 
in the dorsum sellae, posterior to the pituitary gland ii. Sella MRI wo/w contrast: 6mm abnormality 
persists; no evident deviation of the pituitary stock, 
extrasellar extension, or mass effect on the optic 
chiasm; favors a microadenoma c. Optom: i. OHx: hx 
BCC excision from face; CE OU ii. CHA/pseudophakia OU, BCVA 20/20 OD/OS iii. 
NCT: 19mmHg/16mmHg@1027 iv. SLE: mult large 
fluid filled cysts lower fornix OU; small concretions 
LL OD; mult small facial lesions left temple and left 
ear v. DFE: c/d 0.30 OU; unrem fundus OU vi. HVF 
30-2 SS: 1. OD elev FP/FN; suspect sup. arcuate 
defect, some respect of vert. midline; GHT WNL 2. 
OS elev FP/FN; suspect sup. arcuate defect; GHT 
ONL d. Labs: i. TSH, lipids, comp met panel, PSA, 
alb/creat all normal ii. ALT slightly elevated III. Diff 
 Dx: microadenoma vs. bone neoplasm IV. Dx and 
discussion: a. Microadenoma: Per Kanski, “the 
chromophobe adenoma is the most common primary 
intracranial tumor to produce neuro-ophthalmological 
features.”V. Tx, management a. MRI spine wo 
contrast to r/o neural compression b. Refer to Derm to 
assess facial lesions c. Neurosurgery: f/u prn d. PCM: 
f/u prn e. Optom: RTC HVF 30-2 to assess 
repeatability 
Conclusion: The astute clinician will establish a 
reliable visual field baseline and consider atypical 
presentations of a lesion on the optic chiasm

"To Vault or Not Vault" A case of conjunctivochalasis recession and the challenge to fit an MSD lens
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Antonio, Texas

Background: Discussion of conjunctival tissue profile challenges in fitting a Mini Scleral Device (MSD) on a patient with severe dry eye syndrome (DES), ocular rosacea (OR) and blepharitis post conjunctivochalasis (CCH) recession OU

Case Summary: 62yo WF• Referred for corneal prosthetic device due to uncontrolled FBS, injection, DES, despite max therapy• OHx: DES, MGD, Blepharitis, OR, Canalicular ligation OU, CCH Sx OU • MHx: Depression, migraines, hyperlipidemia, pre-DM• Meds: Oracea, lacrilube, celluvise, restasis, flaxseed oil• Prior Treatment: Lacriserts, Azasite, Lotemax, no improvement. II. Pertinent findings• BVA: OD 20/20-, OS 20/20• K’s OD 42.6/45.7 @095 OS 42.1/45.6 @070• Lids: thickened, telangectasia lid margins OU• Cornea: PEE inferior OU; TIBUT 2-3 sec OU• Conjunctiva: 1+ injection, scar tissue temporal limbus OU III. Differential diagnosis• DES -OR -Blepharitis -CCH IV. Diagnosis and discussion• Severe DES uncontrolled on therapy • Prior interventions unsuccessful• Plan: Corneal prosthetic device to shield cornea • Conjunctival elevation impacts landing area of lens edge causing potential impingement or irritation; vaulting onto smoother conjunctiva with larger diameter lens is an option V. Management• Fit MSD lens: 15.8mm • Vault acceptable; slight impingement; staining after 1hr wear at 3- 9 o’clock; VA 20/20• Fit MSD lens: 18.0mm• Vault acceptable; no impingement as lens vaulted over scar tissue; no staining after 1 hr wear; VA 20/20-; more difficulty with insertion. Conclusion: Always consider conjunctival and corneal topography when fitting a MSD. Conjunctival tissue irregularities can affect lens choices.
Prosthetic corneal devices can act as therapeutic chambers and improve comfort by shielding the corneal nerves from external triggers and should be considered early in the treatment of chronic corneal disease despite the difficulties presented by ocular anatomy.

Progression of Adult Onset Vitelliform Macular Dystrophy
Diana Johns, O.D., Kelly R. Thompson, O.D., Cincinnati VA Medical Center, Cincinnati, Ohio

**Background:** Adult Onset Vitelliform Macular Dystrophy (AVMD) is a relatively rare dystrophy that appears as a yellow round macular lesion in patients within the fifth to sixth decade of life. This case reports discusses the progression of AVMD from diagnosis to end stage atrophy.

**Case Summary:** A 70-year-old white male initially presents for a general eye exam with decreased central vision, OS. The patient’s systemic health is positive for hypertension, hypercholesterolemia, osteoarthritis, and congestive heart failure. The patient’s current medications include amlodipine, aspirin, atorvastatin, furosemide, and lisinopril. BCVA was 20/30 OD, and 20/70 OS. The anterior segment was unremarkable, OU. Posterior segment was remarkable for bilateral mildly elevated round yellow macular lesions, OD measuring one-third disc diameter, and OS measuring a full disc diameter. The patient came in for regular follow-up exams over the next several years, including photos and OCT documentation. The lesion OD began to collapse 5 years after diagnosis, with a resulting VA of 20/50, and the lesion OS began to collapse 2 years after diagnosis, with remaining central atrophy and a lamellar macular hole decreasing VA to 20/100. Recent studies have shown the yellow material in AVMD accumulates subretinally. The exact composition of the yellow material is unknown, but the most common hypothesis is an accumulation of melanin and lipofuscin within macrophages of the RPE. It has been theorized that defective phagocytosis of photoreceptor tips by the RPE is the cause. The usual progression involves spontaneous resolution of the lesion leaving an area of atrophy. Although the exact cause is known, resolution of the lesion may occur when a critical number of photoreceptors atrophy and normal phagocytosis is able to catch-up with removal of the lipofuscin. Patients with AVMD are predisposed to macular hole formation due to the focal loss of photoreceptors in the fovea, which results in a worse visual acuity outcome. Sub-retinal fluid and choroidal neovascular membranes are possible, but rarely cause a significant decrease in vision. Currently, there is no treatment for AVMD, unless sub-retinal fluid or a choroidal neovascular membrane develop.

**Conclusion:** AVMD generally progresses from the initial presentation of a yellow macular lesion to collapse of the lesion with residual atrophy in the area. End-stage AVMD is often confused with age-related macular degeneration, but has a clinically distinct presentation.

Progressive Optic Neuropathy secondary to Chronic Ischemia due to Renal Failure Induced Uncontrolled Systemic Hypertension
Lauren R. Hunt O.D., Tam Peony, O.D., Jennifer Melsness, O.D., Walla Walla VA Medical Center, Walla Walla, Washington

**Background:** Progressive optic neuropathy occurring secondary to presumed vascular perfusion deficits due to co-morbidities of hypertension with kidney failure resulting in intermittent hypotension leading to poor optic nerve perfusion in a 75 year old male.

**Case Summary:** Pertinent findings Best corrected visual acuity was 20/30 OD and 20/60 OS. Ocular segment evaluation was significant for mixed senile cataracts OU and scattered macular hard and confluent drusen. Optic nerve head assessment of the
right eye revealed a large c/d ratio of 0.65/0.65, prominent optociliary shunt vessels temporally, and rim tissue thinning superiorly and inferiorly with trace pallor of the rim. Optic nerve head assessment of the left eye revealed a large c/d ratio of 0.6/0.6 and healthy rim tissue. Intraocular pressures were 8mmHg in each eye. Blood pressure readings were variable, ranging from 106/55mmHg to 136/79mmHg per review of primary care notes. Chart review showed pre-treatment intraocular pressures from 8-12mmHg OU with a maximum of 12mmhg OU. Pachymetry showed slightly above average central corneal thickness measurements of 570 microns OU. Gonioscopic measurements were open to ciliary body in all quadrants without pigment OU except for the nasal quadrant OD showing posterior trabecular meshwork. rNFL OCT analysis showed inferior and temporal thinning OD and inferior thinning OS. FDT visual field screening showed superior arcuate and inferior nasal visual field defects OD and superior and inferior nasal step visual field defects OS. The current exam’s reliable visual field defects were consistent with previous, although unreliable, HVF 24-2 visual field defects. Calculated ocular perfusion pressure based on daytime recorded blood pressure readings ranged from 56mmHg to 67mmHg. Differential diagnosis Primary differentials included progressive optic neuropathy, normal tension glaucoma, and previous ischemic event OD. The patient was diagnosed with progressive optic neuropathy secondary to chronic renovascular ischemia. While glaucomatous in appearance, the patient’s optic nerve appearance OU and progression of nerve fiber thinning with correlative visual field defects OU are likely secondary to vascular perfusion deficits due to co-morbidities of hypertension with kidney failure. These co-morbidities result in intermittent hypotension likely exacerbated by normal diurnal variances leading to poor optic nerve perfusion causing optic neuropathy. Treatment Brimonidine 0.2% twice a day OU for neuroprotection was initiated with a target intraocular pressure goal of less than 9mmHg OU, a 25% drop from the maximum intraocular pressure measured. **Conclusion:** Ocular perfusion pressure should be closely monitored in patients with cardiovascular co-morbidities and signs of optic neuropathy.

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The human lens: A living biometric indicator of health status and successful aging


**Background:** Age-related cataract (ARC) is differentiated from congenital, ocular, systemic, and environmental etiologies (i.e. metabolic dysglycemia, retinal disease, alcoholism, drugs, malnutrition, and smoking). Despite protection by the eyelid / facial structures, the human lens is prone to ultraviolet radiation. Various studies state that cataracts to be more prevalent in countries with more sunlight exposure. (i.e. The Beaver Dam Eye Study, 1992). Early ARC is not typically noticeable to the patient, but quantified with emerging in-office and mobile technologies. We present the combined use of in-office lens densitometry and aberrometry in a patient with subclinical ARC.

**Case Summary:** Auxiliary tests evaluate vision at different contrasts that account for the effect of light scatter. Contrast sensitivity, however, can also be degraded by age-related macular degeneration, glaucoma, and neurodegenerative diseases. There are various office devices for evaluating lens transparency directly. The Pentacam® 5th function provides precise geometric information on a cataract’s location, density, thickness, and
consistency. It utilizes a densitometer to measure lenticular density variation. Lenticular induced astigmatism and (coma aberration) or myopia (spherical aberration) can be mapped and followed using Zernicke analysis. The latest software version can also monitor lens opacification. The Dynamic Light Scattering system from NASA projects infrared light into the eye for two to five seconds and collects the light beams scattered by lens proteins. As opposed to the optical imaging techniques which detect visible cataracts, the DLS system can detect and quantify cataracts in their early stages. CATRA is a compact mobile cellular phone device that generates collimated patterns on the fovea. The user sees these patterns and presses a few buttons to map opacity, attenuation, contrast, and point spread function. It combines optics, mathematical knowledge, and computer graphics to address cataracts through patient interaction and education. A consistent and objective system that automatically detects and grades nuclear cataracts could be very useful. The slit lamp photograph system created by Huiqi Li and his colleagues in Singapore is very simple and user-friendly, and is the first one that can automatically detect and grade nuclear cataracts. MAPCATsf is a non-intrusive, non-mydriatic device tracking macular pigmentation and cataract density changes.

**Conclusion:** Lens transparency is an important living biometric indicator of health status. The Pentacam® PNS 5th function and NIDEK OPD 3 scanner have been used together to document lens aging. Dynamic light scattering, CATRA, mobile phone imaging, MAPCATsf, and slit lamp photography are additional techniques that can be employed.

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**Late Onset Schnyders Corneal Dystrophy in a 68 year old Caucasian Male**

Lauren R. Hunt, O.D., Jodi Moore, O.D., Walla Walla VA Medical Center, Walla Walla, Washington

**Background:** Clinical appearance and significance of late-onset Schnyders Corneal Dystrophy presenting in a 68 year old male.

**Case Summary:** A 68 year old Caucasian male presented to the clinic for a comprehensive diabetic eye exam with a chief complaint of severe light sensitivity. His medical history was significant for type I diabetes mellitus, hyperlipidemia, and coronary artery disease. His ocular history was significant for corneal abnormalities, cataract extraction with posterior chamber intraocular lens implant OU, and quiescent proliferative diabetic retinopathy OU. Pertinent findings Best-corrected visual acuity was 20/20 in each eye at distance and near. Anterior segment findings were significant for arcus OU, 2mm of diffuse crystalline opacities in the anterior stroma slightly inferior to the visual axis OD and 3mm of the same findings slightly inferior to the visual axis OS. Both the corneal epithelium and endothelium were clear OU. Intraocular pressures were within normal limits OU. Posterior segment findings were significant for posterior vitreous detachment, posterior chamber intraocular implant, and inactive proliferative diabetic retinopathy OU. An epiretinal membrane was noted OD. Differential diagnosis Primary differentials included Schnyders corneal dystrophy, lattice dystrophy, granular dystrophy, and macular dystrophy. Diagnosis and discussion The patient was diagnosed with late onset Schnyders corneal dystrophy. The condition usually presents in the 1st decade of life with central corneal crystals or haze. Arcus and mid- peripheral haze develop in the third of fourth decade, respectively. Visual loss does not occur until middle age and surgical intervention is not required until the seventh decade. This patient's slit lamp examination was consistent with the fine...
crystal deposition within Bowmans layer and the anterior stroma visualized in Schnyders corneal dystrophy. Medical history is positive for hyperlipidemia, commonly associated with the condition. Previous corneal assessments included stromal haze, corneal scarring, and band keratopathy, suggesting that the conditions classic crystalline appearance is a more recent change. Therefore, the patients corneal appearance has followed the expected clinical course, but at a later age of onset. Treatment: Monitor. Patient educated as to possible genetic linkage, progression, and the need for future surgical intervention by corneal transplant or phototherapeutic keratectomy if the condition changes.

**Conclusion:** Schnyders corneal dystrophy may present in geriatric patients as a late onset, progressive corneal condition.

Works Cited