AMD A-to-OCT-to-RI-to-Z

2-Hour Course

Greg Caldwell, OD, FAAO

grubod@gmail.com

814-931-2030 cell

**Course Description:**

This course will evaluate Age Related-Macular Degeneration (AMD) from subclinical to advanced AMD. It will emphasize structure (OCT) and function rod intercept (RI-dark adaptation) testing to provide early detection and proper staging of AMD. Once accurately diagnosed the course will discuss applying current clinical guidelines in the treatment of subclinical to advanced AMD. The course will help every optometrist by calling attention to OCT structural changes indicating progression.

**Course Objectives:**

1. Show how to diagnose the often missed subclinical or early AMD
2. Increase ones understanding on function testing for AMD
3. Increase ones understanding on the structure changes to properly stage AMD
4. Show how to treat subclinical or early AMD
5. Discuss OCT Angiography utilization in AMD
6. Review treatments for all stages of AMD
7. Review treatments for exudative/choroidal neovascular AMD

**Outline:**

1. AMD demographics
	1. Prevalence
	2. Disease state comparison
		1. Dry eye, glaucoma, DM, Thyroid
2. Eye care professional landscape
	1. 58,000 eye care professionals
		1. 40,000 optometrists
		2. 18,000 ophthalmologists
			1. About 10% are retinal specialists
3. AMD history
	1. Historically 2 Types
		1. Dry versus wet
	2. Current thinking 2 types
		1. Early disease state – preventing avoidable vision loss
		2. Intermediate-advance disease state -preventing avoidable vision loss
4. AMD considerations and pearls
	1. There is currently no cure for AMD
		1. Proper detect and care many prevent significant visual acuity loss in many patients
		2. 1288 eyes (644 adults)
		3. Eye care professionals are missing AMD about 25% of the time
		4. 30% of the undiagnosed eyes had large drusen
			1. Well-known risk factor for progression to advance disease
	2. 78% of AMD patients seek their first treatment after having suffered irreversible vision loss in one eye
		1. 50% have an acuity of 20/200 or worse
	3. Are anti-VEGF injections our patients’ best hope?
	4. Late-stage treatments, albeit are necessary, they have little impact on central acuity
		1. Impacting our ability to intervene in in early to intermediate AMD?
5. Optometrists and eye care professional responsibility
	1. Rethink our responsibility related AMD diagnosis and management
	2. Commit to you can and will do better in early detection and treatment
	3. Implement current clinically appropriate Practice Guidelines
		1. Those that preserve vision, don’t wait until vision has been lost
	4. Closely monitor and treat the early detected disease
		1. If progresses to advanced AMD, better opportunity to save vision
6. Early onset pathogenesis
	1. Drusen small or large are not makers for early stage AMD
		1. Visible structural evidence of a pathological process
			1. Underway for quite some time
	2. Cholesterol deposits exist beneath the surface long before drusen form
		1. Cannot be seen with structure-based methods
		2. Cholesterol produced by RPE and deposits into Bruch’s membrane
		3. Continue to layer in Bruch’s membrane
	3. As this cholesterol accumulates the process unfolds with compromise to the outer retina
		1. Inflammation
		2. Oxidative stress
		3. Disruption of oxygen and nutrients
		4. Drusen formation
	4. Impaired Vitamin A across Bruch’s membrane
		1. Functional impairment can occur to dark adaptation
7. Staging of drusen
	1. Sub-clinical or sub-structural – cholesterol layer
	2. Small drusen < 63 microns
	3. Medium drusen > 63 – <125 microns
	4. Large drusen > 125 microns
8. Beckman Committee Classification of AMD – based on presence of lesions within 2 DD of fovea in either eye
	1. No AMD
		1. None or few small drusen
		2. No AMD pigmentary abnormalities
	2. Early AMD
		1. Medium drusen
		2. No AMD pigmentary changes
	3. Intermediate AMD
		1. 1 large drusen
		2. Any AMD pigmentary changes
	4. Advanced AMD
		1. Any geographic atrophy
		2. Choroidal neovascularization (CNV)
9. Choroidal neovascularization (CNV)
	1. Type I – Occult
	2. Type II- Classic
	3. Type III- RAP
	4. Type IV- Mixed
10. Predictors of progressing to advanced disease
11. Tool for diagnosis, management, and treatment of AMD
	1. Comprehensive eye exam – structural, some functional
	2. Fundus photography and FAF - structural
	3. OCT and OCT Angiography – structural
	4. Dark adaptation – functional
12. Dark Adaptation – Function test
	1. Measures how long to recover from bright light to darkness
		1. Rod intercept line (RI) time
	2. Functional test that can help overcome the challenges in diagnosing AMD
	3. Alabama Study on Early Are-Related Degeneration (ALSTAR)
		1. Able to detect subclinical 3 years before clinically visible
		2. 325 adults without clinically detectable AMD
	4. Rod deterioration happens in earliest stages of AMD
		1. Earlier defection before visual acuity
		2. Rod intercept
		3. Sensitivity 90.6%
		4. Specificity 90.5%
13. OCT in AMD – Structure test
	1. Need spectral domain to follow intermediate or worse AMD, especially in identifying OCT predictors of progression
		1. Hyper-reflective foci
		2. Reticular pseudodrusen
		3. Nascent geographic atrophy
		4. Sub-RPE hyper-reflective columns
		5. Drusen substructures
		6. Drusen load and regression
14. OCT Angiography in AMD
	1. Able to identify OCT predictors of progression and identify occult or classic CNV before they leak
	2. Non-invasive technique
	3. Subclinical CNV or “Occult non-exudative CNV”
		1. Risk of exudation at 12 months is 15.2 times greater compared to eyes without subclinical CNV
15. Treatments for AMD
	1. Early defection meaningful treatments with significant value, not a cure, have been shown to slow or halt progression. Not limited to early stages but all stages of AMD
		1. Prescribe smoking cessation programs
			1. Smoking and AMD
				1. Depletes serum antioxidants
				2. Decreases pigmentary density
				3. Increases risk to advanced AMD
		2. Lifestyle changes
			1. Diet
			2. Exercise
		3. Systemic disease management
			1. Cardiovascular disease, DM, obesity, high cholesterol
		4. Nutritional supplements
			1. Sub-clinical/sub-structural or early disease
				1. Controversy flourishes

No definitive guideline exists

Despite consensus evidence suggests using supplements

* + - 1. Intermediate – advance disease
				1. No controversy on advocating for supplements
			2. AREDS 1
				1. Contains Beta-carotene and no lutein or zeaxanthin, no longer recommended
				2. Investigated early AMD, no statistically significant benefit
			3. AREDS 2
				1. Recommended for intermediate and advanced AMD, study protocol
			4. The Practical Guide for the Treatment of AMD - 3 primary options
				1. Macular pigment supplement

Carotenoids: lutein, zeaxanthin, meso-zeaxanthin

* + - * 1. Carotenoids, antioxidants, zinc, and vitamins C&E

AREDS 2

* + - * 1. Carotenoid macular supplement in subclinical and early AMD. Carotenoid and antioxidant is intermediate and AMD that is progressing
		1. Retinal light protection
			1. Sun exposure
		2. Closer follow up
			1. 12 months is currently accepted as being too long to defect progression
			2. 6 months or sooner based on risk of CNV
	1. Treatments for choroidal neovascularization (CNV)
		1. Current Anti-VEGF treatments
			1. Bevacizumab (Avastin)
				1. Humanized full length monoclonal antibody
				2. AMD
			2. Ranibizumab (Lucentis)
				1. Humanized monoclonal antibody fragment
				2. AMD, DME, DR, RVO
			3. Aflibercept (Eylea)
				1. Fusion protein
				2. AMD, DME, DR
			4. brolucizumab-dbll (Beovu)
				1. Humanized single-chain antibody fragment
				2. Up to 3 months dosing intervals, most are 4-6 weeks

50% remained 3 months after 1 year

* + - 1. Pegaptanib (Macugen)
				1. RNA aptamer
				2. AMD
	1. Low vision consultation
	2. Investigation treatments in AMD
1. Home monitoring devices for AMD
2. Masqueraders of AMD
3. Thank you – Greg Caldwell