

AMD: How to Improve Outcomes and Prevent Blindness

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2 hours

Course Description

Age-related macular degeneration (AMD) is the leading cause of blindness in older adults. This course reviews best practices in diagnosis, risk stratification, and patient management, emphasizing earlier detection, imaging strategies, nutritional supplementation, genetic testing, home monitoring, and emerging therapies to reduce visual loss and improve patient outcomes.

Course Learning Objectives

Differentiate stages of AMD using current classification schemes, clinical findings, and imaging modalities.

Interpret evidence for early detection strategies including dark adaptometry, OCT, OCTA, and functional testing.

Understand progression risk through analysis of patient history and advanced OCT interpretation.

Apply evidence-based risk reduction interventions including lifestyle modifications, nutrition, and AREDS-based supplementation.

Evaluate new and emerging technologies such as home monitoring, genetic testing, and photobiomodulation, to enhance long-term visual outcomes.

Demonstrate understanding of best practices to improve outcomes and prevent blindness by reviewing real world case examples.

Outline

I. Introduction and Epidemiology

- **Public health burden**
 - AMD affects 14 million Americans; prevalence rises sharply with age
 - Leading cause of blindness among Caucasians in the U.S.
- **Patient impact**
 - Vision loss affects independence, driving, mental health, and quality of life

- Vision loss is often severe prior to diagnosis of AMD (IRIS registry, anti-VEGF trial data)
- Lecture goals
 - Review diagnosis and classification of AMD
 - Present best practices for monitoring and utilization of diagnostic imaging
 - Discuss prevention and evidence-based recommendations to reduce progression and prevent blindness

II. Pathogenesis and Risk Factors

- Pathophysiology overview
 - Cholesterol deposits in Bruch's membrane → visible drusen
 - Oxidative stress, inflammation, impaired nutrient transport (vitamin A deficiency, dark adaptation impairment)
- Established risk factors
 - Age, family history, smoking, nutrition
- Putative risk factors
 - Cardiovascular disease, obesity, diabetes, low macular pigment
- Key message: AMD is multifactorial and progressive, requiring early identification of modifiable risks

III. Clinical Classification of AMD

- Beckman Committee classification
 - Early: medium drusen, no pigment abnormalities
 - Intermediate: large drusen and/or pigment abnormalities
 - Advanced: geographic atrophy (GA) or choroidal neovascularization (CNV)
- Alternative classification systems
 - CARMS
 - AREDS simplified
 - ICGS
- Progression rates
 - 5-year progression risk to advanced AMD based on Beckman/AREDS
- Case examples
 - Illustrative OCT and clinical exam cases

IV. Early Detection Strategies and Imaging Strategies

- Dark adaptometry
 - Dark adaptation impairment is earliest functional biomarker
 - CPT code 92284; rapid (<6.5 min) vs. extended protocols
 - Sensitivity/specificity >90%
 - Detects subclinical AMD 3+ years before fundus signs (ALSTAR study)
- Imaging innovations
 - OCT: case examples of structural biomarkers of progression

- Hyperreflective foci
 - Represent migrating RPE cells or inflammatory activity
- Reticular pseudodrusen/subretinal drusenoid deposits
 - Located above RPE rather than beneath it
 - Greater impairment in dark adaptation and higher progression risk to advanced AMD
- Nascent/impending geographic atrophy
 - Emphasis on OCT as primary method to assess for geographic atrophy
- Drusen substructures and volume
 - Increased risk of progression to advanced AMD
- Key message: structural OCT findings often precede symptoms and visual acuity loss, making it essential tool for proactive management
- OCTA: non-invasive blood flow visualization; avoids dye injection risks
 - Case example – non-exudative choroidal neovascularization (CNV) – identification and management strategies
 - Case example – differentiating geographic atrophy from CNV utilizing OCTA
 - Key message: OCTA allows clinicians to detect CNV activity before fluid develops, shifting management from reactive to preventative

V. Risk Reduction and Lifestyle Counseling

- AMD progression is influenced by modifiable systemic and environmental risk factors
 - Most effective when initiated in early to intermediate AMD
- Smoking cessation – most impactful intervention to reduce progression risk
 - Increases oxidative stress and chronic retinal inflammation
 - Depletes serum antioxidants and reduces macular pigment density
 - Dose dependent relationship between smoking exposure and AMD severity
- Diet
 - Role of diet in AMD pathophysiology
 - Decreased risk of progression with high intake of green leafy vegetables, increased consumption of fruits and vegetables, reduction of processed foods
- AREDS/AREDS2 supplements
 - Reduction in risk of progression to advanced disease in intermediate AMD
 - AREDS2 modification – removal of beta-carotene, addition of lutein and zeaxanthin
 - Conflicting evidence re: geographic atrophy progression (AREDS2 vs GATHER data)
 - Controversy re: use in at-risk patients, early AMD
- Macular carotenoids

- Improve macular pigment density, visual function, and slow drusen progression
- Omega-3 fatty acids
 - Observational data supportive; supplementation less consistent
- Emerging evidence suggests that certain systemic medications may influence progression risk
 - Metformin – reduces oxidative stress, lower incidence of AMD in observational study
 - Anti-depressants – current evidence is emerging but inconclusive
- Clinical application
 - Counseling strategies to improve adherence

VI. Advanced Concepts in Management of Intermediate AMD

- AREDS2 supplementation – Is zinc appropriate for everyone?
 - Evidence-based use; zinc controversy (80 mg vs. 25 mg; genotype-specific risks)
- Genetic testing
 - Genotype + phenotype improves risk stratification
 - Goal: personalized supplementation and follow-up frequency
 - Clinic application: genetic testing as an adjunct, not a replacement, for clinical judgment
- Home monitoring (ForeseeHome PHP)
 - Rationale and mechanism of action (hyperacuity perimetry)
 - HOME and ALOFT studies: earlier CNV detection, better preservation of 20/40 vision
 - Real-world adherence and outcomes
- Case management
 - Counseling example: diabetic patient with large drusen and abnormal dark adaptation, high-risk OCT features

VII. Emerging Therapies and Future Directions

- Photobiomodulation (PBM, Valeda system)
 - Mechanism: mitochondrial stimulation, nitric oxide activity, anti-inflammatory effects
 - Clinical trials (LIGHTSITE III): BCVA improvements, drusen volume reduction, reduced GA incidence, safety profile
 - Early implementation feedback
 - Patient counseling strategies
- New pharmacologic options for Geographic Atrophy
 - Pegcetacoplan
 - C3 inhibitor; intravitreal injection every 25–60 days
 - Based on OAKS/DERBY/GALE data
 - Avacincaptad pegol

- C5 inhibitor; monthly intravitreal dosing
 - GATHER1/GATHER2 trial evidence
 - Clinical benefit: *slows lesion growth*, does not restore vision.
 - Patient counseling and co-management pearls
- Other experimental approaches
 - Gene therapy and sustained-release anti-VEGF agents (brief mention for context)

VIII. Summary and Clinical Take-Home Points

- AMD is (possibly) preventable, detectable, and manageable when addressed early
- Earlier detection = better outcomes (dark adaptation, OCT/OCTA, home monitoring)
- Lifestyle and supplements matter (smoking cessation, nutrition, AREDS)
- Genetic testing and PBM represent new frontiers
- Primary care optometrists play a pivotal role in preserving vision and reducing blindness