

Age-related macular degeneration

Screen all your patients regularly with Amsler grid

■ by Tom Sheidow, MD and Jason Giroux, MD

Age-related macular degeneration (AMD) is the leading cause of irreversible blindness in people over age 50. A simple test using the Amsler grid can detect the condition early on, when treatment may still prevent further deterioration. Family practitioners can use this as a quick and easy screen at their regular patient visits. By the year 2020, the prevalence is expected to comprise 50% of patients age 70 and older. Although the neovascular type accounts for only 10% of AMD cases, it's responsible for more than 80% of individuals with severe visual loss associated with this disease.

Types of AMD

NON-EXUDATIVE OR "DRY"

- early stage — presence of few drusen (Fig. 1)
- intermediate stage — numerous drusen with geographic atrophy
- advanced stage — many drusen or large ones with geographic atrophy within macula
- may lead to blindness over the years
- can progress to wet AMD

EXUDATIVE OR "WET"

- choroidal neovascularization (CNV) — growth of new, abnormal blood vessels (Fig. 2)
- subretinal blood or leakage of fluid
- distortions and scotomas
- late form — disciform scarring

Risk factors

- age
- genetics
- history of smoking
- race — Caucasian
- diet low in antioxidants

Symptoms

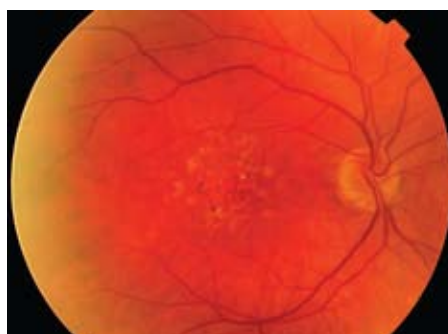
NON-EXUDATIVE

- range from none to a gradual decrease in central vision
- blank spots or scotomas
- loss of contrast, with difficulty adjusting from bright to dim light
- distortion of objects, termed metamorphopsia

EXUDATIVE

- sudden loss of vision
- metamorphopsia

Figure 1



Moderate dry age-related macular degeneration

Tom Sheidow, MD, FRCS is an associate professor of ophthalmology and vitreoretinal surgeon at the Ivey Eye Institute at the University of Western Ontario (UWO) in London, ON.

Jason Giroux is a 4th-year resident in ophthalmology at UWO.

Etiology

NORMAL CHANGES WITH AGE — DRUSEN

- focal deposition of debris
- look like pale, yellow lesions (Fig. 1, light spots)
- located between retinal pigment epithelium (RPE) and Bruch's membrane (Fig. 4)

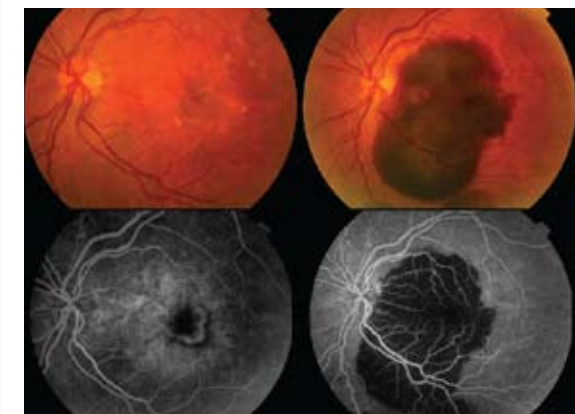
PATHOPHYSIOLOGY OF AMD

- first clinical sign — numerous drusen
- multiple factors
 - heredity
 - inflammation
 - ischemia
 - vascular endothelial growth factor (VEGF) — angiogenic cytokine; main driving force in exudative disease
- choroidal neovascularization
 - due to inflammation and release of VEGF
 - may result in subretinal hemorrhaging, fluid leakage

EYE PHYSIOLOGY (Fig. 4)

- Retinal pigment epithelial cells
- posterior to the photoreceptors
 - responsible for
 - blood-ocular barrier
 - photoreceptor phagocytosis
 - nutrient support
 - part of vitamin A cycle
- Bruch's membrane
- semi-permeable membrane
 - lies between RPE cells and layer of capillaries (choriocapillaris)
 - dysfunction with age leads to drusen and choroidal neovascularization

Figure 2



Left and right upper frames — colour photographs, wet AMD. Note subretinal blood from CNVM in right frame. Left lower frame — intravenous fluorescein angiography (IVFA), wet AMD corresponding to colour image, left above. Clearly visible classic CNVM present (white network) centrally. Right lower frame — IVFA, wet AMD, as in right colour image above. CNVM not visible or occult due to blocking from subretinal blood.

Diagnosis

AMSLER GRID (Fig. 3)

- one eye at a time
- reading glasses on, normal reading distance, no set measurement
- have the patient stare at the dot and evaluate the completeness of the grid and straightness of lines
- refer to ophthalmologist
 - significant change in central vision
 - if there's any doubt

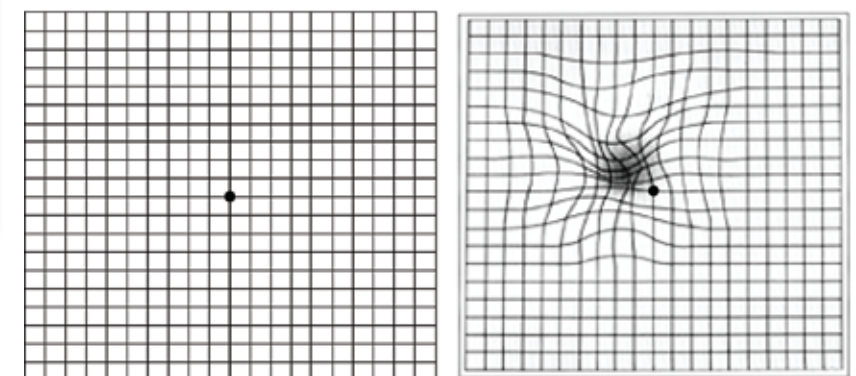
FUNDOSCOPIC EXAM

- drusen
- RPE hyperplasia
- RPE atrophy
- CNV membrane

IMAGING

- only for suspected wet AMD
- intravenous fluorescein angiography (IVFA — see Fig. 2)
- optical coherence tomography

Figure 3



Normal Amsler grid

Abnormal, distortions

Counselling

- advise against excessive exposure to sunlight
- discuss vitamin supplementation, family history and risks
- referral for resources
- Canadian National Institute for the Blind (www.cnib.ca)
- AMD Alliance (www.amdalliance.org)

Management

Based on Age-Related Eye Disease Study — AREDS

NON-EXUDATIVE

Early AMD

- no treatment
- low vision assessment
- magnifiers if needed

Intermediate and advanced AMD

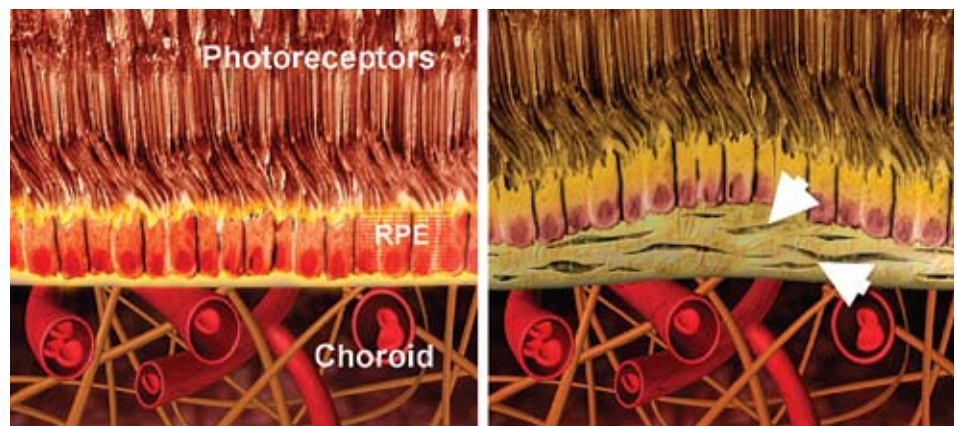
- daily antioxidant supplementation — AREDS ocular combo
 - vitamin C 500 mg
 - vitamin E 400 IU
 - beta-carotene 15 mg
 - zinc oxide 80 mg with cupric oxide 2 mg
- note:
 - beta-carotene — raised risk of lung cancer in smokers
 - high doses of vitamin E — may be associated with heart failure in diabetic disease or cardiac conditions (controversial)

NEOVASCULAR

- anti-angiogenic drugs (anti-VEGF)
 - intravitreal injection

- first-line therapy
- ranibizumab, bevacizumab
- possibly a limited visual gain from baseline
- photodynamic therapy (PDT)
 - verteporfin intravenous injection — 15 minutes before laser treatment
 - light-sensitive dye concentrates in abnormal vessels and, when activated by laser beam, results in thrombosis
- effective at limiting visual loss
- ineffective at providing visual improvement
- Argon laser photocoagulation
 - previously the most common treatment
 - now surpassed by intravitreal anti-VEGF therapy
 - can result in visual loss and retinal scarring
 - permanent scar at site of laser
 - only certain patients are candidates
- future advances — topical and oral agents

Figure 4



Thickening of Bruch's membrane, and developing drusen (short arrows)