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### **Multispectral Imaging (MSI) of choroidal ischemic melanin disruption and inflammatory hyperpigmentation features of atrophic AMD (AAMD) and Glaucoma Optic Neuropathy (GON)**

#### **Abstract:**

**Introduction:** Recent research has noted the association of AAMD with GON and has identified choriocapillaris ischemia as not only causative of outer retinal and RPE apoptosis that incite inflammation in AMD but also ischemia of prelaminar capillaries in GON. However clinical imaging assessment of color fundus photographs or OCT B sectors has been limited to identifying only late manifestations of drusen accumulation and outer retinal structural atrophy in AMD and of optic nerve sectoral atrophy in GON, both of which occur after irreversible death of neuronal sectors. MSI of extended wavelengths provides imaging of chorio-retinal lesions such as ischemia induced melanin disruption and lesions of inflammation. Herein, MSI is used to assess the amount of melanin disruption and features of inflammation in the progressive atrophic stages of AAMD and association with pre-laminar ONH sectoral microvascular ischemia of GON.

**Methods:** A retrospective image-analysis study was conducted of eyes with clinical AMD from two retina practices imaging with both SDOCT and MSI (AISpectral). Eyes were excluded if poor image quality or manifested signs of exudative AMD. The SDOCT images were overlaid upon the MSI to further define aspects of the lesions identified in the enface MSI (after selecting the best image within 550-850nm). Ischemia induced melanin disruption of RPE and choriocapillaris was graded 0-4 based upon the degree of reduced veiling of underlying choroidal vessels. The induced inflammation was graded 0-4 defined by the size and boundaries of hyperpigmented lesions: 1) RPE punctate pigment, 2) pigment macrophage migration within the RPE generating granular alterations, 3) melanocyte proliferation and migration to produce nummular lesions between choroidal vessels with granules upon the vessels. ANOVA testing compared these grades among the AREDS updated staging of AAMD, combining AREDS1&2 due to similarity of images. The association of peri-papillary ischemic melanin disruption and inflammatory pigment grading was also assessed with segmental microvascular loss within the optic nerve head.

**Results:** 49 eyes of 49 patients were analyzed (13,12, and 24 eyes in the AREDS 1-2, 3, and 4 categories). Significantly greater, widespread melanin disruption was noted within short posterior ciliary artery distribution, worse in AREDS stage 3 ( $p=0.04$ ) and 4 ( $p=0.02$ ) compared with stage 1-2. Greater severity of inflammatory pigmented lesions were observed in AREDS 3 ( $p=0.007$ ) and 4 ( $p=0.008$ ) compared with AREDS 1-2. Focal, deep, prelaminar ONH microvascular reduced density was noted in 47% of eyes, and in all but one eye was associated in the same quadrant with significant parapapillary localized ischemic melanin loss and dense granular hyperpigmentation.

**Conclusions:** Detailed, retinal, enface, MSI enables visualization of ischemic melanin disruption and inflammatory hyperpigmented lesions in AAMD and with secondary ONH focal reductions in microvascular density. This study demonstrates that both ischemic and inflammatory lesions worsened in the later stages of AAMD, revealing this aspect of the pathophysiology and the utility of MSI to map the progressive pathology of AAMD as well as with associated microvascular reduced density within the optic nerve, now recognized in the pathophysiology of GON. Certainly, to examine further, ICG IV and continuous flow studies are required to examine further the ischemia along with longitudinal studies to establish progression risks.

### **Biography**

After finishing medical school at Harvard, ophthalmology residency at Massachusetts Eye and Ear Infirmary, and a vitreo-retinal fellowship at the Medical College of Wisconsin, Dr. Sinclair spent 8 years as Assistant Professor of Ophthalmology and head of the vitreo-retinal service at the Hospital of the University of Pennsylvania in Philadelphia. He then migrated across town to Hahnemann University School of Medicine as Professor and of Ophthalmology to establish a new department as Chairman and then spent 6 years as vice-chairman of the Department. He left academia in 1993 and currently maintains a referral, private practice in vitreo-retinal diseases and surgery at in Media, Pennsylvania with affiliation at Riddle Memorial Hospital in the western and southwestern suburbs of Philadelphia. He remains as Adjunct Professor of Ophthalmology at Hahnemann, University teaching residents and students at Hahnemann and Temple Universities and at the Pennsylvania College of Optometry.