Supplementary Online Content


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This supplementary material has been provided by the authors to give readers additional information about their work.
**eFigure 1.** Stacked images of the en face image of Spectral domain optical coherence tomography and fundus photography. The hyperreflective materials are seen in areas with retinal pigment epithelium atrophy. Note that the orientations of the folds are different from those of choroidal vessels. Shadows of retinal vessels are less apparent in areas with outer retinal corrugation, because there is a hyporeflective tissue between the folds.
eFigure 2. High-resolution histology of basal laminar deposit (BLamD) associated with intact RPE in an advanced age-related macular degeneration (AMD) eye. Tissues were post-fixed with osmium tannic acid paraphenylenediamine, and 0.8 µm-thick sections were stained with toluidine blue. The principal components of BLamD are basement membrane proteins that stain blue. BLamD separates RPE from the true basal lamina, which is only 0.15 µm thick and thus visible only by electron microscopy. The principal component of basal linear deposit (BLinD) is lipoprotein-derived debris\textsuperscript{15} (originally called membranous debris\textsuperscript{12}) that stains gray, tan, or brown. Late BLamD is scalloped (convex towards the RPE) and is located closer to the RPE. It has little lipoprotein-derived debris within it. Early BLamD is palisade-like and located closer to Bruch’s membrane. It has noticeable lipoprotein-derived debris within it, apparently in transit to Bruch’s membrane. BLinD is an undulating band several µm thick between early BLamD and the inner collagenous layer of Bruch’s membrane (subretinal space). SDD, subretinal drusenoid deposit (green asterisk); BLamD-L; late basal laminar deposit; BlamD-E; early basal laminar deposit; BLinD, basal linear deposit; arrowheads, calcified Bruch’s membrane. Neurosensory retina is detached and is not shown.
**eFigure 3.** High-resolution histology sections showing persistent BLamD in advanced AMD. Tissue preparation was described in eFigure 2 caption. **A,** Persistent BLamD (yellow asterisk) has lipofuscin and melanosomes of RPE origin (L, M) internal to it and Müller cells (Mü) external to it. Cells of the inner nuclear layer (INL) remain. Choriocapillaris endothelium is absent, leaving empty bays between Bruch’s membrane intercapillary pillars. Neovascular AMD, fovea, 94-year-old white female. **B,** Persistent BLamD is breaking up (arrows), apparently due to undermining action of Müller cell processes. Geographic atrophy, 87 year-old-white male. **C,** Thinned, persistent BLamD (yellow asterisk) is lifted by cells of apparent retinal origin surrounding a calcific nodule (Ca), within a druse-like dome. Geographic atrophy, 90 year-old-white male. **D,** Dotted line separates sub-laminas of late (above) and early (below) BLamD (yellow asterisk). In the corrugation concavity are neo-vessels (red arrow) and cellular infiltrate (white arrow), overlying a fibrocellular scar. Choriocapillaris is mostly ghosts. Neovascular AMD, 90 year-old-white female. **E,** A layer of rivulet BLamD has multiple fine gray lines (likely lipid)\(^\text{22}\) crossing it and overlies late and early BLamD. RPE cellular fragments localize to the BLamD and overlying neurosensory retina. Another neovascular AMD case with similar persistent rivulet BLamD was previously published (Figure 2I of Curcio et al\(^\text{34}\)). Neovascular AMD, 96 yr old white female. R, RPE; L, late BLamD; E: early BLamD.