OCT imaging has numerous roles in AMD management – detecting early choroidal neovascularization, enhancing views of confounding fundoscopic findings, charting progression, assessing the efficacy of anti-VEGF treatments, among others. As such, OCT has established itself as a vital tool for managing all forms of AMD. This course offers an in depth review of OCT findings at all stages of the disease, how these relate to its pathophysiology, and strategies for using OCT to successfully manage your AMD patients.

*All slides will have high-resolution OCT images.*

**Quick review of OCT basics**
1) Normal retinal layers, choroidal layer
2) OCT imaging not just a stand-alone technique.
3) Multimodal imaging enhances OCT interpretation: near infrared reflectance (NIR), autofluorescence (AF), red-free reflectance.

**OCT findings in dry AMD**
1) Drusen
   a) Hard drusen
      i) Not a risk factor for AMD.
   b) Soft drusen
      i) Variable SDOCT reflectivity owing to variable drusen composition.
      ii) Focal elevation of RPE with intact underlying Bruch’s.
   c) Reticular pseudodrusen (subretinal drusenoid deposits)
      i) Visible with NIR (blue light)
      ii) Clinical appearance: Invisible or may appear as small focal whitish accumulations.
      iii) Best seen/detected using SD-OCT.
      iv) Unlike drusen, located above RPE.
      v) Risk factor for advanced AMD, but risk not thought to be as high as with soft drusen.
   d) Drusenoid PED
      i) Homogenous, slightly hyper-reflective signal fills PED space.
      ii) High risk feature of dry AMD.
      iii) Natural course: overtime may spontaneously resolve with resultant RPE atrophy.
      iv) Important to differentiate with PED from wet AMD and CSR.
      v) Subretinal or intraretinal fluid overlying PED and/or hyporeflective composition should increase suspicion for PED secondary to CNV.
      vi) Large drusen (125µm) vs. Drusenoid PED (>350µm)
2) RPE abnormalities
a) Hyperpigmentation or pigment clumping
b) RPE atrophy/hypopigmentation
c) Geographic atrophy
   i) Increased choroidal reflectivity
   ii) Subretinal hyperreflective deposit at margins may be present.
   iii) Vitreoretinal traction may potentiate GA progression and is best detected using SD-OCT.

**OCT findings in wet AMD**

1) Indirect signs
   a) Intraretinal and subretinal fluid
   b) Neurosensory retinal detachment
   c) Serous PED
      i) Indicative of occult CNVM.
   d) Vascularized PED

2) Direct signs
   a) Choroidal neovascular complex
      i) Hyper-reflective band = ill-defined visualization of components of membrane
         (neovessels, fibrin complex, blood, exudation)

**Classifying the type of wet AMD using OCT**

1) Why is classification important?
   a) Response to treatment and natural course (prognostic factors) may differ depending on the classification.

2) Type 1 CNV: neovascularization is confined to the sub-RPE space but that has eroded through Bruch’s membrane.
   a) Most common form of CNV.
   b) This is the occult NV previously identified using FA.
   c) Lesion may be present for months/years without affecting vision.
   d) PED may signify presence of type 1 CNV.
   e) Exudative signs tend to involve subretinal space, as opposed to intraretinal fluid.
   f) In dark pigmented persons, chronic Type 1 CNV may result in anomalous vascular changes, thought to lead to polypoidal choroidal vasculopathy more common in African-American and Asian populations.
      i) Polypoidal choroidal vasculopathy (PCV)
         (1) First described in 1980’s.
         (2) Abnormal inner choroidal vascular network with exudative aneurysmal terminal ends
         (3) Best diagnosed using indocyanine green angiography.
         (4) Clinical phenotype:
            (a) Onset typically 60-70 years old, earlier than exudative ARMD.
            (b) Affects Asians and blacks more than whites
            (c) Subretinal reddish orange polyps may be seen funduscopically.
            (d) Polypoidal lesions in whites tend to have a peripapillary location.
(e) Choroidal polyps result in exaggerated exudation with overlying neurosensory retinal detachment with heavy lipid deposition and blood (serosanguineous).

(5) EDI-OCT findings:
(a) Choroidal thickness may help differentiate PCV from wet ARMD.
(b) PCV > normal > exudative/dry AMD.
(c) Potential for identifying subset of patients with PCV
(d) Chung et al.
   (i) PCV eyes: 438 µm
   (ii) Fellow eyes: 372 µm
   (iii) Normal controls: 224 µm
   (iv) Exudative AMD eyes: 171 µm
   (v) Early dry AMD eyes: 177 µm
   (vi) Cystic retinal changes
   (vii) PCV < AMD

(6) Treatment:
(a) Differs from exudative AMD.
   (i) Tends to be recalcitrant to anti-VEGF treatments.
(b) Use photodynamic therapy with verteporfin.

3) Type 2 CNV: neovascularization has breached the RPE and is visible in the sub-retinal space.
   a) Classic CNV subtype that typically progresses rapidly.
   b) Less common than type 1 (type 2 represents about 15% of new CNVM’s)
      i) However, most type 2 lesions will have associated type 1 CNV.
   c) RPE remains attached (absence of PED).
      i) RPE reflectivity may be diminished due to overlying CNV/edema/hemorrhage.
   d) Disorganization of outer retinal layers with overall displacement of retina anteriorly.
   e) Dense zone of hemorrhage overlying CNVM may be present.
   f) Increased retinal thickness.
      i) Fluid accumulates primarily within the retina and not in the subretinal space.
      ii) Beware of auto-segmentation line errors.

4) Type 3 CNV: neovascularization is thought to develop within the retina, versus the choroid.
   a) Estimated to account for 15% of new CNVM’s.
   b) More likely to develop intraretinal edema and intraretinal hemorrhaging.
   c) Disorganization of outer retinal layers seen with OCT.
   d) Retinal-choroidal anastomosis may be evident with SD-OCT

**Early diagnosis of wet AMD**

1) Thorough knowledge of OCT characteristics of 3 types of CNV described above should facilitate earlier recognition of wet AMD.

2) When to scan dry AMD patients?
   a) Patient’s with high-risk characteristics: coalesced soft drusen, wet AMD in fellow eye.
   b) New onset metamorphopsia
   c) New onset reduced BCVA.

3) Presence of fluid does not equate to activity.
Monitoring anti-VEGF treatment effects using OCT

1) Typical treatment protocol: Initial injection (Day 0), Upload phase (injections Day 30 and Day 60).

2) OCT findings following treatment:
   a) Typically see dramatic decrease in intraretinal cysts and retinal thickness.
   b) Hyper-reflective CNV band often remains unchanged.
      i) CNV diameter: does not change in most cases and is independent from associated retinal edema.
      ii) CNV thickness: decreases significantly, but only by about 25% at most.
      iii) Overall, CNV membrane persists despite anti-VEGF therapy.
         (1) Despite resolution of retinal edema, may see persistent hyper-reflective complex that is thought to represent fibrosed CNV.
   c) Associated hemorrhage may begin to resolve.
   d) May see improved organization of retinal layers concomitant with treatments.

3) Identifying recurrent CNV s/p anti-VEGF treatments.
   a) Recurrence of small subretinal fluid or intraretinal edema may warrant re-treatment.

CONCLUSIONS

1) OCT imaging provides a wealth of microstructural information that sheds light on the pathophysiological changes associated with all forms of AMD.

2) OCT imaging is crucial for identifying the type of wet AMD, which may influence the prognosis and treatment choice.

3) Characteristic AMD OCT findings can alert clinicians to otherwise subclinical CNVM.

4) OCT is critical for guiding frequency of anti-VEGF treatments and monitoring for recurrence of CNV activity.

REFERENCES


