Commentary: Role of choroidal thickness assessment in acute anterior uveitis

Choroidal thickness (CT) is known to be affected in acute phase inflammation of various posterior uveitic disorders, as choroid or retina is the primary site of involvement in these diseases. Although by definition, anterior uveitis (AU) denotes involvement of the anterior segment of the eye, a limited number of studies have also reported changes in the CT in patients with acute anterior uveitis (AAU) suggesting possible choroidal involvement in AU.^[1-10] Recent advances in spectral-domain optical coherence tomography (SD-OCT) with enhanced depth imaging (EDI) mode and swept-source OCT (SS-OCT) have led to better imaging of the choroid providing further insight to pathogenesis of such entities.

Majority of the studies assessing CT in AAU have evaluated unilateral nongranulomatous AAU or selective HLA-B27-associated AAU patients and compared the CT with either healthy fellow eye or a separate group of age-matched healthy control additionally.[1-4,6-9] Few of these studies are retrospective, [1,3,7,8] while others are prospective; [2,4-6,9] the sample size varied between 16 and 120 patients of AAU across these studies.[1-9] Measurement of the CT in these studies was derived either from single point subfoveal CT or from multiple Early treatment of Diabetic Retinopathy Study (ETDRS) subfields with the help of in-built caliper-based manual measurement or automated integrated software-based segmentation of the sclero-choroidal interface (SCI).[1-9] Most of the authors have reported increased CT in the active inflammatory phase of AAU^[1-4,6,7] and few of them further demonstrated subsequent decrease in CT with successful treatment. [2-5] The findings in the present study are consistent with the above reports. [6] Moreover, the authors in this study showed increased CT in the affected eyes is positively correlated with decreased visual acuity. [6] Ahn et al. have also shown greater choroidal thickening by SS-OCT in eyes with more severe anterior chamber inflammation as compared to the fellow eyes in HLA-B27-associated AAU patients.[3]

However, Gehl *et al.* and Wiacek and Machalińska have reported no significant changes in the CT in patients with AAU.^[8,9] Yan *et al.* also reported reduced CT in patients with inactive AU, which was associated with disease duration and frequency.^[10]

Few authors have also studied simultaneous changes in the retinal thickness (RT) in addition to changes in the CT. While Balci $et\ al.^{[1]}$ and Kim $et\ al.^{[7]}$ have reported simultaneous increase in RT in addition to increased CT in active phase, no significant change in RT corresponding to increased CT was reported by Gabriel $et\ al.^{[2]}$ and Basarir $et\ al.^{[4]}$

Few hypotheses have been postulated for the probable increase in CT in AAU.[2,3,7] Although the blood supply and drainage of anterior and posterior part of the uvea are different, anastomotic connections do exist between the two. Anterior uvea is supplied by the anterior ciliary arteries, long posterior ciliary arteries, and anastomotic connections from the anterior choroid. Venous drainage from anterior uveal tract goes to the choroid and vortex veins. Hence, inflammatory mediators arising due to AAU could travel to the posterior segment and cause breakdown of both the blood-aqueous barrier as well as the blood-retinal barrier. Increased hydrostatic pressure in anterior uvea could possibly be transmitted posteriorly to the choroid causing choroidal congestion also. Another explanation could be increased CT secondary to higher blood flow in the choroid. This is substantiated by indirect demonstration of dilated large choroidal vessels in the acute phase of inflammation in en-face choroidal imaging with SS-OCT without any other simultaneous structural changes in the choroidal morphology.[3]

The decrease in CT following treatment, as also shown in multiple studies^[2-5] including the present one, may be a consequence of the topical treatment or decrease in the disease activity. Ideally, longitudinal follow-up of these patients with serial measurement of CT even after stoppage of topical treatment can reveal long-term morphological changes in the choroid in AAU.

During measurement of the CT, one needs to be cautious about taking into account of other parameters which can affect the CT like age, axial length, refractive error, intraocular pressure, associated ocular or systemic disease, diurnal variation, etc. Moreover, the measurement of point subfoveal CT has the potential to be influenced by regional SCI alterations due to wide topographical variations of the choroid. Including ETDRS grid defined multisectoral choroidal volume assessment could give more accurate data. Use of SS-OCT may enable better penetration through the retinal pigment epithelium (RPE) and clearer delineation of the SCI; simultaneous integrated automated software instead of manual caliper-based measurement can minimize inter and intra observer variation also.

Thus, it can be concluded that AAU, which was previously considered as a disease of anterior segment only, may have more widespread but reversible subclinical spectrum of changes in the posterior segment in the form of increased CT. These findings may necessitate OCT-guided measurement of CT in both affected and fellow healthy eyes of all AAU patients in addition to a routine slit-lamp biomicroscopic examination, as also highlighted in the present study. Future larger prospective studies are warranted to further strengthen these findings.

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