

EDITORIAL

Choroid in central serous chorioretinopathy

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Central serous chorioretinopathy (CSCR) is a chorioretinal disease characterized by serous detachment of the neurosensory retina, retina pigment epithelium, from the choroid. The disease shows a predilection for middle-aged males and is associated with steroid use, obstructive sleep apnea, pregnancy, and type “A” personality traits.^[1] The acute disease can resolve spontaneously, recur, or progress to a chronic form. The “central” in CSCR is as a result of the vision loss from serous macular detachment. The exact molecular mechanism of CSCR is not clear, however, several hypotheses have been made with regard to CSCR pathogenesis. A genetic pathway has been described by Lehman *et al.*^[1] The mineralocorticoid pathway has been described extensively by Daruich *et al.* in their review on CSCR pathophysiology. The resultant serous detachment in CSCR has been attributed to the hyperpermeability of the choroid. The choroid is implicated in the pathogenesis of CSCR and choroidal imaging findings have provided an invaluable information for the diagnosis and treatment of CSCR.^[2]

Several imaging modalities such as fluorescein angiography (FA), indocyanine green angiography (ICGA), adaptive optics scanning laser ophthalmoscopy (AO-SLO), optical coherence tomography (OCT) (spectral-domain OCT [SD-OCT] and swept-source OCT [SS-OCT]), and OCT angiography (OCTA) have been employed in literature to aid in CSCR diagnosis. These imaging modalities have captured useful information such as leakage points, choroidal thickness (CT), choroidal vascularity index (CVI), and hyperreflective dots (HRD) which have been used as imaging biomarkers for CSCR. For example, the FA can detect leakage points around and within areas of serous retinal detachments. In acute or early phase of CSCR, FA shows an “inkblot” or “smokestack” pattern mostly form a single leakage site. In later stages, it shows a diffuse circular hyperfluorescence from single or multiple leakage sites. ICGA is used to image choroidal vasculature. ICGA is able to detect leakage sites long after the initial insult than FA because FA is most effective during the active leakage phase. In an early disease state, ICGA shows mild hyperfluorescent and marked hyperfluorescence during later stages. In mid-phase of CSCR, there is usually variable

hyperfluorescence reported in literature. AO-SLO has limited use in CSCR due to the presence of fluid accumulation in the retina. In recent times, the OCT (SD, SS, and OCTA) has become the primary imaging modality for the diagnosis of CSCR. It provides useful and detailed information on the functional and anatomical characteristics of the retina and mostly the choroid. Several of these choroidal imaging findings in CSCR will be discussed below.

CT is one of the imaging biomarkers for CSCR. There is increased CT in CSCR mostly due to the dilation of the choroidal vessels. In a study by Hanumunthadu *et al.*, the increase in CT was greatest in acute CSCR followed by chronic CSCR. Normal eyes had the least CT in the study population. They also reported increased subfoveal CT (SFCT), medium and large choroidal vessel thickness in the same order (acute>chronic>normal eyes). The study also found that CT findings were consistently higher in the same locus of the fovea (750 μm nasal to the fovea) and hence suggested that CT findings might not be diffused but localized.^[3] Sahoo *et al.* also found increased CT at leakage sites in acute or resolved CSCR.^[4] These findings support the localized nature of CT findings in CSCR and the need to repeat testing in the same locus during follow-up visits.

CVI is an imaging biomarker in CSCR. It is defined as the ratio of the total choroidal vascular luminal area and the total choroidal area on OCT. The choroid comprises choroidal vasculature and interstitial stroma. An increase in CVI corresponds to an increased vascular luminal area. Agrawal *et al.* found an increased CVI and SFCT in acute compared to fellow eyes and age-matched normal eyes. The CVI in fellow eyes was higher than in normal eyes and accounts for the bilateralism of CSCR.^[5] It is important to note that CVI in CSCR does not change or improve with laser photocoagulation.^[6]

HRD is another imaging biomarker in CSCR. In a recent study, it was found to correlate with several parameters such as central macular thickness and SFCT which are characteristic findings in CSCR. The presence of HRDs is known to be as a result of chorioretinal remodeling. In acute CSCR, the ongoing inflammatory response could contribute to the development of

HRDs. In a study by Hanumunthadu and Chhablani, HRDs in acute CSCR were higher than chronic CSCR. The mean duration of disease did not correlate with the presence of HRDs.^[7]

Most recently, there have been several advances in imaging techniques which have given rise to wide field and en face choroidal imaging in CSCR. In the past, OCTs could only visualize about 6–9 mm central macula region. However, with the wide-field technique, we can extract structural and anatomical details of the perimacular and peripheral retina as well as choroid. We can now visualize up to 80° in the primary gaze and up to 200° in vertical and horizontal meridians. Singh *et al.* used the wide-field technique to determine CVI in healthy eyes. They found that the area with the least CVI was the macula. The highest CVI was found in the nasal region. Furthermore, in all regions of the choroid, the vertical meridian had a higher CVI than the horizontal meridian. An interesting finding from their study was the correlation between age and CVI. They reported a negative correlation (coefficient of -0.04) between the two parameters.^[8] In a study by Rasheed *et al.*, the vertical meridian had a higher CT than the horizontal meridian. The macular region in this study had the highest CT. The macula has high metabolic demand mostly derived from the choroid, hence the increased number of medium vessels, thick choriocapillaris and increased stroma accounts for the low macula CVI and high macula CT.^[9]

En face is another technique which has been employed in swept-source OCT and enhanced-depth imaging (EDI) OCT. En face imaging technique provides high-quality vascular anatomy images comparable to ICGA. There is also the possibility of assessing each choroidal layer in detail with this technique. Ferrara *et al.* used this technique to characterize the features of retinal pigment epithelium and the choroid. There was a loss of signal in CSCR eyes at the RPE indicating detachment and/or loss. With this technique, they were able to highlight CSCR with focal versus diffuse enlargement at different layers of the choroid. There were an increased number of focally enlarged vessels at the choriocapillary and Sattler's layers compared to diffuse enlargement at these layers (53% vs. 47%). At the level of the Haller's layer, diffuse enlargement was 4 times that of focal enlargement (80% vs. 20%) in this population. There is reported similarity in vascular pattern distribution in acute and chronic CSCR.^[10]

The choriocapillaris or small choroidal vessels play an important role in the pathogenesis of CSCR. A lot is known about the choroidal vessel hyperpermeability in CSCR, especially within the lobules of choriocapillaris. En face imaging techniques can be used to characterize choroidal vessels in acute and chronic CSCR most importantly at the level of the choriocapillaris. OCTA provides information of flow voids in the choriocapillaris. Matet *et al.* evaluated choriocapillaris flow voids in acute, chronic, and healthy eyes. They found increased flow voids in CSCR compared to healthy eyes. Furthermore, the total area of flow voids correlated with the duration of the disease (chronic > recurrent > acute CSCR).^[11]

The retina has well-delineated layers unlike the vessel laden choroid. Although it is known in literature that CT is a characteristic finding of CSCR, the different layers have not been delineated to explore the contribution of each layer. The CT has been attributed to the thickness of Haller's layer which is increased in disease eyes followed by fellow eyes and healthy eyes. The interobserver reliability in this finding is 0.795, which is not strong enough to make absolute deductions. Hence, Uppugunduri *et al.* used an algorithm to delineate the layers of the choroid. The algorithm was validated quantitatively and qualitatively. It achieved an intraobserver dice coefficient of 0.89. Further studies are required to delineate the choroidal layers.^[12]

In conclusion, the choroid is implicated in the pathogenesis of CSCR. Advancement in imaging has led to the identification of choroidal imaging biomarkers such as the CT, CVI, and HRD. Choroidal imaging findings provide valuable information for the diagnosis and treatment of CSCR. The development of techniques such as wide-field and en face EDI and SS-OCT has given detailed findings of the perimacular, peripheral retina, and choroid. These findings in healthy eyes, fellow eyes, and acute and chronic CSCR can direct the diagnosis and management of CSCR.

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