



Retinal Examination for Plaque Burden

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Editorial

A recent article authored by an experienced team of authors suggests that retinal amyloid burden is correlated with brain amyloid burden in Alzheimer’s Disease (AD). Moreover, it is stated that retinal amyloid plaques appear before they start to invade the hippocampus [1]. Screening Amyloid Beta (AB) in the retina is definitely a major breakthrough in Alzheimer’s Disease (AD). Comparing retinal amyloid burden with brain amyloid is even going one step further in the trials for the early diagnosing of AD. Imaging the retina is easy, non-invasive, cheap and does not involve radiation.

But, there are two important questions that come to mind when considering the recent developments in AD studies:

1. Mayo Clinic study of thousands of brains revealed Tau as driver of AD [2]. So, do we need to show Tau protein along with AB in the retina to be able to make correct assumptions about the disease process?

2. Postmortem studies have demonstrated that people with AD have far more AB plaques in their brains than healthy people. But, roughly 30% of people without any signs of dementia have brains “chock-full” of AB at autopsy [3]. So, the presence of AB may not be enough to prove that any patient will develop the disease in the future.

Increased deposition of AB isoforms have been described on photoreceptor outer segments and along the RPE–Bruch’s membrane interface in the aging human and mouse retina [4]. Another study using immunostaining revealed AB on photoreceptor outer segments all through the retina in humans as an ageing process [5]. This deposition process may very well be only a sign of ageing and not related to AD. Analyses of drusen components have shown deposition of AB within vesicles in eyes of Age Related Macular Degeneration (AMD) patients. We also managed to show AB in drusen of AMD patients [6]. A better approach may be to demonstrate and quantify both proteins in the retina [7,8]. We have been able to image Tau aggregates in the retina of live patients (Figures 1 and 2).

Tau deposits have been found in the temporal lobe early in the disease which proves that Tau PET imaging is also necessary to be able to correlate the abnormal protein burden with brain plaques.

Conclusion

Recent studies confirmed that combination of Tau and AB aggregates caused the disease. Another proof of this theory is the unsuccessful results of AB aimed therapies in AD. We believe

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Figure 1: Fibrillary Tau in the Retina (SD- Optical Coherence Tomography).



Figure 2: Advanced Tau tangle in the Retina (SD-OCT).

more collaborative work is necessary for the definite answers to the remaining questions.

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