Recent studies suggest that amyloid beta (AB) induced Tau pathology is responsible for the severe outcome of Alzheimer's Disease (AD) process. Data from different models support the thesis in which AB accumulation acts as a triggering event in the pathogenetic process by accelerating antecedent Tau [1]. Abnormal aggregation of tau protein ultimately leads to the formation of tangles within nerve cells. Once initiated, the tau aggregation process continues and spreads into previously healthy cells [2]. There are three main characteristics for a tauopathy: (a) an increase in tau levels; (b) a modification, like hyperphosphorylation, sometimes related to another posttranslational modifications such as truncation or acetylation; and (c) an abnormal tau aggregation [2].

It is possible to detect abnormal protein aggregation in patients with early or advanced AD by spectral domain optical scanning tomography (SD-OCT) and fundus autofluorescein (FAF) tests. Retinal regions with hyper or hypofluorescence can be inspected by OCT and neurofibrillary filaments (Figure 1) or advanced Tau tangles (Figure 2) can be observed by experienced posterior segment clinicians.

In patients with early AD or mild cognitive impairment (MCI), mostly thin filaments are visible on OCT. But, in patients with PET-proven advanced AD, apart from AB plaques thick tangles can be detected. Some of the thick tangles have a reverse E or number 3 shape (Figure 2).

Retinal examination for amyloid beta is important, but may not be enough to diagnose AD since it can be found in other diseases or just ageing [3]. Detecting AB triggered Tau aggregates may be more specific. And staging of AD may be possible by retinal examination and detection of Tau in different development stages [4].

Citation: Kayabasi A Umur. "Retinal Examination by OCT to Reveal Neurodegeneration in the Brain". EC Ophthalmology 6.5 (2017): 139-140.
Thus, retinal examination by OCT and FAF is safe, non-invasive and cheap; plus, OCT and FAF are valuable and trustable biomarkers in the diagnosis of AD.

**Bibliography**


