INTRODUCTION
Recent studies suggest that amyloid beta (BA) induced Taupathology is responsible for the severe outcome of Alzheimer’s Disease (AD) process. Data from different models support the thesis in which BA 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 with in 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 post translational modifications such as truncation or acetylation; and (c) an abnormal tau aggregation.(2)

METHODS
Patients with early or advanced AD had spectral domain optical scanning tomography (SD-OCT) and fundus auto fluorescein (FAF) tests. Retinal regions with hyperor hypo fluorescence were in spected by OCT and neuro fibrillary filaments (Figure 1) and advanced Tau tangles (Figure 2) were detected in a masked fashion.

Key words:
Tau; OCT; FAF; Retina; Alzheimer's
RESULTS

Inpatients with early AD or mild cognitive impairment (MCI), mostly thin filaments were seen on OCT. But, in the patients with PET-proven advanced AD, apart from beta amyloid plaques thick tangles could be detected. Some of the thick tangles had a reverse E or number 3 shape. (Figure 2)

DISCUSSION

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 BA 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)

CONCLUSION

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.

References


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