

Neurodegenerative Diseases of the Brain using real Imaging Techniques

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Background: Recent research suggests that Tau in Alzheimer's Disease (AD) etiology is the culprit lesion along with neuroinflammation. Retina is the extension of the brain and is the most easily approachable part of the central nervous system. Spectral domain optical coherescent tomography (SD-OCT) and fundus autofluorescein (FAF) can be used to detect the pathological protein accumulations. There is evidence showing that retinal plaques start accumulating even earlier than the ones in the brain. Most recent images of the Tau protein in the brain are normal or reverse C-shaped paired helical filaments. Methods: SD-OCT and FAF looked at 20 patients with PET confirmed AD. The median age was 72 years. SD-OCT scanned hypo or hyperfluorescent retinal lesions, and paired helical filaments formed in C were examined in a masked fashion. The researchers agreed on the shape of the lesions taking into account both the C-shaped filaments (normal or reverse) and the thinner fibrillary structures.

With improving life expectancies and therefore the increase worldwide, the entire burden of neurologic disorders has increased significantly within the past few decades. Neurologic diseases are currently among the leading causes of adult morbidity and mortality, and therefore the debilitating course of those diseases contributes substantially to financial burden. There's a growing suggestion in recent years that cerebral microvascular changes could also be contributing to the pathophysiology of some neurologic disorders. Visualization of cerebral microcirculation remains challenging, however, even with highly specialized modalities for brain imaging. Embryologically, since the retina is an extension of the brain, cerebral and retinal tissues share common characteristics, thereby making retina a potential tissue within the physical body to make inferences regarding changes in cerebral microvasculature. As retinal imaging and image analysis techniques evolve over the approaching years, their utility as a screening tool and also as a tool for understanding the pathogenesis of certain neurologic diseases is probably going to enhance. The present literature on the role of retinal imaging, mainly color fundus photography, optical coherence tomography (OCT) and OCT-angiography (OCT-A) in studying neurologic diseases.

Damage to the brain from Alzheimer's disease occurs years before patients begin to exhibit symptoms. To date, numerous attempted therapies are unsuccessful, largely because there's no measurable indicator or biomarker for Alzheimer's disease before it's already symptomatic and advanced. The eye's retina is taken into account the developmental extension of the brain and may be accessed non-invasively. The research team examined the potential of retinal hyperspectral imaging technology to detect biochemical changes present at the first stages of Alzheimer's disease. Specifically, the technique characterizes light scatter changes within the retina of Alzheimer's disease patients in comparison with healthy participants. The method, which has been utilized in pre-clinical trials and a person's pilot study, scans a patient's eye to detect small quantities of a protein long before they collect in large enough clusters to make plaques within the brain, which may be a biological sign of Alzheimer's disease progression.

Life expectancy has increased substantially during past few decades, mainly due to advancements of health care and lifestyle. Following upward shift in demographic distribution has resulted during a rising

prevalence of aging-related diseases, like dementia. Consistent with the report in Prince et al. (2015), dementia affects approximately 46.8 million people worldwide. AD, because the most prevalent sort of senile psychosis far and away, accounts for 60–80% of all cases with dementia (Prince et al., 2015). The prevalence of AD is estimated to quadruple and intensive health care are going to be needed for 43% of those patients by 2050.

Despite of the heavy public disease burden of AD, there's no effective treatment for AD. Advances within the effective treatment and prevention of AD are hampered by challenges in diagnosing the disease at the preclinical trial, during which subjects are still asymptomatic in clinical settings but may have subtle evidence of early cognitive deficits within the context of neuropathology specific to AD (Sperling et al., 2011a). Currently, diagnosis was based on cognitive assessments among patients with symptomatic cognitive and behavior deficits (Dubois et al., 2014). Studies suggested that measurable changes in PET, MRI and CSF biomarkers occurred predates the onset of clinical symptoms (Beckett et al., 2010). Unfortunately, these are costly and/or invasive procedures that aren't appropriate for screening at a population level.

As a projection of the CNS via the nervus opticus, the retina has been described as a "window to the brain" and investigated intensively the potential of serving as a marker for AD (Guo et al., 2010; MacCormick et al., 2015). Low cost, simple usability and non-invasive features make retina testing ideal for screening large populations and preclinical AD investigations. Furthermore, variety of novel approaches in retina imaging, like OCT, are developed and made it possible to see lesions and changes of the retina at a really fine resolution.

The history to promoting the use of retina imaging in clinical practice for the treatment and management of AD. Then, we specialize in recent findings on the appliance of retina imaging to research AD and supply suggestions for future research directions.

Results: There were paired helical filaments in all patients that exactly matched Tau's histopathological and cryo-EM images in terms of form and scale along with thin fibrils and amyloid beta-like lesions. The disease is defined by the presence of abundant neurofibrillary lesions and neuritic plaques within the cerebral mantle. Neurofibrillary lesions consist of paired helical and straight tau filaments while other neurodegenerative disorders are distinguished by tau filaments with different morphologies. Filament cores are made of two identical protofilaments consisting of tau protein residues 306-378 which adopt a combined cross- β / β -helix structure and define the tau aggregation seed. Paired helical and straight filaments differ in their inter-protofilament packing, showing that they're ultrastructural polymorphs. The number of the retinal filaments and other abnormal proteins was in concordance with the severity of the disease process. The advanced retinal filaments had normal or reverse paired C forms and thin fibrils had the shape of histopathologic images seen in the disease's early stages of development.

Conclusions: Retinal images of Tau were disclosed for the first time in live AD patients. Retinal neuroimaging is a trustable biomarker and tool for monitoring the disease