Translating neuropathological marker of dementia syndromes into structural MRI

Norzaini Rose Mohd Zain[1,2]

[1]Department of Radiology, National Cancer Institute Putrajaya, Malaysia, [2]Head of Specialty for Clinical Radiology, Ministry of Health Malaysia, Malaysia

ABSTRACT

Alzheimer's disease is the most common cause of dementia characterized by progressive neurodegeneration accompanied by cognitive impairments. With the rapid development of neuroimaging technology, it is possible to diagnose AD through neuroimaging. The images commonly used in the clinical detection of AD include MRI, which permits the anatomical imaging of neurodegenerative disease with improved resolution, and Positron Emission Tomography (PET). Development of PET ligands specific for pathological substances such as β-amyloid (Aβ) and phosphorylated tau provides new perspectives on the diagnosis of dementia. Accumulation of amyloid plaques and neurofibrillary tangles in AD is contemplated to induce neural and synaptic loss that finally leads to cortical atrophy. Specific patterns of cortical atrophy distinguish typical AD from other neurodegenerative dementias such as frontotemporal dementia (FTD), corticobasal degeneration (CBD), progressive supranuclear palsy (PSP), and vascular dementia. Cortical atrophy patterns also potentially estimate preclinical or prognostic tissue damage in vulnerable regions such as the hippocampus and entorhinal cortex. Despite advancements made in structural MR imaging, radiologists are often reluctant to make specific diagnosis of clinical dementia based on subtle findings such as mild regional atrophy, which makes early diagnosis of AD often neglected or missed. Therefore, the introduction of a systematic and practical approach to the structural imaging diagnosis in AD is required as it remains the workhorse within the clinical practice. If done well, structural imaging plays a role in the identification and classification of dementia syndromes. Understanding the specific patterns of atrophy allows us to assess pathological progression over time and even physiological change in dementia syndromes.