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Correlation of Leukoaraiosis and Alzheimer's Disease Neuropathology

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Description

Great strides have been made in the accurate *in vivo* diagnosis of Alzheimer's disease (AD), in part to advances in biomarkers that can detect AD-specific neuropathology, such as PET imaging and Cerebrospinal Fluid (CSF) protein analysis of amyloid and tau. Downstream biomarkers are biomarkers that are not directly connected to amyloid or tau and play an important role in the identification and monitoring of AD severity and development. Patterns of volume loss (e.g., hippocampal atrophy) on structural Magnetic Resonance Imaging (MRI), for example, have long been utilised to aid in the differential diagnosis of Alzheimer's disease dementia. However, atrophy is not unique to AD-related neuropathology, but is a universal sign of neurodegeneration and is thus seen as a downstream biomarker of AD.

White Matter Hyperintensities (WMH) are MRI indicators of non-specific diseases that have long been studied in clinical trials for cognitive impairment and Alzheimer's disease. WMH are white matter hyperintense areas seen on T2 fluid attenuated inversion recovery (FLAIR) sequences. WMH has several etiologies (e.g., gliosis, axonal loss), although it is commonly associated with age and cardiovascular disease, and it is typically assumed to be of vascular origin and to represent small artery Cerebrovascular Disease (CBVD). WMH diseases (together with Cerebral Amyloid Angiopathy (CAA), microbleeds, and microinfarcts, among others) are highlighted as causes of vascular cognitive impairment in international consensus-based guidelines.

WMH predicts rapid cognitive decline and a higher likelihood of Alzheimer's disease dementia, and MRI WMH may be a downstream biomarker for Alzheimer's. Based on observations indicating individuals with autosomal-dominant AD had an elevated burden of WMH long before clinical symptom onset and WMH predicted CSF beta-amyloid levels in mutation carriers alone, a recent research argued that the underlying diseases of WMH constitute a key component of AD. WMH has been demonstrated to predict higher CSF total tau and progressive medial temporal lobe atrophy in Alzheimer's disease, as well as PET cortical amyloid uptake in older adults and patients with Alzheimer's dementia, even more than standard AD neuroimaging and cognitive biomarkers. The geographical distribution of WMH in AD has also been demonstrated to differ from that reported in "normal ageing," implying that WMH may be particular to AD. The connection between WMH and AD is frequently linked to WMH being a marker of CBVD. CBVD (e.g., arteriosclerosis, CAA) and AD neuropathology are significantly correlated, and CBVD may have a role in the aetiology of AD. CBVD (and subsequent MRI WMH) can, however, constitute white matter sequelae of AD neuropathology (ADNP), and whether CBVD in AD is additive or synergistic remains unclear.

Although there is much evidence linking WMH to in vivo AD biomarkers, association of ante-mortem MRI WMH with postmortem AD neuropathology (ADNP) is required to establish MRI WMH as a possible diagnostic for AD. Few investigations have looked into the clinicopathological link between WMH and ADNP. A research indicated that greater severity of visually-rated WMH was linked with more severe ADNP, based on a higher CERAD neuritic plaque score and Braak stage in 50 patients from the Baltimore Longitudinal Study of Aging Autopsy Program; WMH were also associated with a dementia diagnosis. In an advanced age autopsy sample of 66 people (mean age at death=95), ante-mortem buildup of WMH over time also indicated a higher Braak stage. A recent ex vivo study linked parietal white matter lesions to demyelination and axonal loss (rather than ischemic injury) in participants with AD, potentially reflecting Wallerian degeneration following cortical AD pathology; cortical phosphorylated tau (p-tau) pathology was also linked to white matter lesion severity. Previous research indicated an inverse link between quantitatively measured WMH and Braak stage, while Ischemic Vascular Dementia Program Project research found no correlation between quantitated WMH and ADNP.