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Vascular and Perivascular Perspectives in Alzheimer's Disease Neuroimaging

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Alzheimer's disease has traditionally been viewed as a neurodegenerative disorder characterized by amyloid-beta deposition, tau pathology, and subsequent neurodegeneration. Biomarker frameworks such as the AT(N) system have reinforced this protein-centered model. However, growing evidence suggests that vascular factors also play an important role in disease progression, and neuroimaging offers a unique opportunity to investigate these processes in vivo. This lecture presents Alzheimer's disease from a broader MRI perspective, focusing on vascular and microstructural markers that complement conventional amyloid- and tau-based imaging.

The first part of the lecture examines large vessel disease, particularly carotid and intracranial arterial stenosis, and its relationship with Alzheimer's disease biomarkers. Cross-sectional analyses showed no significant association between arterial stenosis and global amyloid burden. In contrast, intracranial stenosis, especially involving multiple vessels, was associated with neurodegenerative changes such as hippocampal atrophy. Longitudinal analyses further demonstrated that intracranial stenosis predicted greater progression of both amyloid and tau accumulation. These findings suggest that large vessel disease may contribute to Alzheimer's disease progression through mechanisms including chronic hypoperfusion, blood-brain barrier dysfunction, neuroinflammation, and impaired protein clearance.

The lecture then focuses on MRI-visible perivascular markers. Enlarged perivascular spaces (PVS) are increasingly recognized as potential indicators of altered perivascular fluid dynamics, although their biological significance depends on anatomical location. Previous studies have reported inconsistent associations with Alzheimer's disease biomarkers. In our analysis, enlarged PVS were not associated with amyloid burden, whereas greater basal ganglia PVS burden was associated with lower tau deposition, particularly among cognitively impaired, amyloid-positive, and APOE4-positive individuals. Although seemingly paradoxical, these findings suggest that enlarged PVS may, in certain contexts, reflect altered or compensatory perivascular clearance rather than simply representing structural damage.

The final section addresses the glymphatic system using the DTI-ALPS index, a diffusion MRI-derived marker that indirectly reflects glymphatic function. Unlike PVS, which are structural markers, the ALPS index provides a functional assessment. Lower ALPS index values were associated with higher amyloid burden, whereas no independent association with tau was observed after adjustment. This relationship was significantly stronger in APOE4 carriers, suggesting that glymphatic dysfunction may preferentially contribute to amyloid accumulation in genetically susceptible individuals.

Overall, this lecture highlights the value of integrating vascular, perivascular, and glymphatic MRI markers into the study of Alzheimer's disease. Rather than replacing conventional amyloid and tau biomarkers, these imaging measures provide complementary insights into disease heterogeneity and may improve our understanding of the vascular contributions to Alzheimer's disease pathophysiology.

Keywords: *Alzheimer's disease, MRI biomarkers, Intracranial arterial stenosis, Perivascular spaces, DTI-ALPS index*