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Candidate biomarkers and CSF profiles for alzheimer's disease and CADASIL

Bowirrat Abdalla
EMMS Nazareth Hospital, Israel

The differential diagnosis between Alzheimer's Disease (AD) and Vascular Dementia (VaD) are still roughly problematic in clinical practice, despite the widely used diagnostic criteria to differentiate between the two disorders. There is an increasing evidence that cerebrovascular dysfunction plays a role not only in vascular causes of cognitive alterations but also in AD. Cognitively patients, with AD, show sometimes-mixed degrees of associated vascular lesions in 30-60% of AD cases. In opposition, AD pathology may be present in 40%-80% of VaD patients, thus impeding diagnosis accuracy. Therefore, to eliminate this bewilderment and discrepancies in the diagnosis between the AD and VaD, it is worthy to shed light firstly on a disease that is a microangiopathy and represents VaD with clear milestones and features, as is the case of Cerebral Autosomal Dominant Arteriopathy with Subcortical Infarcts and Leukoencephalopathy (CADASIL). Studying CADASIL CSF biomarkers profile will help in the differential diagnosis between both diseases sharing the coexisting neurodegeneration, furthermore, CADASIL is a dominantly inherited mid-adult life disorder causing ischemic strokes, which belongs to vasculopathies and symbolizes a genuine prototype of VaD that provides a valuable opportunity for studying its CSF biomarkers. Secondly, examining and evaluating the CSF biomarkers of AD compared to that of CADASIL. The pathogenesis similarities between CADASIL and early onset AD affecting the small vessels of the brain have suggested plausible molecular mechanisms involved in vascular damage and their impact on brain function and come from the fact that in both diseases genetic mutations occur. CADASIL mutations in NOTCH3 gene generate toxic protein aggregates (Granular Osmiophilic Material-GOM) in the vicinity of Vascular Smooth Muscle Cells (VSMCs) causing degeneration and loss of VSMCs in small arteries and arterioles of white matter regions of the brain that lead to dementia, similar to those attributed to mutant forms of the Amyloid Precursor Proteins (APP) and presenilins genes who cause overproduction and accumulations of the toxic Aβ42 protein in the brain and collapse of Aβ42 clearance mechanisms in AD. Despite the presumed pathological similarities, substantial differences between the two phenomena may exist especially in the CSF neurochemical phenotypes.

bowirrat@gmail.com