Positron emission tomography for Alzheimer's disease diagnosis

Positron emission tomography (PET) imaging of [18F]-2-fluoro-2-deoxy-D-glucose (FDG) is accurate in the early detection of Alzheimer's disease (AD) and in the differentiation of AD from the other causes of dementia. FDG-PET imaging is available widely and is performed easily. Different patterns of abnormality with the various causes of dementia are well described. Semiquantitative methods of image interpretation are available. Medicare covers FDG-PET imaging for the narrow indication of differentiation of possible AD from frontotemporal dementia [1].

Ferrari-Souza et al. assessed 121 individuals across the aging and Alzheimer's disease clinical spectrum with positron emission tomography (PET) brain imaging for Aβ ([18F]AZD4694) and tau ([18F]MK-6240), as well as CSF GFAP and YKL-40 measures. They observed that higher CSF GFAP levels were associated with elevated Aβ-PET but not tau-PET load. By contrast, higher CSF YKL-40 levels were associated with elevated tau-PET but not Aβ-PET burden. Structural equation modeling revealed that CSF GFAP and YKL-40 mediate the effects of Aβ and tau, respectively, on hippocampal atrophy, which was further associated with cognitive impairment. The results suggest the existence of distinct astrocyte biomarker signatures in response to brain Aβ and tau accumulation, which may contribute to the understanding of the complex link between reactive astrogliosis heterogeneity and AD progression [2].

1)
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2)
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