Poster | Disorders of Nervous Systems and Treatment

[3P]Alzheimer's Disease and Dementia

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*Videos are available throughout the meeting period.

[3P-211]In vivo association of mitochondrial dysfunction with tau pathology in early Alzheimer's disease

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Background
In addition to amyloid and tau aggregation, mitochondrial dysfunction is important in the Alzheimer's disease (AD). However, it remains unclear how these abnormal proteins are associated with mitochondrial dysfunction in vivo. The purpose of this study is to clarify the mutual relationships among mitochondrial dysfunction and AD pathologies in the patients with early stage AD using positron emission tomography (PET).
Methods
Sixteen amyloid positive AD patients at the CDR 0.5 or 1 (mean age ± SD: 73.2 ± 6.3 years) underwent a series of PET measurements with [11C]PiB for amyloid deposition, [11C]PBB3 for tau deposition, and [18F]BCPP-EF for mitochondrial function. Associations among these three PET measures were evaluated by voxel-based regression and regions of interest methods.
Results
AD group showed decreased [18F]BCPP-EF SUVR especially in the medial temporal cortex including parahippocampus. Increased [11C]PBB3 BPND was observed in the medial and lateral temporal and parietal lobes. There was significant negative correlation of [18F] BCPP-EF SUVR with [11C]PBB3 BPND in the Braak stage 1/2 area, but not with [11C]PiB SUVR.
Conclusion
Our results indicated that mitochondrial dysfunction is closely associated with tau pathology.
No correlation of [18F]BCPP-EF with [11C]PiB indicates that mitochondrial dysfunction in the trans-entorhinal and entorhinal regions is not directly linked to amyloid deposition.