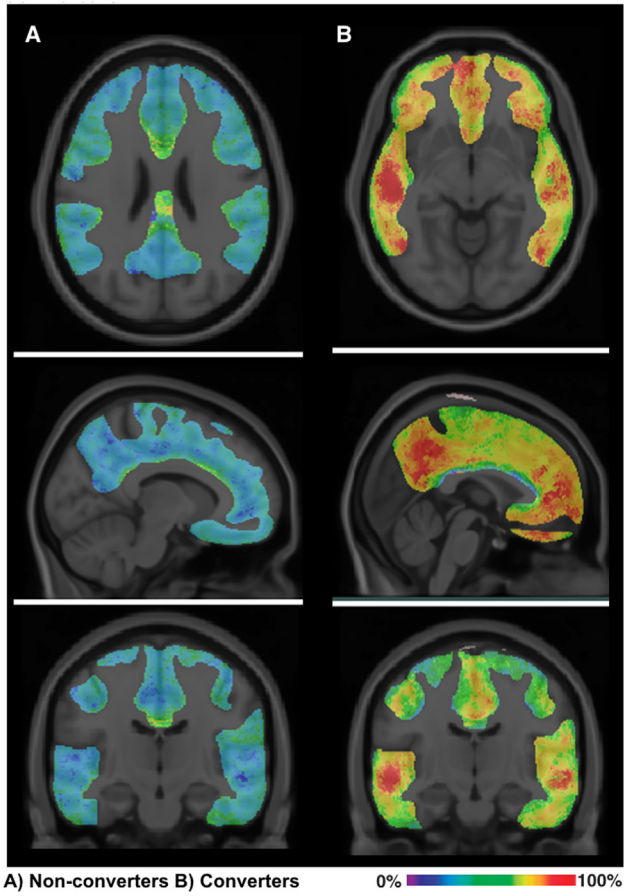

Figure 3. Probabilistic maps of [^{18}F]Florbetapir SUVR positivity at every voxel for MCI Non-converters and converters.

vealed that the analysis of amyloid-PET cut-offs at every voxel might provide important information regarding the patterns of regional Aβ abnormalities associated with the clinical progression to AD.

IC-P-026 PROBABLE CAA AND CLINICAL IMPLICATIONS IN A LARGE MEMORY CLINIC COHORT

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Background: Cerebral amyloid angiopathy (CAA) is associated with cognitive impairment, and especially with Alzheimer's disease. CAA manifests as neuroimaging markers of small vessel disease, and a conspicuous marker for CAA is lobar cerebral microbleeds (CMBs). We aimed to study differences in patients with and without probable-CAA and cognitive impairment in a large memory clinic. **Methods:** A total of 1504 patients undergoing memory investigation were recruited in our study. All patients underwent MR imaging with hemosiderin sensitive sequences and 1039 patients had cerebrospinal fluid (CSF) biomarker analysis. Probable-CAA was classified according to the Boston criteria using microbleeds as hemorrhagic markers. MR images were further analyzed for all markers of small vessel disease including superficial siderosis, white matter hyperintensities (WMH), lacunes and enlarged perivascular spaces. Generalized linear models with appropriate corrections were used to assess data. **Results:** A total of 99 patients (46 Alzheimer disease, 27 mild cognitive impairment, 6 subjective cognitive impairment, 9 vascular dementia, and 11 with other diseases) were classified as having probable-CAA. Patients without probable-CAA had lower CSF amyloid β (Aβ) 42 levels ($P<0.001$); patients with probable-CAA had higher CSF/ serum albumin ratios, reflecting blood brain barrier dysfunction, ($P=0.04$), as well as a tendency for higher total tau and P-tau levels ($P=0.05$). Patients with probable CAA had significantly higher burden of WMH (OR: 2.7, 95CI: 1.6-4.5), cortical superficial siderosis (OR: 6.6, 95CI: 2.3-14.8), lacunes 2.2 (1.3-3.8), and significantly lower amount of deep CMBs (OR: 0.04, 95CI: 0.01 - 0.31). There was no difference in amount of enlarged perivascular spaces, as well as MMSE score between patients with and without probable-CAA. **Conclusions:** Patients with cognitive impairment and probable-CAA likely have higher associated neurodegeneration with their disease, and a higher number of associated small vessel disease markers of CAA.

IC-P-027 AMYLOID-INDUCED MICROGLIAL ACTIVITY IN THALAMOCORTICAL CIRCUITS PREDICTS SUBSEQUENT COGNITIVE DECLINE

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Background: Although the signature properties of Alzheimer's disease (AD), such as the formation of amyloid plaques, activation of inflammatory responses, and hyperphosphorylation have been well studied, the explanation illustrating a clear relationship specifically between neuroinflammation and cognitive impairment has not yet been discovered. Thus in this article, we employed the McGill-R-Thy1-APP transgenic (Tg) rat model to observe for activation of inflammatory responses and the presence of cognitive decline. We hypothesize that early expression of neuroinflammatory signals will lead to future cognitive decline in the amyloid expressing animals. **Methods:** Microglial activation was measured using the ligand [^{18}F]PBR06 for PET, while cognitive performance was measured with the MWM task in 5 wild-type (Wt) and 8 Tg rats at baseline (BL; 11.5 months) and at follow-up (FU; 16 months). PBR-PET images were processed using the cerebellar grey matter as the reference region and cognitive measurement was calculated as the average times of day 3 and 4 of the water maze task. The baseline as well as the change (FU – BL) in the cognitive measurements were correlated at voxel level using "VoxelStats" toolbox to observe for the effect of group and PBR interaction on the change in the water maze performance. The PBR values were corrected for