deficits at age 35. Genetic analysis revealed a I229F mutation in PSEN1 gene and she was diagnosed as having FAD. Significant [C-11]PiB PET uptake was observed bilaterally in frontal, parietal, and temporal lobes, as well as in the basal ganglia, but not in the cerebellum. By histopathology, Aβ immunoreactive deposits were severe in all gray matter structures of cerebrum and cerebellum. Cerebral cortical Aβ deposits consisted of cored, diffuse, and cotton wool plaques (CWP). 6-CN-PiB signal was strong in cored plaques, weak-to-moderate in diffuse plaques, and absent in CWPs. In the cerebellum, Aβ IHC detected a heavy burden of diffuse deposits in the parenchyma and in blood vessels, while 6-CN-PiB fluorescence was detected only in the leptomeningeal and parenchymal vessels and infrequently in small compact Aβ plaques. **Conclusions:** As progress in imaging techniques allows us to use new tracers for misfolded proteins accumulating in neurodegenerative disease, neuropathology becomes more relevant for defining the efficiency of tracers in identifying specific lesions. The relative contribution of different Aβ plaque types to [C-11]PiB PET retention in vivo is currently being investigated. Funding sources: P30AG010133, P01AG025204, U19AG032438.

IC-P-033 CORRESPONDENCE BETWEEN [C-11]PIB PET AND POST-MORTEM MEASURES OF AMYLOID LOAD IN THE PRECUNEUS: THE ROLE OF DIFFUSE Aβ PLAQUES

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Background: The relationship between in vivo imaging measures of fibrillar amyloid-beta (Aβ) deposition and postmortem assessments of amyloid load is not fully understood. This study examined correspondence between in vivo [C-11]PiB PET retention measures and postmortem assessments of Aβ plaque load in the precuneus. **Methods:** [C-11]PiB PET was performed for 12 subjects representing a range of clinical states from cognitively normal to AD dementia. Subjects later came to autopsy with an average imaging-to-death interval of 36±24 months. Postmortem Aβ plaque load was assessed using Aβ immunohistochemistry (IHC, antibody clone 4G8) and a highly fluorescent derivative of PiB (6-CN-PiB) applied to 12-µm paraffin sections of the precuneus. A systematic anatomical match of postmortem dissected precuneus region was identified on the antemortem MR and co-registered [C-11]PiB PET. PiB SUVR values were corrected for atrophy-related cerebrospinal fluid (CSF) dilution using a 2-component (gray+white matter and CSF) correction. PiB SUVR and histopathology correlations were assessed using Pearson correlations. **Results:** In the precuneus region from 12 PiB PET imaged autopsy cases, SUVR PET corresponded with 6-CN-PiB plaque load (R^2 =0.802) and less strongly with Aβ IHC plaque load (R^2 =0.65). However, in cases where total plaque load exceeded 7% area by either Aβ IHC or 6-CN-PiB, SUVR values appeared to plateau. Separate analysis of plaque load with respect to either diffuse or cored plaques revealed that total plaque load was dominated by diffuse plaques (R^2 =0.94), particularly for cases at the SUVR plateau level. **Conclusions:** These preliminary results suggest that in brain regions where pathology load exceeds 7% area covered with plaques, and the majority of plaques are diffuse, there is no corresponding increase in [C-11]PiB retention. This could be due to saturation of the in vivo PiB PET signal with very high Aβ plaque load, but more likely is due to: 1) low PiB PET detection sensitivity for diffuse plaques at nM in vivo PiB concentrations and 2) overestimation of fibrillar Aβ content by semi-quantitative plaque load analyses. This observation also needs to be considered when investigating the detection threshold of [C-11]PiB PET positivity in relation to underlying diversity of Aβ plaque pathology.

IC-P-034 COMPARISON OF [18F] FLUTEMETAMOL AND [11C] PIB PET IMAGES

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Background: The half-life of [11 C] PiB is short while that of [18 F] flutemetamol is long. [11 C] PiB has been used widely as an amyloid imaging agent for brain PET. In recent years, [18 F] flutemetamol has been developed as a new PET amyloid imaging agent. In this study, PET images taken from the same patient using [18 F] flutemetamol and [11 C] PiB were compared so that the amyloid deposition in the brain could be visually and quantitatively evaluated. **Methods:** PET was conducted in three patients with amyloid-positive Alzheimer's disease (AD) and one patient with amyloid-positive mild cognitive impairment (MCI), using [18 F] flutemetamol and [11 C] PiB. Ninety minutes after intravenous administration of 185 MBq of [18 F] flutemetamol, dynamic PET imaging was performed for 30 minutes. For [11 C] PiB, dynamic PET imaging was performed for 70 minutes immediately after administration of 350 MBq of [11 C] PiB. The distribution of amyloid deposition in the region of interest in each cortex was quantitatively evaluated using the standard uptake value (SUV) sum images taken in the latter half of the PET session when a higher level of brain amyloid deposition was more readily detectable. The SUV ratio (SUVR) was calculated using the SUV in the cerebellum as the reference value. Moreover, the mean cortical SUVR (MSUVR) was calculated from the SUVR data in the cortex where a high level of amyloid deposition was detected. **Results:** When both [18 F] flutemetamol and [11 C] PiB PET images were evaluated, high levels of amyloid deposition were observed in the frontal lobe, posterior cingulate gyrus (PCG), precuneus, parietal lobe, and lateral temporal lobe. On the other hand, amyloid deposition was only weakly evident in the medial temporal lobe, occipital lobe, and the cerebellar cortex. In [18 F] flutemetamol PET, the MSUVR was 2.32, and the MSUVR in the PCG was 2.68. In [11 C] PiB PET, the MSUVR was 2.04, and the MSUVR in the PCG was 2.26. These data indicate that, although the dose of radioisotope was lower in [18 F] flutemetamol PET, the SUVR in each cortex were generally high in [18 F] flutemetamol PET. **Conclusions:** [18 F] flutemetamol has a longer half-life and can be delivered for a longer duration. The data indicate that, as an amyloid imaging agent, the performance of [18 F] flutemetamol is similar to that of [11 C] PiB in AD and AD-associated MCI.

IC-P-035 CORRELATION BETWEEN CSF BIOMARKERS AND QUANTIFIED PIB BURDEN IN ALZHEIMER'S DISEASE PATIENTS

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Background: The diagnostic criteria for Alzheimer's disease (AD) suggested by the National Institute on Aging and the Alzheimer's Association (NIA-AA) included biomarkers related to amyloidopathy and neuronal injury. Among those AD biomarkers, the correlation between low cerebrospinal fluid (CSF) concentration of amyloid-β1-42 (Aβ1-42) and plaque burden measured by amyloid imaging as well as between increased levels of CSF tau and the extent of neuronal degeneration measured by MRI has