1010 Monday, October 15, 2018, 16:30 - 18:00, Hall 28

Neuroimaging - Rapid Fire Session: Cognitive Impairment & Neuro-Degeneration

OP-407

Amyloid Imaging using 18F-FIBT PET: a pilot study

K. Shi[1], T. Grimmer[2], S. Förster[3], H. Förstl[2], I. Yakushev[2], M. Schwaiger[2], W. Weber[2], A. Kurz[2], B. H. Youseft[2]; [1]University of Bern, Bern, SWITZERLAND, [2]Technical University of Munich, Munich, GERMANY, [3]Klinikum Bayreuth, Bayreuth, GERMANY.

Purpose: 2-(p-Methylaminophenyl)-7-(2-[18F]fluoroethoxy) imidazo[2,1-b]benzothiazole (18F-FIBT) has been reported as a promising marker for imaging cerebral amyloid deposition using PET. For further clinical integration, the imaging protocol and corresponding evaluation method are compared with ^{11}C-PiB and optimized in this pilot study. **Methods:** Six patients with different clinical-pathophysiological phenotypes underwent dynamic PET imaging for 90 min in Siemens Biograph mMR. For the comparison of different calculation methods, the patients imaged with dynamic ^{18}F-FIBT were compared to four patients scanned with dynamic ^{11}C-PiB. For the comparison of SUVRs, each case imaged with ^{18}F-FIBT was compared to a group of matched patients (five patients/group) imaged with ^{11}C-PiB. The image data were spatially normalized based on MRI using PMOD. Images were analyzed by comparing standardized uptake value ratios (SUVR), binding potentials (BP) obtained using reference tissue model, and distribution volume ratio (DVR). Cerebellum was selected as reference tissue region. The effect of MRI-based partial volume correction (PVC) on interpretation was also compared. **Results:** Specific binding was detected in the cases with underlying AD pathology. The intensities of BP and SUVR were associated with clinical severity for ^{18}F-FIBT, which was not observed for ^{11}C-PiB. SNRs were substantially higher in FIBT than in PiB imaging. The optimal imaging time for ^{18}F-FIBT was found between 40-60 min. Cases with non-AD pathology did not show specific binding. BP has higher contrast than SUVR. However, SUVR is more consistent with the clinical interpretations. **Conclusion**: SUVRs PVC correction seemed to be an easy and robust analyzing technique, making FIBT a favorable amyloid marker for clinical routine.

OP-408

Clinical validation of ^{18}F-PI-2620 for *in vivo* quantification of tau in subjects with Alzheimer's disease

A. Mueller[1], S. Bullich[1], M. Berndt[1], O. Barret[2], J. Seibyl[2], J. Madonia[2], H. Kroth[3], A. Muhs[3], A. Pfeifer[3], G. Tamagnan[2], K. Marek[2], L. Dinkelborg[1], A. Stephens[1]; [1]Piramal Imaging, Berlin, GERMANY, [2]Invicro, New Haven, CT, UNITED STATES OF AMERICA, [3]AC Immune SA, Lausanne, SWITZERLAND.

Aim: Intracellular tau deposition is a key pathologic feature of Alzheimer's disease (AD) and other neurodegenerative disorders. ^{18}F-PI-2620 is a novel tau PET-tracer with a high binding-affinity for aggregated tau. Pre-clinically, ^{18}F-PI-2620 binds to 3R and 4R tau isoforms and is therefore able to depict tau-deposits in AD brain sections from different Braak stages, as well as deposits in PSP. To extend the utility of PI-2620 for use in therapeutic clinical trials, a Test/Retest study with arterial sampling was performed and simplified quantification methods were explored. **Materials and Methods:** In an ongoing clinical imaging study, participants diagnosed with mild AD, as well as non-demented controls (NDCs) undergo dynamic PET imaging for 180 minutes. 3 NDC and 3 AD underwent a Test/Retest study including repeat-scanning and arterial sampling with metabolite correction. Distribution volume ratios (DVRs) were determined using full tracer kinetic models and reference tissue models. Standardized uptake value ratios (SUVR) were determined at different time points p.i. using cerebellar cortex as reference region. Test-retest variability was calculated as percent difference between test and retest scans. **Results:** Imaging data show robust brain uptake and fast washout in non-target regions with peak SUVs > 4 similar to what has been overserved before. There was no increased off-target binding, and no age-related increase seen in cortical and subcortical brain regions like basal ganglia as noted for first generation tau agents. Focal asymmetric uptake was evident in temporal and parietal lobes, precuneus, and posterior cingulate in AD subjects. The time-activity curves were well described by the 2-tissue-compartment model (2TC). SUVR measured at 60-90 min p.i. correlated well with 2TC (slope 1.37, $r^2 = 0.97$) and non-invasive Logan plot (slope 1.3, $r^2 = 0.93$). The average test-retest variability in AD was 2.1 % ± 8.0 (2TC), 3.3 % ± 6.1 (non-invasive Logan) and 7.8 % ± 8.6 (SUVR). SUVRs at 60-90 min were significantly lower in non-demented controls (SUVR 1.0-1.2) than in AD subjects (SUVRs up to 4) in the same brain regions. **Conclusion:** ^{18}F-PI-2620 PET data in AD and NDC demonstrate favorable kinetics and high target specificity with low off-target activity and high signal in regions of expected tau pathology. Non-invasive quantification using SUVR at 60-90 min p.i. provides significant discrimination between NDC and AD subjects. The excellent test-retest variability confirms the utility of PI-2620 to evaluate change of tau deposition in longitudinal studies.

OP-409

Association between White Matter Lesions and Cerebral Glucose Metabolism in Patients with Cognitive Impairment

B. Kim, H. kim, H. No, H. Lee, K. Won, H. Jang; Dongsan medical center, Daegu, KOREA, REPUBLIC OF.

Background: White matter lesions (WMLs), detected as hyperintensities on T2-weighted MRI, are considered potential risk factors for memory and cognitive impairment in the elderly. It has not been sufficiently evident that cognitive impairment in patients with Alzheimer's disease is caused by WMLs as well as amyloid pathology. The aim of this study was to evaluate relationship between WMLs and cerebral glucose metabolism in patients with cognitive impairment after adjustment of cerebral β-amyloid (Aβ) burden. **Methods:** Eighty-three patients with cognitive impairment, who underwent brain MRI, F-18 florbetaben positron emission tomography (PET), and F-18 fluorodeoxyglucose PET, were included prospectively: 19 patients