CORRELATION OF GLUCOSE HYPOMETABOLISM ON $^{18}$F-FDG PET WITH NEUROPSYCHOLOGICAL RESULTS IN POSTERIOR CORTICAL ATROPHY AND TYPICAL ALZHEIMER’S DISEASE

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Posterior cortical atrophy (PCA) is a degenerative brain disease where the lesion is focused at the cerebral region responsible for visual processing. The underlying cause of PCA is Alzheimer’s disease (AD) in the majority of cases. Despite being caused by the same disease process, the clinical characteristics of PCA and typical AD are different. Our objectives are to know the regions of glucose hypometabolism and whether each region is correlated with the neuropsychological results in each disease. Six patients with PCA and 11 with typical AD underwent $^{18}$F-FDG PET and neuropsychological test, and $^{18}$F-FDG PET was made in 18 control group. Imaging data were analyzed using SPM2 with MATLAB 6.5. We compare neuropsychological data between two groups using PASW18. Comparing with normal subjects, PCA showed significant glucose hypometabolism in the occipitoparietal, and posterior part of temporal lobe, and typical AD have significant reduction of glucose metabolism in cingulated gyrus, precuneus, temporal, and frontal lobe (uncorrected p < 0.001). Global glucose metabolism of PCA is similar with typical AD. In neuropsychological results, RCFT copy and the Stroop Word test are more severe impaired in PCA than in typical AD (p < 0.05). In contrast, typical AD had a significantly lower score in comparison to PCA in digit span forward and verbal memory (p < 0.05). The regional glucose hypometabolism in patients with PCA seems to be a different pattern compared with that in typical AD, and these hypometabolic areas are well correlated with the cognitive impairments of PCA and typical AD.