Different pattern of atrophy in early- and late-onset Alzheimer□fs disease

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Introduction

Voxel-based morphometry (VBM) is a computational approach to neuronal structure that measures differences in local concentrations of brain tissue, through a voxel-wise comparison of multiple brain images [1]. The process is semiautomated, bias-free and less time-consumptive, and enables rapid and extensive survey of brain pathology. However, arbitrary setting of the significance threshold may affect the detection of atrophied region. To make up for this flaw, we measured z-scores by placing regions of interest (ROIs) in the areas that significantly different from normal healthy subjects and clarify the variability of atrophic pattern in AD.

Methods

Fifty patients with AD (20 men, 30 women; age range = 58-85 years; mean age = 73.5 ± 8.4 years, MMSE=19.4 ± 4.2) underwent a study to identify the area with significant brain atrophy by comparing to 83 age-matched control subjects (37 men, 46 women; age range = 60-83 years; mean age = 70.6 ± 6.4 years, MMSE=28.9 ± 1.5). MR images were obtained with a 1.5-T MR scanner and regional z-score was obtained by VBM on Medx software. ROIs were set manually on the areas showing z-scores over 4.0. To identify cluster among explanatory variables, principal component analysis (PCA) was applied.

Results

Ten ROIs were conducted in the right and left amygdala, anterior hippocampus, posterior hippocampus, and temporal cortex and subcallosal cortex, and left posterior cingulate cortex (PCC) (Fig. 1). The results of regional z-scores in AD and control groups were shown in figure 2. The second component of PCA was composed of counter direction of eigenvectors in bilateral posterior hippocampus and temporal cortex and left PCC. Since the eigenvalues in PCC and temporal cortex are large, AD with younger onset has more advanced atrophy in those regions and less atrophy in amygdala and hippocampus (Table 1).

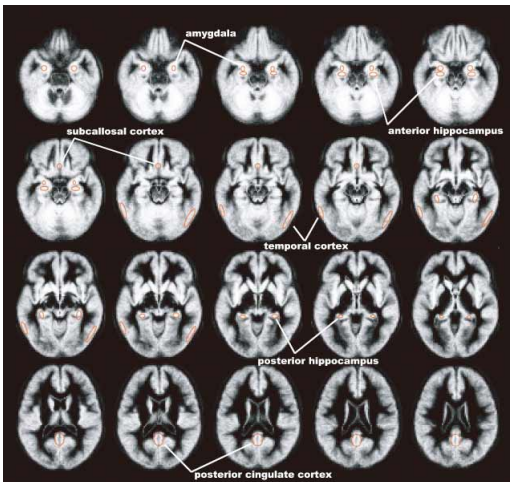

Figure 1. Ten ROIs were set on amygdala, hippocampus (anterior/posterior), temporal cortices, subcallosal cortex and posterior

Table 1. Results of principal component analysis

	PC 1	PC 2	PC 3
Eigenvalue	5.9699	1.7198	1.0767
% total variance	49.7493	14.3316	8.9724
age	0.18308	-0.43986	0.02330
Lt-amygdala	0.33664	-0.19120	-0.03990
Rt-amygdala	0.32559	-0.18363	0.21417
Lt-ant. hippocampus	0.35431	-0.15481	-0.14909
Rt-ant. hippocampus	0.34433	-0.08410	0.15863
Lt-post. hippocampus	0.34951	0.01113	-0.27449
Rt-post. hippocampus	0.32990	0.11495	-0.09637
Lt-temporal cortex	0.25222	0.42378	-0.22615
Rt-temporal cortex	0.22399	0.46687	-0.26793
Post. cingulated cortex	0.13866	0.48802	0.30756
Succallosal cortex	0.34969	-0.15813	0.11558
Total gray volume	0.12945	0.17648	0.76752

Principal component was omitted from the table after component fifth. PC, principal component; ant, anterior; post, posterior.

Discussion

Our PCA revealed characteristic pattern of atrophy in AD patients with early-onset, th
is preferentially associated with the atrophy of the PCC and temporal cortices a
atrophy of the amygdala/anterior hippocampus is less than th
in late-onset AD. This atrophic pattern has similarity with that of familial A
suggesting a possibility of phenotype gene factors that facilitate vulnerability of neurons [2-3].

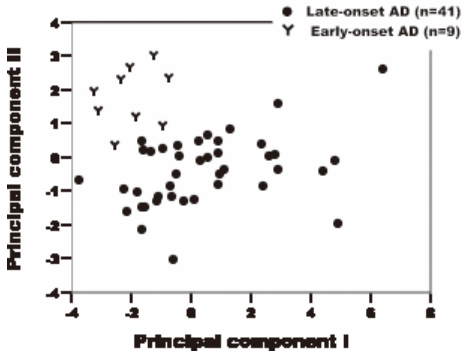

Figure 2. Scatter plot of the first two principal component
of the AD patients. There was different pattern in

References

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