Differences of Cortical Thickness in Patients with Late-life Depression Relative to Healthy Aging Measured Using MRI

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Introduction

MRI methods have been recently applied in morphometric measures, such as regional brain volume in depressive disorders (1). They found gray matter abnormalities in frontal, cingulate and hippocampal regions in the patients with depression. Depression and anxiety are the most common psychiatric conditions in late life. Late-life depression is a heterogeneous disorder that has become a major public health concern as the population has aged. Despite their prevalence, we know relatively little about their unique manifestation in older adults (2,3). Previous researches have found decreased regional cerebral volume, involving frontal, temporal, parietal cortex in the late-life depression (4). This study aimed to analyze the regional cortical thickness in patients with late-life depression and compare with normal aged subjects.

Methods

We enroll 12 patients of late-life depression (3 male, 9 female, mean age = 66.3 yrs) and 12 age-matched controls (5 male, 7 female, mean age = 67.7 yrs). All the subjects are restricted to aged 55 years or older, interviewed by using MINI International Neuropsychiatric Interview and diagnosed based on DSMIV (Diagnosis and Statistical Manual of Mental Disorder, 4th edition). The exclusion criteria include: 1) another major psychiatric illness; 2) current active alcohol or drug dependence; 3) primary neurologic illness, including dementia; 4) medication or medical illness that may affect cognitive function; 5) physical disability that precludes cognitive testing; and 6) metal in the body that precludes MRI. T1-weighted MR images were acquired at the Chang Gung Memorial Hospital, using 3 Tesla Trio Tim System (Siemens, Erlangen, Germany), with a 3D gradient echo sequence (TR/TE/FA = 2100ms/3.04ms/13°, matrix size = 256 x 256, 1 mm^3 isotropic voxels covering the whole brain). Cortical thickness analysis was done by using the Freesurfer v.4.1.0 software (http://surfer.nmr.mgh.harvard.edu/). Cortical thickness measurements were obtained by constructing the gray/white surface and pial (gray/CSF) surface. First, to find the point on gray/white surface that is closest to a given point on the pial surface, vice versa. Then, the thickness is computed as the average distance of this distance. Pre-processing involves the volumetric and surface processing stages, which including: Talairach normalization, intensity normalization, skull stripping, segmentation, fill and separate left and right hemispheres, tessellate, smoothing and inflation, Topology correction, generation of final surfaces and spherical morphometry. Finally, the QDEC software was applied for analyzing group data. Spatial smoothing with 10-mm FWHM was used reduce the anatomical differences of each subject and a significance thresholds of $p < 0.01$ to display the difference of the two groups.

Results

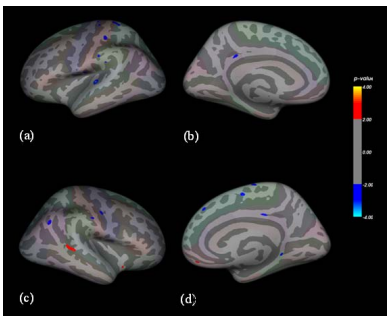

Figure 1 shows the results of group comparisons of cortical thickness. The hot color indicates thinner in patients whereas the cold colors thicker than the controls.

The areas with significant cortical thickness differences are summarized in the Table (BA = Broadman area, and x, y, and z are Talairach coordinates).

	Cortex area	BA	x	y	z
Normal > Patient					
Left hemisphere					
Frontal Lobe	lateralorbitfrontal	11	-29	38	-9
Right hemisphere					
Frontal Lobe	medialorbitofrontal	11	8	42	-16
Temporal Lobe	middle temporal gyrus		45	-41	4
Sub-lobar	Insula		35	8	-10
Patient > Normal					
Left hemisphere					
Frontal Lobe	precentral	4	-22	-25	49
Parietal Lobe	postcentral	2	-43	-27	46
	inferior parietal lobule	40	-54	-29	23
Temporal Lobe	superior temporal gyrus	22	-51	-19	3
Limbic Lobe	isthmuscingulate	31	-5	-37	31
Sub-lobar	insula	13	-46	-9	15
Right hemisphere					
Frontal Lobe	superiorfrontal	8	8	37	38
	precentral	6	40	-9	30
Parietal Lobe	postcentral		54	-15	33
Temporal Lobe	superior temporal gyrus	39	46	-56	30
Limbic Lobe	parahippocampal	30	17	-36	-4
	posteriorcingulate	23	5	-24	34

The orbitofrontal regions, previously found an important area for patients with depression orders, were found thinner in our patients as compared to the aged group. In addition, our results show many different regions arose in patients thicker than controls, such as in cingulate gyrus and parietal lobule etc. Those areas may play important roles as compensatory to the behavior changes in attention and executive functions in patients with late-life depression.

Conclusion

Our study showed reduced cortical thickness in orbitofrontal regions in patients with late-life depression, which agreed well with previous studies. In addition, we found several important functional areas appeared to be thicker cortical thickness in the patient group. Further study correlates with fMRI will be conducted to explore these areas in more details.

References

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