Research Article

MORPHOMETRIC ALTERATIONS OF ANTERIOR SUPERIOR TEMPORAL CORTEX IN OBSESSIVE–COMPULSIVE DISORDER

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The superior temporal gyrus (STG) may be involved in the pathophysiology of obsessive–compulsive disorder (OCD). Moreover, the anterior STG has rich interconnections with the orbitofrontal cortex and the amygdala, and plays a role in visuospatial processing, which is impaired in patients with OCD. This study was designed to examine the morphological abnormalities of the anterior STG and their relationships with visuospatial function and clinical symptom in patients with OCD. We measured gray matter volumes of the anterior STG (rostral STG and planum polare (PP)) by three-dimensional (3D) magnetic resonance imaging in age- and sex-matched groups, which consisted of 22 patients with OCD and 22 normal volunteers. Visuospatial function and clinical symptom were assessed using the Rey–Osterrieth Complex Figure (ROCF) test, the Yale–Brown Obsessive Compulsive Scale, and the Maudsley Obsessive Compulsive Inventory. We found significant volume reductions in bilateral PPs, but there were no significant correlations between brain volumes and the ROCF copy score, immediate or delayed recall score, and clinical symptom in patients with OCD. These results suggest that volume reduction of the anterior STG, especially the PP, may be related to the pathophysiology of OCD, but further research may be needed to explore a relationship of the PP volume change with cognitive impairment observed in patients with OCD. Depression and Anxiety 23:290–296, 2006. © 2006 Wiley-Liss, Inc.

Key words: obsessive–compulsive disorder; anterior superior temporal cortex; magnetic resonance imaging

INTRODUCTION

Obsessive–compulsive disorder (OCD) is characterized by recurrent intrusive thoughts and repetitive, ritualistic behaviors that cause anxiety or distress and are debilitating for patients. Many researchers have focused on the role of the frontosubcortical circuits in the pathophysiology of OCD. Hyperactivities of the frontosubcortical circuits including the orbitofrontal cortex (OFC), the anterior cingulate cortex, and/or the basal ganglia in patients with OCD have been consistent with functional imaging findings [Baxter et al., 1988; Maltby et al., 2005; Rauch et al., 1994; Swedo et al., 1989; van der Wee et al., 2003]. In addition, after successful treatment with pharmacotherapy, behavioral therapy, or psychosurgery, normal-

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ization of the metabolism was found [Benkelfat et al., 1990; Hansen et al., 2002; Kang et al., 2003; Nakao et al., 2005; Saxena et al., 1998; Swedo et al., 1992]. Also, some researchers have suggested the involvement of parietocerebellar dysfunction in the pathophysiology of OCD [Kang et al., 2003; Kwon et al., 2003]. On the other hand, other evidence indicate that the superior temporal cortex may be involved in the pathophysiology of OCD. Cottraux et al. [1996] reported that patients with OCD with checking rituals have higher regional cerebral blood flow (rCBF) in the superior temporal regions in a resting state, and that processing of both obsessive and neutral stimulation increases rCBF in the superior temporal regions, based on the findings of a functional magnetic resonance imaging (fMRI) study. Adler et al. [2000] suggested that OCD symptomatology is mediated by multiple brain regions, including the anterior cingulate, as well as the frontal and the temporal brain regions. However, though fMRI studies in OCD have reported activations in the superior temporal gyrus (STG), few have considered the role of the superior temporal cortex in the pathophysiology of OCD. In particular, no structural study in OCD has reported changes of the STG using an MRI parcellation method.

The STG is not a unitary region. It contains the primary auditory cortex and the auditory association cortical areas. Heschl's gyrus (HG) and the planum temporale are located in the middle to posterior part of supratemporal plane in the STG, whereas the planum polare (PP) is located in the anterior part of the STG. The STG is involved in visual, as well as auditory, processing [Ungerleider and Haxby, 1994]. In particular, the anterior portion of the STG receives input from both the dorsal and ventral visual streams, and therefore represents a site of multimodal sensory convergence [Oram and Perrett, 1996]. In other words, the anterior STG might act as an interface between the dorsal and the ventral pathways of input processing, and allow an exploration of object- and space-related information [Karnath, 2001]. In addition, the STG has rich connections to temporolimbic areas, neocortical association areas in the prefrontal and the parietal cortices, and the thalamus. Moreover, the anterior part of the STG projects to the orbitomedial frontal areas; the middle part of the STG projects to the dorsal aspects of the medial frontal lobe; and the posterior part of the STG projects to the lateral frontal cortex [Pandya, 1995]. All major subdivisions of the temporal neocortex, especially the rostral STG, receive projections from the amygdaloid complex [Amaral and Price, 1984].

There has been significant evidence to date indicating a role of the STG in the pathophysiology of symptoms of schizophrenia. Especially, some studies have noted a correlation between the left STG volume and the severity of hallucinations. Barta et al. [1990] first reported a volume reduction of the left anterior and middle STG in patients with schizophrenia and found a negative correlation between left anterior STG volume and auditory hallucinations. On the other hand, musical hallucinations as a particular type of auditory hallucinations are a disorder of complex sound processing in which the perception is formed by instrumental music, sounds, or songs [Berrios, 1990]. Hermesh et al. [2004] reported that musical hallucinations are frequent in patients with OCD and more suggestive of OCD than of other mental disorders. In addition, brain imaging studies of patients with musical hallucinations revealed a dysfunction of the temporal cortex [Griffiths, 2000; Kasai et al., 1999].

Patients with OCD have impairments in cognitive function. To date, many researchers have examined the association between OCD and cognitive impairment. In particular, some neuropsychological studies have demonstrated significant impairment of visuospatial function in patients with OCD [Galderisi et al., 1995; Okasha et al., 2000]. Given the interconnection of the anterior part of the STG with the OFC, and its involvement in visual processing and musical hallucination, it is possible that the anterior STG is involved in the pathophysiology of OCD.

In this study, we hypothesized that the gray matter volume of the anterior part of the STG, including the PP and the rostral STG, would be reduced in patients with OCD, and that there would be a relationship between regional brain volume changes and visuospatial dysfunction in patients with OCD. To test this hypothesis, we measured the gray matter volumes of the PP and the rostral STG in patients with OCD and normal comparisons using three-dimensional (3D) MRI.

**MATERIALS AND METHODS**

**SUBJECTS**

This study included 22 patients with OCD and 22 healthy normal subjects, matched for age, socioeconomic status (SES), and sex [Hollingshead and Redlich, 1958]. The demographic characteristics of the subjects are shown in Table 1. Each group contained 15 men and 7 women, who were all right-handed. There was a significant difference in IQs of patients with OCD and normal subjects ($t = 2.214, df = 42, P = .032$). Patients were recruited from the inpatient unit and from the outpatient clinics of Seoul National University Hospital (SNUH), and fulfilled DSM-IV criteria for OCD, as diagnosed using the Structured Clinical Interview for DSM-IV [SCID-IV; First et al., 1996]. The exclusion criteria included any lifetime history of neurological or significant medical illnesses and any history of substance abuse. We recruited normal volunteers from the community by placing newspaper advertisements. The exclusion criteria for normal subjects included any current or lifetime history of a DSM-IV Axis I disorder screened using the SCID-IV.
TABLE 1. Demographic and clinical characteristics in subjects

<table>
<thead>
<tr>
<th></th>
<th>OCD (N = 22)</th>
<th>Normal (N = 22)</th>
<th>Analysis</th>
<th>t</th>
<th>P</th>
</tr>
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<tbody>
<tr>
<td>Demographic</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Age</td>
<td>26.727 (7.239)</td>
<td>26.182 (6.052)</td>
<td></td>
<td>-0.271</td>
<td>.788</td>
</tr>
<tr>
<td>Sex</td>
<td>–</td>
<td>–</td>
<td></td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>SES</td>
<td>3.182 (0.665)</td>
<td>3.000 (0.690)</td>
<td></td>
<td>-0.890</td>
<td>.378</td>
</tr>
<tr>
<td>Education</td>
<td>14.182 (1.918)</td>
<td>14.818 (1.468)</td>
<td></td>
<td>1.236</td>
<td>.223</td>
</tr>
<tr>
<td>IQ</td>
<td>107.682 (10.490)</td>
<td>114.318 (9.357)</td>
<td></td>
<td>2.214</td>
<td>.032</td>
</tr>
<tr>
<td>Clinical</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Duration of illness</td>
<td>8.263 (6.410)</td>
<td>–</td>
<td></td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>YBOCS</td>
<td>–</td>
<td>–</td>
<td></td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Obsession</td>
<td>12.737 (2.786)</td>
<td>–</td>
<td></td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Compulsion</td>
<td>10.632 (4.462)</td>
<td>–</td>
<td></td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Total</td>
<td>23.316 (6.913)</td>
<td>–</td>
<td></td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>MOCI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>12.600 (6.793)</td>
<td>–</td>
<td></td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>ROCF</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Copy</td>
<td>33.119 (2.828)</td>
<td>33.568 (1.841)</td>
<td></td>
<td>0.620</td>
<td>.539</td>
</tr>
<tr>
<td>Immediate recall</td>
<td>15.381 (6.991)</td>
<td>22.773 (5.686)</td>
<td></td>
<td>3.812</td>
<td>.000</td>
</tr>
<tr>
<td>Delayed recall</td>
<td>16.833 (5.425)</td>
<td>22.614 (5.092)</td>
<td></td>
<td>3.604</td>
<td>.001</td>
</tr>
</tbody>
</table>

OCD, obsessive-compulsive disorder; SES, socioeconomic status; YBOCS, Yale-Brown Obsessive Compulsive Scale; MOCI, Maudsley Obsessive Compulsive Inventory; ROCF, Rey-Osterrieth Complex Figure.

*Independent sample t-test was used. Data are given as mean (SD) unless otherwise noted.

b df = 42, P < .050.

Two patients with OCD had a comorbidity of major depressive disorder. Seven patients with OCD were drug-naive, and 15 had been administered antiobsessional medication (including a history of combined therapy with neuroleptics in four patients). However, all had remained psychotropic drug free for at least 4 weeks prior to the time of neuropsychological and clinical symptom measurements. In addition, there was an interval of about 2 weeks between neuropsychological and clinical symptom measurements and MRI scanning.

Clinical and cognitive assessments were conducted using the Yale–Brown Obsessive Compulsive Scale [Y-BOCS; Goodman et al., 1989] and the Maudsley Obsessive Compulsive Inventory [MOCI; Rachman and Hodgson, 1980] for OCD symptom severity, and the Rey–Osterrieth Complex Figure (ROCF) test for visuospatial construction and nonverbal memory [Lezak, 1995], respectively. To obtain an IQ estimate, the Vocabulary, Arithmetic, Block Design, Picture Arrangement, and Digit Span subscales, which were included in the Korean version of the Wechsler Adult Intelligence Scale (K-WAIS), were administered to all subjects.

We obtained written informed consent from all subjects after they received a complete description of the scope of the study. This study was carried out under the guidelines established by the institutional review board of the SNUH for the use of human subjects.

MR IMAGE ACQUISITION AND PROCESSING

Image acquisition and processing have been described previously [Choi et al., 2004]. Briefly, we acquired 3D T1-weighted spoiled gradient echo magnetic resonance (MR) images on a 1.5 Tesla GE SIGNA Scanner (GE Medical System, Milwaukee, WI) using the following imaging parameters: 1.5 mm sagittal slices, echo time 5.5 ms, repetition time 14.4 ms, number of excitations 1, rotation angle 20°, field of view 21 x 21 cm, and a matrix of 256 x 256. MR images were processed using an image-processing software package, ANALYZE (Version 4.1, Mayo Foundation, Rochester, MN). Images were resampled to 1.0 mm3 voxels, reoriented, and spatially realigned to the conventional position. The data sets were then filtered using anisotropic diffusion methods [Perona and Malik, 1990] to improve the signal to noise ratio. In order to extract the brain, tissues exterior to the brain were removed by the semiautomated region growing method. By employing the fuzzy C-means algorithm [Bensaid et al., 1996; Cannon et al., 1986], the extracted brain images were segmented into gray matter, white matter, and cerebrospinal fluid. Intracranial volume was calculated by summing up the subtotal volumes of these three components.

VOLUME MEASUREMENTS

We focused upon the four anterior portions of the STG, namely, the right and left rostral STG, and
the right and left PP. Boundary definitions and the method of tracing each brain region were performed as described by Kim et al. [2000], with minor modifications.

**Rostral STG.** A coronal plane including the most anterior point of the Heschl’s sulcus (HS; plane A) divides the STG into rostral and caudal portions. The rostral STG was traced on serial coronal slices. Tracing was done along the lateral rim of the supratemporal plane dorsally and along the deepest point of the superior temporal sulcus (STS) ventrally (Fig. 1). Tracing was started at plane A and continued rostrally to the coronal plane, including the temporofrontal junction.

**Planum polare.** The PP was traced on the coronal plane. Tracing began at the level of the posterior end of the first transverse sulcus (FTS) and continued, following the FTS laterally and the circular sulcus of the insula (CSI) medially. On the level of the anterior tip of the HS, the lateral border of the PP was changed from the FTS to the lateral rim of the supratemporal plane (Fig. 1). Tracing ended on the coronal plane, including the temporofrontal junction.

A reliability study was performed upon structural volume determinations in 10 subjects by two raters. The intraclass correlation coefficients (ICCs) for the reliability of gray matter volume determinations were as follows: the rostral STG (.91, .98 [right and left, respectively]), the PP (.99, .99). Raters were unaware of the names and diagnoses of subjects.

**STATISTICAL ANALYSIS**

All measures of regional brain gray matter volumes were subjected to multivariate analysis of variance with group (OCD, normal subjects) as between-subjects factor, with the intracranial volume (ICV) as a covariate. In the exploratory analysis, we used the Pearson product correlation method to see the relationship between regional brain volumes, clinical symptoms, and cognitive performances. All analyses were two-tailed and the significance level was set at \( \alpha = .05 \).

**RESULTS**

A comparison of the volumetric measures of the two groups is presented in Table 2. A significant group effect was observed with respect to the right and left PP volume. The gray matter volumes of both PPs were significantly reduced in patients with OCD.

As shown in Table 1, patients with OCD were impaired in terms of the immediate and delayed recall of the ROCF. However, there was no significant difference between the two groups in terms of the ROCF copy score. In the exploratory analysis, no significant correlation was found between brain volumes and the ROCF copy score and immediate or delayed recall score in patients with OCD. In addition, regional brain gray matter volumes were not correlated with the MOCI score and the Y-BOCS scores in patients with OCD. There were also no significant correlations between IQ and regional brain volumes.

**TABLE 2. Anterior superior temporal cortex gray matter volumes (ml)**

<table>
<thead>
<tr>
<th></th>
<th>OCD (N = 22)</th>
<th>Normal (N = 22)</th>
<th>Analysis</th>
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<tbody>
<tr>
<td>ICV</td>
<td>1420.705 (134.100)</td>
<td>1354.632 (98.891)</td>
<td></td>
</tr>
<tr>
<td>Planum polare</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right</td>
<td>1.188 (0.351)</td>
<td>1.480 (0.470)</td>
<td>7.224</td>
</tr>
<tr>
<td>Left</td>
<td>1.250 (0.361)</td>
<td>1.618 (0.542)</td>
<td>7.962</td>
</tr>
<tr>
<td>Rostral STG</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right</td>
<td>0.647 (0.417)</td>
<td>0.928 (0.548)</td>
<td>3.876</td>
</tr>
<tr>
<td>Left</td>
<td>0.916 (0.585)</td>
<td>1.089 (0.497)</td>
<td>0.566</td>
</tr>
</tbody>
</table>

aData are given as mean (SD); and \( df = 1, 42 \).

b\( P < .050 \).

Figure 1. Tracing of the planum polare (a) and the rostral superior temporal cortex (b) on the coronal slice.
DISCUSSION

To our knowledge, this is the first MRI study to investigate abnormalities of the superior temporal cortex subregion in patients with OCD. Based on current findings, we suggest that volume reduction of the anterior STG, especially of the PP gray matter volume, may be involved in the pathophysiology of OCD.

In this study, a significant decrease in the bilateral PP gray matter volume was found in patients with OCD. The STG is located along the Sylvian fissure dorsally and the STS ventrally, and is subdivided into several regions both structurally and functionally. Of these regions, the PP subregion is located anterior to the HG and in the supratemporal plane of the anterior STG. Though the PP is considered to serve as a secondary auditory cortex, its specific function remains unknown. Moreover, no study has independently investigated the PP. To date, many researchers have been concerned about a role of the STG in the auditory processing [Brugge et al., 2003; Howard et al., 2000], whereas few have studied the implications of the visual processing of the STG from a psychiatric standpoint. The visual processing system is composed of two different pathways. The ventral pathway, from the primary visual cortex to the inferior temporal cortex, is thought to process form, or “what” an object is, whereas the dorsal pathway, which projects into the posterior parietal cortex, provides information about “where” an object is [Baizer et al., 1991; Ungerleider and Haxby, 1994; Young, 1992]. The STG receives afferent inputs from the inferior temporal cortex, as well as from the inferior parietal lobe and the intraparietal sulcus [Seltzer and Pandya, 1994]. In particular, the anterior part of the STG is located at a transitional position between the two visual processing pathways. Therefore, diverse views have been expressed upon the function of the anterior portion of the STG. For example, Bruce et al. [1981] suggested that the function of the anterior STG concerned visuospatial analyses, whereas others presumed that it was involved in complex visual object recognition [Baylis et al., 1987]. However, it might be considered that both types of information from both pathways converge in the anterior portion of the STG [Oram and Perrett, 1996]. Thus, the anterior STG might act as an interface between the dorsal and the ventral pathways of input processing, and allow an exploration of object- and space-related information [Karnath, 2001].

On the other hand, the anterior part of the STG has rich reciprocal interconnections with other cortical and subcortical areas, that is, with the orbitomedial frontal areas [Pandya, 1995], the ventral portion of the putamen and the caudate nucleus [Veteran and Pandya, 1998], and the amygdala [Amaral and Price, 1984]. With regard to the OFC, our group [Choi et al., 2004] reported that the left anterior OFC gray matter volume was correlated positively with the ROCF copy score, suggesting that volume reduction of this brain region might be related to impaired organizational strategies in patients with OCD. Therefore, based on the role of the anterior STG upon an exploration of object- and space-related information and the interconnection between the anterior STG and the OFC, it is possible that volume reduction of the anterior STG is associated with visuospatial dysfunction in patients with OCD. To examine the possible relationship of the anterior STG gray matter volume with the ROCF copy score, we used an exploratory analysis preliminarily in this study. However, there was no significant correlation between the gray matter volume of the PP and the rostral STG and the ROCF copy score. Previous studies have suggested that patients with OCD were primarily impaired in strategic processing, which then had secondary effects on immediate and delayed recall, suggesting the hypothesis of frontostriatal dysfunction in OCD [Savage et al., 1999, 2000]. In addition, Boldrini et al. [2005] reported that visuoconstructive deficits seemed specific in OCD and supported the ventral frontostriatal circuit involvement in OCD. According to Aycicegi et al. [2003], patients with OCD demonstrated performance deficits on measures of some neuropsychological tests, including visuoconstructual ability, that were consistent with the contention that dysfunction of the orbitofrontal-limbic network underlies OCD. However, Shin et al. [2004] reported that using a qualitative scoring system for the ROCF, patients with OCD had impairments only in planning and organization, not in visuo-perceptual and visuoconstructual ability during copying. Although visuospatial deficits linked to organizational problems in OCD may be largely mediated by the OFC, visuospatial ability may be independently influenced by other brain regions, such as the temporal cortex or the parietal cortex [Coull and Nobre, 1998; Nobre et al., 1997; Rushworth et al., 2001]. To evaluate specifically visuospatial function in OCD, further research with various neuropsychological tests is needed.

In terms of musical hallucinations, this phenomenon may be experienced under a variety of conditions, including diseases of the ear, neurological and psychiatric disorders, toxic states, and as a side effect of antidepressant treatment. However, there is no clear evidence as to which of these is the underlying etiology of musical hallucinations. Some aspects of musical hallucinations might be explained by the concept of OCD [Matsui et al., 2003]. Moreover, Hermesh et al. [2004] reported that musical hallucinations are more common among psychiatric patients than previously reported, and more suggestive of OCD than of other mental disorders. Brain imaging studies of patients with musical hallucinations have indicated dysfunction of the STG, especially the auditory association cortex [Griffiths, 2000; Kasai et al., 1999]. However, other brain areas are also involved, in particular, distinct parts of the frontal lobe. Izumi et al. [2002] reported that
rCBF is increased in the bilateral lower frontal area and the bilateral basal ganglia during musical hallucinations, indicating that the patterns of brain activations are different between musical and verbal hallucinations within one patient. Therefore, further research is needed to clarify the brain substrate associated with musical hallucinations observed in patients with OCD.

The limitation of this study is that patients with OCD who participated in this study were not drug naïve, and 15 of 22 patients had a history of antiobsessional medication. Therefore, we could not exclude completely the possibility of the effects of various medications on cognitive performances and brain volumes, although we made efforts to minimize the effects of medications.

**CONCLUSIONS**

These results indicate that a volume reduction of the anterior part of the STG, especially the PP, may be related to the pathophysiology of OCD, and further studies are needed to investigate the relationship between deficits in the anterior STG and cognitive impairment in OCD.

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