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ORIGINAL PAPER

MRI study of the cavum septum pellucidum in obsessive–compulsive disorder

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Abstract The cavum septum pellucidum (CSP), a putative marker of neurodevelopmental anomaly, has been associated with an increased risk of several psychiatric disorders. The purpose of this study was to evaluate the CSP in patients with obsessive–compulsive disorder (OCD) compared with healthy control subjects. Seventy-one patients with OCD and 71 healthy volunteers matched for age and sex were evaluated with magnetic resonance imaging. We evaluated the CSP using criteria employed in previous studies: presence of the CSP, length of the CSP, and overall size of the CSP, measured in five grades, ranging from grades 0 (no CSP) to 4 (severe CSP). We evaluated OCD symptom severity using the Yale–Brown Obsessive–Compulsive Scale (Y-BOCS). The CSP presence was significantly greater in the OCD group (60.6%) than in control subjects (29.6%), and CSP size grade was significantly larger in the OCD group ($\chi^2 = 15.609$, $P = 0.004$). CSP length showed no significant group difference. Among patients with OCD, those with a CSP had higher scores on the obsession subscale of the Y-BOCS than those without a CSP ($Z = -2.358$, $P = 0.018$), while they did not show significant difference from those without a CSP in the compulsion subscale of the Y-BOCS, age, duration of illness, or age at onset. These results indicate that neurodevelopmental alterations in midline structures might contribute to the pathogenesis of OCD.

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Introduction

Obsessive–compulsive disorder (OCD) is a disabling psychiatric disorder, often with a chronic course [6]. It is quite common in the general population, with a 1-month prevalence of 0.3–3.1% [14]. Its pathogenesis is considered to be a combination of both genetic vulnerability and environmental influence, although relatively little is known about how the two factors interact [19].

Some studies have suggested that a neurodevelopmental model may facilitate understanding of OCD [5, 39]. The onset of OCD is seen in children as young as 5–8 years of age [15], and it is often comorbid with Tourette's syndrome, a known neurodevelopmental disorder [37]. Many family studies have found increased incidence of OCD in first-degree relatives of patients with OCD [14], and twin studies show a greater concordance rate for monozygotic than for dizygotic twins (67.5 vs. 31.0%, respectively) [3]. These findings suggest the presence of pathophysiological processes early in life, sometimes before the onset of overt obsessive–compulsive symptoms and implicate neurodevelopmental disturbance as one of the likely pathogenetic contributors to OCD.

Both structural and functional neuroimaging studies have shown the dysfunction [19], or more specifically, the imbalance [21], of the fronto-striatal-thalamic loop in patients with OCD, identifying the neural circuit underlying OCD as a neurobiological phenomenon. The fact that neurological soft signs, which are known to be suggestive of dysfunctional cortico-cerebellar-thalamic-cortical circuit

in patients with schizophrenia [9, 47] are sometimes seen in healthy subjects who later develop OCD [34], also points to this direction. However, whether this dysfunctional circuit is evidence of disturbed neurodevelopment is unclear, as both structural and functional abnormalities in OCD can be reversed by pharmacotherapy [17, 23, 48].

The cavum septum pellucidum (CSP) has been identified as a putative marker of disrupted earlier neurodevelopment, and its presence has been examined in several psychiatric disorders, including schizophrenia and schizotypal personality disorder [11, 30], bipolar disorder [28], and Tourette's syndrome [27].

The septum pellucidum, a thin vertical partition of white matter separating the two lateral ventricles, is composed of two laminae [42]. During the fifth and sixth weeks of gestational age, these laminae usually become fused. Nonunion of the two laminae of the septum pellucidum results in CSP, a space between them [4].

Less is known about the role of the fiber tracts and sparse neurons contained in the septum pellucidum other than the structure's role as a "relay station" for the limbic system, with connections to the hippocampus and hypothalamus [42]. However, the septum pellucidum is linked closely with the corpus callosum and the hippocampus in development [43], and it probably closes due to their rapid growth [42]. Although some reports have identified incidences of a CSP after blows to the head in boxers [33] or as a complication of surgical procedures [44], the CSP is thought to occur primarily due to the developmental nonunion of the two septal laminae. Hence, the CSP appears to reflect abnormal growth of the limbic structure.

Given the importance of the limbic structure in the pathophysiology of OCD [40], the presence of the CSP in patients with OCD could help in clarifying the role of neurodevelopment in OCD. To our knowledge, no one has yet studied the presence of the CSP in patients with OCD, with the exception of one study whose focus was on Tourette's syndrome [27]. The present study sought to compare the incidence and size of the CSP in patients with OCD and normal control subjects, with our primary hypothesis being that a CSP would be more prevalent among patients with OCD and/or that measures of CSP magnitude would be greater in these patients than in the control group.

Methods

Subjects

Seventy-one patients with OCD were recruited from Seoul National University Hospital; an equal number of and age- and sex-matched healthy control subjects were recruited from the community, through an Internet advertisement or the social networks of hospital staff. These were the same subjects as those described in a previous report by our group [49]. Each group was composed of 47 men and 24 women. The mean ages of the patients and the control subjects were 25.68 years (SD = 6.09) and 26.61 years (SD = 7.50), respectively.

All patients met the criteria set by the Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV) for OCD diagnosis using the Structured Clinical Interview for DSM-IV (SCID) [12]. Patients' mean age at the onset of OCD was 18.59 years (SD = 6.52). At the time of the study, patients had been ill for an average of 8.02 years (SD = 6.10). The severity of OCD symptoms was assessed using the Yale–Brown Obsessive–Compulsive Scale (Y-BOCS) [18]. Mean scores for obsessive symptoms, compulsive symptoms, and total scale were 12.35 (SD = 4.31), 10.49 (SD = 5.00), and 22.84 (SD = 8.40), respectively. Four patients with OCD had a history of major depressive disorder and three had a history of another anxiety disorder. Eighteen patients were taking a selective serotonin-reuptake inhibitor (fluoxetine equivalent mean daily dose = 57.8 mg, SD = 25.1 mg) and eight of these 18 subjects were also taking an atypical antipsychotic agent (chlorpromazine equivalent mean daily dose = 75.0 mg, SD = 57.7 mg). The remaining 53 patients had been medication free for at least 4 weeks at the time of this study.

We screened control subjects using the SCID to exclude any concurrent or lifetime history of DSM-IV Axis I Disorders. None of the control subjects had a history of any major neurological or medical illnesses or of substance abuse. Neither did they have any relatives with psychiatric disorders within the third degree. The study protocol and forms were approved by the institutional review board and we obtained written informed consent from all subjects.

Magnetic resonance imaging acquisition and image processing

We used a 1.5-T GE SIGNA Scanner (GE Medical Systems, Milwaukee, MI, USA) to obtain three-dimensional T1-weighted spoiled gradient-echo magnetic resonance (MR) images. The following parameters were used in obtaining sagittal images: slice thickness = 1.5 mm, echo time = 5.5 ms, repetition time = 14.4 ms, number of excitations = 1, rotation angle = 20°, field of view = 21 × 21 cm, and matrix size = 256 × 256. We used the imageprocessing software ANALYZE (version 7.0; Mayo Foundation, Rochester, MN, USA) to process the MR images. We resampled all MR images to 1.0 mm^3 iso-voxels, and spatially realigned images along the anteroposterior axis of the brain parallel to the

intercommissural line; the other two axes were aligned along the interhemispheric fissure.

Measurements of the CSP

To determine the presence and dimensions of the CSP, we integrated criteria that had been used frequently in previous studies, just as we had in our previous work focusing on the CSP in individuals at high risk for developing psychosis [8]. First, the presence of the CSP was defined as its appearance on at least one coronal magnetic resonance imaging slice. Second, length of the CSP was determined to the nearest 1.0 mm by counting the number of contiguous slices on which it was present. We defined any CSP equal to or greater than 6 mm in length, that is, occurring on six or more slices, as being abnormally large [11, 30, 36]. Third, to establish the grade of the CSP, we used a system based on considerations of length, width, and overall size. The grades were as follows: 0 (absent), 1 (questionable), 2 (mild), 3 (moderate), and 4 (severe). We assigned a rating of 0 if an intact septum pellucidum was clearly visible in all slices (i.e., no CSP was present), and a rating of 1 if the septum was unclear (i.e., questionable CSP). We assigned a rating of 2 if a distinct CSP space with cerebrospinal fluid signal intensity was present, but the space was not wider than the thickness of the septum, 3 if CSP space was wider than the septum thickness, but occupied less than half of the ventricular space, and 4 if the CSP occupied at least half of the ventricular space [8, 27].

Reliability of the ratings was aided by comparison with a set of standard CSP images for each ordinal grade (Fig. 1). Based on the definition provided by Degreef et al. [10], we defined grades 0–1 as normal CSP and grades 2–4 as abnormal CSP. For all cases, a single rater (M.W.C), who was blind to the group membership of the cases, determined the presence and grade of the CSP as well as the number of slices showing the CSP. We selected 50 cases at random for reliability measures; all of these cases were reviewed by two individual raters (M.W.C, J.S.C.). The intra-class correlation coefficient for inter-rater reliability was 0.97 for the number of slices and 0.95 for the grade.

Statistical analysis

We used χ^2 and linear by linear association tests to assess differences between the OCD and control groups in the presence and grade of the CSP. We also assessed sex differences in the presence of the CSP using a χ^2 test. Mann–Whitney tests assessed differences between the patient and control groups, or between patients with CSP and those without, on the dimensions of age, duration of illness, age at onset, and symptom severity. Spearman's correlation was used to assess if age, duration of illness, age at onset or dose of medication was correlated with CSP grade. Two-tailed $P < 0.05$ was considered statistically significant.

Results

Data on the presence, length and grade of the CSP in the two groups are presented in Table 1. The presence of any CSP was significantly more frequent in the OCD group ($\chi^2 = 13.768$, $P < 0.001$), but the presence of an abnormally large ($\geq$6 mm) CSP was not ($\chi^2 = 0.530$, $P = 0.467$). The results showed no significant difference between groups in the presence of an abnormal CSP, defined as grade $\geq$2 ($\chi^2 = 1.659$, $P = 0.193$).

All three patients with OCD who were left handed had a CSP. However, the more frequent appearance of CSP in the OCD group compared with controls was still significant ($\chi^2 = 10.374$, $P = 0.0012$) even after these three subjects were excluded from the analysis. Male patients appeared more likely to have a CSP, but not to a statistically significant degree ($\chi^2 = 3.294$, $P = 0.070$). We compared patients with a CSP and those without a CSP in terms of age at onset, duration of illness, and Y-BOCS scores (total score, obsession subscale, and compulsion subscale) (Table 2). Mann–Whitney test indicated that the mean Y-BOCS obsession score was significantly higher in patients with OCD who had a CSP than in those without a CSP ($Z = -2.358$, $P = 0.018$), even after Bonferroni correction. This finding remained still significant ($Z = -2.530$, $P = 0.011$) even when the medicated subjects ($n = 18$) were excluded from the analysis. However, differences were not significant in the mean Y-BOCS compulsion subscale or mean total scores. In medicated subjects, Pearson's correlation showed that the grade of the CSP was not significantly correlated with the dose of either selective serotonin-reuptake inhibitor ($P = 0.390$) or atypical antipsychotic agent ($P = 0.498$). No significant differences were seen between the CSP and non-CSP groups in terms of age, duration of illness or age at onset, and the CSP grade bore no correlation with these variables.

Discussion

We found a significant difference in the presence of any CSP between patients with OCD and normal subjects, but not in the frequency of an abnormally large CSP. Patients with OCD who had a CSP scored higher on the Y-BOCS obsession subscale, suggesting a possible relationship between the presence of any CSP and the severity of symptoms.

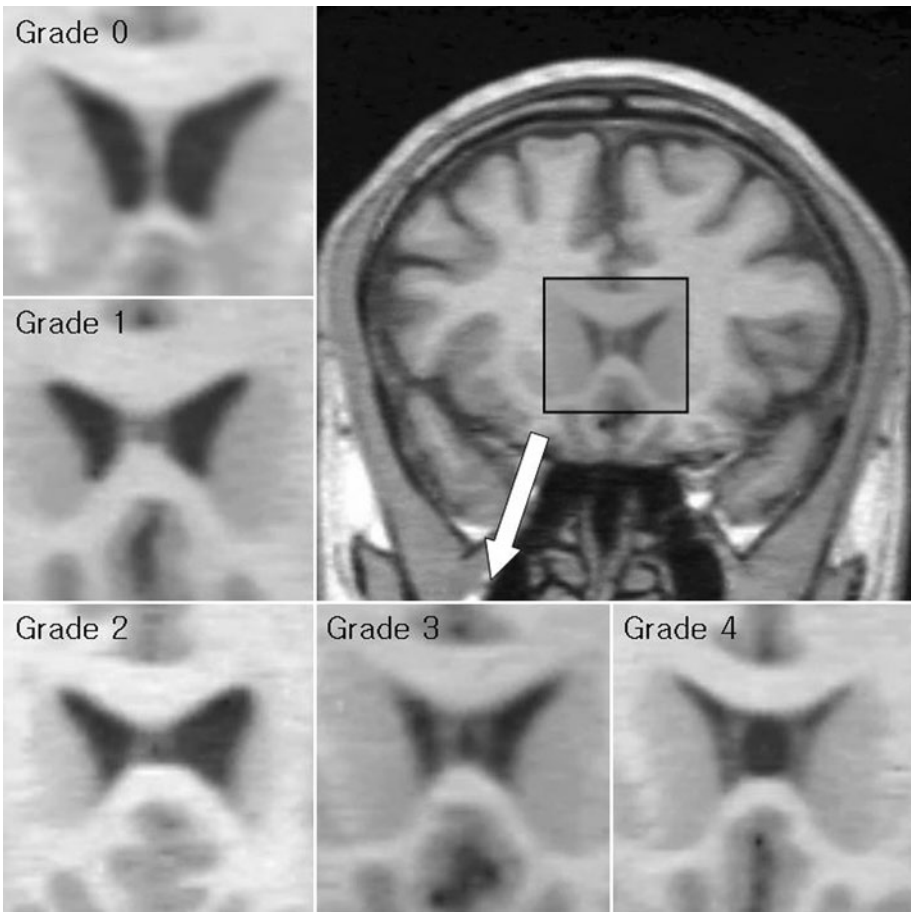

Fig. 1 Illustrative examples of the different grades of the cavum septum pellucidum

Table 1 Comparison of CSP in OCD and control group

	Control ($n = 71$)	OCD ($n = 71$)	Analysis	
			χ^2	P
Grade				
0	50 (70.4%)	28 (39.4%)		
1	11 (15.5%)	27 (38.0%)		
2	7 (9.9%)	14 (19.7%)		
3	2 (2.8%)	1 (1.4%)		
4	1 (1.4%)	1 (1.4%)	15.609*	0.004*†
CSP presence (grade ≥1)	21 (29.6%)	43 (60.6%)	13.768	<0.001†
Abnormal CSP (grade ≥2)	10 (14.1%)	16 (22.5%)	1.695	0.193
Abnormally large CSP (≥6 mm)	3 (4.2%)	5 (7.0%)	0.530	0.467

CSP cavum septum pellucidum, *OCD* obsessive–compulsive disorder

*For a 2 x 5 table, $df = 4$

†$P < 0.05$

Because previous studies found that the occurrence of a grade 1 CSP was no more frequent in patients with schizophrenia or bipolar disorder than in normal control subjects [13, 26, 30, 35], having a grade 1 CSP has often been regarded as being within the range of normal variance. However, because patients with bipolar disorder showed a greater prevalence of a CSP, including a grade 1 CSP [45], we did not exclude the possible significance of a

Table 2 Demographic and clinical data within OCD group according to the presence of CSP

OCD ($n = 71$)	CSP present ($n = 43$)	CSP absent ($n = 28$)	Analysis	
			χ^2	P
Handedness				
Right handed	40 (93.0%)	28 (100.0%)	2.040	0.153
Left handed	3 (7.0%)	0 (0.0%)		
Sex				
M	32 (74.4%)	15 (53.6%)	3.294	0.070
F	11 (25.6%)	13 (46.4%)		
OCD ($n = 71$)	CSP present ($n = 43$)	CSP absent ($n = 28$)	Z	P
Age	26.86 ± 8.13	26.21 ± 6.52	−0.112	0.911
Onset age	19.02 ± 7.03	17.93 ± 5.70	−0.549	0.583
Duration of illness	8.06 ± 6.52	7.98 ± 5.52	−0.183	0.855
YBOCS				
Obsession	13.64 ± 2.66	11.69 ± 4.37	−2.358	0.018*
Compulsion	11.26 ± 4.44	10.46 ± 4.96	−0.842	0.400
Total	24.90 ± 5.98	22.15 ± 8.30	−1.375	0.169

CSP cavum septum pellucidum, *OCD* obsessive–compulsive disorder, *YBOCS* Yale–Brown Obsessive–Compulsive Scale

* $P < 0.05$

grade 1 CSP in the present research. Our results suggest that a grade 1 CSP (i.e., a questionable CSP) might have clinical significance in OCD.

The presence of a CSP may be a marker for developmental dysgenesis of midline structures rather than for a specific disorder. In this respect, our results could be in accordance with the previous neuroimaging studies on the shared abnormality of patients with OCD and patients with schizophrenia. Structural defect common to these two disorders occur not only in some parts of the prefrontal cortex [20], but also in a number of limbic structures. A nonspecific bilateral hippocampal volume reduction [31] and shape deformity of the hippocampus [24, 32] or thalamic surface [25] in both patients with schizophrenia and those with OCD support the possible common role of limbic dysgenesis in the pathogenesis of these two disorders. As mentioned above, most previous studies found CSP of at least grade 2 to be associated with schizophrenia, whereas our OCD patients showed increased prevalence of CSP only when grade 1 was included. Therefore, our results cannot be the direct evidence that the two disorders share the same limbic pathology. However, from the volume reduction in specific brain regions [38] to fractal dimensions of the skeletonized cerebral cortical surface [22], patients with OCD show abnormalities quite similar to, but less in severity than, those seen in patients with schizophrenia. Therefore, our findings suggesting an association between OCD and a subtler form of CSP might reflect a difference in the degree of structural anomaly between the two disorders. Had we included patients with schizophrenia in our study, we could have examined this postulated difference across diagnoses.

In patients with schizophrenia spectrum disorders, the presence of a CSP has been correlated with cognitive dysfunction, specifically, poor performance in the Wechsler Adult Intelligence Scale–Revised Full Scale IQ [35] and verbal learning and memory [13]. Patients with OCD also tend to show both verbal and nonverbal memory deficits [7, 40], but the cognitive deficit is not as severe as in patients with schizophrenia [29]. Perhaps, the neurocognitive deficits shared by schizophrenia and OCD constitute evidence of common limbic dysgenesis, with the occurrence of the CSP in both disorders being one piece of indirect anatomical evidence. This hypothesis suggests further studies examining the relationship between cognitive function and the presence of CSP in patients with OCD.

In contrast to the previous studies involving schizophrenia or bipolar disorder [26, 28], we failed to demonstrate any significant difference in the occurrence of a CSP (grade ≥2) or in the incidence of an abnormally large CSP (≥6 mm). Yet, patients with OCD in the current study showed a slightly more frequent abnormal CSP or an abnormally large CSP than the healthy control group, although the difference was not statistically significant. The number of subjects in the present study may have been insufficient to detect subtle differences in the occurrence of an abnormal or abnormally large CSP between the OCD and control groups. However, given that the number of subjects in the present study was similar to that in most previous studies on the CSP, our results are more likely to

reflect a lesser degree of structural abnormality in patients with OCD than has been reported for patients with schizophrenia or bipolar disorder.

Our findings show that patients having OCD with a CSP had higher scores in the Y-BOCS obsession subscale, suggesting that the CSP has an effect on symptom severity in these patients. This is in accordance with one previous diffusion-tensor imaging study [41], in which fractional anisotropy in the rostrum of corpus callosum negatively correlated with Y-BOCS score.

The current study has some limitations. Although MR scanning with a slice thickness of 1.5 mm is often used in the current literature [46], MR studies with thinner slices [8] might reveal what ones with thicker slices do not. In this respect, thinner slices could have resulted in a more accurate analysis. Also, subjects with OCD in this study were heterogeneous in their medication status, and the medication status in patients with OCD is known to affect structural imaging findings [17], although our results show that medication did not significantly affect our findings. In addition, previous studies on patients with bipolar disorder showed different patterns of CSP occurrence depending on the disease course and duration of illness [28, 45]. Similarly, specific obsessive–compulsive symptom dimensions such as hoarding [2, 16], or the absence of insight [1], independently affect the structural abnormality. However, the design of the current study did not allow for determining the impact of medication status, duration of illness, or disease course. These forms of heterogeneity among subjects with OCD may have contributed to the dilution of our results. Subsequent studies with larger samples or more homogeneous subject groups would further elucidate these limitations. Lack of measures for other evidences of early neurodevelopmental disturbances, or the missing correlation between the age at onset and presence or grade of CSP also precludes from reaching a firm conclusion, although this remains to be validated in further studies.

Conclusion

Patients with OCD in our study more frequently showed the presence of a CSP than the normal control subjects. Moreover, the presence of a CSP may be associated with the severity of obsessive symptoms. Our findings add to existing morphologic evidence supporting the role of subtle neurodevelopmental abnormalities in the pathogenesis of OCD.

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