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Neuroanatomical study

Hippocampus and amygdalar volumes in patients with obsessive-compulsive personality disorder

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ABSTRACT

Objectives: Moving from the point that there might be an association between the neuroanatomy of obsessive-compulsive disorder (OCD) and obsessive-compulsive personality disorder, we decided to examine the volumes of hippocampus and amygdala of patients with obsessive-compulsive personality disorder, which was previously evaluated in OCD patients by us.
Methods: Volumes of the hippocampus, and amygdala were measured by magnetic resonance imaging (MRI) in patients with obsessive-compulsive personality disorder and healthy control subjects. Manual tracing was used.
Results: We detected that the mean left and right sides of hippocampus and amygdala volumes of the patients with obsessive-compulsive personality disorder were smaller than those of the healthy controls.
Conclusion: Consequently, our present results suggest that hippocampal and amygdalar structural abnormalities may be related to the neuroanatomy of obsessive-compulsive personality disorder. However, it is required novel studies with larger sample.

1. Introduction

In the classification of psychiatric disorders, personality disorder is defined as a severe disturbance in characterological constitution and behavioral tendencies, usually consisting of several areas of the personality and leading to considerable personal and social problem. On the othre hand, the main clinical characteristics of them are an resistant, pervasive, and inflexible patterns of inner experience and behavior that detach from cultural expectations and lead to distress or impairment. Basically, features of personality disorders appear in late childhood or adolescence and continue in a stable manner in the period of adulthood. Obsessive-compulsive personality disorder (OCPD) has been established in this name in the DSM-IV [1] and DSM-5 [2]. However, it has been named as anankastic personality disorder in the ICD-10 [3]. According to the DSM system, it is an Axis II disorder mainly characterized by perfectionism, preoccupation with orderliness, and mental and interpersonal control, at the expense of flexibility, openness, and efficiency. OCPD has been classified under C Cluster Personality Disorders in last version of DSM, DSM 5. Nestadt et al. reported that OCPD had a prevalence rate of 1–2% in general population [4]. In daily clinical practice, we can easily observe that OCPD has discriminative features rather than those of other personality disorders. Grant et al. [5] reported that the OCPD was the most prevalent personality disorder in ourpatient settings. It should be [4] emphasized that growing knowledge leads to the fact that OCPD seems to be a neurocognitive function disorder rather than a personality disorder [6]. Skodol et al. [7] reported patients with OCPD to have less association with functional disability compared to other DSM 5 personality disorders. In clinical practice, it is clear that when mentioning about a personality disorder, particularly B Cluster and then A Cluster ones came in to mind rather than OCPD and other C Cluster personality disorders. We should admit that our knowledge on how to occur personality disorders is limited to psychoanalytical school. In this context, we know a little about the neurobiological and neuroanatomical etiopathogenesis of OCPD. Thus, there is not enough structural and functional neuroimaging study directly related to the OCPD. Payer et al. [8] investigated thirty-seven individuals who implicated personality disorder symptomatology exceeding DSM-IV Axis-II screening thresholds, who were Cluster B (n = 20), from Cluster C (n = 28) and comorbidity of Cluster A and previous both groups (n = 11), and thirty-five age, and gender matched healthy subjects. The authors revealed that Cluster C personality disorder symptoms were associated with larger striatal surface area localized to the caudate tail, smaller ventral striatum volumes, and greater cortical

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thickness in the right prefrontal cortex compared to healthy comparison subjects whle both Cluster B and C personality disorder symptoms were linked to trends to greater posterior caudate volumes and orbitofrontal surface area abnormalities, concluding that morphological abnormalities could be extensively contributing to Cluster C personality disorder symptoms. In addition, Kim et al. [9] evaluated the relationship between potential gray and white matter brain structures and intolerance of uncertainty, which is an important clinical aspect of patients with OCPD, in the healthy subjects who were sixty individuals. The authors investigated them by utilization of voxel-based morphometric analysis and reported a clear positive correlation between intolerance of uncertainty and striatal volume, particularly the putamen, considering that the volumetric properties of the striatum might reflect the processing of uncertainty per se. We previously volumetrically examined some key brain regions such as OFC, thalamus, caudate nucleus and anterior cingulate cortex (ACC) concurrently in first-episode patients with obsessive-compulsive disorder (OCD) and determined that patients had increased white matter volums, greater left and right thalamus volumes and significantly reduced left and right OFC volumes compared with healthy controls, with a near-significant difference between the patients and healthy controls on left side for the ACC and with significant correlations were found between Y-BOCS scores and left OFC, and right OFC and between Y-BOCS and left thalamus volumes in the patient group [10]. In a another study, we reported also that among these key brain regions OFC and thalamus could account for the resistancy to the treatment of OCD [11]. In an unpublished study, study team measured orbito-frontal cortex (OFC) and thalamus volumes of patients with OCPD and healthy control subjects, which are accepted as two important ones of key brain regions of OCD [12]. We found that patients with OCPD had considerably smaller left and right OFC volumes compared to those of healthy control subjects. We also observed that thalamus volumes of patients were statistically significantly greater than those of healthy comparisons for both sides of region of interest, considering that volumetric alterations determined in the present study may be involved in the pathophysiology of the OCPD, considering that OCPD might be related to OCD spectrum disorders neuroanatomically. Lawrence et al. [13] and Phillips et al. [14] studied hippocampus, anterior cingulate and basolateral amygdala regions because of the fact that these structures have connections with the orbito-frontal cortex which may have in the pathophysiology of OCD. Moreover, Gray [15] and Pitman [16] both reported that the hippocampus could have a role in the occurrence of compulsive behaviors. In this context, an study team investigated hippocampus and amygdala volumes in refractory OCD patients and founda that the mean left and right hippocampal and amygdala volumes of the patients were smaller than those of the healthy controls [17].

Associaitons afrementioned led us to examine hippocampal and amygdalar volumes in patients with obsessive-compulsive personality disorder to contribute the neuroanatomy of the disorder (see Table 1).

Table 1
Clinical and demographic characteristics of the groups.

Item	OCPD group (n = 16)	Control group (n = 18)	*p*
Age (years)	32.5 ± 8.9	29.5 ± 5.1	>0.05
Gender (Female/Male)	11/5	10/8	>0.05
Education High school			>0.05
High school	9	15	
Elemantary school	3	3	
First school	4		
Handedness (right)	16	18	>0.05
Length of illness (years)	6.4 ± 4.1	–	
Hamilton Depression Rating Score	9.8 ± 2.2	3.3 ± 1.7	<0.01

2. Methods

The patient group was composed of twenty patients with OCPD. We took an approval from the Local Ethics Committee placed at the Firat University School of Medicine (Elazig, Turkey). We performed our syudy in accordance with ethical standards of the Declaration of Helsinki which had been revised in 1983. We selected our patient population from the out and in-patient clinics of Firat University School of Medicine Department of Psychiatry. Healthy control subjects were composed of the individuals who studied in our hospital and accepted to participate into the study. After complete description of the study, all volunteers gave written informed consent to participate into the study. All diagnoses were done by the Diagnostic and Statistical Manual of Mental Disorders Fourth Edition (DSM-IV) and the Structured Clinical Interview for the Diagnostic Schedule for Mental Disorders-Fourth Edition (SCID) [18]. Comorbid depressive symptoms was assessed by using the Beck Depression Rating Scale [19]. On the other hand, a group of healthy controls were matched on age, sex, education and handedness. To exclude patients from the study, following exclusion criteria were used which were also used in our previous studies: The presence of any current or history of comorbid Axis I psychiatric disorder apart from depression, the presence of current severe medical problems, or alcohol/substance abuse within the 6 months preceding the study, any contrendication for suffering from MRI investigation such as cardiac stent, and use of psychoactive medication within four weeks of the study. As for healthy control subjects, several excluion criteria were also used for them. Firts of al, it should not be any presence of any current or history of psychiatric disorder in self and in their first-degree relatives, the presence of current severe medical illnesses, or alcohol/substance abuse within the 6 months preceding the study, and any contrendication for suffering from MRI investigation such as cardiac stent. At this point, we should mention that patients and healthy control ones were the subjects of an another study in which we examined OFC and thalamus volumes in patients with OCPD [12] (see Table 2).

2.1. MRI procedure

MR images of patients with OCPD and healthy control subjects were obtained at a Signa 1.5 Tesla MRI scanner (General Electric Medical Systems, Milwaukee, WI, USA) by using a high-resolution T1-weighted spoiled gradient recalled acquisition sequence. We used earphones to decrease noise during the MRI acquisition. Patients with OCPD and healthy controls were informed about the duration and process of MRI. It was told that at the beginning of the process there might be a bit scare but it would decrease after a period of time. Following parameters were used for acquisition of MRI: repetition time [TR] = 2000 msfield of view [FOV] = 240 mm, echo time [TE] = 15.6 ms, flip angle = 20°,

Table 2
The Volumes of The Structures Evaluated in Patients with OCPD *versus* Controls Subjects (Mean ± Standard Error).

	OCPD Group (*n* = 16)	Healthy Control Group (*n* = 18)	*p*
Hippocampsu Volumes			
Left	3.58 ± 0.46	4.24 ± 0.35	=0.000
Right	3.78 ± 0.44	4.19 ± 0.30	=0.003
Amygdalaa Volumes			
Left	1.66 ± 0.28	1.92 ± 0.10	=0.001
Right	1.67 ± 0.30	1.94 ± 0.13	=0.002

Volumes presented are in cm^3.

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bandwidth = 20.8, slice thickness = 2.4 mm, echo spacing = 15.6 ms, 8 echoes, resolution = 0.9375 × 0.9375 × 2.4 mm.

We utilized a computer workstation program called as GE Volume Viewer voxtool 4.6 64q program (Chicago, IL, USA). By using this program, it was semi-automatically measured the regions of interest, that were hippocampus and amygdala. Two of our study team (H.Y. and M.C.K.) used the manual tracing method to determine the boundaries of hippocampus and amygdala. They did not know the diagnosis of patients and the group of subjects to provide the objectivity of the measurements. The boundaries of hippocampus and amygdala were delineated on the coronal MR images according to standard brain atlases [20–22] and were adapted from Caetano et al. [23] and Brambilla et al. [24]. When tracing the hippocampus, the process was started on the coronal slice where the superior colliculus completely connected with the thalamus and finished one slice before the mammilary bodies were seen. On the other hand, the corona radiata and ambient cistern were accepted as the superior border. The inferior border was indicated as the white matter. At last the inferior horn of the lateral ventricle was accepted as the lateral border of hippocampus. As for the amygdala, tracing process was started when the mamillary body can be observed. Temporal lobe white matter indicated the superior and lateral limits of hippocampus while the white matter of parahippocampal gyrus was selected as inferior border. According to references aforementioned, anterior border of the amygdala was accepted when the amygdala could not be seen. White matter of the temporal lobe was accepted as the superior and lateral boundaries of the amygdala. On the other hand, at lateral, it was taken into consideration that the gray matter of the amygdala blended together with the ventral putamen, this point can make some confusing about the tracings of amygdala. The white matter of parahippocampal gyrus was accepted as inferior border, under the light of references cited above. The limit that the amygdala could not be observed was accepted as anterior border of the amygdala region. The posterior boundary of the amygdala was taken as the coronal plane, at the point where gray matter was firstly seen superior to the alveus. The interrater (HY, MCK) and intrarater reliabilities for hippocampus and amygdala measurements were good (0.90 and 0.95 for hippocampus and 0.85 and 0.90 for amygdala, respectively). Sample images were added to the last paper (Fig. 1).

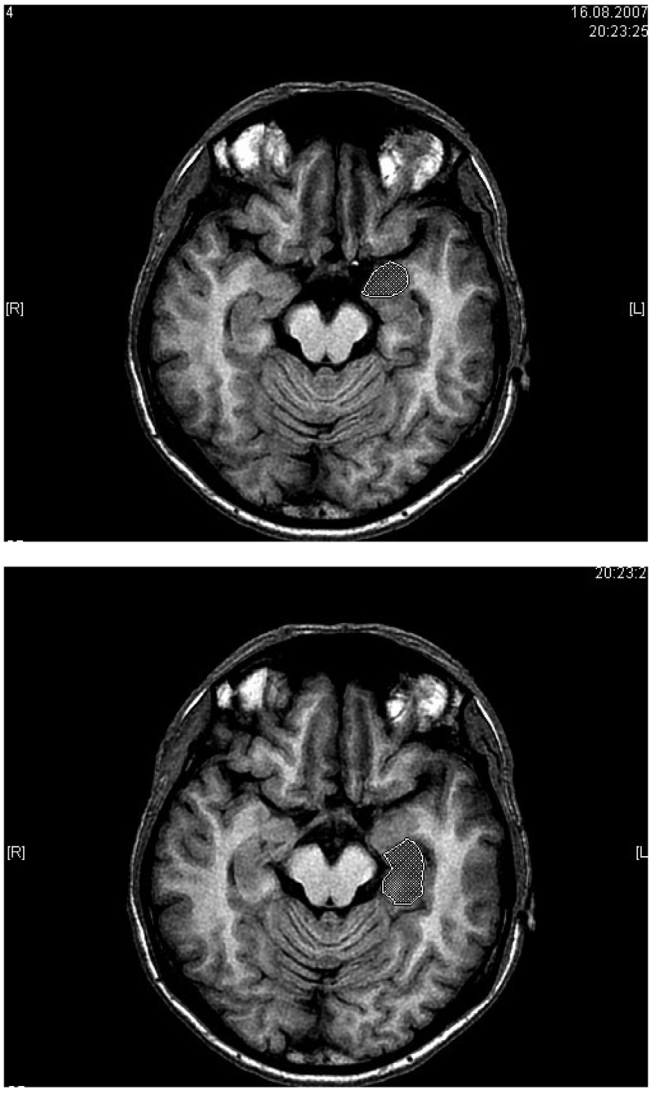

Fig. 1. Sample imagings of hippocampus and amygdala.

2.2. Statistical analysis

To perform on all statistical analyses, the Statistical Package for Social Sciences version 16.0 was utilized (SPSS, Chicago, IL, USA). It was used independent sample *t* test specifically to compare hippocampus and amygdala volumes and other continous variables between patients with OCPD and healthy control comparisons. For categorical variables, we used the chi-square test. In addition, analysis of covariance (ANCOVA), was done by using the SPSS program version 16.0, to control age, gender and whole brain volumes as covariates. In addition, we used correlation analyses by using Pearson's correlation test to examine correlational relationships. For all analyses, statistical significance was accepted as $p < 0.05$.

3. Results

We did not find any considerable differences in respect to demographic variables such as age, gender composition, educational level, handedness and intracranial volume (ICV), whole brain volume, gray and white matter volumes between patients with OCPD and healthy controls ($p > 0.05$).

We found that patients with obsessive compulsive personality disorder had smaller hippocampus volumes in both sides compared to those of healthy control subjects (for the left side of hippocampus; t = 4.68, p < 0.001; for the right side of hippocampus; t = 3.18, $p = 0.003$). As for the ANCOVA, age, gender and whole brain volume as covariates, we observed that the significant differences continued (for the left side of hippocampus, $F = 6.78$, $p < 0.01$; for the right side of hippocampus, $F = 6.98$, $p < 0.01$).

We found that patients with obsessive compulsive personality disorder had smaller amygdala volumes in both sides compared to those of healthy control subjects (for the left side of amygdala; t = 3.78, $p = 0.001$; for the right side of amygdala; t = 3.44, $p = 0.002$). As for the ANCOVA, age, gender and whole brain volume as covariates, we observed that the significant differences continued (for the left side of amygdala, $F = 7.03$, $p < 0.01$; for the right side of amygdala, $F = 6.91$, $p < 0.01$).

We did not find any correlation between hippocampus and amygdala volumes and any clinical and demographic variables, when used Pearson's correlation test.

4. Discussion

The present study is the first one evaluating hippocampus and amygdala volumes in patients with obsessive-compulsive

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personality disorder. The study found followings: (i) patients with obsessive compulsive personality disorder had smaller hippocampus volumes in both sides compared to those of healthy control subjects (for the right side of hippocampus, for patient group: mean = 2479.34 mm^3, SD = 188.77; healthy control subjects: mean = 2781.51 mm^3, SD = 292.34; t = 2.29, p < 0.05; for the left side of hippocampus, for patient group: mean = 2511.29 mm^3, SD = 203.09; comparison subjects: mean = 2865.63 mm^3, SD = 346.70; t = 3.14, $p < 0.05$), as supported by the ANCOVA analysis under, age, gender and whole brain volume as covariates (for the left side of hippocampus, $F = 4.29$, $p < 0.05$; for the right side of hippocampus, $F = 4.20$, $p < 0.05$). Consequently, we determined that patients with obsessive compulsive personality disorder had smaller hippocampus and amygdala volumes in both sides compared to healthy control subjects, as just we had found in OCD patients [17].

As much there have been contradictory results, it has been proposed that OCD patients might have volumetric abnormalities of the hippocampus and amygdala. It was reported that hippocampal volumes were bilaterally smaller in OCD patients compared to those of healthy controls, whereas left amygdala volumes were significantly enlarged in patients with OCD, contrary to our findings [25]. On the other hand, Szezsko et al. [26] and Hong et al. [27] detected that patients with OCD had considerably smaller both sides of amygdala and hippocampal volumes. Previously, we performed a study on refractory OCD patients. In that study, we detected the hippocampus and amygdala volumes in a sample of fourteen refractory OCD patients and fourteen healthy control subjects and determined that the mean left and right hippocampal and amygdala volumes of the patients were reduced compared to those of the healthy controls, with a correlation between OCD severity and left hippocampus but amygdala volumes, and with a correlation between the duration of illness and both hippocampus and left amygdala volumes. We commented that the hippocampus and amygdalar volume abnormalities might be related to refractoriness to OCD. When we take into consideration that hippocampal projections lie down the orbitofrontal cortex (OFC) with topographical specificity in both the hippocampus and the OFC, the role of hippocampus may be understood well in the neuroanatomy of OCD [28,29]. In addition, it has been reported that serotonin reuptake inhibitors, an important choice fort he treatment of OCD, have an important amount of receptors to bind on the amygdala [30,31]. In fact, as can be seen in these studies aforementioned, it can be speculated that hippocampus and amygdala regions seem to be important in the neuroanatomy of the OCD. The fact that we have detected similar results in patients with obsessive-compulsive personality disorder on hippocampus and amygdala volumes like in OCD patients led us to speculate that hippocampus and amygdala volumes might be important in the neuroanatomy of obsessive-compulsive personality disorder.

As in our team's previous neuroimaging studies, the present study also have some similar limitation factors. Firts of all, the study has small number of patients and control subjects. When considering that the study was supported by the University Project Office, limited occasion given should be take into consideration. Second, we did not evaluate apart from the hippocampus and amygdala regions. Third, we have also limitations of manual tracing itself. Fourth, for personality disorders including obsessive-compulsive personality disorder, the validity of diagnosis should be questioned.

Consequently, our present results suggest that hippocampal and amygdalar structural abnormalities may be related to the neuroanatomy of obsessive-compulsive personality disorder. However, it is required novel studies with larger sample.

Conflict of interest

No conflict of interest.

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