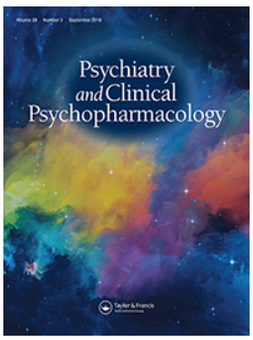

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CASE REPORT

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Remission of obsessive-compulsive symptoms following temporoparietal haemorrhage: a case report

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ABSTRACT

Many etiological factors have been implicated in the pathophysiology of obsessive-compulsive disorder (OCD). The non-invasive neuroimaging studies regarding the pathophysiology of OCD indicate the abnormalities in distinct brain regions. Cortico-striato-thalamo-cortical (CSTC) tracts are proposed to be involved in OCD symptomatology. Reports of OCD cases after a brain injury including cerebrovascular accident (CVA) also support the involvement of CSTC pathways in OCD. Moreover, a few cases of OCD whose OC symptoms displayed an improvement after CVA in the brain regions related CSTC tracts. These reports will guide the surgical interventions in OCD. Here, we present a case of OCD whose symptoms resolved within a few days after a haemorrhage in the left temporo-parieto-occipital lobes. In this case, we supposed that the compression of the subcortical striatal tissues which is known to be involved in the OCD pathophysiology might be associated with the resolution of OCD symptoms.

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Obsessive-compulsive disorder; remission; cortico-striato-thalamic circuits; cerebrovascular accident

Introduction

Obsessive-compulsive disorder (OCD) is typically a chronic disorder and affects 2–3% of the population. It is characterized by persistent, intrusive thoughts and impulses (obsessions), and repetitive intentional behaviours (compulsions). These symptoms persist despite individuals' attempts to resist them and are accompanied by marked and often overwhelming anxiety [1–3]. It causes significant impairments in occupational and social life areas [4]. Several neural systems have been implicated in the pathophysiology of OCD. The results of neuroimaging studies in OCD patients have implicated most consistently the orbito-frontal cortex, the cingulate cortex, and the basal ganglia (mainly the caudate nucleus), and more recently also regions within the parietal lobe, in the pathophysiology of obsessions and compulsions [5,6]. The dysregulation of the serotonergic (5-HT) system has been suggested primarily on the basis of the effectiveness of serotonin reuptake inhibitors (SRIs) and selective serotonin reuptake inhibitors (SSRIs) in alleviating obsessions and compulsions in patients [7,8]. Abnormalities of the dopaminergic system have also been implicated in the pathophysiology of OCD, based on surplus therapeutic benefits obtained with co-administration of SSRIs and dopamine blockers [9], as well as on clinical observations of obsessions and compulsions in basal ganglia-related disorders, such as Tourette's syndrome [10,11]. More recently, an increasing body of evidence points also to the involvement of the glutamatergic system in OCD [12], elevated glutamate levels in the cerebrospinal fluid of drug-naive patients [13], correlations between symptom severity and the level of several glutamatergic metabolites [14]. The frontal orbito-striatal areas (including the caudate nucleus) and the dorsolateral prefrontal cortex have been implicated in the inhibition of responses and in planning, organization, and verification of previous actions [15]. Previously, some cases were reported to develop OCD after ischaemic or haemorrhagic stroke. A 37-year-old man without a history of OCD reported contamination obsessions and compulsive washing after an infarction in the left temporal and parietal cortices (at the territory of middle cerebral artery) [16]. In a 40 year-old woman who had a childhood history of frontoparietal haematoma occurred disruptive obsessions and shopping compulsions following a right temporal lobe haemorrhage [17]. A 73-year-old woman had ego-syntonic contamination obsessions and cleaning compulsions after a left-sided basal ganglia haemorrhage [18]. Previously, a few OCD cases were reported after lesions in putamen, striatum and parietal lobe [19]. In two children, obsessive-compulsive symptoms (OCS), Tourette's syndrome (TS), dystonia and attention-deficit/hyperactivity disorder (ADHD) developed after right subcortical strokes in the regions of right caudate and putamen [20]. There are some reports which

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demonstrated that OCD disappeared within first weeks of a brain injury. To the best of our knowledge, previously only two such cases were reported. In the first case, the well-known symptoms of OCD remitted within a few weeks following a CVA in the right posterior frontal and anterior parietal areas. That patient had a lifetime diagnosis of ADHD and a previous history of which resolved in her early twenties. Her aggressive obsessions and checking compulsions remitted within a few weeks after stroke, but no change was observed in her ADHD symptoms. However, her OCS relapsed 24 months later [21]. In a 33-year-old man with a previous history of OCD, contamination and aggressive obsessions disappeared totally following a haemorrhagic infarction in the left basal ganglia. A few months later, his OCS symptoms gradually relapsed [22]. Here, we present a case of OCD whose symptoms resolved within a few days after a haemorrhage in the left temporo-parieto-occipital lobes. In this case, we supposed that the compression of the subcortical striatal tissues, which is known to be involved in the OCD pathophysiology, might be associated with the resolution of OCD symptoms.

Case presentation

The patient was a 51-year-old, right-handed, married woman with 17 years of diagnosis of OCD. Her first symptoms began after the delivery of her second child upon seeing blood on her hand. She started to imagine that her hands were not cleaned sufficiently and developed contamination obsessions and cleaning compulsions. These compulsions were repeated for three times and every washing period was taking at least 40–45 minutes. She was administered several antidepressant and antipsychotics and cognitive behavioural therapy (CBT). She reported that her symptoms partially improved with these treatments. Her OCD displayed a chronic course. On May 2016, she was admitted to emergency clinic with headache, dizziness, forgetfulness, slurred speech, and inappropriate answers. Her neurological examination revealed disorientation, confusion, and dysarthric speech. Eye movements were normal with isochoric pupils. Muscle tone was 5/5 in every extremity and there was no sensory loss. The MRI scan showed intraparenchymal haemorrhage areas in the left parietal, temporal, and occipital lobes. In the left temporoparietal area, there was hemosiderin collection of 46 × 32 mm dimensions which was hypointense in density together with vasogenic oedema causing a compression on subcortical striatal tissues on the left and therefore a shift to the right side (Figure 1). During her treatment at Neurology Department, her relatives noticed that her OCD symptoms suddenly disappeared 3–4 days after her haemorrhagic stroke. We retrospectively and currently assessed the patient through DSM-IV, SCID-I, and made a diagnosis of OCD. Her prestroke Y-BOCS score was 32 and decreased to 6 on the fourth day of CVA. Her OCD symptomatology maintained stable nearly for 2 months (Figure 2).

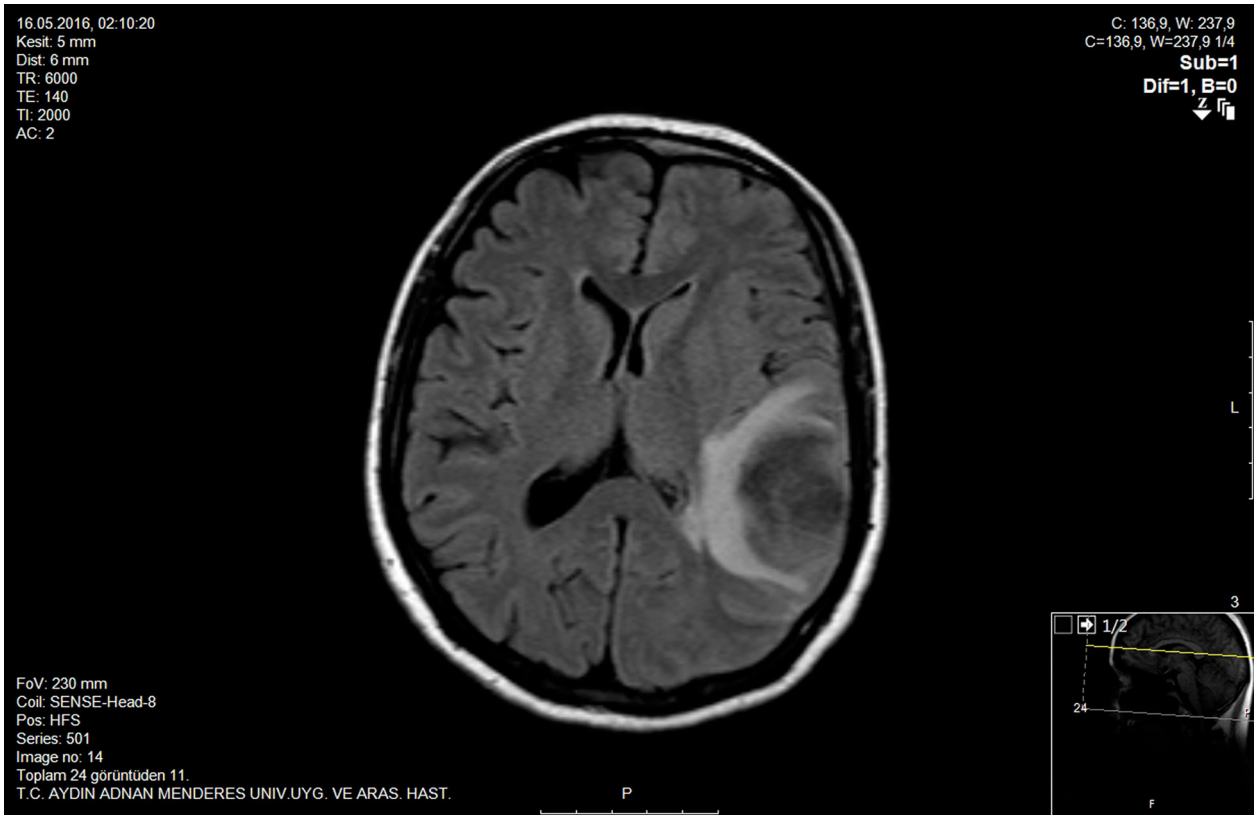

Figure 1. Intraparanchymal hematoma and vasogenic edema in the left parieto-occipital lobe.

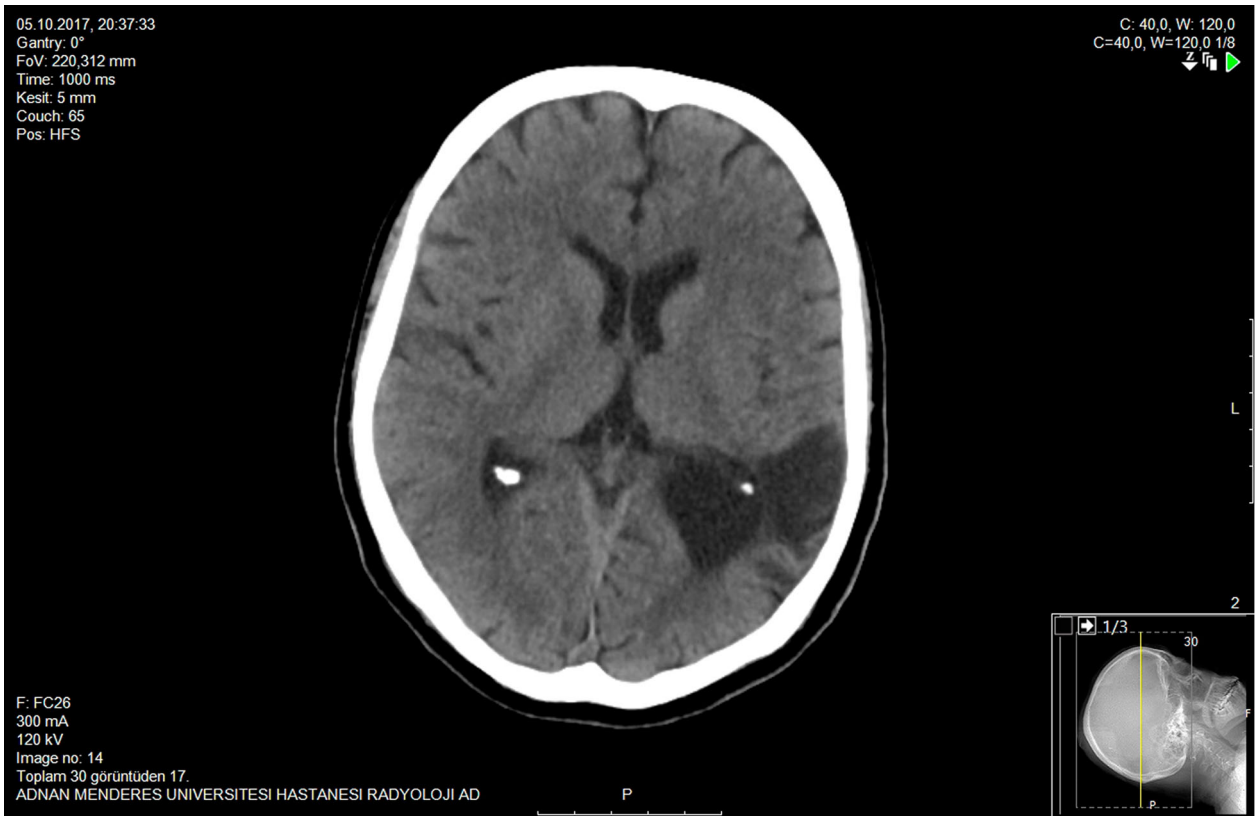

Figure 2. Area of encephalomalacia and resolved hematoma in the left parieto-occipital lobe. Temporal and occipital horns of the lateral ventricul became prominent.

Discussion

Over past 30 years, non-invasive research demonstrated that cortico-striato-thalamo-cortical (CSTC) circuits are implicated in the pathophysiology of OCD. PET, SPECT and fMRI scans showed increased neuronal activities in the caudate nucleus, orbitofrontal cortex, and cingulate gyrus of OCD patients compared to normal controls [23]. In addition, numerous parallel and partially closed loops connected to CSTC circuits affecting motor and cognitive functions were also described [24]. Some of the previous studies found that resting-state functional connectivity (rsFC) between nucleus accumbens (NA) and lateral orbitofrontal cortex was increased in OCD patients in resting state. In contrast, rsFC of NA to the amygdala was decreased during incentive processing pointing deficits [25]. This abnormal interaction between these regions modulates the affective and motivational symptoms of OCD. Besides these imaging studies, EEG findings showed most significant activation in the thalamus, the corpus striatum (including the caudate nucleus), the orbitofrontal and temporoparietal regions with dominance in the right hemisphere in OCD patients. These findings denoted that OFC and ACC are the mainstays of OCD with their connections to striatum thalamus, caudate nucleus, and DLPFC [26]. This finding supports the impaired decision making and reward anticipation in OCD patients. Functional neuroimaging studies have consistently documented hyperactivity at rest in CSTC circuits when comparing OCD subjects with controls. Further, this regional hyperactivity is accentuated during provocation of the OCD symptomatic state versus control states [27]. Human neuroimaging data suggest that abnormalities in OFC/ACC–basal ganglia–thalamic circuitry are central to the pathophysiology of OCD and responses to treatment. In particular, the magnitude of OFC activity is proportional to symptom severity and pretreatment activity within this same region predicts subsequent medication response. Several studies have consistently found reduction in activity in these same regions after successful treatment of OCD, regardless of the mode of treatment, including pharmacological [28], behavioural [29,30], and neurosurgical [31] therapies. In addition, elevated glutamatergic transmission from OFC/ACC to striatum has been inferred from MRS measurements of an elevated glutamate index within the striatum that is correlated with OCD symptom severity and returns toward normal with successful treatment [32]. In our patient, there were intraparenchymal haemorrhage areas in the left parietal, temporal, and occipital lobes. The vasogenic oedema in the left temporoparietal area caused a compression on subcortical striatal tissues on the left and therefore a shift to the right side was observed. We supposed that the essential cause of remission in OC symptoms in our patient was the compression of the striatal tissue known to be involved in OCD

pathophysiology. As a result of this compression, a reduction in the activity of right strial regions might have caused a temporary amelioration in OCD symptoms. As the effects of compression ameliorated by the resolution of haemorrhage and oedema, the symptoms emerged again.

Disclosure statement

No potential conflict of interest was reported by the authors.

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