Case Report

Obsessive-Compulsive Disorder Following Cerebrovascular Accident: A Case Report and Literature Review

Rie Akaho, MD, PhD,* Ichiro Deguchi, MD, PhD,† Hiroshi Kigawa, MD,‡ and Katsuji Nishimura, MD, PhD*

Background: Cases of obsessive-compulsive disorder (OCD) following cerebrovascular accident (CVA) have rarely been reported. *Methods*: Case report and literature review. *Results*: We describe the case of a 58-year-old, right-handed man developed OCD 17 months after stroke resulting from lesion of the right middle cerebral artery infarction. The patient was successfully treated with sertraline up to 50 mg per day. His OCD behaviors largely reduced in 6 weeks, and the Yale-Brown Obsessive Compulsive Scale score was reduced from 29 to 12 in 1 year. A literature review revealed 21 previous cases of OCD following CVA. Among these, consistent with our case, the basal ganglia was the most common site of the lesion responsible for the development of this rare disorder. We discuss the patient's treatment and outcomes. *Conclusions*: Our present case and a literature review suggest that OCD can manifest following CVA, although further studies are necessary. Selective serotonin reuptake inhibitors appear to be effective in treating this rare disorder.

Key Words: Obsessive-compulsive disorder—cerebrovascular accident—stroke—basal ganglia—sertraline

Introduction

Obsessive-compulsive disorder (OCD) can manifest following several brain injuries with structural brain lesions, including traumatic brain injury, brain infections, and brain tumors.[1] In contrast to depression, which frequently occurs following cerebrovascular accident (CVA), cases of OCD following CVA have rarely been reported, and its clinical characteristics, clinical course, treatment, and outcomes remain unknown. Furthermore, both structural and functional neuroimaging findings in such cases may enable us to understand the pathogenesis underlying idiopathic OCD.

Here, we present the case of a man who developed OCD after stroke resulting from lesion of the right middle cerebral artery (MCA) and who was successfully treated with sertraline. We subsequently review the extant literature for cases of OCD following CVA.

Case Report

The patient was a 58-year-old, right-handed, married Japanese man working in a factory, with no previous history of psychiatric disorders. In May 2015, the patient suffered acute atherothrombotic brain infarction of the right MCA at the age of 57. Thrombolytic therapy with tissue plasminogen activator was administered, and a stent was placed at the M1 distal lesion. After rapid and effective treatment, the patient's hemiplegia was eliminated. One month after the stroke, neurological assessment suggested that only mild hemispatial neglect and mild facial palsy

From the *Department of Psychiatry, School of Medicine, Tokyo Women's Medical University, Shinjuku-ku, Tokyo, Japan; †Department of Neurology, Saitama Medical University International Medical Center, Saitama, Japan; and ‡Rehabilitation Center, Hanno-Seiwa Hospital, Saitama, Japan.

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Address correspondence to Katsuji Nishimura, MD, PhD, Department of Psychiatry, School of Medicine, Tokyo Women's Medical University, 8-1 Kawada-cho, Shinjuku-ku, Tokyo 162-8666, Japan. E-mail: nishimura.katsuji@twmu.ac.jp.

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on the left side was present. Although the patient had a Mini-Mental State Examination score of 29 of 30, attention deficit was detected in neuropsychological examinations. He returned to work in August 2015.

In October 2016, at the age of 58, the patient gradually developed obsessive-compulsive symptoms. He frequently checked things more than 10 times, including locks on doors, light switches, water faucets, and taking medicine. The patient often checked these things with finger pointing. He measured his blood pressure frequently, wrote down the value, and read it aloud repeatedly. He frequently washed his hands while he counted to 10. The duration of the patient's baths increased to over an hour. However, the patient continued to work regularly without lateness or absence. Moreover, he realized that the behaviors did not make sense. His family physician prescribed imipramine 10 mg per day with partial remission of symptoms.

However, in June 2017 the patient was referred to our psychiatric clinic because of exacerbation of his obsessive-compulsive symptoms. He was diagnosed with OCD based on the Diagnostic and Statistical Manual of Mental Disorders, fifth edition criteria. No other psychiatric comorbidities were observed. His wife confirmed that he had no premorbid obsessive-compulsive traits. The patient had a Yale-Brown Obsessive Compulsive Scale (a test to measure the severity of OCD symptoms, in which higher scores indicate more severe OCD) score of 29 (obsessions, 14; compulsions, 15). Moreover, the patient's Mini-Mental State Examination score was 29 of 30 once again. Routine biochemical and hematological profiles were normal. Brain magnetic resonance imaging revealed an old cerebral infarct with lesions in the right insular cortex, basal ganglia, and temporo-occipital lobe. Magnetic resonance angiography showed good patency of stenting vessels in right MCA (Fig 1, A). Tc-99mECD single photon emission computed tomography showed a large area with a high degree of hypoperfusion in the right MCA territory, particularly in the right insular cortex, basal ganglia, and right dorsal temporal lobe (Fig 1,B).

The symptoms were treated with sertraline (up to 50 mg/day), while imipramine was stopped. Six weeks after starting treatment with sertraline, the patient's wife recognized that his OCD behaviors largely reduced. No adverse effects of sertraline were observed. In June 2018, a year after the patient's admission to our clinic, he had a Yale-Brown Obsessive Compulsive Scale score of 12 (obsessions, 4; compulsions, 8).

Literature Review

To review published literature for cases of OCD following CVA, we conducted a PubMed search on October 23, 2018, using the search terms "obsessive-compulsive" and ("stroke" or "infarct" or "infarction" or "hemorrhage" or "cerebro-vascular"). Our search initially yielded 138 results. Upon detailed review of titles, abstracts, and full texts, we identified 17 articles (17 cases) describing OCD following CVA (13 ischemic[2-14] and 4 hemorrhagic).[15-18] In addition, among these articles, we found 2 articles describing 1 case[19] and 3 cases,[20] respectively, that linked OCD to CVA. All 21 cases are presented in Table 1.

Discussion

Our review of the literature suggested that the most common site of lesion following CVA in the 21 cases was the basal ganglia (12 cases, 57%).[2,4,7,11-13,15,18-20] The next most common was the temporal lobe (4 cases, 19%),[2,6,11,17] posterior frontal lobe (4 cases, 19%),[3,6,9,20] parietal lobe (4 cases, 19%),[2,6,9,16] thalamus (2 cases, 10%),[12,14] prefrontal cortex (1 case, 5%),[8] corona radiata (1 case, 5%),[19] and pons (1 case, 5%).[10]

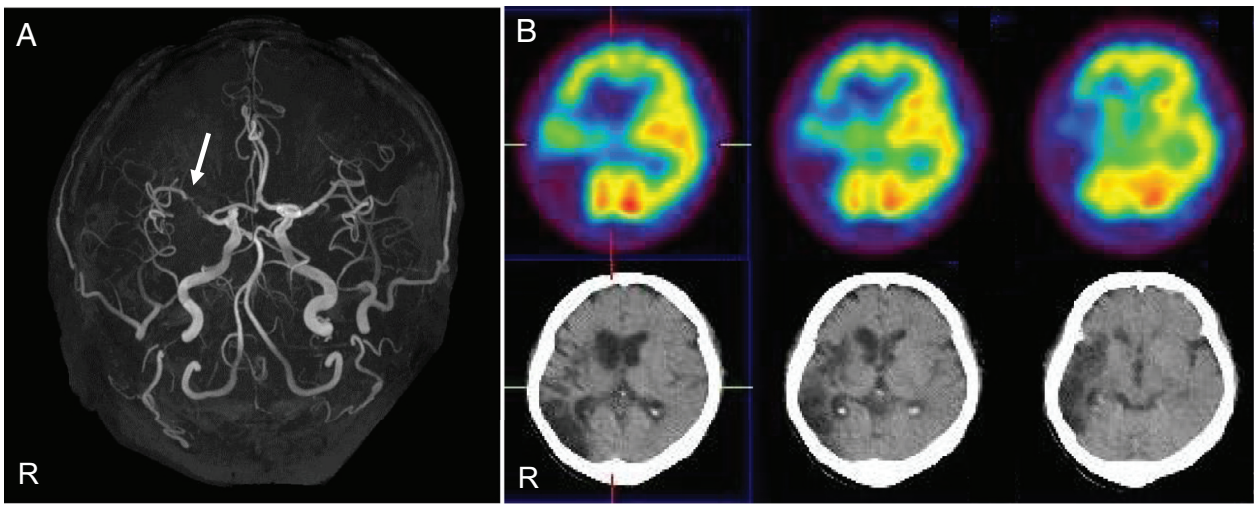

Figure 1. *(A) Brain magnetic resonance angiography showed good patency of stenting vessels in right middle cerebral artery (MCA) (arrow). (B) Tc-99mECD single photon emission computed tomography showed a large area with a high degree of hypoperfusion in the right MCA territory, particularly in the right insular cortex, basal ganglia, and right dorsal temporal lobe.*

Table 1. *Cases of obsessive-compulsive disorder following cerebrovascular accident obtained from the literature review*

Author(s)	Year	Age at onset	Gender	Lesion location	Duration*	Treatment	Y-BOCS scores, before/after treatment	Outcome
Ischemic stroke								
Swoboda and Jenike	1995	62	M	Right posterior frontal lobe	Unknown	Clonazepam, small dose	-/-	Minimally improved
Simpson and Baldwin	1995	71	M	Right inferior parietal, right basal ganglia, and right medial temporal lobe	I month	Clomipramine 100 mg/day	-/-	Very much improved
Lopez-Rodriguez et al.	1997	73	F	Left basal ganglia	6 months	Lithium 600 mg/day	-/-	Very much improved†
Rodrigo Escalona et al.	1997	34	M	Bilateral globus pallidus	Within 6 months	Fluoxetine 20 mg/day	-/-	Not assessed
Philpot and Banerjee	1998	83	F	Left basal ganglia and corona radiata	Unknown	Paroxetine, non-adherent	-/-	No change
Weiss and Jenike (case 1)	2000	53	F	Head of the left caudate	Unknown	Clonazepam 0.25 mg/day	24/-	Not assessed
Weiss and Jenike (case 2)	2000	52	M	Bilateral caudate nuclei	Unknown	Fluvoxamine up to 150 mg/day	-/-	Minimally improved
Weiss and Jenike (case 3)	2000	62	M	Rigt posterior frontal lobe	Unknown	Clonazepam, dose unknown	-/-	Minimally improved
Mahendran	2000	37	M	Left frontal, temporal, and parietal lobes	2 years	Clomipramine, up to 150 mg/day	15/-	Very much improved
Carmin et al.	2002	78	M	Basal ganglia	Unknown	Exposure and ritual prevention (ERP)	24/2	Very much improved
Kim et al.	2002	66	M	Left medial orbitofrontal cortex	Unknown	Sertraline 50 mg/day	15/-	Minimally improved
Sun et al.	2006	65	F	Bilateral deep parietal area and right frontal lobe	Within 2 years	Clozapine 125 mg/day	-/-	Very much improved‡
Matsui et al.	2007	71	M	Right pons	1 month	Paroxetine up to 30 mg/day	14/-	Very much improved
Muneoka et al.	2011	68	F	Left basal ganglia and right temporal lobe	4 months	Paroxetine up to 40 mg/day	31/8	Much improved
Khairkar and Diwan	2012	60	F	Left thalamo-striatal	Unknown	Paroxetine 25 mg/day	-/-	Very much improved
Van Roie et al.	2013	15	M	Right caudate nucleus	Unknown	Anticoagulant treatment	-/-	Much improved
Cooper and Grant	2017	24	M	Left thalamus	Within 1 month	Dronabinol up to 20 mg/day	39/10	Very much improved§
Hemorrhagic stroke								
Thobois et al.	2004	24	M	Left caudate nucleus	3 months after surgery	Refused treatment	12/-	Not assessed
Ros and Podgorski	2007	39	M	Right parietal lobe	Unknown	Sertraline up to 200 mg/day	-/-	Very much improved

(*Continued*)

Table 1 (*Continued*)

Author(s)	Year	Age at onset	Gender	Lesion location	Duration*	Treatment	Y-BOCS scores, before/after treatment	Outcome
Rai et al.	2011	40	F	Right temporal lobe	Unknown	Citalopram up to 100 mg/day	-/-	Very much improved
Katz and Flemming	2015	31	F	Left caudate nucleus	Unknown	Sertraline, dose unknown	-/-	Very much improved

OCD = obsessive-compulsive disorder; Y-BOCS = Yale-Brown Obsessive Compulsive Scale.
*Duration between stroke and manifestation of OCD.
†No response to fluoxetine 40 mg/day, risperidone 1 mg/day, and electroconvulsive therapy.
‡Accompanied by blepharospasm and psychotic symptoms.
§No response to high dose medication with 3 antidepressants (fluvoxamine, clomipramine, and mirtazapine), 6 antipsychotics, clonazepam, ketamin, and traditional mood stabilizing agents.

Recent imaging, surgical, and lesion studies of idiopathic OCD suggest that the prefrontal cortex, basal ganglia, anterior cingulate cortex, and thalamus may be associated with the pathogenesis of idiopathic OCD.[21] Consistent with findings in idiopathic OCD, the most common lesion site in OCD following CVA was in the basal ganglia (including in our case), while the other areas (prefrontal cortex, anterior cingulate cortex, and thalamus) were lesioned in only a few cases. This suggests that the basal ganglia might be the primary site of pathogenetic lesion in OCD.

Although the time course of OCD following CVA has not been well characterized, particularly in cases of lacuna infarction, the duration between incident stroke and manifestation of OCD varies from within 1 month to 2 years. Our patient developed OCD symptoms 17 months after stroke. Similarly, poststroke depression can manifest at any time up to 5 years after stroke.[22]

Consistent with our case, selective serotonin reuptake inhibitors (SSRI) and tricyclic antidepressants (TCA) appear to be effective in treating poststroke OCD. In several studies, paroxetine[10-12] was reported to be moderately to very effective in 3 of 4 cases, sertraline[16,18] was effective in 2 of 3 cases, and citalopram[17] was effective in one of one cases. However, fluvoxamine and fluoxetine were not effective in any cases (both administered in 2 cases). Clomipramine[2,6] was reported to be very effective in 2 cases, but was ineffective in 1 case. In contrast, clonazepam was only minimally effective in 2 of 3 cases, and effectiveness was not assessed in a further case. Efficacy of lithium,[4] clozapine,[9] and dronabinol[14] were also reported each in 1 case. Finally, exposure and ritual prevention[7] was also reported to be effective in 1 case. Overall, OCD symptoms in 13 (62%) of the total of 21 cases were much or very much improved. The efficacy of such antidepressants, including SSRIs and TCAs, has been also reported in the treatment of poststroke depression.[22]

Finally, in a recent case-control study in Sweden, the point prevalence of OCD after stroke was 9% versus 2% in the general population,[23] suggesting that the condition remains underdiagnosed.

In conclusion, we report a case of OCD following stroke in the right MCA, including the basal ganglia, which was successfully treated with sertraline. Our literature review of this rare disorder revealed that the basal ganglia is the most common site of the lesion responsible for the development of OCD following CVA. SSRIs and TCAs may be effective in treating patients with both idiopathic and poststroke OCD. Further case studies of OCD following CVA are needed to provide further details of the clinical features, structural/functional imaging findings, and treatment strategies.

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