Case Report

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Late-Onset Obsessive-Compulsive Disorder Secondary to Caudate Lacunar Infarct: A Case Report

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ABSTRACT

Due to low prevalence of late onset Obsessive-Compulsive Disorder (OCD), it is more likely to have an organic etiology. In this article we present a 59-year-old man, referred for recently developed obsessive compulsive symptoms, with no prior history of OCD in the past. The symptoms were included ego-dystonic recurrent obsessional doubts, leaded him to compulsive checking several times a day and caused a remarkable distress and functional impairment. His neurological exam revealed no deficit. The most prominent finding in MRI was two large areas of focal gliosis and porencephaly located at head of right caudate. The size was 12 mm which is compatible with lacunar infarct. Frontal subcortical areas of signal change were noted. The symptoms were almost recovered only after 2 months treatment with sertraline 100 mg/daily and bupropione 150 mg/daily. It seems that there is a remarkable relationship between basal ganglia and frontal lesions, particularly infarction, with late-onset OCD.

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Introduction

Obsessive compulsive disorder (OCD) is one of the chronic and often disabling psychiatric disorders. OCD patients suffer from overwhelming recurrent persistent intrusive thoughts, ideas, images or urges (called obsessions) which can be followed by repetitive behaviors or mental acts (named compulsions) usually with the aim of tension reduction caused by obsessions (1). According to recent Iranian epidemiological study it has about 5.1% twelve-month prevalence (2). OCD is typically diagnosed within the age of 20 to 25 and half of them develop some symptoms before 20. Adult males and females are equally affected (3).

Onset of symptoms after the age of 35 is uncommon and only 15% of cases present symptoms after this age for the first time (3). Onset of OCD above the age of 50 is rare. It is important to note that our data is limited to case reports/series. Given its rare prevalence, late onset OCD may be more likely to have an organic etiology (4).

Various organic disorders have been reported in late-onset OCD, including focal cerebral lesions, most commonly involving the frontal lobes and/or the basal ganglia have also been

related to OCD (5, 6).

This report presents a new late-onset OCD with underlying some infarct lesions, particularly caudate lacunar infarct.

Case Report

A 59-year-old white married man is referred to psychiatric clinic. He was referred for recently developed obsessive compulsive symptoms with no prior history of any kind of OCD-like symptoms before.

He had some interpersonal and familial stressors 5 months ago in his history. His symptoms included ego-dystonic recurrent thoughts like doubting whether he closed the doors or turned off oven or not. These obsessional doubts leaded him to compulsively repetitive checking several times a day causing him remarkable distress and functional impairment. His Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) score was 28 (scale 0–40).

He had no prominent affective symptoms and his mood was euthymic but he claimed occasional problems in sleep pattern and complains of recent tension like headache and dizziness.

In his past medical records, he had a history of chronic hypertension which was well controlled with losartan 25 mg HS (Hour of sleep). He was taking prazosin 1 mg HS as well because of some symptoms secondary to benign prostate hypertrophy.

He had no major psychiatric disorder or OCD in his family history.

To rule out organic causes a complete neurological evaluation, laboratory tests and Brain MRI/DWI were done in the first visit.

His neurological exam revealed no deficit. Mini mental status examinations and different lobar function examinations including attention, working and semantic memory, language, executive functions, praxis, spatial visual ability, problem solving, insight and judgment were all intact. Laboratory findings were within normal range for CBC, renal and hepatic functions as well.

However, MRI analysis revealed finding was two large areas of focal gliosis and porencephaly located at head of right caudate with the size about 12 mm which was compatible with lacunar infarct. Besides, anterior limb of right internal capsule and right putamen and the interface between left internal capsule and left putamen had similar but less prominent lesions. Frontal subcortical areas of signal changes were noted due to headache sequela. Thalami and basal ganglia and periventricular high T2 signal change was notable secondary to small vessel disease (Figures 1, 2).

He was treated with sertraline 100 mg daily which was accompanied later with Bupropion 150 mg/daily as an adjunctive to minimize sexual problems occurred during SSRI treatment. He tolerated the treatment and had a good response within 3 months follow-up. After 1 month YBOCS reduced to 8 and after 2 months' obsessive symptoms recovered and YBOCS decreased to 0 and stable state continued to our last follow-up, 6 months after initiation of treatment.

Discussion

OCD is a psychiatric condition which its etiology is one of the challenging aspects of neuropsychiatry. Complex role of serotonergic

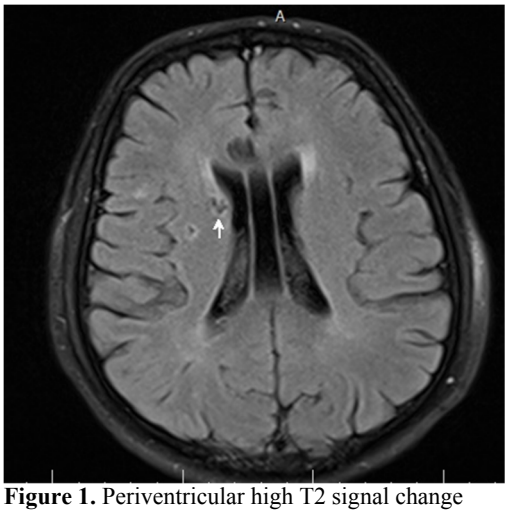

Figure 1. Periventricular high T2 signal change

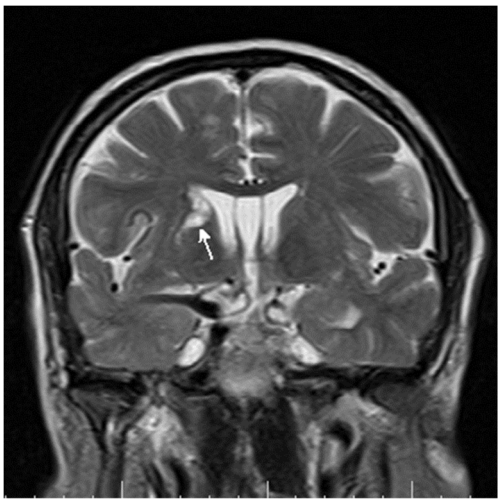

Figure 2. Thalami and basal ganglia signal change

system is most evidence-based accepted etiology (7). In addition, recent studies suggested other plausible etiological processes for this condition such as inflammatory (8) and glutamergic system mediated processes (9, 10).

In this case some brain lesions were reported according to MRI. Most noticeable lesion is the one, probably infarction, in head of right caudate. There are some reported cases that have been presented obsessive symptoms underlying to unilateral or bilateral lesions in basal ganglia (5, 11, 12) and most reported cases are late-onset patients without history of previous symptoms (5, 12, 13) similar to our case.

Moreover, in our case frontal subcortical changes were seen. There are some case reports that have reported obsessive-compulsive features concordant with frontal lobes lesions (4, 12).

Although one of the rationales for our findings is accidental concordance of initiation of psychiatric symptoms and imaging changes, according to previous mentioned reports it seems that there is a significant relationship between basal ganglia and frontal lesions, particular infarction, with late-onset OCD. On the other hand, we found a case report that presented a patient who relieved from obsessive symptoms after a left capsular genu infarction (14).

All of these reports support the role of neural connections between basal ganglia and frontal areas of brain in pathogenesis of OCD. On the other hand, we chose medication instead of Cognitive-Behavior Therapy (CBT) because of financial concerns and preference of patient. As mentioned in case presentation, he was almost recovered only after 2 months treatment with sertraline 100 mg and bupropione150 mg/daily. There is a noticeable gap about treatment of late-onset OCD caused by organic lesions in the literature. A paper reported a late-onset OCD patient following basal ganglia infarct who has been treated with CBT (13) and another case was reported to be responding to fluoxetine 80md/daily. More studies are required to come up with a reliable treatment for these patients.

Our case report is limited to relatively short period of follow-up, limited MRI imaging studies and lack of serological or functional studies.

In summery we conclude that late-onset OCD could be related to basal ganglia and frontal infarcts and physicians should pay more attention to probable underlying brain lesions in these patients, even at the absence of neurological signs.

Acknowledgments

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Conflict of interests

The authors declare no conflict of interests.

References

1. American Psychiatric Association Diagnostic and statistical manual of mental disorders (DSM-5®): American Psychiatric Pub; 2013.
2. Sharifi V, Amin-Esmaeili M, Hajebi A, Motevalian A, Radgoodarzi R, Hefazi M, et al. Twelve-month prevalence and correlates of psychiatric disorders in Iran: The Iranian Mental Health Survey, 2011. Arch Iran Med. 2015 Feb;18(2):76-84.
3. Rasmussen SA, Eisen JL. The epidemiology and clinical features of obsessive compulsive disorder. Psychiat Clin North Am. 1992 Dec;15(4):743-58.
4. Swoboda KJ, Jenike MA. Frontal abnormalities in a patient with obsessive-compulsive disorder: the role of structural lesions in obsessive-compulsive behavior. Neurology. 1995 Dec;45(12):2130-4.
5. Lopez-Rodriguez F, Gunay I, Glaser N. Obsessive compulsive disorder in a woman with left basal ganglia infarct: A case report. Behav Neurol. 1997;10(2):101-3.
6. Hofer H, Frigerio S, Frischknecht E, Gassmann D, Gutbrod K, Muri RM. Diagnosis and treatment of an obsessive-compulsive disorder following traumatic brain injury: a single case and review of the literature. Neurocase. 2013 Aug;19(4):390-400.
7. Askari N, Moin M, Sanati M, Tajdini M, Hosseini SM, Modabbernia A, et al. Granisetron adjunct to fluvoxamine for moderate to severe obsessive-compulsive disorder: a randomized, double-blind, placebocontrolled trial. CNS Drugs. 2012 Oct 1;26(10):883-92.
8. Shalbafan M, Mohammadinejad P, Shariat SV, Alavi K, Zeinoddini A, Salehi M, et al. Celecoxib as an Adjuvant to Fluvoxamine in Moderate to Severe Obsessive-compulsive Disorder: A Double-blind, Placebo-controlled, Randomized Trial. Pharmacopsychiatry. 2015 Jul;48(4-5):136-40.
9. Esalatmanesh S, Abrishami Z, Zeinoddini A, Rahiminejad F, Sadeghi M, Najarzadegan MR, et al. Minocycline combination therapy with fluvoxamine in moderate-to-severe obsessive-compulsive disorder: A placebo-controlled,

double-blind, randomized trial. Psychiat Clin Neurosci. 2016 Nov;70(11):517-26.
10. Arabzadeh S, Shahhossenie M, Mesgarpour B, Rezaei F, Shalbafan MR, Ghiasi Z, et al. L-carnosine as an adjuvant to fluvoxamine in treatment of obsessive compulsive disorder: A randomized double-blind study. Hum Psychopharmacol. 2017 Jul;32(4).
11. Yaramis A, Herguner S, Kara B, Tatli B, Tuzun U, Ozmen M. Cerebral vasculitis and obsessivecompulsive disorder following varicella infection in childhood. Turk J Pediat. 2009 JanFeb;51(1):72-5.
12. Weiss AP, Jenike MA. Late-onset obsessive-compulsive disorder: a case series. J Neuropsychiat Clin Neurosci. 2000 Spring;12(2):265-8.
13. Carmin CN, Wiegartz PS, Yunus U, Gillock KL. Treatment of late-onset OCD following basal ganglia infarct. Depres Anxiety. 2002;15(2):87-90.
14. Oh JH, Ahn BY, Jo MK, Yoon YJ, Park KH, Na DL, et al. Obsessive-compulsive behavior disappearing after left capsular genu infarction. Case Report Neurol. 2011 Jan 14;3(1):18-20.