



New Onset of Obsessive-Compulsive Disorder Post Cingulate Surgery for Refractory Epilepsy

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Abstract

We report the case of a 52 year-old man presenting obsessive-compulsive behaviors following a temporal anterior lobectomy in the context of refractory epilepsy. The patient's evolution and management are discussed. We elaborate on the pathophysiology of OCD (obsessive-compulsive disorder) and the potential mechanisms explaining this unusual scenario. We focus mainly on the implication of various pathways, neurotransmitters and disruptions along the striatal-orbitofrontal-thalamic interconnections.

Keywords: Obsessive-compulsive disorder; Refractory epilepsy; Structural epilepsy; Temporal epilepsy; Serotonin; Selective serotonin reuptake inhibitor; 5-HT

Abbreviations

OCD: Obsessive-Compulsive Disorder; SSRI: Selective Serotonin Reuptake Inhibitor; CNS: Central Nervous System; 5-HT: Serotonin Neurotransmitter

Case Report

A 52 year-old right-handed man was referred to neuropsychiatry regarding his compulsive behaviors. His history includes temporal lobe epilepsy, refractory to medication, since two years of age. The seizures occurred up to fifteen times per day, typically presenting as a rising epigastric sensation, sudden fear, speech arrest, followed by a dyscognitive phase and subsequent oral-buccal automatisms. Serial EEGs demonstrated a left temporal electrophysiological correlation.

Prior to surgery, his treatment consisted of phenytoin and carbamazepine at optimal doses. His family history was unremarkable for obsessive-compulsive or tic disorders. Apart from a remote history of recreational cocaine use in the adolescence, he had no psychiatric history. Three decades ago, he underwent an eventful left anterior temporal lobectomy. Immediately, the seizures frequency decreased drastically. Over the upcoming years, phenytoin has been tapered since he became seizure-free. Carbamazepine has been maintained at the same dose. About 20 years ago, a flood occurred in one of the buildings where he had been employed as a maintenance worker. This event triggered checking compulsions, increasingly incapacitating throughout the years. It took him 20 years to disclose this information to his neurologist.

This condition impacted his life significantly. Before leaving work, he would routinely check the taps several times to ensure that they were turned off, even with the insight that he already underwent this verification. Skipping a routine check induced debilitating anxiety. As a consequence of his inner doubts regarding the taps, he took pictures of the faucets used that day to demonstrate that they were effectively turned off. However, this ritual was insufficient to relieve his anxiety, as he still needed to return to work in the middle of the night to perform supplemental check-ups. Even if he had an ego dystonic perception of the overall situation, it was impossible to suppress those rituals.

There was no evidence of depression at the neuropsychiatric evaluation. He was prescribed citalopram, which was discontinued due to excessive nausea. No subsequent serotonergic medication was considered, as he refused to take additional drugs. Cognitive behavioral therapy has been the cornerstone of the treatment, leading to a moderate improvement in his symptoms & quality of life.

Discussion

Epilepsy results from aberrant synchronous discharges of the neuronal network. Focal temporal

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lobe epilepsy, is mainly associated with hippocampal sclerosis, previous complex febrile seizures and developmental abnormalities.

Obsessive-compulsive disorder is a psychiatric condition typically characterized by the coupling of two major components: intrusive compulsive thoughts, accompanied by ritualistic behaviors leading to an emotional relief. Anatomic circuits involved in OCD are still controversial; multiple possible theories coexist without a unifying consensus. Most models suggested disruptions along the striatal-orbitofrontal-thalamic interconnections. Along these interconnections, various neurotransmitters are well balanced in a finely regulated environment, including glutamate, serotonin, dopamine, and gamma-aminobutyric acid [1].

Serotonin has numerous functions within the central nervous system, mentioning: appetite regulation, sleep, memory, learning consolidation, mood, behavior, as well as the maturation of neuronal cells and synaptic connections. Amongst postulated theories pertaining to OCD pathophysiology, the most longstanding implicates serotonin. In the 1960s, scientists noted that the use of clomipramine, a tricyclic antidepressant and serotonin reuptake inhibitor, resulted in obsessions and ritualistic behaviors [2,3]. Consequently, high doses of SSRIs (selective serotonin reuptake inhibitors) show efficacy in the treatment of OCD [4,5].

Interestingly, an increased prevalence of obsessive-compulsive symptoms have been documented in treatment-refractory temporal lobe epilepsy [6]. Even more, agents elevating the extracellular 5-HT levels, such as 5-hydroxytryptophan and 5-HT reuptake blockers, prevent both focal and generalized seizures. However, depletion of serotonin CNS levels lowers convulsions threshold [6]. Interestingly, several anti-seizure drugs, including phenytoin, generate an increased concentration of extracellular brain 5-HT [7]. In our case, the use of phenytoin, although not particularly efficacious for the complete control of the patient's focal seizures, could have had a certain role in modulating and even suppressing OCD. However, the medication was tapered after clinical resolution of the seizures and the OCD behavior was present before and after the discontinuation of this drug. The subtle variations in symptomatology is difficult to evaluate retrospectively. Furthermore, it is also possible that our gentleman presented an undiagnosed OCD background from an early age, only revealed when serotonin became significantly dysregulated.

OCD and epilepsy may be linked according to the theory of an "interrupted pathological shared organization" resulting from neurosurgery. The temporal lobectomy leads to seizure control, but on the other hand contribute to the emergence of latent OCD traits [8,9]. Along the same line, the sudden postoperative seizure cessation might be perceived as "forced normalization". The presence of sustained excitatory epileptic form activity could have inhibited

the underlying psychiatric disorder. In this "latent disease theory", surgery represents the removal of this excitatory activity, potentially allowing for the OCD emerge [10].

Conclusion

Epileptic patients should be carefully screened for concomitant psychiatric disorders. In addition, prophylactic use of SSRIs could potentially prevent the emergence and decrease the severity of OCD in this specific patient population. Also, slow tapering of anti-seizure medication following epilepsy surgery is recommended. Overall, the role of neuropsychiatry in this patient population is of utmost importance and this expertise could prevent unexpected complications in the long-term management of refractory epilepsy.

References

1. Modell JG, Mountz JM, Curtis GC, Greden JF. Neurophysiologic dysfunction in basal ganglia/limbic striatal and thalamocortical circuits as a pathogenetic mechanism of obsessive-compulsive disorder. *J Neuropsychiatry ClinNeurosci*. 1989;1(1):27-36.
2. Fernandez CE, Lopez-Ibor JJ. Monochlorimipramine in the treatment of psychiatric patients resistant to other therapies. *ActasLusoEspNeurolPsiquiatriCiencAfines*. 1967;(26):119-147.
3. Lopez-Ibor JJ. Intravenous perfusion of monochlorimipramine: technique and results. In Cerletti A, Bove FJ, Editors. *The presentstatus of psychotropic drugs*. Amsterdam: Excerpta Medica, 1969.
4. Goodman WK, Price LH, Rasmussen SA, Delgado PL, Heninger GR, Charney DS. Efficacy of fluvoxamine in obsessive-compulsive disorder. A double-blind comparison with placebo. *Arch Gen Psychiatry*. 1989;46(1):36-44.
5. Jenike MA, Buttolph L, Baer L, Ricciardi J, Holland A. Open trial of fluoxetine in obsessive-compulsive disorder. *Am J Psychiatry*. 1989;146(7):909-11.
6. Isaacs KL, Philbeck JW, Barr WB, Devinsky O, Alper K. Obsessive-compulsive symptoms in patients with temporal lobe epilepsy. *Epilepsy Behav*. 2004;5(4):569-74.
7. Modell JG, Mountz JM, Curtis GC, Greden JF. Neurophysiologic dysfunction in basal ganglia/limbic striatal and thalamocortical circuits as a pathogenetic mechanism of obsessive-compulsive disorder. *J Neuropsychiatry ClinNeurosci*. 1989;1(1):27-36.
8. Liddle PF. Obsessive compulsive disorder. In: Liddle PF, editor. *Disordered Mind and Brain: the Neural Basis of Mental Symptoms*. London, UK: Gaskell; 2001:214-220.
9. Kulaksizoglu IB, Bebek N, Baykan B, Imer M, Gürses C, Sencer S, et al. Obsessive-compulsive disorder after epilepsy surgery. *Epilepsy Behav*. 2004;5(1):113-118.
10. Mace CJ, Trimble MR. Psychosis following temporal lobe surgery: report of six cases. *J Neurol Neurosurg Psychiatry*. 1991;54(7):639-44.