# Improvement in Obsessive-Compulsive Disorder Following Right Anterior Temporal Lobectomy and Amygdalohippocampectomy in a Patient with Refractory Temporal Lobe Epilepsy with Right Mesial Temporal Sclerosis

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## Abstract

There are reports of co-occurrence of obsessive–compulsive disorder (OCD) in patients with temporal lobe epilepsy (TLE). We present a report of a patient with refractory TLE due to hippocampal sclerosis with concomitant OCD on pharmacotherapy for both. She underwent surgery for standard anterior temporal lobectomy with amygdalohippocampectomy and reported improvement in obsessive–compulsive symptoms subsequently. We seek to further evidence of interaction between the two conditions and argue to undertake future research exploration on the same.

**Keywords:** Anterior temporal lobectomy and amygdalohippocampectomy, obsessive-compulsive disorder, temporal lobe epilepsy, mesial temporal sclerosis

## Introduction

Obsessive–compulsive disorder (OCD) is one of the common neuropsychiatric disorders affecting up to 1%–3% of the population. The key features in OCD are recurrent intrusive ideas, impulses, or urges (obsessions) along with overt or covert behaviors (compulsions) aimed at reducing the distress.[1] Epilepsy affects up to 1% of the population and is one of the common of neurological disorders among adults.[2] Partial-onset epilepsies account for about 60% of all adult epilepsy cases, among which temporal lobe epilepsy (TLE) is the most common type. TLE is often refractory to antiepileptic drugs (AEDs) and may require epilepsy surgery.

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Epilepsy is frequently associated with wide-ranging neuropsychiatric manifestations,[3], incidence of which is much higher among the treatment-resistant epilepsy and TLE.[4] There is also an increased risk for emotional, behavioral, cognitive, and perceptual psychopathology. TLE has a higher frequency of lifetime psychiatric disorders of up to 70%, with mood disorders (49.3%) being the most frequent along with OCD in 11.0% of participants as compared to 2.3% (0.3) of lifetime prevalence among normal adult population.[5] This association of TLE and OCD has been described in relatively few studies.[6]

We describe a patient with drug-resistant TLE due to hippocampal sclerosis with OCD who had improvement of her neuropsychiatric symptoms of OCD following surgery for temporal epilepsy.

## Case Report

A 33-year-old right-handed female, with normal development, had presented with recurrent episodes of seizures since 11 years of age. The seizure frequency at presentation was twice a month while on optimal antiepileptic medication, namely, lamotrigine 150 mg/day, levetiracetam 1.5 g/day, and clobazam 30 mg/day. The semiology was in the form of an aura of fearfulness followed by behavioral arrest, bimanual automatisms with hypersalivation, and subsequently, left upper-limb posturing with rare secondary generalization, suggestive of right medial temporal involvement.

In addition, she had behavioral symptoms, for the past 16 years, in the form of interictal fear of contamination with bodily fluids and excreta, compulsive rituals of washing after defecation and spending most of the time in the washroom. She also had aggressive, harmful obsessions with magical thinking and cognitive compulsions. Her primary obsession was excreta sticking to body parts after passing stools. These symptoms were suggestive of OCD. She scored 25 on the Yale–Brown obsessive–compulsive scale (Y-BOCS). On Selective Serotonin Reuptake Inhibitor (SSRI) trial, there was about 40% improvement in her behavioral symptoms.

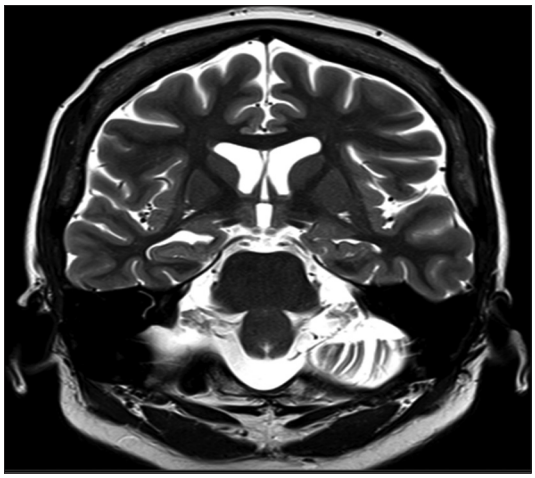

**Figure 1:** T-2 weighted MRI showing right hippocampal volume loss and a mild dilation of the right temporal horn

The patient underwent evaluation for drug-resistant focal epilepsy. Magnetic resonance imaging (MRI) brain [Figure 1] was suggestive of right mesial temporal sclerosis (MTS), and video-telemetric electroencephalogram (EEG) [Figure 2] showed right anterior and middle temporal onset of seizures. Positron emission tomography (PET)-computed tomography revealed moderate-to-severe hypometabolism of the right temporal region. Neuropsychological evaluation showed right temporal involvement. In view of drug resistance and clinico-electro-radiological and neuropsychological concordance, she underwent right anterior temporal lobectomy with amygdalohippocampectomy. There was no immediate postoperative complication. The histopathological report was suggestive of the WHO Type 1a MTS.

Her medication was not changed, and at 6-month postsurgery, she was on lamotrigine 300 mg/day, levetiracetam 1.5 g/day, and clobazam 30 mg/day. After 10 months of surgery, levetiracetam was tapered and topiramate was introduced. At 12 months, she was on clobazam 30 mg/day, lamotrigine 300 mg/day, and topiramate 250 mg/day.

At 6-month follow-up while on the same AEDs, she reported a significant decrease in seizure frequency and duration (International League Against Epilepsy [ILAE] Class 2 outcome), but interestingly, she also reported subjective improvement with respect to her OCD symptomatology which was noted objectively with improvement of her Y-BOCS score to 3. The repeat neuropsychological evaluation at 6-month post-operation showed dorsolateral prefrontal and temporal lobe involvement. At 12-month follow-up, her Y-BOCS score was recorded at 2, and the seizure outcome was ILAE Class 2. She reported that her subjective quality of life had significantly improved due to decrease of OCD symptoms.

## Discussion

The highlight of this report is the remission of symptoms of OCD after anterior temporal lobectomy with amygdalohippocampectomy, which was primarily performed for achieving seizure freedom. TLE has been associated with obsessive–compulsive syndrome quite prominently, especially refractory epilepsy of the temporal lobe.[7] While there is no known overt locus of neuronal degeneration in OCD, functional imaging studies suggest an abnormal metabolic activity in the orbitofrontal cortex, anterior cingulate/caudal medial prefrontal cortex, and caudate nucleus.[8]

Till date, several researchers have proposed the involvement of the temporal lobe in obsessive–compulsive syndrome, with EEG abnormalities being reported over the temporal lobe.[9] Imaging studies[10] demonstrate voxel-based comparison of changes in cerebral glucose metabolism rates pre- and posttreatment in PET, which showed significantly correlated metabolic changes in the hippocampus along with putamen and cerebellum. fMRI studies have also shown activation in the anterior, medial, and lateral temporal cortex along with other areas when provocative stimuli were presented to patients with

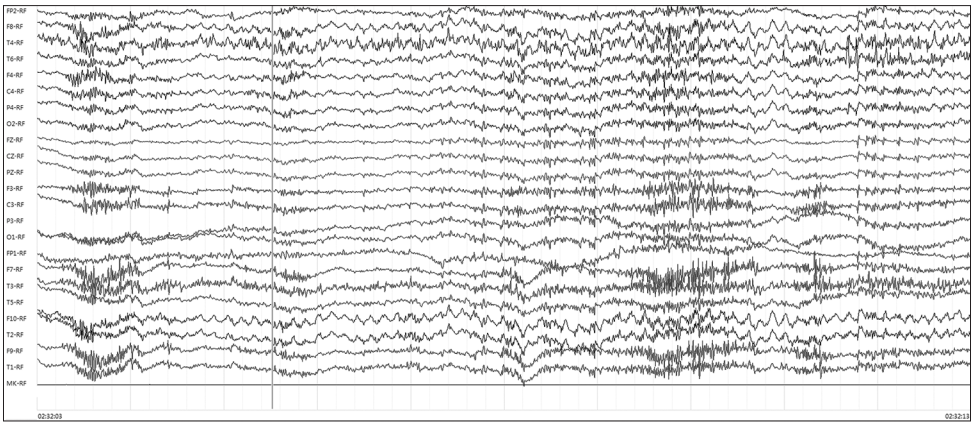

**Figure 2:** Ictal scalp electroencephalogram: Average referential montage showing buildup of theta rhythm in the right anterior and middle temporal region; Sensitivity 10uv, low-pass filter 50, high-pass filter 0.3

OCD.[11] It has also been observed that there is an increase in the regional cerebral blood flow by PET in posterior cingulate cortex and hippocampus in OCD patients in whom a brief exposure to a hierarchical contaminant was given.[12]

A few case reports also suggest a similar dynamic relationship between the temporal lobe and OCD where remission of OCD symptoms was seen after temporal lobectomy. Guarnieri *et al.*[13] described two such patients with benefit of OCD symptoms following temporal lobectomy: one with complex partial seizures with OCD who underwent anterior temporal lobectomy and another male who had simple partial seizures and OCD symptoms who underwent left functional hemispherotomy with temporal lobectomy. Similarly, remission of OCD symptoms after temporal lobectomy was first reported by Kanner *et al*.[14]

These findings, along with the accumulating evidence of structural and electrophysiological abnormalities in the temporal lobe[9,15] in OCD patients, have prompted a more through look into the network dysfunction in OCD. Classical models of OCD suggest involvement of two principal cortical loops. These are the thalamo-orbitofrontal loop mediated by glutamate and a collateral loop that includes striatal-thalamic-cortical (STC) interconnections mediated additionally by serotonin, dopamine, and gamma-aminobutyric acid (GABA).[16]

STC circuits are noted to be involved in higher-order cognitive functions such as inhibition of impulsive behavior, action selection/modulation of motor activity, and attentional allocation. The STC circuits involve mainly the frontal cortex. These loops are comprised of (1) glutamatergic cortico-striatal projections synapsing onto striatal spiny projection neurons and/or interneurons; (2) GABAergic spiny projection neurons targeting basal ganglia output structures (globus pallidus pars internalis [GPi] and substantia nigra pars reticulate [SNr]); (3) GABAergic output neurons from GPi/SNr projecting to thalamic regions; and (4) glutamatergic neurons from thalamus projecting back to cortex.[17]

The communication fibers from the frontal cortex travel to the basal ganglia through the internal capsule and the anterior cingulate. Disruption of this pathway has been implicated in the organization of OCD symptoms. Anterior cingulate cortex dysfunction may serve as the platform for the shared neurobiology between TLE and OCD. This is supported by the evidence of improvement in OCD symptoms followed by capsulotomy and anterior cingulate resection.[18] A shared network disruption of the anterior cingulate ties in with coexistence of affective disorders with TLE. Evidence shows increased default mode network deactivation in resting-state studies in TLE patients with affective disorders, particularly in the subgenual anterior cingulate cortex.[19]

## Conclusion

The occurrence of OCD in patients with TLE and its remission rarely with temporal lobectomy have both been well documented. With the severity of OCD increasing with greater duration of uncontrolled seizures, such an undertaking will not only be important for prognostication but also for improving the quality of life for the patients concerned. It will also provide opportunity to understand the biological basis of OCD.

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### Conflicts of interest

There are no conflicts of interest.

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