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Deep Transcranial Magnetic Stimulation Effects on the Electrophysiological Parameters in Obsessive-Compulsive Disorder

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Abstract

Backgrounds. Deep Transcranial Magnetic Stimulation (dTMS) is a non-invasive treatment cleared by FDA as a safe and efficient intervention for the treatment of depression and obsessive-compulsive disorder (OCD). *Objectives.* In this retrospective single-center study, the effects of dTMS on the electrophysiological parameters and the clinical outcomes of patients with OCD were tested. *Methods.* Thirty sessions of dTMS were administered to 29 OCD patients (15 female and 14 male). Quantitative electroencephalography (QEEG) recordings and Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) were measured at baseline and endpoint. Paired sample t-test was used to measure the change in Y-BOCS scores and QEEG activity after dTMS practice. *Results.* All 29 patients responded to the dTMS intervention by indicating at least 35% reduction in Y-BOCS scores. QEEG recordings revealed a significant decrease in theta, alpha and the beta rhythms. The decrease in the severity of OCD symptoms correlated with the decrease in beta activity at left central region. *Conclusions.* Historically, excess fast oscillations in OCD are correlated with the unresponsiveness to selective serotonin reuptake inhibitor (SSRI) treatment. We hypothesize that the decrease in the power of beta bands by deep TMS is related to the mechanism of the therapeutic response.

Keywords

deep TMS, H7 coil, quantitative EEG, OCD, beta oscillations, Y-BOCS

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Introduction

Deep Transcranial Magnetic Stimulation (dTMS) is a non-invasive brain stimulation technique that uses repetitive magnetic field pulses to induce ionic currents in the pyramidal neurons of the cortex. By repetitive training sessions, cortical excitability on the targeted areas of the brain can be maintained beyond TMS sessions .[1]

The H7 coil OCD protocol designed by Brainsway in 2018,[2] also called as deep TMS, directly stimulates the medial prefrontal cortex (mPFC), dorsal anterior cingulate cortex (dACC), dorsolateral prefrontal cortex (dlPFC) orbito frontal cortex (OFC), inferior frontal gyrus (IFG) and pre supplementary motor area (pre SMA); thereby, helps to modulate the dysfunction of cortico-striato-thalamo-cortico (CSTC) circuit in which underlying pathophysiology of OCD resides.[2,3] Deep TMS differs from the standard rTMS treatment in reaching deeper and wider structures of the brain.[2] In 2018, The U.S Food and Drug Administration (FDA) approved the therapeutic use of H7-coil protocol in OCD patients who failed to benefit from the traditional first-line treatments.[4] Two years later another coil, D-B80 coil, was cleared by FDA for therapeutic use, however, a recent study compared the both FDA-cleared coils and proposed that some OCD specific prefrontal regions which constructs CSTC can be stimulated by H7 coil but not by D-B80 coil.[5]

The clinical trial investigating clinical and electrophysiological effects of deep TMS effects on OCD patients applied 25 sessions H7 coil OCD protocol five times per week for 5 weeks.[2] Before the first sessions and after the last session, event related potentials (ERPs) of the patients were recorded using an electroencephalogram (EEG). By this application, patients' electrophysiological responses to making an error, called as error-related negativity (ERN) were extracted from

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the ERP recordings. The results revealed that neurostimulation of mPFC and ACC activity with high frequency (20 Hz) leads to a change in the error-related negativity (ERN), an electrophysiological endophenotype of OCD.[2] A recent multisite study demonstrated that the durability of the treatment is generally close to two years.[6]

One of the many remaining questions is how deep TMS for OCD interacts with the general electrophysiological activity rather than electrophysiological response to stimulus investigated before.[2] A study on electrophysiological activity of OCD patients reveals that the cortical and subcortical circuits constructing the pathophysiology of OCD is hyperactive both in resting-state and during exposure to OCD related autobiographical events.[7] Hence, obsessive-compulsive symptom improvement after a treatment course can be related with the change in that hyper synchronicity. Like previous clinical study which use the EEG to measure deep TMS effects on brain response to making an error in OCD patients, the resting-state electrophysiological activities of OCD patients after deep TMS treatment can also be observed via quantitative electroencephalography, a non-invasive, affordable technique. There are several publications on the electrophysiological outcomes of conventional repetitive TMS in OCD patients using resting-state QEEG.[8,9] The first one is had small sample size (n = 5) and could not give reliable result.[8] The second one had good sample size but had only pretreatment results to differentiate responsive OCD patients to rTMS treatment from non-responsive ones.[9] As for deep TMS, resting-state QEEG activities of OCD patients has yet to be investigated. The primary aim of the present study is to observe the effects of the high frequency deep TMS on the electrophysiological and clinical outcomes of OCD patients.

Methods

Participants

This retrospective study included patients who consulted Kemal Arıkan Psychiatry Clinic (a private psychiatric practice) in Istanbul, Turkey. Each patient had been informed of the procedures and potential side effects of deep TMS before informed consent was obtained. Local ethics committee permission was received for the study. Twenty-nine OCD patients (15 female and 14 male) aged at 18 - 60 (mean ± SD = 32.30 ± 11.17 years) providing the following criteria were included:

1. Outpatients diagnosed by an experienced psychiatrist, with a diagnosis of OCD by DSM-5 (Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition) criteria.[10]
2. Non-response to at least 2 selective serotonin reuptake inhibitor (SSRI) group medications.
3. Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) scores at baseline and endpoint.
4. QEEG at baseline and after a course of deep TMS.

Further demographic information can be seen in Table 1. None of the participants have acknowledged any neuropsychological or organic diseases (eg, epilepsy), yet some of the patients had also unipolar depression. Patients, except one, were using SSRIs during 30-session deep TMS treatment. The primary clinical measure of OCD was Yale-Brown Obsessive-Compulsive Scale (Y-BOCS).[11] Response was defined as at least a 35% reduction in Y-BOCS scores from baseline to endpoint. In addition to Y-BOCS, depressive and anxiety symptoms evaluated by 17-item Hamilton Depression Rating Scale (HAMD),[12] Hamilton Anxiety Rating Scale[13] at baseline and endpoint were also investigated. Half of the patients had also clinical measurements after dTMS intervention (Table 2).

QEEG Recording

All subjects underwent QEEG recording before and after deep TMS. Resting-state QEEG recordings were recorded in a silent, dimly lit, air-conditioned room. A 19-channel (FP1, F7, T3, T5, F3, C3, P3, O1, FZ, CZ, PZ, F4, C4, P4, O2, FP2, F8, T4, and T6) electro-cap was positioned on the head of the participants in accordance with the 10–20 international system. A transparent electro-gel was injected into the scalp electrodes to increase conductivity. The ground electrode was placed in the FPz position. Mastoid electrodes were positioned to both earlobes as reference electrodes. The impedance of electrodes was controlled <5 kΩ for each electrode. A Neuron-Spectrum-4/P device was utilized to record resting-state QEEG activity while patients were in a comfortable sitting-positioned, closed-eye state. The total duration of records was approximately 7 minutes, including a three-minute background recording, a thirty-second open eyes condition, and a three-and-half-minute closed-eyed condition. Data were sampled at 500 Hz rate; signals were bandpass

Table 1. Demographic Information of Patients.

	Gender	M	SE	Median	SD
Age	M (n = 14)	32.64	2.71	32.50	10.14
	F (n = 15)	32.13	3.39	27.00	13.57
Session Count	M (n = 14)	31.29	1.40	30.00	5.23
	F (n = 15)	32.56	1.34	30.00	5.35

Abbreviations: M, Male; F, Female.

Table 2. Clinical Scores of Patients who Came to Further Examination After dTMS Treatment (n = 15).

	M	SD
Duration (days)	49.33	35.63
Y-BOCS	3.71	7.70
HAMD	0.53	1.55
HARS	2.73	5.61

Abbreviations: Y-BOCS, Yale-Brown Obsessive Compulsive Rating Scale. HAMD, 17-item Hamilton Depression Rating Scale; HARS, Hamilton Anxiety Rating Scale.
Duration represents the number of days since the last dTMS session.

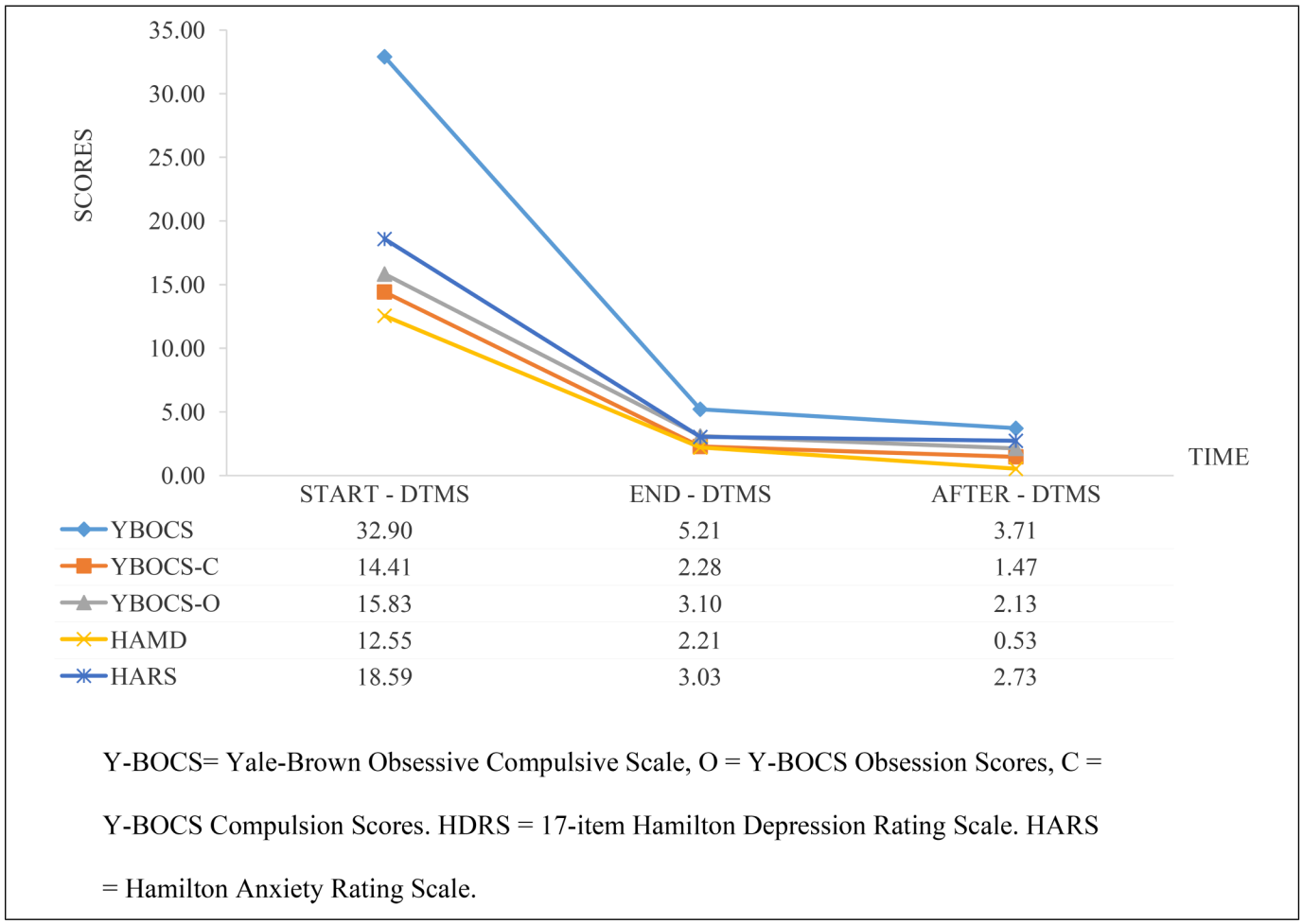

Figure 1. Mean scores on clinical measurements based on three time points.
Abbreviations: Y-BOCS, Yale-Brown Obsessive Compulsive Scale; O, Y-BOCS Obsession Scores; C, Y-BOCS Compulsion Scores; HAMD, 17-item Hamilton Depression Rating Scale; HARS, Hamilton Anxiety Rating Scale.

filtered at 0.15-70 Hz and notch filtered at 50 Hz. Muscle artifacts were eliminated from the raw QEEG recordings. The elimination of artifacts was done manually by an experienced EEG reader. Samples with artifacts were deleted and a minimum of 3-minute edited data was obtained. Each patients' data were averaged across the recording epochs for each electrode, and the absolute power was computed for the following bands: delta (1-4 Hz), theta (4-7 Hz), alpha (8-12 Hz), alpha1 (8-10 Hz), alpha2 (10-12 Hz), beta (12-25 Hz), beta1 (12-15 Hz), beta2 (15-18 Hz), beta3 (18-25 Hz), high beta (25-30 Hz).

Deep TMS Intervention

For the deep TMS sessions, Brainsway's H7 coil deep TMS System was used (Brainsway, Har Hotzvim, Jerusalem, Israel). OCD protocol was applied according to the guidelines of the manufacturing company.

The measurement of the motor threshold and the intervention were conducted by two certified clinical practitioners. The motor threshold (MT) was measured with the H7 coil positioned over the leg area of the motor cortex and a minimum threshold was detected when an observable twitch is seen in either resting leg. The motor threshold was reassessed in each treatment session. Treatment position of the coil for OCD is 4 cm anterior the location where maximum stimulation is observed for the motor threshold measurements.

Deep TMS was administered at 100% of the resting leg motor threshold in 20-Hz 2-second trains, with 20-second intertrain intervals, for fifty trains totaling 2000 pulses. Prior to the stimulation, patients' individualized OCD symptoms were provoked.[14]

Statistical Analysis

QEEG data were analyzed by Neuroguide Deluxe v.2.5.1 program (Applied Neuroscience, Largo, FL). The absolute power of each frequency band was calculated for all electrodes. To achieve normality, logarithmic transformation was applied. Since EEG absolute powers can take place between 0 and 1 and

logarithmic transformation converts values between 0 and 1 to negative values, all EEG absolute power values are transformed by adding 1 number so that all transformed values are ensured to be positive values. Then, data became has been made to convenient to statistical analysis. Statistical analyzes were computed in SPSS version 25. For both clinical scales and QEEG data normality was checked via the Kolmogorov-Smirnov test and the normality assumption was not rejected. Therefore, paired sample t-test was selected to measure pre-post intervention change in QEEG absolute powers for all frequency bands.

As for the clinical measurements, repeated measures analysis of variance (ANOVA) was selected to investigate the change across three time points, i.e, start, end, after deep TMS session. Bonferroni correction was used for post-hoc analysis. The significance level was decided to be set at $P < .01$ level.

Further, the relationship between the change in Y-BOCS scores and the change in qEEG absolute power was investigated for the significant electrode-band pairs. As the normality assumption was rejected for the change in Y-BOCS scores and QEEG electrode band pairs, Spearman's rank-order correlation was selected. For this analysis, the significance level was set at $P < .05$, two-tails.

Results

Clinical Measurements

All the patients responded well to the dTMS based on the 35% reduction in Y-BOCS criteria. Repeated measures ANOVA showed that dTMS course had an effect on the Y-BOCS, HAMD and HARS scores ($P < .001$). Post-hoc analysis gave the expected result. The statistically significant difference was observed between first-second scores and first-third scores ($P < .01$). There was no change between second and third scores (Figure 1 and Table 3).

QEEG Band Powers

Paired sample t-test revealed that there was a significant ($P < .01$) difference between pre-dTMS and post-dTMS QEEG absolute powers for patients with OCD. After 30-session dTMS treatment course, patients with OCD exhibited a decrease in the in the theta (4-7 Hz), alpha (8-12 Hz) beta (12-25 Hz), beta1 (12-15 Hz), beta2 (15-18 Hz), beta3 (18-25 Hz) activity in C3, C4, P3, P4, Pz, O1, O2, F7 and T5 regions (Table 4).

Correlations

Spearman correlations indicated that there is a positive relationship between the change in the severity of OCD symptoms, both obsessive and compulsive subscores and the change in qEEG beta absolute power at left central (C3) region after dTMS intervention (Table 5).

Discussion

The present study demonstrates the effect of deep TMS on SSRI resistant OCD patients, reducing the power of high

Table 3. Post-hoc Analyses for Clinical Measurements Based on Time.

					95% CI	
(I) Time	(II) Time	Mean Difference (I-II)	SE	*P*	Lower Bound	Upper Bound
Y-BOCS 1	Y-BOCS 2	28.93	2.10	.000	23.17	34.69
Y-BOCS 1	Y-BOCS 3	29.79	2.69	.000	22.41	37.16
Y-BOCS 2	Y-BOCS 3	0.86	2.28	1.000	−5.40	7.11
Y-BOCS-C 1	Y-BOCS-C 2	13.13	1.90	.000	7.97	18.30
Y-BOCS-C 1	Y-BOCS-C 3	13.67	1.93	.000	8.41	18.92
Y-BOCS-C 2	Y-BOCS-C 3	0.53	1.16	1.000	−2.63	3.69
Y-BOCS-O 1	Y-BOCS-O 2	15.13	1.52	.000	11.02	19.25
Y-BOCS-O 1	Y-BOCS-O 3	15.40	1.78	.000	10.56	20.24
Y-BOCS-O 2	Y-BOCS-O 3	0.27	1.14	1.000	−2.84	3.38
HAMD 1	HAMD 2	10.73	2.90	.010	2.84	18.63
HAMD 1	HAMD 3	10.67	2.99	.010	2.55	18.78
HAMD 2	HAMD 3	−0.07	0.41	1.000	−1.18	1.04
HARS 1	HARS 2	15.20	3.22	.000	6.44	23.96
HARS 1	HARS 3	13.73	3.48	.000	4.29	23.18
HARS 2	HARS 3	−1.47	1.28	.81	−4.94	2.01

Abbreviations: Y-BOCS, Yale-Brown Obsessive Compulsive Scale; O, Y-BOCS Obsession Scores; C, Y-BOCS Compulsion Scores; HAMD, 17-item Hamilton Depression Rating Scale; HARS, Hamilton Anxiety Rating Scale. The first scores for each scale are pre-dTMS, the second scores are end-dTMS, and the third scores are after-dTMS measurements.
P < .01.

Table 4. Paired Sample t Test Results for EEG Measures at the Beginning and end of dTMS Intervention.

				Paired t-test			
Measures[a]	M	SD	SE	M difference	t-value	df	*P* (two-tailed)
AP C3 Beta 1	2.482	0.604	0.112	0.121	3.148	28	.004
AP C3 Beta 2	2.360	0.480	0.089				
AP C3 Beta 1.1	1.549	0.492	0.091	0.081	3.324	28	.002
AP C3 Beta 1.2	1.467	0.444	0.082				
AP C4 Beta 2.1	1.365	0.485	0.090	0.114	3.137	28	.004
AP C4 Beta 2.2	1.250	0.410	0.076				
AP P3 Beta 1	2.630	0.543	0.100	0.143	3.664	28	.001
AP P3 Beta 2	2.487	0.454	0.084				
AP P3 Beta 2.1	1.389	0.479	0.089	0.123	3.405	28	.002
AP P3 Beta 2.2	1.265	0.414	0.076				
AP P3 Beta 3.1	1.742	0.518	0.096	0.156	3.357	28	.002
AP P3 Beta 3.2	1.585	0.388	0.072				
AP P4 Beta 1	2.687	0.524	0.097	0.121	3.417	28	.002
AP P4 Beta 2	2.565	0.444	0.082				
AP P4 Beta 2.1	1.439	0.457	0.084	0.113	3.38	28	.002
AP P4 Beta 2.2	1.325	0.378	0.070				
AP P4 Beta 3.1	1.796	0.490	0.091	0.134	3.363	28	.002
AP P4 Beta 3.2	1.661	0.389	0.072				
AP Pz Beta 1	2.775	0.504	0.093	0.122	3.527	28	.001
AP Pz Beta 2	2.653	0.425	0.078				
AP Pz Beta 2.1	1.521	0.458	0.085	0.125	3.683	28	.001
AP Pz Beta 2.2	1.395	0.382	0.070				
AP Pz Beta 3.1	1.855	0.496	0.092	0.131	3.433	28	.002
AP Pz Beta 3.2	1.724	0.382	0.071				
AP O1 Beta 1	2.890	0.553	0.102	0.170	4.057	28	.000
AP O1 Beta 2	2.720	0.545	0.101				
AP O1 Beta 1.1	2.112	0.573	0.106	0.139	3.226	28	.003
AP O1 Beta 1.2	1.973	0.604	0.112				
AP O1 Beta 2.1	1.522	0.482	0.089	0.149	4.342	28	.000
AP O1 Beta 2.2	1.373	0.453	0.084				
AP O1 Beta 3.1	1.933	0.551	0.102	0.173	3.856	28	.001
AP O1 Beta 3.2	1.760	0.504	0.093				
AP O2 Beta 1	2.968	0.519	0.096	0.162	4.407	28	.000
AP O2 Beta 2	2.805	0.535	0.099				
AP O2 Beta 2.1	3.533	1.023	0.189	0.124	4.134	28	.000
AP O2 Beta 2.2	3.321	1.139	0.211				
AP O2 Beta 3.1	2.017	0.535	0.099	0.194	4.945	28	.000
AP O2 Beta 3.2	1.822	0.471	0.087				
AP F7 Beta 1.1	1.194	0.430	0.079	0.087	3.378	28	.002
AP F7 Beta 1.2	1.106	0.343	0.063				
AP T5 Theta 1	2.265	0.537	0.099	0.185	3.106	28	.004
AP T5 Theta 2	2.080	0.525	0.097				
AP T5 Beta 1	2.376	0.537	0.099	0.179	3.15	29	.004
AP T5 Beta 2	2.196	0.489	0.090				
AP T5 Alpha 2.1	2.601	0.760	0.141	0.245	3.241	28	.003
AP T5 Alpha 2.2	2.355	0.848	0.157				
AP T5 Beta 2.1	1.197	0.439	0.081	0.134	3.153	28	.004
AP T5 Beta 2.2	1.063	0.384	0.071				

AP, Absolute power. For each electrode-band pairs, the first row indicates pre-dTMS measurement and the second row represents end-dTMS measurement. $P < .01$.

Table 5. Spearman's Rank-Order Correlations Between the Change in Y-BOCS Scores and the Change in qEEG Electrode-Band Pairs.

	Spearman's rho (ρ)				
Measures[a]	Y-BOCS Chg	Y-BOCS-O Chg	Y-BOCS-C Chg	C3 Beta Chg	C3 Beta 1 Chg
Y-BOCS Chg	1				
Y-BOCS-O Chg	.568**	1			
Y-BOCS-C Chg	.494**	0.329	1		
C3 Beta Chg	.403*	0.259	.396*	1	
C3 Beta 1 Chg	.524**	.387*	.524**	.868**	1

Note: [a]Y- BOCS, Yale-Brown Obsessive Compulsive Scale; Y-BOCS-O, Obsession score; Y-BOCS-C, Compulsion score; Chg, Change. The change in scores was calculated by subtracting pre-treatment scores from the post-treatment measures. $n = 29$ for all variables. * $P < .05$, ** $P < .01$.

frequency EEG activities and the severity of symptoms. After 30- treatments, OCD patients exhibited decreased EEG activity particularly in beta band at bilateral centro-parieto-occipital regions which are the endpoints of OCD related cortico-straito-thalamo-cortico circuit. After deep TMS treatment, the decrease in qEEG beta activity observed in the left central region was associated with the decrease in the severity of OCD symptoms. A prior qEEG study reveals that OCD patients showed increased beta-3 activity in occipital lobe, lingual gyrus and limbic lobe during exposure to OCD related autobiographical scripts.[15] Other studies report the association between increased beta activity, impulsivity and preparation of movements.[15,16] Accordingly, the present findings, ie, the reduced beta activity in centroparietal and occipital regions along with obsessive-compulsive symptom improvement may be the electrophysiological marker of response to deep TMS treatment. These findings indicate that deep TMS is an effective intervention for the treatment resistant OCD patients by decreasing the fast electrophysiologic oscillations.

One of the interesting results is that cortical excitability induced by high frequency TMS was not observed, on the contrary a decrease in fast oscillations was found. An analysis of resting-state QEEG data by low resolution electromagnetic tomography revealed that OCD patients who did not respond to sertraline treatment and behavioral therapy had higher beta 1, beta 2 and beta 3 activity at baseline compared to responders[17] including parietal regions.[18] Another study measuring electrophysiological biomarkers of response to SSRIs in OCD patients associates the excess in QEEG absolute power in high frequency bands with non-response to paroxetine treatment.[19] Given that the excess in QEEG fast oscillations are the biomarkers of unresponsiveness to SSRI treatment in OCD patients, the findings of the present study revealed that the decrease in the power of beta by the help of dTMS influenced therapeutic response.

It is noteworthy to mention that many of the dTMS patients use concomitant medications. The initial TMS studies in depression,[20] required patients be washed off of medications primarily for safety purposes. There was a concern of higher seizure risks when combining patients with psychotropic medications. Nevertheless, subsequent real-world utilization studies showed the risk of seizures to be very low as long as medication dosages were not changed, and the motor thresholds were rechecked frequently.[21] So, more recent TMS studies in numerous indications have allowed concomitant medications provided the dosages remain stable to allow conclusions to be made.[22] To the best of our knowledge, there is no evidence of a synergistic effect between TMS and medications, however, there is a general advantage from not having to wash patients off of their medications which can sometimes worsen patients.

The limitations of this study stem from the single center, retrospective design. Since there was no sham-control condition, it is possible that the observed EEG changes were non-specific. Furthermore, because all the patients were responders, it remains unclear if non-responders would have been associated with a lack of changes in beta EEG power. Therefore, these results should be considered preliminary and require confirmation in a prospective study.

The efficacy of deep TMS on OCD has been reported by the decrease in symptom severity measurements in several studies. QEEG is also a technique used in psychiatric practice to pursue changes in the electrophysiological activity of patients after clinical interventions. In the present study, the effects of deep TMS were observed in electrophysiological parameters and clinical outcomes. Considering the electrophysiological correlation of treatment resistant OCD to high frequency EEG oscillations, the deep TMS effect may act through slowing potency. We propose more broadly that deep TMS is an electrophysiological intervention that leads to significant changes in EEG activity and clinical outcomes in treatment resistant patients.

Author Contributions

Author 1 designed the research and wrote the protocol. Author 2, author 3, author 4 and author 5 conducted literature searches and provided summaries of previous research studies. Author 6 and author 7 conducted the statistical analysis. Author 2 and author 6 designed the Figure 1. Author 2 wrote the first draft of the manuscript. Author 8 revised and wrote the second draft of the manuscript. All authors contributed to and have approved the final manuscript.

Declaration of Conflicting Interests

The author(s) declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: Only the last author has a financial interest in BrainsWay, a clinical and research TMS center and the Clinical TMS Society. He is a consultant to BrainsWay, and he met the authors through a BrainsWay funded study. In no way did this impact the integrity of the data of this study.

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Ethical Approval

Not applicable, because this article does not contain any studies with human or animal subjects.

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