Efficacy of Adjunctive Deep Transcranial Magnetic Stimulation in Obsessive-Compulsive Disorder

A Randomized Sham-Controlled Study

Sachin Reddy, MD, Nishant Goyal, MD,† and Umesh Shreekantiah, MD†*

Objectives: Obsessive-compulsive disorder is one of the most common neuropsychiatric disorders with a higher lifetime prevalence than schizophrenia, often showing inadequate response to pharmacological and psychotherapeutic treatments. This study aimed to examine the efficacy of adjunctive deep transcranial magnetic stimulation (dTMS) in a randomized, sham-controlled set-up, addressing inadequate response to standard treatments.

Methods: Forty-nine obsessive-compulsive disorder patients were randomly allocated to receive either high-frequency dTMS (20 Hz) or sham stimulation and received 10 sessions of treatment using the H7 coil to target the dorsal anterior cingulate cortex and the medial prefrontal cortex over a period of 2 weeks. Change in Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) scores was the primary outcome measure. Secondary efficacy measures were changes in Hamilton Anxiety Rating Scale and Hamilton Depression Rating Scale scores and response rates as measured by Y-BOCS.

Results: The active dTMS group demonstrated a significant reduction in Y-BOCS scores compared with sham (−10.4 vs −2.6 points; $P < 0.001$), with an effect size of 1.39. Full response rates were 75% in the active group versus 5% in the sham group ($P < 0.001$). Anxiety and depressive symptoms also improved significantly in the active group (Hamilton Anxiety Rating Scale: −9.1 vs −2.4 points, $P < 0.001$; Hamilton Depression Rating Scale: −5.9 vs −1.8 points, $P < 0.001$).

Conclusion: Our study demonstrated that dTMS targeting the dorsal anterior cingulate cortex and medial prefrontal cortex significantly improved obsessive-compulsive, anxiety, and depressive symptoms, with faster response rates and fewer sessions compared with previous trials, suggesting that dTMS may serve as an effective early intervention for a wider range of obsessive-compulsive disorder patients.

Key Words: deep transcranial magnetic stimulation, obsessive-compulsive disorder, medial prefrontal cortex, TMS

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Obsessive-compulsive disorder (OCD) is a chronic neuropsychiatric condition with a lifetime prevalence of approximately 1% to 3% in India and globally.[1] The disorder imposes significant disability, impairing patients' quality of life and burdening their families at levels comparable to other severe mental illnesses such as schizophrenia.[2] Serotonin reuptake inhibitors (SRIs) and cognitive behavioral therapy (CBT) are first-line treatments for OCD. However, a substantial proportion of patients either do not achieve adequate symptom relief from SRIs or discontinue them due to side effects. Similarly, CBT can be inaccessible, intolerable, or ineffective for some individuals, leaving many patients without satisfactory treatment options.[3–5] The OCD pathophysiology is strongly linked to the dysfunction in the corticostriatopallidothalamocortical loop,[6–8] notably involving cortical regions such as the anterior cingulate cortex (ACC) and medial prefrontal cortex (mPFC).[9] Functional magnetic resonance imaging studies in OCD have reported both increased and decreased activation of the ACC, mPFC, and caudate nucleus, with increased activity during cognitive tasks and decreased activity at rest.[10,11]

Several brain stimulation techniques have been explored to target the neural structures implicated in OCD. Deep brain stimulation, involving surgically implanted electrodes stimulating regions such as the subthalamic nucleus or ventral striatum, has shown promising results in treatment-resistant OCD.[12,13] However, deep brain stimulation carries inherent surgical risks and practical challenges related to maintaining implanted devices. Traditional repetitive transcranial magnetic stimulation, although noninvasive, targets only superficial cortical regions and has demonstrated modest efficacy in OCD.[14,15] Its clinical utility is limited by the shallow penetration depth.[16] Deep transcranial magnetic stimulation (dTMS) addresses these limitations by using the H (Hesed) coil, whose unique winding structure enables stimulation of deeper brain regions, such as the dorsal ACC (dACC) and mPFC, by penetrating up to 5 cm beneath the skull.[17] Initial studies using high-frequency (HF; 20 Hz) dTMS to stimulate the dACC and mPFC with H7 coil have shown promising outcomes in OCD.[18,19] More recently, a postmarketing study evaluating real-world data further supported the efficacy of dTMS in alleviating OCD symptoms.[20] Additionally, dTMS has demonstrated effectiveness even in patients who have not responded to selective SRIs or CBT.[21]

However, the original multicenter randomized controlled trial by Carmi et al[19] was conducted by researchers, several of whom reported affiliations or relationships with the device manufacturer. The study sample was also limited to Western populations. Hence, there is a need to validate and generalize these findings through independently conducted studies across more diverse sociocultural settings, especially within the Asian context. The current randomized controlled trial aims to address these gaps by examining the dTMS efficacy in OCD patients in Indian population. Here we present the findings from a prospective, randomized, sham-controlled study assessing the outcomes of stimulating bilateral dACC and mPFC compared with sham stimulation. Change in OCD symptoms at the end of 2 weeks of dTMS treatment as measured on the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS) was the primary outcome variable.

MATERIALS AND METHODS

Subjects and Data Collection

The study was conducted at a tertiary care psychiatric hospital in Eastern India as a prospective, sham-controlled trial with purposive sampling. The trial was approved by the Institute Ethics

From the *Department of Psychiatry, National Institute of Mental Health and Neurosciences, Bengaluru; and †Department of Psychiatry, Central Institute of Psychiatry, Ranchi, Jharkhand, India.

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Reprints: Sachin Reddy , MD, Department of Psychiatry, NIMHANS, Bengaluru, India 560027 (e-mail: snsachinnandu@gmail.com).
Sachin Reddy ORCID: 0000-0002-7797-2663

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Committee and was registered with the Clinical Trials Registry of India (CTRI registration no. CTRI/2020/07/026407). The study was approved by the institutional ethics board, and all procedures adhered strictly to ethical standards for biomedical research. Data were collected from July 2020 to December 2021. Sample size was calculated a priori using G*Power software (Heinrich-Heine-Universität Düsseldorf, Düsseldorf, Germany) for repeated-measures analysis of variance model assuming 2 groups over 3 time points with modest effect size of 0.4 and significance level at 0.05 and power at 80%. Estimated sample was 18 in each group. Considering a conservative attrition rate of 10%, sample size was fixed at 20 in each group.

Patients were recruited for the study from the outpatient department of the hospital after their OCD diagnosis was confirmed by at least 1 qualified psychiatrist. Patients meeting the diagnostic criteria for OCD according to the *International Statistical Classification of Diseases, Tenth Revision Diagnostic Criteria for Research*, aged 18 to 60 years of either sex, with Y-BOCS scores of 15 or more and Hamilton Depression Rating Scale (HAM-D) scores of 22 or less were included in the study. We excluded from the study patients suffering from any neurological disorder or other psychiatric disorders including severe depressive disorder.

Clinical Procedure

A semistructured interview format was used to collect the clinical and sociodemographic data of the patients at baseline. All the patients signed a written, informed consent form during the recruitment process. Y-BOCS, Hamilton Anxiety Rating Scale (HAM-A) and HAM-D were applied, and the patients were assigned to receive daily sessions of active or sham dTMS 5 days a week for 2 consecutive weeks amounting to a total of 10 sessions. Patients were allocated into active or sham groups using a computer-generated randomization sequence. Block randomization with variable block sizes of 4 and 6 was used to ensure balanced allocation. The sequence was concealed using opaque, sealed, and sequentially numbered envelopes, which were opened only after participant enrollment to the study. Clinical rating scales were reapplied after completing 5 and 10 sessions of dTMS. Repetitive transcranial magnetic stimulation side effects checklist was applied after each dTMS session to monitor any adverse events. Clinical rating scales were applied by trained clinicians. Patients and the clinicians applying the rating scales were blinded to the type of stimulation delivered.

dTMS Procedure

We used the proprietary H7 dTMS coil (Brainsway, Israel) attached to the PowerMag EEG 100 stimulator (MAG & More GmbH, Germany) to deliver the treatment in our study. When fitted 4 cm anterior to the leg motor area, the H7 coil maximally stimulates bilateral dACC and mPFC. The H7 coil is nested inside a helmet connected to the TMS stimulator. The stimulator is designed to deliver various patterns of stimulation such as HF, low-frequency, and theta burst stimulation.

Measurement of Motor Threshold

The resting motor threshold is the minimum intensity of stimulation that generates visible twitch in the right foot in at least 3 of the 6 trials. The helmet with the H7 coil is fitted over the subject's head, around 9 cm superior to nasion. This approximately corresponds to the leg motor area in most people. Biphasic TMS pulses are delivered around this region to determine the area where maximal twitch can be obtained in the right foot. Resting motor threshold is then determined by gradually decreasing the stimulation intensity until the minimum intensity, which produces a visible twitch.

Active dTMS Treatment

The helmet is fitted 4 cm anterior to the leg motor area to deliver the dTMS treatment. Stimulation is delivered at 20-Hz frequency in 50 trains of 2-second duration with an intertrain interval of 20 seconds. Each session lasts for about 18 minutes and accounts for 2000 TMS pulses.

Sham dTMS

The helmet is fitted on patient's head, and the H7 coil is disconnected from the stimulator. A figure-of-8 coil is then connected to the stimulator and placed in contact with the helmet of the H7 coil. The stimulator is set up to deliver the same protocol described above at 50% to 60% intensity of the stimulator output. The patients receiving sham stimulation thus experience sound, vibration, and duration similar to that experienced during the active stimulation.

Drug Treatment

Treating psychiatrists decided the appropriate drug treatment protocols for each patient, but they were maintained on stable dose of medications for at least 2 weeks before starting the dTMS treatment and throughout the study period. However, most patients were on stable doses of medications for more than 2 months.

Outcome Measures

Change in Y-BOCS total score with dTMS treatment was considered as the primary outcome measure. The secondary outcome measures included the changes in HAM-A and HAM-D scores across the 3 time points of assessment and response rates as measured by Y-BOCS score. We considered ≥35% decrease in Y-BOCS score as the criterion for full response.[22]

Statistical Analysis

We included all the patients who were randomized and had received at least 1 session of dTMS in the intention-to-treat (ITT) dataset. Statistical analysis was performed using Statistical Package for Social Sciences version 28.00 (IBM Corp, Armonk, NY). Group differences for sample characteristics were examined with independent t test and χ^2 test wherever applicable. To compare the effect of treatment over time between the 2 groups, linear mixed-effects (LME) model analysis was used. Each clinical outcome variable was assessed on a separate mixed-effects model. In the repeated-measures LME model, a random intercept for each subject was included to account for individual differences in baseline values and to appropriately model the correlation between repeated measurements within subjects. Group, time, and group-time interaction were modeled as fixed effects in the LME model (using relevant covariates). Unstructured covariance type was used to model the repeated-measures data. Parameter estimation was conducted using restricted maximum likelihood, which provides unbiased estimates of variance components by accounting for the loss of degrees of freedom associated with estimating fixed effects. Effect sizes (Cohen f^2) were calculated for each outcome variable in the linear mixed-effects model, based on the F statistic for each fixed effect (main effect of group, main effect of time, and the group × time interaction). The method for calculating Cohen f^2 followed the approach described by Selya et al.[23] The response rates between the 2 groups were compared using sensitivity analysis approach. From the ITT sample, 5 different scenarios were assessed: per-protocol, worst case (all dropouts in the active group as nonresponders and in the sham group as responders), best case (all dropouts in the active group as responders and in the sham group as nonresponders), all dropouts as responders, and all

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dropouts as nonresponders. The response rates in each of these scenarios were compared between the groups using a χ^2 test.

RESULTS

Of the 80 patients with OCD that were assessed for eligibility, 49 patients fulfilling the inclusion and exclusion criteria were recruited to the study: 24 in the active group and 25 in the sham group. Intention-to-treat sample was defined as any patient who received at least 1 dTMS session. Two patients in the sham group and 1 patient in the active group dropped out before receiving any treatment due to conflict in schedule. Thus, the ITT sample included 46 patients (Fig. 1).

Baseline Characteristics

Baseline clinical and sociodemographic characteristics of the sample are presented in Table 1. The 2 groups did not differ significantly on any sociodemographic variables. However, HAM-A score showed a trend level significance at baseline between the groups. Hence, this was used as a covariate in the LME model for the primary outcome measure, Y-BOCS. There was a nearly equal distribution of sex among the participants. Majority were urban dwellers, employed, and married. All of the participants were currently on SRI treatment, and they all had at least 1 adequate trial of an SRI,[24] and most of them were on a stable dose of current SRI for more than 2 months.

Primary Outcome Measures

At end-point analysis after 2 weeks, there was a significant decrease in total Y-BOCS scores in both active (−10.4 points, standard error [SE] = 0.97) and sham (2.6 points, SE = 0.97) groups (Table 2). However, the difference between the 2 groups across time as assessed by the group × time interaction was significant

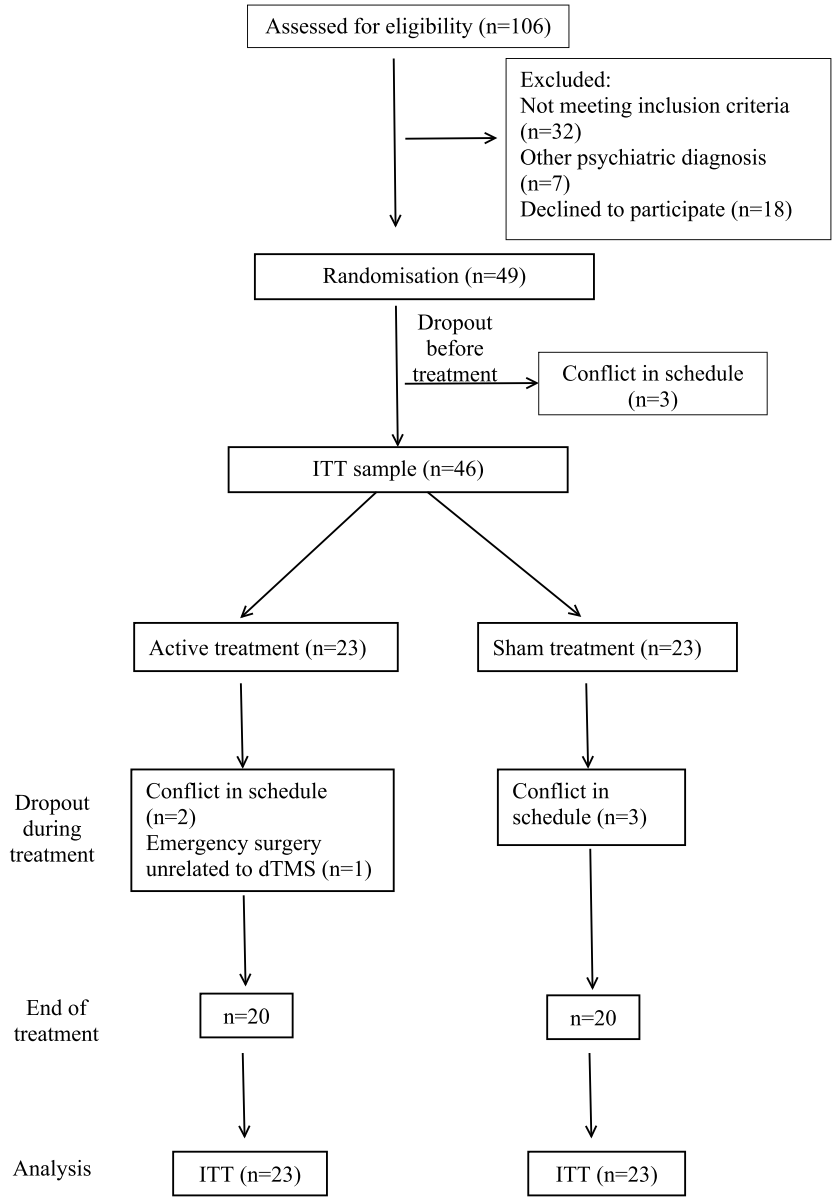

FIGURE 1. Flowchart of the study according to the CONSORT statement. CONSORT, Consolidated Standards of Reporting Trials.

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TABLE 1. Comparison of two Groups on Various Sociodemographic, Clinical, and Treatment Variables

Variables	Active (n = 23) Mean (SD)/n (%)	Sham (n = 23) Mean (SD)/n (%)	t/χ^2	P/Fisher Exact Test
Age, y	31.17 (9.98)	32.57 (8.82)	−0.501	0.619
Education, y	13.35 (2.60)	11.78 (2.79)	1.965	0.056
Sex (female)	10 (43%)	13 (57%)	0.783	0.556
Habitat (urban)	14 (61%)	13 (57%)	0.368	0.824
Family history of psychiatric illness (present)	5 (22%)	4 (17%)	0.138	1.000
Occupation (unemployed)	11 (48%)	8 (35%)	0.867	0.369
Marital status (unmarried)	11 (48%)	8 (35%)	0.807	0.550
Age at illness onset, y	22.83 (9.15)	24.13 (7.03)	−0.542	0.591
Duration of illness, y	8.35 (7.07)	8.43 (5.11)	−0.048	0.962
Duration of prior SRI treatment, y	3.82 (3.25)	3.13 (1.81)	0.895	0.376
Duration of current SRI trial, mo	3.16 (1.51)	3.54 (2.12)	−0.699	0.488
Lifetime SRI trials	1.22 (0.6)	1.39 (0.58)	−0.997	0.324
Y-BOCS score	27.52 (3.14)	27.09 (3.09)	0.473	0.639
HAM-D score	14.17 (4.62)	12.26 (3.06)	1.653	0.105
HAM-A score	19.22 (4.55)	16.57 (4.30)	2.030	**0.048**

Data in bold indicate a significant difference between two groups.

SRI, Serotonin Reuptake Inhibitor; YBOCS, Yales Brown Obsessive Compulsive Scale; HAM-D, Hamilton Depression Rating Scale; HAM-A, Hamilton Anxiety Rating Scale.

(7.80 points, 95% confidence interval [CI] = 5.52–10.07, $P < 0.001$) with an effect size of 1.39 (Fig. 2).

Secondary Outcome Measures

The groups differed significantly on the decrease in depression and anxiety severity across time (Table 2). The mean difference between the groups was significant for both HAM-D scores (4.15 points, 95% CI = 2.76–5.14, $P < 0.001$, Cohen $f^2 = 0.98$), as well as HAM-A scores (6.65 points, 95% CI = 4.91–8.39, $P < 0.001$, Cohen $f^2 = 1.81$).

The sensitivity analysis for response rates revealed significantly better outcomes under all 5 scenarios we modeled (Table 3). In the per-protocol analysis, 15 of 20 patients (75%) in the active group met the response criteria, compared with only 1 of 20 patients (5%) in the sham group ($P < 0.001$, χ^2 test) (Fig. 3).

Safety Assessment

Most patients from both groups tolerated the treatment well with no major/serious adverse events. Among completers, 9 patients in the active group (45%) and 7 patients in the sham group (35%) reported minor side effects with the treatment, which was not significantly different ($P = 0.556$, χ^2 test). Most patients typically complained of self-limiting headache, and 2 patients in the active group reported nighttime sleep disturbance, which resolved after the initial 2 to 3 sessions. A patient reported increase in anxiety at the very first session of dTMS, which resolved shortly after

TABLE 2. Comparison of two Groups on Clinical Rating Scales Using Linear Mixed Effects (LME) Models for Intent-to-Treat Analysis

		Predicted Mean Estimates (SE)			Main Effect of Group, Main Effect of Time and Group × Time Interaction		
Variables		Active dTMS Group	Sham dTMS Group	Mean Difference (95% CI)	*F*	*P*	Cohen f^2
Y-BOCS total score	Baseline	27.18 (0.59)	27.32 (0.59)	0.435 (−1.418 to 2.287)	21.22	**<0.001**	0.56
	After 5 sessions	22.28 (0.61)	25.62 (0.61)	−1.952 (−3.855 to −0.049)	68.11	**<0.001**	3.59
	After 10 sessions	16.78 (0.79)	24.72 (0.79)	−6.800 (−9.143 to −4.457)	26.36	**<0.001**	1.39
	Pre-post	10.40 (0.97)	2.60 (0.55)	7.80 (5.52–10.07)			
HAM-D total score	Baseline	14.17 (0.82)	12.26 (0.82)	1.913 (−0.419–4.245)	0.009	0.926	0.0002
	After 5 sessions	11.26 (0.70)	11.34 (0.70)	0.286 (−1.789–2.361)	75.94	**<0.001**	3.63
	After 10 sessions	8.28 (0.64)	10.39 (0.64)	−1.750 (−3.735–0.235)	20.40	**<0.001**	0.98
	Pre-post	5.95 (0.61)	1.80 (0.29)	4.15 (2.76–5.14)			
HAM-A total score	Baseline	19.22 (0.92)	16.57 (0.92)	2.652 (0.019–5.285)	0.053	0.819	0.0012
	After 5 sessions	15.11 (0.85)	14.86 (0.85)	0.810 (−1.716–3.335)	91.64	**<0.001**	4.67
	After 10 sessions	10.42 (0.84)	14.13 (0.84)	−3.250 (−5.753 to −0.747)	35.39	**<0.001**	1.81
	Pre-post	9.10 (0.74)	2.45 (0.43)	6.65 (4.91–8.39)			

Data in bold indicate a significant difference between two groups.

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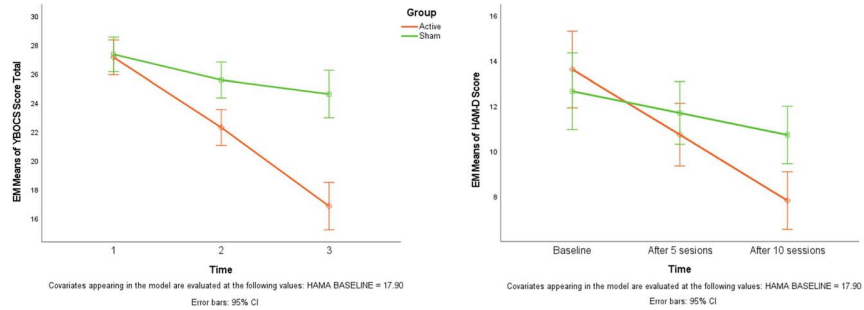

FIGURE 2. Estimated marginal means (EM means) of Y-BOCS (left) and HAM-D (right) scores across 3 time points for active and sham dTMS groups.

the session. None of these were serious enough to warrant discontinuation of dTMS treatment.

Three patients in each group (13%) dropped out primarily due to conflicting schedules and change of plans. A patient from active dTMS group dropped out as she had to undergo an emergency eye surgery due to blunt trauma. None of the dropouts in either group were due to side effects of the treatment.

DISCUSSION

In our study, there was a significant improvement in Y-BOCS scores in the active group compared with the sham group after 10 sessions of dTMS targeting bilateral dACC and mPFC. On average, the active group showed a 10.4-point improvement on the Y-BOCS scale (SE = 0.97), whereas the sham group only showed a 2.6-point improvement (SE = 0.55). Additionally, 75% of the active group achieved a full response, compared with only 5% in the sham group (Fig. 3). The sensitivity analysis for the ITT sample further demonstrated that these response rates remained significant across all scenarios modeled (Table 3).

In the previous multicenter dTMS-OCD trial, Carmi et al reported a 6-point improvement for the active group and a 3.3-point improvement for the sham group after 29 sessions of dTMS.[19] Similarly, in the first dTMS-OCD pilot trial, 43.75% of patients in the HF active group achieved a response compared with 7.14% in the sham group.[18] Moreover, a postmarketing trial that examined real-world efficacy data found that patients showed their first response after an average of 18.5 sessions, whereas sustained response was observed after 20.5 sessions. By the 10th session, the patients in that study showed an 18.8% reduction in baseline Y-BOCS scores.[20] Compared with these studies, our findings demonstrate better outcomes in terms of percentage improvement in Y-BOCS scores, full-response rates, and the number of sessions required to achieve a first response.

A possible explanation for the superior efficacy in our study may be the difference in the sample population. In the previous studies, the sample consisted of OCD patients who had previously failed to respond to at least 1 adequate trial of SRIs or CBT. In fact, most patients had failed 3 or more SRI trials and/or CBT, classifying them as treatment-resistant.[21] In contrast, only 5 patients in the active group and 7 in the sham group had undergone 2 or more adequate SRI trials. On average, our participants had completed 1.3 adequate SRI trials (SD = 0.59), which was not significantly different between the groups. Furthermore, the average illness duration was 8.4 (SD = 6.1) years, but the duration of prior treatment was only 3.5 (SD = 2.6) years. Thus, our sample, although chronically ill, was not treatment-resistant, with relatively shorter durations of prior pharmacological treatment. Together, these factors may account for the superior efficacy of dTMS observed in this population relative to previous studies.

These findings suggest that dTMS may be an effective intervention for a broader range of OCD patients, potentially including those who are not yet treatment-resistant as suggested by a recent meta-analysis.[25] This may especially benefit more severely ill patients who require faster symptom alleviation.

Our study demonstrated significant improvements in both anxiety and depression symptoms in the active dTMS group compared with the sham group. These findings are consistent with prior studies showing that OCD patients often exhibit high anxiety levels,[26] and reductions in Y-BOCS scores are linked with anxiety improvements.[27] Additionally, HF dTMS has been shown to alleviate anxiety in major depressive disorder[28] and reduce depressive symptoms across both OCD and major depressive disorder populations.[29] The improvement in depressive symptoms in our study may therefore be independent of OCD symptom reduction, potentially involving distinct mechanisms.

The dACC and mPFC, the primary targets of dTMS in this study, are crucial to OCD treatment. Although HF stimulation of

TABLE 3. Comparison Between Active and Sham Groups on Response Rates in Various Scenarios (Intent-to-Treat Analysis)

Scenario	Active Group (Responders/n) (%)	Sham Group (Responders/n) (%)	χ^2	P	Risk Ratio (95% CI)
Per-protocol (completers)	15/20 (75%)	1/20 (5%)	20.41	**<0.001**	15.00 (2.18–103.03)
Worst case (all active dropouts as nonresponders, sham dropouts as responders)	15/23 (65.2%)	4/23 (17.4%)	10.85	**0.002**	3.75 (1.49–9.59)
Best case (all active dropouts as responders, sham dropouts as nonresponders)	18/23 (78.2%)	1/23 (4.3%)	25.94	**<0.001**	18.00 (2.61–123.88)
All dropouts as responders in both groups	18/23 (78.2%)	4/23 (17.4%)	17.07	**<0.001**	4.50 (1.80–11.25)
All dropouts as nonresponders in both groups	15/23 (65.2%)	1/23 (4.3%)	18.78	**<0.001**	15.00 (2.15–104.38)

Data in bold indicate a significant difference between two groups.

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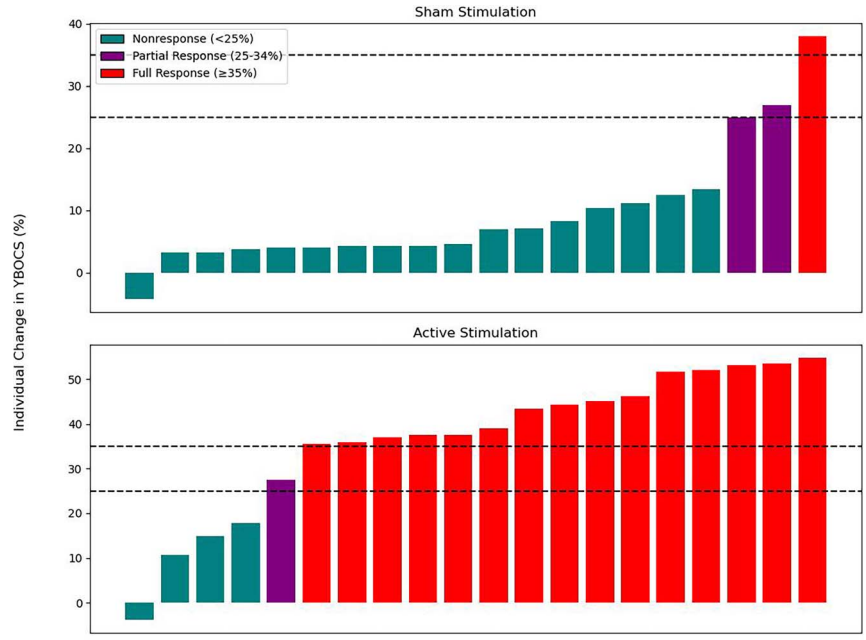

FIGURE 3. Distribution of responders and nonresponders based on Y-BOCS score at the end of study in the active and sham groups. ≥35% Reduction in Y-BOCS score was considered as full response, and ≥25% reduction as partial response.

these already hyperactive regions might seem counterintuitive, it is possible that the hyperactivity represents a compensatory mechanism aimed at controlling obsessive-compulsive phenomena. By modulating the function of these regions through HF stimulation, dTMS may help restore a more functional response to OCD symptoms. Moreover, the mPFC seems to play a crucial role in extinction learning,[30] a fundamental mechanism in reducing obsessive-compulsive behaviors.

Deep transcranial magnetic stimulation was generally well tolerated in both groups. The dropout rates in our study are in line with those of the previous studies.[18–20] Notably, none of the patients cited side effects of dTMS as a reason for discontinuing the study.

Affective state dependency in noninvasive neurostimulation has been studied widely, and the learnings from this have been applied in TMS research, especially in disorders such as OCD, posttraumatic stress disorder, and addiction.[31–33] However, unlike the previous multicenter trial,[19] we did not use individualized symptom provocation for the patients before each dTMS session. Despite this, our patients experienced the benefit with dTMS. This warrants a further systematic study of the effect of various mental states on the outcomes of neuromodulation treatments in clinical populations, especially in OCD.

Limitations

We acknowledge several limitations in this study. First, the sham procedure used in this study differed from that employed in previous dTMS trials, which resulted in operators not being blinded to the procedure. Furthermore, we did not assess the efficacy of blinding, leaving open the possibility of bias affecting the outcomes. Additionally, patients in our study received fewer dTMS sessions compared with the 25 to 30 sessions delivered in prior studies and the 29 sessions recommended by the Food and Drug Administration–approved protocol. This is due to the logistical limitations in our study. This premature termination of treatment may have impacted the full potential efficacy of dTMS in our population.

Another limitation is the absence of follow-up assessments, which prevents us from determining the long-term maintenance effects of the treatment. Although the population in this study was not treatment-resistant, it remains unclear whether the observed effects would be sustained in patients with more severe or long-standing treatment histories. Future studies with more treatment sessions, head-to-head comparison between different stimulation protocols, and follow-up assessments are needed to address these gaps.

CONCLUSIONS

Our study demonstrated that dTMS targeting the dACC and mPFC significantly improved obsessive-compulsive, anxiety, and depressive symptoms, with faster response rates and fewer sessions compared with previous trials, particularly in a non–treatment-resistant population. These findings suggest that dTMS may be an effective early intervention for a broader range of OCD patients, potentially shifting its use earlier in the treatment pathway. Future research should focus on identifying biomarkers of response and nonresponse to dTMS, as well as exploring the potential benefits of combining dTMS with psychotherapy to enhance treatment outcomes.

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