

Effectiveness of an Integrated Cognitive Control–Metacognitive Intervention on Obsessive–Compulsive Symptoms: The Roles of Executive Function, Inhibitory Control, Metacognition, and Neurophysiological Indices

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This study aimed to evaluate the effectiveness of an integrated cognitive control–metacognitive intervention (ICCM) for reducing obsessive–compulsive symptoms and improving executive function, inhibitory control, metacognitive beliefs, and neurophysiological indices in adults with obsessive–compulsive disorder (OCD). An assessor-blinded, parallel-group randomized controlled trial was conducted with 80 adults aged 18–55 years who met DSM-5-TR criteria for OCD and had Yale–Brown Obsessive–Compulsive Scale (Y-BOCS) scores ≥ 16 . Participants were randomly assigned to ICCM or a wait-list control group in a 1:1 ratio. The intervention comprised eight weekly 120-minute group sessions integrating metacognitive therapy, attention regulation, goal-management strategies, response-inhibition training, and adaptive error monitoring. Assessments were conducted at baseline, post-treatment, and 8-week follow-up; EEG/ERP measures were obtained at baseline and post-treatment. Outcomes included Y-BOCS, Metacognitions Questionnaire-30, an executive-function composite, stop-signal reaction time (SSRT), Stroop interference, and NoGo-N2, NoGo-P3, ERN, and Pe amplitudes. Generalized estimating equations, baseline-adjusted EEG models, Hedges' g , and exploratory bootstrap mediation analyses were used. Significant group $\times$ time effects favored ICCM for Y-BOCS at post-treatment ($B = -8.92$, $SE = 0.47$, $p < .001$) and follow-up ($B = -10.16$, $SE = 0.53$, $p < .001$). Significant improvements were also observed for MCQ-30, executive functioning, SSRT, and Stroop interference (all $p < .001$). Baseline-adjusted EEG analyses showed significant effects for NoGo-N2 ($+1.14 \mu V$), NoGo-P3 ($+1.43 \mu V$), and Pe ($+0.57 \mu V$), all $p < .001$, whereas ERN change was nonsignificant ($p = .062$). Mediation analyses identified significant indirect effects through metacognitive belief change (0.53 , 95% CI $[0.16, 0.98]$) and SSRT improvement (0.41 , 95% CI $[0.19, 0.69]$). ICCM produced substantial and sustained reductions in OCD symptoms while improving metacognitive, executive, inhibitory, and selected neurophysiological processes, with metacognitive change and response inhibition emerging as particularly important candidate mechanisms of therapeutic improvement.

Keywords: obsessive–compulsive disorder; metacognitive therapy; cognitive control; executive function; response inhibition; EEG; event-related potentials

1. Introduction

Obsessive-compulsive disorder (OCD) is a chronic and often disabling psychiatric condition characterized by the presence of recurrent, intrusive, and unwanted thoughts, images, or urges and by repetitive behaviors or mental acts performed in response to these experiences or according to rigidly applied rules. Although obsessions and compulsions vary considerably in content, they commonly involve contamination, checking, symmetry, responsibility, harm, taboo thoughts, and the need for certainty or completeness. According to the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition, Text Revision, these symptoms are typically time-consuming, cause clinically significant distress, and interfere with social, occupational, academic, or other important areas of functioning (American Psychiatric, 2022). The clinical burden of OCD is intensified by its frequently persistent course, heterogeneous symptom profile, high levels of functional impairment, and tendency to coexist with depressive, anxiety-related, and other psychiatric symptoms. Accordingly, contemporary research has increasingly moved beyond a purely symptom-descriptive understanding of OCD toward identifying the cognitive, metacognitive, executive, and neurophysiological mechanisms that contribute to symptom persistence and may serve as modifiable therapeutic targets.

A substantial body of evidence indicates that cognitive-control dysfunction represents one important dimension of OCD. Rather than being restricted to distressing beliefs or maladaptive interpretations, OCD is associated with difficulties in the regulation of goal-directed behavior, inhibition of prepotent responses, flexible shifting between mental sets, planning, working toward superordinate goals, and disengaging from repetitive response patterns. A major meta-analysis of neuropsychological functioning in adults with OCD demonstrated broad but variable impairments across several cognitive domains, particularly executive functions, memory-related processes, and processing efficiency (Abramovitch et al., 2013). These deficits should not be interpreted simply as generalized intellectual impairment; rather, they appear to reflect disruptions in specific control processes that may become especially consequential when an individual attempts to resist compulsions, terminate checking, shift away from obsessional doubt, or maintain goal-directed behavior in the face of intrusive internal signals. Evidence from Iranian clinical populations similarly suggests that individuals with OCD exhibit difficulties in executive functioning, selective

attention, and information processing, indicating that these cognitive-control problems are not limited to Western samples and may be relevant across cultural contexts (Yazdi-Ravandi et al., 2018).

Among executive-control processes, response inhibition has received particular attention because compulsive behavior can be conceptualized partly as a failure to suppress actions that are no longer adaptive or consistent with higher-order goals. Response inhibition refers to the capacity to stop, cancel, or withhold a response when circumstances require behavioral control. The stop-signal paradigm has become one of the most widely used experimental methods for examining this function, with stop-signal reaction time providing an estimate of the latent speed of the inhibitory process (Verbruggen & Logan, 2008). In OCD, impaired response inhibition may manifest clinically as difficulty interrupting checking, washing, repeating, reassurance seeking, mental neutralization, or other habitual sequences despite awareness that these actions are excessive or unnecessary. A meta-analysis focused specifically on the stop-signal task found evidence of impaired response inhibition in individuals with OCD, supporting the hypothesis that inefficient inhibitory control is a reproducible cognitive feature of the disorder (Mar et al., 2022). At the neural level, meta-analytic evidence from task-based functional neuroimaging studies further indicates altered activation in brain systems implicated in inhibitory control among individuals with OCD, reinforcing the view that impaired response suppression has a neurobiological as well as behavioral basis (Li et al., 2024).

Executive difficulties in OCD are, however, unlikely to be adequately captured by inhibitory control alone. Many patients report becoming absorbed in repetitive routines, losing sight of broader goals, becoming trapped in detail-focused monitoring, and repeatedly rechecking actions that have already been completed. Such patterns point toward difficulties in maintaining goal hierarchies, monitoring ongoing behavior efficiently, and switching from habitual or stimulus-driven actions to consciously selected goals. Goal Management Training provides a useful cognitive-rehabilitation framework for addressing these higher-order executive processes. Originally developed for individuals with executive dysfunction following frontal-lobe injury, Goal Management Training emphasizes interruption of automatic behavior, periodic stopping, conscious reactivation of goals, decomposition of complex activities into manageable subgoals, monitoring of task performance, and flexible correction when behavior deviates from

intention (Levine et al., 2011). Although developed outside the OCD context, these strategies are conceptually relevant to compulsivity because compulsive actions frequently involve automaticity, goal neglect, repeated checking, and difficulty returning to task-relevant behavior once attention has been captured by uncertainty or perceived error.

The potential relevance of cognitive rehabilitation to OCD has increasingly been recognized. A systematic review of cognitive-training studies in OCD found that interventions targeting cognitive processes may produce measurable improvement in selected neuropsychological functions and, in some studies, symptom-related outcomes, although the literature remains heterogeneous in training methods, outcome selection, and methodological quality (Bhattacharya et al., 2024). This emerging evidence supports the possibility that directly strengthening cognitive-control processes may complement conventional psychological treatments. At the same time, cognitive training in isolation may have limited transfer if improvements achieved in structured tasks are not connected to the metacognitive beliefs and symptom-related interpretations that drive repetitive behavior in daily life. A patient may improve on a laboratory inhibition task while continuing to believe that intrusive thoughts signal danger, that uncertainty is intolerable, or that a ritual must be completed until an internally defined stopping criterion is reached. This limitation highlights the need to integrate cognitive-control training with interventions that directly modify the beliefs governing engagement with intrusive experiences.

The metacognitive model of OCD provides a particularly relevant framework for this purpose. From a metacognitive perspective, intrusive thoughts are not considered pathological simply because they occur. Rather, distress and compulsive responding arise when individuals interpret intrusive thoughts according to dysfunctional beliefs about the importance, power, danger, controllability, or required management of mental events. Early experimental work demonstrated that modifying metacognitive beliefs relevant to OCD can produce changes in obsessive-compulsive responding, providing support for a causal role of these beliefs rather than treating them merely as correlates of symptom severity (Fisher & Wells, 2005). The model emphasizes beliefs such as thought-action fusion, thought-event fusion, the perceived necessity of rituals, beliefs that particular cognitive or behavioral acts prevent feared outcomes, and rigid internal criteria regarding when it is safe or appropriate to stop a ritual. Empirical testing of this framework has shown that fusion beliefs, beliefs about

rituals, and internal stop signals are closely associated with obsessive-compulsive symptoms and may help explain why compulsive behaviors persist even after an individual recognizes their irrational or excessive character (Myers et al., 2009).

The broader construct of metacognition also encompasses beliefs about uncontrollability, danger, cognitive confidence, the need to control thoughts, and heightened self-focused cognitive monitoring. These dimensions may amplify the significance assigned to intrusive thoughts and increase attempts to monitor, suppress, neutralize, or correct internal experience. Research using the Metacognitions Questionnaire has demonstrated that maladaptive metacognitive beliefs are associated with emotional distress and anxiety-related vulnerability, including within Iranian samples (Bahrami & Yousefi, 2011). More recent Iranian work has likewise indicated that psychological interventions capable of changing maladaptive cognitive schemas can influence metacognitive beliefs among individuals with obsessive-compulsive symptoms, underscoring the potential modifiability of these processes (Tajizadegan & Sharifi Isfahani, 2026). Within OCD specifically, reducing beliefs about the danger or significance of thoughts may decrease the perceived need to engage in compulsions, whereas enhancing attentional flexibility and detached responding may reduce perseverative self-focused monitoring.

Metacognitive therapy has consequently emerged as a promising treatment approach for OCD. In an early clinical case series, metacognitive therapy was associated with substantial reductions in obsessive-compulsive symptoms, providing preliminary support for interventions that directly target metacognitive beliefs and maladaptive modes of responding to intrusive cognitions (Fisher & Wells, 2008). Subsequent controlled research has strengthened this evidence. A pilot randomized trial comparing metacognitive therapy with exposure and response prevention showed that metacognitive treatment was feasible and clinically promising for individuals with OCD (Glombiewski et al., 2021). More recently, a non-inferiority randomized controlled trial directly comparing metacognitive therapy with exposure and response prevention provided further evidence that metacognitive therapy can produce substantial therapeutic effects and may represent a credible alternative or complementary approach within OCD treatment (Exner et al., 2024). These findings are important because exposure and response prevention remains one of the most established psychological treatments for OCD, yet not all patients respond adequately, tolerate exposure-based procedures, or

maintain optimal gains. Identifying interventions that address alternative mechanisms of symptom persistence therefore has both theoretical and clinical significance.

Despite this progress, metacognitive and executive-control approaches have often developed in parallel rather than as components of a unified therapeutic model. This separation may be theoretically limiting because metacognitive dysfunction and cognitive-control deficits are likely to interact. A person who believes that an intrusive thought is dangerous or meaningful may allocate excessive attention to it, repeatedly monitor internal states, and experience increased difficulty inhibiting a ritualized response. Conversely, weak inhibitory control, reduced cognitive flexibility, or inefficient goal maintenance may make it more difficult to apply adaptive metacognitive strategies even when the person intellectually understands them. From this perspective, symptom persistence may emerge from an interaction between dysfunctional beliefs about thoughts and compromised mechanisms for regulating attention, suppressing habitual responses, monitoring behavior, and returning to goal-directed action. An intervention that addresses only one level may therefore leave another important maintenance mechanism insufficiently treated.

An integrated approach combining cognitive remediation and metacognitive therapy has recently been proposed specifically for adults with OCD. Zargarinejad and colleagues developed a randomized controlled trial protocol in Tehran integrating cognitive remediation with metacognitive therapy to improve cognitive functioning while reducing symptom severity (Zargarinejad et al., 2025). This framework is particularly relevant because it recognizes that cognitive impairment and maladaptive metacognitive processing may be mutually reinforcing. However, the mechanistic architecture of such integrated interventions remains insufficiently established. It remains necessary to determine not only whether a combined intervention reduces obsessive-compulsive symptoms, but also whether it changes the specific cognitive-control and metacognitive processes it is designed to target and whether these changes statistically account for clinical improvement. Measuring executive function, response inhibition, and maladaptive metacognitive beliefs concurrently can therefore provide a more informative test of the intervention model than symptom ratings alone.

Neurophysiological measures offer an additional level of mechanistic analysis. Event-related potentials derived from electroencephalography provide temporally precise

indicators of cognitive-control operations that may not be fully captured by behavioral performance. Inhibitory-control tasks commonly elicit the NoGo-N2 and NoGo-P3 components, which are associated with conflict detection, recruitment of inhibitory processes, and successful suppression of prepotent responses. Error-processing tasks also elicit the error-related negativity and error positivity, which index rapid error detection and later conscious evaluation of errors, respectively. A growing electrophysiological literature suggests that these processes are altered in OCD. A contemporary systematic and mechanistic review of EEG, ERP, and quantitative EEG evidence concluded that abnormalities in components related to inhibitory control, conflict processing, and performance monitoring are among the most consistent neurophysiological findings in OCD (Chmiel & Kładna, 2026). Such measures are particularly valuable in intervention studies because they can help determine whether symptom improvement is accompanied by changes in the neural processes that support response inhibition and adaptive monitoring.

The error-related negativity is especially relevant to OCD because excessive error monitoring has long been proposed as a neurocognitive feature of the disorder. A review and meta-analysis of ERN findings characterized enhanced error-related negativity as a potential endophenotypic marker of OCD and highlighted its relative stability across symptom states and contexts (Riesel, 2019). This possibility has important implications for treatment research. If ERN reflects a relatively trait-like vulnerability marker, symptom improvement may occur without substantial normalization of ERN amplitude. In contrast, other components, such as NoGo-N2, NoGo-P3, or Pe, may be more sensitive to intervention-related changes in inhibitory recruitment or conscious post-error processing. Examining several ERP components within the same trial can therefore help distinguish potentially modifiable state-related mechanisms from more stable neurophysiological characteristics.

The integration of clinical, behavioral, metacognitive, executive, and electrophysiological outcomes is also important for addressing the persistent gap between efficacy research and mechanism research. Demonstrating that a treatment reduces Y-BOCS scores provides evidence of clinical benefit but does not explain how improvement occurs. Conversely, improvement on a cognitive task or ERP component does not establish that the change is clinically meaningful. A mechanistically informative trial should therefore examine whether symptom reduction co-occurs

with improvements in executive functioning, response inhibition, and metacognitive beliefs and whether changes in these domains statistically mediate treatment response. This approach is particularly valuable for an intervention explicitly constructed around cognitive control and metacognitive regulation because it allows the theoretical targets of treatment to be evaluated directly rather than inferred from symptom change alone.

Accordingly, an integrated cognitive control–metacognitive intervention may offer several complementary therapeutic pathways. Metacognitive procedures may weaken beliefs concerning the dangerousness, importance, or uncontrollability of intrusive thoughts and reduce ritualized attempts to neutralize them; attention-training procedures may improve flexible allocation of attention; goal-management strategies may reduce automatic responding and goal neglect; inhibitory-control exercises may strengthen the ability to withhold compulsive responses; and performance-monitoring strategies may help patients respond to errors without excessive checking or corrective repetition. At the same time, EEG/ERP measures can provide objective indices of whether these behavioral and cognitive changes are accompanied by altered recruitment of inhibitory-control and error-monitoring systems. Evaluating these domains together may therefore provide a more comprehensive account of treatment response than either purely symptom-based or purely neurocognitive approaches.

The aim of the present study was to evaluate the effectiveness of an integrated cognitive control–metacognitive intervention for reducing obsessive–compulsive symptoms and improving executive function, inhibitory control, maladaptive metacognitive beliefs, and neurophysiological indices of cognitive control in adults with OCD, while also exploring whether changes in these proposed mechanisms statistically mediated symptom improvement.

2. Methods and Materials

2.1. Study Design and Participants

This study was designed as an assessor-blinded, parallel-group randomized controlled trial to evaluate the effectiveness of an integrated cognitive control–metacognitive intervention for reducing obsessive–compulsive symptoms and improving executive functioning, inhibitory control, maladaptive metacognitive beliefs, and neurophysiological indices of cognitive control in adults

with obsessive–compulsive disorder (OCD). Participants were randomly assigned in a 1:1 ratio to either the integrated cognitive control–metacognitive intervention group or a wait-list control condition receiving stable treatment as usual. Assessments were conducted at three time points: baseline before randomization, immediately after completion of the 8-week intervention, and 8 weeks after the end of treatment. Neurophysiological assessment using electroencephalography and event-related potentials was conducted at baseline and immediately after treatment because the electrophysiological component of the study was intended primarily to examine proximal changes in neural mechanisms of response inhibition, interference control, and performance monitoring. Recruitment and treatment were conducted through outpatient psychiatric and psychological services in Tehran, Iran, including Iran Psychiatry Hospital and the Brain and Cognition Clinic. The study protocol was structured in accordance with the principles of randomized clinical trial methodology, including concealed allocation, blinded outcome assessment, standardized intervention delivery, and systematic monitoring of treatment adherence. Adults aged 18–55 years who met DSM-5-TR diagnostic criteria for a primary diagnosis of OCD were considered eligible. A total of 80 participants were recruited, with 40 participants allocated to each study condition. Diagnosis was established through a structured clinical interview conducted by a trained clinical psychologist and, when clinically indicated, confirmed by a psychiatrist. Inclusion criteria consisted of a Yale–Brown Obsessive–Compulsive Scale score of at least 16, indicating at least moderate symptom severity, sufficient proficiency in Persian to participate in assessment and treatment procedures, ability to complete computerized cognitive tasks, and provision of written informed consent. Participants receiving serotonin reuptake inhibitors or other established pharmacological treatments for OCD were required to have maintained a stable medication dose for at least 8 weeks before baseline assessment and to agree, in consultation with their treating physician, to avoid changes in medication during the active treatment phase whenever clinically feasible. Exclusion criteria included current psychotic disorder, bipolar disorder, a severe current major depressive episode requiring immediate modification of treatment, active suicidal risk, a current substance use disorder of moderate or greater severity, epilepsy, major neurological disease, a history of moderate or severe traumatic brain injury, intellectual disability that prevented completion of study procedures, and uncorrected visual or

auditory impairments that interfered with computerized cognitive assessment. Individuals initiating another structured psychotherapy specifically targeting OCD during the trial were also excluded. Additional criteria for inclusion in EEG analyses required technically adequate recordings and the availability of the minimum number of artifact-free trials specified for each ERP component. Following completion of baseline assessments, eligible participants were randomized using concealed permuted blocks. Randomization was stratified according to baseline OCD severity, categorized as a Y-BOCS score of 16–23 versus 24 or higher, and according to stable medication status to reduce the possibility of clinically meaningful baseline imbalance. The allocation sequence was generated by a researcher who was not involved in treatment delivery or outcome assessment. Clinical assessors remained blind to allocation throughout the study, and participants were instructed not to disclose their treatment assignment during follow-up assessments. EEG preprocessing files and statistical datasets were coded with anonymized group labels until the principal preprocessing and scoring decisions had been finalized. Post-treatment assessment was performed within 7 days after the final intervention session, whereas follow-up clinical and cognitive assessment was conducted 8 weeks after completion of treatment. Participants who attended at least six of the eight intervention sessions were categorized as treatment completers; however, the primary analyses retained all available observations from randomized participants according to an intention-to-treat-oriented analytical framework.

2.2. Data Collection Tools

The Yale–Brown Obsessive–Compulsive Scale was used as the principal clinical measure of obsessive–compulsive symptom severity. The Y-BOCS is a clinician-administered instrument consisting of 10 severity items, five assessing obsessions and five assessing compulsions. Each item is rated from 0 to 4, producing a total score ranging from 0 to 40, with higher scores representing greater symptom severity. The scale assesses dimensions such as time occupied by obsessive thoughts and compulsive behaviors, interference with functioning, subjective distress, resistance, and perceived control and was developed to provide an index of OCD severity that is relatively independent of the particular content of obsessions or compulsions. The instrument has demonstrated strong interrater reliability, internal consistency, sensitivity to therapeutic change, and

convergent validity in clinical OCD research. The Persian version has also demonstrated satisfactory psychometric characteristics in Iranian clinical populations, including strong internal consistency and test–retest reliability. The total Y-BOCS score was defined as the primary clinical outcome of the study. In addition to continuous symptom change, clinical response was operationalized as a reduction of at least 35% from the baseline Y-BOCS score, whereas remission was defined as a post-treatment or follow-up Y-BOCS score of 12 or lower.

Maladaptive metacognitive beliefs were assessed using the 30-item Metacognitions Questionnaire. The MCQ-30 evaluates five theoretically distinct but related dimensions of metacognition: positive beliefs about worry and perseverative thinking, negative beliefs concerning the uncontrollability and dangerousness of thoughts, cognitive confidence, beliefs concerning the need to control thoughts, and cognitive self-consciousness. Items are rated on a 4-point Likert-type response scale, and higher total and subscale scores indicate stronger endorsement of dysfunctional metacognitive beliefs. The measure was selected because the metacognitive model proposes that persistent obsessive–compulsive symptoms are influenced not merely by the occurrence of intrusive cognitions but by beliefs regarding the significance, controllability, dangerousness, and required management of those cognitions. The MCQ-30 has demonstrated a stable five-factor structure, satisfactory internal consistency, convergent validity, and acceptable temporal stability in international research, and Persian-language applications have also reported adequate-to-strong reliability for the total scale and constituent domains. The total score was used as the primary metacognitive outcome, while subscale scores were examined in supplementary analyses to determine whether treatment effects were particularly pronounced for beliefs concerning uncontrollability, cognitive confidence, and the need to control thoughts.

Executive functioning was assessed through complementary neuropsychological measures emphasizing planning, rule maintenance, cognitive flexibility, and goal-directed problem solving. Planning ability was evaluated using the Tower of London task, in which participants were required to reproduce goal configurations through a limited sequence of moves while observing task-specific rules. Primary indices included planning accuracy, number of correctly solved problems, excess moves, and planning latency, thereby providing measures of preplanning, sequencing, maintenance of task rules, and efficient goal

attainment. Cognitive flexibility and set-shifting were assessed using the Wisconsin Card Sorting Test. Participants were required to infer changing sorting principles on the basis of corrective feedback, with principal indices including the number of categories achieved, perseverative responses, perseverative errors, and failure to maintain set. To obtain a broader measure of executive functioning and reduce dependence on a single task-specific indicator, selected indices from the Tower of London and Wisconsin Card Sorting Test were direction-corrected so that higher values consistently represented superior performance, standardized relative to the baseline sample, and averaged to produce an executive-function composite score. This composite was considered particularly relevant to OCD because difficulties with cognitive flexibility, planning, goal maintenance, and disengagement from previously reinforced response patterns may contribute to persistent checking, repetitive corrective behavior, and difficulty terminating compulsive routines.

Response inhibition was primarily measured using a computerized stop-signal task. Participants were instructed to make rapid left- or right-handed responses to visually presented go stimuli, while a proportion of trials were followed by an auditory stop signal indicating that the initiated motor response should be inhibited. The interval between the presentation of the go stimulus and the stop signal was dynamically adjusted through a tracking procedure designed to produce successful inhibition on approximately 50% of stop trials. Stop-signal reaction time was calculated using the integration method and served as the primary measure of motor response inhibition, with shorter values representing more efficient inhibitory control. Quality-control procedures included examination of go-trial accuracy, mean go reaction time, failed-stop reaction time, and the probability of successful stopping to ensure compatibility with the assumptions of the independent race model. Because inhibitory dysfunction in OCD can involve both the suppression of an initiated motor response and resistance to interference from competing stimulus dimensions, interference control was additionally evaluated through a computerized Stroop task. Participants responded to the ink color of congruent and incongruent color words while attempting to suppress automatic reading responses. A Stroop interference cost was derived from the difference in performance between incongruent and congruent conditions, with lower interference costs representing more efficient conflict resolution and inhibitory control.

Neurophysiological functioning was assessed using electroencephalography and event-related potentials

recorded during computerized cognitive-control paradigms. EEG signals were acquired from 64 Ag/AgCl scalp electrodes positioned according to the extended international 10–20 system while participants completed a visual Go/No-Go paradigm and a speeded flanker task. Data were recorded at a sampling rate of 1000 Hz, filtered using a 0.1–30 Hz band-pass filter, and notch-filtered at 50 Hz to attenuate line-frequency interference. Signals were subsequently re-referenced to the average reference, and ocular and other stereotyped artifacts were identified and corrected using independent-component analysis. Continuous data were segmented into task-relevant epochs, baseline-corrected, and screened for remaining artifacts, with epochs exceeding ± 100 μ V after correction rejected from subsequent averaging. During the Go/No-Go paradigm, the NoGo-N2 component was quantified as mean amplitude within approximately 250–350 ms following stimulus presentation at frontocentral electrodes, particularly FCz and Cz, and was interpreted as an index of conflict detection and recruitment of inhibitory control. The NoGo-P3 component was quantified within approximately 350–550 ms over central and centroparietal electrodes and was considered an index of successful inhibitory processing and evaluation of response suppression. Performance-monitoring activity was assessed primarily during the flanker paradigm. The error-related negativity was defined within approximately 0–100 ms following erroneous responses at FCz and adjacent frontocentral sites, whereas error positivity was quantified within approximately 200–400 ms after erroneous responses over centroparietal electrodes. Participants were required to contribute at least 30 artifact-free correct NoGo trials for inhibitory-control components and at least six artifact-free error trials for analyses of error-related activity. Amplitudes rather than peak latencies were designated as the principal ERP outcomes because mean-amplitude scoring tends to provide greater reliability in clinical psychophysiological research. These electrophysiological indicators were selected because altered N2, P3, ERN, and Pe responses have been repeatedly associated with inhibitory control, conflict monitoring, error processing, and excessive performance monitoring in OCD and therefore provided theoretically relevant neural markers through which the mechanisms of the integrated intervention could be examined.

2.3. *Intervention*

The integrated cognitive control–metacognitive intervention consisted of eight weekly group sessions of approximately 120 minutes, conducted in groups of 8–10 participants by a clinical psychologist trained in metacognitive therapy and cognitive rehabilitation, with psychiatric supervision available when clinically necessary. The protocol was designed to combine metacognitive modification of dysfunctional beliefs about intrusive thoughts with explicit training of attentional regulation, response inhibition, goal maintenance, performance monitoring, and flexible behavioral control. The first session established a shared formulation of the obsessive–compulsive cycle by distinguishing intrusive thoughts from metacognitive interpretations of those thoughts and examining how threat monitoring, attempts to obtain certainty, neutralization, ritualized responding, and rigid stopping criteria maintain symptoms. Participants identified their characteristic sequences of intrusive cognition, metacognitive appraisal, emotional response, and compulsive or neutralizing behavior and began daily monitoring of these sequences. The second session targeted dysfunctional metacognitive beliefs, including thought–action or thought–event fusion, beliefs concerning the necessity of controlling thoughts, assumptions that rituals prevent adverse outcomes, and beliefs that intrusive cognitions must be neutralized. Detached mindfulness and decentered responding were introduced to encourage observation of intrusive mental events without engaging in extended appraisal, suppression, reassurance seeking, or neutralization, and behavioral experiments were used to examine predictions regarding the consequences of allowing thoughts to remain unresolved. The third session focused on attentional flexibility through procedures derived from attentional training, including selective attention, rapid shifting of attentional focus, divided attention, and deliberate reorientation from internally focused threat monitoring toward external task-relevant cues. Participants practiced brief attentional exercises each day and progressively applied them in situations likely to trigger obsessional perseveration. The fourth session introduced a goal-management STOP routine in which participants learned to interrupt automatic responding, orient attention to the present situation, explicitly state the current goal, and evaluate whether the intended behavior served that goal. The STOP procedure was repeatedly rehearsed before checking, washing, repeating, reassurance seeking, mental

neutralization, and other habitual responses. The fifth session developed hierarchical goal representation and subgoal by teaching participants to translate vague intentions into explicit superordinate goals and manageable subgoals, recognize competing habitual routines, identify contexts in which goal neglect occurred, and deliberately reactivate task-relevant goals following attentional capture by obsessional doubt. The sixth session directly targeted response inhibition and interference control through therapist-guided and computerized stop/change exercises, Stroop-like conflict tasks, delayed-response procedures, and deliberate pause-before-action strategies. Participants were encouraged to tolerate the discomfort and uncertainty associated with withholding a habitual response rather than immediately resolving the internal signal through a compulsion. The seventh session addressed performance monitoring and adaptive use of errors. Participants learned to distinguish informative errors from threat signals, limit perfectionistic overchecking, apply a “check once, update, continue” principle, and use mistakes as cues for proportionate behavioral correction rather than restarting tasks or entering repetitive verification cycles. The final session integrated the metacognitive, attentional, inhibitory, and goal-management strategies into a personalized relapse-prevention framework. Participants identified high-risk situations and rehearsed an individualized sequence involving interruption of autopilot responding, attentional reorientation, activation of a relevant goal, toleration of uncertainty, inhibition of ritualized action, and resumption of valued behavior. Between-session assignments consisted of short, repeated applications of treatment strategies in naturally occurring situations involving intrusive thoughts, compulsive urges, attentional capture, and habitual responding rather than prolonged decontextualized cognitive exercises. Attendance and homework completion were recorded throughout the intervention. Treatment delivery was manualized, and a randomly selected proportion of audio-recorded sessions was independently evaluated using a 12-item adherence checklist representing essential metacognitive and cognitive-control components. The control group remained on an 8-week wait-list while continuing stable treatment as usual. Changes in medication and initiation of additional structured OCD psychotherapy were discouraged during the controlled phase, and participants assigned to the control condition were offered the integrated intervention after completion of their follow-up assessment.

2.4. Data Analysis

Statistical analyses were conducted using a two-sided significance level of .05. Prior to hypothesis testing, data were examined for coding errors, implausible values, missing observations, distributional characteristics, and assumptions relevant to each inferential model. Baseline demographic and clinical characteristics were summarized separately for the intervention and control groups using means and standard deviations for continuous variables and frequencies and percentages for categorical variables, and the magnitude of clinically meaningful baseline imbalance was considered when specifying adjusted models. The primary analysis examined longitudinal change in obsessive-compulsive symptoms using generalized estimating equations with a Gaussian distribution, identity link function, and exchangeable working-correlation structure. The model included treatment group, assessment time, and the group-by-time interaction, with baseline specified as the reference occasion. The group-by-time interaction represented the principal test of intervention effectiveness because it assessed whether temporal change differed systematically between the integrated intervention and control conditions. The same longitudinal framework was applied to secondary clinical and cognitive outcomes, including MCQ-30 scores, executive-function composite scores, stop-signal reaction time, and Stroop interference costs. Where multiple outcomes were examined within the same conceptual family, secondary *p* values were adjusted using the Holm procedure to reduce inflation of the familywise Type I error rate while preserving greater statistical power than conventional Bonferroni correction. Post-treatment and follow-up contrasts were estimated with 95% confidence intervals. Between-group effect sizes were calculated using Hedges' *g*, with the direction of scoring standardized when necessary so that larger absolute values consistently reflected stronger intervention-control separation. Categorical clinical response and remission rates were calculated using the prespecified Y-BOCS criteria and compared descriptively between groups, with risk ratios and corresponding 95% confidence intervals reported where appropriate. EEG and ERP outcomes were analyzed primarily using baseline-adjusted post-treatment models in which the post-treatment amplitude of each electrophysiological component was entered as the dependent variable and treatment condition and the corresponding baseline ERP amplitude were entered as predictors. This approach was applied separately to NoGo-

N2, NoGo-P3, ERN, and Pe amplitudes after exclusion of datasets that failed prespecified signal-quality or minimum-trial requirements. The primary longitudinal analyses used all available repeated observations from randomized participants without restricting the sample to treatment completers. Missing outcome observations were therefore accommodated within the available-data GEE framework, while multiple imputation was used as a sensitivity analysis to evaluate the robustness of principal findings to assumptions regarding missingness. In addition to efficacy analyses, exploratory mechanism analyses examined whether improvement in obsessive-compulsive symptoms was statistically mediated by changes in metacognitive beliefs, executive functioning, inhibitory control, or neurophysiological cognitive-control indices. Parallel mediation models were constructed using standardized baseline-to-post-treatment change scores. Improvement in metacognition was coded so that larger values represented greater reductions in maladaptive MCQ-30 beliefs, improvement in inhibitory control was coded so that larger values represented greater shortening of stop-signal reaction time, executive-function change was coded so that larger values represented improved performance, and NoGo-P3 change was coded so that larger values represented greater post-treatment amplitude enhancement. Improvement in Y-BOCS total score served as the dependent variable, and baseline Y-BOCS severity was included as a covariate. Indirect effects were evaluated through 5,000 nonparametric bootstrap resamples with bias-corrected confidence intervals. Because the sample size and trial design were optimized primarily to detect treatment efficacy rather than to establish definitive temporal causal mediation, these mediation analyses were interpreted as exploratory evidence concerning potential mechanisms of therapeutic change rather than confirmatory causal tests.

3. Findings and Results

Eighty participants were randomized, with 40 assigned to each group. Seventy-seven participants provided post-treatment clinical data, and 74 provided data at the 8-week follow-up. EEG data that met the predefined artifact-rejection and minimum trial-count criteria at both baseline and post-treatment were available for 72 participants, comprising 36 participants in each group. Attrition was not disproportionately concentrated in the intervention group. Baseline demographic and clinical characteristics were broadly comparable between the two groups, and mean Y-

BOCS scores indicated severe obsessive-compulsive symptomatology in both groups. Most participants were

receiving a stable pharmacological regimen for OCD at baseline.

Table 1

Baseline Demographic and Clinical Characteristics

Characteristic	Control (n = 40)	ICCM I (n = 40)	Overall
Age, years	31.1 ± 8.0	32.3 ± 8.4	31.7 ± 8.2
Female, n (%)	21 (52.5)	28 (70.0)	49 (61.3)
Stable OCD medication, n (%)	31 (77.5)	24 (60.0)	55 (68.8)
Illness duration, years	7.4 ± 4.0	8.4 ± 4.9	7.9 ± 4.5
Y-BOCS total	26.8 ± 4.7	27.8 ± 4.7	27.3 ± 4.7
MCQ-30 total	71.7 ± 8.6	73.0 ± 9.0	72.3 ± 8.8
SSRT, ms	257.0 ± 21.4	262.7 ± 19.4	259.8 ± 20.5

Y-BOCS severity decreased substantially in the ICCMI group, whereas only modest improvement was observed in the control group. The GEE group × time interaction was statistically significant at post-treatment ($B = -8.92$, $SE = 0.47$, $p < .001$) and remained significant at the 8-week follow-up ($B = -10.16$, $SE = 0.53$, $p < .001$), indicating a substantially greater reduction in obsessive-compulsive symptoms in the ICCMI group over time. The post-treatment between-group effect size was large ($|g| = 1.50$). At post-

treatment, 62.5% of participants in the ICCMI group met the predefined criterion for clinical response, defined as a reduction of at least 35% in Y-BOCS scores, compared with 0% of observed participants in the control group. At follow-up, the corresponding response rates were 85.0% and 0%, respectively. Remission, defined as a Y-BOCS score ≤ 12 , was observed in 15.0% of ICCMI participants at both post-treatment and follow-up.

Table 2

Clinical and Cognitive Outcomes Across Assessment Points

Outcome	Control Baseline	Control Post-treatment	Control Follow-up	ICCM I Baseline	ICCM I Post-treatment	ICCM I Follow-up
Y-BOCS total	26.76 ± 4.68	24.26 ± 5.27	24.79 ± 5.74	27.77 ± 4.71	16.74 ± 4.69	15.90 ± 4.61
MCQ-30 total	71.70 ± 8.64	68.17 ± 8.22	68.61 ± 7.84	72.99 ± 9.00	58.12 ± 9.02	56.14 ± 8.87
Executive composite (z)	-0.16 ± 0.64	-0.11 ± 0.69	-0.14 ± 0.66	-0.23 ± 0.70	0.36 ± 0.69	0.40 ± 0.72
SSRT (ms)	256.97 ± 21.39	251.88 ± 22.64	252.92 ± 22.58	262.73 ± 19.41	233.75 ± 21.80	230.83 ± 20.96
Stroop interference	43.05 ± 9.91	41.28 ± 10.93	41.25 ± 11.76	43.27 ± 8.43	34.28 ± 8.79	33.61 ± 9.30

The intervention also produced marked improvements in the hypothesized cognitive and metacognitive processes. Relative to the control group, MCQ-30 total scores decreased significantly in the ICCMI group at post-treatment (group × time $B = -12.14$, $p < .001$) and follow-up ($B = -14.04$, $p < .001$), indicating a substantial reduction in maladaptive metacognitive beliefs. Executive-function composite scores increased significantly at post-treatment ($B = 0.57$ SD units, $p < .001$) and follow-up ($B = 0.61$ SD units, $p < .001$). Similarly, SSRT decreased significantly at

post-treatment ($B = -25.57$ ms, $p < .001$) and follow-up ($B = -28.32$ ms, $p < .001$), indicating more efficient response inhibition. Stroop interference costs also decreased significantly at post-treatment ($B = -7.88$, $p < .001$) and follow-up ($B = -8.58$, $p < .001$). Taken together, these findings indicate improvements in metacognitive regulation, executive functioning, inhibitory control, and interference resolution, with treatment gains maintained beyond the active intervention period.

Table 3*Group × Time Effects From Generalized Estimating Equations and Post-treatment Effect Sizes*

Outcome	Post-treatment Interaction B (SE)	p	Follow-up Interaction B (SE)	p	Post-treatment Hedges' g
Y-BOCS total	-8.92 (0.47)	< .001	-10.16 (0.53)	< .001	-1.50
MCQ-30 total	-12.14 (0.70)	< .001	-14.04 (0.76)	< .001	-1.15
Executive composite (z)	0.57 (0.04)	< .001	0.61 (0.04)	< .001	0.67
SSRT (ms)	-25.57 (2.05)	< .001	-28.32 (2.19)	< .001	-0.81
Stroop interference	-7.88 (0.55)	< .001	-8.58 (0.76)	< .001	-0.70

Baseline-adjusted EEG models demonstrated significant intervention-related changes in ERP components associated primarily with inhibitory control and post-error processing. NoGo-N2 amplitude became significantly less negative in the ICCMI group relative to the control group, with an adjusted group effect of +1.14 μ V (SE = 0.10, $p < .001$). This pattern was consistent with reduced excessive conflict-related recruitment following treatment. NoGo-P3 amplitude increased significantly in the ICCMI group relative to the control group, with an adjusted group effect of +1.43 μ V (SE = 0.13, $p < .001$), suggesting enhanced

recruitment of neurocognitive resources associated with successful response inhibition. Pe amplitude also increased significantly following the intervention (+0.57 μ V, SE = 0.08, $p < .001$), indicating greater post-error evaluative processing. In contrast, the baseline-adjusted group effect for ERN amplitude was small and did not reach conventional statistical significance (+0.18 μ V, SE = 0.09, $p = .062$). This dissociation was consistent with the a priori possibility that ERN may reflect a comparatively stable performance-monitoring disposition in OCD rather than a symptom-proximal neurophysiological state marker.

Table 4*EEG/ERP Outcomes at Baseline and Post-treatment*

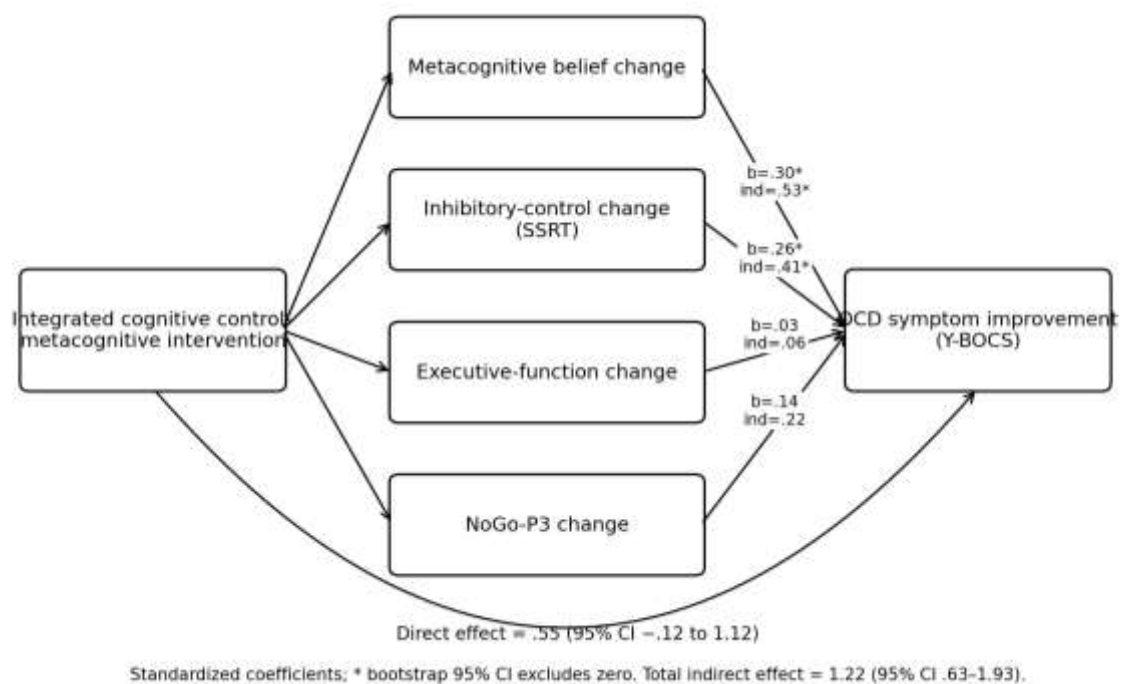
Component	Control Baseline	Control Post-treatment	ICCM I Baseline	ICCM I Post-treatment	Adjusted Group Effect	p	Post-treatment Hedges' g
NoGo-N2 (μ V)	-5.98 $\pm$ 0.72	-5.97 $\pm$ 0.76	-5.92 $\pm$ 0.95	-4.76 $\pm$ 1.10	+1.14 $\pm$ 0.10	< .001	1.26
NoGo-P3 (μ V)	8.80 $\pm$ 1.03	8.90 $\pm$ 1.25	8.45 $\pm$ 0.92	9.96 $\pm$ 1.07	+1.43 $\pm$ 0.13	< .001	0.90
ERN (μ V)	-7.91 $\pm$ 1.20	-7.85 $\pm$ 1.24	-8.35 $\pm$ 1.06	-8.12 $\pm$ 1.17	+0.18 $\pm$ 0.09	.062	-0.22
Pe (μ V)	4.45 $\pm$ 0.70	4.60 $\pm$ 0.83	4.37 $\pm$ 0.74	5.08 $\pm$ 0.93	+0.57 $\pm$ 0.08	< .001	0.54

The exploratory parallel mediation model suggested that improvement in obsessive-compulsive symptoms was statistically transmitted primarily through changes in maladaptive metacognitive beliefs and response inhibition. The standardized indirect effect through reduction in maladaptive metacognitive beliefs was 0.53, with a 95% bootstrap confidence interval of [0.16, 0.98], indicating a statistically significant indirect pathway. The indirect effect through improvement in SSRT was also significant (standardized indirect effect = 0.41, 95% bootstrap CI [0.19, 0.69]). In contrast, the indirect effect through improvement in the executive-function composite was small and nonsignificant (0.06, 95% bootstrap CI [-0.28, 0.38]), and the indirect effect through increased NoGo-P3 amplitude likewise did not reach statistical significance (0.22, 95%

bootstrap CI [-0.06, 0.50]). The total indirect effect across the four proposed mediators was statistically significant (1.22, 95% bootstrap CI [0.63, 1.93]), whereas the residual direct effect of the intervention on Y-BOCS improvement was not statistically significant because its bootstrap confidence interval included zero (0.55, 95% bootstrap CI [-0.12, 1.12]). These results suggest that reductions in maladaptive metacognitive beliefs and improvements in inhibitory control may represent particularly important mechanisms through which ICCMI influences obsessive-compulsive symptom severity. Nevertheless, because mediator and outcome changes were assessed over the same treatment interval, these findings should be interpreted as exploratory mechanistic signals rather than definitive evidence of causal mediation.

Table 5*Exploratory Parallel Mediation of Post-treatment Y-BOCS Improvement*

Path	Standardized Indirect/Direct Effect	95% Bootstrap CI	Interpretation
Metacognitive belief change	0.53	[0.16, 0.98]	Significant
SSRT improvement	0.41	[0.19, 0.69]	Significant
Executive-function improvement	0.06	[-0.28, 0.38]	Not significant
NoGo-P3 increase	0.22	[-0.06, 0.50]	Not significant
Residual direct effect	0.55	[-0.12, 1.12]	Not significant
Total indirect effect	1.22	[0.63, 1.93]	Significant

Figure 1*Exploratory Mechanistic Model of Symptom Improvement*

4. Discussion

The present study evaluated the effectiveness of an integrated cognitive control-metacognitive intervention (ICCM) for adults with obsessive-compulsive disorder and examined whether clinical improvement was accompanied by changes in metacognitive beliefs, executive functioning, inhibitory control, and electrophysiological indices of cognitive control. The findings showed a clear and clinically meaningful advantage for ICCM over the control condition. Obsessive-compulsive symptom severity decreased substantially in the intervention group, whereas the control group showed only modest change. The group $\times$ time effects were significant at both post-treatment and the 8-week follow-up, indicating not only an immediate treatment

benefit but also short-term maintenance of clinical gains. The magnitude of the post-treatment between-group effect was large, and a substantial proportion of participants in the ICCM condition met the predefined criterion for clinical response. These changes were accompanied by significant reductions in maladaptive metacognitive beliefs, improvements in executive-function performance, faster stop-signal inhibition, reduced Stroop interference, and changes in several ERP components. Exploratory mediation analyses further suggested that reductions in maladaptive metacognitive beliefs and improvement in response inhibition represented the strongest candidate mechanisms linking the intervention to symptom reduction.

The pronounced reduction in Y-BOCS severity is consistent with the broader literature indicating that

interventions targeting metacognitive mechanisms can produce substantial improvements in OCD. The metacognitive model proposes that intrusive thoughts become clinically problematic when individuals interpret them as meaningful, dangerous, uncontrollable, or requiring neutralization, and when these interpretations generate repetitive monitoring and ritualized responding. Experimental evidence has shown that manipulating beliefs about intrusive thoughts can alter obsessive-compulsive responding, supporting the proposition that metacognitive beliefs are not merely correlates of OCD but may play an active role in maintaining symptoms (Fisher & Wells, 2005). Clinical evidence has also demonstrated that metacognitive therapy can reduce obsessive-compulsive symptoms, initially in case-series research and subsequently in randomized comparisons (Fisher & Wells, 2008; Glombiewski et al., 2021). More recent evidence indicating that metacognitive therapy can perform comparably with exposure and response prevention further supports the therapeutic relevance of directly addressing beliefs about thoughts, rituals, uncertainty, and cognitive control (Exner et al., 2024). The current findings extend this line of work by suggesting that metacognitive procedures may become especially effective when they are combined with structured cognitive-control training rather than delivered as an exclusively belief-focused intervention.

The reduction in MCQ-30 scores is particularly important because the intervention was designed to weaken dysfunctional beliefs concerning the significance and controllability of thoughts while encouraging detached responding to intrusive mental events. Participants were trained to distinguish the occurrence of an intrusion from the meaning assigned to it and to reduce the tendency to use compulsions as a strategy for controlling internal experience. The strong improvement in metacognitive beliefs is therefore consistent with the intended treatment mechanism. Previous studies have shown that fusion beliefs, beliefs about rituals, and internal stopping criteria are closely related to obsessive-compulsive symptom expression (Myers et al., 2009). From this perspective, repeated checking or neutralization may continue not because the individual lacks insight, but because a metacognitive rule such as “I must be completely certain before I stop” remains active. ICCMI directly targeted such beliefs while simultaneously training participants to disengage from repetitive cognitive processing and resume goal-directed behavior. The present results are therefore compatible with the idea that modifying how patients regulate their

relationship with intrusive thoughts may be as important as modifying the content of the thoughts themselves.

The observed metacognitive improvements are also consistent with findings from Iranian research indicating that maladaptive metacognitive beliefs are closely associated with emotional distress and may be amenable to psychological intervention (Bahrami & Yousefi, 2011). More recent Iranian work has also demonstrated that therapeutic modification of maladaptive cognitive structures can influence metacognitive beliefs in individuals with obsessive-compulsive symptoms (Tajizadegan & Sharifi Isfahani, 2026). These findings are relevant because the present study was conducted in an Iranian clinical context and therefore provides additional support for the cross-contextual applicability of metacognitive treatment principles. The current findings suggest that interventions addressing beliefs about thought control, danger, and uncertainty can be integrated successfully with cognitive-rehabilitation procedures within this cultural and clinical setting.

A second major finding was the improvement in executive functioning. Participants receiving ICCMI demonstrated significant gains in the executive-function composite at both post-treatment and follow-up. This result is compatible with the substantial body of evidence showing that adults with OCD exhibit difficulties in planning, set-shifting, cognitive flexibility, and related executive processes (Abramovitch et al., 2013). Iranian studies have similarly reported executive and selective-attention difficulties among individuals with OCD, suggesting that deficits in these domains may have direct relevance to daily functional impairment (Yazdi-Ravandi et al., 2018). The intervention specifically targeted several processes likely to be reflected in executive performance, including interruption of automatic action, explicit goal reactivation, hierarchical goal organization, flexible re-planning, and adaptive performance monitoring. The observed improvement may therefore reflect enhanced capacity to sustain goal-directed behavior rather than remaining captured by rigid, repetitive, or previously reinforced action sequences.

The use of goal-management strategies may have been particularly relevant to these gains. Goal Management Training was developed to improve executive functioning by helping individuals interrupt automatic responding, identify current goals, organize subgoals, and monitor whether behavior remains aligned with intended objectives (Levine et al., 2011). These principles map closely onto several behavioral characteristics of OCD. Compulsive actions often

become detached from broader goals and are maintained by moment-to-moment attempts to reduce uncertainty or discomfort. By teaching participants to stop, orient to the present task, identify the current goal, and evaluate whether a proposed action serves that goal, ICCMI may have reduced the degree to which behavior was governed by obsession-driven automaticity. The maintenance of executive-function gains at follow-up also suggests that the intervention may have promoted generalizable strategies rather than only task-specific improvements.

The results for inhibitory control were similarly notable. SSRT decreased significantly in the intervention group relative to the control group, indicating faster stopping of an initiated response. Stroop interference also decreased, suggesting improved ability to manage conflict between competing response tendencies. These findings align with evidence that response inhibition is impaired in OCD. Meta-analytic research using the stop-signal task has shown that individuals with OCD typically demonstrate less efficient stopping than healthy comparison groups (Mar et al., 2022). The stop-signal framework conceptualizes inhibition as a race between go and stop processes, with shorter SSRT reflecting a more efficient inhibitory mechanism (Verbruggen & Logan, 2008). Within the clinical context of OCD, inefficient stopping may translate into difficulty interrupting checking, washing, repeating, reassurance seeking, or covert neutralization once these actions have been initiated.

The current results are also compatible with neuroimaging evidence indicating altered recruitment of inhibitory-control networks in OCD (Li et al., 2024). ICCMI did not treat inhibition as an abstract laboratory skill alone. Instead, response-inhibition exercises were integrated with symptom-relevant strategies such as pausing before habitual checking, delaying a ritualized response, tolerating uncertainty during response withholding, and reactivating a competing goal. This integration may have strengthened the likelihood that improved inhibitory control transferred from training tasks to clinically relevant situations. The broader cognitive-training literature in OCD has produced encouraging but heterogeneous results, with some studies showing gains in targeted cognitive processes and variable transfer to symptoms (Bhattacharya et al., 2024). The present study suggests that cognitive training may achieve greater clinical relevance when explicitly linked to metacognitive beliefs and real-world compulsive behavior.

The neurophysiological findings provide additional evidence that the intervention influenced cognitive-control

mechanisms beyond self-reported or behavioral change. NoGo-N2 became less negative in the ICCMI group relative to control, while NoGo-P3 amplitude increased significantly. These components are commonly interpreted in relation to conflict detection, inhibitory recruitment, and successful implementation of response control. Recent electrophysiological reviews of OCD have identified abnormalities in N2 and P3 responses among the more consistent neural correlates of impaired cognitive control (Chmiel & Kładna, 2026). The reduction in excessive NoGo-N2 negativity observed in the present study may reflect more efficient conflict processing or reduced need for compensatory recruitment during inhibition. At the same time, the increase in NoGo-P3 may indicate more effective allocation of resources to successful suppression of prepotent responses. These neural changes are broadly congruent with the behavioral evidence of shorter SSRT and reduced Stroop interference, suggesting convergence across multiple levels of measurement.

The increase in Pe amplitude also warrants attention. Pe is generally associated with conscious evaluation and processing of errors after an incorrect response. The intervention explicitly trained participants to interpret errors as informative signals rather than as evidence of threat, incompleteness, or the need for repetitive correction. Participants were encouraged to adopt a “check once, update, continue” approach rather than returning repeatedly to completed actions. The increase in Pe may therefore reflect more adaptive post-error evaluation rather than heightened pathological monitoring. This interpretation is consistent with the intervention’s aim of transforming the functional meaning of errors, rather than simply suppressing awareness of them.

In contrast, ERN did not show a statistically significant treatment-related change. This finding is theoretically informative because enhanced ERN has been proposed as a relatively stable neurophysiological marker of OCD vulnerability rather than a direct index of current symptom severity. A review and meta-analysis identified error-related negativity as a potential endophenotypic feature of OCD and suggested that it may remain elevated even when clinical symptoms improve (Riesel, 2019). The present dissociation between marked symptom improvement and nonsignificant ERN change is compatible with this interpretation. It suggests that some neurophysiological characteristics may represent relatively enduring dispositions, whereas other indices, including NoGo-N2, NoGo-P3, and Pe, may be more responsive to cognitive and metacognitive

intervention. This distinction is important because it cautions against assuming that successful treatment must normalize every neural abnormality associated with OCD.

The exploratory mediation findings provide further insight into how ICCMI may have produced clinical change. Reductions in maladaptive metacognitive beliefs and improvement in SSRT each showed significant indirect effects on Y-BOCS improvement, whereas the executive-function composite and NoGo-P3 did not show individually significant indirect effects. This pattern is consistent with the theoretical structure of the intervention. Metacognitive beliefs influence how intrusive thoughts are interpreted and whether they trigger ritualized responding, while inhibitory control determines whether an already activated habitual response can be withheld. Together, these mechanisms may represent particularly proximal determinants of whether an intrusive thought progresses into a compulsion. The metacognitive model has long emphasized the role of fusion beliefs, ritual beliefs, and subjective stop criteria in maintaining OCD (Myers et al., 2009), while the response-inhibition literature highlights the difficulty patients may experience when attempting to suppress habitual actions (Mar et al., 2022). Their joint contribution in the present mediation model therefore provides a coherent account of clinical improvement.

The nonsignificant indirect effects for executive functioning and NoGo-P3 should not be interpreted as evidence that these changes were unimportant. Both domains improved significantly at the group level, but their unique mediation effects may have been smaller, overlapping with other mechanisms, or insufficiently powered within the present sample. Executive-function gains may support broader behavioral organization and maintenance of treatment strategies without serving as the most immediate pathway to symptom reduction. Similarly, NoGo-P3 may reflect neural efficiency associated with successful inhibition but may not independently account for clinical change once behavioral inhibition and metacognitive change are entered into the same model. The significant total indirect effect nevertheless supports the broader proposition that treatment response was distributed across multiple interacting mechanisms rather than arising from a single process. This interpretation is consistent with recent efforts to combine cognitive remediation and metacognitive treatment in OCD rather than treating the disorder as exclusively cognitive, behavioral, or neurophysiological (Zargarinejad et al., 2025).

5. Conclusion

The present findings therefore support an integrated conceptualization of OCD in which maladaptive beliefs about thoughts and deficits in cognitive control mutually reinforce one another. Intrusive thoughts may activate beliefs about danger, responsibility, or the need for certainty; these beliefs may intensify attention to internal threat cues and increase the urge to perform a ritual; inefficient inhibitory control may then reduce the capacity to stop the ritualized response; and repeated checking or neutralization may strengthen the belief that continued monitoring is necessary. ICCMI appears to intervene at several points within this cycle by weakening maladaptive metacognitive beliefs, improving attentional and inhibitory control, reactivating explicit goals, and teaching more adaptive error-monitoring strategies. The large clinical effect observed in the present study is therefore plausible within a multilevel treatment model that simultaneously targets beliefs, behavior, and cognitive control.

Several limitations should be considered when interpreting the findings. First, the sample size was adequate for the primary efficacy analyses but relatively modest for complex mediation and electrophysiological models, which limits precision in estimating indirect effects and subgroup differences. Second, the study was conducted in a limited number of outpatient centers in Tehran, which may restrict generalizability to other clinical settings, cultural contexts, or patients with different levels of comorbidity and treatment history. Third, the control condition was a wait-list with stable treatment as usual rather than an active psychological comparison condition, so nonspecific effects of therapist attention, group participation, treatment expectancy, and structured weekly engagement cannot be fully excluded. Fourth, EEG outcomes were assessed only at baseline and post-treatment, preventing evaluation of the longer-term stability of neurophysiological changes. Fifth, the exploratory mediation analyses used mediator and outcome changes measured across the same treatment interval, meaning that temporal precedence could not be established and causal mediation cannot be inferred. Finally, although adherence and fidelity procedures were incorporated, the intervention combined several active components, making it impossible to determine the independent contribution of metacognitive therapy, goal-management strategies, attentional training, or inhibitory-control exercises.

Future research should replicate these findings in larger multisite samples and include active comparator conditions

such as exposure and response prevention, metacognitive therapy alone, or cognitive remediation alone to clarify the incremental value of treatment integration. Dismantling designs would be particularly useful for identifying which components are necessary, additive, or redundant. Longer follow-up periods should be included to determine whether clinical, cognitive, and electrophysiological gains remain stable over 6 or 12 months. Future trials should also collect mediator variables at multiple intermediate time points so that the temporal order of metacognitive, inhibitory, executive, and symptom changes can be tested more rigorously. Larger samples would permit examination of moderators such as symptom dimension, baseline cognitive impairment, medication status, illness duration, comorbid depression, and severity of metacognitive dysfunction. Additional neurophysiological and neuroimaging measures could also help determine whether treatment changes are localized to specific inhibitory or error-monitoring systems or reflect broader network-level reorganization.

From a clinical perspective, the findings suggest that treatment planning for OCD may benefit from assessing both metacognitive beliefs and cognitive-control functioning rather than focusing exclusively on symptom frequency or content. Patients who display pronounced ritual stopping difficulties, rigid checking, goal neglect, poor response inhibition, or dysfunctional beliefs about thought control may particularly benefit from an integrated approach. Clinicians may consider combining metacognitive techniques with structured exercises that strengthen response inhibition, attentional flexibility, goal reactivation, and adaptive error use, while ensuring that these exercises are directly linked to real-world obsessive-compulsive situations. Brief, repeated practice embedded in daily symptom contexts may enhance transfer more effectively than decontextualized cognitive exercises alone. The intervention may also be useful as an adjunct to established OCD treatments when residual symptoms appear to be maintained by rigid metacognitive rules or executive-control difficulties.

Authors' Contributions

Authors equally contributed to this article.

Declaration

In order to correct and improve the academic writing of our paper, we have used the language model ChatGPT.

Transparency Statement

Data are available for research purposes upon reasonable request to the corresponding author.

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Declaration of Interest

The authors report no conflict of interest.

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Ethical Considerations

The study protocol adhered to the principles outlined in the Helsinki Declaration, which provides guidelines for ethical research involving human participants.

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