Research Article

NEUROPSYCHOLOGICAL PREDICTORS OF TREATMENT RESPONSE TO COGNITIVE BEHAVIORAL GROUP THERAPY IN OBSESSIVE–COMPULSIVE DISORDER

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Background: ***The available research on the relationship between neuropsychological functioning and the therapeutic outcome of obsessive–compulsive disorder (OCD) has yielded inconsistent results. In this study, our aim was twofold. First, we sought to evaluate the effects of cognitive behavioral group therapy (CBGT) on neurocognitive functions in OCD patients. Second, we assessed the viability of neuropsychological test performance as a predictor of treatment response to CBGT.*** **Methods:** ***One hundred fifty carefully screened OCD patients were randomized to receive either 12-week CBGT (n = 75) or to remain on a waiting list (WL; n = 75) for the corresponding time. Forty-seven participants dropped out of the study, leaving 103 participants that were included in the analysis (CBGT, n = 61; WL, n = 42). Participants had several neuropsychological domains evaluated both at baseline and at end-point.*** **Results:** ***A significant difference in obsessive–compulsive, anxiety, and depression symptoms was observed between treated patients and controls favoring the CBGT group, but no significant differences were found on neuropsychological measures after 3 months of CBGT. In addition, there were no differences between treatment responders and nonresponders on all neuropsychological outcome measures. Employing a conservative alpha, neuropsychological test performance did not predict CBGT treatment response.*** **Conclusions:** ***Although the CBGT group demonstrated significant improvement in OCD symptoms, no significant difference was found on all neuropsychological domains, and test performance did not predict treatment response. Depression and Anxiety 33:848–861, 2016.*** *© 2016 Wiley Periodicals, Inc.*

Key words: ***OCD; CBT; group therapy; neuropsychology; executive function; treatment response***

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BACKGROUND

Obsessive–compulsive disorder (OCD) is a prevalent disorder (2.5%),[1] characterized by obsessions and/or compulsions that cause marked distress, consume time, and interfere significantly with daily routines, work, social, or family life.[2] Unsurprisingly, OCD is associated with reduced quality of life in multiple domains.[3] Brain imaging studies strongly link OCD with abnormal activation of the orbitofrontal cortex, anterior cingulate cortex, lateral frontal and temporal cortices, caudate nucleus, thalamus, amygdala, and insula.[4–7] In addition, functional connectivity studies reveal aberrant cortico-striato-thalamo-cortical (CSTC) connectivity in OCD.[8,9] Taken together, the prevailing CSTC neurobiological model of OCD[10] suggests that OCD may be associated with neuropsychological deficits, particularly in the domain of executive functions.

However, neuropsychological findings in OCD are extremely inconsistent. Some neuropsychological studies in adult OCD patients describe impairment in nonverbal memory and deficits in organizational strategies,[11–14] while others suggest that adults with OCD have deficits in cognitive flexibility and response inhibition,[15–17] and yet others report no neuropsychological deficits in any of the aforementioned domains.[18–21] In a recent meta-analysis of neuropsychological functions of OCD patients, Abramovitch et al.[22] found effect sizes indicating reduced performance on all neuropsychological domains of OCD patients when compared to healthy controls. However, the magnitude of neuropsychological deficits across domains varied. A medium to large effect size was found for the memory domain, while medium effect sizes were reported for attention, executive function, and processing speed. Finally, small effect sizes were found for working memory and visuospatial abilities. Furthermore, this study reported that statistically significant heterogeneity was found across effect sizes representing most neuropsychological domains. Nevertheless, a comprehensive moderator analysis attempting to account for this heterogeneity revealed no statistically significant moderator effect across neuropsychological domains.[22]

Although neuropsychological deficits have been identified in OCD, little research has focused on whether these deficits are associated with active symptoms of the disorder. In other words, it remains unclear whether neuropsychological deficits in OCD are state-dependent[23] or an innate trait feature of OCD.[24,25] Some studies have observed persistent cognitive dysfunction following successful pharmacological treatment for OCD for different periods of time,[26–28] suggesting that cognitive impairments are not secondary to symptoms but rather are an OCD trait feature. However, others have reported improved performance on tests such as the Stroop test and Wisconsin Card Sorting Test (WCST) following aripiprazole augmentation of serotonin reuptake inhibitors (SRIs) at week 16.[29] Nonpharmacological treatment studies examining neuropsychological correlates of treatment response to cognitive behavioral therapy (CBT) found significantly improved performance on neuropsychological tests among treatment responders,[29] supporting the view that neuropsychological performance may be a state-dependent epiphenomenon of OCD.[24,31] Abramovitch et al.[32] provided a theoretical rationale for such improvement. The authors described an executive overload model of OCD in which an overflow of obsessive thoughts causes an overload on the executive and attentional systems, consuming valuable resources needed for normative cognitive functioning. This leads to reduction in the amount of resources available for cognitive function, which would be most pronounced in neuropsychological assessment settings, where tests of varying cognitive loads require the full capacity of cognitive systems. Following this logic, it is plausible that reduction in OCD symptom severity may correspond to increased availability of cognitive resources and thus to improved test performance. In addition, several studies indicate that reduction in neuropsychological test performance may be common across numerous disorders,[33,34] in line with the work of Caspi et al.,[35] indicating that the P factors (i.e., having a psychopathology) are predictive of reduced cognitive functioning. It is thus possible that a general improvement in clinical status may be associated with improvement in neuropsychological test performance.

Another important question that remains controversial is whether cognitive functioning predicts treatment outcome in OCD. Although only a limited number of studies addressed this question, it has been speculated that deficits in nonverbal memory and execution function (e.g., impaired decision making, set shifting, or mental flexibility), could hypothetically limit patients' ability to comprehend, remember, and implement the issues raised during therapeutic interventions. In an attempt to test this hypothesis, some studies reported that individuals with OCD that did not respond to treatment with SRIs also relatively underperformed when compared to treatment responders on a gambling task.[36] Others reported that impairments in set-shifting ability (i.e., lower number of categories completed and increased perseverative responses in the WCST) were associated with a positive treatment response to SRIs.[37]

Studies investigating neuropsychological predictors of treatment response to CBT in OCD found that underperformance on neuropsychological tests before treatment was more common among subsequent nonresponders.[29] Conversely, Bolton et al.[38] found that neither neuropsychological test performance nor neurological soft signs predicted response to behavioral treatment in OCD. In one recent clinical trial investigating neuropsychological test performance as a predictor of response to fluoxetine and CBT, the authors found that those OCD patients who performed better on tests of executive functions at baseline subsequently showed better response to treatment.[39] Interestingly, mental flexibility predicted a favorable response to CBT,

but also heralded a worse response to fluoxetine. The authors suggest that depending on individual neuropsychological profiles, treatment response to a given intervention may differ. For example, OCD patients with higher Verbal IQ and reduced perseveration scores on the WCST tend to respond better to CBT, whereas those participants with better inhibitory control, but more perseverative responses/errors may respond better to fluoxetine. Consequently, the authors suggested that particular treatments can be assigned according to specific neuropsychological features.[37] For pediatric OCD patients, one study showed that poorer nonverbal memory at baseline predicted poorer response to either sertraline, CBT, or both, with a larger effect size for CBT alone.[40]

In light of the limited and divergent literature concerning neuropsychological predictors of treatment response to CBT, the aim of the present investigation was to examine neuropsychological predictors of response to cognitive behavioral group therapy (CBGT) for OCD. Although CBGT has been found to be an effective treatment for OCD,[41–44] to our knowledge no study to date has examined neuropsychological predictors for treatment response to CBGT. Considering that aspects of nonverbal memory and executive function may be associated with treatment response,[39,40,45] we hypothesized that CBGT would be an effective treatment for OCD and that reduced nonverbal memory and executive functions would be predictive of poorer treatment response. Finally, in light of research findings, and the predictions put forth by the executive overload model, we hypothesized that executive functions, nonverbal memory, and attention would improve following successful treatment.

MATERIALS AND METHODS

STUDY DESIGN AND TREATMENT PROTOCOL

Subjects were randomly assigned to CBGT or to waiting list (WL) conditions. CBGT consisted of twelve 2-hr weekly sessions spread over 3 months, conducted according to a previously developed and standardized protocol (see Cordioli et al.[41,46]). This protocol included psychoeducation about OCD, exposure and response prevention (ERP) techniques, cognitive restructuring (techniques targeting dysfunctional beliefs), and group therapeutic factors such as universality, setup of hope, altruism, etc.[47] Cognitive techniques used in this protocol are generally adaptations of those originally described by Beck[48] for depression treatment and by Foa et al.[49–53] for OCD. In the initial sessions patients received information on OCD and were taught to identify its symptoms. In these sessions, patients were provided with live exhibitions of ERP. In subsequent sessions, commonly held dysfunctional beliefs were explained and cognitive techniques for their corrections were given, followed by practical exercises of ERP and cognitive restructuring. At the end of each session, homework exercises were assigned to each patient. In the final sessions, the focus was on strategies for relapse prevention; patients were educated on the differences between lapses and relapses and trained in coping strategies for risky situations.

From August 2008 to August 2010, 11 successive groups were carried out with six to eight participants in each. All sessions were conducted by the same trained Ph.D. and masters level clinicians and assisted by an equally experienced cotherapist. All clinicians specialized in CBT for OCD, with at least 5 years of clinical experience. Following the waiting period, patients in the WL group were offered the same treatment but these data are not included in the present study.

PARTICIPANTS

Recruitment for the present study was conducted using newspaper, television, and radio advertisements. Inclusion criteria were age 18–65 years; a primary diagnosis of OCD obtained using the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I);[54] and a score of 16 or more on the Yale-Brown Obsessive–Compulsive Scale (Y-BOCS).[55] Exclusion criteria were a lifetime diagnosis of neurological disorders (e.g., traumatic brain injury, epilepsy), any psychotic or autism spectrum disorders, intellectual disabilities, and current abuse of alcohol or other substances (excluding nicotine).

Initial screening of 169 subjects led to exclusion of 19 individuals. Subsequently, 150 patients were randomized. Seventy-five OCD patients were allocated to CBGT and 75 to WL. The screening, randomization and dropout in the follow-up period are depicted in Fig. 1. Written informed consent was obtained from all patients before entering the study. The Institutional Review Board of the Hospital de Clínicas of Porto Alegre approved the study.

MEASURES

Clinical Measures. The *SCID-I* was used to confirm a diagnosis of OCD and other comorbid Axis I disorders at intake.[54] The Portuguese version of the *Y-BOCS*[56] was used in order to assess the severity of OCD symptoms. The Y-BOCS comprises ten items: five items assessing obsessions and five assessing symptom severity associated with compulsions. Each item is scored using a Likert scale ranging from 0 to 4 (thus leading to a total score of 0 to 40).

The *Clinical Global Impression—Severity Scale* (CGI-S)[57] was used to assess the severity of OCD symptoms. Its scores range from 1 (minimal) to 7 (very severe) symptoms.

The *Beck Anxiety Inventory* (BAI)[58] and the *Beck Depression Inventory* (BDI)[59] are each 21-item self-report questionnaires that assess symptom severity for anxiety and depression, respectively. Both scales have a total score that ranges between 0 and 63, where higher scores represent more severe symptoms. For the present study, we used the Brazilian-Portuguese version of the two scales.[60]

Functional impairments were assessed using the *Functioning Assessment Short Test* (FAST).[61] The FAST is a structured interview that includes items pertaining to autonomy, work, cognitive functioning, socioeconomic issues and interpersonal relationships. Each item is scored using a 4-point Likert scale, ranging between 0 (no difficulty) and 3 (severe difficulty). The FAST provides a total score of functioning in addition to six specific subscores.

Neuropsychological Measures. *The Wechsler Abbreviated Scale of Intelligence (WASI)*[62] is a short and reliable measure of intelligence that consists of four subtests: Vocabulary, Block Design, Similarities, and Matrix Reasoning. Verbal IQ is calculated from the Similarities and Vocabulary subscores, and Performance IQ is calculated from the scores of the Block Design and Matrix Reasoning subscores.

Digit Span (DS) and Letter-Number Sequencing (LNS) are subtests of the Wechsler Adult Intelligence Scale—Third Edition (WAIS-III).[63] The DS task evaluates attention and working memory and requires the repetition of sequences of digits of increasing length forward and backwards. Both DS subtests (i.e., forward and backwards) consist of seven random number sequences that the examiner reads aloud at the rate of one per second. The LNS assesses processing speed and verbal working memory.[64,65] It requires listening to an auditory presentation of alternating numbers and letters and

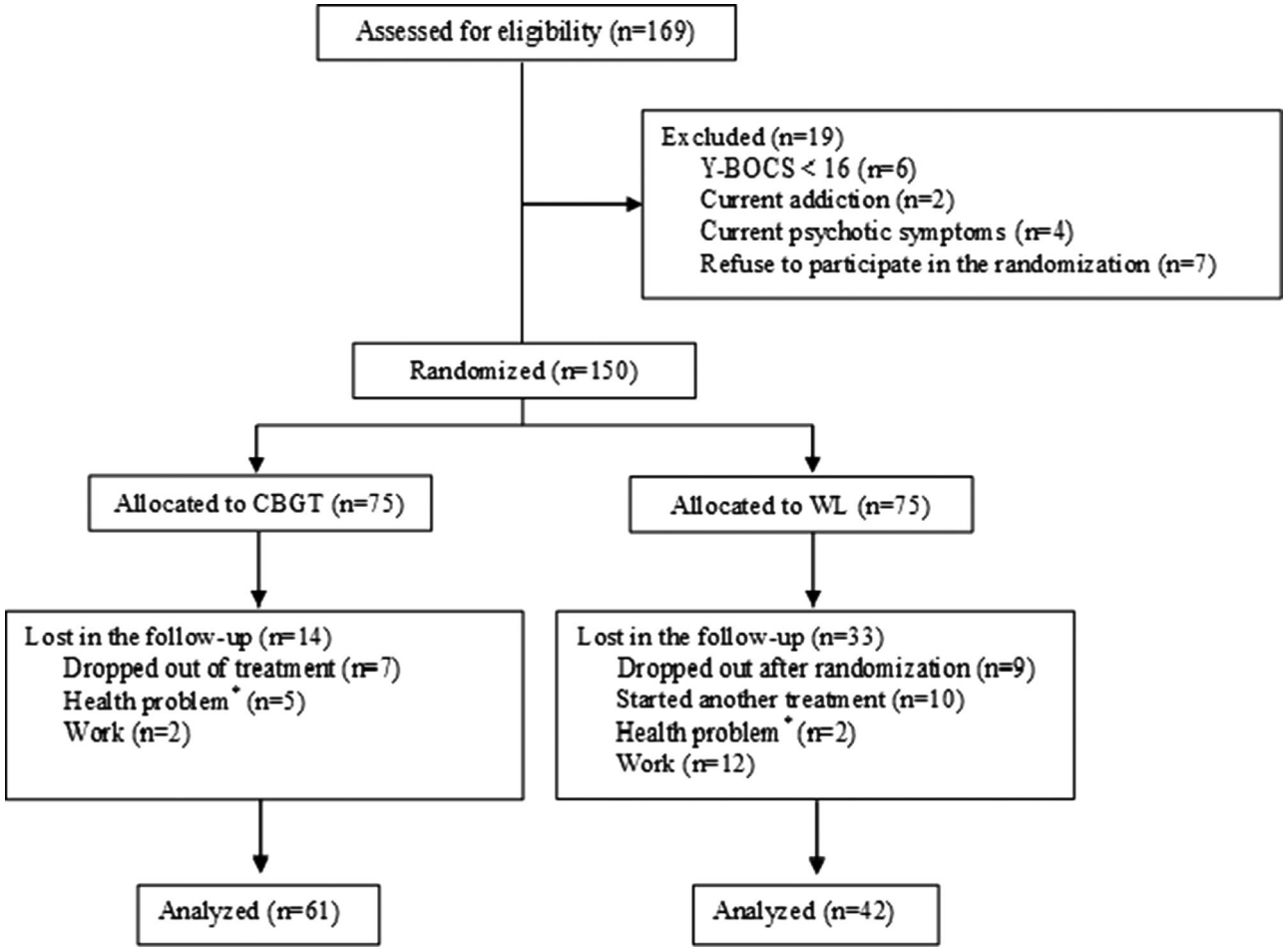

Figure 1. Flow diagram of the progress through the phases of a parallel randomized trial of two groups (i.e., enrollment, intervention allocation, follow-up, and data analysis).
***Fall, infarct.**

repeating back the numbers in ascending order, followed by the letters alphabetically.

The *Logical Memory AB (LM)* is a subtest of Wechsler Memory Scale III[66] that assesses auditory verbal memory. Participants are requested to remember and reproduce two short stories, both immediately after these are read to them and then after a 30 min interval.

The *Brief Visuospatial Memory Test—Revised* (BVMT-R)[67] is a test of visuospatial memory that consists of three learning trials in which six abstract designs are presented for 10 s. A learning score is calculated by summing all designs recalled across the three encoding trials (maximum score of 36). Delayed recall of the designs takes place after a 25-min delay. Subsequently, a recognition trial is administered, where subjects are requested to recognize six core stimuli of 12 designs (maximum score 6).

The *Rey-Osterrieth Complex Figure Test* (RCFT)[68] is a test of nonverbal memory. To assess organizational ability, we used the organizational strategy used to copy the figure.[69] Participants can obtain six points for different structural elements (central square, vertical and horizontal lines, central cross and final angle) drawn during the copying task.

The *Stroop Color and Word Test (Stroop)*,[70] was used to measure selective attention, mental flexibility, and inhibitory control. In this test, patients must inhibit an automatic response (read the word) and make a controlled response (to say the color of the word). The Charles Golden version of the Stroop test includes three trials. This test includes three classic trials: the Word Trial, in which participants are asked to read as many words out of 100 color words printed in black ink within 45 s; the Color Trial, in which participants are requested to name as many colors out of 100 color hues printed as "XXXX" within 45 s; and the Color-Word Incongruent Trial, in which participants are requested to name as many hues as possible out of 100 color hues printed in competing color words within 45 s. The interference score is produced by subtracting the Incongruent Trial score from the Congruent score using the Charles Golden formula.

The *Trail Making Tests A and B (TMT)*[71] were used to assess grapho-motor information processing speed, attention, and mental flexibility. It requires participants to connect 25 encircled numbers randomly arranged on a page in proper order (Part A) and 25 encircled numbers and letters in alternating order (Part B). The score for each version (A and B) corresponds to the number of seconds taken for completing the task. The executive function score was obtained on the basis of the differences between TMT-B and TMT-A times (TMT B–A).

PROCEDURE

Eligible participants were interviewed and completed the clinical and neuropsychological assessment in one session, with a mean session duration of 3.5 hr (range 3–4 hr). All interviewers had been rigorously trained in the use of diagnostic instruments in order to guarantee satisfactory inter-rater reliability.[72] Judgments made by the reviewers were comparable 96% of the time. The neuropsychological assessment was administered individually by four masters- and Ph.D.-level trained psychologists who were blinded to group assignment. A standardized protocol of neuropsychological assessment and a guide to the applicator were made. The application of neuropsychological tests followed a set presentation order. The interpretation of the results was made by the same psychologists, but in pairs to ensure consistency of interpretation.

STATISTICAL ANALYSIS

Statistical analyses were performed using the Statistical Package for Social Sciences (SPSS) for Windows, version 16.0.[73] To examine differences on continuous clinical and demographic variables between the groups at baseline, *t*-tests for independent samples were used. The generalized estimating equations (GEE)[74] was used to verify the mean differences within and between groups (i.e., CBGT and WL or responders and nonresponders). This analysis was adjusted by baseline Color-Word Stroop, Backward and Total DS when comparing CBGT and WL groups. Scores were also adjusted by baseline TMT trial B and TMT trial B-A when comparing CBGT responders and nonresponders. In this type of analysis, the Wald chi-square (χ^2) is used to test all variables, for both continuous and categorical parameters. The comparison between groups before and after 12 weeks of treatment was performed using pairwise comparisons with Bonferroni adjustment. Variables with asymmetric distribution, as detected by the Kolmogorov–Smirnov test, were submitted to logarithmic transformation and then analyzed using parametric tests.

Multiple Linear Regression analysis was used to adjust for the effects of possible confounding variables associated with neuropsychological functioning, as well as to verify the accountability of neuropsychological functioning for the variation of Y-BOCS scores posttreatment. Pearson correlation coefficient was used to verify the association between the changes in neuropsychological scores and the percentage of improvement in response to treatment in the CBGT group. Pearson's χ^2 was used to compare categorical variables between groups. Yates χ^2 test was used for dichotomous data (responders vs. nonresponders). We employed a conservative criterion for improvement of ≥35% on the Y-BOCS and CGI-S ≤ 2 at end point. This analysis was adjusted by age, age of onset of obsessive–compulsive symptoms, and education in years. The intention to treat analysis was not employed because we focused on the relationship between treatment and neuropsychological function. In order to correct for family-wise inflation of type I error, we employed a conservative adjustment for multiple comparisons where a significance threshold of 0.01 was set throughout the study.

RESULTS

LOST AT FOLLOW-UP

In the present study, 31.3% of the participants were lost at follow-up, the majority of which were randomized to the WL group ($n = 33$) instead of the treatment group ($n = 14$). No significant differences were observed at baseline between study completers and noncompleters in terms of gender, age, employment status, marital status, laterality, duration of OC symptoms, years of education, socioeconomic status, functional assessment staging (FAST), severity of depression (BDI) or anxiety (BAI) symptoms, the scores assessing OCD severity (Y-BOCS and CGI) or comorbidity (e.g., unipolar depression, bipolar disorder, anxiety disorders). However, a significant difference between groups regarding age at onset was observed, where the mean age at onset among noncompleters ($M = 11.5$ years, $SD = 5.8$) was significantly lower compared to completers ($M = 14$ years, $SD = 9.6$; $t(1) = 3.63$, $P = .044$).

SOCIODEMOGRAPHIC AND CLINICAL FEATURES (BASELINE DATA)

One hundred three participants completed the study (68.7%). Sixty-nine (67%) female and 34 (33%) male participants with OCD were included. Our analysis revealed no significant effect of gender on either the percentage treatment response rate to CBGT ($P = 0.791$) or neuropsychological test performance (P range, .119–.898).

The mean age of the entire sample was 42.6 years ($SD = 14.1$). Patients had obsessive–compulsive (OC) symptoms for a mean period of 28.5 years ($SD = 13.4$). The mean age at onset was 14.1 years ($SD = 9.6$). Although 32 patients (52.5%) in the treatment group and 17 (40.5%) in the control group were medicated with SRIs and mood stabilizers, the doses were stable for at least 3 months ($M = 24.1$ months) before starting the CBGT. In addition, within the CBGT group eight participants (13.1%) were medicated with benzodiazepines, and eight participants (13.1%) with antipsychotics. Among the WL group, 12 participants (28.6%) were medicated with benzodiazepines, and five participants were medicated with antipsychotics (11.9%). Chi-squared analysis revealed that there was no significant difference in the response rate between medicated ($n = 32$; response rate = 53.6%) and unmedicated ($n = 29$; response rate = 47.8%) participants within the CBGT group ($\chi^2 = 1.87$; $P = 0.393$) and the WL group: medicated ($n = 17$; response rate = 4.7%) and unmedicated ($n = 25$; response rate = 11.9%; $\chi^2 = 2.29$; $P = 0.318$). Furthermore, comparing performance across neuropsychological tests between medicated and unmedicated participants yielded a single difference on one outcome measure, namely the RCFT memory trial ($t = 2.2$; $P = 0.033$). However, this P value did not survive correction for multiple comparisons and was therefore deemed not significant.

In terms of past treatments, 33 participants from the CBGT group (54.1%) and 30 participants from the control group (48.8%), reported receiving treatment for OCD in the past. There was no difference in the response rate between those who had undergone prior psychotherapeutic treatment versus those who were not treated in the past ($\chi^2 = 0.13$; $P = 0.935$). Eighty (77.7%) showed comorbidity with any disorder; 70 (68.%) with depressive disorders (major depressive disorder and dysthymia); 18 (17.5%) with bipolar disorders (bipolar I and II disorders and cyclothymic disorder); 53 (51.5%)

with anxiety disorders (specific and social phobia, panic disorder, generalized anxiety disorder, posttraumatic stress disorder); 27 (26.2%) with obsessive–compulsive related disorders (body dysmorphic disorder, trichotillomania, skin picking disorder), and 8 (7.8%) with tic disorders (chronic tic disorder and Tourette's syndrome). Within the CBGT group, no significant difference in treatment response was found between participants with a diagnosed comorbid condition ($n = 33$, 67.34%), and individuals diagnosed solely with OCD ($n = 10$, 83.33%: Yates' $\chi^2 = 0.54$; $P = 0.462$). For the present study, we excluded comorbid neurological conditions. Given that tic disorders are considered neuropsychiatric conditions, we have assessed the impact of tic disorders on treatment response, and neuropsychological test performance. No significant differential response to treatment ($\chi^2 = 1.70$; $P = 0.428$) was found between participants with comorbid tic disorder or Tourette's syndrome ($n = 8$) and those without comorbid tic disorder or Tourette's syndrome ($n = 95$). Furthermore, Independent Samples *t*-test revealed that there was no significant difference in neuropsychological test performance between participants who had comorbid tic disorder and/or Tourette's syndrome and participants without such comorbidities: WASI: Verbal IQ ($P = 0.803$), Performance IQ ($P = 0.207$); Total IQ ($P = 0.571$); BVMT: Total Recall ($P = 0.751$); Total Learning ($P = 0.532$); RCFT: Copy ($P = 0.682$), Memory ($P = 0.872$), Organization ($P = 0.461$); LM AB: Immediate Recall ($P = 0.475$), Late Recall ($P = 0.229$); TMT: trial A ($P = 0.968$), trial B ($P = 0.404$), trial B-A ($P = 0.609$); Stroop: Color-Word (incongruent; $P = 0.364$), Interference ($P = 0.405$); DS Total ($P = 0.841$) and LNS ($P = 0.756$).

Notably, the prevalence of medicated patients between the groups did not differ and was not modified during treatment. Furthermore, as presented in Table 1, the CBGT and WL groups did not differ on any demographic or clinical factors.

NEUROPSYCHOLOGICAL MEASURES (BASELINE DATA)

Significant differences were observed between the CBGT and WL groups at baseline on the Stroop Incongruent trial ($t(100) = 2.829$; $P = 0.006$), indicating reduced processing speed among the CBGT group, and on the DS Backward ($t(101) = 3.012$; $P = 0.003$), which assesses working memory (see Table 2), indicating reduced performance on this task among the WL group. No significant differences were observed between the groups on all other outcome measures at baseline.

CLINICAL AND NEUROPSYCHOLOGICAL COMPARISON BETWEEN CBGT AND WL CONTROL GROUPS AT PRE- AND POSTTREATMENT

GEE revealed a significant difference in symptom reduction between treated patients and controls on the Y-BOCS Obsessions subscale ($\chi^2(1) = 53.58$; $P < .001$), Y-BOCS Compulsions subscale ($\chi^2(1) = 43.51$; $P < .001$), Y-BOCS total score ($\chi^2(1) = 73.99$; $P < .001$), CGI-S ($\chi^2(1) = 124.38$; $P < .001$), BAI ($\chi^2(1) = 6.05$; $P = 0.003$), and BDI ($\chi^2(1) = 7.26$; $P = 0.005$), indicating a larger reduction among the CBGT group (see Table 2).

No significant difference between the pre- and posttreatment performance was found between the groups on the WASI's Vocabulary, Block Design, Similarities, Matrix Reasoning, Verbal IQ, Performance IQ, Total IQ; BVMT-R's Total Recall, Total Learning, Delayed Recall, and Recognition Hits; RCFT Copy; RCFT Immediate Memory and Organization; LM Immediate and Delayed Recall; TMT parts A and B and B-A; Stroop Color-Word and Interference; DS Forward, Backward, and Total; and LNS and FAS (see Table 2).

ASSOCIATION BETWEEN NEUROPSYCHOLOGICAL FUNCTION AND THE CLINICAL OUTCOME FOR CBGT

The variables for age, years of education, and age at onset of obsessive–compulsive symptoms were controlled in a multiple regression model in order to be linearly related to T1 and T2-1 scores. To examine whether neuropsychological performance at baseline was predictive of response to CBGT, we compared treatment responders and nonresponders on CBGT. Responders displayed better performance before the intervention in set shifting (over and above processing speed) as measured by the TMT B ($F_{57} = 16.12$; $P = .025$) and TMT B-A ($F_{57} = 13.5$; $P = .011$), suggesting that these functions may predict response to CBGT. The TMT B model was responsible for 16.5% of the variation in Y-BOCS total scores posttreatment and showed a weak but significant partial association, independent of the other variables (adjusted $\beta = 0.211$; $P = .035$). Likewise, the TMT B-A model was responsible for 17.5% of the variation in Y-BOCS scores posttreatment and showed a weak association, direct and independent of the other variables (years of education, age at onset of obsessive–compulsive symptoms and age; adjusted $\beta = 0.254$; $P = 0.011$). No significant differences were observed at baseline between the responders and nonresponders regarding other neuropsychological measures (Table 3).

COMPARISON BETWEEN RESPONDERS AND NONRESPONDERS TO CBGT: PRE- AND POSTTREATMENT

Among the CBGT group, 72.1% responded favorably to treatment (using the conservative definition of improvement: a reduction > 35% on the Y-BOCS and CGI-S < 2). The comparison between treatment responders and nonresponders for the TMT-B and TMT B-A was adjusted to TMT-B and TMT B-A baseline scores. As presented in Table 4., there were no significant differences in other neuropsychological domains: BVMT-R's Total Recall ($\chi^2(1) = 0.06$; $P = 0.811$), Total Learning ($\chi^2(1)$

TABLE 1. Sociodemographic and clinical features: comparing CBGT group and WL group ($n = 103$)

	CBGT group ($n = 61$)	WL group ($n = 42$)	Statistics	p
Age in years, mean (*SD*)	43.4 (14.0)	41.4 (14.3)	$t(101) = 0.736$	0.463
Gender, *n* (%)				
Male	23 (37.7)	11 (26.2)	$\chi^2(1)\text{Yates} = 1.016$	0.313
Female	38 (62.3)	31 (73.8)		
Marital status, n (%)				
Married	38 (62.3)	21 (50.0)	$\chi^2(3) = 3.996$	0.262
Single	16 (26.2)	14 (33.3)		
Widowed	0 (0)	2 (4.8)		
Divorced/separated	7 (11.5)	5 (11.9)		
Education in years, mean (*SD*)	14.2 (4.1)	13.7 (3.3)	$t(100) = 0.602$	0.548
Currently employed, n (%)	24 (39.3)	15 (35.7)	$\chi^2(1)\text{Yates} = 0.028$	0.868
Laterality (right-handed), *n* (%)	54 (88.5)	41 (97.6)	$\chi^2(1)\text{Yates} = 2.872$	0.147
Age at onset, mean (*SD*)	14.2 (10.2)	13.9 (8.5)	$t(101) = 0.195$	0.846
Duration of OC symptoms, mean (*SD*)	29.2 (12.7)	27.5 (14.5)	$t(101) = 0.634$	0.527
Currently medicated, *n* (%)	32 (52.5)	17 (40.5)	$\chi^2(1)\text{Yates} = 0.992$	0.319
FAST, mean (*SD*)	25.0 (11.5)	21.9 (11.3)	$t(98) = 1.339$	0.184
Severity of symptoms				
BAI, mean (*SD*)	18.2 (10.0)	19.4 (11.4)	$t(98) = -0.562$	0.575
BDI, mean (*SD*)	18.0 (8.9)	18.9 (9.9)	$t(98) = -0.465$	0.643
Y-BOCS				
Obsessions, mean (*SD*)	13.5 (3.1)	12.6 (3.1)	$t(101) = 1.439$	0.153
Compulsions, mean (*SD*)	14.0 (3.4)	13.6 (3.0)	$t(101) = 0.475$	0.636
Total, mean (*SD*)	27.4 (5.8)	26.2 (5.0)	$t(101) = 1.091$	0.278
CGI-S (OCD)	5.1 (0.8)	4.9 (1.0)	$t(99) = 1.255$	0.213
Life time comorbidity, *n* (%)				
Depressive disorders	45 (73.8)	25 (59.5)	$\chi^2(1)\text{Yates} = 1.710$	0.191
Bipolar disorders	8 (13.1)	10 (23.8)	$\chi^2(1)\text{Yates} = 1.301$	0.254
Anxiety disorders	31 (50.8)	22 (52.4)	$\chi^2(1)\text{Yates} = 0.000$	1.000
Other disorders	16 (26.2)	11 (26.2)	$\chi^2(1)\text{Yates} = 0.000$	1.000
Socioeconomic levels, *n* (%)				
Class A	5 (8.2)	1 (2.4)	$\chi^2(3) = 3.291$	0.349
Class B	28 (45.9)	15 (35.7)		
Class C	24 (39.3)	22 (52.4)		
Class D or lower	4 (6.5)	4 (9.5)		

CBGT, cognitive-behavioral group therapy; WL, wait list; FAST, Functioning Assessment Short Test; BAI, Beck Anxiety Inventory; BDI, Beck Depression Inventory; Y-BOCS, Yale-Brown Obsessive–Compulsive Scale; CGI-S, Clinical Global Impression—Severity Scale, depressive disorders (major depressive disorder and dysthymia), bipolar disorders (bipolar I and II disorders and cyclothymic disorder), anxiety disorders (specific and social phobia, panic disorder, generalized anxiety disorder, posttraumatic stress disorder), other disorders (body dysmorphic disorder, trichotillomania, skin picking disorder); *n*, sample; *SD*, stander deviation; *t*, independent samples test; χ^2, Pearson chi-square.

= 0.39; $P = 0.539$), Delayed Recall ($\chi^2(1) = 0.82$; $P = 0.364$), and Recognition Hits ($\chi^2(1) = 0.11$; $P = 0.741$); RCFT Copy ($\chi^2(1) = 0.02$; $P = 0.884$); RCFT Memory ($\chi^2(1) = 3.64$; $P = 0.057$) and Organization Strategies (RCFT; $\chi^2(1) = 4.36$; $P = 0.037$); LM AB Immediate ($\chi^2(1) = 0.05$; $P = 0.817$) and Delayed Recall ($\chi^2(1) = 0.23$; $P = 0.630$); TMT-A ($\chi^2(1) = 4.32$; $P = 0.038$); TMT-B ($\chi^2(1) = 1.27$; $P = 0.260$) and TMT B-A ($\chi^2(1) = 3.61$; $P = 0.057$); Stroop Color-Word ($\chi^2(1) = 0.03$; $P = 0.765$) and Interference ($\chi^2(1) = 1.16$; $P = 0.281$); DS Forward ($\chi^2(1) = 0.01$; $P = 0.562$), Backward ($\chi^2(1) = 1.37$; $P = 0.241$), and Total ($\chi^2(1) = 0.34$; $P = 0.923$); and LNS ($\chi^2(1) = 1.81$; $P = 0.179$).

The Pearson correlation coefficient was used in 20% of the sample with "pure OCD" to verify the association between the changes in neuropsychological scores and the percentage of improvement in response to treatment within the CBGT group. Only one positive correlation was observed. Patients in the CBGT group who exhibited a higher percentage of improvement also had a better percentage performance change in processing speed and verbal working memory by LNS test ($r = .434$; $P = .049$).

The Pearson correlation coefficient was used to verify the association between the changes in neuropsychological scores and the percentage of improvement in response to treatment in the CBGT group. One significant inverse correlation was observed concerning the RCFT organization score ($r = -.272$; $P = .037$) where better response to treatment was associated with poorer RCFT copy organizational score. In addition, a positive correlation was observed between response to treatment and performance on the immediate logical memory test ($r = .343$; $P = 0.007$; Table 5).

TABLE 2. Clinical and neuropsychological comparison between CBGT group and WL group at pre- and posttreatment ($n = 103$)

	CBGT group ($n = 61$)				WL Group (n = 42)				
	Pre Mean (*SE*)	Post Mean (*SE*)	p^1	Dif (CI95%)	Pre Mean (*SE*)	Post Mean (*SE*)	p^1	Dif (CI95%)	p^2
Y-BOCS									
Obsessions	13.5 (0.39)	7.24 (0.55)	<0.001	6.23 (5.3 to 7.2)	12.6 (0.47)	11.1 (0.56)	<0.001	1.52 (0.7 to 2.4)	<0.001
Compulsions	13.2 (0.42)	6.70 (0.60)	<0.001	6.50 (5.4 to 7.6)	12.6 (0.52)	11.3 (0.60)	0.023	1.36 (0.2 to 2.5)	<0.001
Total	27.3 (0.72)	14.1 (1.08)	<0.001	13.2 (11.5 to 14.9)	26.0 (0.74)	23.5 (0.98)	0.005	2.53 (0.8 to 4.3)	<0.001
CGI-S	4.96 (0.10)	2.45 (0.17)	<0.001	2.51 (2.2 to 2.8)	5.23 (0.14)	4.86 (0.17)	0.006	0.37 (0.1 to 0.6)	<0.001
BAI	18.2 (1.28)	11.1 (1.38)	<0.001	7.1 (3.0 to 11.1)	19.4 (1.77)	19.4 (2.10)	1.000	0.0 (−4.8 to 4.9)	0.003
BDI	17.9 (1.14)	11.3 (1.23)	<0.001	6.64 (3.5 to 9.8)	18.9 (1.55)	18.2 (2.16)	1.000	0.7 (−3.9 to 5.3)	0.005
WASI									
Vocabulary	51.1 (1.41)	49.5 (1.35)	0.274	1.61 (−1.3 to 4.5)	48.8 (1.78)	49.6 (1.51)	0.621	−0.80 (−3.9 to 2.4)	0.272
Block Design	32.5 (1.75)	37.4 (1.70)	<0.001	−4.94 (−7.1 to −2.8)	41.8 (2.41)	44.2 (2.38)	0.066	−2.35 (−4.9 to 0.2)	0.126
Similarities	33.5 (0.92)	34.8 (0.68)	0.142	−1.25 (−2.9 to 0.4)	32.9 (0.84)	35.1 (0.94)	0.001	−2.17 (−3.4 to −0.9)	0.385
Matrix Reasoning	20.9 (1.04)	22.2 (0.94)	0.077	−1.18 (−2.5 to 0.1)	23.7 (1.39)	24.6 (1.26)	0.194	−0.98 (−2.5 to 0.5)	0.841
Verbal IQ	105.6 (1.65)	105.6 (1.25)	1.000	0.00 (−4.3 to 4.3)	103.5 (1.71)	105.8 (1.61)	0.545	−2.3 (−5.9 to 1.3)	0.278
Performance IQ	103.1 (1.86)	106.3 (1.77)	0.008	−3.25 (−5.7 to −0.8)	99.6 (2.45)	102.3 (2.47)	0.010	−2.73 (−4.8 to −0.6)	0.752
Total IQ	104.8 (1.65)	106.8 (1.37)	0.074	−2.02 (−4.2 to 0.2)	102.0 (1.98)	104.6 (1.97)	0.004	−2.61 (−4.4 to −0.8)	0.682
BVMT-R									
Total Recall	19.6 (1.00)	22.3 (0.98)	0.001	−2.69 (−4.3 to −1.1)	19.8 (1.23)	23.3 (1.19)	<0.001	−3.53 (−5.5 to −1.6)	0.520
Total Learning	4.19 (0.30)	3.22 (0.31)	0.011	0.98 (0.2 to 1.7)	4.24 (0.32)	3.88 (2.34)	0.361	0.36 (−0.4 to 1.1)	0.260
Delayed Recall	8.53 (0.41)	8.98 (0.39)	0.194	0.45 (−1.1 to 0.2)	7.83 (0.46)	9.34 (0.43)	<0.001	−1.51 (−2.4 to −0.7)	0.056
Recognition Hits	5.27 (0.15)	5.46 (0.10)	0.200	−0.19 (−0.5 to 0.1)	5.33 (0.17)	5.32 (0.18)	0.936	0.02 (−0.4 to 0.4)	0.410
RCFT									
Copy	32.2 (0.66)	33.4 (0.50)	0.089	−1.2 (−2.6 to 0.2)	31.7 (0.85)	32.2 (0.85)	0.552	−0.5 (−2.2 to 1.2)	0.530
Memory	15.0 (0.94)	19.0 (1.01)	<0.001	−4.0 (−5.4 to −2.5)	16.3 (1.1)	17.2 (1.31)	0.384	−0.9 (−3.0 to 1.2)	0.018
Organization	3.18 (0.24)	3.82 (1.24)	0.040	0.6 (−1.3 to −0.03)	3.74 (0.28)	4.10 (0.28)	0.263	−0.4 (−1.0 to 0.3)	0.537
LM AB									
Immediate Recall	23.1 (0.87)	24.3 (0.90)	0.158	−1.2 (−2.9 to 0.5)	21.6 (1.19)	22.3 (1.12)	0.399	−0.7 (−2.5 to 1.0)	0.707
Late Recall	19.3 (0.85)	21.2 (0.92)	0.004	−1.9 (−3.3 to −0.6)	18.0 (1.21)	19.6 (1.11)	0.067	−1.7 (−3.4 to 0.1)	0.813
TMT									
Trial A	44.4 (1.90)	43.0 (3.65)	0.693	−1.4 (−5.6 to 8.4)	45.6 (4.04)	49.3 (4.53)	0.273	−3.7 (−10.3 to 2.9)	0.297
Trial B	100.3 (7.71)	94.8 (8.54)	0.513	5.5(−11.0 to 22.0)	111.7 (12.6)	103.3 (8.87)	0.419	8.4 (−12.0 to 28.8)	0.829
Trial B-A	56.2 (6.89)	57.3 (8.12)	0.879	−1.2 (−16.3 to 13.9)	65.1 (9.38)	48.1 (5.52)	0.067	18.4 (−1.3 to 38.0)	0.122
Stroop									
Color Word (incongruent)	39.6 (1.60)	39.1 (0.88)	1.000	0.5 (−3.1 to 4.0)	32.6 (1.57)	36.4 (1.25)	0.029	−3.8 (−7.4 to −0.2)	0.026
Interference	1.23 (1.00)	3.35 (1.61)	0.107	−2.1 (−4.7 to 0.5)	−3.32 (1.25)	1.48 (1.44)	0.003	−4.8 (−7.9 to −1.7)	0.195
DS									
DS Forward	8.8 (0.33)	9.10 (0.33)	0.241	−0.3 (−0.8 to 0.2)	7.86 (0.31)	8.13 (0.34)	0.405	−0.3 (−1.9 to 0.4)	0.944
DS Backward	6.48 (031)	6.30 (0.32)	0.502	0.2 (−0.3 to 0.7)	5.12 (0.31)	5.38 (0.32)	0.403	−0.3 (−0.9 to 0.3)	0.284
Total	15.3 (0.58)	15.5 (0.63)	0.562	−0.3 (−1.1 to 0.6)	13.0 (0.51)	13.5 (0.59)	0.327	−0.5 (−1.6 to 0.5)	0.697
LNS	9.29 (0.41)	9.80 (0.46)	0.105	−0.5 (−1.1 to 0.1)	8.17 (0.39)	8.48 (0.43)	0.398	−0.3 (−1.0 to 0.4)	0.682

CBGT, cognitive-behavioral group therapy; WL, wait list; Y-BOCS, Yale-Brown Obsessive–Compulsive Scale; CGI-S, Clinical Global Impression—Severity Scale; BAI, Beck Anxiety Inventory; BDI, Beck Depression Inventory; WASI, Wechsler Abbreviated Scale of Intelligence; BVMT-R, Brief Visuospatial Memory Test—Revised; RCFT, Rey Complex Figure Test; LM AB, Logical Memory AB; TMT, trial making test; Stroop, Stroop Color and Word Test; DS, Digit Span; LNS, Letter-Number Sequencing; *SE*, standard error; p^1, pairwise comparisons with Bonferroni adjustment; p^2, generalized estimating equations.
All analyses were adjusted by baseline Color-Word Stroop, Backward, and Total DS.

DISCUSSION

The results of the present study show that CBGT is an effective treatment for OCD, with 72.1% of patients responding favorably to treatment. In addition, a significant improvement was observed on all clinical and functional measures. These results are in accordance with other reports employing CBGT for OCD.[41,42,75,76]

The first aim of this study was to evaluate whether CBGT could improve neuropsychological functioning among OCD patients. Our results demonstrate that the CBGT group did not exhibit performance improvement across cognitive domains (i.e., executive functions, Performance IQ, learning and memory, organization, and Late Recall) after 3 months of treatment. Only Muscatello et al. (2011)[29] observed that attentional interf

TABLE 3. Neuropsychological domains of responders and nonresponders at baseline assessment ($n = 61$)

	Responders ($n = 44$) Mean (*SD*)	Nonresponders ($n = 17$) Mean (*SD*)	*T*	*p*
WASI				
Vocabulary	50.6 (9.78)	52.4 (14.4)	0.56	0.577
Block Design	36.8 (13.6)	36.2 (15.7)	−0.15	0.879
Similarities	33.9 (6.12)	32.5 (9.68)	−0.55	0.590
Matrix	23.3 (6.90)	21.7 (10.5)	0.30	0.768
Reasoning				
Verbal IQ	105.2 (9.74)	106.7 (19.4)	−0.60	0.557
Performance	103.3 (11.8)	102.6 (20.7)	−0.12	0.908
IQ				
Total IQ	104.7 (9.83)	105.0 (19.4)	0.06	0.949
BVMT-R				
Total Recall	19.5 (7.89)	19.8 (7.91)	0.13	0.899
Total Learning	4.02 (1.91)	4.65 (3.28)	0.74	0.782#
Delayed Recall	8.51 (2.90)	8.59 (3.87)	0.08	0.934
Recognition	5.23 (1.15)	5.35 (1.22)	0.36	0.721
Hits				
RCFT				
Copy	32.5 (5.45)	31.4 (4.42)	−0.80	0.429
Memory	14.9 (7.57)	15.4 (7.14)	0.23	0.646#
Organization	2.91 (1.87)	3.88 (1.87)	0.23	0.103#
LM AB				
Immediate	23.6 (6.41)	21.9 (7.89)	−0.83	0.408
Recall				
Late Recall	19.6 (6.91)	18.4 (6.20)	−0.66	0.514
TMT				
Trail A	44.4 (13.5)	44.5 (18.1)	0.02	0.981
Trail B	89.8 (35.4)	128.7 (95.3)	1.59	0.025#§
Trail B-A	45.5 (32.7)	84.8 (82.6)	1.85	0.011#§
Stroop				
Color-Word	40.1 (12.6)	38.3 (12.4)	−0.47	0.639
Interference	1.09 (7.10)	1.61 (9.84)	0.23	0.713#
DS				
DS Forward	8.68 (2.45)	9.12 (3.00)	0.59	0.561
DS Backward	6.52 (2.12)	6.35 (3.08)	−0.25	0.807
Total	15.2 (4.04)	15.5 (5.82)	0.20	0.840
LNS	9.16 (3.07)	9.65 (3.74)	0.52	0.603

CBGT, cognitive-behavioral group therapy; WASI, Wechsler Abbreviated Scale of Intelligence; *SD*, standard deviation; *T*, Independent Samples Test; *p*, Independent Samples Test; BVMT-R, Brief Visuospatial Memory Test—Revised; RCFT, Rey Complex Figure Test; LM AB, Logical Memory AB; TMT, trail making test; Stroop, Stroop Color and Word Test; DS, Digit Span; LNS, Letter-Number Sequencing; #, nonparametric variable (Mann–Whitney test)

§, adjusted by education in years, age at onset of obsessive–compulsive symptoms and age.

erence stimuli—as measured by the Stroop test and perseverative errors on the WCST—significantly improved after a 16-week treatment with aripiprazole. Park et al. (2006)[30] conducted a study testing a cognitive training program for OCD patients, focusing on the improvement of organizational cognitive strategies. They concluded that the training program improved patients' memory function and clinical symptoms. Other treatment modalities have not yet been tested in OCD, but have had positive results for schizophrenia,[30] including the improvement of neuropsychological domains such as verbal and nonverbal memory, and executive function when compared to CBT; one such example is cognitive remediation therapy (CRT).[77]

Although the difference was not statistically significant, our results indicate a trend wherein patients with OCD who responded to CBGT displayed greater post-treatment improvement in organization strategy on the RCFT and attention and executive function as measured by the TMT than did nonresponders, suggesting that the neuropsychological test performance in these domains could be associated with symptom severity. Vandborg et al. (2014)[78] observed that patients with OCD performed worse on the RCFT compared to healthy controls both before and after CBT. They concluded

TABLE 4. Comparison between nonresponder and responder patients to CBGT: pre- and posttreatment ($n = 61$)

	Responders ($n = 44$)				Non responders ($n = 17$)				
	Pre Mean (*SE*)	Post Mean (*SE*)	p^1	Dif (CI 95%)	Pre Mean (*SE*)	Post Mean (*SE*)	p^1	Dif (CI 95%)	p^2
WASI									
Vocabulary	50.6 (1.46)	49.3 (1.50)	0.350	1.34 (−1.5 to 4.2)	52.4 (3.38)	49.9 (2.98)	0.492	2.53 (−4.7 to 9.7)	0.763
Block Design	36.8 (2.03)	41.8 (1.70)	<0.001	−5.02 (−7.7 to −2.3)	36.2 (3.69)	38.5 (4.11)	0.129	−2.29 (−5.3 to 0.7)	0.183
Similarities	33.9 (0.91)	35.3 (0.72)	0.176	−1.36 (−3.3 to 0.6)	32.5 (2.28)	33.5 (1.55)	0.504	−1.01 (−3.9 to 1.9)	0.845
Matrix Reasoning	23.3 (1.03)	24.7 (0.98)	0.026	−1.32 (−2.5 to −0.2)	21.7 (2.46)	23.8 (1.93)	0.245	−2.11 (−5.7 to 1.5)	0.678
Verbal IQ	105.2 (1.45)	106.2 (1.41)	0.556	−1.00 (−4.3 to 2.3)	106.7 (4.57)	104.1 (2.57)	0.488	2.59 (−4.7 to 9.9)	0.382
Performance IQ	103.3 (1.76)	107.3 (1.85)	<0.001	−4.02 (−6.2 to −1.8)	102.6 (4.88)	103.9 (4.08)	0.706	−1.24 (−7.7 to 5.2)	0.421
Total IQ	104.7 (1.47)	107.6 (1.43)	0.010	−2.93 (−5.2 to −0.7)	105.0 (4.57)	104.6 (3.16)	0.896	0.35 (−4.9 to 5.6)	0.261
BVMT-R									
Total Recall	19.3 (1.10)	22.5 (1.10)	0.002	−3.20 (−5.2 to −1.2)	19.9 (1.08)	22.8 (1.05)	<0.001	−2.89 (−4.4 to −1.4)	0.811
Total Learning	4.15 (0.28)	3.23 (0.35)	0.021	0.91 (0.1 to 1.7)	4.27 (0.33)	3.70 (0.31)	0.148	0.57 (−0.2 to 1.3)	0.539
Delayed Recall	8.45 (0.41)	9.06 (0.43)	0.126	−0.62 (−1.4 to 0.2)	8.07 (0.45)	9.19 (0.39)	0.003	−1.12 (−1.8 to −0.4)	0.364
Recognition Hits	5.23 (0.16)	5.38 (0.12)	0.357	−0.15 (−0.5 to 0.2)	5.35 (0.15)	5.42 (0.14)	0.696	−0.07 (−0.4 to 0.3)	0.741
RCFT									
Copy	32.5 (0.75)	33.5 (0.56)	0.241	−1.01 (−2.7 to 0.7)	31.5 (0.72)	32.4 (0.69)	0.219	−0.85 (−2.2 to 0.5)	0.884
Memory	15.4 (1.08)	19.4 (1.12)	<0.001	−4.00 (−5.7 to −2.3)	15.6 (0.99)	17.2 (1.13)	0.061	−1.64 (−3.4 to 0.1)	0.057
Organization	3.04 (0.27)	4.06 (0.26)	0.002	−1.02 (−1.7 to −0.4)	3.73 (0.24)	3.81 (0.26)	0.779	−0.08 (−0.7 to 0.5)	0.037
LM AB									
Immediate Recall	23.7 (0.91)	24.6 (0.95)	0.405	−0.85 (−2.9 to 1.2)	21.4 (1.04)	22.6 (1.02)	0.115	−1.15 (−2.6 to 0.3)	0.817
Late Recall	19.9 (0.96)	21.5 (1.02)	0.070	−1.54 (−3.2 to 1.2)	17.7 (1.00)	19.8 (0.98)	0.003	−2.07 (−3.4 to −0.7)	0.630
TMT									
Trails A	44.4 (1.95)	39.7 (1.84)	0.027	4.67 (0.5 to 8.8)	45.4 (3.34)	50.9 (5.11)	0.213	−5.47 (−14.1 to 3.1)	0.038
Trails B	90.3 (5.04)	91.2 (8.11)	0.914	−0.92 (−17.5 to 15.7)	118.3 (12.0)	104.6 (9.3)	0.163	13.7 (−5.5 to 32.9)	0.260
Trails B-A	46.0 (4.57)	51.5 (7.82)	0.534	−5.46 (−22.7 to 11.8)	73.4 (9.55)	55.7 (7.43)	0.036	17.7 (1.1 to 34.3)	0.057
Stroop									
Color Word (incongruent)	40.1 (1.88)	39.4 (1.04)	1.000	0.63 (−3.8 to 5.0)	38.3 (3.01)	38.5 (1.65)	1.000	−0.18 (−5.8 to 5.5)	0.765
Interference	0.79 (1.08)	2.90 (1.67)	0.188	−2.11 (−5.2 to 1.0)	−1.92 (1.18)	2.44 (1.59)	0.001	−4.36 (−7.0 to −1.7)	0.281
DS									
DS Forward	8.52 (0.35)	8.94 (0.33)	0.168	−0.42 (−1.0 to 0.2)	8.33 (0.32)	8.51 (0.36)	0.338	−0.18 (−0.7 to 0.3)	0.562
DS Backward	6.46 (0.29)	6.21 (0.29)	0.423	0.25 (−0.7 to 0.3)	5.46 (0.33)	5.68 (0.36)	0.386	−0.23 (−0.7 to 0.3)	0.241
Total	15.0 (0.57)	15.3 (0.61)	0.533	−0.34 (−1.4 to 0.7)	13.8 (0.59)	14.2 (0.66)	0.338	−0.41 (−1.2 to 0.4)	0.923
LNS	9.15 (0.43)	9.92 (0.45)	0.025	−0.77 (−1.4 to −0.1)	8.56 (0.41)	8.70 (0.47)	0.678	−0.13 (−0.8 to 0.5)	0.179

CBGT, cognitive-behavioral group therapy; WASI, Wechsler Abbreviated Scale of Intelligence; BVMT-R, Brief Visuospatial Memory Test—Revised; RCFT, Rey Complex Figure Test; LM AB, Logical Memory AB; TMT, trail making test; Stroop, Stroop Color and Word Test; DS, Digit Span; LNS, Letter-Number Sequencing; *SE*, standard error; p^1, pairwise comparisons with Bonferroni adjustment; p^2, generalized estimating equations.

All analysis were adjusted by baseline trail making B and trail making B-A.

that impaired visuospatial memory and organizational skills in patients with OCD could be trait-related rather than state-dependent.

Patients with OCD who responded to CBGT performed better on the TMT-B and TMT B-A at baseline, suggesting that increased information processing speed, set shifting, and working memory were predictors of good therapeutic outcome in OCD. Although a number of studies investigated the relationship between neuropsychological test performance and treatment outcome in OCD, the results are mixed, making it difficult to extract a specific pattern of features that may be associated with a better treatment response. For instance, while most studies reported that patients with OCD who did respond to treatment exhibited similar[79] or better[28,30] baseline cognitive performance as compared to poor responders, at least one study found that increased perseverative errors and decreased number of categories completed were associated with better response to SRIs in OCD.[37] We believe that the heterogeneity of findings described in the literature may be ascribed to the different populations,[80] comparison groups, executive tests, and therapeutic strategies[25–27,38,79] employed. However, two alternative explanations exist. First, given that the vast majority of studies did not find an association between neuropsychological test performance and OCD symptom severity, it is possible that our results support the notion that neuropsychological deficits may be state-independent in OCD. However, given the design of the present study, we are unable to indicate whether cognitive functions in our sample were objectively deficient. A second

TABLE 5. Association between the changes in neuropsychological scores and the percentage of improvement in response to treatment in the CBGT group

	Improvement (%)	
	Pearson correlation coefficient (*r*)	*P*
WASI		
Verbal IQ	–.134	.304
Performance IQ	.026	.841
Total IQ	–.109	.405
BVMT–R		
BVMT TR	–.076	.562
BVMT TL	–.078	.563
RCFT		
Copy	–.152	.242
Memory	–.190	.143
Organization	–.272	.037
LM AB		
Immediate Recall	.343	.007
Late recall	.141	.277
TMT		
Trails A	.097	.479
Trails B	–.100	.464
Trails B-A	–.114	.402
Stroop		
Color-Word (incongruent)	.229	.092
Interference	.001	.997
DS		
DS Forward	–.154	.241
DS Backward		
Total		
LNS	–.147	.261

WASI, Wechsler Abbreviated Scale of Intelligence; BVMT-R, Brief Visuospatial Memory Test—Revised; RCFT, Rey Complex Figure Test; LM AB, Logical Memory AB; TMT, trail making test; Stroop, Stroop Color and Word Test; DS, Digit Span; LNS, Letter-Number Sequencing.

alternative explanation pertains to a recent study concluding that OCD may be associated with underperformance on neuropsychological tests, but to an extent that does not meet the definition of a clinically significant impairment (i.e., OCD patients underperform but do so within the normative range).[22] In this case, where baseline symptoms are clinically meaningful but neuropsychological test performance is within a normative range, a significant change in cognitive functions is not expected even in light of a significant reduction in symptom severity. However as noted above, this explanation remains speculative given that this study did not allow for objective inferences regarding participants' neuropsychological test performance.

The present study has several strengths including a relatively large and carefully screened clinical sample, and employment of a conservative definition of treatment response with multiplicity corrections. More importantly, to our knowledge the present study is the first to assess cognitive correlates of CBGT treatment response for OCD, adding to the small and contrasting body of literature that has been focusing on individual CBT or pharmacological treatment or their combination.[81] However, our study is not without limitations. First, it did not include a sample of healthy controls. Thus, we were unable to speak of the presence of objective neuropsychological underperformance, deficits, or impairments. This limitation hinders our ability to make cogent inferences concerning the association between neurocognitive *deficits* and treatment response in OCD, but only regarding the change (of lack thereof) in neuropsychological test performance following CBGT. Notwithstanding, we believe that the present study is of contribution to the small and highly inconsistent body of literature [25] investigating the association between neuropsychological test performance and treatment response in OCD. Second, it used a WL control group that is associated with increased number of dropouts. However, as indicated, analysis of dropouts did not reveal any systematic difference. That being said, the percentage of dropouts in the present study was elevated (31.3%), particularly in the WL group. However, similar dropout ratios have been reported in other OCD clinical trials.[41,42,82] Moreover, the present study is characterized by elevated rates of comorbidity (80%) and medication use (47.6%) and although the analyses revealed no significant moderating effects of medications or comorbidity, the study may not have been sufficiently powered to detect such differences. Thus, the possibility of a residual confounding effect of medications or comorbidity cannot be unconditionally ruled out. A third potential limitation of this study would be that examiners administering the neuropsychological battery at T2 were not blinded to participants' assigned condition. However, in the context of neuropsychological tests, the administration instructions and scoring processes are very much standardized and the assessors' subjective inferences are minimal, reducing the impact of this limitation upon the results of this study. Finally, we did not assess OCD symptom dimensions, which potentially may moderate the association between OCD symptoms and neuropsychological test performance. However, the majority of studies did not find such an association.

CONCLUSION

No meaningful association between response to CBGT and neuropsychological test performance has been identified in the present study. However, whereas the present study adds to the small and contrasting body of literature exploring this association[81] at this time the available research does not permit a definite conclusion regarding lack of such association. Further research assessing cognitive correlates of treatment response is highly needed, particularly clinical trials utilizing different treatment strategies and/or novel pharmacological agents, ideally addressing OCD symptom dimensions.

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Conflict of interest. The authors report no conflict of interest. Those who funded the study had no further role in its design, nor in the collection, analysis, and interpretation of data, in the writing of the report, or in the decision to submit the paper for publication.

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