MERAL Publications and Solutions Pvt. Ltd.

Original Article

Effectiveness of Neurofeedback in Reducing Rumination and Cognitive Avoidance in Patients with Obsessive Compulsive Disorder: A Clinical Trial

Elahe Azizi[1], Seyed Hossein Hasani[2], Mehdi Emami Kalan[2], Masoumeh Kalhor[1], Sedigheh Ebrahimi Alavikolaei[1]*

[1]Department of Clinical Psychology, Islamic Azad University, Amol, Iran. [2]Department of Clinical Psychology, Islamic Azad University, Tonekabon, Iran.

Correspondence: Sedigheh Ebrahimi Alavikolaei, Department of Clinical Psychology, Islamic Azad University, Amol, Iran. sedigheh.ebrahimialav@gmail.com

ABSTRACT

Obsessive-compulsive disorder (OCD) is a psychiatric condition characterised by persistent, intrusive thoughts and ritualistic behaviours. This study assesses the impact of qEEG-assisted neurofeedback on two critical components of OCD: rumination, a maladaptive focus on problem causes and consequences, and cognitive avoidance (CA), the tendency to evade distressing thoughts aiming to evaluate neurofeedback's effectiveness in reducing rumination and CA severity in patients with OCD. This controlled prospective clinical trial with parallel design included patients diagnosed with OCD, with Yale-Brown Obsessive Compulsive Scale (YB-OCS) scores ≥16. Subjects were alternately assigned to either the neurofeedback or control groups maintaining a 1:1 ratio. The neurofeedback group underwent 25 sessions over six weeks, with outcomes measured through the Rumination Response Scale (RRS) and the Cognitive Avoidance Questionnaire (CAQ) pre- and post-intervention. Of the initial cohort, 30 participants finished the study. Significant reductions in Rumination and CA were observed in the neurofeedback group with multivariate ANCOVA showing a significant impact on CAQ and RRS scores (Lambda Wilks $p=0.001$) and univariate ANCOVA indicating marked decreases in CA ($p=0.001$, $Eta^2=0.687$) and Rumination ($p=0.001$, Eta Squared=0.636) compared to controls. The findings substantiate qEEG-assisted neurofeedback's role in significantly reducing rumination and cognitive avoidance in OCD, indicating neurofeedback's potential to modulate brain regions implicated in OCD pathology, such as orbitofrontal cortex and anterior cingulate, thus enhancing self-regulation and reducing symptoms. Limitations include no long-term follow-up, reliance on self-report measures, a small, single-centred sample, and convenience sampling, all of which affect the generalizability of the results.

Keywords: Neurofeedback, Obsessive Compulsive Disorder, OCD, EEG Biofeedback, Rumination, Cognitive avoidance

Introduction

Background

Categorised as Obsessive Compulsive Disorder and Related Disorders (OCD&RD) in the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), Obsessive Compulsive Disorder (OCD) is the fourth most prevalent psychiatric illness and causes a tenth of all disability cases worldwide [1, 2]. A pattern of recurrent thoughts, known as obsessions or ruminations, accompanied by high anxiety is a defining feature of OCD, which often leads to compulsion - repetitive behaviours or mental acts aimed at alleviating anxiety- also called rituals [1].

Neurophysiologically, anxiety symptoms in patients with OCD are associated with an overactive orbitofrontal-striatal circuit, anterior cingulate, and thalamus, which present as dysregulated autonomic nervous system, resulting in increased heart rate, elevated skin electrodermal activity, and augmented breathing rate [3]. Psychologically, patients with OCD suffer from dysregulation in data processing and display marked distress when making value-based judgments, seeking extra information to make choices, which outwardly manifests as indecisiveness, doubt, and behavioural inflexibility, which, coupled with

behavioural inflexibility, shapes a neuro-cognitive endophenotype of OCD [4].

The primary treatments for OCD&RD are cognitive-behavioural therapy (CBT) and antidepressants; however, these interventions only provide symptomatic remission to a limited number of patients, with other patients requiring alternative therapies, one of which is biofeedback [1, 3]. Among CBT methods, exposure and response prevention (ERP), a form of confrontation behavioral therapy, is considered the gold standard, demonstrating significant efficacy in reducing OCD symptoms by directly confronting feared stimuli and preventing the subsequent compulsive behaviors [5, 6]. Nevertheless, some patients do not fully respond to ERP or experience substantial residual symptoms, necessitating adjunctive or alternative treatments.

Biofeedback, as a general term, involves inhibiting excessive or reinforcing impaired physiological signals, which aim to teach the patient to regulate their emotions, thoughts, or behaviours in response to specific stimuli, helping patients manage their symptoms [1, 7]. In the case of psychiatric disorders such as anxiety, depression, and schizophrenia, biofeedback is applied with a specific type of feedback from neural activity in target brain regions, i.e., neurofeedback [7].

Initially, neurofeedback was described as a method in which specific frequency bands of the electroencephalograph (EEG) are used to select operant brainwave frequencies by giving real-time audio and/or visual feedback cues [8, 9]. Because the technique is non-invasive and side effects such as headache or fatigue due to attentional demands are minimal, EEG neurofeedback is generally considered a viable alternative, nonmedical treatment option.

Using fMRI as an alternative to EEG, the effectiveness of neurofeedback was demonstrated by showing that patients learned to modulate brain regions (the anterior insula and orbitofrontal cortex) through which later studies have suggested reductions in rumination and anxiety, which has demonstrated a superior effect compared with sham neurofeedback (false feedback signal used as placebo) with similar effectiveness to medication, particularly in reducing compulsion [8, 9].

Comparatively, pharmacological treatments, particularly selective serotonin reuptake inhibitors (SSRIs), are widely used and have demonstrated efficacy in reducing OCD symptoms by 40-60% in responder [10]. However, they often come with side effects and a variable response rate. Combining neurofeedback with pharmacotherapy has shown potential synergistic effects, although more controlled studies are necessary to validate these findings.

Rationale and Objectives

Rumination, defined as passive thinking about the causes and consequences of problems without proactive resolution, exacerbates anxiety and often precedes and fuels obsessive-compulsive behaviour. Cognitive Avoidance (CA), the tendency to repress unwanted thoughts and avoid disturbing thoughts/images, is a principal component of anxiety-related conditions by acting as a mediator of rumination and is crucial in OCD [4]. Avoiding upsetting internal events, including obsessive thoughts, leads to excessive cognitive loads and disruptions in the processing of multiple cortical and subcortical regions, significantly predicting autonomic neural responses to threats [3]. This results in brief moments of reduced fear and anxiety, subsequently giving way to further rumination and worry. Consistent patterns of OCD-related activity have been identified through quantitative EEG (qEEG) of patients with OCD, showing an excess of alpha brain waves across most regions of the brain and increased theta activity in frontal and posterior temporal areas [11].

While the number of treatment sessions in qEEG-assisted neurofeedback is limited and the effects are short-lasting initially, changes become more permanent as the brain learns to self-regulate, similar to skill acquisition and habit formation [11]. Previous studies on the psychological mechanism of neurofeedback's effectiveness on OCD have illustrated that rumination is a key predictor of anxiety, reduced through treatment sessions [4]. Considering the cyclical interaction between rumination—conceptualized as disengagement or avoidance of specific problems—and OCD symptoms, assessing changes in rumination and CA severity during neurofeedback treatment provides a better understanding of the psychological underpinnings of OCD treatment using qEEG neurofeedback. Thus, the current study aimed to determine the effectiveness of qEEG-assisted neurofeedback on rumination and cognitive avoidance in patients with OCD.

Materials and Methods

Design and Sampling

This study adopted an applied, naturalistic, prospective parallel trial structure with a control group and alternate treatment allocation.

Participants were selected via convenience sampling from 130 individuals aged 18-50 years with OCD who visited the Rahnemon Counseling and Psychological Services Center during the assessment period from December 21, 2022 to May 20, 2023. Participants were required to have a Yale-Brown Obsessive Compulsive Scale (YB-OCS) score of higher than 16 as a baseline for the presence of obsessive-compulsive symptoms. As inclusion criteria, to participate in the study, the individuals were required to a. provide written informed consent, b. score at least 16 on the YB-OCS, c. be able to share information and experiences, and d. have no history of somatic comorbidities that might affect their mental activity. The exclusion criteria included a. missing more than one session, b. lack of active participation by failing to complete assignments, and c. unwillingness to continue participation. They were then alternatively assigned to each of the two groups: half were assigned to the experimental

group, engaging in neurofeedback exercises, and the other half served as the control group, resulting in an allocation ratio of 1:1.

Intervention

In this randomised clinical trial, the experimental group received neurofeedback exercises in 25 sessions over a six-week period, amounting to three sessions per week. The treatment was performed using an Iranian 12-channel biofeedback equipment, which records EEG and assesses it using the dual-channel Clinical Q diagnostic system to carry out the OCD treatment protocol. The neurofeedback sessions involved placing sensors on the participant's scalp, connected to sensors through which the computer software detected, reinforced, and recorded specific brain activities, and fed it back immediately to the patient, indicating whether the brain activity was within the set range, which enabled them to gradually develop mental strategies to change their brain activity and maximise rewards. The control group received sham neurofeedback sessions, wherein the feedback provided was non-contingent and based on random signals, thereby controlling for placebo and expectancy effects. After completing the treatment sessions, both groups were re-assessed for dependent variables.

In choosing C4 SMR feedback and bipolar mode at F3-PZ with visual feedback to reduce beta waves, we relied on studies indicating that SMR enhancement at C4 is associated with improvements in attentional processes and anxiety reduction [12, 13]. The decrease of beta activity, particularly in the frontal regions, has been linked to the attenuation of symptoms in anxiety disorders, including OCD [11]. The rationale is based on the understanding that excessive beta activity in these regions correlates with heightened anxiety and compulsive behaviour, while SMR enhancement supports better regulation of sensorimotor processes, thus aiding in overall symptom improvement [14, 15]

Each session began with a 120-s baseline recording at specific EEG recording points (C4, F3-PZ) in an eyes-open condition and relaxed muscle state, followed by 15 min of neurofeedback at point C4 using visual feedback to reinforce the Sensorimotor Rhythm (SMR) wave and reduce alpha and high beta waves. Another 15 min were dedicated to bipolar mode at F3-PZ using visual feedback to reduce beta waves (18-30 Hz).

Outcomes

The two primary tools, which were administered at both the pre-treatment and post-treatment stages, were used to measure outcomes in this study.

The primary outcomes included the Rumination Response Scale (RRS), developed by Nolen-Hoeksema and Morrow in 1993, and the Cognitive Avoidance Questionnaire (CAQ), developed by Sexton and Dugas in 2004. RRS is a 22-item scale rated on a 4-point scale, with total scores ranging from 22 to 88 and a reported Cronbach's alpha ranging from 0.55 to 0.92. The CAQ features a Likert scale ranging from 1 (never) to 5 (always) with a total score reliability of 0.9 and subscale reliability ranging from 0.71 to 0.90.

The Yale-Brown Obsessive-Compulsive Scale (Y-BOCS), a 5-item Likert scale that assesses five key obsessional and five key compulsion parameters, was also used as one of the eligibility criteria. The scale's total score ranges from 0 to 40, with severity gradings from sub-threshold (0-7) to very severe (32-40) and high reliability and validity, with an inter-rater reliability of 0.98, internal consistency of 0.89, and test-retest reliability of 0.84 over a 2-week period.

Statistical Methods

Statistical analysis was conducted using the Statistical Package for the Social Sciences (SPSS), version 27. Both descriptive (mean and standard deviation) and inferential statistical methods were employed to assess the outcomes. The inferential analysis primarily focussed on comparing the mean scores between the experimental and control groups, which involved applying a one-way analysis of covariance (ANCOVA) and the Shapiro-Wilk test alongside a multivariate analysis of covariance. All statistical tests were conducted at a significance level of $\alpha = 0.05$.

Results and Discussion

Participant flow and recruitment

The study population comprised individuals aged between 18 and 50 years who had obsessive-compulsive disorder. During the assessment period from December 21, 2022 to May 20, 2023, approximately 130 individuals were identified. Of these, 55 scored the highest on the Yale-Brown Obsessive Compulsive Scale, which, after applying all other inclusion criteria, amounted to a final sample of 30 participants randomised into the experimental group (n=15) and the control group (n=15).

Baseline Data

The baseline demographic and clinical characteristics of the participants are summarised in Table 1. The experimental group consisted of 6 men (40%) and 9 women (60%), whereas the control group comprised 7 men (46.7%) and 8 women (53.3%), showing a comparable distribution of sexes in both groups. The average age of the participants ranged from 18 to 50 years.

Table 1. Baseline demographic and clinical characteristics

Variable	Group	Frequency	Percentage
Gender: Male	Control	7	23.3%
	Experimental A	6	20.0%
Gender: Female	Control	8	26.7%
	Experimental A	9	30.0%
Age: 18-30	Control	8	26.7%
	Experimental A	7	23.3%
Age: 31-45	Control	4	13.3%
	Experimental A	8	26.7%
Age: 45-50	Control	3	10.0%

Total	Experimental A	0	0.0%
	Control	15	100%
	Experimental A	15	100%

Numbers Analysed

All 30 participants were analysed with no exclusions or losses reported post-randomization, and the analysis was conducted according to the initially assigned group.

Outcomes

The primary outcomes were rumination and cognitive avoidance, which were assessed using RRS and CAQ. The results are presented in Table 2, which shows the mean of pre-treatment and post-treatment scores with standard deviations.

Table 2. Mean and SD of Rumination Response Scale (RRS) and Cognitive Avoidance Questionnaire (CAQ) scores

Group	Time Point	Variable	Count	Lowest Score	Highest Score	Mean	SD
Control	Pre-treatment	Rumination	15	46	67	58.47	6.413
		Cognitive Avoidance	15	75	111	96.33	9.612
	Post-treatment	Rumination	15	46	71	59.20	7.692
		Cognitive Avoidance	15	75	116	97.87	10.723
Experimental	Pre-treatment	Rumination	15	45	72	60.07	9.483
		Cognitive Avoidance	15	73	105	91.27	10.879
	Post-treatment	Rumination	15	34	66	51.47	10.302
		Cognitive Avoidance	15	59	98	80.67	11.146

Multivariate ANCOVA was conducted (Table 3) to evaluate the effectiveness of neurofeedback intervention on both CAQ and RRS. Before the analysis, the assumptions for ANCOVA were tested using Shapiro-Wilk and Levene's tests, confirming the normal distribution and homogeneity of variance across groups, respectively. The significant outcome of the Lambda-Wilks' test in the multivariate ANCOVA (p-value = 0.001) indicates a statistically significant overall effect of neurofeedback on the combined outcomes of CAQ and RRS.

Table 3. Multivariate ANCOVA Results

Test	Value	F-Statistic	df1	df2	p-value
Lambdas Wilks' Test	0.216	45.43	2	25	0.001

Tables 4 and 5 show the univariate ANCOVAs conducted separately for cognitive avoidance and rumination. The results of the Shapiro-Wilk test indicated that the scores for both CAQ and RRS were normally distributed, and the homogeneity of regression slopes was also confirmed by the interaction term in the ANCOVA model. The results of ANCOVAs showed that neurofeedback had significant effects on both CA (p-value = 0.001, Eta Squared = 0.687) and rumination (p-value = 0.001, Eta Squared = 0.636).

Table 4. Cognitive Avoidance Questionnaire (CAQ) Univariate ANCOVA Results

Source	Sum of Squares	df	Mean Square	F	p-value	Eta Squared
Between Groups	1048.04	1	1048.04	59.34	0.001	0.687
Within Groups	476.81	27	17.66	-	-	-
Total	244624.00	30	-	-	-	-

Table 5. Rumination Response Scale (RRS) Univariate ANCOVA Results

Source	Sum of Squares	df	Mean Square	F	p-value	Eta Squared
Between Groups	652.92	1	652.92	47.25	0.001	0.636
Within Groups	373.03	27	13.81	-	-	-
Total	94616.00	30	-	-	-	-

The current study investigated the efficacy of qEEG-assisted neurofeedback in reducing rumination and cognitive avoidance in patients with OCD. Our results demonstrated a significant decrease in both rumination and cognitive avoidance in the experimental group compared with the control group, as measured by the CAQ and RRS questionnaires.

Previous research has examined the potential of neurofeedback as a treatment for OCD and related disorders, indicating it as a safe alternative with similar efficacy to behavioural and pharmaceutical interventions [1, 16, 17]. As these studies outline, neurofeedback seems to facilitate a reduction in OCD symptomatology by modulating specific brain wave frequencies which might be associated with rumination and cognitive avoidance as vital contributors to the disorder's persistence, explaining the improvement observed in the current study, which can be attributed to neurofeedback's effect enhancing self-regulation capacities, enabling patients to better manage intrusive thoughts and maladaptive avoidance strategies, associated with rumination and cognitive avoidance, respectively [11, 16, 18].

Our findings support the theory that neurofeedback modulates neural activity in specific brain regions implicated in OCD. Studies have shown that neurofeedback targeting the orbitofrontal cortex and anterior cingulate cortex can reduce the hyperactivity typically observed in these areas among OCD patients [15, 19]). This modulation is associated with a decrease in excessive beta wave activity (18-30 Hz), which is linked to cognitive rigidity and obsessive rumination thereby facilitating more flexible thinking [20]. Additionally, increasing sensorimotor rhythm (SMR) through neurofeedback is correlated with a stabilization of emotional and cognitive processes, reducing cognitive avoidance and anxiety [15]. This is evidenced by the significant reduction in RRS and CAQ scores observed in our study, suggesting that neurofeedback enhances the brain's ability to regulate itself, mitigating the core symptoms of OCD.

Consistent with the theory of cognitive avoidance, our results follow studies illustrating that avoiding threatening stimuli and internal distress contributes to the maintenance of OCD symptoms [21, 22]. As such, the observed effectiveness of neurofeedback in reducing cognitive avoidance suggests its potential role in disrupting this maladaptive cycle. Moreover, the reduction in rumination aligns with the concept that ruminative responses, while often perceived as problem-solving efforts, exacerbate distress and contribute to the chronicity of OCD, thus reduction in rumination is associated with improvement in other OCD symptoms [17, 21, 22].

Comparatively, our findings align with previous studies showing that neurofeedback can reduce OCD symptoms by modulating specific neural circuits. For instance, a study by Gruzelier (2014) found that neurofeedback significantly reduced symptoms in OCD patients by targeting the orbitofrontal cortex and anterior cingulate cortex [15]. Similarly, Micoulaud-Franchi et al. (2015) demonstrated the effectiveness of neurofeedback in reducing anxiety and rumination, further supporting our results [19].

The findings of this study can be translated into practical implications for mental health practitioners. Neurofeedback presents as a promising non-pharmacological intervention for reducing rumination and cognitive avoidance in patients with OCD. Considering its demonstrated safety and efficacy, it is usually considered an adjunct or alternative to traditional treatments, especially for those who are medication-resistant or prefer non-medication approaches.

Limitations and suggestions

Although the study's outcomes are statistically meaningful and translatable to clinical practise, several limitations warrant attention. The absence of follow-up evaluation precludes insights into the long-term stability and sustainability of the treatment effects. In addition, relying solely on self-report measures might have introduced response biases, albeit minimised by incorporating a control group. Furthermore, considering the small sample size, single-centred nature of the study, and convenience sampling method, the results may not be generalizable across different populations.

As such, we believe that subsequent research should explore the applicability of neurofeedback with greater sample sizes among different patient populations and varying study settings, broadening the scope and understanding of neurofeedback effectiveness. Future studies might also benefit from a more comprehensive methodological approach, including a longer follow-up period and a multimodal assessment strategy.

Conclusion

This study demonstrates the potential of neurofeedback as an effective intervention for reducing rumination and cognitive avoidance in patients with OCD. The implications of these findings extend to both clinical practise and future research, highlighting the need for further exploration of the role of neurofeedback in the treatment of OCD and related disorders.

Patient Consent

All participants were informed about the objective and the methods of the study and provided written informed consent prior to enrolment in the study.

Data Availability

The data that support the findings of this study are not openly available due to reasons of privacy and sensitivity, and so are not publicly available. They are, however, available from the authors upon reasonable request through the corresponding author.

Author contributions

All authors contributed to the study's conception and design. The study was supervised by S.E.A., patient recruitment and interventions were carried out by E.A. assisted by M.E.K., and M.M.D collected the data. Analyses were performed M.K. The first draft of the manuscript was written by A.M.J., and all authors commented on pre-final versions of the manuscript. All authors have read and approved the final manuscript.

Acknowledgments: The authors acknowledge Seyed Hossein Hassani's Contributions as the director of the Rahnemon Clinic where patients were recruited from.

Conflict of interest: Authors Elahe Azizi, Mehdi Emami Kalan, and Masoumeh Kalhor have been employed at the Rahnemon Clinic since prior to the conceptualisation of the study.

Other authors have no conflicts of interests to declare.

Financial support:. While the study was not funded by any institution, participant were recruited from the Rahnemon Clinic with approval from the director of the clinic and following the ethical considerations below.

Ethics statement: The objectives and methodology of this study were approved by the Islamic Azad University, Amol Branch's Ethics Committee under the registration number IR.IAU.BABOL.REC.1402.016, granted on July 7, 2023 and all steps have been performed in accordance with the 1964 Declaration of Helsinki and its later amendments.

References

1. Ferreira S, Pego JM, Morgado P. The efficacy of biofeedback approaches for obsessive-compulsive and related disorders: A systematic review and meta-analysis. Psychiatry Res. 2019;272:237-45.
2. Association AP. Diagnostic and Statistical Manual of Mental Disorders (DSM). 5th ed. Washington, DC2013.

3. Seibell PJ, Hollander E. Management of obsessive-compulsive disorder. F1000Prime Rep. 2014;6:68.
4. Cheon EJ, Koo BH, Seo WS, Lee JY, Choi JH, Song SH. Effects of neurofeedback on adult patients with psychiatric disorders in a naturalistic setting. Appl Psychophysiol Biofeedback. 2015;40(1):17-24.
5. Foa EB, Yadin E, Lichner TK. Exposure and Response (Ritual) Prevention for Obsessive Compulsive Disorder: Therapist Guide: Oxford University Press; 2012 01 Jan 2015.
6. Abramowitz JS. Variants of exposure and response prevention in the treatment of obsessive-compulsive disorder: A meta-analysis. Behavior Therapy. 1996;27(4):583-600.
7. Schoenberg PL, David AS. Biofeedback for psychiatric disorders: a systematic review. Appl Psychophysiol Biofeedback. 2014;39(2):109-35.
8. J.H. Begemann M, J.R. Florisse E, van Lutterveld R, Kooyman M, E. Sommer I. Efficacy of EEG neurofeedback in psychiatry: A comprehensive overview and meta-analysis. Translational Brain Rhythmicity. 2016;1(1).
9. Micoulaud-Franchi JA, McGonigal A, Lopez R, Daudet C, Kotwas I, Bartolomei F. Electroencephalographic neurofeedback: Level of evidence in mental and brain disorders and suggestions for good clinical practice. Neurophysiol Clin. 2015;45(6):423-33.
10. Fineberg NA, Reghunandanan S, Brown A, Pampaloni I. Pharmacotherapy of obsessive-compulsive disorder: evidence-based treatment and beyond. Aust N Z J Psychiatry. 2013;47(2):121-41.
11. Hammond DC. Neurofeedback with anxiety and affective disorders. Child Adolesc Psychiatr Clin N Am. 2005;14(1):105-23, vii.
12. Vernon D, Egner T, Cooper N, Compton T, Neilands C, Sheri A, et al. The effect of training distinct neurofeedback protocols on aspects of cognitive performance. Int J Psychophysiol. 2003;47(1):75-85.
13. Sterman MB. Physiological origins and functional correlates of EEG rhythmic activities: implications for self-regulation. Biofeedback Self Regul. 1996;21(1):3-33.
14. Egner T, Gruzelier JH. EEG Biofeedback of low beta band components: frequency-specific effects on variables of attention and event-related brain potentials. Clinical Neurophysiology. 2004;115(1):131-9.
15. Gruzelier JH. EEG-neurofeedback for optimising performance. I: A review of cognitive and affective outcome in healthy participants. Neuroscience & Biobehavioral Reviews. 2014;44:124-41.
16. Zafarmand M, Farahmand Z, Otared N. A Systematic Literature Review and Meta-analysis on Effectiveness of Neurofeedback for Obsessive-Compulsive Disorder. Neurocase. 2022;28(1):29-36.
17. Hammond DC. QEEG-Guided Neurofeedback in the Treatment of Obsessive Compulsive Disorder. Journal of Neurotherapy. 2003;7:25-52.
18. Beigi Harchegani N, sharifi T, Nikkhah M, Ahmadi R. comparison of the effectiveness of neurofeedback therapy and schema therapy on perfectionism of people with social anxiety in shahinshahr. medical journal of mashhad university of medical sciences.65(3):1020-35 , year = 2022.
19. Micoulaud-Franchi JA, McGonigal A, Lopez R, Daudet C, Kotwas I, Bartolomei F. Electroencephalographic neurofeedback: Level of evidence in mental and brain disorders and suggestions for good clinical practice. Neurophysiologie Clinique/Clinical Neurophysiology. 2015;45(6):423-33.
20. Coben R, Evans JR. Neurofeedback and neuromodulation techniques and applications. 2011.
21. Dickson KS, Ciesla JA, Reilly LC. Rumination, worry, cognitive avoidance, and behavioral avoidance: examination of temporal effects. Behav Ther. 2012;43(3):629-40.
22. Kyrios M, Iob MA. Automatic and strategic processing in obsessive-compulsive disorder: attentional bias, cognitive avoidance or more complex phenomena? J Anxiety Disord. 1998;12(4):271-92.