RESEARCH PAPER

Effects of Transcranial Direct Current Stimulation on MoCA-INA Scores and DTABR in Patients with Ischemic Post-Stroke Cognitive Impairment

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ABSTRACT

Introduction: Post-stroke cognitive impairment (PSCI) is a consequence of ischemic stroke, reducing independence and life quality. Although transcranial direct current stimulation (tDCS) may improve cognition, evidence linking quantitative electroencephalography (qEEG) to outcomes is limited. This study evaluated tDCS effects on DTABR and MoCA-INA performance.

Materials and Methods: This prospective quasi-experimental single-blind pretest–posttest study included 30 patients with PSCI allocated to an intervention group (n = 15) or a sham control group (n = 15). The intervention group received active tDCS (2 mA, 20 minutes/session, 10 sessions over two weeks), while the control group received sham stimulation. Cognitive function was assessed using MoCA-INA and neurophysiological changes were evaluated using qEEG before and after intervention.

Results: Active tDCS reduced slow-wave activity, increased alpha–beta activity, and significantly decreased global DTABR compared with sham stimulation (1.16 ± 0.58 vs. 3.97 ± 3.12, p = 0.004). Mean MoCA-INA scores increased from 19.73 ± 6.13 to 24.87 ± 4.73 in the intervention group, compared with 19.40 ± 3.58 to 20.27 ± 3.90 in the control group. Significant within-group cognitive improvement was observed only after active tDCS (p = 0.001). Compared with sham stimulation, active tDCS produced greater improvements in visuospatial/executive function, attention, language, and delayed recall.

Conclusion: Active tDCS improved both neurophysiological and cognitive outcomes in patients with PSCI, as demonstrated by reduced global DTABR and enhanced MoCA-INA performance. These findings support tDCS as a promising adjunctive neuromodulation strategy for post-stroke cognitive rehabilitation.

Keywords: transcranial direct current stimulation; ischemic stroke; cognitive impairment; DTABR; MoCA-INA

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INTRODUCTION

Ischemic stroke remains one of the leading causes of disability worldwide and is frequently associated with long-term cognitive impairment that negatively affects functional independence and quality of life (1). Post-stroke cognitive impairment (PSCI) commonly involves deficits in memory, attention, executive function, and visuospatial abilities, creating major challenges during neurological rehabilitation (2). Stroke continues to represent a substantial public health burden in Indonesia. Data from the Indonesian Basic Health Research Survey reported a national stroke prevalence of 10.9 per 1,000 population with approximately 713,783 new cases annually, predominantly affecting older adults (3). Given the substantial burden and geographic variability of stroke incidence in Indonesia, effective post-stroke rehabilitation strategies addressing neurological sequelae, including cognitive impairment, remain essential (4). Among stroke subtypes, ischemic stroke accounts for most cases and results from reduced cerebral blood flow causing neuronal injury, metabolic dysfunction, and secondary brain damage (5).

Beyond stroke incidence, clinical outcomes after ischemic stroke are influenced by several vascular and stroke-

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related characteristics (6). Recent evidence suggests that stroke subtype may contribute more strongly to post-stroke functional outcomes than blood pressure variability, with lacunar stroke demonstrating better modified Rankin Scale outcomes compared with non-lacunar stroke (7). Nevertheless, functional recovery alone may not fully reflect cognitive recovery after stroke, highlighting the importance of evaluating post-stroke cognitive impairment (8,9).

Cognitive impairment after ischemic stroke is associated with disruption of cortical and subcortical networks, particularly involving the prefrontal cortex, hippocampus, parietal lobe, and thalamus, which are essential for higher cognitive functions (2,10). Therefore, interventions that promote cortical reorganization and neuroplasticity have become an important focus in post-stroke rehabilitation (1,11). Transcranial Direct Current Stimulation (tDCS) is a non-invasive neuromodulation technique that delivers low-intensity electrical currents to alter cortical excitability and facilitate neuroplasticity (12). Recent studies demonstrated that repeated tDCS sessions improved cognitive performance in stroke survivors as measured using the Montreal Cognitive Assessment (MoCA) (13). Similarly, anodal tDCS targeting the left lateral prefrontal cortex significantly enhanced cognitive domains including fluency and orientation in patients with post-stroke cognitive dysfunction (14). Neuroplasticity after stroke is driven by structural and functional reorganization of surviving neural networks through mechanisms including changes in neuronal excitability, adaptive long-term potentiation/depression (LTP/LTD), cortical remodeling, and reperfusion-mediated recovery within peri-infarct regions. These processes may facilitate restoration of cognitive and functional performance and provide a biological basis for neuromodulation approaches such as tDCS (15).

Beyond clinical assessment, objective neurophysiological evaluation is increasingly important to understand cortical responses following neuromodulation (16). Quantitative electroencephalography (qEEG) provides quantitative analysis of brain electrical activity and has shown potential for evaluating stroke severity and predicting functional outcomes (17). Among qEEG parameters, the delta–theta/alpha–beta ratio (DTABR) has been associated with cortical dysfunction and post-stroke recovery. Previous evidence demonstrated that tDCS may induce favorable changes in qEEG patterns, including increased alpha activity and reduced delta activity following intervention (18).

However, evidence integrating objective neurophysiological measures using qEEG with clinical cognitive evaluation using the Indonesian version of the Montreal Cognitive Assessment (MoCA-INA) remains limited in patients with ischemic stroke. Therefore, this study aimed to evaluate the effects of tDCS on qEEG outcomes, particularly DTABR, and cognitive performance measured by MoCA-INA in patients with ischemic stroke and cognitive impairment.

MATERIALS AND METHODS

Study Design and Setting

This experimental study used a prospective pretest–posttest control group design to evaluate the effects of tDCS on cognitive function and brain electrical activity in patients with ischemic stroke and cognitive impairment. Outcome assessments were conducted before and after intervention using the Indonesian version of the MoCA-INA and qEEG.

Participants were blinded to group allocation, whereas investigators administering the intervention were aware of treatment assignment. Outcome assessments, including MoCA-INA and qEEG evaluations, were performed by assessors blinded to treatment allocation. The study was conducted from November 2025 until the required sample size was achieved at Dr. Wahidin Sudirohusodo General Hospital and affiliated hospitals.

Sample Size

The minimum sample size was determined based on a comparison between two study groups in a pretest–posttest design, considering the correlation between paired measurements. The calculation used a two-sided significance level of 5%, a statistical power of 90%, a standard deviation of 7.27, an expected correlation coefficient of 0.38 between repeated measurements, and a minimum clinically meaningful difference of 6.8 points in the primary outcome based on previous studies. The estimated minimum sample size was 14.88 participants per group, which was rounded up to 15 participants per group. Therefore, the study included a total of 30 participants, comprising 15 participants in the active tDCS group and 15 participants in the sham control group.

Participants

Participants were recruited consecutively from eligible patients attending the study sites during the recruitment period. Eligible participants met the following inclusion criteria: (1) first-ever ischemic stroke confirmed clinically and by neuroimaging, with post-stroke cognitive impairment defined as a MoCA-INA score <26; (2) stroke onset ≤6 months; (3) aged 18–65 years; (4) at least 9 years of formal education; (5) adequate hearing ability; (6) willingness to complete all study procedures including qEEG, MoCA-INA, and tDCS sessions; and (7) provision of written informed consent.

Exclusion criteria included: post-stroke aphasia, recurrent stroke, history of epilepsy, metallic implants, cardiac pacemaker, cochlear implant, previous head injury, severe hearing impairment, severe psychiatric disorders or advanced dementia limiting participation, pregnancy, and participation in other neuromodulation interventions within the previous month. Participants were withdrawn if they requested discontinuation, developed acute medical conditions preventing continuation, experienced adverse effects related to tDCS, or failed to complete intervention and post-treatment evaluations.

Assignment Method

Eligible participants were recruited consecutively from the study sites during the recruitment period. Because this was a prospective quasi-experimental study, participants were assigned to either the active tDCS group or the sham control group using a quasi-experimental assignment rather than individual randomization. Both groups underwent identical assessment procedures and intervention schedules, differing only in the delivery of active or sham stimulation.

Intervention Protocol

Participants were allocated into two groups.

The intervention group received active tDCS with direct current stimulation at 2 mA for 20 minutes per session. A total of 10 sessions were administered over two weeks. The anodal electrode was positioned over the left dorsolateral prefrontal cortex (DLPFC; F3), while the cathodal electrode was positioned over the right DLPFC (F4). The control group received sham tDCS. Stimulation at 2 mA was delivered only during the initial 30 seconds while maintaining electrode placement for the remaining session duration. Treatment duration, number of sessions, and electrode placement were identical to the intervention group.

Outcome Measurements

Primary Outcomes

The primary outcomes of this study were changes in cognitive function measured using the Indonesian version of the MoCA-INA and neurophysiological changes assessed using the DTABR derived from qEEG.

Secondary Outcomes

Secondary outcomes included changes in global DTABR, absolute qEEG power across the delta, theta, alpha, and beta frequency bands, and changes in individual MoCA-INA cognitive domains. Significant improvements in cognitive domains were observed in visuospatial/executive function, attention, language, and delayed recall.

Cognitive Assessment

Cognitive function was evaluated using the Indonesian version of the MoCA-INA. MoCA-INA assesses multiple cognitive domains including attention, executive function, memory, language, visuospatial ability, abstraction, and orientation. Cognitive impairment was defined as a MoCA-INA score <26.

Quantitative Electroencephalography Assessment

Brain electrical activity was assessed using qEEG recorded with the Cadwell Easy III EEG system and analyzed using NeuroGuide Deluxe qEEG software. Participants were instructed to wash their hair before examination and avoid applying hair products. Recordings were performed in a quiet environment with participants resting in a supine position. Electrodes were positioned according to the international 10–20 system using 19 channels. Recording was initiated after electrode impedance reached <20 Ω. Signal acquisition used a low-cut filter of 0.53 Hz, high-cut filter of 70 Hz, and notch filter activation. Raw EEG data were converted into EDF format and processed automatically for artifact removal. The primary qEEG parameter analyzed was the DTABR.

Study Procedure

After eligibility screening and informed consent acquisition, baseline assessments including MoCA-INA and qEEG were performed. Participants subsequently underwent active or sham tDCS intervention for two weeks. Following completion of all treatment sessions, MoCA-INA and qEEG assessments were repeated. Collected data were recorded and analyzed statistically.

Statistical Analysis

Data analysis was performed using IBM SPSS Statistics version 29. Baseline characteristics, including age, sex, educational level, stroke onset, comorbidities, and the distribution of impaired MoCA-INA domains, were summarized descriptively. Data distribution was evaluated using the Shapiro–Wilk test because the sample size was fewer than 50 participants. Categorical variables were compared between groups using the Chi-square test or Fisher's exact test, as appropriate. Continuous within-group comparisons were analyzed using paired samples t-test for normally distributed data or the Wilcoxon signed-rank test for non-normally distributed data. Between-group comparisons were performed using the independent samples t-test for normally distributed data or the Mann–Whitney U test for non-normally distributed data. Changes in individual MoCA-INA domain scores were also compared using the Mann–Whitney U test. Statistical significance was defined as $p < 0.05$. All participants who completed the intervention were included in the final analysis.

Ethics Approval and Consent to Participate

This study was approved by the Health Research Ethics Committee of the Faculty of Medicine, Hasanuddin University, Makassar, Indonesia (Approval No. 1121/UN4.6.4.5.31/PP36/2025; Protocol No. UH25121176). Ethical approval was granted on 31 December 2025 and remained valid until 31 December 2026. All procedures were conducted in accordance with institutional ethical standards and the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment.

RESULTS

This study was conducted at Dr. Wahidin Sudirohusodo General Hospital and affiliated hospitals in Makassar, Indonesia, from November 2025 to May 2026. Data were collected prospectively through pre- and post-intervention assessments using the Indonesian version of the MoCA-INA and qEEG. Changes in cognitive performance and brain electrical activity were evaluated by comparing measurements obtained before and after intervention in both study groups. Cognitive outcomes were assessed using MoCA-INA scores, while neurophysiological outcomes were evaluated using qEEG parameters.

Participant Flow

A total of 40 patients were assessed for eligibility during the study period. Of these, 10 patients were excluded prior to enrollment. The remaining 30 eligible participants were enrolled and allocated to either the active transcranial direct current stimulation (tDCS) group (n = 15) or the sham control group (n = 15). All participants received the allocated intervention, completed the follow-up assessments, and were included in the final statistical analysis. No missing outcome data were identified, and all enrolled participants completed the study protocol. No serious adverse events related to tDCS were observed during the intervention period. The participant flow is presented in Figure 1.

Participant Characteristics

Baseline demographic and educational characteristics are summarized in Table 1. Mean age was 53.13 ± 9.63 years in the intervention group and 52.60 ± 11.15 years in the control group, with a mean age of 52.87 ± 10.24 years; the difference was not significant (p = 0.642). Sex distribution was identical, with 8 male participants (53.3%) and 7 female participants (46.7%) in each group. Senior high school was the most common educational level (43.3%), followed by bachelor's degree (33.3%). Stroke onset and comorbidity profiles were comparable. Most participants had a one-month stroke onset (90.0%), and hypertension was the most common comorbidity (50.0%), followed by hypertension and diabetes mellitus (26.7%). No significant between-group differences were observed for stroke onset (p = 0.483), comorbidity status (p = 1.000), sex (p = 1.000), or educational level (p = 0.338), indicating comparable baseline characteristics before the intervention.

Baseline cognitive impairment across individual MoCA-INA domains is summarized in Table 2. Delayed recall was the most frequently impaired domain in both groups (93.3%), followed by visuospatial/executive function (90.0%), attention (76.7%), and orientation (76.7%). No significant between-group differences were observed in the baseline distribution of impaired cognitive domains (all p > 0.05), indicating comparable cognitive profiles before intervention.

Quantitative EEG Outcomes

Absolute Power in the Intervention Group

Figure 2 shows absolute EEG power before and after active stimulation in the intervention group. Before stimulation, delta activity had the highest power across frontal and prefrontal electrodes (F3, F4, F7, F8, FP1, and FP2), peaking at FP1 and FP2 (approximately 35 μV). Theta was the second dominant band, while alpha and beta showed lower power. After tDCS administration, spectral distribution shifted (Figure 2): delta power decreased at all electrode sites, while alpha and beta activity increased in most frontal regions. Alpha reached the highest post-intervention value at F3 (21.83 μV). Overall, post-intervention qEEG showed reduced slow-wave and increased fast-wave activity after active stimulation.

Absolute Power in the Control Group

Figure 3 presents the absolute EEG power distribution in the control group before and after sham stimulation. Before stimulation, delta activity remained predominant across all frontal electrode sites, with the highest values recorded at FP1 (52.74 μV) and FP2 (47.95 μV). After sham stimulation, only minimal spectral alterations were observed (Figure 2). Delta activity remained dominant and showed slight increases at several recording sites, whereas theta, alpha, and beta bands demonstrated only minor changes. Alpha activity remained relatively stable post-stimulation. These findings suggest persistence of slow-wave predominance without meaningful electrophysiological modulation.

Delta–Theta/Alpha–Beta Ratio (DTABR)

Figure 4 illustrates changes in DTABR across frontal and prefrontal electrode sites following stimulation. In the intervention group, DTABR decreased consistently after active tDCS at F3 (1.86 to 1.06), F4 (1.78 to 1.23), F7 (2.20 to 1.34), F8 (2.08 to 1.15), FP1 (2.51 to 1.31), and FP2 (2.42 to 1.26), with the greatest reductions observed in the prefrontal regions. In contrast, the control group demonstrated increased DTABR values following sham stimulation at FP1 (5.17 to 6.23), FP2 (4.35 to 5.18), F3 (2.92 to 3.54), F4 (2.09 to 2.76), F7 (3.55 to 4.09), and F8 (2.62 to 3.19), indicating opposite electrophysiological trends between the two groups.

Overall changes in global DTABR are presented in Figure 5. The intervention group demonstrated a marked reduction in median global DTABR following active stimulation, whereas the control group showed no significant within-group change after sham stimulation. Table 3 summarizes the statistical analysis of global DTABR. Before stimulation, mean global DTABR did not differ significantly between the intervention and control groups (2.08 ± 0.99 vs. 3.25 ± 2.40, p = 0.098). After stimulation, the intervention group demonstrated significantly lower global DTABR than the control group (1.16 ± 0.58 vs. 3.97 ± 3.12, p = 0.004). Within-group analysis showed a significant reduction in global DTABR following active stimulation (p = 0.001), whereas no significant change was observed in the control group (p = 0.204).

Cognitive Outcomes Based on MoCA-INA

Baseline MoCA-INA domain impairment is presented in Table 2. In the intervention group, the most frequently impaired domains were delayed recall (93.3%), visuospatial/executive function (86.7%), attention (73.3%), and orientation (66.7%). In the control group, they were visuospatial/executive function (93.3%), delayed recall (93.3%), orientation (86.7%), and attention (80.0%). No significant between-group differences were observed in baseline MoCA-INA domains (all p > 0.05), indicating comparable cognitive profiles before intervention. Overall cognitive outcomes before and after stimulation are presented in Table 4 and Figure 6. Mean MoCA-INA scores were comparable before stimulation (19.73 ± 6.13 vs. 19.40 ± 3.58, p = 0.348) but significantly higher in the intervention group after stimulation (24.87 ±

4.73 vs. 20.27 ± 3.90, p = 0.002). Within-group improvement was significant only in the intervention group (p = 0.001 vs. p = 0.078 for controls).

Further analysis of individual cognitive domains is presented in Table 5. In the intervention group, significant improvements after active tDCS were observed in executive function (p = 0.018), attention (p = 0.003), language (p = 0.034), delayed recall (p = 0.001), orientation (p = 0.016), and overall MoCA-INA score (p = 0.001). In contrast, no significant changes were found in naming (p = 0.317) or abstraction (p = 0.157). In the control group, none of the cognitive domains, including the total MoCA-INA score (p = 0.078), demonstrated statistically significant pre–post improvement (all p > 0.05). These findings indicate that active tDCS produced significant within-group improvements across multiple cognitive domains, whereas sham stimulation did not result in significant cognitive changes.

DISCUSSION

This study demonstrated that active tDCS was linked to enhancements in both neurophysiological and cognitive outcomes in patients with ischemic stroke and cognitive impairment. In comparison to sham stimulation, active tDCS consistently reduced the DTABR and significantly improved MoCA-INA scores and various cognitive domains. The baseline characteristics showed that the intervention and control groups were comparable regarding age, sex, and educational background, minimizing the possibility that post-intervention differences were due to demographic imbalances. In addition, stroke onset and comorbidity profiles were comparable between groups, further reducing potential baseline confounding factors that could influence neurophysiological and cognitive outcomes. The predominance of male participants in this study aligns with findings by Karimah et al. (2025) and Pinzon and Anggraini (2021), who reported a higher burden of ischemic stroke among males due to earlier accumulation of vascular risk factors (19). Similarly, the mean age of approximately 53 years corresponds to the age range commonly associated with increased stroke incidence, as previously noted by Harahap et al. (2021). The educational level was also relatively balanced across groups, which is significant because cognitive reserve may affect post-stroke recovery, as suggested by Buvarp et al. (2021).

Prior to stimulation, both groups exhibited similar levels of cognitive impairment, as indicated by mean MoCA-INA scores falling below the normal threshold. Baseline domain analysis further demonstrated that delayed recall and visuospatial/executive function were the most frequently impaired cognitive domains in both groups, with no significant between-group differences across any MoCA-INA domain. These results corroborate the findings of Karimah et al. (2025) and Pinzon and Anggraini (2021), who identified that post-stroke cognitive dysfunction frequently impacts memory, attention, and executive function due to disruptions in large-scale cortical networks (5,19). The elevated baseline DTABR values observed in both groups further suggest a predominance of slow-wave activity and diminished cortical efficiency. Comparable electrophysiological patterns were reported by Williamson et al. (2025) and Zakaria et al. (2021), who demonstrated that increased delta and theta activity signifies impaired neuronal metabolism and cortical dysfunction following ischemic injury (19,20). Furthermore, Liu et al. (2022) and Wang et al. (2025) established that elevated DTABR is linked to impaired interregional communication and poorer neurological outcomes (21,22).

Following active tDCS, there was a reduction in DTABR values across all frontal and prefrontal electrode sites, which was also reflected by a significant reduction in global DTABR compared with the control group. These findings coincided with significant enhancements in cognitive performance. These results align with the findings of Liu et al. (2022), who indicated that neuromodulation can restore cortical excitability and facilitate functional reorganization post-stroke. Zakaria et al. (2021) similarly reported that reductions in slow-wave dominance were linked to improved electrophysiological recovery (23). Mechanistically, Yang et al. (2022) suggested that repetitive stimulation enhances synaptic efficacy and promotes neuroplastic adaptation via long-term potentiation-related pathways .

The improvement in neurophysiological markers was accompanied by clinically significant gains in cognitive function. The observed increase in MoCA-INA scores in this study corroborates the findings of Wang et al. (2022), who demonstrated that stimulation targeting the dorsolateral prefrontal cortex enhanced executive performance and memory-related functions in stroke survivors. Similarly, Yoon et al. (2021) reported improvements in cognitive processing and information integration following repeated tDCS sessions (24). Furthermore, active tDCS produced significantly greater improvements than sham stimulation in visuospatial/executive function, attention, language, and delayed recall, whereas naming, abstraction, and orientation did not differ significantly between groups. These findings suggest that tDCS preferentially enhances cognitive domains that rely heavily on prefrontal cortical networks and working memory processes.

In contrast, the sham stimulation group exhibited a sustained elevation in DTABR and only minor, non-significant alterations in MoCA-INA scores. This phenomenon aligns with the findings of Chen et al. (2022), who reported that sham stimulation did not elicit measurable electrophysiological adaptation (25). Similarly, Antonenko et al. (2024) proposed that the lack of sustained electrical modulation hinders the induction of cortical plasticity (26). The slight improvement in cognitive scores may be attributed to repeated cognitive testing exposure or spontaneous recovery, as previously noted by Qurat-ul-Ain et al. (2022) and Yoon et al. (2021) (24,27).

Overall, the current findings reinforce existing evidence supporting the therapeutic efficacy of tDCS in post-stroke cognitive rehabilitation. Consistent with the observations of Wang et al. (2022) and Yang et al. (2022), active neuromodulation was linked to concurrent enhancements in both objective electrophysiological measures and clinical cognitive outcomes (28,29). The reduction in DTABR, coupled with improved MoCA-INA performance, suggests that tDCS may facilitate the restoration of cortical dynamics and support cognitive recovery following ischemic stroke. The concordance between reduced global DTABR and improvements in specific cognitive domains further supports the potential of qEEG-derived DTABR as an objective biomarker for monitoring neurophysiological responses to tDCS during post-stroke cognitive rehabilitation.

LIMITATIONS

This study has several limitations. First, the sample size and single-region recruitment may limit generalizability to populations with post-stroke cognitive impairment. Second, the intervention and follow-up lasted only two weeks, so the durability and long-term sustainability of cognitive and neurophysiological improvements following tDCS could not be determined. Third, although qEEG and MoCA-INA provided clinical assessments, additional neuroimaging modalities and functional outcome measures not included may have limited characterization of mechanisms underlying cognitive recovery. Finally, restricting participants to first-ever ischemic stroke with cognitive impairment within six months of onset may limit applicability to recurrent stroke or other recovery stages. Future studies are warranted for validation.

CONCLUSIONS

This study demonstrated that active tDCS improved both cognitive and neurophysiological outcomes in patients with ischemic stroke and cognitive impairment. Before intervention, participants exhibited cognitive deficits accompanied by predominant slow-wave activity on qEEG, reflected by elevated DTABR values. Following intervention, the active tDCS group showed a significant reduction in global DTABR compared with the sham group, suggesting favorable modulation of cortical activity. These neurophysiological changes were accompanied by significant improvements in overall MoCA-INA performance and greater gains in visuospatial/executive function, attention, language, and delayed recall compared with sham stimulation. Overall, these findings suggest that tDCS may serve as a promising adjunctive neuromodulation strategy for post-stroke cognitive rehabilitation, with benefits demonstrated by both qEEG-derived neurophysiological markers and clinical cognitive assessments.

Declarations

Consent for Publication

Not applicable.

Availability of Data and Materials

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing Interests

The authors declare that they have no competing interests.

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Authors' Contributions

TGS: Conceptualization, methodology, investigation, data curation, formal analysis, writing—original draft.

JT: Conceptualization, supervision, validation, writing—review and editing.

MIB: Methodology, investigation, supervision.

AAZ: Statistical analysis, formal analysis.

AKB: Investigation, data interpretation.

NM: Review and editing, supervision.

All authors read and approved the final manuscript.

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REFERENCES

1. Rost NS, Meschia JF, Gottesman R, Wruck L, Helmer K, Greenberg SM. Cognitive Impairment and Dementia After Stroke: Design and Rationale for the DISCOVERY Study. Stroke. Stroke; 2021;52(8):E499–516. DOI: 10.1161/STROKEAHA.120.031611
2. Sloane KL, Hamilton RH. Transcranial Direct Current Stimulation to Ameliorate Post-Stroke Cognitive Impairment. Brain Sci. Brain Sci; 2024;14(6). DOI: 10.3390/BRAINSCI14060614
3. Elsa MC, Siahaan YMT, Lumenta M. Mary Christina Elsa [PDF] from semanticscholar.org Visuospatial function impairment in patients with subacute-chronic isolated frontal lobe ischemic stroke: A cross-sectional study using MoCA-INA in a private hospital. Romanian JouRnal of neuRology. 2024;23(3):243–53.
4. Feigin VL, Abate MD, Abate YH, ElHafeez SA, Abd-Allah F, Abdelalim A, et al. Global, regional, and national burden of stroke and its risk factors, 1990–2021: a systematic analysis for the Global Burden of Disease Study 2021. Lancet Neurol. Elsevier Ltd; 2024;23(10):973–1003. DOI: 10.1016/S1474-4422(24)00369-7
5. Pinzon RT, Anggraini C. The Predictor Factors of Cognitive Disorders Thirty Days Post Mild-Moderate

Ischemic Stroke. J Indon Med Assoc. 2021;71(5):220–7.

6. Cao Y, Chen Y, Zhang X, Wang Y. Analysis of Prognostic Risk Factors for Ischemic Stroke in China: A Multicentre Retrospective Clinical Study; A National Survey in China. Curr Neurovasc Res. Bentham Science Publishers Ltd.; 2022;19(1):117. DOI: 10.2174/1567202619666220331160024
7. Maulida, Akbar M, Bahar A, Masadah R, Tammasse J, Kaelan C. Analysis of blood pressure variability in lacunar and non-lacunar types acute ischemic strokes patients as a predictor of clinical outcomes. Acta Biomed. Mattioli 1885; 2024;95(6):e2024187–e2024187. DOI: 10.23750/ABM.V95I6.16635
8. Lingo VanGilder J, Hooyman A, Peterson DS, Schaefer SY. Post-stroke cognitive impairments and responsiveness to motor rehabilitation: A review. Curr Phys Med Rehabil Rep. Springer Science and Business Media B.V.; 2020;8(4):461. DOI: 10.1007/S40141-020-00283-3
9. El Husseini N, Katzan IL, Rost NS, Blake ML, Byun E, Pendlebury ST, et al. Cognitive Impairment After Ischemic and Hemorrhagic Stroke: A Scientific Statement From the American Heart Association/American Stroke Association. Stroke. Wolters Kluwer Health; 2023;54(6):E272–91. DOI: 10.1161/STR.0000000000000430;CTYPE:STRING:JOURNAL
10. Tsiakiri A, Giantsios C, Vlotinou P, Nikolaidou A, Atanbori J, Sohani B, et al. Mapping brain networks and cognitive functioning after stroke: A systematic review. Brain Organoid and Systems Neuroscience Journal. Elsevier; 2024;2:43–52. DOI: 10.1016/J.BOSN.2024.08.001
11. Karaganova I, Mindova S. Neuroplasticity after stroke: Adaptive and maladaptive mechanisms in evidence-based rehabilitation. Journal of Stroke and Cerebrovascular Diseases. W.B. Saunders; 2026;35(6):108634. DOI: 10.1016/J.JSTROKECEREBROVASDIS.2026.108634
12. Bo W, Lei M, Tao S, Jie LT, Qian L, Lin FQ, et al. Effects of combined intervention of physical exercise and cognitive training on cognitive function in stroke survivors with vascular cognitive impairment: a randomized controlled trial. Clin Rehabil. Clin Rehabil; 2019;33(1):54–63. DOI: 10.1177/0269215518791007
13. Xue X, Zhang S, Guo Q, Wu J, Zhang J, Wang C, et al. Protocol for a randomized controlled trial: efficacy and mechanisms of dual-target anodal tDCS in post-stroke cognitive impairment. Front Hum Neurosci. Frontiers Media SA; 2025;19:1603797. DOI: 10.3389/FNHUM.2025.1603797/TEXT
14. Najibi AM, Rahnemayan S, Poursoleimani A, Heshmati R, Nazari MA, Shali EG, et al. Transcranial Direct Current Stimulation and Mindfulness for Cognitive and Mood Recovery in Stroke Survivors: A Pilot Randomized Controlled Study. Stroke Res Treat. Stroke Res Treat; 2025;2025(1). DOI: 10.1155/SRAT/3893469
15. Alia C, Spalletti C, Lai S, Panarese A, Lamola G, Bertolucci F, et al. Neuroplastic Changes Following Brain Ischemia and their Contribution to Stroke Recovery: Novel Approaches in Neurorehabilitation. Front Cell Neurosci. Front Cell Neurosci; 2017;11. DOI: 10.3389/FNCEL.2017.00076
16. Cox SS, Wood M, Sutton F, Tucker K, Cash N, Gong R, et al. Neuromodulation approaches and applications in the management of post-stroke pain: a comprehensive review. Front Neurol. 2026;17:1789715. DOI: 10.3389/FNEUR.2026.1789715
17. Zhang Y, Chu H, Qiao Q, Dong S, Liu J, Wang H. Prospective analysis of quantitative EEG indices for predicting functional outcomes in acute ischemic stroke. SLAS Technol. Elsevier; 2025;33:100317. DOI: 10.1016/J.SLAST.2025.100317
18. Liu AL, Du MH, Liu YL, Fei CJ, Xue YQ, Yin R. Research progress of quantitative electroencephalography in post-ischemic stroke mental disorders. Front Neurol. Frontiers Media SA; 2025;16:1445962. DOI: 10.3389/FNEUR.2025.1445962
19. Zahra Khairunnisa Karimah, Nugraha Sutadipura, Alya Tursina. Characteristics of Vascular Dementia in Ischemic Stroke Patients at RSAU Salamun Bandung. Jurnal Riset Kedokteran. Universitas Islam Bandung (Unisba); 2025;31–8. DOI: 10.29313/JRK.V5I1.6687
20. Williamson J, James S, Li S, Sidorov E, Yang Y. Abstract TP105: Non-invasive Brain Stimulation Alters Resting-State Brain Activity in Ischemic Stroke. Stroke. Ovid Technologies (Wolters Kluwer Health); 2025;56(Suppl_1). DOI: 10.1161/STR.56.SUPPL_1.TP105
21. Liu M, Xu G, Yu H, Wang C, Sun C, Guo L. Effects of Transcranial Direct Current Stimulation on EEG Power and Brain Functional Network in Stroke Patients. IEEE Trans Neural Syst Rehabil Eng. IEEE Trans Neural Syst Rehabil Eng; 2023;31:335–45. DOI: 10.1109/TNSRE.2022.3223116
22. Xi W, Song G, Ma Z, Liu L. The effects of transcranial direct current stimulation on EEG power and complexity of ischemic stroke. Front Neurosci. Frontiers Media SA; 2025;19:1688032. DOI: 10.3389/FNINS.2025.1688032/TEXT

23. Zakaria H, Valentine O, Mayza A. Analysis of quantitative EEG (QEEG) parameters on the effect of transcranial direct current stimulation (TDCS) on post-stroke patients. AIP Conf Proc. American Institute of Physics Inc.; 2021;2344(1). DOI: 10.1063/5.0047216/748987

24. Yoon J-Y, Son M-N, Jo Y-J, Kim D-S, Kim G-W, Won Y-H, et al. Feasibility and effectiveness of remotely supervised tDCS combined with computerized cognitive training for patients with stroke: A randomized sham-controlled study. Res Sq. 2021;1–14. DOI: 10.21203/rs.3.rs-146962/v2

25. Chen Y, Zhao Z, Huang J, Wang T, Qu Y. Computer-aided cognitive training combined with tDCS can improve post-stroke cognitive impairment and cerebral vasomotor function: a randomized controlled trial. BMC Neurol. BMC Neurol; 2024;24(1). DOI: 10.1186/S12883-024-03613-3

26. Antonenko D, Fromm AE, Thams F, Kuzmina A, Backhaus M, Knochenhauer E, et al. Cognitive training and brain stimulation in patients with cognitive impairment: a randomized controlled trial. Alzheimers Res Ther. Alzheimers Res Ther; 2024;16(1). DOI: 10.1186/S13195-024-01381-3

27. Qurat-ul-ain, Ahmad Z, Ishtiaq S, Ilyas S, Shahid I, Tariq I, et al. Short term effects of anodal cerebellar vs. anodal cerebral transcranial direct current stimulation in stroke patients, a randomized control trial. Front Neurosci. Frontiers Media S.A.; 2022;16:1035558. DOI: 10.3389/FNINS.2022.1035558/TEXT

28. Wang Y, Xu N, Wang R, Zai W. Systematic review and network meta-analysis of effects of noninvasive brain stimulation on post-stroke cognitive impairment. Front Neurosci. Frontiers Media S.A.; 2022;16:1082383. DOI: 10.3389/FNINS.2022.1082383/TEXT

29. Yang C, Zhang T, Huang K, Xiong M, Liu H, Wang P, et al. Increased both cortical activation and functional connectivity after transcranial direct current stimulation in patients with post-stroke: A functional near-infrared spectroscopy study. Front Psychiatry. Front Psychiatry; 2022;13. DOI: 10.3389/FPSYT.2022.1046849

TABLES AND FIGURES

Table 1. Baseline Characteristics of Study Subjects

Variable	Intervention Group (n=15)	Control Group (n=15)	Total (n=30)	*P* Value
Age (years), Mean ± SD	53.13 ± 9.63	52.60 ± 11.15	52.87 ± 10.24	0.642*
Sex, n (%)				1.000†
Male	8 (53.3)	8 (53.3)	16 (53.3)	
Female	7 (46.7)	7 (46.7)	14 (46.7)	
Educational Level, n (%)				0.338†
Diploma (D3)	1 (6.7)	0 (0.0)	1 (3.3)	
Bachelor's Degree (S1)	5 (33.3)	5 (33.3)	10 (33.3)	
Master's Degree (S2)	0 (0.0)	3 (20.0)	3 (10.0)	
Senior High School	8 (53.3)	5 (33.3)	13 (43.3)	
Junior High School	1 (6.7)	2 (13.3)	3 (10.0)	
Stroke Onset, n (%)				0.483†
1 month	14 (93.3)	13 (86.7)	27 (90.0)	
2 months	1 (6.7)	0 (0.0)	1 (3.3)	
3 months	0 (0.0)	2 (13.3)	2 (6.7)	
Comorbidities, n (%)				1.000†
Hypertension	8 (53.3)	7 (46.7)	15 (50.0)	
Diabetes Mellitus	1 (6.7)	2 (13.3)	3 (10.0)	
Hypertension + Diabetes Mellitus	4 (26.7)	4 (26.7)	8 (26.7)	
None	2 (13.3)	2 (13.3)	4 (13.3)	

*Independent t-test. † Chi-square test.

Table 2. Baseline Distribution of Impaired MoCA-INA Domains

Impaired MoCA-INA Domain	Intervention Group (n=15)	Control Group (n=15)	Total (n=30)	P value
Visuospatial/Executive	13 (86.7%)	14 (93.3%)	27 (90.0%)	1.000†
Naming	3 (20.0%)	7 (46.7%)	10 (33.3%)	0.245†

Attention	11 (73.3%)	12 (80.0%)	23 (76.7%)	1.000†
Language	6 (40.0%)	9 (60.0%)	15 (50.0%)	0.466†
Abstraction	8 (53.3%)	11 (73.3%)	19 (63.3%)	0.269†
Delayed Recall	14 (93.3%)	14 (93.3%)	28 (93.3%)	1.000†
Orientation	10 (66.7%)	13 (86.7%)	23 (76.7%)	0.390†

† Fisher's exact test.

Table 3. Comparison of Global DTABR Before and After Stimulation Between the Intervention and Control Groups

Global DTABR	Intervention Group (n=15) Mean ± SD	Control Group (n=15) Mean ± SD	Between-Group P value
Before stimulation	2.08 ± 0.99	3.25 ± 2.40	0.098*
After stimulation	1.16 ± 0.58	3.97 ± 3.12	0.004*
Within-group P value	**0.001†**	**0.204†**	

**Independent samples t-test.* †Paired samples t-test.

Table 4. Comparison of MoCA-INA Scores Before and After Stimulation Between the Intervention and Control Groups

MoCA-INA	Intervention Group (n=15) Mean ± SD	Control Group (n=15) Mean ± SD	Between-Group P value
Before stimulation	19.73 ± 6.13	19.40 ± 3.58	0.348*
After stimulation	24.87 ± 4.73	20.27 ± 3.90	0.002*
Within-group P value	**0.001†**	**0.078†**	

Comparison between groups using the Mann–Whitney U test. †Comparison within groups using the Wilcoxon signed-rank test.

Table 5. Comparison of Cognitive Domain Scores Before and After Intervention Between Groups

Variable	Intervention		p-value	Control		p-value
	Median	Min–Max		Median	Min–Max	
Executive Function (Pre)	4.00	0.00–5.00	0.018*	3.00	1.00–5.00	0.334*
Executive Function (Post)	5.00	0.00–5.00		3.00	1.00–5.00	
Naming (Pre)	3.00	1.00–3.00	0.317*	3.00	1.00–3.00	0.180*
Naming (Post)	3.00	2.00–3.00		3.00	2.00–3.00	
Attention (Pre)	5.00	1.00–6.00	0.003*	4.00	2.00–6.00	0.670*
Attention (Post)	6.00	1.00–6.00		4.00	3.00–6.00	
Language (Pre)	2.00	1.00–3.00	0.034*	2.00	1.00–3.00	0.317*
Language (Post)	3.00	1.00–3.00		2.00	1.00–3.00	
Abstraction (Pre)	1.00	0.00–2.00	0.157*	1.00	0.00–2.00	0.157*
Abstraction (Post)	2.00	0.00–2.00		1.00	1.00–2.00	
Delayed Recall (Pre)	1.00	0.00–4.00	0.001*	2.00	0.00–3.00	0.180*
Delayed Recall (Post)	5.00	3.00–5.00		2.00	1.00–3.00	
Orientation (Pre)	5.00	1.00–6.00	0.016*	5.00	2.00–6.00	0.129*
Orientation (Post)	6.00	3.00–6.00		5.00	3.00–6.00	
Total Score (Pre)	22.00	7.00–25.00	0.001*	18.00	15.00–26.00	0.078*
Total Score (Post)	28.00	15.00–30.00		19.00	15.00–27.00	

*P-values were calculated using the Wilcoxon signed-rank test for within-group comparisons between pre- and post-intervention measurements. Statistical significance was defined as $p < 0.05$.

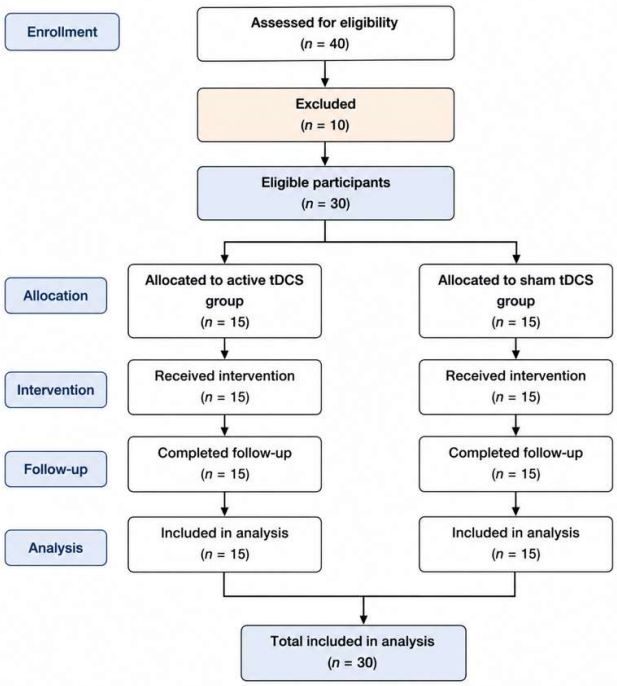

Figure 1. Participant flow diagram of the study.

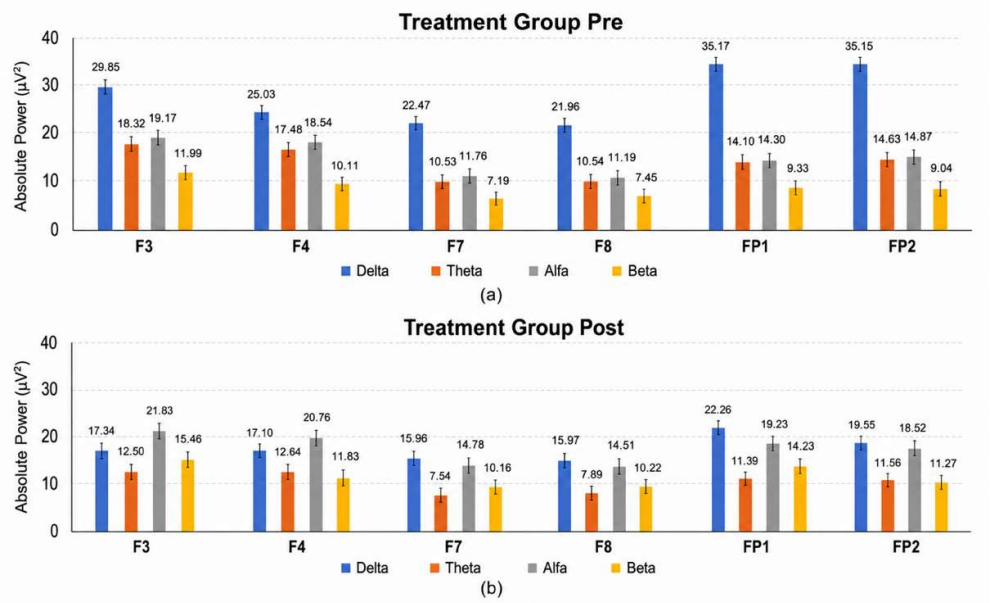

Figure 2. Absolute power values of delta, theta, alpha, and beta waves in the treatment group before and after stimulation. (a) Pre-stimulation absolute power values and (b) post-stimulation absolute power values measured at frontal electrode sites (F3, F4, F7, F8, FP1, and FP2). Values are presented as mean ± SD.

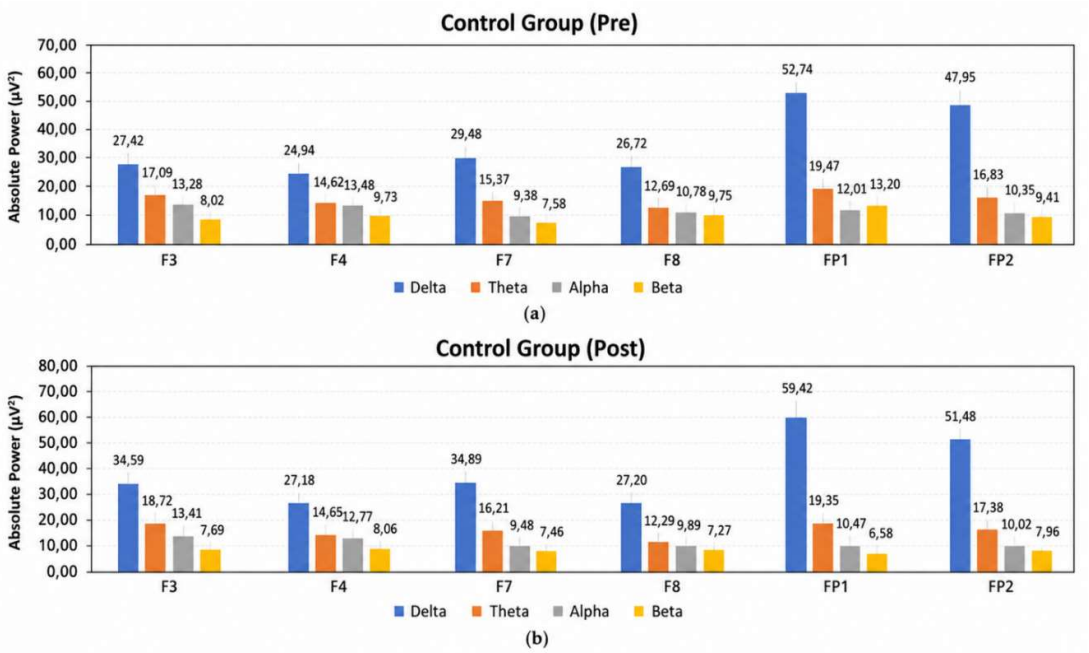

Figure 3. Absolute EEG power of delta, theta, alpha, and beta bands across frontal electrode locations (F3, F4, F7, F8, FP1, and FP2) in the control group before stimulation (a) and after stimulation (b).

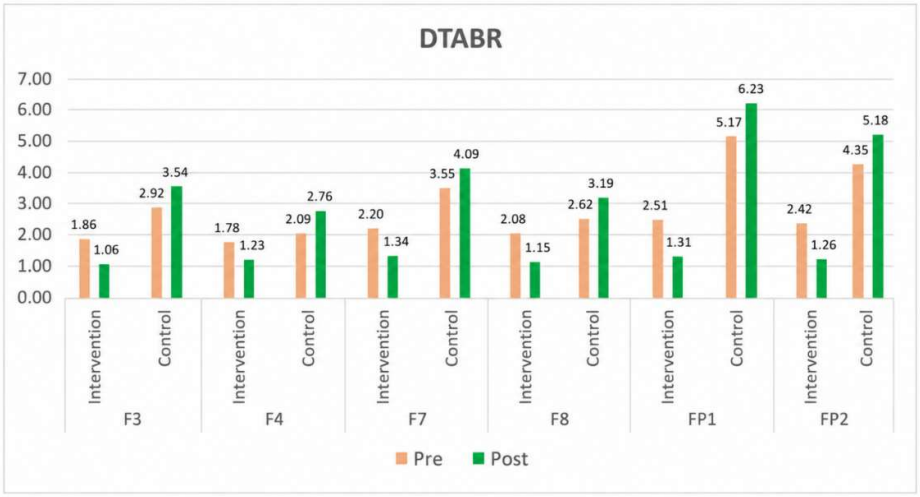

Figure 4. Comparison of delta–theta–alpha–beta ratio (DTABR) between the intervention and control groups at frontal electrode locations (F3, F4, F7, F8, FP1, and FP2) before (Pre) and after (Post) stimulation.

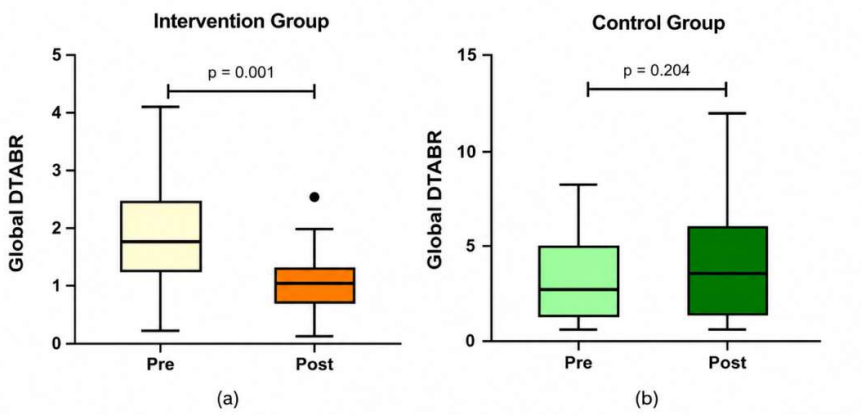

Figure 5. Comparison of median global delta–theta/alpha–beta ratio (DTABR) values before and after stimulation in the (a) intervention group and (b) control group.

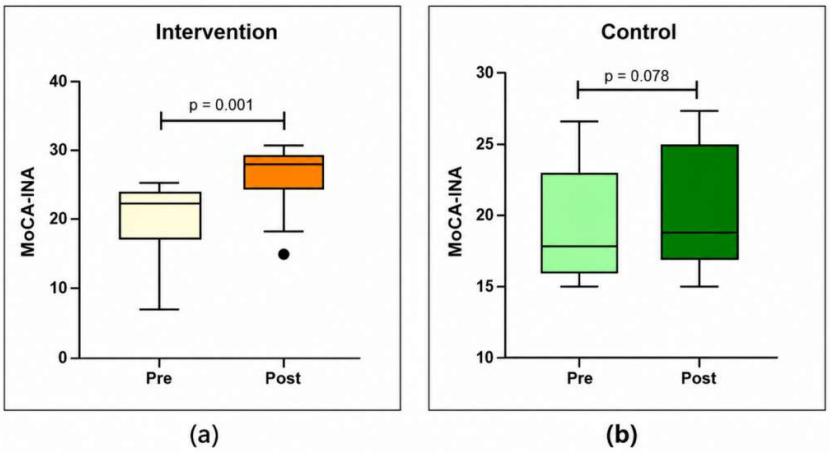

Figure 6. Comparison of median MoCA-INA scores before and after stimulation between the (a) intervention group and (b) control group.