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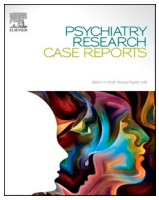

Sequential multimodal neuromodulation for treatment-resistant PTSD: A case report of lasting remission

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ABSTRACT

Treatment-resistant posttraumatic stress disorder (PTSD) often persists despite evidence based treatments. Novel neuromodulation approaches may address this critical unmet need. To describe the feasibility, observed tolerability, and clinical course associated with a sequential multimodal neuromodulation protocol of a sequential multimodal neuromodulation protocol combining transcranial direct current stimulation (tDCS), transcranial magnetic stimulation (TMS), and transcranial pulse stimulation (TPS) in a patient with chronic, treatment-resistant PTSD. A single adult patient with treatment resistant PTSD underwent three sequential neuromodulation phases: (1) tDCS over dorsolateral prefrontal cortex and greater occipital nerve for 12 sessions; (2) high-frequency TMS for 25 sessions; (3) TPS for 9 sessions. Symptom severity was assessed using the PTSD Checklist for DSM-5 (PCL-5) and Hospital Anxiety and Depression Scale (HADS) at baseline, post-each phase, and 3-month follow-up. Symptom reductions exceeded clinical significance after combined treatment: PCL-5 decreased by 60%, anxiety by 23.81%, and depression by 61.9 %. Reductions were maintained at 3 months. No serious adverse events occurred; tolerability was excellent. The Patient Global Impression of Change score was 75%, and the Patient Acceptable Symptom State was reported at 85%, reflecting a high level of patient satisfaction and perceived benefit. Sequential multimodal neuromodulation was safe, well-tolerated, and associated with substantial, sustained symptom improvement in this treatment-resistant PTSD case. These findings warrant controlled trials to optimize protocol synergy and individualize treatment.

Introduction

Posttraumatic stress disorder (PTSD) is a prevalent and often debilitating psychiatric condition that arises following exposure to traumatic events. Epidemiological data suggest that up to 90% of individuals in the United States will experience at least one traumatic event in their lifetime (Kilpatrick et al., 2013). While most individuals demonstrate natural recovery over time, a subgroup develops persistent symptoms that meet the diagnostic criteria for PTSD. The estimated lifetime prevalence of PTSD in the United States is approximately 8% (Kilpatrick et al., 2013).

Although treatments including cognitive behavioural therapy (Bisson et al., 2013) and pharmacological interventions (Hoskins et al., 2021) can be effective for some patients, a significant proportion fails to achieve sustained symptom remission. Consequently, a substantial number of individuals with PTSD remain without adequate therapeutic options, underscoring the urgent need for novel and more effective treatment strategies.

Several clinical investigations have explored neuromodulation techniques—such as transcranial direct current stimulation (tDCS), vagus nerve stimulation, and transcranial magnetic stimulation (TMS)—as promising alternatives to conventional PTSD treatments. Meta-analytic evidence suggests these modalities confer moderate efficacy, demonstrating a small reduction in overall PTSD symptom severity as well as improvements in comorbid anxiety and depressive symptoms (Liu et al., 2024). While these findings are preliminary, they highlight the potential of neuromodulatory interventions to address treatment-resistant cases and expand the current therapeutic arsenal.

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Given the complex and distributed neurocircuitry underlying PTSD—involving dysregulation across the prefrontal cortex, amygdala, hippocampus, and brainstem—targeting a single node with a single neuromodulatory modality may be insufficient to achieve robust and durable clinical effects (Ressler et al., 2022). A growing body of evidence suggests that different neuromodulation techniques, exert their effects through distinct yet complementary mechanisms (Krishna and Fasano, 2024).

Combining these modalities—either concurrently or sequentially—offers a rational strategy to enhance therapeutic efficacy by leveraging their synergistic neurobiological effects. For instance, pairing tDCS with TMS may prime neural networks to respond more robustly to stimulation, while adjunctive other technique such as ultrasound could amplify downstream autonomic and affective regulation (Pellow et al., 2024; Zhou et al., 2024).

Such an integrative approach may not only increase the magnitude and durability of symptom reduction but also improve treatment accessibility by individualizing protocols to target specific symptom clusters or neurophysiological profiles.

Case report

We report the case of patient, 45 years old, male that develop PTSD due to frequent, repeated, and early childhood psychological, physical, and sexual abuse in which the perpetrator was a trusted person. This history and the current triggers impair the this patient's functioning significantly. Because the patient presented with complex PTSD symptoms, including dissociative responses to triggers and episodes of amnesia, repeated self-report measures may not fully capture the clinical picture. The absence of clinician-rated PTSD assessments, dissociation-specific instruments, and a standardized adverse-event checklist is an important limitation. Clinical observations and session-by-session notes were therefore used to contextualize, but not replace, the self-report outcomes.

The patient has previously undergone pharmacological treatment without achieving adequate symptom relief and subsequently received electroconvulsive therapy (ECT). While ECT yielded a positive clinical response, the emergence of adverse effects—namely persistent headache and memory impairment—necessitated discontinuation of this intervention. The patient is also engaged in psychotherapy, which provides supportive coping strategies to a certain extend; however, a referral from the clinical psychologist was made for additional therapeutic options. In view of the persistence of his symptoms, he underwent neuromodulation, after consent to the publication of his case.

At intake, the patient scored 62 on the PTSD Checklist for DSM-5 (PCL-5)(Blevins et al., 2015), representing 77.5% of the maximum possible score. The PCL-5 is a validated self-report measure for assessing PTSD symptom severity, with a diagnostic cut-off range of 31–33. On the Hospital Anxiety and Depression Scale (HADS)(Zigmond and Snaith, 1983), a brief self-assessment tool comprising separate anxiety and depression subscales, the patient scored 12 on the anxiety subscale (57% of the maximum score) and 17 on the depression subscale (80% of the maximum score). Scores of 11 or higher on either subscale are considered clinically significant.

The rationale for combining tDCS, rTMS, and TPS was based on the distributed and multidimensional neurobiology of PTSD. Because chronic PTSD involves dysregulation across prefrontal regulatory systems, limbic threat-processing circuits, memory networks, and autonomic/brainstem pathways, a single stimulation target or modality may be insufficient to induce robust and durable clinical improvement. The protocol was therefore designed as a sequential, multimodal intervention in which tDCS combined with greater occipital nerve stimulation served as a priming and autonomic-regulatory phase, right-DLPFC rTMS targeted prefrontal top–down control of fear and trauma-related cognition, and TPS was used to engage broader frontal, temporal, parietal, and precuneus networks implicated in autobiographical memory, self-referential processing, and dissociation. This staged approach was intended to leverage complementary mechanisms, enhance network plasticity, and address distinct PTSD symptom domains cumulatively rather than relying on a single-node intervention.

The stimulation parameters were selected pragmatically on the basis of available safety data, prior clinical experience with each modality, and the patient's symptom profile, rather than from an established PTSD-specific optimization protocol. At present, no consensus exists regarding optimal stimulation targets, intensity, session number, or sequencing for tDCS, rTMS, or TPS in treatment-resistant PTSD. The initial tDCS phase was intended to modulate prefrontal excitability and affective regulation, while greater occipital nerve stimulation was included to engage afferent pathways potentially relevant to autonomic regulation. The subsequent right-DLPFC rTMS phase was selected because of the role of prefrontal regulatory networks in fear processing, cognitive control, and trauma-related affective dysregulation. TPS was added as a final phase to engage broader cortical networks, including frontal, temporal, parietal, and precuneus regions implicated in autobiographical memory, self-referential processing, and dissociative symptoms. The protocol should therefore be regarded as a personalized, clinically guided intervention rather than an optimized or standardized PTSD neuromodulation protocol.

One month following intake, the patient began a course of bifrontal tDCS consisting of 20-minute sessions at 1.5 mA, followed by 20 min of stimulation targeting the greater occipital nerve at 2.0 mA (To et al., 2017). The rationale for this specific combination was to target both the cognitive-emotional and somatic/arousal-related dimensions of PTSD (Ressler et al., 2022, 2026; Yehuda et al., 2015). PTSD is characterized not only by intrusive memories, hypervigilance, avoidance, and negative mood, but also by disturbances in sleep, bodily tension, pain, fatigue, and autonomic arousal (Ressler et al., 2022; Yehuda et al., 2015). Hence, a neuromodulation approach that engages both frontal regulatory networks and posterior sensory/autonomic pathways may be particularly relevant. Bifrontal stimulation of the dorsolateral prefrontal cortex (DLPFC) was selected because the DLPFC is involved in executive control, emotional regulation, attentional control, and top-down modulation of limbic activity (Banich et al., 2024), processes that are often disrupted in PTSD (Ressler et al., 2022; Yehuda et al., 2015). Enhancing prefrontal regulation may therefore help reduce symptoms such as hyperarousal, intrusive re-experiencing, impaired cognitive control, and affective dysregulation. Occipital stimulation was included because the greater occipital nerve region is connected with brainstem, thalamic, and limbic networks involved in pain perception, arousal, and bodily threat processing (Luckey et al., 2023). This target may be especially relevant for PTSD patients with prominent somatic symptoms, chronic pain, headaches, fatigue, or heightened physiological arousal (Ressler et al., 2022; Yehuda et al., 2015). The combination allows to produce distinct therapeutic effects in PTSD. That is, DLPFC stimulation may primarily influence cognitive-emotional regulation, whereas occipital stimulation may more strongly affect somatic, pain-related, and arousal-related symptoms. This follows the same target-specific logic used in the original fibromyalgia study, where DLPFC and C2 stimulation were compared because they were expected to influence different symptom dimensions (To et al., 2017). This protocol was administered over 20 sessions (three sessions per week). Upon completion, symptom reduction was observed across multiple domains: PCL-5 decreased by 7%, HADS-Anxiety by 33.3%, and HADS-Depression by 23.8% (see also Fig. 1).

Following a one-month treatment-free interval, the patient underwent TMS, targeting the right dorsolateral prefrontal cortex (DLPFC), delivered at 55% of machine output and 10 Hz for 25 sessions (one session per day/5 sessions a week). Many psychiatric rTMS protocols use between 20 and 30 sessions (Perera et al., 2016). At the TMS baseline assessment, conducted immediately before the first TMS session, minimal additional change was observed: PCL-5 scores decreased by 1.25%, depression scores by 4.76%, while anxiety scores increased by 9.52%.

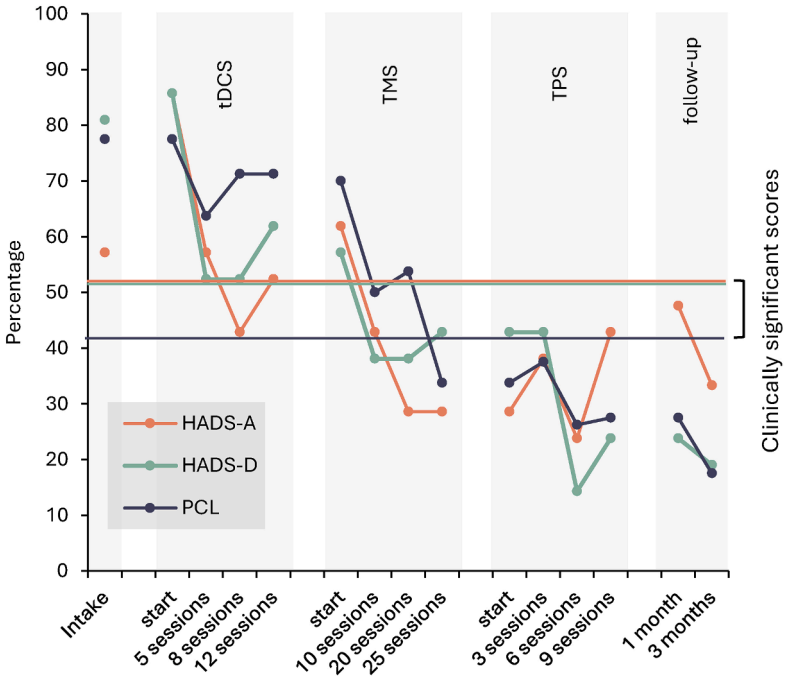

Fig. 1. Longitudinal overview of symptom severity scores on the PCL-5, HADS-Anxiety, and HADS-Depression scales at baseline (intake), during neuromodulation treatment (tDCS, TMS, and TPS sessions), and at follow-up to 3 months post-treatment. The straight horizontal lines indicate the minimal clinically important difference (MCID) thresholds for each questionnaire.

However, by the end of the TMS course, PCL-5 scores had decreased by 36.25%, anxiety by 33.3%, and depression by 14.29%, with all scores falling below clinically significant thresholds (see also Fig. 1). This was again followed by a one-month observation period with no further symptom changes.

Subsequently, the patient received transcranial pulse stimulation (TPS) (Matt et al., 2022), a modality that uses focused ultrasound pulses to target deeper cortical and subcortical structures. TPS was administered in nine sessions, delivering 6000 pulses per session. The dose and regional distribution were selected pragmatically on the basis of available TPS safety and feasibility data, prior clinical protocols, and the aim of targeting cortical networks relevant to PTSD-related cognitive-emotional regulation, autobiographical memory, self-referential processing, and dissociative symptoms: 1000 pulses to the frontal lobe, 600 to the temporal lobe, 800 to the parietal lobe bilaterally, and 1200 to the precuneus (Cheung et al., 2022; Cont-Richter et al., 2025; Dorl et al., 2022; Matt et al., 2022). Although clinical stability following the TMS phase, we decided to include TPS because it formed part of the predefined sequential multimodal neuromodulation plan. The decision to proceed with TPS was made a priori, based on the rationale that TPS could engage broader cortical networks implicated in autobiographical memory, self-referential processing, and dissociative symptoms, rather than being introduced in response to symptom worsening or relapse (Vanneste et al., 2025). After the TPS phase, further reductions were observed: PCL-5 scores decreased by 6.25% (final score: 27.5), and depression by 19.05%. However, anxiety scores increased by 14.29% (see also Fig. 1).

A three-month follow-up post-treatment showed sustained and significant symptom reduction: PCL-5 decreased by 60%, HADS-Anxiety by 23.81%, and HADS-Depression by 61.90% from baseline, indicating lasting therapeutic benefit. All neuromodulation treatments were well tolerated and no adverse events were reported. The *Patient Global Impression of Change* score was 75%, and the *Patient Acceptable Symptom State* was reported at 85%, reflecting a high level of patient satisfaction and perceived benefit.

Adverse events were monitored clinically through session-by-session inquiry and clinical observation, but no standardized adverse-event scale was administered. Mild transient headache was reported during early stimulation sessions, and during TPS the patient reported bodily agitation/tension and transient sleep difficulty. No serious adverse events occurred, and all treatment phases were completed

Discussion

This case report describes clinically meaningful symptom improvement temporally associated with a sequential, multimodal neuromodulation strategy in a patient with treatment-resistant PTSD. The integration of tDCS, rTMS, and TPS—each targeting distinct neurophysiological mechanisms—was associated with clinically meaningful and sustained reductions in PTSD, anxiety, and depressive symptoms. These findings are consistent with the possibility that sequential multimodal neuromodulation may be clinically useful, but they do not establish efficacy, mechanism, or synergy. While encouraging, these results warrant validation through controlled, mechanistic studies to determine optimal protocols, dosing parameters, and individual predictors of treatment response. A multimodal neuromodulation framework represents a promising frontier in PTSD treatment and warrants systematic investigation through well-powered, mechanistically informed clinical trials.

PTSD is characterized by dysfunction across distributed networks involving prefrontal regulatory regions, limbic threat-processing systems, and brainstem/autonomic circuits(Fenster et al., 2018). Each neuromodulation modality used in this case plausibly engages distinct elements of this network. The initial tDCS phase, targeting bilateral prefrontal cortex and the greater occipital nerve, was conceptualised as a possible priming intervention, enhancing cortical excitability and modulating afferent autonomic input (Vanneste et al., 2020). The early reductions observed in anxiety and depressive symptoms following tDCS are consistent with prior work suggesting that low-intensity electrical stimulation can preferentially influence affective regulation and stress reactivity (Zheng et al., 2024).

The subsequent rTMS phase targeting the right DLPFC was

associated with the largest reduction in core PTSD symptoms. This finding aligns with models implicating the DLPFC in top–down regulation of fear responses, cognitive control over trauma-related memories, and emotional reappraisal (Patel et al., 2024). Notably, the delayed but robust response to rTMS suggests that network-level plasticity, rather than immediate symptom suppression, may underlie its therapeutic effects. The relative stability of symptom gains during the post-TMS observation period further supports the notion of enduring circuit reorganization.

Finally, TPS was associated with incremental improvement in PTSD and depressive symptoms, potentially reflecting its ability to engage deeper cortical and subcortical regions not readily accessible via conventional non-invasive stimulation. The inclusion of parietal and precuneus targets is of particular interest, given their involvement in self-referential processing, autobiographical memory, and dissociative symptoms—domains often disrupted in complex PTSD (Agathos et al., 2024). Although anxiety scores transiently increased during the TPS phase, this may reflect heightened interoceptive awareness or emotional processing rather than clinical deterioration, especially in light of the sustained improvements observed at follow-up.

An important observation from this case is the nonlinear and domain-specific trajectory of symptom change. Anxiety, depression, and PTSD severity did not improve uniformly across phases, underscoring the heterogeneity of PTSD symptom clusters and their neurobiological substrates. Fluctuations in anxiety, particularly during transitions between modalities, may reflect adaptive network recalibration rather than treatment failure. This highlights the importance of longitudinal monitoring and avoiding premature conclusions based on short-term symptom variability.

From a clinical perspective, the fact that meaningful improvement emerged cumulatively rather than after a single intervention suggests that sequential neuromodulation may overcome ceiling effects commonly observed with monotherapy in treatment-resistant populations. This is particularly relevant for patients who have failed pharmacotherapy, psychotherapy, and even electroconvulsive therapy, as in the present case.

The maintenance of symptom reduction at three-month follow-up strengthens the clinical relevance of the findings. Sustained improvements across PTSD, anxiety, and depressive symptoms—combined with high Patient Global Impression of Change and Patient Acceptable Symptom State ratings—suggest that the observed effects were not only statistically or numerically meaningful but also personally and functionally significant for the patient. However, because Patient Global Impression of Change and Patient Acceptable Symptom State ratings are patient-reported outcomes, they may be particularly sensitive to expectancy, therapeutic alliance, increased clinical attention, and the perceived novelty of the intervention, these ratings should therefore be interpreted as indicators of perceived clinical benefit rather than objective evidence of treatment-specific efficacy. Durability remains a critical benchmark for neuromodulation interventions, and the present case adds preliminary evidence that multimodal approaches may yield longer-lasting benefits than single-modality treatments.

Several limitations should be acknowledged. First, this is a single case report without a sham condition, control group, or randomized design; therefore, causal attribution cannot be made. Symptom improvement was temporally associated with sequential neuromodulation, but placebo effects, expectancy, regression to the mean, spontaneous fluctuation, increased clinical attention, and nonspecific therapeutic factors cannot be excluded. This is particularly relevant because the intervention was intensive, novel, and delivered in a highly structured clinical context, all of which may influence patient-reported outcomes, including PGIC and PASS ratings. Second, the mechanistic interpretation remains speculative. Although the symptom trajectory is compatible with modulation of prefrontal, limbic, autobiographical-memory, and autonomic networks, no EEG, neuroimaging, autonomic, or other biomarker measures were obtained to confirm target engagement or network-level change. Third, the stimulation parameters and sequencing were clinically selected and informed by prior literature and feasibility considerations, but they cannot be considered optimized for PTSD. Fourth, because tDCS, rTMS, and TPS were delivered sequentially, the independent contribution of each modality and the extent of any additive or synergistic effect cannot be determined. These limitations underscore the need for sham-controlled, biomarker-informed studies comparing unimodal and multimodal protocols. Because PTSD related to childhood trauma may involve dissociation, amnesia, emotional avoidance, and fluctuating self-awareness, repeated self-report questionnaires may not fully capture the patient's clinical state. The absence of clinician-rated PTSD and dissociation-specific instruments is an important limitation

Future studies should systematically evaluate optimal sequencing, dosing, and spacing of neuromodulation modalities, ideally guided by biomarkers such as EEG, functional connectivity measures, or symptom-network profiling. Controlled trials comparing multimodal versus unimodal neuromodulation, as well as adaptive designs that tailor stimulation based on intermediate response, may further clarify which patients are most likely to benefit from this approach.

Conclusion

In conclusion, this case report supports the feasibility and tolerability of a personalized sequential neuromodulation approach in severe treatment-resistant PTSD and describes sustained symptom improvement over three months. However, because this was an uncontrolled single case without objective biomarkers, the findings should be interpreted as preliminary and hypothesis-generating. Controlled, sham-comparator studies with neurophysiological or neuroimaging measures are required to determine efficacy, mechanism, optimal parameters, and the specific contribution of each neuromodulation modality.

Consent

Written informed consent for publication of this case, including anonymized clinical details, was obtained from the patient, and can be submitted at request.

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CRediT authorship contribution statement

Sven Vanneste: Writing – review & editing, Writing – original draft, Visualization, Validation, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Jan Ost:** Writing – original draft, Methodology, Investigation. **Dirk De Ridder:** Writing – review & editing, Writing – original draft, Visualization, Validation, Project administration, Methodology, Investigation, Formal analysis.

Declaration of competing interest

The authors declare that they have no conflicts of interest, financial or personal, that could have influenced the work presented in this paper.

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