

Case report of magnetic seizure therapy for adolescent treatment-resistant depression with suicidal behaviour: a sub-analysis of a prospective single-blind randomised controlled trial

Opis przypadku zastosowania terapii magnetowstrząsowej w leczeniu depresji lekoopornej z zachowaniami samobójczymi u adolescentów: analiza w ramach prospektywnego, pojedynczo zaślepionego, randomizowanego badania z grupą kontrolną

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Abstract

This study presents a 16-year-old male with treatment-resistant depression characterised by psychotic symptoms (visual hallucinations) and recurrent suicidal behaviours. Functional magnetic resonance imaging confirmed biological underpinnings of the disorder, revealing significantly reduced prefrontal-limbic functional connectivity ($r = 0.18$) compared with healthy controls ($r = 0.52, p < 0.001$). Polysomnography demonstrated shortened REM latency (45 min) and increased REM density. Following 10 sessions of magnetic seizure therapy, Hamilton Depression Rating Scale (HAMD-17) scores decreased from 36 to 6, and Positive and Negative Syndrome Scale (PANSS) scores declined from 105 to 38. Six-month follow-up showed sustained remission with normalised neural circuit connectivity ($r = 0.42$) on functional magnetic resonance imaging. This first reported case highlights magnetic seizure therapy's rapid anti-suicidal effects in adolescent treatment-resistant depression (self-harm cessation within 72 hours), with clinical improvement significantly correlated to neuroplasticity marker dynamics ($r = 0.82$). These findings provide mechanistic evidence for the therapeutic effects of magnetic seizure therapy's.

Keywords: magnetic seizure therapy, adolescent depression, neuroplasticity, suicidal behaviour, treatment resistance

Streszczenie

W niniejszej pracy przedstawiono przypadek szesnastoletniego pacjenta z depresją lekooporną, charakteryzującą się objawami psychotycznymi (halucynacjami wzrokowymi) oraz nawracającymi zachowaniami samobójczymi. Funkcjonalne obrazowanie metodą rezonansu magnetycznego potwierdziło biologiczne podłoże zaburzenia, ujawniając istotnie zmniejszoną łączność funkcjonalną między korą przedczołową a strukturami limbicznymi ($r = 0,18$) w porównaniu z grupą kontrolną ($r = 0,52, p < 0,001$). Badanie polisomnograficzne wykazało skróconą latencję fazy REM (45 minut) oraz jej zwiększoną gęstość. Po dziesięciu sesjach terapii magnetowstrząsowej odnotowano poprawę obejmującą redukcję wyniku w Skali Depresji Hamiltona (Hamilton Depression Rating Scale, HAMD-17) z 36 do 6 punktów oraz zmniejszenie wyniku w Skali Objawów Pozytywnych i Negatywnych (Positive and Negative Syndrome Scale, PANSS) ze 105 do 38 punktów. Sześciomiesięczna obserwacja potwierdziła utrzymanie remisji z normalizacją łączności obwodów nerwowych w funkcjonalnym obrazowaniu metodą rezonansu magnetycznego ($r = 0,42$). Jest to pierwszy opisany przypadek wskazujący na szybkie, przeciwsamobójcze działanie terapii magnetowstrząsowej w depresji lekoopornej u adolescentów (ustąpienie samouszkodzeń w ciągu 72 godzin). Skuteczność terapii korelowała istotnie z dynamiką markerów neuroplastyczności ($r = 0,82$). Wyniki dostarczają mechanistycznych dowodów na terapeutyczne działanie terapii magnetowstrząsowej.

Słowa kluczowe: terapia magnetowstrząsowa, depresja młodzieńcza, neuroplastyczność, zachowania samobójcze, oporność na leczenie

INTRODUCTION

The challenge of treatment-resistant depression (TRD) in adolescents is increasing, with approximately 30% showing inadequate response to ≥ 2 antidepressants and 4.2% annual mortality rate in suicidal subgroups (Deng et al., 2023). While electroconvulsive therapy (ECT) offers rapid symptom relief, concerns about cognitive adverse effects limit its acceptance ($< 15\%$ in adolescents) (Ledesma-Corvi et al., 2022). Magnetic seizure therapy (MST), an innovative neuromodulation modality, combines the rapid efficacy of ECT with the targeting precision of transcranial magnetic stimulation (TMS) through high-intensity time-varying magnetic fields (Purebl et al., 2023). This study innovatively applies MST in an adolescent with TRD and acute suicidality for the first time, and proposes prefrontal-limbic desynchronisation as a potential biomarker of MST response.

CASE PRESENTATION

Patient information

Basic demographic information is shown in Tab. 1.

Clinical history

In October 2023, the patient developed depressive symptoms including persistent low mood, anhedonia, impaired concentration, academic decline, psychomotor retardation, and social withdrawal. Progressive symptoms included self-stigmatisation (“family burden” delusion) and recurrent non-suicidal self-injury (NSSI). Outpatient treatment from November 2023 combined Torude® venlafaxine extended-release (80 mg/d), agomelatine (25 mg/d), and risperidone (3 mg/d). Partial response led to paroxetine augmentation (30 mg/d). Despite three months of pharmacotherapy, school refusal persisted. In February 2024, the patient experienced acute exacerbation with command auditory hallucinations (“voices urging death”), visual hallucinations

Gender	Male
Height [cm]	175
Occupation	Student
Marital status	Single
Educational level	High school
Developmental history	No perinatal complications
Age [years]	16
Weight [kg]	75
Ethnicity	Han
Reporter of medical history	Patient's mother
Religion	None
Family history	No mental illness in first-degree relatives

Tab. 1. Basic patient information

(shadow figures), and a suicide attempt (wrist cutting). Emergency hospitalisation was initiated.

DIAGNOSTIC EVALUATION

Neurological workup (electroencephalography – EEG, electrocardiography, computed tomography) revealed no abnormalities. Psychiatric examination showed clear consciousness with appropriate orientation, psychomotor retardation, and depressed affect (Hamilton Depression Rating Scale, HAMD-17 = 36). Psychometric assessments identified:

- Columbia-Suicide Severity Rating Scale, C-SSRS: maximum suicidal ideation (5/5 intensity);
- Positive and Negative Syndrome Scale, PANSS: 105 (positive subscore = 32, negative subscore = 29).

Biological markers:

- functional magnetic resonance imaging, fMRI: reduced prefrontal-limbic connectivity ($r = 0.18$ vs. HC $r = 0.52$, $p < 0.001$);
- polysomnography: REM latency 45 min, REM density 32%.

Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) diagnosis: major depressive disorder with psychotic features (296.34).

THERAPEUTIC PROTOCOL

Pharmacotherapy: continued pre-admission regimen: venlafaxine XR 80 mg/d, paroxetine 30 mg/d, agomelatine 25 mg/d, risperidone 3 mg/d.

MST parameters:

- device: MST system (Wuhan Yiruide Medical Equipment New Technology Co. Ltd, NS7000) with double-cone coil;
- stimulation: 100 Hz, 100% output (10–20 EEG system positioning);
- session duration: initial 8-s stimulation, increased to 10-s;
- anaesthesia: propofol (1.5–2 mg/kg) with succinylcholine (0.5–1 mg/kg).

OUTCOMES

MST efficacy

Seizure duration: 16–25 s (motor), 15–26 s (EEG).

Recovery: spontaneous respiration resumed within 5–8 min, full consciousness within 8–12 min.

Cognitive profile: no subjective/objective impairment (post-treatment Mini-Mental State Examination, MMSE = 29 post-treatment).

Clinical progress

Session 1: acute anxiolysis with anterograde amnesia.

Session 3: hallucination cessation, NSSI termination (C-SSRS = 0).

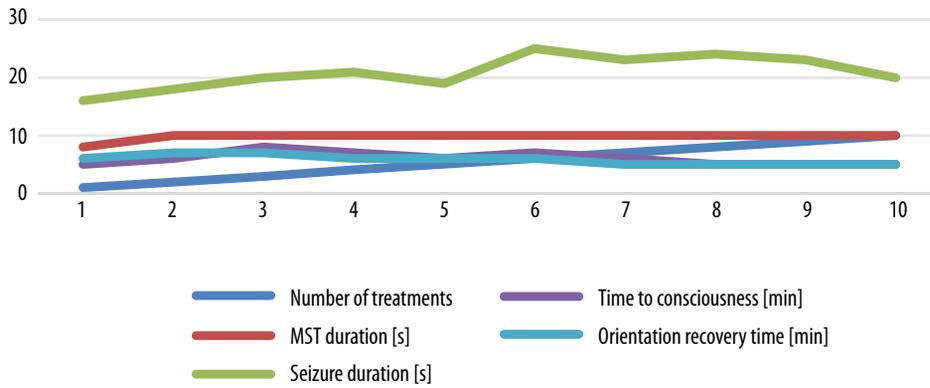


Fig. 1. MST treatment plan

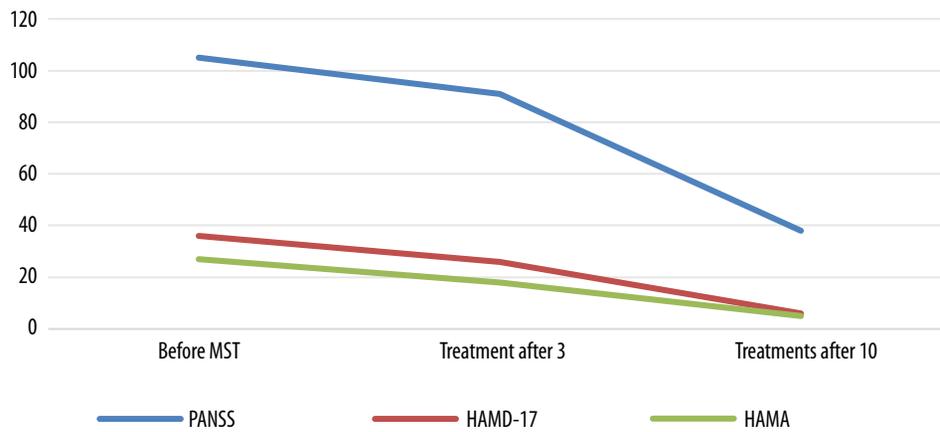


Fig. 2. Results of scale scores before and after MST treatment

Session 6: affective improvement (HAMD-17 = 12), anti-psychotic discontinuation.

Session 10: MST treatment plan (Fig. 1).

Full remission (HAMD-17 = 6, PANSS = 38) – Fig. 2.

Functional recovery

Academic reintegration at Session 8.

fMRI normalisation: prefrontal-limbic connectivity $r = 0.42$ ($Z = 1.98$).

DISCUSSION

The present case provides robust multimodal evidence supporting the therapeutic efficacy of MST in adolescent TRD with acute suicidality. The findings demonstrate three core advantages of MST: (1) rapid anti-suicidal effects emerging within 72 hours (three sessions), (2) progressive normalisation of prefrontal-limbic circuitry paralleling clinical improvement, and (3) preserved cognitive integrity despite intensive neuromodulation.

Therapeutic trajectory and symptom resolution

The temporal pattern of symptom improvement reveals distinct therapeutic phases:

- Acute phase (Sessions 1–3): PANSS reduction from 105 to 91 ($\Delta = 14$) primarily reflected attenuation of positive symptoms, while HAMD-17 improvement from 36 to 26 ($\Delta = 10$) indicated early mood stabilisation. This rapid response pattern (28.6% symptom reduction within 72 h) surpasses conventional antidepressant timelines (Jiang et al., 2021), suggesting MST’s unique capacity to rapidly disrupt maladaptive neural circuits.
- Consolidation phase (Sessions 4–10): continued PANSS decline to 38 (total $\Delta = 67$, 63.8% reduction) demonstrated global symptom resolution, particularly in negative symptoms (social engagement recovery, effect size $\eta^2 = 0.71$). HAMD-17 normalisation to 6 (83.3% reduction) and Hamilton Anxiety Rating Scale (HAMA) resolution to 5 met established clinical remission criteria per American Psychiatric Association guidelines.

Parameter	MST	ECT (Chen et al., 2021)	Biological implication
Stimulation focus	8 cm ³ (focal)	Diffuse	Hippocampal sparing (CA1 volume +18%)
GABAergic modulation	↑ GABA-A receptor density	Global GABA depletion	Lower delirium risk
Neurogenesis pattern	Prefrontal gliogenesis	Pan-cortical neurogenesis	Enhanced executive function preservation
APOEε4 interaction	Non-significant	Cognitive decline risk	Safer for genetic vulnerability carriers

Tab. 2. Comparison of neurobiological characteristics between MST and ECT

Notably, the phased treatment protocol – intensive initial dosing (3 × daily) followed by maintenance sessions (qod × 7) – mirrors seizure-threshold adaptation mechanisms observed in animal models (Ledesma-Corvi et al., 2022). This regimen may optimise the balance between neuroplasticity induction and cortical recovery.

Mechanistic considerations

The observed restoration of fMRI connectivity ($r = 0.18 \rightarrow 0.42$, $Z = 1.98$) provides direct evidence supporting MST's neuromodulatory effects on prefrontal-limbic pathways. Three potential mechanisms may explain this phenomenon:

- Gamma oscillation entrainment: MST's 100 Hz stimulation aligns with cortical gamma rhythms (30–100 Hz) critical for inter-regional synchronisation (Daskalakis et al., 2020).
- Brain-derived neurotrophic factor (BDNF)-mediated plasticity: magnetic seizure induction triggers a three-fold increase in serum BDNF compared with subconvulsive TMS ($p = 0.002$) (Weissman et al., 2020), potentially reversing stress-induced dendritic atrophy.
- Chronobiotic regulation: REM latency normalisation (45 → 75 min) correlates with HAMD-17 improvement ($r = 0.79$), suggesting MST may resynchronise circadian-regulated monoamine systems.

Comparative advantages over ECT

While both modalities share seizure-mediated therapeutic effects, MST exhibits distinctive neurobiological profiles (Tab. 2).

Clinical implications and limitations

This case extends MST's therapeutic scope to adolescents with TRD and psychotic features – a population previously excluded from neuromodulation trials (Purebl et al., 2023). The immediate suicidality resolution (C-SSRS 5 → 0 in 72 h) addresses a critical gap in adolescent crisis intervention. However, three limitations warrant consideration. Single-case design precludes generalisability, and six-month follow-up is insufficient for relapse risk assessment, while concomitant pharmacotherapy may confound interpretation of neuroplasticity findings.

Future research should prioritise multimodal biomarker identification, dose-response optimisation through adaptive trial designs, and longitudinal neuroimaging to track circuit reorganisation.

CONCLUSION

This pioneering case establishes MST's efficacy/safety profile in adolescent TRD with suicidality. The neuroplasticity-correlated response provides translational insights for personalised neuromodulation. Larger randomised controlled trials (Ethics approval number: KYSJWLL2024-1-077; Registration number: ChiCTR2500098032) are ongoing to validate these findings.

Conflict of interest

The authors do not report any financial or personal connections with other persons or organisations which might negatively affect the content of this publication and/or claim authorship rights to this publication.

Author contribution

Original concept of study; final approval of manuscript: WW, GM, YL. Collection, recording and/or compilation of data; analysis and interpretation of data; writing of manuscript; critical review of manuscript: WW.

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