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Restimulation could stop status epilepticus after electroconvulsive therapy: 2 case reports

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Background: Electroconvulsive therapy (ECT) is an effective treatment for severe depression, mania, psychosis and catatonia. While seizures are considered essential for the therapeutic effect of ECT, it concurrently has an anticonvulsant effect which plays a role in its mechanism of action. This property has also prompted the use of ECT in managing status epilepticus (SE).

Case Presentation: We report two distinct cases of prolonged seizures during ECT that persisted for more than 5 min despite administration of propofol and lorazepam, ultimately meeting criteria for status epilepticus (SE). The first case involved an 80-year old woman with severe psychotic depression starting ECT, while the second case involved a 30-year old man receiving maintenance ECT for difficult-to-treat schizophrenic psychosis. In both cases, SE was promptly terminated by restimulation, defined as an additional stimulus delivered within the same ECT session. After epilepsy and intracranial pathology were ruled out, ECT was safely resumed in both patients after switching from etomidate to propofol induction.

Conclusion: Status epilepticus after ECT can be resolved by restimulation when standard interventions are unsuccessful, thereby avoiding potential neurological complications. We provide an overview of the mechanism and current clinical evidence supporting this strategy, and propose an amended clinical practice protocol for SE after ECT.

KEYWORDS

electroconvulsive therapy, status epilepticus, prolonged seizure, mechanism, complication, anticonvulsant hypothesis, restimulation

1 Introduction

Electroconvulsive therapy (ECT) is an effective treatment for difficult-to-treat depression, particularly in older patients and when psychotic features are present (1). It is also a second-line treatment for clozapine-resistant psychosis (2). While there is an abundance of evidence for the efficacy and safety of ECT (3), the mechanism of action remains unresolved. ECT was developed in 1938 as a safe way to elicit a seizure, as it was believed that seizures counteracted psychosis (4). The three currently most accepted hypotheses still stem from the assumption that seizures are directly involved in the therapeutic effect of ECT. The generalized seizure hypothesis posits that the therapeutic effect of ECT is dependent on the elicitation of generalized seizures (5), while the combined anatomical-ictal hypothesis suggests that therapeutic effect is driven by seizure activity in the limbic system which induces neurotrophic effects through brain derived neurotrophic factor (BDNF) (6, 7). The anticonvulsant hypothesis suggests that the therapeutic effect of ECT originates from an increased inhibitory GABA-ergic neurotransmission, as the seizure threshold often rises during a course of ECT (8, 9). This phenomenon has facilitated the use of ECT in status epilepticus (SE) (10). Status epilepticus is defined by the International League Against Epilepsy (ILAE) as a generalized seizure lasting more than 5 min, which is considered a practical time point for initiating treatment, or more than 30 min, beyond which significant risk of long-term neuronal injury and functional deficits arise (11).

Prolonged seizures after ECT are seizures of >180 seconds occurring in 1-2% of ECT courses (12) and are typically managed by intravenous anesthesia or benzodiazepines (13). However, in rare cases these interventions are ineffective leading to SE (14). Tardive seizures after ECT, meaning seizure activity after termination of the therapeutic seizure, can also occur (15). Managing SE poses significant clinical challenges. Evidence guiding interventions is limited and entails general intensive care, antiepileptic drugs and treatment of underlying pathology (11, 16). We illustrate the paradoxical relationship between seizure and ECT by presenting two cases where SE following ECT was promptly managed by restimulation.

2 Case presentation

2.1 Case A

2.1.1 Patient information

Ms. A, an 80-year-old woman, was admitted for severe depression with psychotic features. She had no prior psychiatric or neurological history, and no known family history of depression or epilepsy. She had a history of breast cancer with bone metastasis diagnosed in the previous year. She had been treated with escitalopram 15 mg and mirtazapine 15 mg for three months before admission without any clinical improvement. Further medication consisted of letrozole 2.5 mg.

2.1.2 Clinical findings and diagnostic assessment

The patient exhibited depressed mood, anhedonia, cognitive impairment, and psychotic features such as nihilistic delusions and paranoid behavior. Her Montgomery-Åsberg Depression Rating Scale (MADRS) (17) score was 40/60, and her CORE score was 15, suggestive of a melancholic depression (18). Upon admission, olanzapine 5 mg was added to the regimen which showed no effect after the first week. Given the severity of her symptoms, ECT was advised and started after informed consent by proxy was granted by the patient's family. Pre-ECT evaluations, including EKG and laboratory tests, were unremarkable.

2.1.3 Therapeutic intervention

Right unilateral ECT twice a week was started using a square-wave, brief-pulse, constant-current device (MECTA SR1-5000Q; Lake Oswego, Oregon). Figure 1a shows a timeline of the index ECT. Anesthesia consisted of etomidate 12 mg, succinylcholine 35 mg and 100% oxygen. The seizure threshold was established by empirical titration (see Table 1). The second titration step resulted in a threshold seizure, followed by a therapeutic stimulus at 6 times seizure threshold. The following seizure exceeded 2 min on electroencephalogram (EEG). Per hospital protocol (see Table 2a), propofol 60 mg was administered, followed by lorazepam 2 mg at 4 min and an additional 2 mg at 6 min. Despite these interventions, EEG showed sustained spike and wave activity consistent with SE

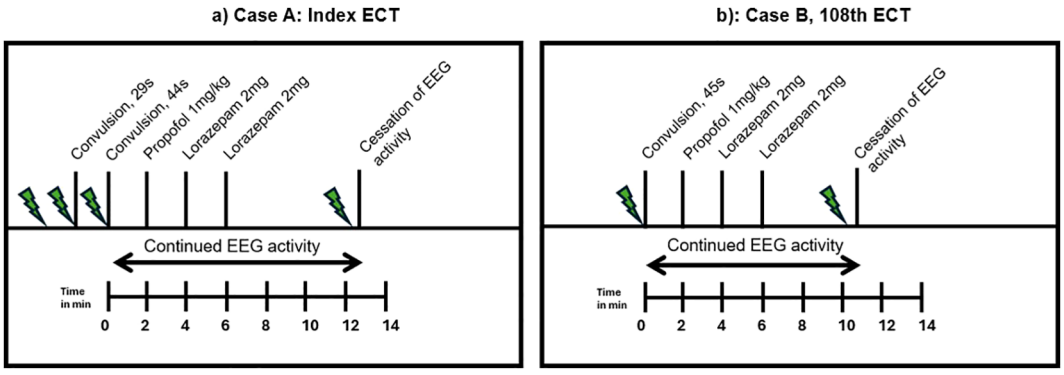

FIGURE 1
(a) Case A, Index ECT. **(b)** Case B, 108th ECT.

TABLE 1 Stimulus parameters for Case A and B.

Stimulus Nr.	Stimulus type	Electrode position	Pulsewidth	Pulse frequency	Train	Charge
Case A						
1	Titration	RUL	0.5ms	20Hz	1.75s	28mC
2	Titration	RUL	0.5ms	20Hz	3.25s	52mC
3	Therapeutic	RUL	0.5ms	50Hz	8s	320mC
4	Termination	RUL	0.5ms	50Hz	8s	320mC
Case B						
1	Therapeutic	BT	0.5ms	20Hz	3.75	60mC
2	Termination	BT	0.5ms	20Hz	3.75	60mC

RUL, Right unilateral electrode position; BT, Bitemporal electrode position.

(Figure 2). The clinical team decided to administer another stimulus using the same parameters applied 15 min after the first. After restimulation, EEG monitoring showed immediate cessation of seizure activity, followed by postictal suppression.

2.1.4 Follow-up and outcomes

Post-ECT, Ms. A was closely monitored and her vital signs and neurological status remained stable. A neurologist (EB) evaluated the patient, and subsequent 24-channel EEG showed no epileptic activity. A CT ruled out intracranial pathology, including breast cancer metastasis. After weighing risks and benefits with the patient's family, ECT was resumed using propofol for induction. The patient tolerated subsequent sessions without complications. After 8 sessions her MADRS score decreased to 4 and CORE score to 0, indicating remission. She was discharged with maintenance ECT and continued to do well at follow-up after 6 months.

TABLE 2 Protocol to manage prolonged seizures after ECT.

a. Current protocol at University Psychiatric Center KU Leuven	
Duration of convulsion	Action
1 min 30 seconds	Prepare propofol 1 mg/kg
2 min	Administer propofol 1 mg/kg
4 min	Administer lorazepam 2 mg
6 min	Administer lorazepam 2 mg
b. Proposed amendment based on current report	
10–15 min	Consider restimulation
15 min	Transfer to expert acute neurological care
Follow up:	· 24h in-hospital monitoring · Neurological consult with 24-channel EEG to rule out epilepsy · Consider intracranial imaging to rule out intracranial pathology · Consider stopping medication that lowers seizure threshold · Switch to propofol induction if ECT would be resumed

2.1.5 Patient perspective

Ms. A recalls little about her depressive symptoms and often wonders what she was doing in the months before her hospitalization. She felt well-informed about side effects before and during treatment. As she was unconscious during the status epilepticus and still severely depressed afterward, she has limited recollection of discussions about the events. Her son, who was also informed, felt he received adequate explanations regarding what happened. Both Ms. A and her family emphasize that they mainly remember the rapid and complete remission of depression after ECT sessions. They also emphasized the importance of the kindness and warmth of the ECT team as a key aspect of her care. At the time of writing maintenance ECT was discontinued and Ms. A remains in remission.

2.2 Case B

2.2.1 Patient information

Mr. B was a 30-year old male diagnosed with schizophrenia, showing first symptoms of disorganization and paranoid delusion at 17 with severe impact on his functioning. Before admission, he was treated with Amisulpride 400 mg, olanzapine 10 mg and paliperidone long acting injection 150 mg with little improvement in functioning, which lead to the diagnosis of difficult-to-treat schizophrenia. Physically he was diagnosed with Juvenile Polyposis Syndrome at 11, for which he received a total colectomy. His current admission started several years before the event for non-suicidal self-injurious behavior and catatonia.

2.2.2 Clinical findings and diagnostic assessment

On admission, Mr. B showed mannerisms, stereotypical behaviors, autonomic instability, perseverations and non-suicidal self-injurious behavior, particularly severe scratching, leading to diagnosis of schizophrenia with catatonia. A CT brain and extensive blood work showed no abnormalities, inferring no organic

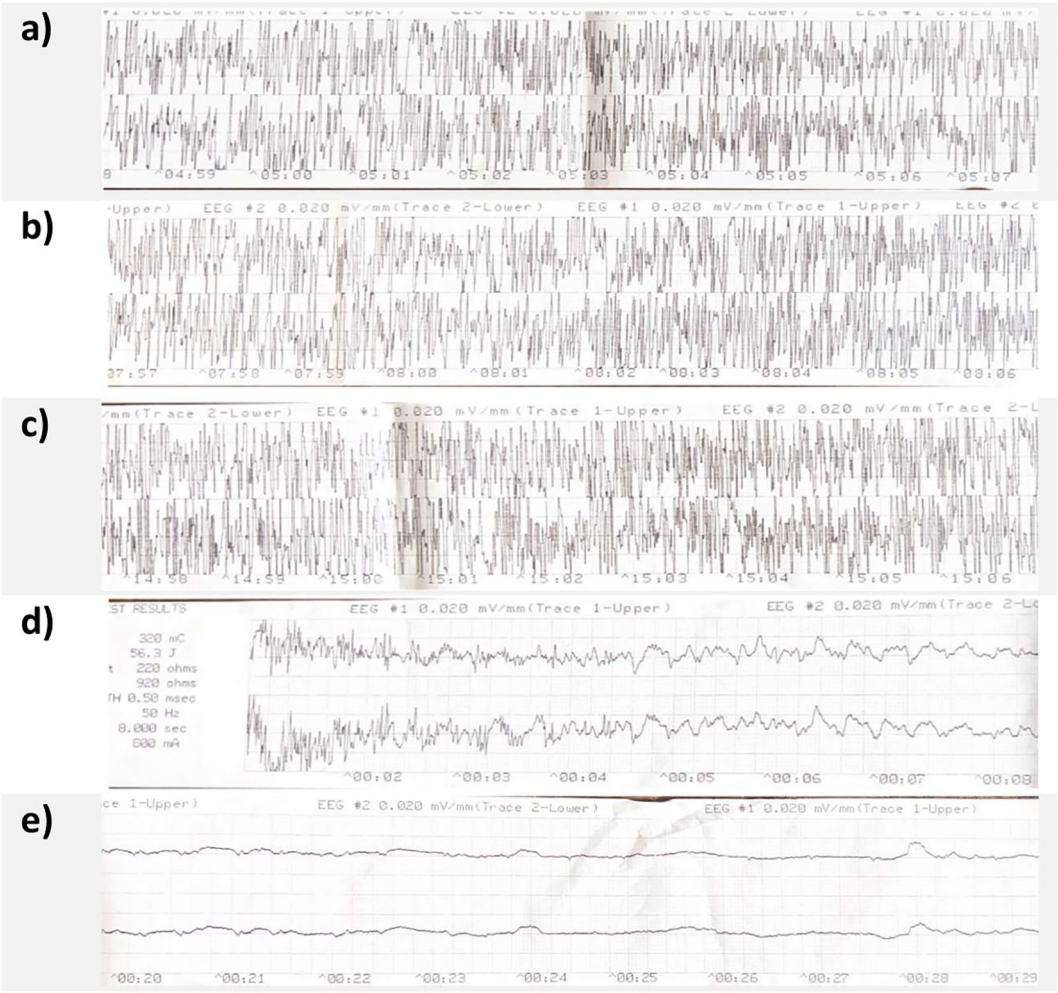

FIGURE 2
Case A's MECTA 2-channel EEG. Panel (**a–c**) show continued spike and wave activity at 5 min, 8 min and 15 min respectively. Panel **(d)** shows restimulation at 15 min with termination of clear spike and wave activity, panel **(e)** shows clear postictal suppression 20 seconds after restimulation.

etiologies of catatonia. Alongside clorazepate 15 mg 3x/day, clozapine 200 mg was initiated and initially provided partial improvement. However, residual stereotypical behaviors and scratching persisted. Clozapine was eventually discontinued because of recurrent gastrointestinal obstruction, which was considered a side effect aggravated by Juvenile Polyposis Syndrome and colectomy. After multidisciplinary discussion and pre-ECT evaluations, ECT was advised and informed consent by proxy was obtained from his family.

2.2.3 Therapeutic intervention

Bitemporal ECT for catatonia in schizophrenia was started with good effect on catatonic symptoms and non-suicidal self-injury. The reduction in symptoms led to an improvement of activities of daily living on the ward. Reduction to biweekly ECT led to an increase in catatonic symptoms, after which weekly ECT was continued. Aside from clorazepate 15 mg 3x/day, he was on aripiprazole 15 mg and clotiapine 20 mg 3x/day. Furthermore he received lorazepam 2.5 mg as needed. ECT was continued with important clinical improvement for 107 sessions. During his weekly maintenance ECT treatment, the patient had a prolonged seizure on session 100 necessitating propofol with successful termination of the seizure.

On the 108th ECT session, he received 16 mg etomidate and 50 mg succinylcholine for induction. Figure 1b shows a timeline of this ECT session. Therapeutic stimulus was given with the same parameters as previous stimulations (see Table 1), and motor convulsions terminated at 45 seconds. He showed epileptic activity on EEG for more than 2 min, after which the same protocol as above was followed (see also Table 2a). EEG showed continued epileptic activity after 8 min. The clinical team decided to administer 8 mg etomidate and 50 mg succinylcholine and stimulate the patient 10 min 43s after the first stimulation with the same dose as the therapeutic stimulus. A 25-second convulsion ensued, after which EEG monitoring showed immediate cessation of seizure activity and postictal suppression.

2.2.4 Follow-up and outcomes

The patient was closely monitored for 24 hours and received a neurological follow up consultation with 24-channel EEG which showed no epileptic activity. After careful consideration, imaging was not performed since there was no indication that the patient had any structural brain abnormality. Discharge of residential hospitalization was already being planned in the time leading up to this event, but was only possible due to continued improvement with weekly ECT. Therefore weekly ECT was resumed with propofol induction. After no prolonged seizures or other complications were reported in the next month, the patient was discharged from residential hospitalization, while continuing weekly maintenance ECT.

2.2.5 Patient perspective

Communicating with Mr. B remained challenging even after symptom improvement with ECT, making it difficult to fully understand his personal experience of the treatment. However, given the severity of his symptoms, it was evident that he endured significant suffering. As he had no recollection of the SE, he expressed no concern about its implications. His father was more worried about potential cognitive side effects of ECT than the prolonged seizures.

With continued maintenance ECT sessions, Mr. B showed noticeable improvement in paranoid delusions, stereotypical behaviors, excessive scratching, and disorganization, allowing for better engagement in activities of daily living. One month after the SE, he was discharged from the hospital after several years of inpatient care and transitioned to a psychiatric care home. However, he continues to experience disorganization and is still receiving maintenance ECT at the time of writing.

3 Discussion

3.1 Mechanism

The anticonvulsant effects of ECT have long been recognized, giving rise to the anticonvulsant hypothesis of its mechanism of action. This hypothesis states that increased inhibitory GABA-ergic neurotransmission is necessary for the therapeutic effect of ECT (8, 9). As genetic deficits of GABA-ergic metabolism lead to epileptic syndromes and many GABA-agonists are anticonvulsants, we know that GABA plays a central role in seizures. Furthermore, GABA might stimulate neuroplasticity (19). During the course of ECT, seizure duration decreases while seizure threshold increases (20), which could be due to increased levels of GABA, GABA-receptor activity and GABA-ergic interneurons (21–24) and may be linked to neuroplastic effects of ECT (20). Postictal suppression, seen at the end of an ECT-induced seizure on EEG, could be the expression of an increased postictal inhibitory process and appears to be a useful predictor of clinical outcome of depression (25, 26).

The anticonvulsant hypothesis has provided a theoretical basis for the use of ECT as a treatment for SE. A recent scoping review describes 28 patients with refractory or super-refractory SE that received ECT, all of which resulted in SE resolution, with clinical improvement reported in 20 patients (10). ECT is classified at a GRADE D/Oxford level 4 evidence for treatment of SE (10, 27) and is mentioned in 5 clinical practice guidelines as alternative therapy for specific cases of refractory and super refractory SE (28). The limited evidence supporting these clinical recommendations highlights the relevance of our report.

3.2 Current findings and clinical practice

The cases described in our study demonstrate that administering an additional ECT stimulus can effectively terminate SE when conventional treatments fail. This approach is theoretically grounded in the anticonvulsive hypothesis and in clinical evidence showing that ECT is an effective treatment for refractory SE. Although propofol induction prevented prolonged seizures in subsequent ECT sessions and is usually sufficient to terminate prolonged seizures, it was ineffective in these two cases. Similarly, the ensuing doses of lorazepam were insufficient. The temporal relationship between the stimulus and the swift cessation of the seizure reinforces this hypothesis. Additionally, this approach was validated in two patients. It should be noted that in case B, we used etomidate in preparation for the terminating stimulus instead of propofol. Although this strengthens the hypothesis that the seizure stopped because of the stimulus and not due to additional anesthesia, propofol bears preference due to its stronger anticonvulsive properties (29).

Our cases can be considered both a prolonged seizure and SE, explaining why terminology in literature of abnormal seizures after ECT is heterogenous. We consider all prolonged or tardive seizures with >5 min of generalized seizure activity, or all partial and absence seizures >10 min as status epilepticus, based on the ILAE classification (11). In a search of the literature we found 35 cases of status epilepticus after ECT meeting these criteria (14, 30–63) (see Supplementary Materials for search method). Only two of these describe restimulation to terminate SE. Hazimeh et al. (28) describe a convulsion starting 11 min after ECT which was initially managed with midazolam and propofol. When convulsions resumed and propofol had no effect, the convulsion ceased after the second stimulus. However, more convulsions followed after 5 min and SE was not resolved by this intervention. Goh et al. (37) describe a prolonged seizure of more than 12 min, constituting SE, which was terminated completely by a second stimulus. In these two cases described in the literature, the eliciting stimulus was the seizure threshold, and both terminating stimuli used were six times seizure threshold equivalent to a therapeutic stimulus. Conversely, in our cases the eliciting stimulus was therapeutic and both terminating stimuli were of the same dose, suggesting the dose of the terminating stimulus is not a critical factor in the mechanism of seizure termination. It has indeed been shown that seizure duration decreases between the first and second treatment, while the relationship between stimulus dosage and seizure duration is less

straightforward (64). It is also known that an effective ECT session results in an immediate and substantial surge of GABA (22). The finding that the second stimulus is more effective than the administered medication could be explained by the electrical stimulus provoking an excitatory wave immediately followed by a massive outpouring of GABA and other inhibitory neurotransmitters (19). However, if the eliciting stimulus dose is the seizure threshold, we would recommend restimulating with a therapeutic stimulus.

Prolonged seizure, SE, and tardive seizures after ECT share risk factors that lower seizure threshold, which should be considered and mitigated following an abnormal seizure after ECT:

- Medication: clozapine (59), lithium (65), bupropion, antibiotics, theophylline (15).

  Seizure risk may increase after recent tapering of antiepileptics or benzodiazepines (15).
- ECT delivery: first sessions (15), multiple monitored ECT (30).
- Anesthesia: etomidate (29), hyperventilation, anesthetic-ECT time interval (66).
- Patient specific factors: younger age (65), women (67), height (64), intracranial pathology like brain metastasis (42), although ECT can remain safe in these patients (68).

Based on these findings, we propose amendments to the protocol used to manage prolonged seizure after ECT (See Table 2b). We suggest considering restimulation after all other treatments have failed. All 4 available reports restimulated 10–15 min after the eliciting stimulus, and it should be considered before 30 min of SE as risk of neurological complications is significant by this time (11). Additionally, we suggest minimizing risk factors for prolonged seizures like intracranial pathology and medication before considering resumption of ECT. Finally, we propose to resume ECT using propofol induction as this raises the seizure threshold (29). Further research is necessary to bolster the evidence for these recommendations, although we report that both of our patients were able to continue ECT without further complications and with important clinical benefit.

3.3 Limitations

Aside from publication bias and possible overinterpretation which are limitations inherent to case series (69), a limitation of our report is that we do not have 24-channel EEG data of the events themselves as patients in our center are monitored through the MECTA 2-channel EEG during ECT. Difficulty in diagnosing SE, particularly non-convulsive SE, has been noted in previous reports, as EEG slowing after ECT is a physiological phenomenon (70–72). Additionally, while ECT has been safely used in SE, our report is not able to offer a comparative analysis of the tolerance of this intervention. However, we offer suggestions on how to manage follow-up.

3.4 Conclusion and implications

This report suggests that status epilepticus after ECT can be safely treated by restimulation, avoiding a longer seizure and potential severe neurological complications. Our report describes the theoretical foundations and acknowledges two previous reports with this finding, leading us to believe this strategy is warranted if this rare complication arises. After risk factors were determined, anesthesia was switched to propofol and both patients resumed ECT without complications. Further research would have to validate this strategy which might offer a safe and effective way to address SE when other treatments fail.

Data availability statement

The original contributions presented in the study are included in the article/Supplementary Material. Further inquiries can be directed to the corresponding author/s.

Ethics statement

The studies involving humans were approved by EC Research UZ/KU Leuven and EC UPC KU Leuven. The studies were conducted in accordance with the local legislation and institutional requirements. The participants provided their written informed consent to participate in this study. Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article.

Author contributions

MP: Conceptualization, Methodology, Project administration, Visualization, Writing – original draft, Writing – review & editing. KH: Supervision, Writing – review & editing. PS: Supervision, Writing – review & editing. EB: Writing – review & editing. FB: Conceptualization, Writing – original draft, Writing – review & editing.

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Conflict of interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

Generative AI statement

The author(s) declare that Generative AI was used in the creation of this manuscript. The authors used AI-assisted proofreading (ChatGPT, OpenAI) only for English language optimalisation of the manuscript. All content was reviewed and approved by the authors.

Publisher's note

Supplementary material

The Supplementary Material for this article can be found online at: https://www.frontiersin.org/articles/10.3389/fpsyt.2025.1576374/full#supplementary-material

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