Received: 15 March 2024 | Revised: 7 June 2024 | Accepted: 2 July 2024

DOI: 10.1002/pcn5.227

PCN Reports
Psychiatry and Clinical Neurosciences
Open Access

CASE REPORT

A case with temporal spikes on electroencephalography induced by over 80 sessions of electroconvulsive therapy

Hisaki Omori MD[1,2] | Masahiro Hata MD, PhD[1] | Matasaburo Kobayashi MD[1] | Yuki Miyazaki MD[1] | Atsuya Hirashima MD[1,2] | Shun Takahashi MD, PhD[1,3,4,5] | Manabu Ikeda MD, PhD[1]

[1]Department of Psychiatry, Osaka University Graduate School of Medicine, Osaka, Japan

[2]Department of Psychiatry, Esaka Hospital, Osaka, Japan

[3]Department of Occupational Therapy, Graduate School of Rehabilitation Science, Osaka Metropolitan University, Osaka, Japan

[4]Department of Clinical Research and Education Center, Asakayama General Hospital, Osaka, Japan

[5]Department of Neuropsychiatry, Wakayama Medical University, Wakayama, Japan

Correspondence
Masahiro Hata, MD, PhD, Department of Psychiatry, Osaka University Graduate School of Medicine, 2-2 Yamadaoka, Suita, Osaka 565-0871, Japan.
Email: mhata@psy.med.osaka-u.ac.jp

Funding information
None

Abstract

Background: Electroconvulsive therapy (ECT) is widely recognized as one of the most effective treatments for various psychiatric disorders and is generally considered safe. However, a few reports have mentioned that multiple ECT sessions could induce electroencephalography (EEG) abnormalities and epileptic seizures, a serious side effect of ECT. We experienced a case with EEG abnormalities after multiple ECT sessions and aimed to share our insights on conducting ECT safely.

Case Presentation: We present the case of a 73-year-old female diagnosed with major depressive disorder. She underwent regular ECT sessions to alleviate her psychiatric symptoms. However, after more than 80 sessions, previously undetected EEG abnormalities were observed. Since the patient did not have clinical seizures, we were able to continue ECT at longer intervals without the use of antiepileptic drugs.

Conclusion: Our case suggests the importance of routine EEG testing in patients undergoing prolonged ECT. While careful monitoring is necessary, continuing ECT without antiepileptic medication in patients with EEG abnormalities could be permissible.

KEYWORDS
electroconvulsive therapy, electroencephalography, epileptic seizures, ECT

BACKGROUND

Electroconvulsive therapy (ECT) is considered one of the most effective treatments for various psychiatric disorders, including treatment-resistant depression, bipolar disorder, mania, and catatonia.[1] Although concerns about side effects such as cognitive impairment and cardiovascular problems have been raised, ECT is generally considered to be safe.[2] While its therapeutic mechanisms are still under discussion, one of them is the anticonvulsant hypothesis.[3] This hypothesis derived from the observation that there is an increase in seizure threshold and a decrease in seizure duration in patients who have undergone multiple sessions of ECT. Contrary to this, a few cases that developed temporal lobe epilepsy during ECT have been reported. Some reports have demonstrated that the prevalence of spontaneous seizures after ECT ranged from approximately 1% to 2%, although a large 5-year-long study of psychiatric patients who received ECT showed no such cases.[4] In this article, we report a case with electroencephalography (EEG) abnormalities while undergoing over 80 sessions of ECT, contributing to the consideration of measures to perform ECT safely.

CASE PRESENTATION

This 73-year-old female was diagnosed with major depressive disorder in her 30s, and she had received outpatient treatment for over 30 years. However, at the age of 66, she had suicidal thoughts and refused to eat anything. Simultaneously, she had delusional thoughts such as "I'm already dead" and "My whole body is gone," which were considered Cotard's syndrome.[5] Since pharmacotherapy did not lead to an improvement in both mood and psychotic symptoms, she was referred to our hospital for intensive treatment, including ECT.

She received 10 sessions of ECT and her psychiatric symptoms improved remarkably. However, her symptoms deteriorated rapidly in 2 months. Thus, she had 10 more ECT sessions and her symptoms improved markedly again. After that she had 17 maintenance ECT sessions every 1–2 months for about a year and half to prevent her symptoms worsening. While performing maintenance ECT, we switched primary antipsychotic medication for the patient from perospirone to olanzapine. When she was 68 years old, her symptoms stabilized and she temporarily ended maintenance ECT, but her depressive symptoms worsened and she required hospitalization in 3 years later. She needed to have more 10 sessions of ECT and continued regular maintenance ECT for 11 sessions. When she was 72 years old, she underwent two sets of 10 ECT sessions and three maintenance ECT sessions.

The patient occasionally underwent routine EEG testing, but no abnormalities were observed. However, when she was 72 years old, her EEG showed sporadic spike-wave discharges on frontotemporal electrodes (F7·F8) (Figures 1 and 2). At that time, she had received 81 sessions of ECT. The maximum frequency was twice a week. She had no personal or family history of epilepsy, and both head computed tomography and blood tests showed no abnormalities. Her medication had not been changed since before the abnormal EEG activities appeared. She was taking asenapine, brexpiprazole, lemborexant, ramelteon, and vortioxetine.

Considering the course of the treatment so far, we concluded that maintaining a remission state through pharmacotherapy was challenging, therefore we decided to continue ECT at longer intervals with careful EEG monitoring and not to use antiepileptic drugs because it was confirmed that she did not have any clinical seizures after a detailed interview by a certified epilepsy specialist (M.H.). She subsequently underwent eight ECT sessions, but no clinical seizures were observed and spike-wave discharges in the EEG decreased.

DISCUSSION AND CONCLUSION

We report a case with temporal spike-wave EEG discharges after more than 80 sessions of ECT.

We found only 10 case reports about patients who were suspected of epileptic seizures during the period of undergoing ECT treatment and had confirmed spike waves in the temporal region in EEGs.[6–8] In these cases, a minimum of 36 and a maximum of over 1100 sessions had been administered.[7]

One possible underlying mechanism of epileptic seizures or abnormalities in EEGs after multiple sessions of ECT is electrical kindling. The kindling effect was observed in animal experiments by Goddard and colleagues in 1969.[9] They demonstrated that by

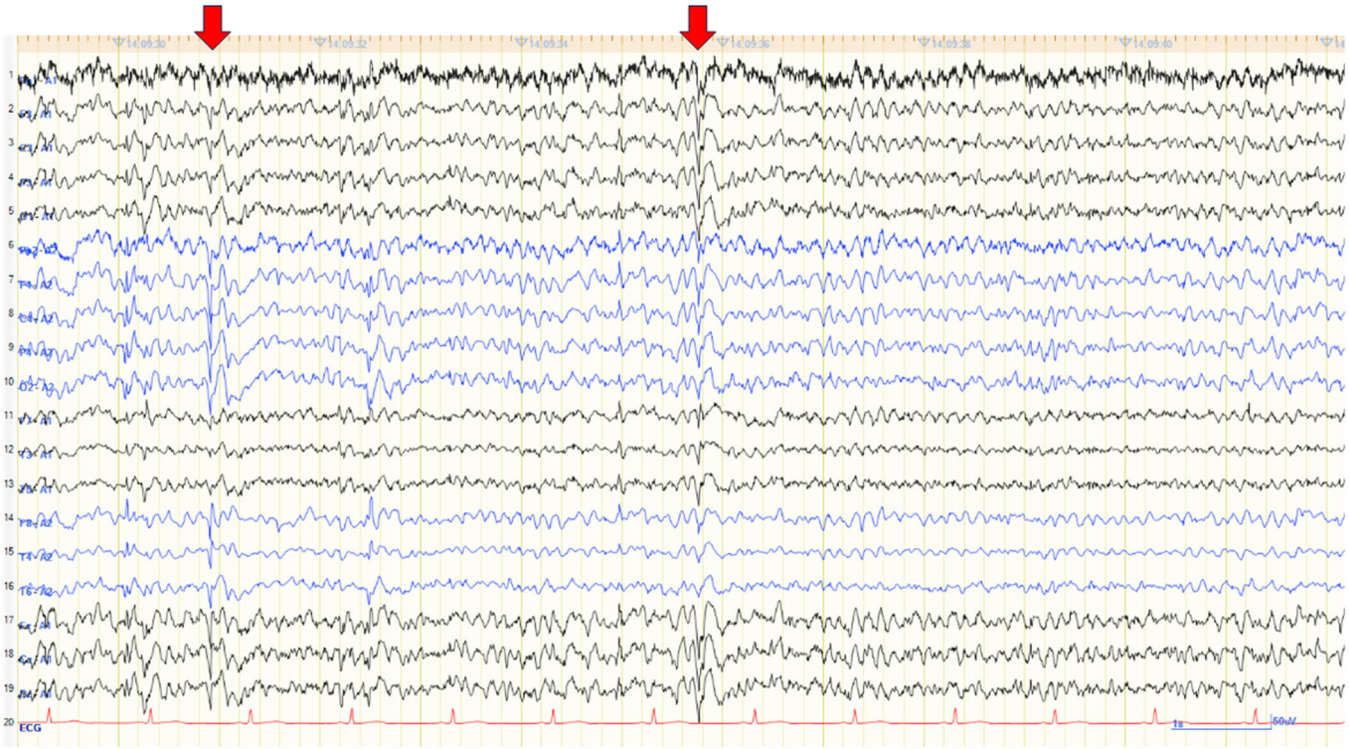

FIGURE 1 Routine electroencephalography after 81 sessions of electroconvulsive therapy, monopolar derivation. Generalized positive spikes are observed in the monopolar derivation. The red arrow indicates the location of the spike.

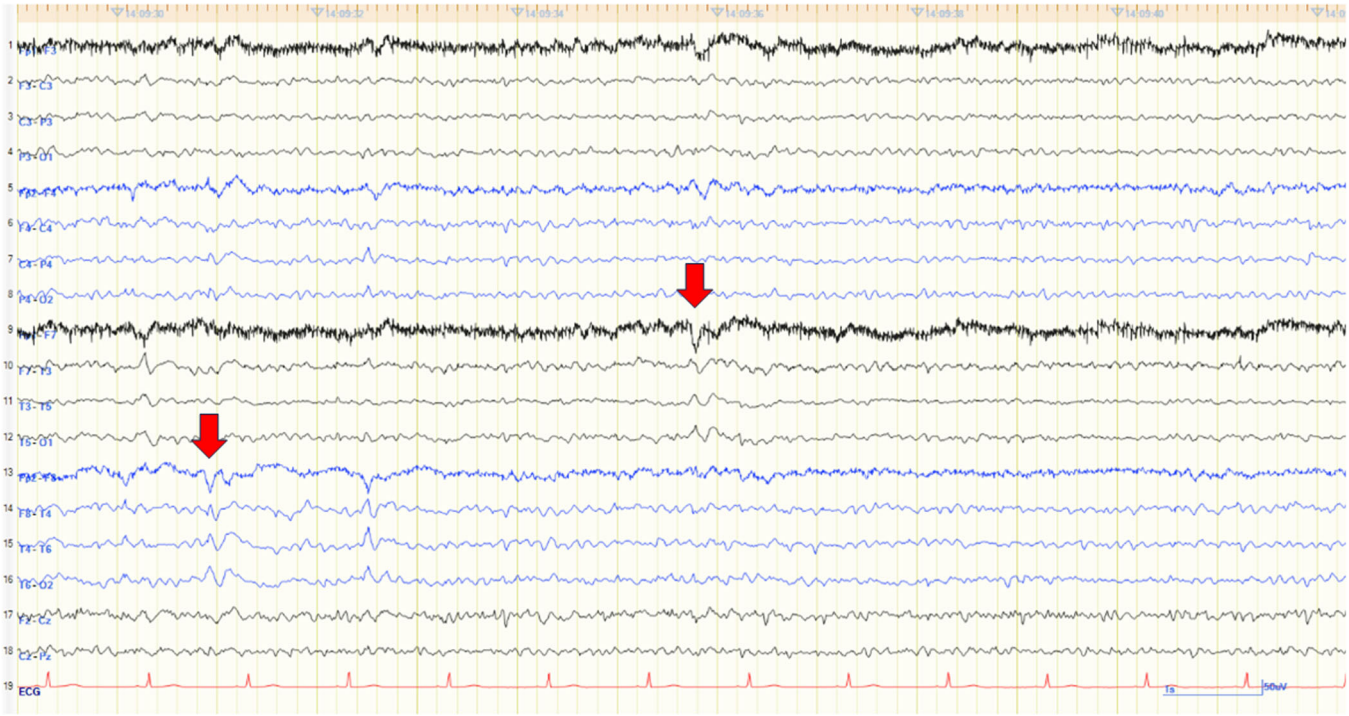

FIGURE 2 Routine electroencephalography after 81 sessions of electroconvulsive therapy, bipolar derivation. Phase reversals over the right or left frontotemporal region (F7·F8) are observed in the bipolar derivation. The red arrow indicates the location of the spike.

TABLE 1 Cases with epileptic seizures induced by ECT.

Author	Number of cases	Number of ECT sessions until EEG abnormalities appeared	Clinical symptoms	ECT	Antiepileptic drugs
Rasmussen and Lunde (2007)[8]	4	Case 1: 124 times	Brief spells of unresponsiveness	Discontinue	Use
		Case 2: 39 times	Staring spells	Discontinue	Use
		Case 3: 69 times	Spells of shaking	Continue	Use
		Case 4: 86 times	Spells of unresponsiveness	Continue	Use
Bryson et al. (2016)[6]	5	Case 1: 106 times	Intermittent blank spells	Discontinue	Use
		Case 2: 331 times	Deterioration in cognition	Discontinue	Disuse
		Case 3: 36 times	Motor and speech arrest	Discontinue	Use
		Case 4: 52 times	Confusion and psychomotor slowing	Discontinue	Disuse
		Case 5: 348 times	Deterioration in cognition	Discontinue	Use
Schotte et al. (2019)[7]	1	Over 1100 times	Focal to bilateral tonic-clonic seizure	Continue	Use
Our case	1	81 times	None	Continue	Disuse

Abbreviations: ECT, electroconvulsive therapy; EEG, electroencephalography.

repeating electrical stimulations, the response to these stimulations progressed from little effect at first to localized discharges in EEGs, eventually evolving into convulsions. The effect was obtained by stimulating regions associated with the limbic system, which was not achieved by stimulations of other areas.

There are some similarities in the processes through which electrical kindling and ECT induce seizures. First, anatomical changes correlate with the number of treatment sessions and particularly in large-scale data, consistent increases in the volume of the medial temporal lobe structures such as the hippocampus and amygdala have been demonstrated.[1] This correlates with the fact that the kindling phenomenon needs stimulation of the limbic system. Second, all reported cases so far exhibited abnormalities not in the early stages of treatment but after multiple sessions. This is analogous to the kindling phenomenon, which requires repetitive stimulations.

While this mechanism has been discussed thus far, the interpretation of this phenomenon requires careful consideration. Due to the limited number of reported cases, it remains uncertain whether there is a causal relationship between ECT and epileptic seizures. In addition, the kindling phenomenon was observed only in animal experiments. Furthermore, in ECT sessions, strong electrical stimulations are applied from the beginning to induce generalized tonic-clonic seizures in EEGs, although the kindling phenomenon adopts weaker ones.

Considering our case, the number of ECT sessions and the EEG abnormality detected are consistent with previous reported cases. In past cases, patients were suspected of having epileptic seizures and then underwent further examination, revealing abnormalities in EEGs. In past reports of 10 cases,[6–8] ECT was discontinued in seven cases and continued in three cases with the use of antiepileptic drugs which could lower the susceptibility to seizures during ECT sessions (Table 1). On the other hand, although EEG abnormalities were induced in our patient, she did not exhibit clinical epileptic seizures, therefore antiepileptic drugs were not administered. Considering the difficulty in achieving therapeutic efficacy with treatments other than ECT and the severity of psychiatric symptoms, we decided to continue ECT sessions with careful EEG monitoring. This approach represents a different course from that in previous reports in that we continued ECT without using antiepileptic drugs. Although several ECT sessions with longer intervals were performed on this patient after EEG abnormalities were detected, the patient did not have clinical epileptic seizures. The absence of clinical consensus on how to modify the treatment plan when EEG abnormalities are detected during the ECT course presents an unresolved challenge that demands future consideration.

In conclusion, our case suggests the importance of regular EEG assessments for patients undergoing prolonged ECT because if EEG monitoring had not been performed, temporal lobe epilepsy seizures might have occurred, potentially leading to accidents or other disadvantages for the patient. While careful monitoring is necessary, continuing ECT without antiepileptic medication in patients with EEG abnormalities could be permitted.

AUTHOR CONTRIBUTIONS

Hisaki Omori and Masahiro Hata designed the work. Hisaki Omori, Masahiro Hata, and Matasaburo Kobayashi described the case. Hisaki Omori and Masahiro Hata interpreted EEG data. Hisaki Omori wrote the first draft, and Masahiro Hata, Shun Takahashi, and Manabu Ikeda assisted in writing of the manuscript. Yuki Miyazaki and Atsuya Hirashima provided feedback on the manuscript. All authors contributed to and approved the final version of the manuscript.

ACKNOWLEDGMENTS

N/A.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

All data generated or analysed during this study are included in this article. Further enquiries can be directed to the corresponding author.

ETHICS APPROVAL STATEMENT

This study was approved by the Ethical Committee of Osaka University Hospital.

PATIENT CONSENT STATEMENT

Written informed consent for the publication of this report was obtained from the patient and patient's family.

CLINICAL TRIAL REGISTRATION

N/A.

ORCID

Masahiro Hata http://orcid.org/0000-0003-3837-1504
Yuki Miyazaki http://orcid.org/0000-0003-4952-0886
Shun Takahashi http://orcid.org/0000-0002-9049-4694

REFERENCES

1. Kritzer MD, Peterchev AV, Camprodon JA. Electroconvulsive therapy: mechanisms of action, clinical considerations, and future directions. Harvard Rev Psychiatry. 2023;31(3):101–13.
2. van der Wurff FB, Stek ML, Hoogendijk WJG, Beekman ATF. The efficacy and safety of ECT in depressed older adults: a literature review. Int J Geriatr Psychiatry. 2003;18(10):894–904.
3. Abbott CC, Gallegos P, Rediske N, Lemke NT, Quinn DK. A review of longitudinal electroconvulsive therapy: neuroimaging investigations. J Geriatr Psychiatry Neurol. 2014;27(1):33–46.
4. Shin HR, Kim M, Park KI. Electroconvulsive therapy and seizure: a double-edged sword? Encephalitis. 2023;3(4):103–8.
5. Berrios GE, Luque R. Cotard's delusion or syndrome?: a conceptual history. Compr Psychiatry. 1995;36(3):218–23.
6. Bryson A, Gardner H, Wilson I, Rolfe T, Archer J. Temporal lobe epilepsy following maintenance electroconvulsive therapy—electrical kindling in the human brain? Epilepsia. 2016;57(11):e216–20.
7. Schotte C, Cleeren E, Goffin K, Hunyadi B, Buggenhout S, Van Laere K, et al. Development of temporal lobe epilepsy during maintenance electroconvulsive therapy: a case of human kindling? Epilepsia Open. 2019;4(1):200–5.
8. Rasmussen KG, Lunde ME. Patients who develop epilepsy during extended treatment with electroconvulsive therapy. Seizure. 2007;16(3):266–70.
9. Goddard GV, McIntyre DC, Leech CK. A permanent change in brain function resulting from daily electrical stimulation. Exp Neurol. 1969;25(3):295–330.

How to cite this article: Omori H, Hata M, Kobayashi M, Miyazaki Y, Hirashima A, Takahashi S, et al. A case with temporal spikes on electroencephalography induced by over 80 sessions of electroconvulsive therapy. Psychiatry Clin Neurosci Rep. 2024;3:e227. https://doi.org/10.1002/pcn5.227