

Case Report

Convulsive Status Epilepticus After Electroconvulsive Therapy

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Abstract

Status epilepticus is a very rare complication of electroconvulsive therapy. A case of a 58-year old patient with catatonic schizophrenia is reported who developed a status epilepticus after ECT. At a neurological critical care unit the status finally could be stopped. The origin of the epileptic status lastly remained unclear (German J Psychiatry 2013; 16(2): 81-83).

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A 58 year old woman suffering from catatonic schizophrenia (ICD-10 F20.2) was admitted to an inpatient psychiatric care with catatonic symptoms like mutism, echopraxia and catalepsy. At the first admission in 1977, the patient showed a paranoid syndrome with the strong conviction of being a follower of a maharaja. In addition, she reported to be able to make contact with him by singing. She meditated to acquire illumination. Her line of thought was disjointed and she showed religious mania. A diagnosis of paranoid schizophrenia was made and the patient was treated with haloperidol and diazepam. Within 1979 and 1989 five rehospitalizations followed with clinical symptoms of religious mania, psychomotor inhibition and excitation in turns. Mainly she was medicated with haloperidol, benperidol, chlorprothixene and flupentixol. Since then the patient lived with psychiatric outpatient care until she was rehospitalized in 2011 with a catatonic syndrome described above.

Pharmacological treatment (e.g. with risperidone, olanzapine and lorazepam) did not succeed. Therefore, unilateral electroconvulsive therapy (ECT) was started.

Before that, an EEG was conducted with normal results and without typical epileptic patterns. Furthermore, the patient did not have a history of seizures. An MRT scan of the brain showed normal findings without an epileptic focus or signs of encephalitis. The neurological examination also was unremarkable apart from the reported abnormalities of psychomotor activity as well as the laboratory results.

ECT was conducted with anesthesia consisting of etomidate, succinyl chloride and an opioid analgetic.

After the first session, the catatonic symptoms improved. ECT was conducted with etomidate (with a dose ranging from 18 to 20 mg), alfentanil (with a dose ranging from 0.5 to 2 mg) and succinyl chloride (with a dose ranging from 30 to 60 mg). Twice the therapeutic seizure had to be stopped by injecting midazolam because of its extended duration (Table 1).

Table 1: Overview ECT time schedule

Number of ECT	Max. Device output (%) applied	Impedance	Postictal suppression index	Time	Dose of etomidate (mg)	Dose of alfentanil (mg)	Dose of succinyl chloride (mg)	Dose of midazolam (mg)	Oxygen saturation
1	55	1900	29,40%	170	20	2	40	2	97
2	50	1810	13,40%	69	18	0,75	60	0	98
3	50	1610	49,80%	94	20	0,5	40	0	96
4	50	1840	32,60%	170	20	1	30	2	96
5	50	1280	–	20	18	0,5	30	0	100
6	50	1560	22,40%	55	18	0,5	30	0	98
7	50	1190	41,70%	13	–	–	–	–	–

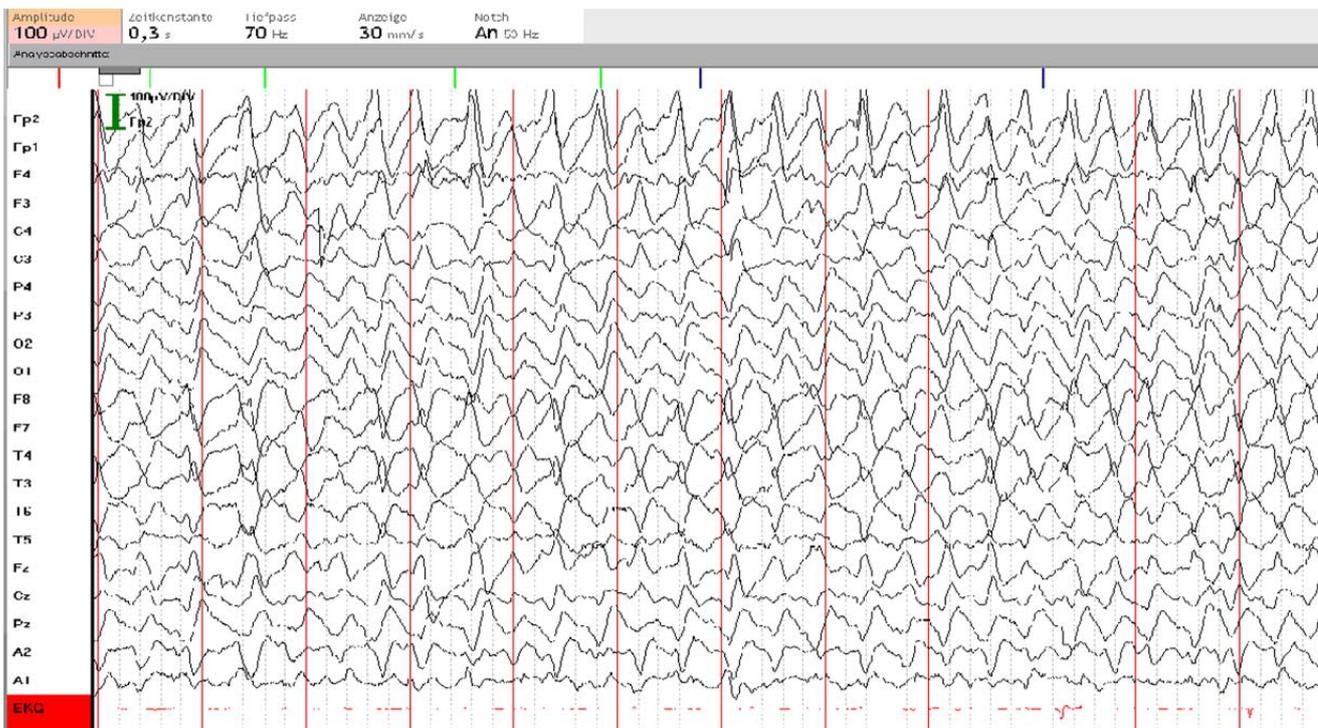
However, the patient suffered from short epileptic seizures with secondary generalization a few hours after the second and third session of ECT. Because of a considerable amelioration of psychopathological symptoms during the application of ECT, it was continued in spite of these adverse events. Similar side effects have been described in previous case reports (Rasmussen & Lunde, 2007). However, two hours after the eighth session of ECT, an epileptic status with coma, eye deviation to the right, and convulsions in the right face emerged (Figure 1).

The status could not be terminated with benzodiazepines and rapid intravenous valproate titration. The epileptic status could be stopped only after anesthetizing the patient with propofol at a neurological critical care unit. After termination of the anaesthetic 24 hours later the status re-occurred. Five days later, the EEG still showed burst-suppression

and deep midazolam sedation, which gradually could be tapered off. After discharge from the neurological hospital, an alpha EEG without any pathological findings was seen. Unfortunately, complications occurred including pneumonia, cystitis and pulmonary embolism. After several months recovery occurred with a pharmacological treatment including lorazepam, benperidol, phenytoin, levomepromazine, melperone and valproate. Finally, the patient could be discharged from hospital without catatonic symptoms. She was able to perform activities of daily life.

The origin of the epileptic status remained unclear. On the one hand, epileptic seizures during a catatonic stupor have been described without concurrent specific neurological disorder (Suzuki et al., 2006). On the other hand, a paraneoplastic genesis of epileptic seizures has been discussed in terms of limbic encephalitis. Positive neuropil antibodies

Figure 1. Epileptic status EEG



activity. Finally, the status could be disrupted with phenytoin

which indicate neurological paraneoplastic disease patterns

(e.g. Anti-Hu, Anti-Ri, Anti-Jo, Anti-Tr VGKCC-AK) supported this assumption (Kleyensteuber et al., 2010, Lee et al., 2006, Samarasekera et al., 2007). The pulmonary embolism could also be attributed to a paraneoplastic process (Cafagna & Ponte, 1997, Zerbino et al., 1994). Because of suspect lymph nodes and swollen cervix uteri in computer tomogram a cervical carcinoma was assumed. However, this was excluded by a gynecological consultation.

Prolonged seizures occur in up to 19% of the cases (Benbow et al., 2003). Typically, electrically induced seizures do not last much longer than 60 seconds (Weiner, 1980). Prolonged seizures are defined as seizure activity over 180 seconds (APA, 2001). To stop prolonged seizures, it is recommended to administer 1–2 mg of midazolam. After 2 minutes of EEG recording and persistence of seizure activity, midazolam should be administered again in the same dosage. In case of further persistence, fosphenytoin 20 mg/kg or propofol can be administered (Krystal, 2010). Indeed, on an overall basis, status epilepticus after ECT seems to be a very rare complication. Convulsive status epilepticus is even more exceptional, reported in the last 15 years only by Dersch et al. (2011).

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