

Successful Electroconvulsive Therapy in a Clozapine-Refractory Schizophrenia Patient with Meningioma

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Introduction

Electroconvulsive therapy (ECT) is a highly effective treatment in mood disorders and also somewhat effective in schizophrenia patients (1-5). In schizophrenia, patients with catatonic and affective symptoms seem to benefit most (6). Several case reports have shown that the safety of patients with cerebral tumors is not compromised by this intervention (7-10) as long as intracranial pressure is not increased (11). However, it is important that contraindications for ECT are reviewed carefully in order to make it available for patients who require it.

Case Report

We report here on a 58-year-old female schizophrenia patient (Mrs. A) with right parietal meningioma who underwent a series of ECT without complications. She had been known in our clinic since 1991. At that time, she presented to our outpatient clinic with symptoms of depressed mood, agitation, and insomnia. Within two years, she developed the complete clinical picture of schizophrenia with persecutory delusions, auditory hallucinations, and conceptual disorganization. Prescription of fluphenazine depot (25 mg every three weeks) worked for several years, but was unsuccessful

after a relapse due to noncompliance and was followed by olanzapine (10 mg/d), which led to symptomatic remission. After two years of treatment with olanzapine, the patient relapsed again in 2001 due to noncompliance. Re-introduction of olanzapine up to 20 mg/d was unsuccessful and medication was subsequently switched to clozapine. Ultimately, she was maintained on a dosage of 200 to 450 mg clozapine per day and remained clinically stable over the following years. Prior to the most recent admission to our ward, the patient had been prescribed clozapine (400 mg/d) and citalopram (40 mg/d) as well as lorazepam (6.25 mg/d) for the treatment of comorbid generalized anxiety disorder.

Several case reports have shown that the safety of patients with cerebral tumors is not compromised by this [ECT] intervention as long as intracranial pressure is not increased.

In October 2011, she was admitted to our ward due to reoccurrence of auditory hallucinations, persecutory delusions and anxiety. The patient refused to increase the dose of clozapine because of sedation. Initially, she participated in a randomized, double-blind, placebo-controlled Phase 2 trial. In addition to 400 mg of clozapine she had received the study medication for thirteen days. During this trial, the patient developed pronounced psychomotor agitation and suicidal ideation and an admission to a locked unit became necessary. Due to legal restrictions she had to be excluded from the clinical trial. Next to reinstating previous treatment

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with clozapine and citalopram, the dosage of lorazepam was increased from 2 mg/d to 4 mg/d. Mrs. A could be transferred back to an open ward after two weeks. As symptoms persisted we attempted to augment the ongoing medication with paliperidone palmitate (75 mg/month). In spite of adequate plasma levels, the patient's condition did not improve and only an increased benzodiazepine dose resulted in a slight alleviation of symptoms.

A Cochrane review about ECT for schizophrenia concludes that ECT in combination with antipsychotic drugs may be considered an option for treatment-resistant schizophrenia.

In light of the severe psychiatric illness, we decided to stop treatment with paliperidone palmitate after one month and to administer ECT. With the patient's consent we subsequently began with the preparations for a series of ECT. These preparations included cerebral magnetic resonance imaging (MRI), electroencephalography (EEG), standard laboratory tests, and anesthesiological consultation. There was no contraindication for ECT in the patient's physical state, with the exception of an asymptomatic right parietal meningioma, 8 x 2 mm in diameter. Neurosurgical and ophthalmological consultations resulted in the decision to perform ECT treatment. Because of its potential to lower the seizure threshold, clozapine was reduced to 200 mg/d, treatment with citalopram 40 mg/d was continued, lorazepam was discontinued. Following standard oxygenation, thiopental was used for anesthesia and succinylcholine was used for muscle relaxation.

Altogether, five ECT treatments were administered (2/wk) with a right unilateral electrode positioning. Doses ranged from 252 mC to 302 mC. Pulse-width was 0.5 ms, and we used a frequency between 50 and 60 Hz. Electrocardiogram, heart rate, blood pressure and blood oxygenation were continuously monitored and remained within the ranges to be expected during seizure. The patient developed no side effects such as headache or other neurological symptoms. Neurological examinations, which had been performed at regular intervals on the ward, showed no abnormalities; EEG was unremarkable. Following two treatments, we observed a marked improvement of positive symptoms and affective modulation. After completing the ECT series, Mrs. A was symptomatically remitted and could be discharged. Subsequently, we conducted three maintenance ECTs once a

month while continuing psychopharmacological treatment with clozapine 200 mg/d and citalopram 40 mg/d. Eighteen months after discharge Mrs. A is still in remission.

A Cochrane review about ECT for schizophrenia concludes that ECT in combination with antipsychotic drugs may be considered an option for treatment-resistant schizophrenia (2, 12). The present case report shows that even clozapine-refractory patients may remit following this intervention. In addition, this case report is in line with prior reports, which suggest that ECT in patients with asymptomatic meningioma can be safely administered and that a beneficial effect can be expected despite the intracranial tumor. However, it has to be emphasized that the topic clearly warrants further study in terms of larger trials and more valid study designs.

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