Successful ECT treatment of a treatment resistant manic bipolar patient with intracranial mass and pulmonary embolism history: Case report

İntrakranial kitlesi ve pulmoner emboli öyküsü olan tedaviye dirençli manik bipolar hastada başarılı EKT uygulanması: Olgu sunumu

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SUMMARY

Electroconvulsive therapy (ECT) is an effective and safe treatment method used in many psychiatric disorders. The efficacy and safety of ECT in the presence of an intracranial mass is controversial. The presence of a space-occupying mass was a contraindication to ECT until the 1980s. With the changes in ECT protocols over time and its application to more patients, positive data on the safety of ECT in intracranial masses have begun to accumulate. Available data suggest that ECT can be safely used in patients with benign, small, and otherwise clinically insignificant tumors. A history of pulmonary embolism (PE) accompanying a psychiatric disorder may cause clinicians to avoid ECT. There are some concerns regarding the use of ECT in patients with a history of PE as there is little evidence for the safety of the implementation of ECT. Although there is limited evidence regarding the safety of ECT in the presence of comorbidities, ECT can be successfully implemented where there is no response to other treatments with the necessary consultations and close follow-up, without delaying the treatment. In this article, a case who had been followed up with a bipolar affective disorder diagnosis, was hospitalized for manic episode, who had comorbidities such as intracranial tumor and PE history, who did not benefit from psychotropic treatments and who responded significantly to nine sessions of ECT without any complications is presented. The case presented here is, as far as is known, the first report where comorbidities such as intracranial tumor and pulmonary embolism history, which may cause clinicians to hesitate to implement ECT, co-exist.

Keywords: electroconvulsive therapy, ECT, bipolar, intracranial mass, tumor, pulmonary embolism

ÖZET

Elektrokonvülsif terapi (EKT), birçok psikiyatrik hastalıkta kullanılan etkin ve güvenli bir tedavi yöntemidir. İntrakranial kitle varlığında EKT'nin etkinliği ve güvenilirliği tartışmalıdır. 1980'li yıllara kadar yer kaplayan kitle varlığı, EKT için kontrendikasyon olarak kabul edilmekteydi. Zaman içinde EKT protokollerinin değişmesi ve daha çok hastaya uygulanmasıyla birlikte intrakranial kitlesi olan hastalarda EKT'nin güvenilirliği ile ilgili olumlu veriler birikmeye başlamıştır. Eldeki veriler EKT'nin benign, boyut olarak küçük ve klinik olarak önemsiz tümörleri olan hastalarda güvenle uygulanabileceğini göstermektedir. Psikiyatrik bir bozukluğa eşlik eden pulmoner emboli (PE) öyküsü klinisyenlerin EKT uygulamasından kaçınmasına sebep olabilir. PE öyküsü olan hastalarda EKT uygulamasının güvenliğine ilişkin az sayıda kanıt olması nedeniyle hastalarda EKT uygulanmasıyla ilgili bazı endişeler vardır. Komorbid durumlar birlikteliğinde EKT'nin güvenliğiyle ilgili sınırlı sayıda kanıt olmasına rağmen, diğer tedavilerden yanıt alınamadığında, gerekli konsültasyonlar ve yakın takip eşliğinde tedavide gecikmeye izin vermeden EKT başarıyla uygulanabilmektedir. Bu yazıda, bipolar affektif bozukluk tanısı ile takip edilen, manik atak nedeniyle yatarak tedavi gören, intrakranial tümör, geçirilmiş PE gibi komorbiditeleri olan, uygulanan psikotrop tedavilerden fayda görmeyen ve 9 seans EKT uygulamasından komplikasyon gelişmeden belirgin yanıt alınan bir vaka sunulmaktadır. Burada sunulan vaka bilindiği kadarıyla, klinisyenlerin EKT uygulamasında tereddüt edebildiği intrakranial tümör ve pulmoner emboli öyküsü gibi komorbiditeleri bir arada bulunduran ilk bildirimdir.

Anahtar Kelimeler: elektrokonvülsif terapi, EKT, bipolar, intrakranial kitle, tümör, pulmoner emboli

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INTRODUCTION

Electroconvulsive therapy (ECT) is an effective and safe treatment method used in many psychiatric disorders. The most common adverse effects of ECT includes headache and memory loss, which improves quickly after treatment.

The efficacy and safety of ECT in the presence of an intracranial mass is controversial. The presence of a space-occupying mass was a contraindication to ECT until the 1980s. Maltbie et al. reported that more adverse effects were observed during or after ECT in patients with invasive and aggressive intracranial tumors. In the same study, morbidity with predominant neurological symptoms were reported in 74% of 35 patients with intracranial tumors who received ECT, and one-month mortality rates were reported in 28% of all patients; however, the presence of a brain tumor was detected after ECT in 34 of these cases (1). It has been reported that during seizure activity in ECT, cortical blood flow increases by approximately 300% and cerebral metabolic rate for glucose and oxygen increases by approximately 200% (2). In addition, magnetic resonance imaging (MRI) studies have shown that ECT causes temporary functional breakdown in the blood-brain barrier through increased vascular permeability (3). As a result, it is considered that ECT may cause increased edema around the mass and neurological symptoms related to increased intracranial pressure (4).

With the changes in ECT protocols over time and its application to more patients, positive data on the safety of ECT in intracranial masses have begun to accumulate. The American Psychiatric Association report (1990) concluded that although space-occupying intracranial lesions pose a risk, they are not an absolute contraindication for ECT (5). Available data suggest that ECT can be safely used in patients with benign, small, and otherwise clinically insignificant tumors. Buday et al. reviewed 33 publications involving 75 patients who underwent ECT in the presence of intracranial tumors and observed that no serious adverse effects developed except for complications such as postictal confusion in four patients, short-term delirium in one, and secondary myoclonic seizures with Todd's paralysis in one, all of which were reversible (6).

A history of pulmonary embolism (PE) accompanying a psychiatric disorder may cause clinicians to avoid ECT. Cases of PE developing shortly after ECT have been implemented have also been reported, and the relationship between ECT and PE has not been clearly demonstrated (7). There are some concerns regarding the use of ECT in patients with a history of PE as there is little evidence for the safety of the implementation of ECT. ECT causes hypertension, tachycardia and low ejection fraction by causing a sympathetic surge; therefore, it may lead to possible complications in patients with PE who have impaired cardiopulmonary function (8). It has been reported that ECT is safe for patients with a recent history of PE under anticoagulant therapy (9). A case in which ECT was implemented after PE and thus the placement of the inferior vena cava filter following consultation with other departments, with no complications developing was reported (10).

In this article, a case who had been followed up with a bipolar affective disorder diagnosis, was hospitalized for manic episode, who had comorbidities such as intracranial tumor and PE history, who did not benefit from psychotropic treatments and who responded significantly to nine sessions of ECT is presented.

CASE HISTORY

A 36-year-old female patient, who was in follow-up with the diagnosis of bipolar affective disorder (BD) type 1 for 11 years and has been hospitalized many times, admitted to our emergency department with symptoms of manic episode. The patient, who has used antipsychotics, mood stabilizers and their combinations in the past at sufficient doses and for sufficient periods of time, had been receiving treatment with valproate 1500 mg/day, lithium 900 mg/day, risperidone 2 mg/day, biperiden 4 mg/day, zuclopenthixol depot 200 mg/2 weeks for the last one year and was in remission. In addition to BD, the patient had a history of epilepsy characterized by absence seizures, hypothyroidism, obesity, chronic obstructive pulmonary disease (COPD), asthma, low-grade glioma in the right frontal lobe with a size of 3*2 cm, pulmonary embolism history (history of pulmonary embolism in 2015, follow-up and treatment with a preliminary diagnosis of chronic pulmonary embolism until 2017, regression in embolic findings in imaging after 2017), diabetes mellitus type 1, obstructive sleep apnea syndrome (OSAS) diagnoses but was not using any of the

treatments determined by the relevant departments dealing with these diseases.

The patient, who was evaluated by a psychiatrist in the emergency department, and whose blood tests and cranial computed tomography (CT) did not reveal any pathology except low-grade glioma, was admitted to psychiatry clinic. The result of the Young Mania Rating Scale (YMRS), which was applied to the patient on the day of hospitalization, was 40. The patient's valproate level was 2 mg/L, and lithium level was 0.1 mmol/L; TSH level of 8.51 mU/L and free T4 level of 1.02 ng/dl supported subclinical hypothyroidism. The patient was treated with zuclopenthixol acetate 50 mg intramuscularly (IM) on the day of hospitalization, then haloperidol 10 mg/day and oral lorazepam 4 mg/day for 7 days. On the 7th day, haloperidol and lorazepam were discontinued and aripiprazole long-acting IM injection was administered, and oral aripiprazole 10 mg/day was added. Thyroid replacement therapy was determined by the endocrinology department due to subclinical hypothyroidism. Due to the patient's history of epilepsy, she was consulted to the neurology department, and in her electroencephalography (EEG), focal theta activity and sharp wave activity were detected in the right posterior temporal region. The patient was put on valproate 1000 mg/day on the 13th day of her hospitalization, which was increased to 1500 mg/day on the 19th day, and it was observed that this dose reached the therapeutic blood level (98 mg/L). The EEG taken two weeks after the start of the valproate treatment was reported as normal. The patient, who was evaluated by the neurosurgery department due to a history of intracranial glioma, did not show any change in the glioma in the right frontal lobe in the contrast-enhanced cranial MRI, and continuing routine controls were recommended. The patient who had a history of pulmonary embolism, asthma, COPD and OSAS in the past and who was evaluated by the pulmonary diseases and cardiology departments was prescribed inhaled bronchodilator treatment. Quetiapine 400 mg/day was added to the treatment on the 17th day as the patient's mania symptoms persisted. Quetiapine was discontinued on the 23rd day, as the patient's symptoms persisted and she scored 35 on the YMRS, 25 mg/day clozapine was added to the treatment, and aripiprazole was increased to 15 mg/day. The dose of clozapine was gradually increased to 150 mg/day on the 40th day. Oral aripiprazole was discontinued and she was given a second dose of aripiprazole long-acting IM injection. Due to the adverse effects of hypersalivation and constipation observed in the follow-ups, the clozapine dose was reduced to 100 mg/day on the 44th day and it was observed that the side effects regressed. It was decided to administer ECT for the patient who did not display the expected benefit from pharmacological treatments and did not have a significant regression in the YMRS score. The patient was re-evaluated by the neurosurgery, neurology and pulmonary diseases departments and it was stated that there was no pathology that would constitute a contraindication for ECT. Nine sessions of bitemporal brief pulse ECT with an alternating current between 20 and 120 Hz were administered to the patient three times a week. Rocuronium bromide, thiopental and succinylcholine were used as the anesthesia protocol. There were no adverse effects during and after the ECT sessions. After the 5th session of ECT, the patient's manic symptoms started to regress and her YMRS score decreased to 24; after the 9th ECT session, it was observed that the patient's wellbeing significantly improved and the YMRS score decreased to 6. The patient, whose manic symptoms regressed significantly, was discharged with a prescription of valproate 1500 mg/day, clozapine 100 mg/day, aripiprazole long-acting IM injection 400 mg/month, and weekly outpatient controls were scheduled.

DISCUSSION

When the medical literature is reviewed, it is observed that there is a significant difference between the case reports up to 1980 and the reports after 1980 in terms of the safety of ECT implementation in patients with intracranial lesions (6). The differences in both ECT and anesthesia protocols in the past, the detection of intracranial masses following clinical worsening after ECT in some patients, may be the reason why ECT was considered as a contraindication in these patients. With the development of imaging techniques over the years, intracranial masses that could not be detected in the past and that were not clinically important have begun to be diagnosed. These factors may help explain the reason why adverse effects of ECT are less common in patients with intracranial masses compared to the past.

Small, calcified, slowly growing, benign tumors without associated edema such as meningioma pose relatively less risk (11). The presence of a

large mass, multiple masses (e.g., metastatic lesions), edema, increased intracranial pressure should probably be considered relative contraindications for ECT (12); for such psychiatric patients with these types of intracranial masses who require ECT, ECT implementation may be considered after all other available treatment options have been tried. During this process, it is absolutely necessary to be in contact with the neurology or neurosurgery departments.

ECT applications after steroid pretreatment was used to decrease cerebral edema around the intracranial tumor have been reported (11). In addition, pretreatment with short-acting beta-blocker agents (esmolol) has been shown to safely and effectively block seizure-induced increases in heart rate and blood pressure without significantly affecting seizure duration (13).

Although neuroimaging is not mandatory before ECT, it is recommended for detailed neurological evaluation and possible organic pathology evaluation if additional neurological symptoms develop during or after ECT (6). The administration of ECT in these patients should be evaluated on a case-by-case basis after interdisciplinary consultation with a neurologist and neurosurgeon, following a good benefit/risk assessment.

The inadequacy of data on the application of ECT in PE patients hinders the use of ECT in patients with PE. The limited data available indicate that ECT is safe in patients with PE (14). It has been recommended to consider the use of a beta-blocker and/or remifentanil during ECT in order to reduce cardiac risk factors in patients with recent PE (7). In this case, starting an additional prophylaxis with the recommendation of the pulmonary diseases department was not considered, since the patient had PE a long time ago and was still not undergoing anticoagulant therapy. Although there are no randomized controlled studies on the safety of the use of ECT in patients with PE, cases in which ECT was used safely and without any problems have been reported with no adverse effects (7,10).

The case presented here is, as far as is known, the first report where comorbidities such as intracranial tumor and pulmonary embolism history, which may cause clinicians to hesitate to implement ECT, co-exist. The patient was evaluated by the relevant departments, their recommendations were followed, and she responded to ECT without any complications. Although there is limited evidence regarding the safety of ECT in the presence of comorbidities, ECT can be successfully implemented where there is no response to other treatments, as in this case, with the necessary consultations and close follow-up, without delaying the treatment.

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