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Case Report / Olgu Sunumu

Electroconvulsive Therapy in A Major Depression Patient with Arachnoid Cyst

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ABSTRACT

Electroconvulsive therapy in a major depression patient with arachnoid cyst

Arachnoid cyst develops when two layers of arachnoid membrane fail to unite in early fetal life causing cerebrospinal fluid accumulation and formation of a cyst. Arachnoid cysts constitute 1% of space occupying lesions in the brain. Diagnosis is made usually with regular medical examinations. They can cause epilepsy, increased intracranial pressure, neurological deficits, macrocranium and growth deficit in children, and subdural hematoma. It has been recommended to be careful when administrating Electroconvulsive therapy (ECT) to the patients with space occupying lesions. ECT can cause adverse effects in patients with arachnoid cyst by increasing intracranial pressure. Arachnoid cyst can rupture and give symptoms by causing subdural effusion, subdural hemorrhage or intracystic hemorrhage. In this case, we diagnosed major depression and arachnoid cyst. We administered ECT to the patient who had suicide ideation and was resistant to pharmacotherapy. Patient's complaints fully recovered with ECT. The size of arachnoid cyst didn't increase and we observed no complication. In this case, we aimed to show that ECT could be successfully administered to the patients with arachnoid cyst.

Key words: Arachnoid cyst, ECT, major depression

ÖZET

Araknoid kisti olan majör depresyon vakasında elektrokonvülsif tedavi uygulaması

Araknoid kist, erken fetal yaşamda araknoid membranın iki tabakasının birleşiminin gerçekleşmemesi sonucu oluşur ve bunu beyin omurilik sıvısı birikimi, kist oluşumu izler. Araknoid kistler kafa içi yer kaplayan lezyonların %1'ini oluşturur. Tanı, genelde genel tıbbi incelemeler sırasında konur. Epilepsi, artmış intrakranial basınç, nörolojik defisit, çocuklarda makrokranium ve gelişme geriliği, subdural hematomlara neden olabilirler. İntrakraniyal yer kaplayan lezyonların varlığında elektrokovülsif tedavi (EKT) uygulanırken dikkatli olunması tavsiye edilmektedir. EKT kafa içi basıncını artırarak, araknoid kisti olan hastalarda yan etkilerin oluşmasına neden olabilmektedir. Rüptüre olan araknoid kist, subdural efüzyon, subdural kanama ya da intrakistik kanama ile semptomatik hale gelebilir. Sunmakta olduğumuz bu olguya majör depresyon ve araknoid kist tanısı kondu. Medikal tedaviye yanıt vermeyen ve intihar düşüncesi olan hastaya EKT uygulandı. EKT ile hastanın şikayetleri tamamen düzeldi. Araknoid kistin boyutlarında herhangi bir büyüme veya komplikasyon görülmedi. Bu olguda, araknoid kist ile beraber EKT'nin başarılı bir şekilde uygulanması tartışıldı.

Anahtar kelimeler: Araknoid kist, EKT, majör depresyon

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INTRODUCTION

Arachnoid cyst can be due to congenital, traumatic or inflammatory etiology. Usually it has a developmental origin. Arachnoid cyst develops when two layers of the arachnoid membrane do not unite in early fetal period, which is followed by accumulation of cerebrospinal fluid and cyst formation (1). Arachnoid cysts account for 1% of the intracranial space occupying lesions (2). The most common localization is middle cranial fossa (1). It is two times more frequent in right side of the brain (2-4). It is usually diagnosed during general medical evaluations (2,3). Main clinical findings include epilepsy, increased intracranial pressure, neurological deficit and macro cranium and developmental delay in children (5). Subdural

hematomas are rare complications of arachnoid cysts (5). In the literature, knowledge on association between arachnoid cysts and psychiatric disorders is based on case reports (1,6,7).

Treatment of arachnoid cysts is controversial. Regarding complications of surgical treatments, conservative approaches are preferred in asymptomatic cases. Surgical interventions are advised in cysts which increase in size and cause neurological effects or become symptomatic via bleeding or hygroma (8).

In this case report, we presented a patient with left temporal, 33x17 mm, arachnoid cyst which does not compress neighboring tissues and does not lead to neurological signs who was treated successfully with ECT for major depressive disorder.

CASE

E.Y. was a patient who was 69 years old, illiterate, married with 6 children, and a housewife. She applied with demoralization, distress and abdominal pain complaints to our outpatient clinic. In her history, she stated sleep problems in the last two months, early waking, demoralization going on all day, significant loss of interest to all activities, abdominal pain, generalized body pain, lack of appetite and weight loss. Pessimism, guilt and suicidal thoughts were also present. She did not describe any psychosocial stress. The patient told that she had been using paroxetine 40mg/day for 2 months and that the treatment was not effective. Her psychiatric history revealed that, there were 3 past depressive episodes in the last 8 years and all episodes fully remitted after paroxetine treatment. She had cholecystectomy 40 years ago. There was no family history of psychiatric disorder.

In her psychiatric examination, she appeared older, her appearance was compatible with her socioeconomic status, and her self-care was decreased. Her mood was depressive and affect was anxious. Speech output was decreased. There were no perception problems and psychotic symptoms. Her thought content involved ideas of death and preoccupation with guilt. Her orientation was intact but she had problem in focusing and maintaining attention. Her sleep was decreased and psychomotor activity was slowed. Abstract thinking and calculation abilities were within normal range considering her age and education. Her insight and reasoning were intact.

Routine biochemistry, hemogram, sedimentation rate and thyroid function tests, vitamin B12 and folic acid levels, blood ammonia level and systemic physical examination were normal. Patient was diagnosed with major depression. Hamilton Depression Rating Scale (HAM-D) and Clinical Global Impression (CGI) scores were 31 and 7, respectively, during inpatient admission. Venlafaxine 75 mg/day and alprazolam 1.5 mg/day were initiated. Venlafaxine dosage was gradually increased to 150 mg/day in the second week of hospitalization. Venlafaxine dose was increased to 225mg for ongoing suicidal ideas and electroconvulsive therapy (ECT) was planned. Routine computerized brain tomography before ECT revealed left temporal, 33x17 mm cystic lesion in CSF density (arachnoid cyst). Neurological examination and consultation did not reveal any increased intracranial pressure signs. Cranial nerve examination was normal, deep tendon reflexes were normoactive and muscle power was 5/5 in upper and lower extremities. Brain surgery consultation did not suggest any surgical intervention. There was no papilledema. Radiology consultation revealed that the arachnoid cyst was not connected with ventricles and CSF. Since the patient did not respond to venlafaxine, paroxetine and alprazolam treatments and her suicidal ideas continued, it was decided to perform ECT. HAM-D was 28 and CGI was 6 before ECT. After informed consent was obtained from the patient and her relatives, bilateral ECT 3 times a week were initiated. Consciousness and orientation were assessed and neurological examination was done after each ECT session, no complication was detected. After second ECT session, suicidal thoughts disappeared and depressive symptoms began to improve. After seventh session of ECT, speech, energy and psychomotor activity of the patient were normal. Death ideas, somatic complaints and her preoccupations with guilt in her thought content improved completely. After thirty days of treatment, HAM-D was 3 and CGI was 1. Patient was discharged with venlafaxine 225 mg/day and

alprazolam 0.75 mg/day treatment. CT imaging after her discharge did not reveal any changes in size or features of the arachnoid cyst.

DISCUSSION

Caution has been suggested when applying ECT to patients with intracranial masses (9). ECT may lead to some side effects by increasing intracranial pressure in patients with arachnoid cyst. Since some arachnoid cysts are connected to CSF, increased intracranial pressure during ECT may lead to enlargement of these cysts (10). After rupture of the cyst, patient may become symptomatic with subdural effusion, subdural hematoma or intrinsic bleeding (5). On the other hand, previous studies have suggested that, ECT may be safely administered, particularly in patients with meningioma or other tumors which do not cause increased intracranial pressure (9).

In the literature, 8 patients with intracranial arachnoid cysts were treated successfully with ECT; of these patients three were diagnosed with major depression and others were diagnosed with major depression with psychotic features, bipolar disorder, schizoaffective disorder, psychosis and catatonia (10-12). In three of these patients, ECT was administered after brain imaging did not reveal any growth of the cyst. In our report, arachnoid cyst was detected during routine pre-ECT CT and there were no prior brain images. ECT treatment was decided for presence of suicidal ideation and lack of treatment response. After seven sessions of ECT, there were no complications in the patient. Regarding this, the present case is the first in our country. This case shows that, if arachnoid cyst does not lead to increased intracranial pressure, edema or neurological signs and the cyst is not connected to CSF or ventricles, ECT may be an effective and safe treatment option.

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