Electroconvulsive Therapy for Depression in a Patient With an Intracranial Arachnoid Cyst

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Abstract: Electroconvulsive therapy (ECT) has been frequently considered relatively contraindicated in patients with space-occupying lesions in the brain. After the 7 cases available in the literature, we describe the safe use of ECT in a depressive patient with arachnoid cyst. We provide a comprehensive review on this clinical association, and we conclude that even if the few data available are reassuring, careful neurological evaluation before the ECT treatment is indicated.

Key Words: electroconvulsive therapy, arachnoid cyst, intracranial lesion

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Electroconvulsive therapy (ECT) is a psychiatric treatment in which seizures are induced with electricity for therapeutic effect. Today, ECT is most often used as a treatment for severe major depression which has not responded to other treatments. Although it is a relative contraindication, the possibility of increased intracranial pressure must be carefully assessed.

After the early case of a patient with an intracranial arachnoid cyst who was successfully treated with ECT[1] and the recent case series of ECT in patients with arachnoid cysts,[2] we report here an additional case and summarize the state of knowledge on this topic.

We describe a man with a major depression and an asymptomatic arachnoid cyst of the right anterior temporal and the right lateral prefrontal lobes who was successfully treated with ECT. Structural brain magnetic resonance imaging (MRI) carried out twice (at 1-year interval) before the ECT did not reveal any evolution in the cyst and allowed the use of ECT.

CASE REPORT

M.A. was a 58-year-old man admitted in the Department of Psychiatry with a diagnosis of recurrent major depression, according to the *Diagnostic and Statistical Manual of Mental Disorders Fourth Edition, Text Revision*, of 4-month duration that had been unresponsive to antidepressant medication. The patient's general medical examination was normal, and a detailed neurological examination was nonfocal.

He had experienced his first depressive episode 20 years before admission and was successfully treated with clomipramine. Clomipramine was retried again recently for approximately 3 months, with no effect at all. The patient was eager to undergo ECT.

Previous structural brain MRI performed for depression assessment revealed an asymptomatic arachnoid cyst of the right anterior temporal and the right lateral prefrontal (dimension, 7.6 × 4.1 × 8.1 cm) (Fig. 1). Moreover, MRI was carried out twice (at 1-year interval) before the ECT and did not reveal any evolution in the cyst and allowed the use of ECT.

On admission, the patient exhibited a profoundly sad mood with diurnal variation. He had morbid ideas, feelings of hopelessness and helplessness. Moreover, psychomotor retardation, social withdrawal, decreased libido, and anorexia with a 10-lb weight loss were present. He woke up early in the morning, and his sleeping time was decreased. His score on the Folstein Mini-Mental Status Examination was 30. His score on the Hamilton Depression Scale was 27.

After providing informed consent, M.A. underwent a course of 7 brief pulse bilateral (temporal) ECT treatments, administered 3 times a week in a specialized treatment suite (pulse width, 1 millisecond; frequency, 60 Hz; duration, 3 seconds; current, 800 mA; charge, 288 mC).

Anesthesia included 100% oxygen by mask, 2 mg/kg propofol 1%, 4 mL lidocaine 1%, and 1 mg/kg succinylcholine chloride 5%.

M.A. tolerated the ECT treatment and did not experience any major systemic side effects. He typically achieved complete recovery of his pretreatment orientation within 1 hour of the ECT treatment and denied any persistent memory disturbance. After 3 ECT treatments, the depressive symptoms improved and fully remitted after 5 ECT treatments.

DISCUSSION

Arachnoid cysts are described as benign, congenital, intra-arachnoidal space–occupying lesions that are filled with clear cerebrospinal fluid.[3] Not communicating with the ventricular system and modeled on surrounding structures, they are common and represent 1% of all intracranial masses.[3] The incidence tends to be higher in men. Most arachnoid cysts are supratentorial and approximately half are found in the middle cranial fossa, anterior to the temporal lobes.

Intriguingly, the precise mechanism for the formation of arachnoid cysts is not yet known. Some hypotheses have been proposed: "splitting" or diverticulum of the developing arachnoid, active fluid secretion by the cyst wall, trauma, mastoiditis, meningitis, and subarachnoid hemorrhage.[3]

Of major concern for our clinical issue, arachnoid cysts seem to be generally stable over time. However, there have been reported cases with sudden or progressive enlargement, as well as spontaneous resolution.[3] Moreover, the size of the cystis sometimes small and not preoccupying or sometimes compresses the underlying brain.[3]

According to Osborn and Preece,[3] "The best diagnostic clue for arachnoid cyst is a sharply demarcated extra-axial cyst that can displace or deform the adjacent brain. Scalloping of

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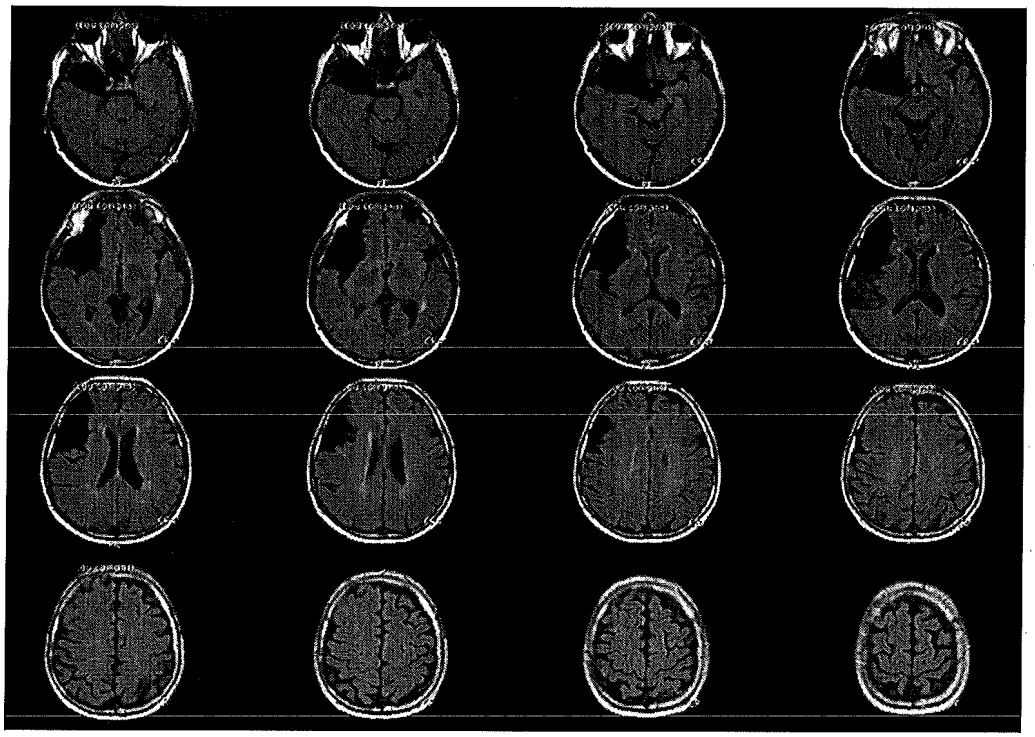

FIGURE 1. Structural MRI showing an arachnoid cyst of the right anterior temporal and the right lateral prefrontal cortex (dimension, 7.6 × 4.1 cm in the axial plane and 8.1 cm in the coronal axis).

the adjacent calvarium is often seen. The classic arachnoid cyst has no identifiable internal architecture and does not enhance. The cyst typically has the same signal intensity as cerebrospinal fluid at all sequences. Occasionally, however, hemorrhage, high protein content, or lack of flow within the cyst may complicate the MR appearance. Arachnoid cysts have an increased prevalence of coexisting subdural hematomas, especially when they occur in the middle cranial fossa."

In addition to the pathophysiology of arachnoid cysts, this case report raises several considerations not only about pathophysiological mechanisms of depression but also about the action and side effects of ECT.

First, to what extent is the arachnoid cyst involved in the pathophysiology of depression? Data on this topic are scarce, and a general conclusion cannot be proposed. Second, to date, there is no consensus about whether there is a hemisphere lateralization in primary and secondary depression. Indeed, as Mayberg[4] stated for depression in lesion-deficit studies, there is no clear consensus as to whether the left or right hemisphere is dominant in the expression of depressive symptoms. She observed in reports of patients with traumatic frontal lobe injury a high correlation between affective disturbances and right hemisphere pathology. In addition, studies in stroke suggest that left-sided lesions of both frontal cortex and the basal ganglia are more likely to result in depressive symptoms than right lesions.[4] Moreover, as for idiopathic depression: "Unlike the lesion-deficit literature, most of the studies report bilateral rather than left-lateralized abnormalities, although asymmetries have been reported."[4] Third, our patient had anarachnoid cyst of the right anterior temporal and right lateral prefrontal cortex. These regions are involved in the pathophysiological network of depression.[4] Thus, it can be hypothesized that alteration of these areas can change the mood.

Second, because the conduction of electrical current from the ECT device may be sensitive to an alteration in brain structure, as in this patient, it may logically be hypothesized that the efficacy of the therapy may be diminished. Nevertheless, congruent with previous results,[1,2] ECT was clinically effective in this patient, as has been described in other patients with organic brain disease.[5]

Third, the side effects of ECT in patients with space-occupying lesions are a long-standing debate because intracranial pressure increases during the treatment. Space-occupying lesions within the brain are classically considered as relative contraindications to ECT.[6] Nevertheless, several clinical reports suggest that ECT may be safely given to patients with these lesions so long as intracranial pressure is not increased. In fact, the contention that ECT is contraindicated for patients who have a space-occupying lesion derives from several observations that the increase in intracranial pressure occurring during ECT treatment is amplified by the space-occupying lesion. In consequence, noncardiogenic pulmonary edema, cerebral edema, brain hemorrhage, and neurological deterioration and death might be precipitated.[5] Maltbie et al[7] confirmed that ECT was contraindicated in patients with intracranial tumor, but it may be permissible in patients with space-occupying lesions if the treatment is strongly indicated.[5] For Abrams,[8] numerous successful prospective administrations of ECT to patients with known brain tumors (mostly meningiomas) largely demonstrate the safety of this procedure when it is performed cautiously and with foreknowledge.

The literature insists on the imperative necessity to check for the presence of increased intracranial pressure and absence of evolution of brain lesions. Intriguingly, the majority of prospective administrations of ECT to patients with brain tumor (mostly meningiomas) were unlikely to have caused increased

intracranial pressure.[8] Moreover, administration of ECT in the presence of a brain tumor, accompanied by increased intracranial pressure, has been shown to be safe and effective after 1-week treatment with parenteral dexamethasone.[9]

Our patient underwent MRI twice, and the results did not show any sign of evolution or displacement of midline structures. Moreover, he did not show any disorientation or short-term memory performance decline after ECT treatment. Congruent with the findings of Escalona et al,[1] our findings show that our patient was not at increased risk for adverse cognitive effects from ECT.

In conclusion, this is an additional case of ECT for depression in a patient with an arachnoid cyst. Despite reassuring data of the 8 cases available in the literature, more systematic research is needed to establish more clearly the risk of ECT in this rare population. Moreover, systematic structural brain imaging before ECT could probably reveal some neurological abnormalities that would have been ignored otherwise.

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