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CASE REPORT

Electroconvulsive therapy (ECT) for intractable depression following epilepsy neurosurgery*

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Psychopathology following epilepsy neurosurgery is a significant risk. Treatment modalities have not been addressed in the literature. As disproportionately elevated suicide rates have been reported, it is critical to treat aggressively any psychiatric illness wherein suicidal ideation is a key component. This case reports the safe utilization of electroconvulsive therapy (ECT) for intractable depression following epilepsy neurosurgery (24 references).

Key words: epilepsy; neurosurgery; electroconvulsive therapy (ECT); depression; neuropsychological testing; amygdalohippocampectomy (AHE); suicide.

INTRODUCTION

Surgical intervention for medically intractable epilepsy is a recognized treatment[1]. Approximately 80% of these surgeries result in total remission or marked reduction in seizure frequency[2]. In the face of such positive results, patient dissatisfaction is most often related to postoperative neurological, cognitive, psychiatric and psychosocial deficits[2]. *En bloc* temporal lobectomy (TL) has been the standard surgical approach for patients with a temporal lobe focus[3]. More recently, selective amygdalohippocampectomy (AHE) has been found to have similar surgical success[4]. Further, AHE has decreased neuropsychologic complications[5–7] when compared to TL. In one series, TL resulted in a 35% prevalence of psychiatric features during an eight-year follow-up with predominantly postoperative affective disorder[8]. In a smaller series with shorter follow-up, AHE resulted in 8% *de novo* affective disorders[9]. Postoperative *de novo* psychoses have also been reported[10]. Thus, the true surgical outcome is a combination of decreased seizures with minimal psychiatric, neurological, neuropsychological, and psychosocial complications. When psychiatric features are present, it is important that the intervention does not compound potential surgical morbidity or result in increased seizures.

This case represents the only known treatment with electroconvulsive therapy (ECT) for intractable depression following AHE.

CASE REPORT

This 26 year old white single female presented to a major medical centre with uncontrolled complex partial seizures of 22 years duration. The patient was not responsive to phenobarbital,

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phenytoin, valproic acid or carbamazepine. Phase I and II evaluations confirmed focal seizure onset within the right mesial temporal lobe, though an MRI had identified a nonepileptogenic left insular cavernous angioma. A selective right AHE was performed with total seizure control now greater than 40 months exluding one seizure while on desipramine.

The patient when fifteen sought counselling for inability to communicate with her parents and difficulty coping with epilepsy. Otherwise, the patient had a good premorbid psychiatric history. Within three weeks of surgery, the patient developed a severe *de novo* major depression[11] characterized by decreased appetite with weight loss, decreased energy, irregular sleep, decreased interests, self-deprecatory thoughts, social withdrawal but no suicidal ideation. During the ensuing year, the patient underwent a series of antidepressant trials including the following psychotropics: nortriptyline, fluoxetine, trazodone, bupropion, lithium (used for synergism), desipramine, doxepin, sertraline, paroxetine, amitriptyline, and methylphenidate.

An unremitting vegetative-affective cluster with suicidal ideation/intent/plan necessitated the urgent hospitalization during which the patient received ECT. The pre-ECT evaluation included normal standard chemistries, CXR, spine series, and EKG. Pre-ECT EEG revealed right temporal delta focus. Pre-ECT SPECT demonstrated right temporal perfusion deficit consistent with AHE. Neuropsychology, neurology, neurosurgery, ECT indications, and ECT competency consultations were performed. This physician reviewed the alternatives, benefits, and risks of ECT as outlined in the California Uniform Informed Consent for ECT with the patient and family[12]. Special circumstances discussed included: potential increased confusion to the point of delirium, increased memory loss to the point of global amnesia, *status epilepticus*[13], and recurrence of seizures secondary to kindling.

The patient underwent a course of 15 ECT treatments utilizing the MECTA-SR1. Power settings were pulse width 1.6–1.8 ms/frequency 70 hz/duration 2.0 s/current 0.7 amp. The second to fourth treatments were right unilateral; all other treatments were bilateral. Total seizure duration from these treatments equaled 535 s. Premedications were 100 mg methohexital and 80 mg succinylcholine. Following the first ECT, rhythmic left hand movement was noted which responded to 3 mg midazolam. Portable 18 channel EEG utilizing the 10–20 electrode placement system[14] revealed the original right temporal delta focus only. Thereafter, each ECT was monitored with 18 channel EEG 2 minutes before, during and for 18 minutes following the treatment. Similar hand movements occurred after the sixth treatment which responded to 3 mg midazolam; however, no neurophysiological correlates were noted. At that juncture, further neurology, neuropsychology, neurosurgery, and external ECT expert consulations occurred. The neurosurgeon was concerned with the risk of producing a kindling effect were further ECT to occur. The neurologist cleared the patient after repeat SPECT was unchanged and an MRI revealed abnormalities consistent with AHE. The ECT consultant strongly recommended a minimum of 15 bilateral ECT. The remaining treatments were uneventful.

Neuropsychometrics were performed pre-surgery, pre-ECT, throughout the ECT course, and serially thereafter. Those tests most sensitive to the learning test–retest phenomenon were alternated. Brief descriptions of those tests reported in this paper are included in the Index[15,16]. The data from these tests are summarized in Tables 1–4.

After the completion of ECT, acute suicidal intent remitted to be replaced with chronic suicidal ideation without active intent or plan. Unfortunately, depressive symptoms with reduced severity also persisted. Further psychotropics and medications known to have positive affective effects were utilized including: phenelzine, imipramine, buspirone, pemoline, bromocriptine, clomipramine, protriptyline, venlafaxine, amoxapine, nefazadone, felbamate, gabapentin, and risperidone. For the past year increasing suicidal ideation with intent and severity of vegetative-affective cluster have returned. At one point the worsening neuropsychometrics led to a PET scan which was consistent with the original surgery but without any structural/functional changes. ECT has again been considered to break the acute suicidal intent.

DISCUSSION

This is a unique case study with obvious design limitations. Specifically, not all neuropsychological tests done pre-surgery were identical to pre-ECT, during the ECT series itself, or post ECT. As a clinical case in progress, basic neuropsychometrics were carried out pre-ECT. Whenever possible, alternative forms of sensitive tests were utilized to minimize the learning test–retest phenomenon. *In toto,* the patient