Retrospective Analysis of the Short-Term Safety of ECT in Patients With Neurological Comorbidities: A Guide for Pre-ECT Neurological Evaluations

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Pre-ECT neurology consultations are often requested to determine the relative risk of the procedure in patients with neurological comorbidities, but there is limited data to guide clinicians. The authors performed a retrospective chart review of all consecutive inpatients at McLean Hospital who underwent a pre-ECT neurological evaluation between January 2012 and June 2014 (N=68). ECT was safe and effective in patients with a wide variety of neurological diseases. Only one minor event was related to a neurological comorbidity, and there were no serious neurological complications. Based on the latest evidence, the authors provide guidance on the pre-ECT evaluation with respect to neurologic status.

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ECT is an effective treatment for major depressive disorder (MDD), bipolar disorder, catatonia, and treatment resistant psychotic disorders.[1,2] ECT is administered in conjunction with a short-acting anesthetic quickly followed by the depolarizing muscle relaxant succinylcholine to minimize discomfort and adverse effects. Brief (0.5–2 msec) or ultra-brief (0.25–0.3 msec) electrical pulses are delivered through two electrodes, utilizing one of three placements [right unilateral (RUL), bitemporal, or bifrontal] two to three times per week, for an average acute course of six to 12 treatments.[1,2] Although ECT performed within an optimal medical setting is considered a very safe procedure,[2–4] it has physiological effects that may lead to undesirable side effects.[5] During the electrical stimulation phase, there is an initial parasympathetic discharge with bradycardia, followed by a surge in sympathetic tone during the seizure, which is associated with hypertension, a transient rise in the intracranial and intravascular pressure, and possibly a disruption of the blood-brain barrier.[2] The acute rise in intracranial and intravascular pressure is the main neurological safety issue, especially in patients with vascular or space-occupying lesions.[2,6] Other concerns include the potentially higher risk of post-ECT delirium and prolonged seizures in the presence of neurological comorbidities.[6,7]

There is a high prevalence of MDD in Parkinson's disease (PD),[8] multiple sclerosis (MS),[9] epilepsy,[10] cerebrovascular accident (CVA),[11] and other neurological diseases. When ECT is indicated to treat psychiatric disorders in patients with neurological comorbidities, neurologists are often consulted prior to treatment to determine the relative risk of the procedure (sometimes inappropriately referred to as "ECT clearance").[6] ECT is thought to be safe with most of the neurological comorbidities,[2,6] but data primarily derives from small case series and case reports.[6,12,13] To address this issue, we performed a systematic retrospective chart review of neurological pre-ECT consultations at a single institution in order to determine the rates of acute complications in patients with neurological comorbidities.

METHODS

Setting

McLean Hospital is a large psychiatric facility (170 inpatient beds) with an active ECT service. All patients are evaluated by internal medicine prior to treatment, and pre-ECT neurology consultations are requested in selected cases. Patients are carefully evaluated by a multidisciplinary behavioral neurology and neuropsychiatry service, and the relative safety of ECT is determined based on the nature, time course, and stability of the neurological disease. Once the assessment is completed, there are three possible outcomes for consultations: 1) low relative risk: the neurological comorbidity has no or minimal impact on the risk of ECT complications; 2) intermediate relative risk: the neurological comorbidity significantly increases the risk of ECT complications and requires specific monitoring (in our setting this sometimes translates into a transfer to a general medical hospital for treatment as McLean is a psychiatric hospital without an intensive care unit); and 3) high relative risk: the neurological

comorbidity could lead to serious post ECT complications (e.g., intracranial bleed), therefore, ECT should be performed only in psychiatrically critical circumstances.

Investigations are sometimes recommended prior to making a final decision on the relative risk of a patient. These can include head neuroimaging (CT-scan or MRI/MRA) to rule out space occupying lesions or vascular lesions, cervical neuroimaging to clarify the nature and stability of cervical injuries, EEG to determine the presence or amount of epileptiform activity when seizure is at issue and to assess for slowing that might result from diverse conditions, and occasionally specific blood tests (e.g., hormonal dosing in patient with a pituitary lesion). Findings can influence the risk stratification in multiple directions (e.g., a small stable meningioma with no mass effect would confirm a low risk, whereas a large undocumented aneurysm would change the category to intermediate or high risk). Neuropsychological testing is sometimes recommended prior to ECT to avoid the confounding effect of treatments on cognitive status but does not have an impact on risk stratification. Modifications to the treatment regimen will sometimes be recommended prior to starting ECT in order to stabilize or optimize the neurological condition of the patient. As an example, prophylactic treatment of headache syndromes could be optimized to minimize the risk of post-ECT exacerbation of headaches. In patients with seizure disorders, modifications to antiepileptic drugs (AEDs) are sometimes suggested in order to decrease the epileptic activity prior to treatment, aiming to minimize the risk of prolonged seizures with ECT.

Chart Review

Approval was obtained from the institutional review board to perform a chart review of all patients who were seen by neurology prior to ECT while being hospitalized at McLean Hospital between January 1, 2012 and June 30, 2014 (2.5 years). A cross-search for patients who were seen in consultation by both the ECT and neurology teams was performed in the electronic medical record. We identified 78 individual patients, of which 68 specifically included a question of pre-ECT assessment. The 68 charts were reviewed by one researcher (S.D.), focusing on the neurological consultation reports, progress notes, ECT procedural notes, and discharge summaries.

Retrieved information included demographics, psychiatric diagnosis, reason for neurological consultation, final neurological diagnosis, ECT risk assessment stratification, results of investigations, number and type of ECT, clinical outcomes, and acute complications of ECT. The goal of this study was to determine the short-term safety of ECT in patients with neurological comorbidities. Potential long-term adverse effects of ECT were outside the scope of this study.

Statistics

Descriptive statistics are provided on the distribution of the reasons for consultations and clinical outcomes. The rate of acute complications was determined. These were classified into minor complications (transient short-term recall impairment, body aches, and headache) and significant adverse events (cardiovascular, psychiatric, and neurological). For neurological complications, we determined whether the event was related or not to the main neurological comorbidity.

RESULTS

The case series included 68 patients (42 women, 26 men) with a mean age of 53.2±16.1 years. The primary psychiatric indications for ECT were MDD (N=39; 57.3%), bipolar I or II disorder – major depressive episode (N=15; 22.1%), bipolar I disorder – mixed episode or delirious mania (N=4; 5.9%), schizoaffective disorder (N=3; 4.4%); treatment refractory schizophrenia (N=3; 4.4%), mood disorder not otherwise specified (N=2; 2.9%), treatment refractory obsessive-compulsive disorder (N=1; 1.5%), and treatment refractory agitation due to Alzheimer's dementia (N=1; 1.5%).

Reasons for Consultation

Table 1 lists the reasons leading to requesting a pre-ECT neurology consult. The most common reason was a documented or possible history of seizure (N=25; 36.8%), followed by traumatic brain injury (TBI) (N=10; 14.7%), prior CVA or transient ischemic attacks (TIA) (N=8; 12.5%), and brain lesion or abnormal MRI (N=7; 10.3%).

Outcomes of Consultation

The risk stratification was determined after the initial assessment without further investigations in 52/68 patients (76.5%). In 15/68 (22.1%), we recommended obtaining specific investigations in order to be able to determine the relative risk. In five subjects, EEG was recommended to evaluate patients with recent possible seizures or unexplained loss of consciousness. One urgent head CT-scan was requested for a possible new-onset partial seizure. Nine patients underwent brain MRIs or MRI/MRAs for reasons such as ruling out brain metastasis, confirm stability of brain lesions, and clarify a history of CVA. Two patients had cervical spine MRIs to assess cervical disc/spine disease. One patient improved without ECT and one was transferred to another hospital (at the patient's request) prior to the investigations, therefore, there was no final risk classification. In one case (1.5%), we deferred to neurosurgery to determine the relative risk of ECT because of the presence of postsurgical intracranial stainless steel material. Consequently, 65 patients had final risk stratification.

In total, 61/65 patients were classified in the low risk category given the stability of their neurological conditions and minimal increase in potential complications from ECT. These included multiple patients with remote mild TBI, patients with remote CVA, stable seizure disorders on AEDs, and various headache syndromes. In some cases, management recommendations to further minimize the risks of neurological complications were provided to the team. This included avoiding neck hyperextension for cervical disease, starting levetiracetam/gabapentin for a probable history of seizure, stopping levetiracetam for an erroneous seizure diagnosis, and various headache management recommendations

TABLE 1. Problems Leading to Pre-ECT Neurology Consultations Listed in Decreasing Order of Frequency[a]

General Category	Frequency (Total Consultations N=68)
Seizure, epilepsy, or unexplained loss of consciousness	N=25 36.8%
Traumatic brain injury or "concussion"	N=10 14.7%
Cerebrovascular accident or transient ischemic attack	N=8 11.8
Brain lesion or abnormal MRI	N=7 10.3%
Headaches or migraine	N=3 4.4%
Parkinson's disease	N=3 4.4%
Cervical spine disease	N=3 4.4%
Dementia or delirium	N=3 4.4%
Abnormal EEG	N=2 2.9%
Miscellaneous - multiple sclerosis - chronic inflammatory demyelinating polyneuropathy - reversible cerebral vasospasm syndrome - possible myopathy - unexplained urinary/fecal incontinence - lymphoma with possible brain metastasis - unexplained neurological symptoms/ somatoform disorder - mild intellectual disability - acute intermittent porphyria	N=1 for each; 1.5%

[a] Some patients had more than one reason for consultation, explaining why the total is above 68.

(e.g., maintain prophylactic treatment, triptans post ECT if needed).

The remaining four patients were classified in the intermediate risk category. One patient suffered from chronic inflammatory demyelinating polyneuropathy (CIDP), which is associated with an increased risk of potentially lethal cardiac arrhythmia with succinylcholine.[14] We recommended that the anesthesiologist avoid or at least minimize succinylcholine during treatment. The anesthesiologist elected to transfer the patient to a general hospital for ECT. One patient had a reversible cerebral vasoconstriction syndrome (RCVS). This patient was classified in the intermediate category given the lack of data on ECT in RCVS. It was recommended to avoid using triptans if the patient developed post-ECT headache, as it is a known trigger of vasospasm. Finally, two patients had stable retro-orbital meningiomas with compression of the optic nerve. In both cases, neuro-ophthalmological follow-up was recommended after ECT (or during treatment in case of new visual symptoms).

No patient was classified in the high risk category over the reviewed 30-month period.

Neurological Diagnosis and Clinical Outcomes

Of the 68 evaluated patients, 13 did not receive ECT because of psychiatric symptom improvement or patient refusal. Five patients were transferred to general medical hospitals for ECT because of nonneurological comorbidities (gastroesophageal reflux, pacemaker). One patient with acute intermittent porphyria elected to receive ECT at another hospital where she had her outpatient management. Table 2 lists final neurological diagnoses in the remaining 49 patients whose post-ECT data were available for review. Eight patients were deemed not to have a neurological disease after reviewing the case. Table 3 provides details on the 41 subjects with neurological comorbidities.

The average ECT course during the inpatient stay was 8.1±3.5 treatments. Forty-four had RUL placement, two started with RUL and were switched to bilateral because of lack of efficacy, and three received bilateral treatments for the complete course.

In 41/49 patients (83.7%), there were no or only well-tolerated side effects post-ECT (transient short-term memory complaints, mild headaches, and transient body/jaw pain). Eight patients had significant adverse events. Three patients had psychiatric complications (two cases of hypomania, one case of agitation/anxiety), and three patients had cardiovascular complications (one case of bradycardia during

TABLE 2. Final Neurological Diagnosis in the 49 Patients With ECT Data[a]

Neurological Diagnosis	Number of Patients (Total N=49)
Cerebrovascular accident or transient ischemic attack	7
Cervical spine disease/radiculopathy	2
Chronic inflammatory demyelinating polyneuropathy (CIDP)	1
Chronic migraines	2
Cisterna magna	1
Cluster headaches	1
Dementia (Alzheimer's)	1
EEG diffuse background slowing secondary to clozapine	1
Epilepsy or probable seizure	14
Intellectual disability - mild	1
Meningioma	2
Multiple sclerosis	1
Paroxysmal hemicrania	1
Parkinson's disease or medication-induced Parkinsonism	2
Postural tremor (essential or medication induced)	2
Syncope	3
Reversible cerebral vasospasm syndrome (RCVS)	1
Traumatic brain injury – mild (remote)	6
Traumatic brain injury – moderate to Severe (remote)	1
No neurological diagnosis	8

[a] Some patients had more than one diagnosis, explaining why the total is above 49.

TABLE 3. Detailed Clinical Information on Patients With Neurological Diseases (N=41) Who Underwent Electroconvulsive Therapy[a]

Subject ID	Age	Gender	Psychiatric Diagnosis	Final Neurological Diagnosis	Final Risk Stratification and Recommendations	Investigations	Number of ECT	Type of ECT	Clinical Outcome	Complications
1	62	M	Schizoaffective disorder	Parkinsonism and akathisia secondary to antipsychotics	Low risk		10	RUL	Significant improvement	None
3	25	M	Schizoaffective disorder	Provoked seizure (Intoxication, alcohol withdrawal)	Low risk		13	RUL	Significant improvement	Transient musculoskeletal pain
4	43	F	MDD	Reversible cerebral vasoconstriction syndrome, restless leg syndrome	Intermediate risk: avoid triptans		3	RUL	Significant improvement	Transient Jaw pain
5	62	M	MDD	Mild traumatic brain injury (remote)	Low risk		18	RUL	Modest improvement	Right ulnar distribution paresthesia (normal cervical MRI), minor transient memory complaints
7	58	M	MDD	C6 neuropathy, mild TBI (remote)	Low risk: avoid hyperextension		12	RUL	Unsustained improvement	None
8	78	F	MDD	Provoked seizures (alcohol)	Low risk		8	RUL	Modest improvement	Minor transient fatigue, pain, and memory complaint
11	47	F	MDD	Syncope	Low risk		5	RUL	No improvement	Premature ventricular contraction after 5th treatment - transfer to general hospital
12	55	F	MDD	Paroxysmal hemicrania	Low risk: indomethacin 50 mg pre and post ECT		8	RUL	Significant improvement	None
14	57	F	Bipolar disorder I - MDE	Mild traumatic brain injury×2 (remote), migraines	Low risk	MRI within normal limits	4	RUL	Marked improved	None
18	65	F	MDD	Stable meningioma with optic nerve compression	Intermediate risk: neuro-ophthalmology follow-up after ECT	MRI with contrast: Right skull base meningioma, origin from sphenoid, compression right optic nerve, no change over time	6	RUL	Significant improvement	Transient confusion and memory loss, hypomania after ECT
19	48	M	MDD	Provoked seizure (tramadol)	Low risk	EEG normal, MRI differed after discharge	7	RUL	Significant improvement	Mild headaches and musculoskeletal aches, brief agitation after last treatment (responded to propofol)

continued

TABLE 3, *continued*

Subject ID	Age	Gender	Psychiatric Diagnosis	Final Neurological Diagnosis	Final Risk Stratification and Recommendations	Investigations	Number of ECT	Type of ECT	Clinical Outcome	Complications
21	19	M	MDD	Enlarged cisterna magna	Low risk		7	RUL	Significant improvement	None
22	78	F	MDD	Alzheimer's disease, syncope	Low risk		7	RUL	Modest improvement	None
23	76	F	MDD	Remote cerebrovascular accident	Low risk		10	RUL	Marked improvement	None
24	76	F	MDD	Remote cerebrovascular accident	Low risk		10	RUL	Significant improvement	None
27	43	F	MDD	Epilepsy (generalized tonic-clonic seizures), narcolepsy	Low risk		8	RUL	Significant improvement	None
30	60	M	Bipolar disorder I - MDE	Provoked seizures (alcohol, benzodiazepines), possible epilepsy	Low risk: continue divalproex sodium	EEG normal, CT-scan (2.5 years prior) normal	18	9 RUL, 9 BL	Modest improvement	Hypertension and premature auricular contraction in the recovery room
35	28	F	Treatment refractory psychosis	Abnormal EEG secondary to clozapine	Low risk: stop oxcarbamazepine given no evidence of seizures		9	BL	Significant improvement	Hypomania - treatment with oxcarbamazepine resumed for hypomania
36	55	F	Bipolar disorder I - MDE	Stable multiple sclerosis, remote seizures	Low risk		8	RUL	Significant improvement	None
37	22	F	MDD	Partial complex seizure×1	Low risk: start levetiracetam/ gabapentin,	MRI recommended but not performed	6	RUL	Modest improvement	Subjective cognitive complaints
39	28	M	Bipolar disorder I - MDE	Possible partial complex seizure	Low risk	EEG and MRI within normal limits	6	RUL	Marked improvement	None
40	40	F	MDD	Mild traumatic brain injury×2 (remote)	Low risk		2	RUL	Significant improvement	None
41	36	F	MDD, suicidal ideation	Complex partial seizures (remote)	Low risk		6	RUL	Significant improvement	Transient post-ECT headaches
45	65	M	MDD	Medication induced tremor, stable cervical disease	Low risk, avoid hyperextension		3	RUL	Significant improvement	None
47	44	M	MDD, suicidal ideation	Mild traumatic brain injury (remote)	Low risk		8	RUL	Significant improvement	None
49	43	M	MDD	Essential tremor	Low risk		12	RUL	Significant improvement	Mild cognitive complaint and post-ECT H/A
50	66	F	MDD	Stable Meningioma	Low risk		11	9RUL, 2BL	Significant improvement	None

continued

TABLE 3, *continued*

Subject ID	Age	Gender	Psychiatric Diagnosis	Final Neurological Diagnosis	Final Risk Stratification and Recommendations	Investigations	Number of ECT	Type of ECT	Clinical Outcome	Complications
51	27	M	OCD	Epilepsy, mild intellectual disability	Low risk		10	RUL	Significant improvement	None
52	67	F	MDD	Motor and sensory chronic inflammatory demyelinating polyneuropathy (CIDP)	Intermediate risk: avoid/minimize succinylcholine		4	RUL	Marked improvement	None
56	60	M	Bipolar disorder I - MDE	Remote cerebrovascular accident, mild TBI (remote)	Low risk		10	RUL	Significant improvement	Mild memory impairment complaints
57	29	M	Bipolar disorder I, mixed episode	Migraines, cluster headache	Low risk: continue verapamil, sumatriptan if needed for post ECT headache		3	RUL	Significant improvement	None
58	60	M	MDD	Parkinson's disease, moderate or severe traumatic brain injury with seizure (remote)	Low risk	EEG with sharp wave (no epileptiform) and intermittent background slowing, MRI with nonspecific FLAIR hyperintensities	10	RUL	Significant improvement	None
61	55	M	MDD	Remote lacunar cerebrovascular accident	Low risk		9	RUL	Significant improvement	None
62	59	M	MDD	Multiple mild traumatic brain injuries (remote)	Low risk	Inconsistent effort on neuropsychological tests, refused MRI (claustrophobia)	4	RUL	Modest improvement	Bradycardia during 3rd treatment - corrected by lowering metoprolol
63	70	F	MD	Remote cerebrovascular accident×2	Low risk	MRI/MRA: old ischemic right posterior circulation CVA, possible narrowing proximal to the right carotid artery	4	RUL	Significant improvement	None
64	62	F	Bipolar disorder I, - MDE, catatonia	Remote transient ischemic attack TIA (4y)	Low risk		12	BL	Significant improvement	None

continued

TABLE 3, *continued*

Subject ID	Age	Gender	Psychiatric Diagnosis	Final Neurological Diagnosis	Final Risk Stratification and Recommendations	Investigations	Number of ECT	Type of ECT	Clinical Outcome	Complications
69	57	F	MDD	Chronic migraines, possible occipital neuralgia	Low risk: NSAIDS and triptans for post-ECT headache		12	RUL	Significant improvement	Increased headache treated with zolmitriptan and butalbital/ acetaminophen/ caffeine, mild cognitive complaints
71	66	F	Bipolar disorder I - MDE	Possible partial complex seizure (tardive seizure post ECT)	Low risk	CT, MRI, and EEG within normal limits	3	RUL	Significant improvement	None after consultation for possible tardive seizure
76	28	M	MDD	Epilepsy in childhood	Low risk	MRI with mild cerebellar vermis atrophy	10	RUL	Modest improvement	None
77	58	M	Dementia due to Alzheimer's disease	Alzheimer's disease, remote cerebrovascular accident and transient ischemic attack	Low risk		9	BL	Significant improvement	None
78	33	F	Bipolar disorder II - MDE	Possible epilepsy versus syncope	Low risk	MRI and EEG within normal limits	6	RUL	Significant improvement	None

[a] BL: bilateral; MDD: major depressive disorder; MDE: major depressive episode; RUL: right unilateral.

ECT, one case of hypertension and premature atrial contractions post-ECT, one case of premature ventricular contractions post-ECT). Two patients (4.1%) had neurological complaints after starting ECT. One developed an ulnar neuropathy, which was unrelated to the initial reason for consultation (mild remote TBI). One patient with chronic migraines suffered an exacerbation of her usual headaches, which was successfully treated with zolmitriptan and butalbital/acetaminophen/caffeine. This was the only case of an ECT induced adverse reaction that was related to the neurological comorbidity.

In terms of treatment efficacy, 38/49 patients (77.6%) had clinically significant to marked psychiatric improvement during the acute course of ECT based on discharge summaries. The remaining 11 patients had modest, unsustained, or no improvement. Of the 41 patients with a confirmed neurological comorbidity, 32 (78.0%) had significant to marked improvement.

DISCUSSION

This is one of the largest case series and the first systematic collection focusing on patients with neurological comorbidities undergoing ECT at a single site. Results support that ECT is safe in the short-term in patients with a wide variety of neurological diseases (Table 2). Out of 41 patients with neurological comorbidities, only one had a mild exacerbation of their condition (2.4%), and there were no serious adverse events. None of the 14 patients with a probable history of seizures had prolonged seizures after ECT. None of the patients with a past history of TBI or CVA/TIA had neurological complications. Our sample included the first reports of safe and successful ECT in one patient with RCVS and one with CIDP. Furthermore, our data suggest that neurological comorbidities do not negatively impact efficacy.[1]

The low frequency of significant complications identified in this cohort of patients with neurological comorbidities is compatible with large studies on the rate of all types of adverse events in patients receiving ECT.[3,4] Nuttall et al. (2004) identified a rate of complications (including prolonged seizures) of 0.92% and no deaths in a sample of over 17,000 ECT treatments. In a large review, Watts et al. (2011) estimated the rate of death to be below 1/73,440 treatments. Overall, cerebrovascular complications (excluding cognitive complaints

and transient confusion) are less common than cardiovascular adverse events.[3,5] It should be noted that a significant proportion of adverse events are related to mistakes during the anesthesia process (including inadequate paralysis), which can be minimized by implementing structured protocols.[4]

In the following sections, we review the literature related to the safety of ECT in patients with specific neurological comorbidities. Integrating the current evidence with our results, we provide the key clinical aspects to review when performing pre-ECT neurological evaluations. A list of relevant single-case reports is provided in supplemental material (S1).

General Principles

Although each neurological disease requires a specific assessment (see discussion below), a few general principles of the pre-ECT neurological evaluation can be outlined. First, it should be determined if the neurological comorbidity is remote, current but stable, or current and active. If the disorder is not well controlled (e.g., epilepsy with frequent seizures), the consultant should try to optimize treatment prior to ECT, if possible. Second, it should be determined if the neurological comorbidity could be exacerbated by the physiological changes induced by ECT (including increased intracranial pressure). If this is the case, the consultant should inform the treating team of the potentially increased risk associated with ECT, and work in concert with the psychiatrist and anesthesiologist to minimize those risks. Third, the consultant should provide ongoing monitoring in patients with neurological diseases at higher risk of complications.

Seizure/Epilepsy

The two main concerns in patients with epilepsy are the potentially higher risk for a prolonged seizure and the impact of antiepileptic drugs (AEDs).[15] In the largest case series, ECT was found to be safe, with only occasional need to reduce AEDs.[15] In fact, ECT leads to an anticonvulsant response in the brain, which increases seizure threshold and might improve seizure control.[2] ECT has even been used as a last resort treatment of status epilepticus.[16] While maintaining AEDs increases the seizure threshold, it has not been associated with added complications.[17] In light of these studies and our results, a history of seizure/epilepsy requires careful consultation and management pre-ECT, but does not usually pose a contraindication. Patients with a history of status epilepticus should be closely monitored for prolonged seizures. AEDs should be maintained in patients with an active seizure disorder.[15] Patients with epilepsy but no recent seizures can skip their dose on the morning of treatment or the previous night (AEDs can be given after treatment).[15] No modification of the treatment protocol is necessary, but ECT might require a higher charge in the presence of AEDs.

Abnormal EEG

Abnormal EEGs in psychiatric inpatients are common, frequently related to psychotropic medications.[18] EEG background slowing could be a risk factor for post-ECT cognitive impairment,[6] but seems to have little impact on outcomes.[19] We do not consider an abnormal EEG because of medications (including clozapine) or other benign factors to be a contraindication. If there is unexplained diffuse background slowing, an investigation for an encephalopathy should be performed to determine the diagnosis prior to ECT. Markers of possible seizures (e.g., spikes, sharp activity) are not contraindications to ECT. Of note, EEGs are expected to be abnormal for up to a few months after ECT.[20]

Traumatic Brain Injury

There is no evidence from the literature and our results that a history of mild TBI increases the rate of complications after ECT.[12,21] Therefore, mild TBI is not considered a contraindication to ECT even when it is recent or multiple,[21] and brain imaging is not necessarily required. It may be helpful to review imaging prior to ECT in moderate or severe TBI cases. Remote hemorrhages and gliosis could theoretically cause cortical irritability and increase the risk of prolonged seizures. However, ECT has been safely performed in this population without excess cognitive adverse effects.[21] If possible, ECT lead placement should be modified to avoid areas of skull defect or abnormal brain tissue (S1).

CVA/TIA and Vascular Lesions

The main concern in patients with vascular problems (such as CVA/TIA, cerebral aneurysms, subdural hematoma, hemangioma, or cerebral amyloid angiopathy) is that the acute increase in intravascular and intracranial pressure during ECT could lead to bleeding or lesion expansion (S1).[6] This is most significant in the acute period. For a recent ischemic CVA, ECT can be performed in critical situations (S1), but preferably delayed due to the risk of hemorrhagic conversion.[6] Strokes that are stable (older than ≈1–2 months) and asymptomatic white matter abnormalities are not contraindications to ECT.[22,23] The only caveat is to ensure that an adequate CVA work-up has already been performed to rule out stroke etiologies that are potentially relevant to ECT. These include cardiac arrhythmias that may be worsened during ECT, critical carotid or basilar stenosis that might be worsened during hyperextension of the neck, or coagulation disorders. There is limited data on the topic, but for hemorrhagic strokes it is advisable to wait for a few months after the event before proceeding with ECT.[6]

Chronic subdural hematoma, stable venous angiomas, and small stable aneurysms not requiring surgical intervention are not considered contraindications to ECT (S1).[24,25] ECT should only be performed in the most critical situations in the presence of an unstable aneurysm. Vascular lesions should be discussed with the anesthesiologist. It is recommended to minimize hypertension and tachycardia by giving a beta-blocker (or another antihypertensive) prior to the procedure.[2]

Brain Lesions

Intracranial space occupying lesions used to be perceived as a contraindication to ECT because of high rates of complications in early reports.[26] However, with modern ECT techniques

the evidence clearly indicates that stable masses without edema, mass effect, or raised intracranial pressure do not substantially increase the risk.[27] There are reported cases of safe and successful ECT in the presence of meningioma, glioblastoma, metastasis, and surgical lesions (S1). Arachnoid cysts could theoretically enlarge because of ECT, but reported cases have not been associated with complications.[28] Masses that are associated with increased intracranial pressure or edema do not constitute absolute contraindications to ECT (S1), however, ECT should only be reserved for critical cases. Treatment should be performed in consultation with neurosurgery and anesthesiology, using strategies to minimize the increase in intracranial pressure (e.g., corticosteroids, diuretics, hyperventilation).[2]

Headaches and Migraines

There is no theoretical contraindication to ECT in patients with headache syndromes. However, ECT frequently causes or exacerbates headaches,[29] such as the case in our series. Patients with tension, migraine, medication overuse, caffeine withdrawal, and cluster headaches do not have an increased risk of significant complications and do not require specific investigations pre-ECT. Patients should be warned that ECT could exacerbate their symptoms, especially in younger patients with disabling headaches.[30] Brain imaging should be obtained for positional headaches suggestive of raised intracranial pressure, or if clinically indicated as per standard guidelines.[31] In patients with frequent headaches, we recommend nonsteroidal anti-inflammatory drugs (NSAID) pre- and post-ECT.[32] Triptans can be used pre-ECT for migraine (S1), but we favor NSAID/acetaminophen due to the associated cardiovascular risk and vasoconstriction. Triptans can be safely used for post-ECT headaches.[33]

Parkinson's Disease and Other Movement Disorders

Patients with PD might be at higher risk for post-ECT delirium,[34] but PD is not a contraindication for having ECT.[6] In fact, there is some evidence that ECT might have a beneficial impact on the motor symptoms of PD.[35] The surge in dopaminergic transmission with ECT could also theoretically exacerbate psychosis and dyskinesia due to PD.[35] However, this has not been an issue in the two cases in our series and in our experience. Should these adverse effects or post-ECT confusion occur, temporarily lowering dopaminergic medications may be considered. ECT has also been safely used in patients with multiple system atrophy.[36] ECT could also theoretically lead to an exacerbation of tardive dyskinesia, but this has not been an issue in our experience. There are reports of ECT leading to improvement of tardive dyskinesia[37] and dystonia (S1). While the data are too limited to reach conclusions in terms of efficacy, movement disorders do not constitute contraindications to ECT.

Neurodegenerative Major Neurocognitive Disorder

Patients with dementia could be at increased risk of transient cognitive worsening post-ECT.[12,38] However, it is overall a safe and effective treatment for depression in this population.[38–40] A proportion of patients can even show some improvement of cognitive function post-ECT.[38] In addition, ECT could be helpful to treat agitation in patients who do not respond to medications.[40] In summary, dementia is not a contraindication to ECT, but it does warrant closer monitoring during treatment.

Multiple Sclerosis

ECT is thought to be effective for MDD associated with MS, however, there are reports of neurological deteriorations in up to 20% of patients.[41] Our approach consists of reviewing the history of symptom exacerbations, relapses, and treatments. In patients not currently in an active episode and on stable treatment, ECT is probably safe.[42] Brain MRI with contrast should be obtained if there are active MS symptoms, as imaging may provide another surrogate marker for disease activity. Active disease might be a risk factor for neurological deterioration post ECT,[41] therefore, ECT should only be considered in psychiatrically critical cases during an acute MS exacerbation.

Intracranial Devices

Intracranial devices can be metallic (e.g., deep brain stimulator, coils/clips) or nonmetallic (e.g., ventriculoperitoneal shunt). The main concerns are that the electricity could be transmitted to the device leading to potentially damaging heat, and increased risk of prolonged seizures. That being said, there have been over 20 reported cases of ECT in patients with metallic devices (including deep brain stimulators), and none have suffered complications.[43,44] Although there is limited experience, normal pressure hydrocephalus and shunts do not appear to increase complications (S1).

Cervical Disease

There are concerns that ECT could aggravate nerve root or spinal compression due to cervical spine disease (S1). Cervical neuroimaging is generally recommended if there are neurological signs on exam, but the presence of stable cervical disease is not a contraindication to ECT. In our practice, we inform the anesthesiologist to ensure adequate paralysis with succinylcholine and minimal cervical extension during treatment. If there is unstable cervical disease, neurology/neurosurgery consultations should be obtained prior to ECT, but treatment could be performed in critical situations (S1).

Neuromuscular Diseases

Potential complications with myasthenia gravis (MG), CIDP, and other neuromuscular diseases is not related to ECT per se, but instead to muscle relaxants.[14] MG is associated with slow recovery from succinylcholine and increased sensitivity to nondepolarizing agents. Importantly, Guillain-Barre, CIDP, and other causes of muscle atrophy/paralysis (including CVA and prolonged catatonia) carry a risk of life-threatening arrhythmias with succinylcholine.[14] These conditions do not constitute absolute contraindications to ECT,[45] but should be

discussed with the anesthesiologist. Strategies include using lower doses of succinylcholine and using a nondepolarizing muscle relaxant (except for MG).

Reversible Cerebral Vasoconstriction Syndrome

There was one case of safe and effective use of ECT in a patient with RCVS in our sample. No other case was found in the literature. RCVS can be triggered by selective serotonin reuptake inhibitors (SSRIs), therefore, ECT could theoretically constitute a trigger. Our case shows that it is feasible to do ECT in this population, but there is too little data to reach conclusions on safety. Close neurological monitoring is recommended.

CONCLUSIONS

This case series adds to the current literature on the short-term safety of ECT in patients with neurological diseases. There are no absolute contraindications to ECT, and treatments can be administered safely in the presence of most neurological comorbidities. A pre-ECT neurology consultation should be considered in patients with a neurological comorbidity that could increase the risk of ECT related complications. The role of the neurology consultant is to clarify the neurological diagnosis, provide a basic risk stratification to help patients and their physician make an informed decision about the treatment, optimize treatment prior to ECT, and ensure ongoing monitoring in higher risk patients.

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