Successful Use of Electroconvulsive Therapy in the Setting of Multiple Risk Factors for Retinal Detachment

To the Editor: Electroconvulsive therapy (ECT) is an effective method of treatment for numerous psychiatric disorders, but it also precipitates distinct effects on intraocular pressure (IOP).[1] Administration of propofol and succinylcholine cause, respectively, a decrease and increase in IOP.[1] During convulsions, IOP further rises before returning to baseline after approximately 10 minutes.[1] The significance of these transient pressure changes remains unknown.

Changes in IOP co-occur with retinal detachment.[2] A majority of patients with unilateral retinal detachment possess relative hypotony in the affected eye.[2] How a change in IOP might cause retinal detachment remains uncertain; a competition between IOP and choroidal pressure might induce exudative detachment.[2] When choroidal pressure exceeds IOP, fluid accumulates behind the retina and delamination ensues.

A patient subjected to ECT might, therefore, be at risk for retinal detachment.[3] If a patient possesses additional risk factors for retinal detachment, one must ask, "Is ECT contraindicated?"

Here, we describe the case of a depressed patient with lattice degeneration of the retina, which predisposes to retinal detachment. The patient underwent ECT for psychotic depression, to which she responded well with no ophthalmologic complications.

Case report. Ms A, a 70-year-old white woman, was hospitalized for worsening psychotic depression. In addition to depressive signs and symptoms, she exhibited a preoccupation with having multiple neurologic and cardiovascular diseases; assessments by neurologists and a cardiologist were inconclusive. Ms A met *DSM-5* criteria for major depressive disorder with psychotic features.

Prior to admission, Ms A was treated for depression with escitalopram 20 mg, olanzapine 10 mg, and psychotherapy. Her outpatient psychiatrist noted that her symptoms had not improved, and she was unable to function in her role as a teacher. Of note, Ms A's past medical history was significant for essential hypertension, hypercholesterolemia, osteoporosis, lattice degeneration of the retina in her left eye, and repair of a retinal detachment in her right eye 30 years prior. She was additionally treated with gabapentin, amlodipine, hydrochlorothiazide, enalapril, and aspirin for various medical ailments.

Clinical assessment revealed a Hamilton Depression Rating Scale (HDRS)[4] score of 32 and a Montreal Cognitive Assessment (MoCA)[5] score of 23. Brain magnetic resonance imaging revealed no pathology. On our unit, Ms A's olanzapine dose was titrated to 20 mg, and her escitalopram was cross-tapered to sertraline 200 mg, but she exhibited no response. Given the severity of symptoms and lack of clinical response, the treatment team recommended ECT. Ms A was provided a daily antihypertensive (lisinopril 10 mg) prior to each treatment, and blood pressures were measured both before and after each session. She underwent 10 ECT treatments with no complication (Table 1).

After completion of her ECT course, Ms A displayed significant improvement in all symptoms, and her HDRS and MoCA scores improved to 6 and 28, respectively. Sertraline and olanzapine were cross-tapered to venlafaxine (225 mg/d) and lithium (600 mg/d). One month postdischarge, her psychiatric symptoms remained in remission with no ophthalmologic complications.

Prior to this report, no evidence has been published describing successful use of ECT in a patient with lattice degeneration. Because ECT induces transient changes in both IOP and blood pressure, it remains unclear whether ECT precipitates retinal tearing in a patient with ophthalmologic comorbidities.[1,2,6] Our patient displayed no complications. This finding is consistent with prior reports[6,7] of successful ECT treatment in patients who had undergone ophthalmologic surgeries.

Further studies must elucidate the risk of ophthalmologic complications from ECT for different ophthalmologic comorbidities. Per this report, however, we can conclude that those patients suffering from treatment-resistant major depressive disorder in the setting of lattice degeneration should not be dissuaded from treatment with ECT.

References

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Table 1. Summary of Electroconvulsive Therapy (ECT) Treatments[a]

Treatment Number	Protocol	Modality	Succinylcholine (mg)	Propofol (mg)	Frequency (Hz)	PW (s)	Duration (s)	Current (A)	Charge (mC)	Energy (%)	Seizure Time		Impedance	
											Motor (s)	EEG (s)	Static (Ω)	Dynamic (Ω)
1	TD	RUL	40	40	10	0.25	5.6	0.9	24.8	5	39	72	1,570	260
2	ST	RUL	40	40	50	0.25	6.7	0.9	151	30	29	74	1,590	260
3	ST	RUL	40	40	50	0.25	6.7	0.9	150	30	23	42	1,500	240
4	ST	RUL	40	40	50	0.25	6.7	0.9	151	30	22	40	NA	230
5	ST	RUL	40	40	50	0.25	6.7	0.9	151	30	18	27	1,310	250
6	ST	RUL	35	40	50	0.25	6.7	0.9	150	30	21	32	1,620	260
7	ST	RUL	35	40	50	0.25	6.7	0.9	151	30	37	42	1,490	250
8	ST	RUL	35	40	50	0.25	6.7	0.9	151	30	19	40	1,560	250
9	ST	RUL	35	40	50	0.25	6.7	0.9	150	30	NA	NA	NA	NA
10	ST	RUL	35	40	50	0.25	6.7	0.9	151	30	23	38	1,120	250

[a]The patient underwent 10 ECT treatments during a 22-day period. Displayed in the table are the protocol type, modality, medications including succinylcholine and propofol, pulse width, current, charge, and percent energy. Outcomes of the session, including the seizure time by motor and EEG activity and both static and dynamic impedances, are shown.

Abbreviations: EEG = electroencephalogram, mC = millicoulomb, NA = not available, PW = pulse width, RUL = right unilateral, ST = suprathreshold treatment, TD = threshold determination.

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