

Electroconvulsive Therapy in a Patient With a Recent Subarachnoid Hemorrhage

Seshagiri Rao Doddi, MBBS, Matthew Lotz, BS, William T. Regenold, MDCM, and Lawrence Adler, MD

Abstract: The safety of electroconvulsive therapy (ECT) is improving with advances in anesthesia and ECT technique. There are published case reports of successful treatment of depression in patients who were once considered at high medical risk. Recent cerebral hemorrhage is one of the conditions considered to significantly increase the risk of ECT treatment. Literature search did not indicate any case reports of ECT treatment in patients with recent subarachnoid hemorrhage. We report the successful ECT treatment of depression in an older man who had developed a subarachnoid hemorrhage after a suicide attempt by ingestion of antifreeze.

Key Words: electroconvulsive therapy, ECT, subarachnoid hemorrhage, depression, older adult, ethylene glycol, suicide

(*J ECT* 2017;00: 00–00)

Electroconvulsive therapy (ECT) is a safe and effective treatment for patients experiencing a major depressive episode.^{1–4} Recent review articles suggest that the safety of ECT has been enhanced over decades because of improvements in anesthesia and ECT techniques with decreasing rates of medical morbidity and mortality due to ECT treatments.^{4–6} These improved outcomes apply even to patients who were once considered high risk for ECT. For example, case reports show that ECT can be safely used to treat depression in patients with recent myocardial infarction,⁷ severe thrombocytopenia,⁸ severe hemophilia,⁹ brain aneurysms,^{10–13} repaired arteriovenous malformations,¹⁴ intracranial tumors,¹⁵ intracranial arachnoid cysts,¹⁶ and subdural hemorrhage.¹⁷ On the other hand, there are also case reports of intracerebral hemorrhage and subdural hematoma after ECT that suggest that individuals with recent intracerebral bleeds are at risk for further bleeding.^{17–20} A literature search using Medline, PubMed, and PsycINFO did not reveal any reports of ECT treatment in patients with a recent subarachnoid hemorrhage. Here, we report a case of an older adult with major depression who sustained a subarachnoid hemorrhage and was successfully treated with ECT.

CASE REPORT

Mr Z, a 72-year-old married white man, presented with a 6-month history of major depression. His depressive symptoms included persistent and pervasive depressed mood, anhedonia, loss of appetite, decreased energy, inappropriate guilt, feelings of hopelessness and helplessness, and preoccupation with death and suicidal ideas. His depression occurred in the context of multiple stressors including chronic pain, physical disability, and financial concerns.

Mr Z had no previous psychiatric history and no history of alcohol or substance use. His past medical history was notable for prostate cancer treated 16 years ago by prostatectomy with resulting incontinence and erectile dysfunction; a lumbar back injury from a fall resulting in chronic pain, numbness, and weakness in his legs with impaired gait requiring a walker, unrelieved by a L4 to L5 fusion surgery several months before admission; mild hypertension treated with amlodipine 2.5 mg daily, and gastroesophageal reflux disease treated with pantoprazole 20 mg daily.

During the second month of his depressive episode, Mr Z made his first suicide attempt by ingesting antifreeze and making multiple deep cuts on several parts of his body. A computerized tomography (CT) scan of his head after this first suicide attempt was read as normal with no evidence for an acute intracranial bleed, acute ischemia, or mass effect. He was treated as an inpatient for 15 days at the Shock Trauma Center (STC) of the University of Maryland Medical Center. After he was extubated, he was seen by psychiatric consultants who did not start any psychiatric medications at that time because of resolving delirium. After medical management and surgical repair of muscles and tendons, he was transferred to an acute rehabilitation facility. He was closely followed by psychiatric consultants who started escitalopram 10 mg daily, which was titrated to 20 mg per day. He also received individual therapy and showed significant improvement in his depressive symptoms. He was discharged home 2 months after his initial suicide attempt and was referred for outpatient psychiatric treatment. He saw a psychiatrist and a psychotherapist as an outpatient but did not fully comply with escitalopram therapy. He relapsed and made a second suicide attempt 4 months after his discharge from the rehabilitation facility.

Mr Z was found by neighbors in his backyard lying unresponsive with an empty antifreeze container next to him. He was taken to a local hospital emergency room by ambulance where he was intubated. Physical examination did not show any bruises, bumps, or external evidence of trauma to his head. A CT scan of the head showed subarachnoid hemorrhage over the left parietal and temporal lobes with no mass effect. The same day, he was transferred to the STC of the University of Maryland Medical Center from the local hospital for further medical management. A repeat head CT scan on the day after admission to the STC showed unchanged volume and distribution of subarachnoid blood over the left parietal and temporal lobes without evidence of interval recurrent intracranial hemorrhage (Fig. 1). He also experienced multiple metabolic and toxic effects due to the ethylene glycol contained in antifreeze, including hypernatremia, hyperkalemia, hyperlactemia, hyperosmolarity, and severe metabolic acidosis. In the STC, he remained intubated with mechanical ventilation. As per advise of poison control, he was treated for 48 hours with fomepizole (4-methylpyrazole), which blocks the breakdown of ethylene glycol into its toxic metabolites. He received multiple rounds of hemodialysis for treatment of metabolic acidosis and acute renal failure. After medical stabilization and extubation, he was evaluated by the psychiatry consult service. One week after his initial admission, he was transferred to the Older Adult Psychiatric Inpatient Unit.

From the Department of Psychiatry, University of Maryland School of Medicine, Baltimore, MD.

Received for publication June 13, 2017; accepted July 21, 2017.

Reprints: Seshagiri Rao Doddi, MBBS, Department of Psychiatry, University of Maryland Medical Center, Room No. S12 A05, 22 South Greene St, Baltimore, MD 21201 (e-mail: sdoddi@som.umaryland.edu).

The authors have no conflicts of interest or financial disclosures to report.

Copyright © 2017 Wolters Kluwer Health, Inc. All rights reserved.

DOI: 10.1097/YCT.0000000000000453

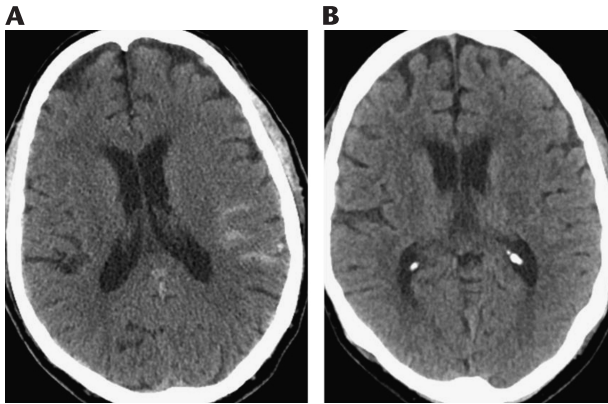


FIGURE 1. Noncontrast CT scan of the patient’s head. A, The day of suicide attempt. Hyperintensities in left temporoparietal region indicate subarachnoid hemorrhage. B, Three days before first ECT. No evidence of subarachnoid hemorrhage.

Upon admission to the psychiatric unit, he remained severely depressed with all symptoms of depression noted earlier as well as psychomotor retardation, a nihilistic delusion that he was hopelessly impoverished, and regret over not succeeding in his suicide attempts. Electroconvulsive therapy was considered, but because of his recent subarachnoid hemorrhage, he was deemed too high risk for ECT, because an extension of his bleed could result in a mass effect and possibly a catastrophic increase in intracranial pressure. He was restarted on escitalopram, and the dose was quickly increased to 20 mg per day. He received individual, group, and milieu therapies. Over 4 weeks on the inpatient unit, he showed minimal improvement. Despite frequent reviews by his managed care company encouraging discharge to outpatient treatment, he was considered to be at too high risk for suicide because of his recent attempts and multiple suicide risk factors including advanced age, male sex, social isolation, psychosis, strong family history of depression and suicide, chronic medical illness and pain, and financial stressors. Electroconvulsive therapy was therefore reconsidered.

To fully assess the current risks of ECT, a repeat head CT and a neurosurgery consult were obtained. The head CT 30 days after his initial CT showed interval resolution of the subarachnoid hemorrhage over the left parietal and temporal lobes with no evidence of new hemorrhage (Fig. 1). The neurosurgery team agreed with our assessment that the possible benefit of ECT for this patient at high risk for suicide now outweighed the risk of a possible new hemorrhage with ECT. The patient and family were informed of the increased risk for adverse events with ECT treatment because of the recent subarachnoid hemorrhage. The patient provided written informed consent, and the family gave verbal assent for ECT. His first treatment occurred 33 days after his subarachnoid hemorrhage was seen on CT scan. Over the next 3 weeks, the patient was continued on escitalopram 20 mg per day and was treated with a total of 9 bifrontal ECT treatments using a Thymatron System IV device (Somatics, LLC, Lake Bluff, Ill). Device settings were as follows: pulse width = 0.5 ms; frequency = 30 Hz; stimulus duration, 5.6 to 6.5 seconds; total energy = 35 to 40 J; and charge = 150.6 to 178.5 mC. Methohexital 70 mg and succinylcholine 60 mg were administered intravenously for anesthesia and neuromuscular blockade, respectively, before stimulation. Labetolol 15 to 20 mg intravenously was given before stimulation to prevent a large increase in blood pressure. A CT scan of the head was repeated after the first procedure to assess for bleeding, which showed no intracranial hemorrhage or any

other acute intracranial process. Neurological assessments were performed every 15 minutes up to 2 hours after each ECT treatment. The patient tolerated the ECT treatments without complications. Montgomery-Asberg Depression Rating Scale (MADRS) and Mini-Mental Status Examination (MMSE) were administered once before ECT treatment, on a weekly basis during ECT treatments, and after the ECT treatment to monitor for changes in severity of depression and cognition, respectively. The patient showed dramatic improvement over the course of ECT treatment, which was reflected in a decrease of MADRS score from 34 pretreatment to 5 posttreatment, indicating remission of his depression (Table 1). There were no significant changes noted in the MMSE, which ranged from 28 to 30 (Table 1). The patient was discharged home in stable condition with outpatient psychiatric and medical follow-up. Mr Z was contacted by phone 5 months after his discharge from the psychiatry unit. He reported continuing escitalopram 20 mg daily, seeing a psychiatrist regularly, and maintaining sustained remission of his depression.

DISCUSSION

To our knowledge, this is the first report of ECT performed on a patient with a recent subarachnoid hemorrhage. With regard to the cause of this patient's subarachnoid hemorrhage, it is noteworthy that this patient attempted suicide by ingestion of antifreeze. Ethylene glycol is one of the ingredients of antifreeze, and its metabolites cause metabolic acidosis and result in formation of calcium oxalate crystals. The metabolites and calcium oxalate crystals cause toxicity resulting in acute renal failure, metabolic acidosis, and central nervous system toxicity.²¹⁻²⁴ Intracerebral bipallidal hemorrhage has been reported with ethylene glycol ingestion and is hypothesized to be related to the deposition of oxalate crystals.²¹ We verified that the antifreeze used in the suicide attempt contained ethylene glycol and not methanol, which is another possible ingredient of antifreeze that has been associated with subarachnoid hemorrhage.^{25,26} Trauma was considered an unlikely cause of the hemorrhage because examination revealed no evidence of head trauma. Rupture of a blood vessel, such as an arteriovenous malformation or cerebral aneurysm, was also deemed unlikely because of the small size and lack of progression of the hemorrhage, although it could not be definitively excluded because the patient did not have a cerebral angiogram.

Active cerebral hemorrhage and recent cerebrovascular accident are considered relative contraindications to ECT.^{27,28} During ECT treatment, there is an initial brief parasympathetic response after electrical stimulation. This is followed by a sympathetic response associated with a hyperdynamic state that is characterized by arterial hypertension, tachycardia, and considerably increased cerebral blood flow rate and velocity.^{29,30} These responses lead to a concern for increased risk of intracranial hemorrhage in

TABLE 1. MADRS and MMSE Scores Before, During, and After ECT

MADRS and MMSE Scores Over ECT Course		
Time	MADRS	MMSE
Pre-ECT	34	29
Week 1	30	29
Week 2	20	30
Week 3	7	28
Week 4 (post-ECT)	5	29

vulnerable patient populations. Administering ECT treatment after recent cerebrovascular hemorrhage increases the risk for new hemorrhage and therefore requires careful assessment of risks and benefits. In the case of this patient, the likely benefit of relieving his depression and the extremely high risk of suicide without depression relief outweighed the risk of morbidity and mortality associated with renewed subarachnoid hemorrhage in light of hemorrhage resolution and advice from the neurosurgery team.

In sum, this case illustrates that ECT can be administered safely to a patient 33 days post-subarachnoid hemorrhage without mass effect by taking essential precautions including follow-up brain imaging, neurosurgical consultation, and the use of β -blocker medication (eg, labetalol) to minimize the risk of intracranial hemorrhage associated with hemodynamic changes accompanying ECT.

REFERENCES

- Fink M. Convulsive therapy: a review of the first 55 years. *J Affect Disord.* 2001;63:1–15.
- van der Wurff FB, Stek ML, Hoogendijk WJ, et al. The efficacy and safety of ECT in depressed older adults: a literature review. *Int J Geriatr Psychiatry.* 2003;18:894–904.
- Abrams R. The mortality rate with ECT. *Convuls Ther.* 1997;13:125–127.
- Watts BV, Groft A, Bagian JP, et al. An examination of mortality and other adverse events related to electroconvulsive therapy using a national adverse event report system. *J ECT.* 2011;27:105–108.
- Rice EH, Sombrotto LB, Markowitz JC, et al. Cardiovascular morbidity in high-risk patients during ECT. *Am J Psychiatry.* 1994;151:1637–1641.
- Nuttall GA, Bowersox MR, Douglass SB, et al. Morbidity and mortality in the use of electroconvulsive therapy. *J ECT.* 2004;20:237–241.
- Magid M, Lapid MI, Sampson SM, et al. Use of electroconvulsive therapy in a patient 10 days after myocardial infarction. *J ECT.* 2005;21:182–185.
- Gonzalez-Arriaza HL, Mueller PS, Rummans TA. Successful electroconvulsive therapy in an elderly man with severe thrombocytopenia: case report and literature review. *J ECT.* 2001;17:198–200.
- Saito N, Shioda K, Nisijima K, et al. Second case report of successful electroconvulsive therapy for a patient with schizophrenia and severe hemophilia A. *Neuropsychiatr Dis Treat.* 2014;10:865–867.
- Leonard SD. Electroconvulsive therapy and intracranial aneurysms. *Proceedings of UCLA Healthcare.* 2016;20.
- Bader GM, Silk KR, Dequardo JR, et al. Electroconvulsive therapy and intracranial aneurysm. *Convuls Ther.* 1995;11:139–143.
- Drop LJ, Viguera A, Welch CA. ECT in patients with intracranial aneurysm. *J ECT.* 2000;16:71–72.
- van Herck E, Sienaert P, Hagon A. Electroconvulsive therapy for patients with intracranial aneurysms: a case study and literature review. *Tijdschr Psychiatr.* 2009;51:43–51.
- Marks JA, Bryson EO, Adams DA, et al. The safe use of electroconvulsive therapy in a patient with a repaired arteriovenous malformation: images showing surgical clips. *J ECT.* 2016;32:3–4.
- Geduldig ET, Kellner CH. Electroconvulsive therapy in the elderly: new findings in geriatric depression. *Curr Psychiatry Rep.* 2016;18:40.
- Desseilles M, Thiry J, Monville J, et al. Electroconvulsive therapy for depression in a patient with an intracranial arachnoid cyst. *J ECT.* 2009;25:64–66.
- Wijeratne C, Shome S. Electroconvulsive therapy and subdural hemorrhage. *J ECT.* 1999;15:275–279.
- Weisberg LA, Elliott D, Mielke D. Intracerebral hemorrhage following electroconvulsive therapy. *Neurology.* 1991;41:1849.
- Kulkarni RR, Melkundi S. Subdural hematoma: an adverse event of electroconvulsive therapy—case report and literature review. *Case Rep Psychiatry.* 2012;2012:585303.
- Saha D, Bisui B, Thakurta RG, et al. Chronic subdural hematoma following electroconvulsive therapy. *Indian J Psychol Med.* 2012;34:181–183.
- Caparros-Lefebvre D, Policard J, Sengler C, et al. Bipallidial haemorrhage after ethylene glycol intoxication. *Neuroradiology.* 2005;47:105–107.
- Barceloux DG, Krenzelok EP, Olson K, et al. American Academy of Clinical Toxicology Practice Guidelines on the treatment of ethylene glycol poisoning. Ad Hoc Committee. *J Toxicol Clin Toxicol.* 1999;37:537–560.
- Battistella M. Fomepizole as an antidote for ethylene glycol poisoning. *Ann Pharmacother.* 2002;36:1085–1089.
- Baud FJ, Galliot M, Astier A, et al. Treatment of ethylene glycol poisoning with intravenous 4-methylpyrazole. *N Engl J Med.* 1988;319:97–100.
- Askar A, Al-Suwaid A. Methanol intoxication with brain hemorrhage: catastrophic outcome of late presentation. *Saudi J Kidney Dis Transpl.* 2007;18:117–122.
- Aisa TM, Ballut OM. Methanol intoxication with cerebral hemorrhage. *Neurosciences (Riyadh).* 2016;21:275–277.
- American Psychiatric Committee on Electroconvulsive Therapy. *The Practice of Electroconvulsive Therapy: Recommendations for Treatment, Training and Privileging.* 2nd ed. Washington, DC: American Psychiatric Association Publishing; 2001.
- Waite J, Easton A. *The ECT Handbook.* 3rd ed. London, UK: Royal College of Psychiatrists Publications; 2013.
- Viguera A, Rordorf G, Schouten R, et al. Intracranial haemodynamics during attenuated responses to electroconvulsive therapy in the presence of an intracerebral aneurysm. *J Neurol Neurosurg Psychiatry.* 1998;64:802–805.
- Viguera A, Welch C, Bigatello L, et al. Hemodynamic responses to electroconvulsive therapy in a hypertensive patient with end-stage pulmonary fibrosis. *Anesth Analg.* 1998;87:737–741.