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Case Report Open Access

Electroconvulsive Therapy for Depression When Substance Use Disorders are Comorbid: A Case Report and Review of the Literature

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Abstract

Substance use Disorder (SUD) is a common disorder in the United States and is frequently comorbid with depression. Electroconvulsive therapy (ECT) is the most effective treatment for depression. However, literature offers an unclear picture of whether patients with comorbid SUD are less likely to receive, or benefit from, ECT. Limited data exists regarding alcohol use disorder in ECT, while the impact of other types of SUD in ECT patients remains mostly unexplored. In this article, we review the current literature base for SUD in ECT patients and present a case where ECT was used successfully in a patient with bipolar affective disorder, current episode depressed, as well as co-morbid alcohol use disorder and opioid use disorder, in sustained remission on medication assisted therapy. Further research into ECT utilization patterns and response rates for patients with opioid use disorder is indicated.

Keywords: Medication assisted therapy; Electroconvulsive Therapy (ECT); Opioid use disorder; Substance use disorder; Bipolar disorder; Depression

Introduction

Approximately 19.7 million people in the United States suffer from substance use disorder (SUD) [1], characterized by dependence or abuse of alcohol or illicit drugs. Depression and substance use disorder co-occur frequently [2], necessitating antidepressant treatment for patients with SUD. Electroconvulsive therapy (ECT) is the most effective treatment for depression, with response rates of 60% to 90% [3]. Guidelines advise the use of ECT in treatment-refractory patients or in certain life-threatening situations (e.g. acutely suicidal or emaciated secondary to severe depression) [4]. Studies indicate that ECT is equally efficacious for bipolar and unipolar depression [5]. Research surrounding ECT use in the setting of comorbid SUD is limited and contradictory. Furthermore, despite SUD encapsulating 10 different diagnoses in DSM-5, available literature generally fails to distinguish among the different types of substance use. A history of substance use may be associated with whether a clinician initiates ECT, though the direction of that association is unclear. A large retrospective analysis of adult patients admitted to the inpatient psychiatry service found that the likelihood of both prior ECT use and planned ECT use (scheduled in the next 7 days) was significantly lower among patients diagnosed with a substance use disorder [3]. Conversely, an analysis of the 2014 Market Scan database found that patients with a mood disorder who received ECT had substantially higher rates of substance use disorder compared to those that did not receive ECT (RR 1.97) [6]. It is further noted in this study that the ECT group had substantially higher rates of comorbid alcohol use disorder (RR 2.12). Neither of these studies provide clarification regarding substance use remission status or details regarding what type of SUD occurred (other than alcohol use disorder in the latter).

How substance use may impact ECT outcomes is equivocal, and nothing is known about how co-morbid opioid use disorder, in particular, may affect response rates. One of the largest and most influential studies on ECT, the consortium for research in ECT (CORE), does not mention substance abuse in ECT5. One population-based study in Sweden using a sample of patients treated with ECT for depression found similar response rates in patients with and without SUD [7]. The authors do not comment on specific types of substances or on whether the substance use was past or current. Dating back to 1966, Roper proposes use of ECT to treat drug addiction itself [8]. He presents six patients treated with ECT for various substance use disorders, including: meperidine, morphine and barbiturates. He demonstrates mixed results in this small case series. One study of ECT outcomes in 30 psychiatric patients, including 17 patients with comorbid substance abuse, found more pronounced improvement in the suicide and depression items of the Brief Psychiatric Rating Scale (BPRS) in the group with comorbid substance abuse [9]. Conversely, another study of 261 psychiatric patients found that substance abuse correlated with unchanged or worse outcome after ECT [10]. The association between alcohol use disorder and ECT has been somewhat better studied compared to other forms of substance use. A small (n=80) retrospective chart review of patients diagnosed with mood disorder found no significant differences in ECT outcomes when alcohol abuse was comorbid [11]. In contrast, patients with both drug (cocaine, heroin or cannabis) and alcohol abuse had significantly worse ECT outcomes compared to patients without a SUD. Conversely, a study of 141 patients diagnosed with major depressive episode showed that a history of alcohol use disorder predicts improved response to ECT (OR 2.1) [12]. Overall, although the most data exists for comorbid alcohol use disorder and ECT outcomes, studies to date are conflictual. Outside of the research literature, it is notable that this particular topic was highlighted in the public domain by an influential book written by Kitty Dukakis in 2006, Shock: the Healing Power of Electroconvulsive therapy, which details her personal success with

ECT treatment for her co-morbid depression and substance use disorder.

To date, there have been no studies or case reports that specifically evaluate ECT response in the context of concurrent opioid use disorder. The topic is of particular salience as numerous large-scale studies have shown a high prevalence of depression among patients with opioid dependence [13-15], exceeding the overall prevalence rate of depression in the United States [16]. Buprenorphine, a mixed partial mu-receptor agonist and kappa-receptor antagonist, is a widely used agent for use in medication assisted treatment (MAT) to treat opioid use disorder [17]. Our case presents a patient with a history of both alcohol use disorder and opioid use disorder, with active MAT use in the form of buprenorphine-naloxone.

Case Report

The patient is a 64 year old Caucasian female with a history of bipolar affective disorder, type II who presented in a severe depressive episode. She had not experienced a hypomanic episode in several years and had never required inpatient psychiatric admission. She had a history of opioid and alcohol dependence, had undergone multiple inpatient substance abuse treatment programs, and had been treated with buprenorphine-naloxone for over 15 years. At the time of presentation, she was in sustained remission with 9 years of sobriety. Her medical history was significant for obesity, hepatitis C status-post treatment with interferon approximately 20 years prior, hypothyroidism treated with levothyroxine, and essential hypertension, well controlled with antihypertensive medication. She had had inadequate response to multiple trials of antidepressants and augmenting agents, including fluoxetine, sertraline, paroxetine, venlafaxine, duloxetine, amitriptyline, bupropion, lamotrigine, quetiapine, and aripiprazole. She decided with her outpatient team to pursue ECT as an outpatient. A standard pre-procedure medical workup was unremarkable. Throughout the course of her ECT treatment, the patient continued to take a low dose of buprenorphine-naloxone (3-0.75 mg film to 4-1 mg film daily).

ECT was performed using a MECTA device. Anaesthesia was performed with methohexital and succinylcholine. The patient received a total of 13 treatments administered over 7 weeks. All ECT therapy was performed right unilaterally and, after the third treatment, remained at the following parameters: pulse width 0.3 msec, frequency 50 Hz, duration 8 sec, current 800 mA. By the fifth treatment, the patient reported improvement in her mood. In subsequent treatments, she reported improvement to her energy, affect, and activity level. At the end of her ECT course, her depression was in remission (Quick Inventory of Depressive Symptomology score of 2). She denied any manic or hypomanic symptoms. She indicated that her decision to halt ECT treatments was due to her significant mood improvement and to anxiety related to anesthesia. The latter had emerged after she experienced an incident of accidental awareness during general anesthesia (AAGA).

Subsequent to the ECT, the patient did not suffer a relapse of either AUD or OUD. She was eventually able to taper off buprenorphine-naloxone. Buprenorphine-naloxone was later briefly reinitiated as an adjunct treatment when she had a recurrence of depression but was discontinued for lack of effectiveness. She does admit to marijuana use, roughly on a weekly to biweekly basis, which preceded the ECT. The patient did not report or demonstrate any episodes of hypomania or mania following the ECT.

Discussion

Substance use disorders and depression commonly co-occur. Despite the high prevalence of SUD in patients with depression, there is limited understanding of how SUD impacts ECT patient referrals and outcomes. The frequency of SUD comorbidity in this population is particularly important given the current opioid crisis in the United States. CDC reported that opioid overdoses accounted for the majority of overdose deaths in 2017. Furthermore, patients with alcohol or other SUD have elevated rates of suicide compared to the general population [18,19]. Given that ECT is the most effective treatment for depression and can be lifesaving in instances of acute suicidality, it is critical that we understand how various types of substance use may impact ECT outcomes.

Current literature offers limited and contradictory information on utilization and response profiles for ECT in the setting of SUD. There is some evidence that polysubstance abuse predicts worse outcomes for patients [11], but comorbid alcohol use disorder alone may have either no impact or a positive impact on ECT outcomes [11,12]. SUD in general may not impact ECT outcomes at all [7]. No clear conclusions can be drawn in this area given unclear and contradictory evidence to date. At the same time, there is no compelling evidence that suggests ECT should be avoided in SUD. Further studies would have improved clinical relevance if specific SUD are reported along with remission status of the use disorder(s). Furthermore, because substance use disorders are a diverse group of diagnoses, each with different challenges and recommended treatments, it is important for future publications in this area to clearly report the substance of abuse as well as the extent of any comorbidities.

In the case reported here, our patient responded favorably to ECT, achieving clinical remission of depression while being simultaneously treated with an opioid agonist. This provides evidence that ECT can be used safely and effectively in patients with comorbid SUD, in remission on MAT. Fully generalizable conclusions are limited by the narrow scope of this one case. Our patient was in sustained remission for both alcohol use disorder and opioid use disorder, and on partial agonist therapy. It is unclear how active illicit opioid use would have impacted her clinical response to ECT. Our patient declined future ECT partially due to mood improvement but also because of peri-procedure anxiety that developed after an incident of AGAA. Our patient's occurrence of AGAA may or may not have been related to MAT (buprenorphine-naloxone). At the same time, our case highlights the importance of monitoring for potential adverse events in ECT patients with substance use disorder. Risk factors for increased anesthetic requirements include chronic use of opioids and obesity [20], both of which may have contributed to this patient's experience of AAGA. Further studies specifically addressing the ECT response in patients with opioid use disorder and other SUDs are indicated.

References

1. Substance Abuse and Mental Health Services Administration (2018) Key substance use and mental health indicators in the United States: Results from the 2017 National Survey on Drug Use and Health.
2. Currie SR, Patten SB, Williams JV, Wang J, Beck CA, et al. (2005) Comorbidity of major depression with substance use disorders. Can J Psychiatry 50: 660-666.
3. Knight J, Jantzi M, Hirdes J, Rabinowitz T (2018) Predictors of electroconvulsive therapy use in a large inpatient psychiatry population. J ECT 34: 35-39.

4. American Psychiatric Association (2010) Practice Guideline for the treatment of patients with major depressive disorder 3rd edn.
5. Fink M (2014) What was learned: Studies by the consortium for research in ECT (CORE) 1997-2011. Acta Psychiatrica Scandinavica 129: 417-426.
6. Wilkinson ST, Agbese E, Leslie DL, Rosenheck, RA (2018) Identifying recipients of electroconvulsive therapy: Data from privately insured Americans. Psychiatr Serv 69: 542-548.
7. Nordenskjold A, von Knorring L, Engstrom I (2012) Predictors of the short-term responder rate of electroconvulsive therapy in depressive disorders--a population-based study. BMC Psychiatry 12: 115-244.
8. Roper P (1966) Drug addiction, psychotic illness and brain self-stimulation: Effective treatment and explanatory hypothesis. Can Med Assoc J 95: 1080-1086.
9. Patel M, Patel S, Hardy DW, Benzies BJ, Tare V (2006) Should electroconvulsive therapy be an early consideration for suicidal patients? J ECT 22: 113-115.
10. Small IF, Milstein V, Miller MJ, Malloy FW, Small JG (1986) Electroconvulsive treatment--indications, benefits, and limitations. Am J Psychother 40: 343-356.
11. Moss L, Vaidya N (2014) Does comorbid alcohol and substance abuse affect electroconvulsive therapy outcome in the treatment of mood disorders? J ECT 30: 22-25.
12. Aksay SS, Hambsch M, Janke C, Bumb JM, Kranaster L, et al. (2017) Alcohol use disorder as a possible predictor of electroconvulsive therapy response. J ECT 33: 117-121.
13. Brooner RK, King VL, Kidorf M, Schmidt CW Jr, Bigelow GE (1997) Psychiatric and substance use comorbidity among treatment-seeking opioid abusers. Arch Gen Psychiatry 54: 71-80.
14. Ross J, Teesson M, Darke S, Lynskey M, Ali R, et al. (2005) The characteristics of heroin users entering treatment: Findings from the australian treatment outcome study (ATOS). Drug Alcohol Rev 24: 411-418.
15. Litz M, Leslie D (2017) The impact of mental health comorbidities on adherence to buprenorphine: A claims based analysis. Am J Addict 26: 859-863.
16. Richard M. Glass (2013) Diagnostic and statistical manual of mental disorders. American Psychiatric Association.
17. Nyhuis PW, Gastpar M, Scherbaum N (2008) Opiate treatment in depression refractory to antidepressants and electroconvulsive therapy. J Clin Psychopharmacol 28: 593-595.
18. Flensborg-Madsen T, Knop J, Mortensen EL, Becker U, Sher L, et al. (2009) Alcohol use disorders increase the risk of completed suicide-irrespective of other psychiatric disorders. A longitudinal cohort study. Psychiatry Res 167: 123-130.
19. Harris EC, Baraclough B (1997) Suicide as an outcome for mental disorders: A meta-analysis. Br J Psychiatry 170: 205-228.
20. Ghoneim MM (2007) Incidence of and risk factors for awareness during anaesthesia. Best Pract Res Clin Anaesthesiol 21: 327-343.