


# Treating electroconvulsive therapy–induced mania with more electroconvulsive therapy: Evidence for electroconvulsive therapy as the ultra-mood stabilizer

SAGE Open Medical Case Reports  
Volume 6: 1–4  
© The Author(s) 2018  
Article reuse guidelines:  
sagepub.com/journals-permissions  
DOI: 10.1177/2050313X18799236  
journals.sagepub.com/home/sco



Rejish K Thomas<sup>1</sup>, Patrick J White<sup>1</sup> and Serdar Dursun<sup>2</sup>

## Abstract

Electroconvulsive therapy has been described as a mood stabilizer, as it is effective in all stages of bipolar disorder. Electroconvulsive therapy–induced mania is a known and potentially dangerous risk of treating bipolar depression with electroconvulsive therapy and there are no established guidelines for the management of electroconvulsive therapy–induced mania. We report a case of electroconvulsive therapy–induced mania where electroconvulsive therapy was continued as the sole, effective antimanic agent, which is the first described case in literature.

## Keywords

Electroconvulsive therapy, electroconvulsive therapy–induced mania, bipolar disorder, mood stabilizer

Date received: 6 February 2018; accepted: 14 August 2018

## Introduction

Electroconvulsive therapy (ECT) is the most effective treatment for treatment-resistant bipolar depression among the few options that exist.<sup>1</sup> ECT has been described as a mood stabilizer, with remission rates of 65.3% and 88.0% for bipolar depression and treatment-resistant mania, respectively.<sup>2–4</sup> A known risk of ECT treatment in a bipolar patient is ECT-induced mania, which iatrogenically places a patient in a potentially dangerous phase of their illness. ECT-induced mania has been reported to have an incidence as high as 24.8%, seen in a population of 105 inpatients with diagnoses of bipolar I or II disorder receiving ECT for depression.<sup>5</sup> Although ECT can cause mania, given the efficacy of ECT in all stages of bipolar disorder, it stands to reason that it is also a treatment to bring a patient into euthymia.

Although ECT-induced mania is well known to occur and is treated often with differing strategies, there is little evidence to guide clinicians beyond case reports and clinical experience. A case report by Lee et al.<sup>6</sup> described ECT-induced mania treated by aborting ECT and adding a mood stabilizer. In this case, a 55-year-old with major depressive disorder (MDD) and suicidality that did not respond to antidepressants received ECT. He demonstrated manic symptoms after the third ECT session that worsened over several days and therefore the decision was made to discontinue

ECT and antidepressant medications and initiated quetiapine which the patient responded to. There is also case report evidence for ceasing ECT and adding lithium or antipsychotics for ECT-induced mania.<sup>7,8</sup> Furthermore, DeQuardo and Tandon<sup>9</sup> reported a case of adding lithium and continuing four more ECT sessions with the patient reaching euthymia. The safety of concurrent use of lithium with ECT is controversial, with case reports and reviews from the 1970s and 1980s recommending against concurrent use citing excessive cognitive side effects, prolonged apnea and spontaneous seizures as serious side effects.<sup>10</sup> However, three retrospective studies with a total of 293 comparing ECT and lithium treatment to ECT alone found no significant difference in complications or recovery time.<sup>11,12</sup>

We report the case of a patient who had a second episode of ECT-induced mania where psychopharmacologic treatments failed in the past and were being refused by the patient. ECT was successfully used as the sole, effective antimanic

<sup>1</sup>Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

<sup>2</sup>Neurochemical Research Unit (NRU), Department of Psychiatry, University of Alberta, Edmonton, AB, Canada

## Corresponding Author:

Rejish Thomas, Department of Psychiatry, University of Alberta, 1E5.18 Walter Mackenzie Centre, Edmonton, AB T6G 2B7, Canada.  
Email: rejish@ualberta.ca



agent. To our knowledge, this is the first described of such cases noted in literature.

## Case presentation

A 50-year-old Caucasian male, previously high functioning, married, father presents with a 3-year history of first episode of depression. During this episode, he has never been able to achieve a euthymic state, despite three previous admissions, ranging in durations from 1 month to 6 months long. He presented at a clinic follow-up appointment 1 month after his last discharge with a severely low mood, abulia, severe initial insomnia, severe anorexia, anergia, poor concentration and severe psychomotor slowing. Mental status exam revealed a very thin and disheveled appearance, a flat affect, monotonous speech and no signs of catatonia or psychosis. In addition, his wife feared he was getting worse and he had been catatonic in the past. The decision was made to have him admitted to a psychiatric ward.

His past psychiatric history reveals a patient who has never been in contact with psychiatry or on psychiatric medications prior to 3 years ago. However, his wife reported two episodes of mania in the past 10 years, both lasting over 2 months. In these episodes, his functioning was impaired and he went into extreme debt, attempting to own and run a restaurant without previous training. In both episodes, he demonstrated no psychotic symptoms but manic symptoms such as elevated mood, pressured speech, decreased need for sleep, distractibility and increased goal-oriented activity such as hypersexuality. The patient denies that these episodes were a change from his baseline and attempted to defend his actions despite the severity of symptoms described and many family members describing his actions as not in keeping with his personality. She denied any depressive episodes in the past that impaired his functioning. Prior to this episode, he was functioning at a high level with a long-term occupation, when minor stressors financially and changes to his job led to a slow progression of decreased functionality at work that worsened to a severe depressive episode. At times during the 3-year illness, he presented with catatonic features that resolved with short-term use of benzodiazepines. In the prior admissions, he never reached full remission and was unable to return to work. Treatment was only able to reduce his depression symptoms from severe to moderate severity. In this episode, he was trialed on the following medication regimens: duloxetine 90 mg daily, mirtazapine 15 mg nightly, clonazepam 0.5 mg daily and olanzapine 2.5 mg twice a day; duloxetine 120 mg daily, aripiprazole 5 mg daily, quetiapine 300 mg nightly, dextroamphetamine 40 mg daily and bupropion extended-release 450 mg daily; venlafaxine 375 mg daily and quetiapine 300 mg nightly. There were also trials of pramipexole, lamotrigine and valproic acid. He was once trialed on ECT in his last admission. After two treatments, he exhibited manic features and ECT was held, while valproic acid was initiated. At a dose of

500 mg at night, within days, he fell back into a significant melancholic depression that proved again to be treatment-resistant to psychopharmacological strategies.

On admission in March 2017, the Maudsley Staging Method of treatment-resistant depression score for this patient was 12, which put him in the severe category with a poor prognosis.<sup>13,14</sup> The patient had a Beck Depression Inventory I (BDI-I) score of 30 (severe), a Quick Inventory of Depressive Symptomatology (QIDS-SR16) score of 20 (severe) and a Sheehan Disability Scale score of 8/10 in all domains (markedly impaired functioning).<sup>15–17</sup> Neuropsychological testing ruled out a contributing neurocognitive or personality disorder and the computed tomography head scan was reported as a normal study. Furthermore, screening laboratory testing, in particular, thyroid-stimulating hormone, vitamin B12, calcium, iron studies, complete blood count and C-reactive protein, was normal. The patient did not respond to intensive psychotherapy in addition to valproic acid 1000 mg twice a day and venlafaxine 375 mg daily.

With the patient's and patient's family's consent, bifrontal ECT was commenced on July 2017, after medication was discontinued. The ECT machine used was the MECTA spECTrum 5000Q®. The following parameters were used that were maintained for the four total treatments received which all yielded adequate seizures: pulse width = 1 ms, pulse frequency = 20 Hz, train duration = 6 s, current = 800 mA. The rationale for this dosing is based on the "half-age" stimulation and Devanand et al.'s finding that lower frequencies are more efficient.<sup>18–20</sup> After the first treatment, clinicians noticed the patient's affect was brighter. After 5 days, within hours after his third ECT treatment, he presented with a Young Mania Rating Scale (YMRS) score of 17 (moderate hypomania) demonstrating flight of ideas, pressured speech and imminent plans to build a skating rink in his backyard despite not having the means.<sup>21,22</sup> He remained consistently manic, non-psychotic and oriented for 2 days, sleeping less than 3 h per night, playing his guitar and singing loudly on the ward and drawing plans to build a skating rink in his backyard. In this state, he was assessed to have capacity by two psychiatrists and he refused antimanic medications but was willing to proceed with ECT. His reasons for his refusal were due to a stigma he attached to a psychiatric illness and avoidance of side effects, particularly feeling "blunted." Thus, he received his fourth ECT treatment. He was evaluated 6 h after the fourth ECT to have a YMRS score of 5 (remission) and a BDI-I of 5 (remission).<sup>22,23</sup> His family indicated that he remained "slightly high" but close to his baseline. This was congruent with a mental status exam revealing rapid but not pressured speech and a euphoric affect, but no psychotic signs with logical and realistic thought content. He had insight into his symptoms while manic being a large change from his depressed state and realized his plans were unrealistic. Further ECT was offered, but the patient and his wife chose to hold ECT. This was deemed acceptable as the patient showed newfound insight into

understanding the toll his illness was taking on his family and demonstrated more commitment to his treatment and even to lithium, which he refused in the past. The patient was started on monotherapy lithium carbonate which was titrated to 1200 mg daily and he was later discharged euthymic without any insomnia. He remained euthymic at his 6-month follow-up and was able to return to work.

## Discussion

There is a clinical opinion that ECT after ECT-induced mania will further exacerbate mania; however, there is a dearth of evidence to support this assertion.<sup>7,8</sup> ECT should be considered as it is well known that acute mania responds well to ECT, best seen in a prospective study by Mohan,<sup>24</sup> which found an 88% remission rate with bilateral ECT.

Antidepressant-induced mania is also a well-known phenomenon seen in 44% of 338 patients from the multi-site National Institute of Mental Health (NIMH) Systematic Treatment Enhancement Program for Bipolar Disorder (STEP-BD) study.<sup>25</sup> Although both antidepressants and ECT can induce mania, the major difference is that antidepressants have no mood stabilization properties and are not considered a treatment in mania.<sup>26</sup> Therefore, it would not be appropriate to be continued treatment with antidepressants in antidepressant-induced mania. Guidelines such as the Canadian Network for Mood and Anxiety Treatments (2013) recommend against the use of monotherapy antidepressant treatment in bipolar depression.<sup>26</sup>

Given the proximity of the mania to ECT, the switch was likely not due to the natural history of the bipolar illness. Also, given the mania was consistent until the next ECT treatment 2 days later with clear sensorium, a delirium with euphoria was unlikely.

The second edition of the American Psychiatric Association Task Force Report on Electroconvulsive Therapy (2002) states there is “no established strategy” to treat ECT-induced mania. They list options for treatment as continuing ECT, hold ECT and observing for spontaneous remission with the option of reinstating ECT or abort ECT and start a mood stabilizer.<sup>27</sup> In the case described, the other options were not chosen by the patient or not feasible. The patient had a previous episode of ECT-induced mania and the treatment strategy of ceasing ECT and starting a valproic acid returned the patient to a severely depressed state within days. The patient had also failed antipsychotics, like quetiapine, olanzapine and aripiprazole, in the past, in manic and depressive phases of illness. This left few psychopharmacologic options that could be effective in the acute setting. Furthermore, another case study described a patient whose mania was not resolved by simply ceasing ECT and the patient had a risk of escalating in his behavior.<sup>6</sup> Adding lithium during ECT may have been an option, as it has been described with a positive result in the literature; however, this was refused when offered.<sup>9</sup> The patient’s wish to avoid

potential side effects further supported the use of ECT in this scenario and future scenarios. It is left to be determined whether further ECT beyond the one received in this case would have resulted in faster, sustained euthymia, but shared decision-making by the treating team, the patient’s family and the patient led to ceasing ECT at that point.

## Conclusion

This case demonstrated one additional ECT treatment abruptly treating a manic episode caused by a short course of ECT in a patient with bipolar I disorder. This rapid response led to compliance and successful maintenance treatment with lithium monotherapy. Using further ECT to treat ECT-induced mania is a strategy that warrants further study.

## Author’s Note

This article has not been published, submitted for publication or presented elsewhere to date.

## Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

## Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

## Funding

The author(s) disclosed receipt of the following financial support for the research, authorship, and/or publication of this article: This work was funded by University of Alberta Resident Research Committee.

## Informed consent

Written informed consent was obtained from the patients for their anonymized information to be published in this article. It was obtained after the patient was assessed by two clinicians to have decisional capacity in a euthymic state, approximately 1 month post the last ECT treatment.

## References

1. Schoeyen HK, Kessler U, Andreassen OA, et al. Treatment-resistant bipolar depression: a randomized controlled trial of electroconvulsive therapy versus algorithm-based pharmacological treatment. *Am J Psychiatry* 2015; 172(1): 41–51.
2. Medda P, Toni C and Perugi G. The mood-stabilizing effects of electroconvulsive therapy. *J ECT* 2014; 30: 275–282.
3. UK ECT Review Group. Efficacy and safety of electroconvulsive therapy in depressive disorders: a systematic review and meta-analysis. *Lancet* 2003; 361(9360): 799–808.
4. Medda P, Perugi G, Zanella S, et al. Response to ECT in bipolar I, bipolar II and unipolar depression. *J Affect Disord* 2009; 118(1–3): 55–59.

5. Bost-Baxter E, Reti I and Payne J. ECT in bipolar disorder: incidence of switch from depression to hypomania or mania. *J Depress Anxiety* 2012; 1: 123.
6. Lee J, Arcand L, Narang P, et al. ECT-induced mania. *Innov Clin Neurosci* 2014; 11(11–12): 27–29.
7. Li DJ, Li HJ and Lin CH. Electroconvulsive therapy-induced manic episode for a patient with bipolar depression: a case report. *J ECT* 2015; 31(2): e30–e31.
8. Saatcioglu O and Guduk M. Electroconvulsive therapy-induced mania: a case report. *J Med Case Reports* 2009; 3: 94.
9. DeQuardo JR and Tandon R. Concurrent lithium therapy prevents ECT-induced switch to mania. *J Clin Psychiatry* 1988; 49(4): 167–168.
10. El-Mallakh RS. Complications of concurrent lithium and electroconvulsive therapy: a review of clinical material and theoretical considerations. *Biol Psychiatry* 1988; 23: 595–601.
11. Jha AK, Stein GS and Fenwick P. Negative interaction between lithium and electroconvulsive therapy—a case-control study. *Br J Psychiatry* 1996; 168: 241–243.
12. Dolenc T and Rasmussen K. The safety of electroconvulsive therapy and lithium in combination. *J ECT* 2005; 21: 165–170.
13. Fekadu A, Wooderson SC, Donaldson C, et al. A multidimensional tool to quantify treatment resistance in depression: the Maudsley Staging Method. *J Clin Psychiatry* 2009; 70(2): 177–184.
14. Fekadu A, Wooderson SC, Markopoulou K, et al. The Maudsley Staging Method for treatment-resistant depression: prediction of longer-term outcome and persistence of symptoms. *J Clin Psychiatry* 2009; 70(7): 952–957.
15. Beck AT, Ward CH, Mendelson M, et al. An inventory for measuring depression. *Arch Gen Psychiatry* 1961; 4: 561–571.
16. Rush AJ, Trivedi MH, Ibrahim HM, et al. The 16-item Quick Inventory of Depressive Symptomatology (QIDS), clinician rating (QIDS-C), and self-report (QIDS-SR): a psychometric evaluation in patients with chronic major depression. *Biol Psychiatry* 2003; 54: 573–583.
17. Sheehan DV, Harnett-Sheehan K and Raj BA. The measurement of disability. *Int Clin Psychopharmacol* 1996; 11(Suppl. 3): 89–95.
18. Fink M and Petrides G. The “half-age” stimulation strategy for ECT dosing. *Convuls Ther* 1996; 12(3): 138–146.
19. Fink M. What was learned: studies by the consortium for research in ECT (CORE) 1997–2011. *Acta Psychiatr Scand* 2014; 129(6): 417–426.
20. Devanand DP, Lisanby SH, Nobler MS, et al. The relative efficiency of altering pulse frequency or train duration when determining seizure threshold. *J ECT* 1998; 14(4): 227–235.
21. Young RC, Biggs JT, Ziegler VE, et al. A rating scale for mania: reliability, validity and sensitivity. *Br J Psychiatry* 1978; 133(5): 429–435.
22. Suppes T, McElroy SL, Altshuler LL, et al. Mixed hypomania in 908 patients with bipolar disorder evaluated prospectively in the Stanley Foundation Bipolar Treatment Network: a sex-specific phenomenon. *Arch Gen Psychiatry* 2005; 62(10): 1089–1096.
23. Reeves GM, Rohan KJ, Langenberg P, et al. Calibration of response and remission cut-points on the Beck Depression Inventory-Second Edition for monitoring seasonal affective disorder treatment outcomes. *J Affect Disord* 2012; 138(1–2): 1381–1382.
24. Mohan TS, Tharyan P, Alexander J, et al. Effects of stimulus intensity on the efficacy and safety of twice-weekly, bilateral electroconvulsive therapy (ECT) combined with antipsychotics in acute mania: a randomised controlled trial. *Bipolar Disord* 2009; 11: 126–134.
25. Truman CJ, Goldberg JF, Ghaemi SN, et al. Self-reported history of manic/hypomanic switch associated with antidepressant use: data from the systematic treatment enhancement program for bipolar disorder (STEP-BD). *J Clin Psychiatry* 2007; 68(10): 1472–1479.
26. Yatham LN, Kennedy SH, Parikh SV, et al. Canadian Network for Mood and Anxiety Treatments (CANMAT) and International Society for Bipolar Disorders (ISBD) collaborative update of CANMAT guidelines for the management of patients with bipolar disorder: update 2013. *Bipolar Disord* 2013; 15: 1–44.
27. American Psychiatric Association Task Force on Electroconvulsive Therapy. The practice of electroconvulsive therapy. Recommendations for treatment, training, and privileging: a task force report of the American Psychiatric Association, 2nd ed. *Am J Psychiatry* 2002; 159(2): 331.