Received: 16 April 2023 | Revised: 4 May 2023 | Accepted: 16 May 2023

DOI: 10.1002/ccr3.7450

CASE REPORT

Clinical Case Reports Open Access WILEY

Ventricular tachycardia triggered by electroconvulsive therapy: Case report and review of the literature

Samuel Su[1] | **Pratik Shah**[1] | **Davinder S. Jassal**[1,2,3]

[1]Section of Cardiology, Department of Internal Medicine, University of Manitoba, Winnipeg, Manitoba, Canada

[2]Institute of Cardiovascular Sciences, St. Boniface Hospital, University of Manitoba, Winnipeg, Manitoba, Canada

[3]Department of Radiology, University of Manitoba, Manitoba, Winnipeg, Canada

Correspondence
Davinder S. Jassal, Department of Medicine, Radiology, and Physiology and Pathophysiology, Rm Y3531, Bergen Cardiac Care Centre, Section of Cardiology, Department of Internal Medicine, Max Rady College of Medicine, Rady Faculty of Health Sciences, University of Manitoba, St. Boniface Hospital, 409 Tache Avenue, Winnipeg, Manitoba R2H 2A6, Canada.
Email: djassal@sbgh.mb.ca

Key Clinical Message

We report a case of a 71-year-old male with severe depression treated with electroconvulsive therapy (ECT) in the operating room complicated by monomorphic ventricular tachycardia (MMVT). The clinical presentation, treatment, and outcomes of this catecholamine-mediated cardiac event are reported with a brief review of the literature.

KEYWORDS

antiarrhythmic, cardiomyopathy, electroconvulsive therapy, ventricular tachycardia

JEL CLASSIFICATION

Cardiovascular disorders

1 | INTRODUCTION

Electroconvulsive therapy (ECT) is a widely-practiced procedure in psychiatric medicine that is indicated in various conditions including depression, bipolar disorder, and/or schizophrenia; in particular when refractory to standard pharmacotherapy.[1] ECT is done under general anesthesia and utilizes electrical currents from 25 to 1000 mC to induce generalized cerebral seizures both clinically and electrographically.[2] Serious cardiovascular complications may occur in up to 8% of patients receiving ECT, while those with pre-existing cardiac disease may have an incidence rate as high as 55%.[3] The electrical stimulus of ECT typically causes an initial parasympathetic nervous system surge, followed by a sympathetic surge during the induced seizure phase, which is then followed by a final post-ictal parasympathetic surge. While parasympathetic activation can result in bradyarrhythmias, transient asystole, and/or premature ventricular contractions, sympathetic surges during ECT have been reported to cause tachycardias, hypertension, and/or increased myocardial demand with the possibility for myocardial ischemia.[3] We describe a case of MMVT during ECT in a patient with co-existing psychiatric and structural heart disease.

2 | CASE REPORT

A 71-year-old male was voluntarily admitted to a psychiatric unit following a suicidal overdose attempt on a background history of long-standing depressive symptoms. He received a total of two treatments of ECT for his psychiatric illness. His past medical history was pertinent for heart failure with reduced ejection fraction (HFrEF) with a left ventricular ejection fraction (LVEF) of 35–40%, paroxysmal atrial fibrillation, and prior transcatheter aortic valve intervention. Due to social, financial, and psychiatric circumstances, the patient was only on Candesartan 4 mg po od for his underlying HFrEF. For treatment of his acute suicidal ideation, the patient was prescribed ECT with bitemporal electrode placement, bidirectional pulse width of 0.50 ms, 50 Hz frequency, and 0.8 Amps of current for a total of 5 s (200 mC charge delivered).

The patient's first (out of two) ECT treatments was complicated by rapid atrial fibrillation with a ventricular rate of approximately 180 bpm. He was treated with 50 mg of intravenous esmolol resulting in improved ventricular rate control of his underlying atrial fibrillation. During the second ECT treatment, 10 days later, the patient was pre-treated with 75 mg intravenous esmolol for his underlying atrial fibrillation. Seconds after the ECT stimulus, he was noted to have a short-lasting episode of a wide-complex tachycardia on intraoperative telemetry without a palpable pulse detected by the anesthesiologist (Figure 1A). The morphology was consistent with non-sustained MMVT with an electrocardiographic axis change from the patient's underlying rhythm. After approximately 12 s, the underlying rhythm self-converted to rapid atrial fibrillation (Figure 1B) which was treated with an additional 15 mg of intravenous esmolol. The patient was admitted to the coronary care unit for postoperative monitoring with initiation of intravenous amiodarone. Serial high sensitivity troponins, chest radiography, and extended electrolyte panel testing were normal. The patient was monitored on cardiac telemetry for 7 days with no recurrent episodes of MMVT and converted from atrial fibrillation to normal sinus rhythm. After consultation with the cardiac electrophysiology service, an implantable cardiac defibrillator (ICD) was not indicated due to the catecholamine-triggered nature of the event. Prior to discharge, the patient was established on goal directed medical therapy for his HFrEF including oral candesartan, metoprolol, and spironolactone.

3 | DISCUSSION

Monomorphic ventricular tachycardia, in the setting of a structurally abnormal heart, is most commonly caused by a re-entry circuit through regions of myocardial scar which are associated with altered myocyte action potentials, prolonged refractory periods, and uncouple cell to cell propagation.[4] Various triggers have been identified including hypokalemia, hypomagnesemia, and QT prolongation. Catecholaminergic VT, which is usually genetic, is a polymorphic VT known to be triggered by exercise, stress, and intense emotional states.[4] In this case report, we describe a patient with HFrEF who received ECT complicated by transient MMVT.

A literature search on MEDLINE using the combined terms of ECT and VT, revealed a total of eight relevant published case reports.[5–12] The findings of each study are summarized in Table 1. Of all of these prior case reports of

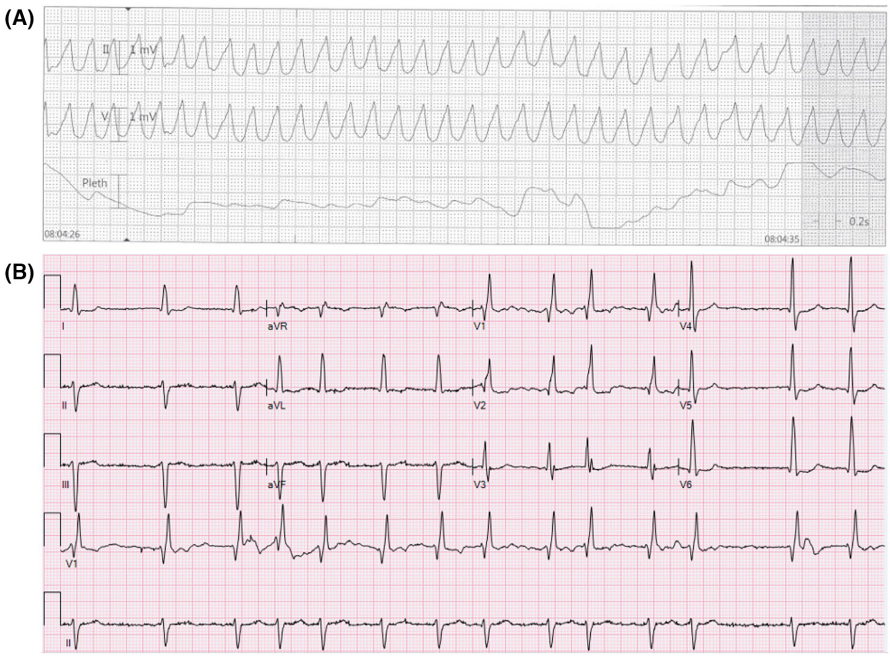

FIGURE 1 Panel (A): Non-sustained MMVT on intra-operative cardiac monitoring shortly after ECT. Panel (B): Atrial fibrillation on 12 lead ECG after episode of non-sustained MMVT was treated with intravenous esmolol and amiodarone.

TABLE 1 Summary of adult case reports of MMVT related to electroconvulsive therapy.

Report	Authors	Year	Age/sex	Pre-existing cardiac disease	LVEF	Arrythmia description	Treatment regimen	Outcome
1	Larsen et al.	1998	62/M	Atrial fibrillation (paroxysmal), "mild heart insufficiency"	Not available	Non-sustained Monomorphic VT (17 s)	None	ECT not contraindicated and safely repeated.
2	Urabe et al.	2001	33/M	Nil	Not available	Non-sustained VT Type unspecified (30 s)	None	Hyperkalemia and catecholamine mediated VT attributed to thiopental and suxamethonium. Subsequent ECT performed with alternate anesthetic and $MgSO_4$ safely
3	Kim et al.	2005	58/M	Nil	Not available	Non-sustained Monomorphic VT (>2 min runs)	Landiolol 15 mg IV	Landiolol appeared to terminate the VT
4	Bailey et al.	2006	40/M	Nil	"Normal"	Pulseless VT Type unspecified (4 min of CPR before ROSC)	None	Hyperkalemia mediated VT attributed to succinylcholine. Treated with atenolol. Succinylcholine avoided in subsequent ECT
5	Usui et al.	2008	67/M	Incomplete RBBB	"Normal"	Unspecified morphology and type	Lidocaine 100 mg IV, Defibrillation at 150 J	Esmolol pre-treatment was used to prevent VT in a subsequent ECT
6	Koga et al.	2011	64/M	Nil	Not available	Pulseless Monomorphic VT (30 s of CPR before ROSC)	None	No alternate cause found. ECT was not re-attempted
7	Pal et al.	2015	28/M	Nil	Not available	Sustained Monomorphic VT (4 min of ACLS)	Defibrillation ×2 at 200 J IV $MgSO_4$ and potassium	VT resolved with electrolyte correction
8	Grover et al.	2020	24/F	Nil	55%	Sustained monomorphic VT (4 min)	Lignocaine 90 mg IV, Defibrillation ×2 at 150 J	1st ECT-induced VT resolved with Lignocaine. 2nd ECT-induced VT required defibrillation x2. Further ECT was not re-attempted. Alternate pharmacotherapy pursued instead

VT associated with ECT, none of the patients were known to have significant underlying structural heart disease. One case report proposed a cause of ECT-induced VT to be due to electrolyte imbalances, as correction of hypokalemia and hypomagnesemia resulted in acute termination of the tachyarrythmia.[10] Additional cases describe that VT can be triggered by the enhanced catecholamine surge of ECT, resulting from hyperkalemia related to various anesthetic induction agents.[5,11] Finally, several authors highlight the possible trigger of atropine use prior to ECT, which is a common practice used to prevent post-ictal bradyarrhythmias and to reduce respiratory secretions that could lead to aspiration events.[13] In these cases, the authors propose that atropine exaggerates the sympathetic response which thereby leads to ventricular ectopy.[7–9,12] As such, the use of beta-adrenergic blockers, such as esmolol[12] or landiolol,[7] have been postulated as a means to mitigate this adverse autonomic response and thereby prevent VT.

To our knowledge, this is the first case report of a patient with pre-existing structural heart disease with an underlying substrate for scar-mediated VT with evidence of a catecholamine-induced event in the setting of ECT. During his second ECT, the patient self-converted out of the MMVT into his pre-existing atrial fibrillation before any acute intervention. His atrial fibrillation was managed appropriately with esmolol and amiodarone to achieve adequate rate and rhythm control. Similar to the previous published case reports,[6–9,12] we did not find an alternative etiology for the MMVT, and given the temporal relationship of this tachyarrythmia during the ECT treatment, and no further plans to re-attempt ECT, implantation of an ICD was not warranted.

While the optimal pre-treatment and acute management of ECT-induced VT is not well studied, prompt recognition of VT, as well as identifying patients at risk for this adverse event, is of utmost importance for ensuring patient safety during ECT. In cases where VT is found to have been triggered by ECT, close cardiac monitoring, immediate access to anti-arrythmics and defibrillation, and/or alternative psychiatric therapies should be considered.

AUTHOR CONTRIBUTIONS

Samuel Su: Conceptualization; data curation; formal analysis; investigation; methodology; project administration; resources; software; validation; visualization; writing – original draft; writing – review and editing. **Pratik Shah:** Conceptualization; data curation; formal analysis; investigation; methodology; project administration; resources; software; validation; visualization; writing – original draft; writing – review and editing. **Davinder S Jassal:** Conceptualization; data curation; formal analysis; funding acquisition; investigation; methodology; project administration; resources; software; supervision; validation; visualization; writing – original draft; writing – review and editing.

ACKNOWLEDGMENTS

There is no acknowledgement for this manuscript.

FUNDING INFORMATION

There is no funding for this manuscript.

CONFLICT OF INTEREST STATEMENT

The authors report no conflicts of interest.

DATA AVAILABILITY STATEMENT

Not applicable.

CONSENT STATEMENT

Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy.

ORCID

Davinder S. Jassal https://orcid.org/0000-0002-3639-9047

REFERENCES

1. Espinoza RT, Kellner CH. Electroconvulsive therapy. *N Engl J Med*. 2022;386(7):667-672. doi:10.1056/NEJMra2034954
2. Scott AIF. *The ECT Handbook*. 2nd ed. England; 2005.
3. Andrade C, Arumugham SS, Thirthalli J. Adverse effects of electroconvulsive therapy. *Psychiatr Clin North Am*. 2016;39(3):513-530. doi:10.1016/j.psc.2016.04.004
4. Foth C, Gangwani MK, Alvey H. Ventricular Tachycardia. StatPearls. Treasure Island (FL): StatPearls Publishing. Copyright © 2022, StatPearls Publishing LLC 2022.
5. Bailey C, Venn R, Panayiotou S, et al. Electroconvulsive therapy for catatonia resulting in cardiac arrest. *Eur J Anaesthesiol*. 2006;23(9):812-814. doi:10.1017/s0265021506241255
6. Grover S, Aggarwal S. Recurrent ventricular tachycardia during the electroconvulsive therapy procedure: a case report. *Indian J Psychiatry*. 2020;62(2):222-224. doi:10.4103/psychiatry.IndianJPsychiatry_412_19
7. Kim C, Sakamoto A, Ogawa R. Effect of landiolol on nonsustained ventricular tachycardia during electroconvulsive therapy. *Anesth Analg*. 2005;101(4):1247. doi:10.1213/01.ane.0000173762.12938.37
8. Koga Y, Mishima Y, Momozaki M, Hiraki T, Ushijima K. A case of nonsustained ventricular tachycardia immediately following modified electroconvulsive therapy in a depressive patient. *J Anesth*. 2011;25(4):595-598. doi:10.1007/s00540-011-1166-8
9. Larsen JR, Hein L, Strömgren LS. Ventricular tachycardia with ECT. *J ECT*. 1998;14(2):109-114.
10. Pal A, Samanta S, Samanta S, Wig J. Sustained ventricular tachycardia after electroconvulsive therapy: can it be prevented? *Indian J Psychol Med*. 2015;37(2):247-248. doi:10.4103/0253-7176.155663

11. Urabe K, Koguchi T, Ishikawa K, et al. A case of ventricular tachycardia immediately after electroconvulsive therapy in a schinzophrenic patient. *Masui*. 2001;50(1):50-52.
12. Usui C, Hatta K, Yokoyama T, et al. Possible effect of beta-blocker on the prevention of ventricular tachycardia during electroconvulsive therapy. *Psychiatry Clin Neurosci*. 2008;62(5):623. doi:10.1111/j.1440-1819.2008.01858.x
13. Levine J, Swartz M, Feibel H, Schreiber G. Premedication with non-selective and M1-selective muscarinic antagonists before ECT. *Isr J Psychiatry Relat Sci*. 1993;30(3):179-182.

How to cite this article: Su S, Shah P, Jassal DS. Ventricular tachycardia triggered by electroconvulsive therapy: Case report and review of the literature. *Clin Case Rep*. 2023;11:e7450. doi:10.1002/ccr3.7450