Anesthetic Management of a Patient with Congenital Heart Disease Undergoing Electroconvulsive Therapy: A Case Report

SYDNA SEBASTIAN
DNAP, CRNA
STAFF CRNA AT THE DEPARTMENT OF ANESTHESIOLOGY AND PAIN MANAGEMENT, UNIVERSITY OF TEXAS, SOUTHWESTERN MEDICAL CENTER, DALLAS, TEXAS

ACKNOWLEDGMENTS: The author would like to thank Huong Giang Brown, MD, Anesthesiologist and Chief of Clinical Operations, Neuroanesthesiology, Department of Anesthesiology and Pain Management at the University of Texas, Southwestern Medical Center, Dallas, Texas and Daiwai Olson, Ph.D., R.N. Research Nurse, Neurocritical Care Department at the University of Texas, Southwestern Medical Center, Dallas for providing guidance and support in the creation of this manuscript.

CONFLICT OF INTEREST: There is no conflict of interest.

AUTHOR STATEMENT: The author meets the authorship criteria, has read and approved this submission's final version, and agrees with the manuscript's content.

ABSTRACT

Objective: Electroconvulsive Therapy (ECT) is an effective treatment for mood disorders, particularly treatment-resistant depression and several psychiatric illnesses. The physiological effects of ECT profoundly impact the cardiovascular system, even in healthy patients. There are few case reports of successful ECT for patients with congenital heart disease. This article aims to discuss a case report of a patient with congenital heart disease who has undergone ECT treatment, focusing on anesthetic management.

Method: This article reports the anesthetic management for a patient with left-sided hypoplastic heart syndrome status post stage III Norwood procedure with major depressive disorder exacerbated by catatonia, who underwent ECT treatment.

Results: The patient received 14 acute bitemporal treatments followed by six maintenance bitemporal treatments, which significantly improved catatonia without any complications.

Conclusion: ECT was administered to a patient with congenital heart disease without any cardiovascular complications, reinforcing the safe use of ECT for patients with congenital heart disease.

KEYWORDS: Congenital heart disease, anesthetic management, electroconvulsive therapy

INTRODUCTION

ECT is a procedure in which an electric current is passed through the brain using electrodes to generate generalized cerebral seizures to manage treatment-resistant depression and several other psychiatric illnesses, including catatonia.[1] Catatonia is a psychomotor syndrome associated with several mental illnesses and some medical conditions characterized by verbigeration and waxy flexibility.[2,3] Urgent medical attention is crucial due to the lack of communication, poor oral intake, electrolyte imbalances, hypokinesis, physical deterioration, potential risk of skin ulceration, and venous thrombosis.[2,3] Benzodiazepines and ECT are the effective treatment options for catatonia.[2,3] Fontan surgery is a staged palliative procedure for a single ventricle congenital heart disease, in which the single ventricle pumps blood systemically, whereas the venous system drains passively into the pulmonary circulation.[4] ECT is known to increase the workload of the heart due to the surges in the parasympathetic and sympathetic nervous system caused by the electric stimulus, potentially detrimental to patients with preexisting heart disease.[5,6] The case report examines ECT administration and anesthetic management of a patient with congenital heart disease.

The case report examines ECT administration and anesthetic management of a patient with congenital heart disease.

CASE REPORT

A 26-year-old male with a complex congenital hypoplastic left heart status post Norwood and Glenn procedure in 1997, whose case was complicated by cardiac arrest with frontal brain anoxic injury, followed by Fontan procedure with stenting for coarctation of the aorta and pulmonary artery stent placement in 1999, presents with major depressive disorder (MDD) and catatonia in August 2022. The patient also has a history of autism and hypertension (HTN), which was treated with Lisinopril 10 mg daily. Before developing catatonia, he functioned independently with minimal assistance while living in assisted care. On admission, he was non-verbal with limited oral intake and required a feeding tube for nutrition. The patient was admitted to the inpatient psychiatric unit for further management. Despite some improvement with intravenous lorazepam, ECT was initiated to achieve complete resolution of catatonia. Consent for procedure and anesthesia was obtained by a court-appointed guardianship.

Preoperative assessments included echocardiography, electrocardiography (EKG), and consultations with cardiology and neurology. Patient was adequately optimized and cleared for ECT from a cardiac perspective. A cardiac anesthesiologist was consulted to formulate an anesthetic plan and was present for the first several treatments of ECT, given the uncertainty of the patient's hemodynamic response to ECT. Due to the concern of the dilated neoaorta and hypertension, an awake arterial line and a pacer pad were placed for the initial ECT session to monitor and treat the acute hemodynamic alteration with autonomic activation. The goal was to have tight control of blood pressure. In addition, a filter was placed on the intravenous line, and caution was taken to avoid air in the lines as the patient had a patent atrial septectomy. After adequate preoxygenation, the anesthetic induction was achieved with etomidate 10 mg and succinylcholine 60 mg. During the first session, blood pressure was controlled with a total dose of 3.75 mg of nicardipine. The airway was managed with a bag valve mask. The patient was hemodynamically stable with no significant arrhythmia or hypertension. Since the patient could tolerate the first ECT without complications, the arterial line and pacer pads were not placed for the subsequent ECTs. However, the patient required a combination of antihypertensive agents like esmolol, labetalol, nicardipine, and clevidipine (ultrashort short-acting calcium channel blocker), with etomidate increased to 14 mg during the series of treatment. The intravenous fluid was given judiciously to maintain preload. The patient tolerated the acute and maintenance phase of

ECT without any complications. The cardiac anesthesiologist was on standby for at least the first eight ECTs at the request of the family and to ensure safety. The patient successfully underwent 14 acute bitemporal treatments followed by six maintenance bitemporal treatments (09/12/22-10/14/22) with significant improvement in catatonia.

DISCUSSION

The physiological effects of the brief session (15-20 minutes) of ECT treatment are profound in the cardiovascular system.[6,7] The acute hemodynamic changes are triggered by the activation of the autonomic nervous system (ANS) by the electrical stimulus.[5-7] The initial response is a parasympathetic discharge, lasting for 10-15 seconds, resulting in bradycardia, hypotension, or even asystole, followed by an exaggerated sympathetic surge, peaking in 3-5 minutes, leading to a transient increase in systolic blood pressure (SBP) by 30% to 40% and an increase in heart rate by 20% or more.[5-7] One of the risk factors that increase the risk of bradycardia and asystole during ECT is pre existing heart disease.6 Increased tissue oxygen consumption with seizure activity can precipitate myocardial ischemia and infarction in patients with compromised hearts.[5,6]

Decreases in left ventricular systolic and diastolic function may persist for 20 minutes and up to 6 hours following ECT, even in patients without cardiac disease.[5,6]

Arrhythmia is one of the most common cardiac complications of ECT, manifested as bradycardia, premature atrial contractions, supraventricular tachycardia, premature ventricular contractions, and ventricular tachycardia or even asystole.[6] There are incidences of varying degrees of heart blocks, including Wolf-Parkinson syndrome[8] and new-onset atrial fibrillation with ECT.[9] Reversible contractile dysfunction of the myocardium, referred to as myocardial stunning, that continues after an episode of ischemia, despite coronary reperfusion, has also been reported after ECT.[6] The increase in vascular permeability caused by the catecholamine discharge from the sympathetic surge can potentially lead to cardiogenic pulmonary edema, which could threaten one with preserved heart function.[10] Takotsubo Cardiomyopathy (TCM), a reversible heart failure characterized by left ventricular hypokinesis and apical ballooning, can also be precipitated by significant emotional and physical stress related to procedures such as ECT.[10,11]

There are very few case reports of ECT for patients with congenital heart disease. Grover et al. reported successful ECT treatment for a patient with atrial septal defect.[12] Yet another case reported on ECT treatment for a patient with Marfan Syndrome by Rao et al.[13] There are no absolute contraindications for ECT, and it is considered a low-risk procedure. During ECT, pre-treatment with anticholinergics, like glycopyrrolate or atropine, to prevent bradycardia and asystole is routinely not given except for the seizure thresholding, a process of determining the lowest energy required to evoke a seizure lasting a minimum of 25 seconds on electroencephalogram (EEG)[7] as the response from the parasympathetic surge is very brief and the catecholamine release compensates it.6 The sympathetic surge resulting in hypertension and tachycardia is routinely treated with beta-blockers and antihypertensives.[7]

Having an understanding of the physiological effects of ECT helps to manage the potential complications effectively. A thorough preoperative evaluation, assessment of the functional status, labs, tests, and medical clearance are imperative for the safe administration of ECT. In addition to standard monitoring, such as EKG, pulse oximeter, end-tidal cardio dioxide (ETCO2), and blood pressure, invasive monitoring like arterial line may also be required in patients with severe cardiac comorbidities. A cardiac anesthesiologist may be a great resource to formulate anesthetic plans. Even though several potential complications are possible, adequate optimization of any preexisting conditions, judicious selection of medications, meticulous monitoring, and an expert team can ensure the safety for the patients.

CONCLUSION

ECT is the standard therapy for treatment-resistant depression and other psychiatric illnesses, including catatonia.[14] The physiologic effect of ECT is significant, and the acute hemodynamic changes can be potentially detrimental to patients with comorbidities, especially pre existing heart disease. A multidisciplinary approach with careful evaluation and monitoring with the right choices of drugs enables the safe administration of ECT for high-risk patients.

References

1. Joung KW, Park DH, Jeong CY, Yang HS. Anesthetic care for electroconvulsive therapy. *Anesth Pain Med (Seoul).* Apr 2022;17(2):145-156. doi:10.17085/apm.22145

2. Walther S, Stegmayer K, Wilson JE, Heckers S. Structure and neural mechanisms of catatonia. *Lancet Psychiatry.* Jul 2019;6(7):610-619. doi:10.1016/s2215-0366(18)30474-7

3. Gandhi K, Nguyen K, Driscoll M, Islam Z, Maru S. Catatonia, Pregnancy, and Electroconvulsive Therapy (ECT). *Case Rep Psychiatry.* 2023;2023:9601642. doi:10.1155/2023/9601642

4. Swain B, Yager A, Wanat-Hawthorne A. Perioperative Care for Electroconvulsive Therapy in an Adult Patient With Fontan Physiology. *J Cardiothorac Vasc Anesth.* Apr 2024;38(4):1011-1014. doi:10.1053/j.jvca.2024.01.008

5. Uppal V, Dourish J, Macfarlane A. Anaesthesia for electroconvulsive therapy. *Continuing Education in Anaesthesia Critical Care & Pain.* 2010;10(6):192-196. doi:10.1093/bjaceaccp/mkq039 6. Hermida AP, Mohsin M, Marques Pinheiro AP, McCord E, Lisko JC, Head LW. The Cardiovascular Side Effects of Electroconvulsive Therapy and Their Management. J ect. Mar 1 2022;38(1):2-9. doi:10.1097/yct.0000000000000802

7. Sebastian S. Anesthetic Management of Patients Undergoing Electroconvulsive Therapy. *Aana j.* Dec 2023;91(6):465-471.

8. Enomoto S, Yoshino A, Takase B, Kuwahara T, Tatsuzawa Y, Nomura S. Electroconvulsive therapy-induced Wolff-Parkinson-White syndrome: a case report. *Gen Hosp Psychiatry.* Sep-Oct 2013;35(5):575.e7-8. doi:10.1016/j.genhosppsych.2013.02.004

9. Baroni M, Albaba D, Ngo A, Livingston RK. Atrial Fibrillation Following Electroconvulsive Therapy: A Systematic Review and Case Report. *J ect.* Jun 4 2024;doi:10.1097/yct.0000000000001029

10. Bryson EO, Aloysi AS, Farber KG, Kellner CH. Individualized Anesthetic Management for Patients Undergoing Electroconvulsive Therapy: A Review of Current Practice. *Anesth Analg.* Jun 2017;124(6):1943-1956. doi:10.1213/ane.0000000000001873

11. Kinoshita M, Kinoshita M, Takahashi R, Mutoh S, Kakuta N, Tanaka K. The Safety and Strategies for Reinitiating Electroconvulsive Therapy After ECT-Induced Takotsubo Cardiomyopathy: A Case Report and Systematic Review. *J ect.* Sep 1 2023;39(3):185-192. doi:10.1097/yct.0000000000000905

12. Grover S, Agarwal M, Hegde A, Malhotra N, Somaiya M, Ghormode D. Electroconvulsive therapy in the presence of atrial septal defect: a case report. *J ect.* Jun 2012;28(2):e19-20. doi:10.1097/YCT.0b013e318249612e

13. Rao NP, Loganathan S, Prakash O, Varghese M. Use of electroconvulsive therapy for schizophrenia with comorbid Marfan syndrome. *J ect.* Dec 2009;25(4):276-7. doi:10.1097/YCT.0b013e3181a09d69

14. Deng ZD, Argyelan M, Miller J, et al. Electroconvulsive therapy, electric field, neuroplasticity, and clinical outcomes. *Mol Psychiatry.* Mar 2022;27(3):1676-1682. doi:10.1038/s41380-021-01380-y

Take the QUIZ *on the next page!*

Continuing Education Quiz

To test your knowledge on this issue's article, provide correct answers to the following questions on the form below. Follow the instructions carefully.

1. Which statement is CORRECT about the autonomic activation by electric stimulus?

A) Initial sympathetic system activation followed by parasympathetic system activation.
B) Initial parasympathetic system activation followed by sympathetic system activation.
C) The electric stimulus does not activate the autonomic nervous system.
D) No special order in the activation.

2) How long does the usual parasympathetic response last?

A) 10-15 minutes
B) 10-15 seconds
C) 3-5 minutes
D) 3-5 seconds

3) When does the sympathetic surge usually peak?

A) 2-3 minutes
B) 10 minutes
C) 3-5 minutes
D) 15 minutes

4) How does parasympathetic activation manifest?

A) Ventricular arrhythmia
B) Hypotension, Bradycardia, Asystole
C) Hypertension
D) Tachycardia

5) How does sympathetic activation manifest?

A) Hypotension
B) Bradycardia and asystole
C) Hypertension and tachycardia
D) Hypertension and bradycardia

6) Which statement is correct?

A) Seizure does not increase myocardial oxygen consumption
B) There is no systolic or diastolic dysfunction with ECT
C) Myocardial stunning is possible with ECT
D) ECT does not have any profound effect on the cardiovascular system.

7) What is one of the most common cardiac complications of ECT?

A) Changes in blood pressure
B) Arrhythmia
C) Decrease in systolic and diastolic function.
D) Increased cardiac output

8) What is the possible cause of cardiogenic pulmonary edema with ECT?

A) Decreased catecholamine release leading to increased vascular permeability
B) Increased catecholamine release leading to increased vascular permeability
C) Decreased catecholamine release leading to decreased vascular permeability
D) Increased catecholamine release leading to decreased vascular permeability

9) The response from the sympathetic response is routinely treated during ECT?

A) True
B) False

10) During seizure thresholding, a process of determining the lowest energy required to evoke a seizure lasting a minimum of 25 seconds on electroencephalogram, anticholinergics are routinely given.

A) True
B) False

Continuing Education Quiz

To apply for Continuing Education/ Contact Hours:

1) Provide all the information requested on this form.

2) Provide correct answers to this issue's quiz in the box to the right

3) Mail this form along with $10 Member or $20 Non-Member
(check or money order, payable to ASATT) to:
ASATT
6737 W Washington St, Ste 4210
Milwaukee, WI 53211

The answers to the Fall 2024 "Anesthetic Management of a Patient with Congenital Heart Disease Undergoing Electroconvulsive Therapy: A Case Report" Quiz 1 are: (circle answers)

1.	A B C D	6.	A B C D
2.	A B C D	7.	A B C D
3.	A B C D	8.	A B C D
4.	A B C D	9.	A B
5.	A B C D	10.	A B

Quiz 1

Name: ASATT

Number: Street

Address:

Phone Number:

City: State: Zip:

Signature: Date:

ACHIEVE 80% IN THIS QUIZ TO EARN ONE (1) CONTINUING EDUCATION CREDIT.