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CASE REPORT

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Extracorporeal membrane oxygenation for catecholaminergic polymorphic ventricular tachycardia: a case report and literature review

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Abstract

Background Catecholaminergic polymorphic ventricular tachycardia (CPVT) is an inherited ion channelopathy characterized by a structurally normal heart sensitive to catecholamines. It primarily presents as Bidirectional ventricular tachycardia (BiVT) and is a significant cause of sudden cardiac death in children.

Case presentation We report our experience with central Extracorporeal Membrane Oxygenation (ECMO) therapy in a 4-year-old boy with CPVT. Despite these measures, his CPVT was refractory to standard medical treatment and mechanical ventilatory support, with symptom progression. Consequently, ECMO support was initiated in addition to existing treatment. The patient was successfully weaned off ECMO on the 10th day of therapy and was discharged in a good condition. Follow-up after discharge showed favorable outcomes.

Conclusions The successful outcome in this case was attributed to the application of ECMO, which helped maintain the patient's circulatory status and address progressively worsening cardiogenic shock and uncontrolled ventricular arrhythmia. In such situations, the early use of ECMO can provide essential circulatory support and stability for patients, as demonstrated in this case.

Keywords Extracorporeal membrane oxygenation (ECMO), Catecholaminergic polymorphic ventricular tachycardia (CPVT), Hemodynamics, Pediatrics

Background

Catecholaminergic Polymorphic Ventricular Tachycardia (CPVT) is a rare but potentially fatal inherited ion channelopathy characterized by heightened sensitivity to catecholamines despite a structurally normal heart [1, 2]. The primary clinical manifestation of CPVT is Bidirectional ventricular tachycardia (BiVT), which predominantly occurs in children and adolescents, and often presents as syncope or sudden cardiac arrest. CPVT is a significant cause of sudden cardiac death in children. Due to its presentation being often triggered by emotional stress or physical exertion, and the fact that the heart appears structurally normal, diagnosing CPVT can be challenging and complex [3, 4]. Genetic testing plays a crucial role in the definitive diagnosis of CPVT, allowing for the identification of specific mutations and guiding personalized treatment.

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The main goal of managing CPVT is to prevent and control episodes of ventricular arrhythmias. Current clinical strategies primarily involve antiarrhythmic medications such as beta-blockers and the implantation of an Implantable Cardioverter Defibrillator (ICD) [5]. However, experience in managing severe CPVT during the acute phase is relatively limited, particularly in patients who do not respond adequately to conventional medications and ICD therapy. In this study, we report the case of a pediatric patient with severe CPVT who was successfully managed with Extracorporeal Membrane Oxygenation (ECMO). Through this case, we aimed to enhance the understanding of the diagnosis and management of CPVT, especially in the context of severe cases.

Case presentation

A 4-year-old male was admitted to the First Hospital of Jilin University following a 9-hour syncope episode. Nine hours prior, the patient experienced sudden abdominal pain, nausea, and vomiting, followed by syncope, loss of consciousness, and weak spontaneous respiration. His parents administered Cardiopulmonary Resuscitation (CPR), briefly restoring consciousness, though he relapsed into syncope shortly after, necessitating further CPR. Emergency services transported him to a local hospital, where resuscitative efforts, including CPR, restored cardiac rhythm with fluctuating rates between 50 and 200 bpm, alternating between sinus and ventricular rhythms. He had a history of unexplained syncope episodes at ages 3 and 4, which resolved without intervention. He had no relevant medication or family history. On admission, physical examination indicated poor general condition, mild conjunctival edema, equal and reactive pupils, a heart rate of 96 bpm with muffled sounds and irregular rhythm, and cold extremities up to the elbows and knees, with a Capillary Refill Time (CRT) of 4 s.

Upon admission, laboratory tests revealed the following: pro-BNP, 2610 pg/ml; troponin I, 5.8 ng/ml, CK-MB mass, 70.92 ng/ml, myoglobin>400 ng/ml; and lactate, 2.4 mmol/L. Infection markers, electrolytes, and immune function were all within the normal ranges. Electrocardiogram (ECG) indicated BiVT (Fig. 1). Preliminary diagnoses were ventricular arrhythmia and cardiogenic shock. The patient was treated with metoprolol and propafenone for arrhythmia, and dopamine combined with mechanical ventilation for cardiogenic shock.

Within the first 24 h of admission, the patient repeatedly experienced BiVT and ventricular fibrillation (VF) occurred twice. After defibrillation, the sinus rhythm was briefly restored but was followed by recurrent BiVT. Concurrently, the cardiogenic shock progressively worsened. Despite the administration of inotropic agents (inotropic score of 44), the blood pressure fluctuated between 83–108/53–79 mmHg. Echocardiography revealed an enlarged Left Ventricular End-Diastolic Diameter (LVEDD) of 37.5 mm (z-score: 2.5), reduced cardiac systolic function, Left Ventricular Ejection Fraction (LVEF) of 23%, and Tricuspid Annular Plane Systolic Excursion (TAPSE) of 8 mm (z-score: -0.67) (Fig. 2). Lactate levels increased to 7 mmol/L, and chest radiography indicated a cardiothoracic ratio (CTR) of 0.55. Given that the primary ventricular arrhythmia was not effectively controlled and the severity of cardiogenic shock progressively worsened, there was a therapeutic conflict, and the patient's circulatory status became difficult to maintain. Consequently, ECMO was initiated in addition to the existing treatment, with femoral artery and vein cannulation in VA-ECMO mode to support cardiac function. The flow rate was set at 80 ml/kg·min, air flow at 60 ml/kg·min, and oxygen concentration at 0.4. During ECMO support, the metoprolol dose was increased. The patient's condition gradually improved, and approximately 20 h after ECMO initiation, the cardiac rhythm transitioned from ventricular tachycardia (VT) to ventricular bigeminy. Echocardiography showed that the LVEDD remained enlarged, with LVEF ranging from 23 to 27%. By day 6 of ECMO, the cardiac rhythm had fully returned to sinus rhythm with occasional premature ventricular beats (PVBs), and the LVEDD had decreased to 36.4 mm (z-score: 2.13), with an LVEF of 51%. On day 10 of ECMO, the cardiac rhythm stabilized at sinus rhythm, echocardiography demonstrated a reduction in the LVEDD to 32.4 mm (z-score: 0.8), and the LVEF improved to 72.3%. The CTR on chest radiography decreased to 0.50, prompting discontinuation of ECMO.

During the diagnostic and treatment processes, thyroid function, immune function, tandem mass spectrometry of blood and urine, cranial MRI, and amplitude-integrated electroencephalography (aEEG) were performed, and no abnormalities were detected. Following parental consent and approval from the medical ethics committee, second-generation sequencing (SGS) was conducted. The results identified a heterozygous mutation c.491 C>A in the Ryanodine receptor 2 (RyR2), leading to a substitution of proline with histidine at amino acid position 164. Parental genetic testing confirmed that this was a de novo mutation in the patient (Fig. 3).

Oral metoprolol was administered, initially at 0.5 mg/kg/day, increased to 1.5 mg/kg/day. The patient showed no palpitations or chest tightness, with improved exercise tolerance. By day 20, Holter monitoring indicated an 85 bpm heart rate, ST-segment depression, bidirectional T wave changes, and no ventricular arrhythmias. Echocardiography showed normal left ventricular function. The patient was hospitalized for 40 days, during which no cardiac symptoms were reported in the later stages, and rehabilitation therapy became the primary focus.

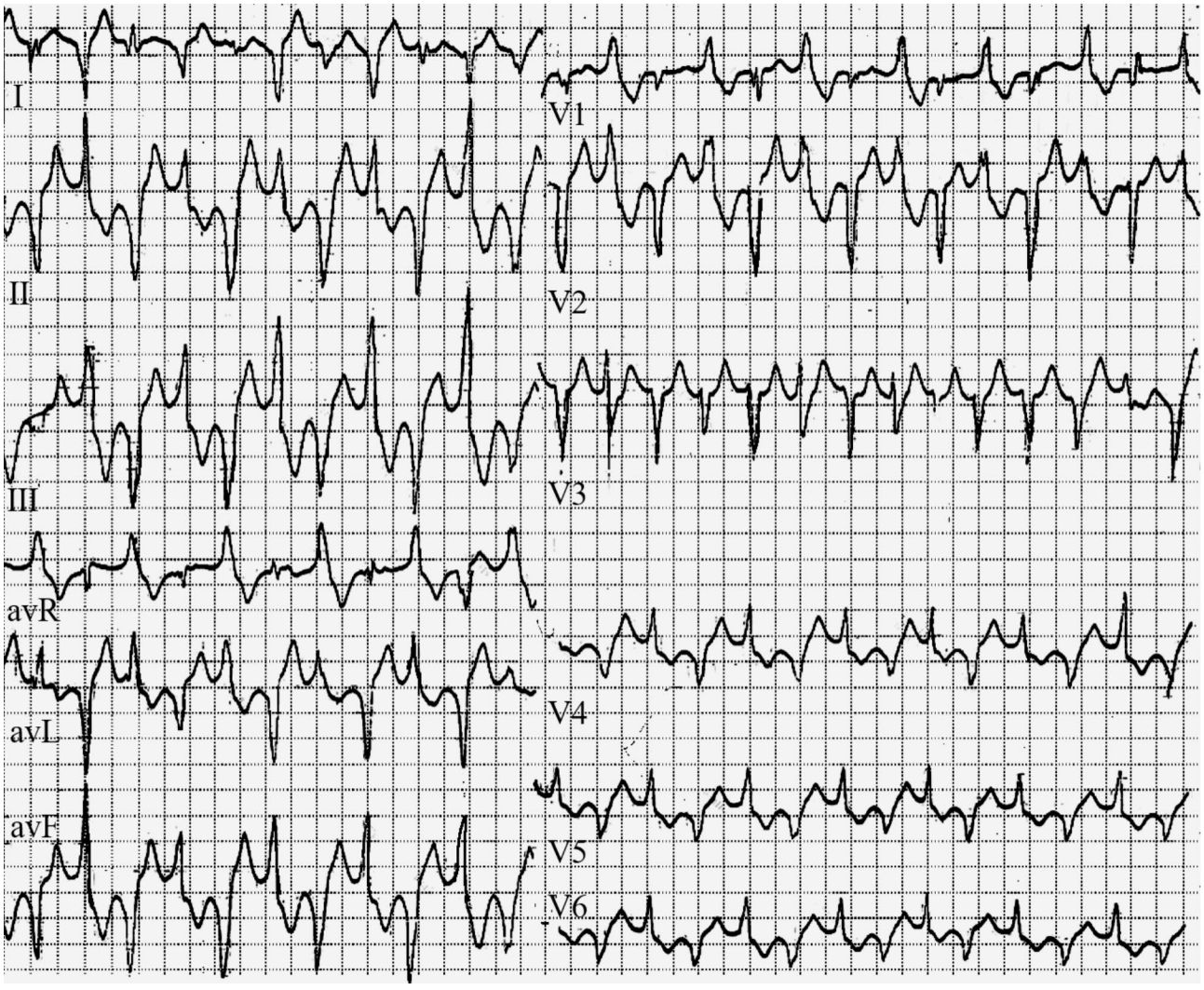

Fig. 1 The Electrocardiogram (ECG) of Bidirectional ventricular tachycardia (BiVT)

At the 1-month follow-up, a single VT episode was noted, resolving after metoprolol adjustment to 2 mg/kg/day. By 2 months, exercise tolerance improved, but Holter monitoring showed frequent polymorphic PVBs, occasional bigeminy/trigeminy, and brief VT episodes with sporadic QT prolongation, though echocardiography was normal. At the 4-month follow-up, echocardiography again showed left ventricular enlargement, prompting the placement of an ICD via the transvenous route, with continued oral administration of beta-blockers and addition of propafenone. Three months post-ICD, all findings normalized. The patient, now one year post-follow-up, remains asymptomatic with marked growth in height and weight.

Discussion and conclusions

CPVT is an inherited primary arrhythmia syndrome categorized as a channelopathy resulting from calcium ion channel dysfunction. It predominantly manifests during adolescence and is associated with high mortality rate [2]. According to the literature, CPVT accounts for approximately 10–15% of cases of sudden cardiac death in children and young adults [6–9]. This condition is primarily characterized by BiVT or polymorphic ventricular tachycardia (PMVT) induced by physical exertion or emotional stress, which can lead to syncope or even sudden death [3]. The true prevalence of CPVT remains unknown, although international studies have estimated an incidence of approximately 1 in 10,000 individuals. However, because the resting ECG and cardiac structure in patients with CPVT are often normal, and some patients present directly with unexplained sudden death, the actual prevalence may be underestimated. In terms of sex differences, the incidence rate is similar between males and females, although some studies suggest that the age of onset may be earlier in males, with a mean age of onset of 8 years [10, 11]. In this case, the patient first presented with symptoms at the age of 3 years, earlier

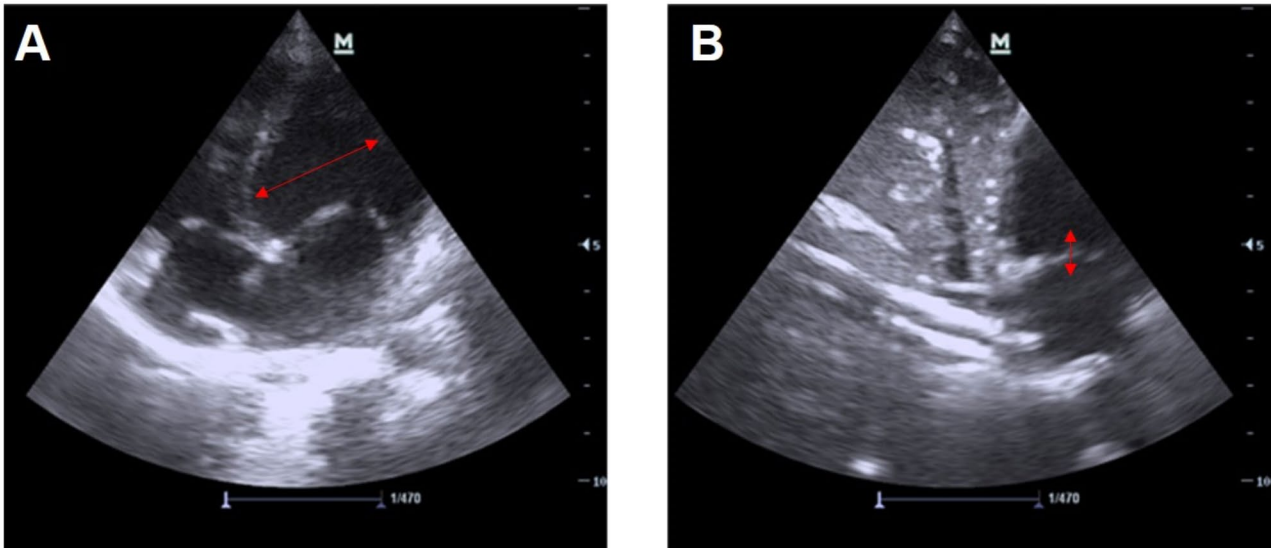

Fig. 2 The Echocardiography on the second day of hospitalization. The arrow in **A** indicates a Left Ventricular End-Diastolic Diameter (LVEDD) of 37.5 mm, while the arrow in **B** indicates a Tricuspid Annular Plane Systolic Excursion (TAPSE) of 8 mm

than the typical age of onset. Survival was dependent on ECMO support, and the early onset may be considered a contributing factor to the poorer prognosis.

CPVT primarily arises from dysfunction of the cardiac RyR2 in cardiomyocytes [12, 13]. Current research has identified that 60–65% of CPVT cases are attributed to genetic mutations that follow either autosomal dominant or recessive inheritance patterns. Based on the currently known pathogenic genes, CPVT is classified into five types: CPVT1 to CPVT5, corresponding to mutations in RyR2, CASQ2, TRDN, CALM1, and TRDN, respectively [14]. Among these, CPVT1, resulting from mutations in RyR2, is the most common, accounting for approximately 55–60% of all cases. CPVT2, associated with CASQ2 gene mutations, has an incidence rate of approximately 1–2%, whereas the incidence rates of the remaining three types are all below 1% [15, 16]. The patient in this case presented with a mutation in RyR2, corresponding to CPVT1, which follows an autosomal dominant inheritance pattern. RyR2 is located in the chromosomal region 1q42-q43 and encodes RyR2, a protein that primarily functions as a calcium release channel in the sarcoplasmic reticulum (SR) of cardiomyocytes [17]. Mutations in RyR2 result in abnormal regulation of this calcium release channel [18]. Under stress or stimulation that leads to sympathetic nervous system activation, abnormal diastolic release of calcium ions from the SR can occur, causing intracellular calcium overload [19]. This process may subsequently trigger delayed after depolarization and triggered activity, leading to VT and VF.

In CPVT patients, cardiac structure and resting ECG are generally normal, though mild sinus bradycardia may occur. Typical ECG findings during CPVT episodes include BiVT and PMVT. This patient predominantly exhibited BiVT, occasionally with VF, aligning with literature reports. However, this case showed more severe arrhythmias, significantly impacting hemodynamics. Hemodynamic instability hindered adequate myocardial perfusion, leading to ischemia and potential secondary myocardial injury. Post-episode ECG revealed VT and sinus rhythm restoration, with pathological Q waves and ST-T changes confirming acute myocardial ischemia. Literature indicates that CPVT patients generally have normal cardiac structure initially. In this case, the patient showed normal structure at onset, but persistent ventricular arrhythmias led to progressive deterioration, marked by reduced contractile function and enlargement of the left ventricle and atrium. The evolution of cardiac function and structure in this patient reflected the severity progression of the disease.

The diagnosis of CPVT is primarily based on ECG/ambulatory ECG monitoring, exercise provocation tests, and genetic testing. The detection or induction (by exercise or epinephrine challenge tests) of characteristic BiVT or PMVT is a hallmark of CPVT [3]. Exercise provocation testing is suitable for older children who are generally in good condition and are able to tolerate exercise. The typical manifestation during the exercise test is the gradual onset of premature ventricular contractions, which progressively evolve into polymorphic premature ventricular contractions or BiVT and PMVT; abnormal rhythms gradually decrease or disappear after cessation of exercise [20]. In this case, the patient was in critical condition following CPR upon admission, rendering them unsuitable for exercise provocation testing. However, typical BiVT changes have already been detected

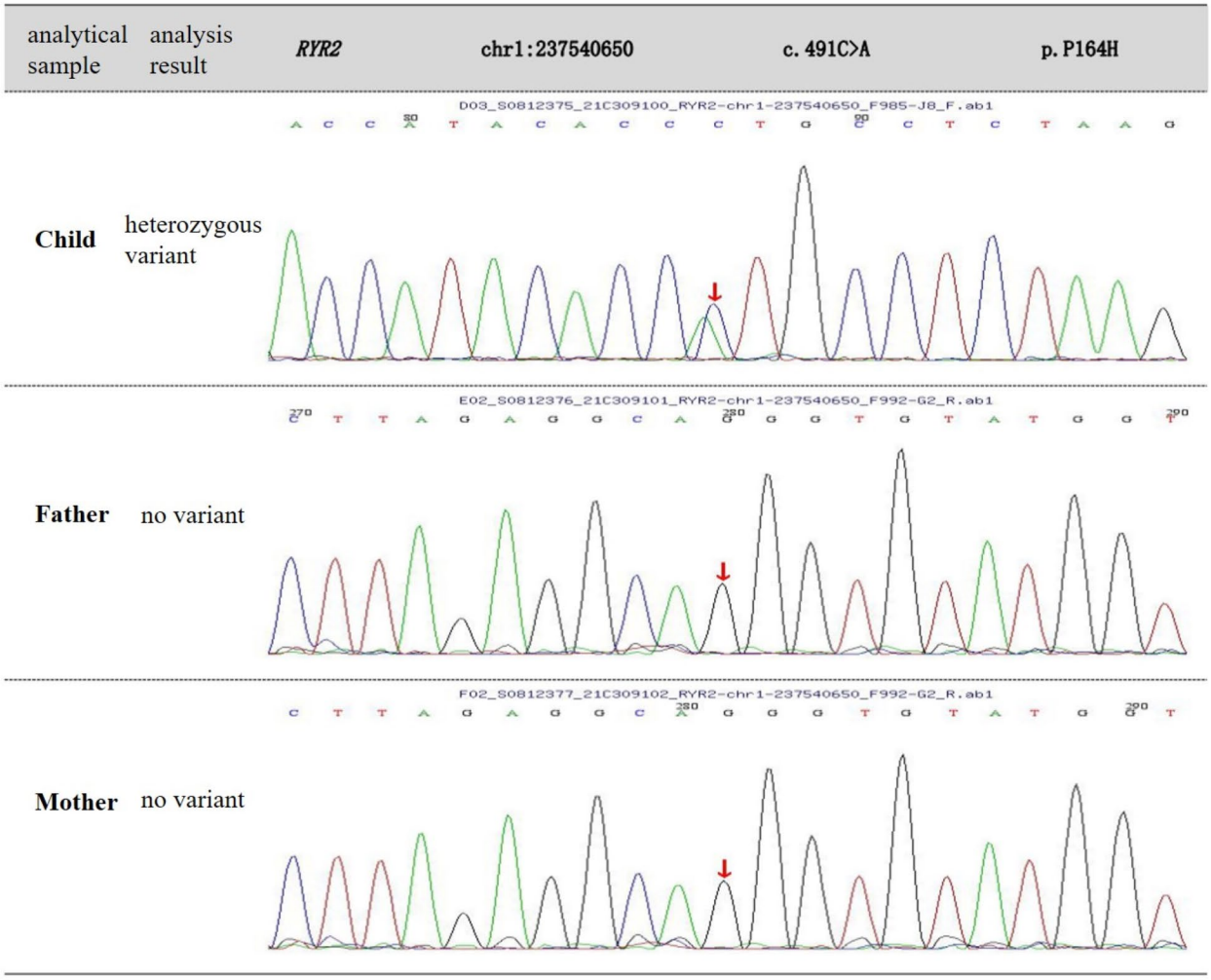

Fig. 3 Second-generation sequencing results. The arrow indicates a heterozygous mutation c.491 C > A in the Ryanodine receptor 2 (RyR2), leading to a substitution of proline with histidine at amino acid position 164

using routine ECG monitoring. Some studies suggest that adrenaline can be used as a provocative agent, but its sensitivity is significantly lower than that of exercise provocation testing, and is therefore not recommended as a primary diagnostic method [21, 22]. Genetic testing is an important adjunctive tool for diagnosing CPVT. Currently, 60–65% of patients with CPVT show positive results in genetic screening [15, 23], and genetic testing is recommended for all probands diagnosed with CPVT. Given that sudden cardiac death may be the first presentation of CPVT, genetic screening is also recommended for first-degree proband relatives.

The primary treatment goal during CPVT onset is to control ventricular arrhythmias and manage complications like cardiogenic shock and hypoxic-ischemic encephalopathy (HIE). This patient presented with severe hemodynamic instability and worsening cardiogenic shock, meeting ECMO support criteria. A search of PubMed and Web of Science databases revealed that literature on the use of ECMO for CPVT treatment is currently limited. One case report described a 9-year-old girl who experienced cardiorespiratory arrest due to CPVT-induced broad and narrow complex tachycardia. During the diagnostic and therapeutic process, the team utilized ECMO as standby support during electrophysiological studies to ensure patient safety while managing complex arrhythmias [24]. The case report emphasizes the optimization of arrhythmia control through electrophysiological studies and combination pharmacotherapy, while this case report highlights the critical role of ECMO in providing acute circulatory support for critically ill patients. Both the reported case and this study highlight the critical role of ECMO in the management of critically ill CPVT patients. This case not only provides practical insights into CPVT treatment but also offers a valuable contribution to the exploration of ECMO applications in CPVT management.

Long-term management for CPVT includes beta-blockers (propranolol, metoprolol, nadolol) to reduce malignant events and, in some cases, adjunctive calcium or sodium channel blockers. Preventive lifestyle modifications to limit sympathetic excitation, like avoiding intense exercise and stress, are essential. For refractory cases, ICD implantation or left cardiac sympathetic denervation (LCSD) may be necessary [5]. ICDs are recommended for high-risk CPVT patients, with genetic testing playing a crucial role in determining the indications for ICD implantation. For instance, patients with RyR2 gene mutations are often better candidates for ICD therapy. While ICDs terminate VF, they don't prevent BiVT or PMVT [25]. Therefore, ICDs are commonly combined with beta-blockers (e.g., propranolol, nadolol) and antiarrhythmic agents such as flecainide to reduce the occurrence of arrhythmic events. In conclusion, ICDs represent a critical therapeutic option for high-risk CPVT patients. Additionally, gene therapy for CPVT is still in the exploratory stage. Researchers are working on gene editing and intervention strategies to correct the associated genetic mutations in an effort to improve cardiac electrophysiological function. With advancements in technology and genetic research, their efficacy and indications are expected to improve, providing safer and more effective treatment strategies for CPVT management.

In conclusion, this case report highlights several key aspects that contribute to the comprehensive understanding of CPVT management. This report presents a complete set of clinical data, including dynamic changes in ECGs that clearly demonstrate the progression of the disease. The inclusion of follow-up data provided a full picture of the patient's clinical course, particularly in the context of severe CPVT complicated by cardiogenic shock. The successful use of ECMO therapy is a notable feature, showing its vital role in stabilizing hemodynamics and managing refractory ventricular arrhythmias. This case underscores the importance of early ECMO intervention in cases where conventional therapies fail, providing essential circulatory support and improving patient outcomes in life-threatening situations.

Abbreviations

aEEG	Amplitude integrated electroencephalography
BiVT	Bidirectional ventricular tachycardia
CPVT	Catecholaminergic polymorphic ventricular tachycardia
CPR	Cardiopulmonary resuscitation
CRT	Capillary refill time
CTR	Cardiothoracic ratio
ECMO	Extracorporeal membrane oxygenation
ECG	Electrocardiogram
HIE	Hypoxic-ischemic encephalopathy
ICD	Implantable cardioverter defibrillator
LVEDD	Left ventricular end-diastolic diameter
LVEF	Left ventricular ejection fraction
LCSD	Left cardiac sympathetic denervation
PVBs	Premature ventricular beats
PMVT	Polymorphic ventricular tachycardia
RyR2	Ryanodine receptor 2
SGS	Second-generation sequencing
SR	Sarcoplasmic reticulum
TAPSE	Tricuspid annular plane systolic excursion
VF	Ventricular fibrillation
VT	Ventricular tachycardia

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12887-024-05357-y.

Supplementary Material 1

Supplementary Material 2

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Not applicable.

Author contributions

Yuna Li, Yao Wu, and Yumei Li analyzed and interpreted the patient data, and are major contributors in writing the manuscript. All authors read and approved the final manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Our manuscript contains individual person's data in form of individual details and images. Written informed consent was obtained from the parents of the patient for publication of this Case report and any accompanying images. A copy of the Written consent is available for review by the Editor-in-Chief of the journal.

Competing interests

The authors declare no competing interests.

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References

1. Gerber DA, Dubin AM, Ceresnak SR, Motonaga KS, Bussineau M, Dunn K, Caleshu C, Shoemaker MB, Lubitz SA, Perez MV. Structural Abnormalities on Cardiac Magnetic Resonance Imaging in patients with Catecholaminergic polymorphic ventricular tachycardia. JACC Clin Electrophysiol. 2020;6(6):741–2.
2. Wleklinski MJ, Kannankeril PJ, Knollmann BC. Molecular and tissue mechanisms of catecholaminergic polymorphic ventricular tachycardia. J Physiol. 2020;598(14):2817–34.
3. Giudicessi JR, Ackerman MJ. Exercise testing oversights underlie missed and delayed diagnosis of catecholaminergic polymorphic ventricular tachycardia in young sudden cardiac arrest survivors. Heart Rhythm. 2019;16(8):1232–9.
4. Roston TM, Kallas D, Davies B, Franciosi S, De Souza AM, Laksman ZW, Sanatani S, Krahn AD. Burst Exercise Testing can unmask arrhythmias in patients with incompletely Penetrant Catecholaminergic polymorphic ventricular tachycardia. JACC Clin Electrophysiol. 2021;7(4):437–41.
5. Roston TM, Jones K, Hawkins NM, Bos JM, Schwartz PJ, Perry F, Ackerman MJ, Laksman ZWM, Kaul P, Lieve KVV, et al. Implantable cardioverter-defibrillator

use in catecholaminergic polymorphic ventricular tachycardia: a systematic review. Heart Rhythm. 2018;15(12):1791–9.
6. Semsarian C, Ingles J, Wilde AA. Sudden cardiac death in the young: the molecular autopsy and a practical approach to surviving relatives. Eur Heart J. 2015;36(21):1290–6.
7. Tester DJ, Ackerman MJ. Postmortem long QT syndrome genetic testing for sudden unexplained death in the young. J Am Coll Cardiol. 2007;49(2):240–6.
8. Hofman N, Tan HL, Clur SA, Alders M, van Langen IM, Wilde AA. Contribution of inherited heart disease to sudden cardiac death in childhood. Pediatrics. 2007;120(4):e967–973.
9. Tan HL, Hofman N, van Langen IM, van der Wal AC, Wilde AA. Sudden unexplained death: heritability and diagnostic yield of cardiological and genetic examination in surviving relatives. Circulation. 2005;112(2):207–13.
10. Liu N, Ruan Y, Priori SG. Catecholaminergic polymorphic ventricular tachycardia. Prog Cardiovasc Dis. 2008;51(1):23–30.
11. Priori SG, Napolitano C, Tiso N, Memmi M, Vignati G, Bloise R, Sorrentino V, Danieli GA. Mutations in the cardiac ryanodine receptor gene (hRyR2) underlie catecholaminergic polymorphic ventricular tachycardia. Circulation. 2001;103(2):196–200.
12. Yin L, Zahradnikova A Jr., Rizzetto R, Boncompagni S, Rabesahala de Meritens C, Zhang Y, Joanne P, Marques-Sule E, Aguilar-Sanchez Y, Fernandez-Tenorio M, et al. Impaired binding to Junctophilin-2 and Nanostructural Alteration in CPVT Mutation. Circ Res. 2021;129(3):e35–52.
13. Wilson AD, Hu J, Sigalas C, Venturi E, Valdivia HH, Valdivia CR, Lei M, Musgaard M, Sitsapesan R. The V2475F CPVT1 mutation yields distinct RyR2 channel populations that differ in their responses to cytosolic ca(2+) and mg(2). J Physiol. 2021;599(23):5179–201.
14. Walsh R, Adler A, Amin AS, Abiusi E, Care M, Bikker H, Amenta S, Feilotter H, Nannenberg EA, Mazzarotto F, et al. Evaluation of gene validity for CPVT and short QT syndrome in sudden arrhythmic death. Eur Heart J. 2022;43(15):1500–10.
15. Priori SG, Napolitano C, Memmi M, Colombi B, Drago F, Gasparini M, DeSimone L, Coltorti F, Bloise R, Keegan R, et al. Clinical and molecular characterization of patients with catecholaminergic polymorphic ventricular tachycardia. Circulation. 2002;106(1):69–74.
16. Katz G, Arad M, Eldar M. Catecholaminergic polymorphic ventricular tachycardia from bedside to bench and beyond. Curr Probl Cardiol. 2009;34(1):9–43.
17. Guo Y, Cao Y, Jardin BD, Zhang X, Zhou P, Guatimosim S, Lin J, Chen Z, Zhang Y, Mazumdar N, et al. Ryanodine receptor 2 (RYR2) dysfunction activates the unfolded protein response and perturbs cardiomyocyte maturation. Cardiovasc Res. 2023;119(1):221–35.
18. Lissoni A, Hulpiau P, Martins-Marques T, Wang N, Bultynck G, Schulz R, Witschas K, Girao H, De Smet M, Leybaert L. RyR2 regulates Cx43 hemichannel intracellular Ca2+-dependent activation in cardiomyocytes. Cardiovasc Res. 2021;117(1):123–36.
19. Dulhunty AF. Molecular changes in the Cardiac RyR2 with Catecholaminergic polymorphic ventricular tachycardia (CPVT). Front Physiol. 2022;13:830367.
20. Peltenburg PJ, Pultoo SNJ, Tobert KE, Bos JM, Lieve KVV, Tanck M, Clur SB, Blom NA, Ackerman MJ, Wilde AAM, et al. Repeatability of ventricular arrhythmia characteristics on the exercise-stress test in RYR2-mediated catecholaminergic polymorphic ventricular tachycardia. Europace. 2023;25(2):619–26.
21. Marjamaa A, Hiippala A, Arrhenius B, Lahtinen AM, Kontula K, Toivonen L, Happonen JM, Swan H. Intravenous epinephrine infusion test in diagnosis of catecholaminergic polymorphic ventricular tachycardia. J Cardiovasc Electrophysiol. 2012;23(2):194–9.
22. Danielsen TK, Manotheepan R, Sadredini M, Leren IS, Edwards AG, Vincent KP, Lehnart SE, Sejersted OM, Sjaastad I, Haugaa KH, et al. Arrhythmia initiation in catecholaminergic polymorphic ventricular tachycardia type 1 depends on both heart rate and sympathetic stimulation. PLoS ONE. 2018;13(11):e0207100.
23. Bai R, Napolitano C, Bloise R, Monteforte N, Priori SG. Yield of genetic screening in inherited cardiac channelopathies: how to prioritize access to genetic testing. Circ Arrhythm Electrophysiol. 2009;2(1):6–15.
24. Sadagopan SN, Yue AM. Broad and narrow complex tachycardia resulting in cardiorespiratory arrest in a child: what is the optimal treatment strategy? Eur Heart J Case Rep. 2023;7(10):ytad490.
25. Roses-Noguer F, Jarman JW, Clague JR, Till J. Outcomes of defibrillator therapy in catecholaminergic polymorphic ventricular tachycardia. Heart Rhythm. 2014;11(1):58–66.

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