ESC European Society of Cardiology

European Heart Journal - Case Reports (2023) **7**, 1–6
https://doi.org/10.1093/ehjcr/ytad064

CASE REPORT
Electrophysiology

Successful management of near-incessant bidirectional ventricular tachycardia in one-year-old child with COVID-19 infection: a case report

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Received 23 August 2022; first decision 13 September 2022; accepted 1 February 2023; online publish-ahead-of-print 4 February 2023

Background The COVID-19 is an infectious disease, caused by SARS-CoV-2 virus. Cardiovascular complications of COVID-19 are reported more often, from inflammatory cardiac diseases to acute coronary syndromes, thromboembolic events and arrhythmias. Sometimes, these arrhythmias may be life threatening and require urgent intervention.

Case summary This is a case of one-year-old boy, who was referred to our hospital because of premature ventricular complexes on ECG. The child had genetic chimerism with a karyotype of 46XY(12)/46XX(3) and small patent ductus arteriosus. We observed non-sustained episodes of bidirectional ventricular tachycardia (VT) on 24 h Holter monitor, which increased over time and caused multiple planned and urgent shocks, despite antiarrhythmic drugs and deep sedation and intubation. Patient was tested positive for COVID-19 using PCR. After thorough echocardiographic testing and a negative genetic analysis for arrhythmogenic disorders he was diagnosed with COVID-19 associated ventricular tachycardia, taking into account that he also developed multisystem inflammatory syndrome. Further, a significant decrease of ventricular activity was observed, which allowed us to implant a cardioverter-defibrillator (ICD). Soon after the implantation the storm of ventricular tachycardia restarted with multiple shocks of the device. This time left partial thoracic sympathectomy was performed and the patient didn't have ICD shocks any more.

Discussion COVID-19 infection can be associated with significant arrhythmias, including fatal ventricular arrhythmias also in children. Left partial thoracic sympathectomy can be a helpful option in patients with sustained ventricular tachycardia and multiple ICD shocks, in whom antiarrhythmic treatment or VT ablation is useless or not available.

Keywords COVID-19 infection • Ventricular tachycardia • Left partial thoracic sympathectomy • Case report

ESC Curriculum 5.6 Ventricular arrhythmia • 5.8 Cardiac ion channel dysfunction • 5.10 Implantable cardioverter-defibrillators

Learning points

- COVID-19 infection can be associated with significant arrhythmias, including fatal ventricular arrhythmias also in children.
- COVID-19 infection is associated with catecholamine burst, which can be a trigger for manifestation of bidirectional ventricular tachycardia in patients with catecholaminergic polymorphic ventricular tachycardia.
- Left partial thoracic sympathectomy can be a helpful option in patients with sustained ventricular tachycardia and multiple ICD shocks, in whom antiarrhythmic treatment or VT ablation is useless or not available.
- Persistent cough can be the single sign of rhythm disorders in particular of PVC's in children.

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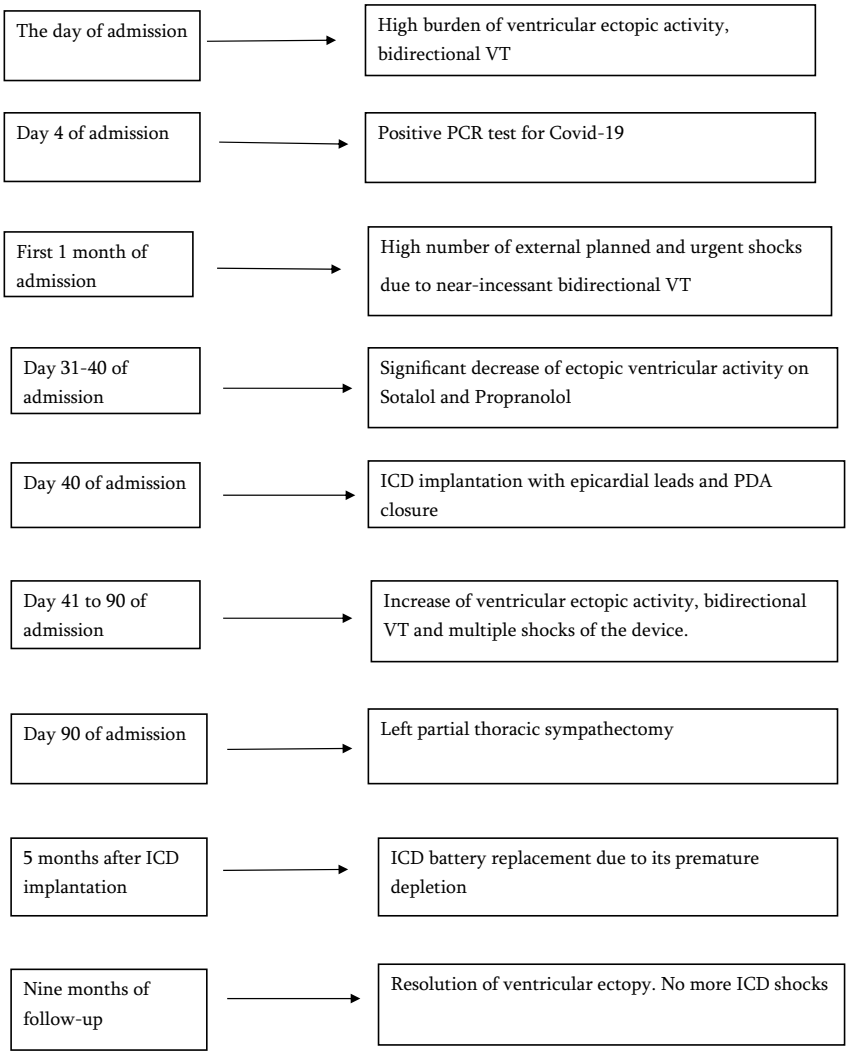
Handling Editor: Robert Schönbauer
Peer-reviewers: Peregrine Green; Ugur Canpolat
Compliance Editor: Emmanouil Mantzouranis
Supplementary Material Editor: Nikesh Jathanna

Introduction

COVID-19 is an infectious disease, caused by SARS-CoV-2 virus (severe acute respiratory syndrome coronavirus 2). It is well known, that COVID-19 significantly affects patient's cardiovascular system causing myocardial injury, myocarditis, arterial and venous thromboembolism, arrhythmias and other conditions.[1] Cardiac involvement, including ventricular dysfunction, coronary artery dilation or aneurysm, and arrhythmias is also found in pediatric patients, who suffered from COVID-19 infection.[2] Most common cardiac arrhythmias in COVID-19 patients are atrial fibrillation (AF), sinus tachycardia, sinusoidal block, sinus bradycardia and QTc prolongation, ventricular fibrillation (VF) and ventricular tachycardia (VT). Several mechanisms for arrhythmias such as cytokine or catecholamine storm, hypoxia, direct myocardial injury and electrolyte imbalance are described.[1,3] Sometimes, these arrhythmias can be life threatening and require urgent intervention.

Timeline



Case presentation

A one-year-old boy was referred to our hospital from pediatrician because of frequent premature ventricular complexes (PVCs) on ECG. The main complaint of the child was persistent cough, which was the reason for a visit to pediatrician. The baby was born at 27 weeks of pregnancy by cesarean section because of placental bleeding. At birth his weight was 1050 g and height 33 cm. The child had genetic chimerism with a karyotype of 46XY(12)/46XX(3) and bilateral groin hernia, which was operated at the age of 4 months.

The parents of the child reported also about frequent hospitalizations because of acute bronchitis and pneumonia. The child was under regular follow-up in our clinic too because of presence of small patent ductus arteriosus (PDA), which was diagnosed soon after birth.

The initial physical examination of the child revealed an arrhythmic pulse and a grade 2/6 continuous murmur in the left sternal border. Auscultation of the respiratory system revealed no abnormalities. The body temperature was 36.7°C.

An initial cardiac examination, including electrocardiogram (ECG) and echocardiogram was performed. The electrocardiogram demonstrated normal sinus rhythm with frequent polymorphic premature ventricular complexes (PVC) (*Figure 1*). The echocardiogram demonstrated a patent ductus arteriosus with restrictive flow from left to right, mild tricuspid insufficiency and moderately enlarged left ventricle with mildly decreased ejection fraction and no signs of elevated pulmonary pressure

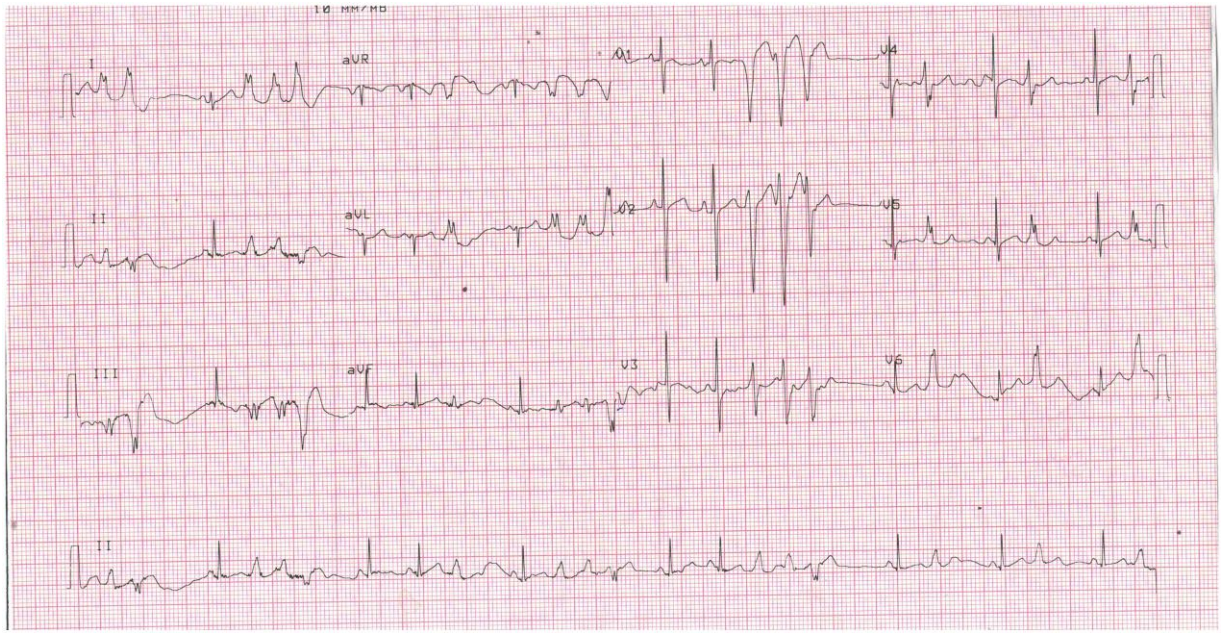


Figure 1 Twelve-lead ECG with polymorphic PVC's.

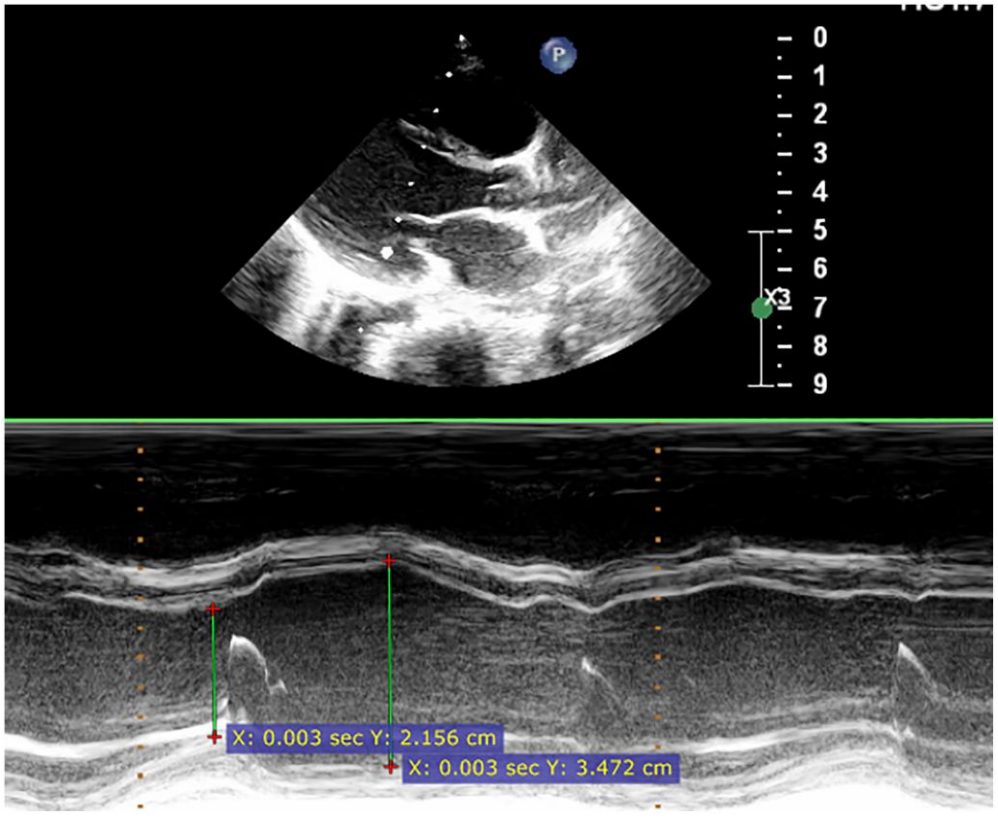


Figure 2 Echo on admission. Parasternal long axis, M Mode.

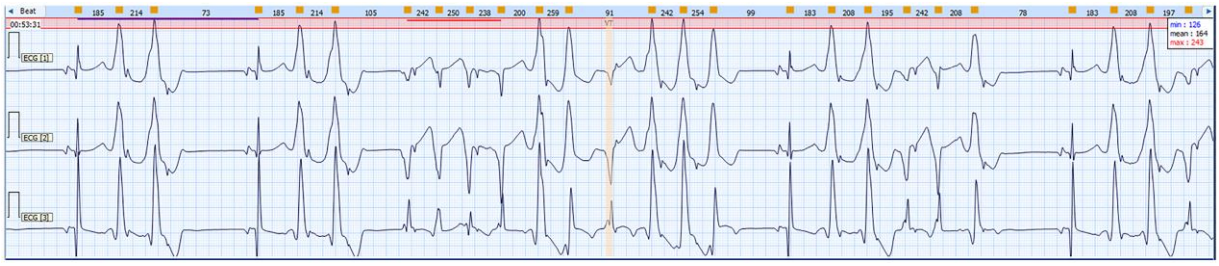


Figure 3 Bidirectional VT on holter monitoring.

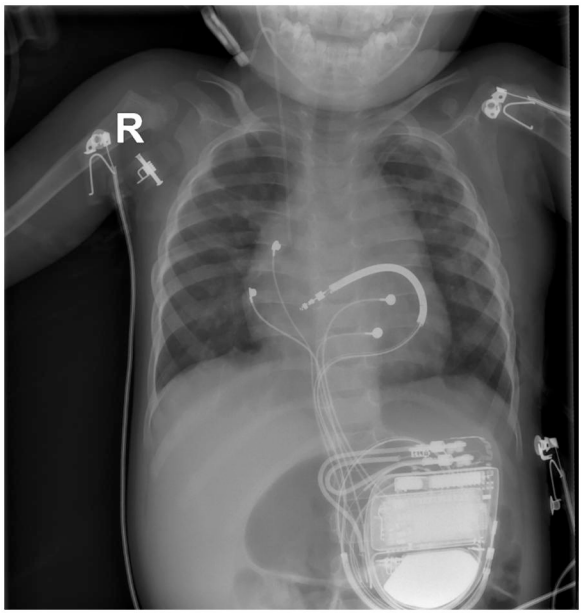


Figure 4 Epicardial ICD.

(*Figure 2*). A 24 h holter monitor was performed, which showed high burden of ventricular ectopic activity with frequent PVC's and non-sustained episodes of bidirectional ventricular tachycardia (VT) (*Figure 3*). A hospitalization and initial treatment with beta-blockers was immediately performed. Further, despite the medical treatment with antiarrhythmic drugs (Sotalol, Propafenone, Amiodarone and Verapamil) the ventricular activity continued to increase becoming sustained, near-incessant ventricular tachycardia with high number of external planned and urgent shocks. Unfortunately shocks had only little success, with short reversal to sinus rhythm. That is why deep sedation and intubation was performed to our patient. Genetic testing for genes responsible for common arrhythmogenic disorders, such as catecholaminergic polymorphic ventricular tachycardia (CPVT), arrhythmogenic right ventricular cardiomyopathy (ARVC), Brugada syndrome, long and short QT syndromes, hypertrophic and dilated cardiomyopathy and others was performed and was negative. Meanwhile a grandmother and parents of the child were diagnosed with COVID-19 infection. A PCR test for COVID-19 was positive in our patient too. Further the child developed symptoms related to COVID-19 infection, such as renal and hepatic impairment with highly elevated levels of creatinin-110 micromol/L (reference interval 9–32 micromol/L), urea-9.9 mmol/L (reference interval 1.8–6 mmol/L), aspartate aminotransferase (AST)-129 U/L (reference interval 8–60 U/L) and alanine aminotransferase (ALT)-71 U/L (reference interval 5–30 U/L). Also severe leucocytosis (WBC-48*10^9/L) with lymphopenia (LYM-17%), elevated levels of inflammatory markers such as C reactive protein (CRP)- 42.4 mg/L (reference interval 0–8.2 mg/L) and procalcitonin-0,8 ng/mL (reference interval < 0,1 ng/mL) occured. The repeated advanced echocardiogram showed also an enlargement of posterior descending coronary arteria (d = 2.4 mm). So, the patient met the criterien of multisystem inflammatory syndrome in children. Treatment with Infliximab was performed, which slowly led to normalization of renal and hepatic function. Inflammatory markers were normalized too. Unfortunately ventricular ectopic activity continued to occur.

The patient was diagnosed with COVID-19 associated ventricular near-incessant tachycardia and multisystem inflammatory syndrome in children. Differential diagnosis from catecholaminergic polymorphic ventricular tachycardia, which was the initial diagnosis, remains the main challenge. Considering COVID-19 infection as a serious stressor and given the predominantly bidirectional appearance of ventricular tachycardia, the presence of CPVT is highly suspected, even in the absence of mutations responsible for CPVT.

During hospitalization due to the further treatment with sotalol and propranolol, a significant decrease of ventricular ectopic activity was observed. Even though, one or two unplanned shock episodes were required because of arrhythmia relapse. In this relatively stable period, the patient underwent implantation of cardioverter-defibrillator (ICD) and PDA closure (*Figure 4*).

Ventricular and atrial epicardial electrodes were placed through a medial sternotomy access. A pocket for the ICD device was created subcutaneously on the anterior abdominal wall, on the left side. Unfortunately, soon after the implantation the storm of ventricular tachycardia restarted and caused multiple shocks of the device, despite treatment with Flecainide and Propranolol. This time left partial thoracic sympathectomy was considered and performed in the operating room by an experienced cardiac surgeon. Transection and removal of the paravertebral sympathetic chain was performed through a left posterolateral thoracotomy. The procedure went well without further side effects such as Horner syndrome. After 3 months, a planned replacement of ICD battery was performed due to its premature depletion.

After the left partial sympathectomy during 9 months of follow-up, the patient did not have ICD shocks any more. The last 24 h holter monitor showed preserved sinus function (daily mean frequency of HR—96 bpm) with complete resolution of ventricular activity (*Figure 5*). The child takes no more antiarrhythmic drugs and is under close follow-up in our clinic. The initially increased left ventricular ejection fraction was fully recovered by the last echocardiogram (*Figure 6*).

Discussion

COVID-19 infection can be associated with significant arrhythmias, such as atrial fibrillation, atrial flutter (AF), sinus tachycardia, sinusoidal block, sinus bradycardia and QTc prolongation, ventricular fibrillation

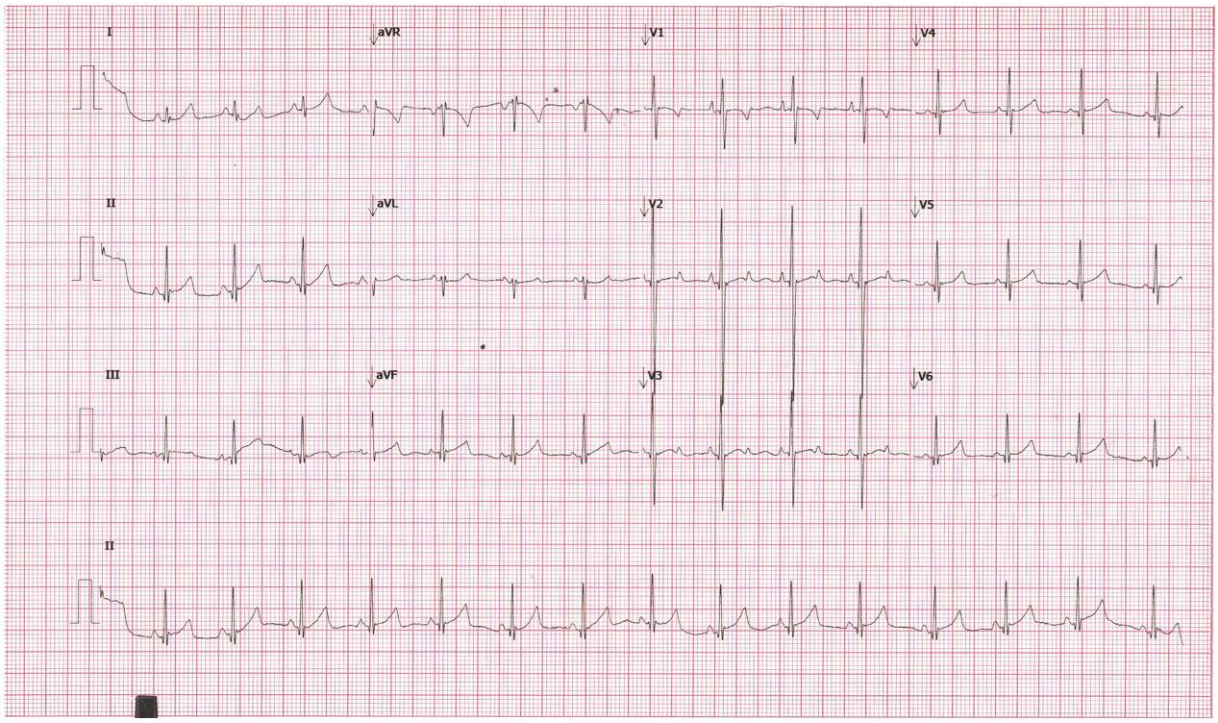


Figure 5 Sinus rhythm after sympathectomy.

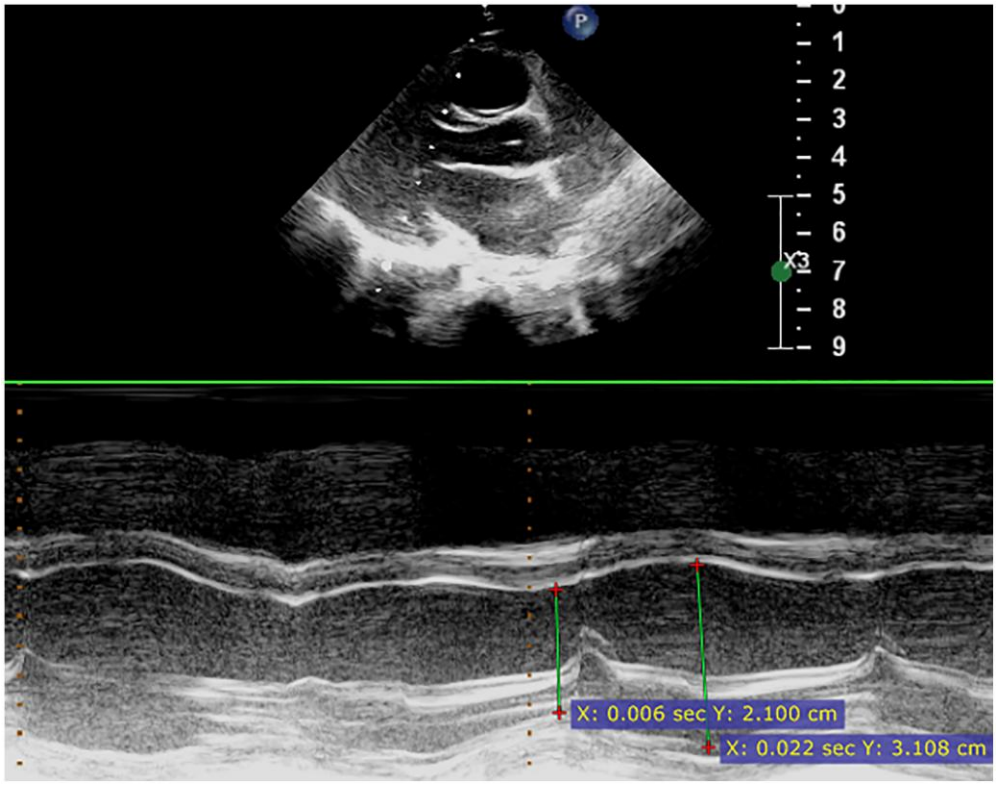


Figure 6 Echo on discharge. Parasternal long axis, M Mode.

(VF), ventricular tachycardia (VT) and others.[1,2,3] However, till now no case report about COVID-19 associated bidirectional VT is available in literature. The inflammatory cytokines can cause arrhythmias, by modifiing the function and expression of cardiac calcium and potassium channels, so affecting the cardiomyocyte action potential.[4] Acute hypoxia in COVID-19 can be another significant reason for cardiac arrhythmias.[5] Myocardial injury due to inflammation ot thrombosis observed in COVID-19 patients, also increase the risk for arrhythmias.[6] Fluid imbalance and electrolyte abnormalities can cause new arrhythmias or exacerbate preexisting conduction disease in COVID-19 patients.[7]

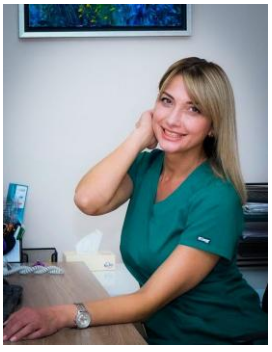
It is established, that COVID-19 infection is also associated with catecholamine burst. Sympathetic overstimulation and storm occurs due to different factors such as emotional stress, hypoxia, immunological and pro-inflammatory factors and other.[8] This catecholamine burst can be a trigger for bidirectional ventricular tachycardia in patients with catecholaminergic polymorphic ventricular tachycardia and a possible explanation for bidirectional VT in our patient. However, absence of mutations responsible for CPVT casts doubt for the diagnosis of CPVT in our case. On the other hand, taking into account the absence of case reports about COVID-19 associated bidirectional VT and the clinical sensitivity of genetic testing for CPVT only 70–80%, the diagnosis of CPVT is still highly suspected.[9]

Left partial thoracic sympathectomy can be a helpful option in patients with sustained ventricular tachycardia and multiple ICD shocks, in whom antiarrhythmic treatment or VT ablation is useless or not available.[10]

The sympathetic overstimulation is well known for increasing the risk for arrhythmias.[11] Predictably, pharmacological or surgical blockade of the sympathetic nervous system should decrease the burden of ventricular arrhythmia events. Cardiac sympathectomy as a treatment option for medically refractory ventricular fibrillation/tachycardia storm, catecholaminergic polymorphic VT and long QT syndrome is recommended in the 2017 American Heart Association (AHA), American College of Cardiology (ACC) and Heart Rhythm Society (HRS) guideline for the management of ventricular arrhythmias.[12]

In some cases the result of sympathectomy is a reduction of the burden of ventricular activity, while in others only slowing down of the rapidity of ventricular tachycardia prevents from multiple shocks. In our case it resulted in complete resolution of ventricular activity.

Lead author biography



My name is Nare Ghazaryan. I am 32 years old. In 2014, I graduated with honors from Yerevan State Medical University and continued my studies in clinical residency in Cardiology. Within the DAAD scholarship, I spent 6 months of my residency in the Department of Cardiology at the University Clinic of Heidelberg. Immediately after completing my residency in 2017, I left for Germany and started to work in the Department of Internal Medicine at Gross-Umstadt Clinic. However, later I decided to return to Armenia to invest my knowledge and potential in my homeland. In 2019, I completed 1 year of Fellowship in Clinical Arrhythmology in Nork-Marash Medical Center and since then I am working as a young cardiologist in the same center.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

Acknowledgments

We would like to express our sincere gratitude to all doctors, who were involved in this case, in particular to cardiac surgeon Hagop Hovaguimian and Gagik Heboyan, to invasive electrophysiologist Armen Khachatryan, to functional diagnostic specialist Tatevik Gabrielyan. We are also very thankful to all other medical stuff for their mindful work and dedication.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Patient consent statement: The patient's parients were informed and have signed the patient consent form, according to COPE guidelines.

Conflict of interests: The authors declare no conflict of interest.

Funding: This report received no external funding.

References

1. Chang WT, Toh HS, Liao CT, Yu WL. Cardiac involvement of COVID-19: a comprehensive review. *Am J Med Sci* 2021;**361**:14–22.
2. Rodriguez-Gonzalez M, Castellano-Martinez A, Cascales-Poyatos HM, Perez-Reviriego AA. Cardiovascular impact of COVID-19 with a focus on children: a systematic review. *World J Clin cases* 2020;**8**:5250.
3. Bhatla A, Mayer MM, Adusumalli S, Hyman MC, Oh E, Tierney A, *et al.* COVID-19 and cardiac arrhythmias. *Hear Rhythm* 2020;**17**:1439–1444.
4. Lazzerini PE, Laghi-Pasini F, Boutjdir M, Capecchi PL. Cardioimmunology of arrhythmias: the role of autoimmune and inflammatory cardiac channelopathies. *Nat Rev Immunol* 2019;**19**:63–64.
5. Clark RE, Christlieb I, Sanmarco M, Diaz-Perez R, Francis Dammannjr J, Zipser ME. Relationship of hypoxia to arrhythmia and cardiac conduction hemorrhage: an experimental study. *Circulation* 1963;**27**:742–747.
6. Babapoor-Farrokhran S, Gill D, Walker J, Rasekhi RT, Bozorgnia B, Amanullah A. Myocardial injury and COVID-19: possible mechanisms. *Life Sci* 2020;**253**:117723.
7. Pandat S, Zhu Z, Fuentes-Rojas S, Schurmann P. Arrhythmias in COVID-19. *Methodist Debakey Cardiovasc J* 2021;**17**:73–82.
8. Porzionato A, Emmi A, Barbon S, Boscolo-Berto R, Stecco C, Stocco E, *et al.* Sympathetic activation: a potential link between comorbidities and COVID-19. *FEBS J* 2020;**287**:3681–3688.
9. Napolitano C, Bloise R, Memmi M, Priori SG. Clinical utility gene card for: catecholaminergic polymorphic ventricular tachycardia (CPVT). *Eur J Hum Genet* 2014;**22**:152.
10. Chihara RK, Chan EY, Meisenbach LM, Kim MP. Surgical cardiac sympathetic denervation for ventricular arrhythmias: a systematic review. *Methodist Debakey Cardiovasc J* 2021;**17**:24.
11. Shen MJ, Zipes DP. Role of the autonomic nervous system in modulating cardiac arrhythmias. *Circ Res* 2014;**114**:1004–1021.
12. Al-Khatib SM, Stevenson WG, Ackerman MJ, Bryant WJ, Callans DJ, Curtis AB, *et al.* 2017 AHA/ACC/HRS guideline for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: a report of the American college of cardiology/American heart association task force on clinical practice guidelines and the heart rhythm society. *J Am Coll Cardiol* 2018;**72**:e91–220.