



Recurrent ventricular tachycardia in a patient with COVID-19 vaccine-associated myocarditis: a case report

Amole Ojo^{1^}, Ilan Goldenberg¹, Valentina Kutyla¹, Mehmet K. Aktas¹, Spencer Rosero¹, Hakeem Ayinde², David T. Huang¹

¹Clinical Cardiovascular Research Center, University of Rochester Medical Center, NY, USA; ²Division of Cardiology, Mary Washington Hospital, Fredericksburg, VA, USA

Contributions: (I) Conception and design: A Ojo; (II) Administrative support: I Goldenberg; (III) Provision of study materials or patients: A Ojo; (IV) Collection and assembly of data: A Ojo; (V) Data analysis and interpretation: All authors; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: Amole Ojo, MD. Clinical Cardiovascular Research Center, University of Rochester Medical Center, 265 Crittenden Blvd., Box 653, Rochester, NY 14642, USA. Email: amole_ojo@urmc.rochester.edu.

Background: The development of coronavirus disease 2019 (COVID-19) vaccine-associated myocarditis has been reported. Most of the reported cases are mild, with quick clinical recovery and excellent short-term outcomes. Cases of COVID-19 vaccine-associated myocarditis presenting with sustained ventricular tachycardia (VT) are rare.

Case Description: A 46-year-old male patient with no prior cardiac history presented following two episodes of syncope. Two days earlier, he had received his second dose of COVID-19 mRNA vaccine (Pfizer)—first dose was administered three weeks earlier. He had an episode of VT while in the emergency room. His cardiac magnetic resonance imaging (MRI) findings were consistent with myocarditis. He was eventually diagnosed with COVID-19 vaccine-associated myocarditis after all other work up were unremarkable [echocardiogram, coronary angiogram, diagnostic electrophysiology study and later ¹⁸F-fluorodeoxyglucose (FDG) metabolism cardiac sarcoid positron emission tomography (PET) study]. An implantable cardiac monitor was implanted to monitor for recurrence of VT. Seven months after initial presentation, he had recurrent VT and he underwent implantation of an implantable cardioverter defibrillator (ICD). He has received appropriate ICD therapies on account of recurrent VT and he is currently maintained on an antiarrhythmic medication.

Conclusions: Excellent short-term outcomes have been reported in patients with COVID-19 vaccine associated myocarditis. Our case shows that long-term outcomes may not be benign in everyone, particularly in those who develop myocardial scar.

Keywords: Case report; COVID-19 vaccine-associated myocarditis; recurrent ventricular tachycardia (VT); wide complex tachycardia; syncope

Submitted Aug 23, 2022. Accepted for publication Dec 03, 2022. Published online Feb 20, 2023.

doi: 10.21037/atm-22-4164

View this article at: <https://dx.doi.org/10.21037/atm-22-4164>

Introduction

The development of coronavirus disease 2019 (COVID-19) vaccine-associated myocarditis has been reported (1-3).

The presenting symptom in many of these cases is chest pain. Other less frequent presenting symptoms or signs include dyspnea, palpitations, fever and pericardial

[^] ORCID: 0000-0002-6082-535X.

effusion (1). Most of the reported cases are mild, with quick clinical recovery and excellent short-term outcomes (4). We present a case of recurrent ventricular tachycardia (VT) in a patient who developed myocarditis following administration of a COVID-19 vaccine. We present the following case in accordance with the CARE reporting checklist (available at <https://atm.amegroups.com/article/view/10.21037/atm-22-4164/rc>).

Case presentation

A 46-year-old male patient with a history of attention-deficit/hyperactivity disorder (managed with amphetamine-dextroamphetamine) presented to our hospital following two episodes of syncope. His other symptoms included palpitations, lightheadedness, nausea and vomiting. Two days earlier, he had received his second dose of COVID-19 mRNA vaccine (Pfizer)—first dose was administered three weeks earlier. He has no family history of sudden cardiac death. His physical exam revealed tachycardia with a rate of 108 beats/min, otherwise normal vital signs, clear lungs and no murmurs. While in the emergency room, he became unresponsive and telemetry showed wide complex tachycardia (*Figure 1*). The wide

complex tachycardia terminated spontaneously and he regained consciousness within a minute. At this time, the underlying rhythm was noted to be atrial fibrillation and an electrocardiogram (ECG) was done (*Figure 2*). Patient later converted back to sinus rhythm. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

High sensitivity troponin T was mildly elevated at 25 ng/L (normal 0–21 ng/L). C reactive protein was elevated at 33 mg/L (normal 0–10 mg/L). Erythrocyte sedimentation rate was elevated at 23 mm/hr (normal 1–15 mm/hr). His COVID-19 polymerase chain reaction was negative. Transthoracic echocardiogram showed normal left ventricular ejection fraction without regional wall motion abnormalities; normal right heart size and function with indeterminate pulmonary artery systolic pressure. There were no significant valvular abnormalities. Coronary angiogram showed angiographically normal coronary arteries. He underwent a diagnostic electrophysiology study to rule out a pre-excited tachycardia and supraventricular tachycardia with aberrancy. He had normal antegrade atrioventricular nodal conduction and concentric decremental retrograde ventriculo-atrial conduction. There was no inducible supraventricular tachycardia. There was no evidence of any accessory pathway. Cardiac magnetic resonance imaging (MRI) subsequently showed linear epicardial delayed gadolinium enhancement in the basal anterior lateral and basal inferior lateral segments of the left ventricle suggestive of focal myocarditis. There was no overlying pericardial enhancement or pericardial effusion to suggest pericarditis (*Figure 3*).

Based on the above diagnostic testing, we concluded that patient's episodes of syncope were due to wide complex tachycardia consistent with VT in the setting of COVID-19 vaccine-associated myocarditis. While there is a possibility that the myocarditis was due to a virus we did not test for or idiopathic and the timing of vaccination was just a coincidence, our case is not the first of such cases to be reported (1–3,5,6). He was discharged on metoprolol and amiodarone (for 3 months) with plan to repeat a cardiac MRI in 3 months for follow up of the delayed gadolinium enhancement. He was also discharged with a wearable

Highlight box

Key findings

- This case shows that patients with COVID-19 vaccine associated myocarditis can present with sustained ventricular tachycardia (VT).

What is known and what is new?

- Cases of COVID-19 vaccine associated myocarditis have been reported. The presenting symptom in many of these cases is chest pain. Other less frequent presenting symptoms or signs include dyspnea, palpitations, fever and pericardial effusion. Presentation with sustained VT is rare. Most of the cases have shown excellent short-term outcomes.
- Our patient presented with sustained VT and he had recurrence of VT, requiring implantation of an implantable cardioverter-defibrillator (ICD).

What is the implication, and what should change now?

- This case demonstrates that the long-term outcome of COVID-19 vaccine-associated myocarditis may not be benign, particularly in those who develop myocardial scar.
- Hence, such patients should be monitored closely for development of ventricular arrhythmias and ICD implantation should be considered if they develop VT.

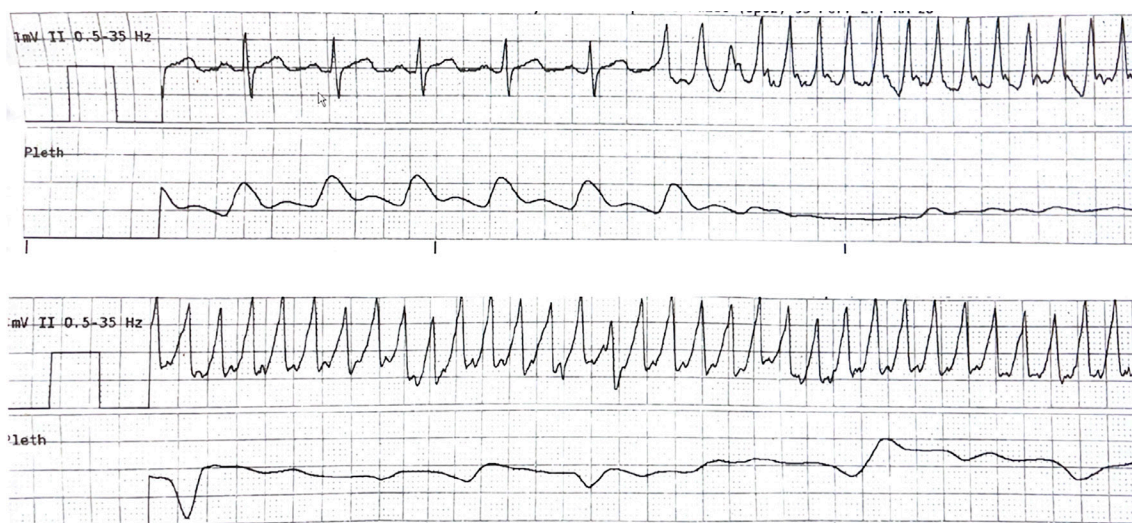


Figure 1 Telemetry strips. The rhythm strips show sinus rhythm at a HR of 98 bpm followed by sudden onset of wide complex tachycardia, HR 274 bpm. HR, heart rate.

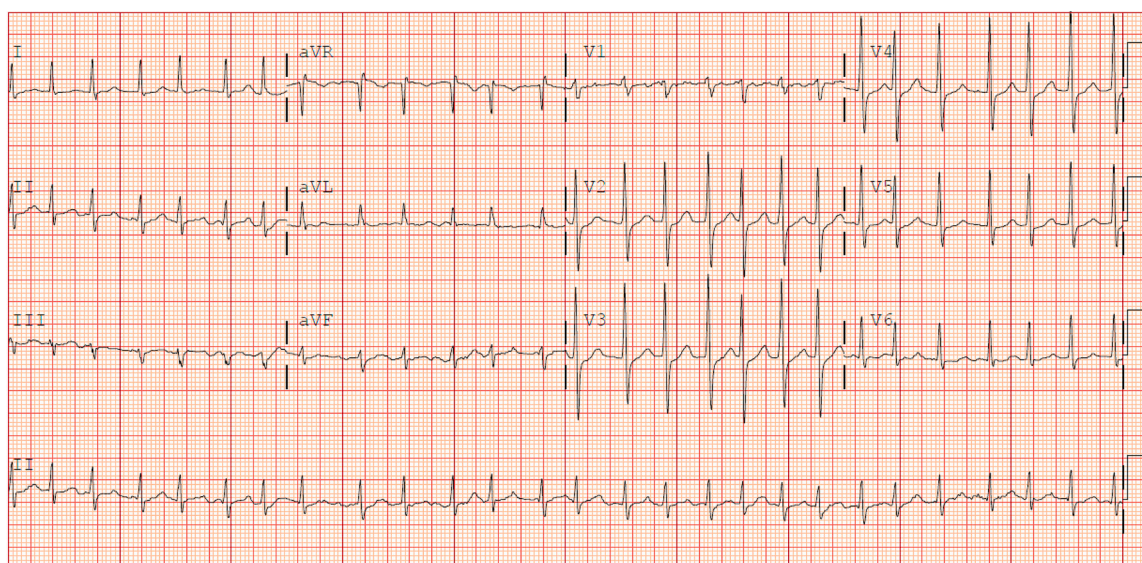


Figure 2 Twelve-lead ECG showing atrial fibrillation with rapid ventricular rate, HR 158 bpm. ECG, electrocardiogram; HR, heart rate.

defibrillator (LifeVest, Zoll, Pittsburgh, PA). Follow up cardiac MRI showed interval decrease in linear epicardial enhancement at the anterolateral and inferolateral walls of the left ventricle at the base. Findings were suspected to represent scarring from prior inflammation. No new areas of abnormal myocardial delayed enhancement were identified (*Figure 4*). He stopped wearing his LifeVest after 3 months and his amiodarone was also stopped. However, given residual late gadolinium enhancement on the cardiac

MRI, we explained to the patient that he remained at risk for ventricular arrhythmia. Risks and benefits of an implantable cardioverter defibrillator (ICD) and implantable cardiac monitor (ICM) were discussed and patient chose to have an ICM implanted.

Seven months after initial presentation, patient presented to the hospital on account of palpitations. His ECG and ICM interrogation showed VT (*Figure 5* and *Figure 6* respectively). He was chemically cardioverted with

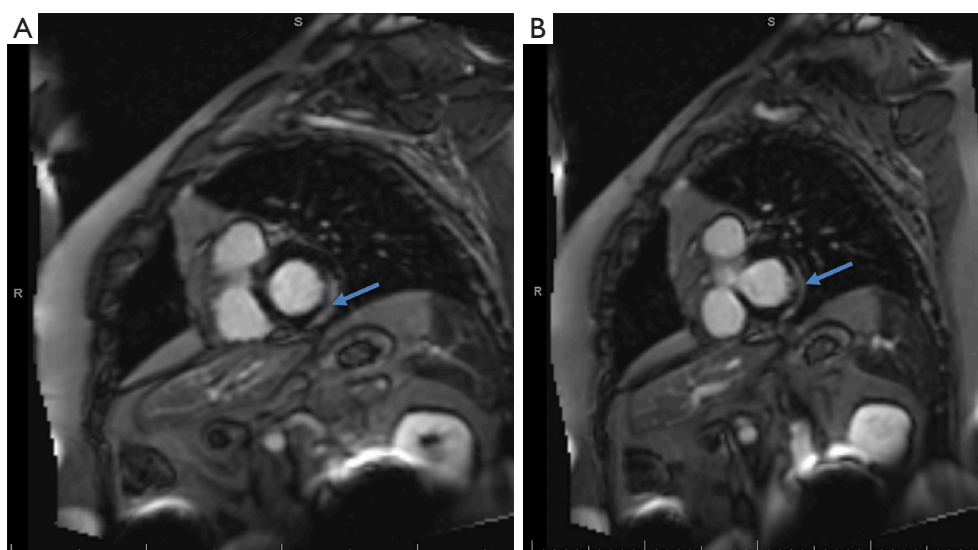


Figure 3 Cardiac MRI showing delayed gadolinium enhancement in the anterior lateral (A) and inferior lateral (B) segments of the left ventricle (arrows). MRI, magnetic resonance imaging.

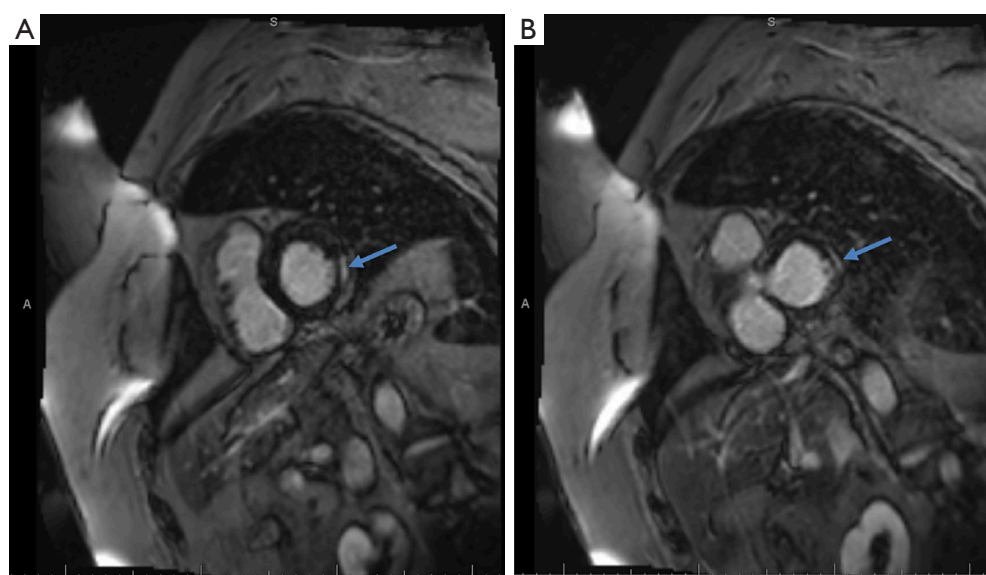


Figure 4 Cardiac MRI showing interval decrease in delayed gadolinium enhancement in the anterior lateral (A) and inferior lateral (B) segments of the left ventricle (arrows). MRI, magnetic resonance imaging.

intravenous amiodarone. An echocardiogram was repeated and this showed unchanged findings compared to the one obtained 7 months earlier. He underwent implantation of a single chamber secondary prevention ICD with a VDD lead [LinxSmart DX (Biotronik SE & Co., Berlin, Germany)] and explant of his ICM. He was discharged home after being initiated on sotalol. Over the following 2 months,

patient had recurrent monomorphic VT which his ICD successfully terminated with one round of anti-tachycardia pacing (*Figure 7*). In addition to adjusting his metoprolol dose, his sotalol dose was up-titrated. There have been no further episodes of VT in the past 6 months. Given that COVID-19 vaccine-associated myocarditis remains a diagnosis of exclusion, we decided to evaluate for cardiac

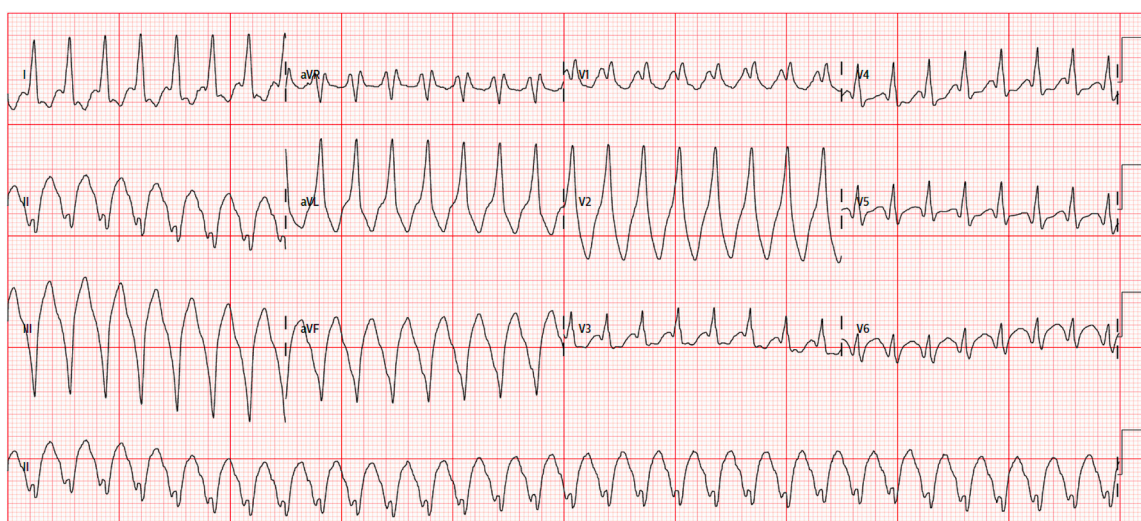


Figure 5 ECG showing ventricular tachycardia. ECG, electrocardiogram.

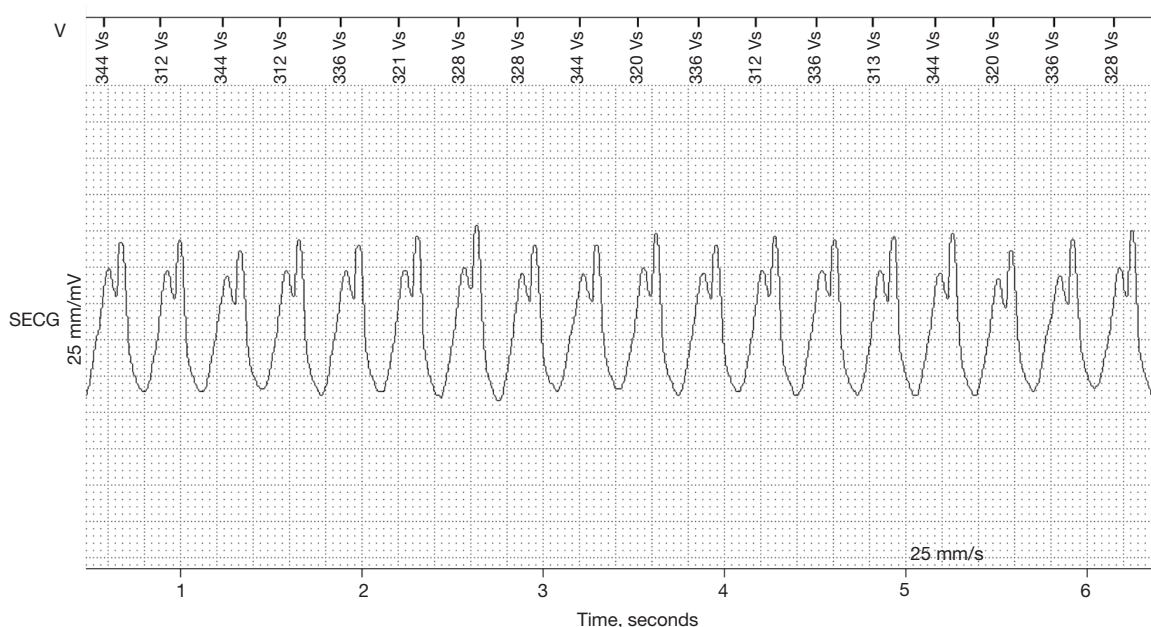


Figure 6 Tracing from an implantable cardiac monitor showing ventricular tachycardia. SECG, standard electrocardiography; Vs, ventricular-sensed.

sarcoidosis. We obtained an ^{18}F -fluorodeoxyglucose (FDG) metabolism cardiac sarcoid positron emission tomography (PET) study. This showed a normal study with fasting FDG blood pool uptake with no myocardial FDG uptake.

Discussion

The development of COVID-19 vaccine-associated

myocarditis has been documented (1-3,5,6). It is very uncommon for initial presentation of COVID-19 vaccine-associated myocarditis to be sustained VT. Based on our literature review, only one such case has been reported in the past (7). Most of the reported cases are mild, with quick clinical recovery and excellent short-term outcomes (4). There is limited data on long-term outcomes. Long-term outcome may not be benign as illustrated in



Figure 7 Intracardiac tracings from the patient's ICD, showing an episode of ventricular tachycardia terminated by anti-tachycardia pacing. ICD, implantable cardioverter defibrillator; FF, far-field; A, atrial channel; V, ventricular channel; As, atrial-sensed; Ars, atrial refractory-sense event; VT1, ventricular tachycardia detection zone; Vp, ventricular-paced; ATP, anti-tachycardia pacing; Vs, ventricular-sensed; VDI, VDI pacing mode gives ventricular pacing (V), while sensing both chambers (D), and inhibits ventricular pacing if an intrinsic R wave is sensed.

our patient, particularly in those who develop myocardial scar. Even though the exit of the VT could only be accurately determined with mapping, the morphology of the VT is suggestive of a basal inferoseptal exit (right bundle morphology with precordial transition beyond V6, left superior axis). This presentation of COVID-19 vaccine-associated myocarditis illustrates the need to better understand the mechanism of this pathology. Potential mechanisms include cytokine-induced hyperinflammatory response, development of autoantibodies and molecular mimicry of the spike glycoprotein of the COVID-19 virus. A report by Won *et al.* demonstrated higher interleukin-18, interleukin-27 and Th1-type cytokines in a patient with COVID-19 vaccine-associated myocarditis (8).

It is well known that some patients from myocarditis due to causes other than related to COVID-19 mRNA vaccine can have residual scar which can predispose them to ventricular arrhythmia in the future (9,10). Our case shows that the same is possible in COVID-19 vaccine-associated myocarditis. Hence, should our patient have VT again, catheter ablation to modify the arrhythmogenic substrate may be an option given that he is young and he is already on maximum dose of one antiarrhythmic medication. Given that patient is on an antiarrhythmic medication, he has office visit every 6 months and remote monitoring of his ICD every 3 months.

Conclusions

Excellent short-term outcomes have been reported in patients with COVID-19 vaccine associated myocarditis. Our case shows that long-term outcomes may not be benign in everyone, particularly in those who develop myocardial scar.

Acknowledgments

Funding: None.

Footnote

Reporting Checklist: The authors have completed the CARE reporting checklist. Available at <https://atm.amegroups.com/article/view/10.21037/atm-22-4164/rc>

Peer Review File: Available at <https://atm.amegroups.com/article/view/10.21037/atm-22-4164/prf>

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at <https://atm.amegroups.com/article/view/10.21037/atm-22-4164/coif>). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editorial office of this journal.

Open Access Statement: This is an Open Access article distributed in accordance with the Creative Commons Attribution-NonCommercial-NoDerivs 4.0 International License (CC BY-NC-ND 4.0), which permits the non-commercial replication and distribution of the article with the strict proviso that no changes or edits are made and the original work is properly cited (including links to both the formal publication through the relevant DOI and the license). See: <https://creativecommons.org/licenses/by-nc-nd/4.0/>.

References

1. Witberg G, Barda N, Hoss S, et al. Myocarditis after Covid-19 Vaccination in a Large Health Care Organization. *N Engl J Med* 2021;385:2132-9.
2. Rosner CM, Genovese L, Tehrani BN, et al. Myocarditis Temporally Associated With COVID-19 Vaccination. *Circulation* 2021;144:502-5.
3. Kim HW, Jenista ER, Wendell DC, et al. Patients With Acute Myocarditis Following mRNA COVID-19 Vaccination. *JAMA Cardiol* 2021;6:1196-201.
4. Jain SS, Steele JM, Fonseca B, et al. COVID-19 Vaccination-Associated Myocarditis in Adolescents. *Pediatrics* 2021;148:e2021053427.
5. Perez Y, Levy ER, Joshi AY, et al. Myocarditis Following Coronavirus Disease 2019 mRNA Vaccine: A Case Series and Incidence Rate Determination. *Clin Infect Dis* 2022;75:e749-54.
6. Minocha PK, Better D, Singh RK, et al. Recurrence of Acute Myocarditis Temporally Associated with Receipt

- of the mRNA Coronavirus Disease 2019 (COVID-19) Vaccine in a Male Adolescent. *J Pediatr* 2021;238:321-3.
7. Lin W, Yip ACL, Evangelista LKM, et al. Ventricular tachycardia from myocarditis following COVID-19 vaccination with tozinameran (BNT162b2, Pfizer-BioNTech). *Pacing Clin Electrophysiol* 2022;45:1097-100.
 8. Won T, Gilotra NA, Wood MK, et al. Increased Interleukin 18-Dependent Immune Responses Are Associated With Myopericarditis After COVID-19 mRNA Vaccination. *Front Immunol* 2022;13:851620.
 9. Androulakis E, Falconer D, Briasoulis A, et al. Long-term Outcomes of Catheter Ablation for Ventricular Arrhythmias in Post-Myocarditis Patients: Insights from a Meta-Analysis of Current Data. *SN Compr Clin Med* 2022;4:62.
 10. Berte B, Sacher F, Cochet H, et al. Postmyocarditis ventricular tachycardia in patients with epicardial-only scar: a specific entity requiring a specific approach. *J Cardiovasc Electrophysiol* 2015;26:42-50.

Cite this article as: Ojo A, Goldenberg I, Kutiyfa V, Aktas MK, Rosero S, Ayinde H, Huang DT. Recurrent ventricular tachycardia in a patient with COVID-19 vaccine-associated myocarditis: a case report. *Ann Transl Med* 2023;11(6):267. doi: 10.21037/atm-22-4164