Mapping out the dots - Could trivial myocarditis emerge as a life-threatening ventricular electrical storm? A case report

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Case Report

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Abstract

Ventricular tachycardia storm is a term used when there are three or more episodes of sustained ventricular tachycardia within 24 hours, each requiring termination by an intervention such as pharmacological cardioversion, anti-tachycardia pacing (ATP), or defibrillation. Broadly classifying, this type of arrhythmia can be due to ischemic heart disease or non-ischemic heart disease. A patient may present with a complaint of shortness of breath, palpitations, chest heaviness, syncope or even it can cause sudden cardiac death. In our case report, the patient had ventricular electrical storm activity and his initial electrocardiogram depicts the source of origin of arrhythmia from the left ventricular outflow tract and septal area. The cardiac magnetic resonance imaging (CMR) was carried out later and showed the foci of hyperenhancement in the epicardium and mid-myocardium of the left ventricle on delayed enhancement sequence suggesting localized scar and relative thinning with trabeculation. These findings more likely pointed toward previous unnoticed subclinical myocarditis that led to fibrosis and ultimately caused life-threatening electrical instability in the left ventricle.

Background

Ventricular arrhythmia can be a life-threatening event. Sustained ventricular tachycardia is one of the manifestations which are encountered in patients with coronary artery disease with myocardial infarction, this phenomenon can also be witnessed in dilated cardiomyopathies, sarcoidosis, post-myocarditis, repaired congenital cardiac diseases, and inherited channelopathies. Ventricular tachycardia storm, commonly abbreviated as VT storm, is coined for ventricular tachycardia occurring in a short frame of time; three or more episodes of sustained VT within 24 hours, each requiring termination by an intervention such as pharmacological cardioversion, anti-tachycardia pacing (ATP) or defibrillation (1).

Ventricular tachycardia storm (VT Storm) is a critical medical situation, a patient may present with syncope, heart failure, or even cardiac arrest and a patient with an implantable cardio-defibrillator (ICD) may present with repeated anti-tachycardia pacing (ATP) or shocks. Thus, initial management includes stabilizing the patient haemodynamically, looking for reversible triggering elements including electrolyte imbalance, heart failure exacerbation, and ongoing ischemia, and therapeutic interventions to reduce the recurrence of VT storms. Therapeutic intervention includes administrating anti-arrhythmic drugs (including beta-blockers, amiodarone, sotalol, and lidocaine/mexiletine) and long-term interventions include ICD placement, catheter ablation, and surgical sympathectomy for secondary prevention (2). Implantable cardiac defibrillator among survivors of ventricular fibrillation or sustained ventricular tachycardia is superior to antiarrhythmic drug therapy (3). In one of the meta-analyses, S. J. Connolly et al studied randomized control trials on ICD therapy vs. medical treatment for the prevention of death in survivors of ventricular fibrillation or sustained ventricular tachycardia and study found that 28% reduction in the relative risk of death with the ICD resulting to 50% reduction in arrhythmic death (4). In Multicenter Automatic Defibrillator Implantation Trial (MADIT), it was found that in patients with a previous history of myocardial infarction and increased risk of ventricular tachyarrhythmia, prophylactic therapy with ICD improved survival than medical therapy with ant-arrhythmic drugs (5).

This case report is about a young male patient who presented through emergency and had multiple episodes of ventricular tachycardia. This case report describes the initial steps that resulted in the survival of this patient and discusses the interesting Cardiac Magnetic Resonance Imaging (CMR) findings and mentions long-term intervention for this patient for secondary prevention.

Case

A 28 years old male patient with no known co-morbidities, not addicted to any substance, presented with complaints of palpitations on and off for 1 month, blackouts and dizziness, and sweating for 1 hour before presentation in the emergency room. He was in the usual state of health one month back then he started feeling palpitation, not related to any activity, can occur at rest. He developed blackouts and felt dizzy before presenting in the emergency room. He denied any syncope episode during this interval; he also denied any complaint of shortness of breath or chest discomfort. He also denied any significant history of joint pain, rash, cough or fever, or any flu-like signs and symptoms before this presentation. He also denied any previous history of ischemic heart disease or premature ischemic heart disease in the family or any sudden cardiac death in the family. He denied any previous hospitalization or any drug history.

Electrocardiogram taken at the emergency room showed sustained monomorphic ventricular tachycardia, he was hemodynamically stable. He was given an initial dose of Inj. Amiodarone 150 mg but the rhythm was not reverted then 3 direct cardioversion shocks with were given (of 150J, 200J &250J respectively) and the patient was reverted to normal sinus rhythm. After stabilization, he was transferred to the intensive care unit where he again developed monomorphic ventricular tachycardia. The rhythm was not reverted with amiodarone infusion and then 2 direct cardioversion shocks were given (150J and 200J respectively) and normal sinus rhythm was achieved.

Left heart catheterization was done after stabilizing the patient which showed normal coronary arteries. The electrophysiology team was taken on board and they advised to start Inj. lidocaine instead of amiodarone (1mg/kg IV bolus followed by 1–4 mg/min continuous infusion) as corrected QT interval was 480 ms. EP team also advised replacing potassium and magnesium and started propranolol 20 mg every 6 hours. There were no episodes of ventricular tachycardia episodes after starting above mentioned regimen.

On general physical examination, the young obese male patient lying on the bed is well oriented with time, place, and person and vitally stable. On cardiovascular examination, the apex beat was located at the 5th intercostal space non forceful in nature, and S1 and S2 were audible of equal intensity and no murmur was appreciated. While abdominal, respiratory and neurological system examinations were unremarkable.

On further laboratory investigation, his baseline blood investigations were within normal limits except for his CRP which was initially 5.5 mg/L and later level was 4 mg/L, his potassium was 3.0 mmol/L and magnesium was 1.7 mg/dL, which were replaced and later levels were with normal limits. His ACE

(angiotensin converting enzyme) levels were also within the normal range. His echocardiography was done which showed the normal biventricular size and systolic function and normal morphology of cardiac valves and ejection fraction of 65%. Cardiac magnetic resonance imaging (CMR) was carried out showing normal-sized cardiac chambers, normal left ventricular systolic function, and an estimated left ventricular ejection fraction of 60%. The left ventricle basal inferolateral segment was noted as hypokinetic. Basal to mid inferior, anterolateral, and inferolateral segments were relatively thin (4–6 mm) with increased trabeculations. The fat suppression sequence showed fat suppression in mid-myocardial region of in the basal inferolateral segment. Delayed enhancement imaging showed the foci of hyperenhancement in epicardium and mid-myocardium. These findings suggested two differential diagnosis based, it could be either due to post myocarditis changes or undifferentiated hereditary cardiomyopathy. Following is the link to the clip of CMR:

https://youtu.be/6-AwyryQpt0

The electrophysiology physician team planned for implantable cardiac defibrillator (ICD) insertion for secondary prevention. The patient was discharged from the hospital after ICD implantation and advised for follow-up in the outpatient department. On his first follow-up, the patient was feeling well and symptoms free and there were no active complaints.

Discussion

This case report pertains to a patient who presented with repeated episodes of ventricular tachycardia requiring pharmacological and mechanical cardioversion. The patient electrocardiogram suggested that arrhythmic rhythm was originating from the left ventricular outflow tract and septal area. After stabilization, the patient was taken for left heart catheterization for any possible ischemia that resulted in the ventricular tachycardiac storm and coronary angiography revealed normal coronary arteries. The baseline laboratory work-up was not pointing towards any pathology. The decision was made to carry out a CMR which disclosed localized scar in basal lateral walls of the left ventricle and relative thinning with trabeculation noted in anterolateral and inferolateral walls and hyperenhancement foci in epicardium and mid-myocardium in delayed enhancement. As per the history of the patient, there was no prior ischemic heart disease and there was no systolic dysfunction, thus this scar was more likely non-ischemic in origin and substrate for the VT storm. Though, the associated history of previous myocarditis episode or any major trauma remained unclear but there was no past surgical history for correction of congenital cardiac diseases. Myocardial biopsy was not done due to a lack of resources and the critical nature of the process. An implantable cardioverter-defibrillator (ICD) was placed for secondary prevention and he was discharged for home on beta blockers.

Generally speaking, myocardial infarction is the main cause of ventricular scars; though scars can also occur in nonischemic cardiomyopathies due to replacement fibrosis, thus scar creates a substrate for reentry and leads to ventricular tachyarrhythmia or even electrical storm (6). Talking about diagnosing and mapping out the ventricular scar, cardiac MRI (CMR) has been the gold standard non-invasive

modality as CMR can image in any desired plane and with an almost impeded field of view (7). The late gadolinium enhancement (LGE)-CMR technique delineates the zones of myocyte necrosis or myocardial fibrosis and visualizes even small subendocardial infarcts that might otherwise be missed (8). Generally, the area of hyperenhancement (HE) of CMR in ischemic cardiomyopathy always involves the subendocardium while the involvement of mid-wall or epicardial HE strongly suggests a non-ischemic cause (7); the latter pattern is seen in this case report. Further discussing, the treatment course of ventricular tachyarrhythmia related to scar, antiarrhythmic drug therapy can reduce VT episodes but has shown poor efficacy to prevent reentry phenomenon because of substantial morphological change(6). An implantable cardioverter-defibrillator (ICD) can successfully terminate ventricular tachycardia event, either through a high voltage shock cardioversion, or repeated anti-tachycardia pacing (ATP) that interrupts reentry (6). Another option can be catheter ablation to reduce VT episodes and can be life-saving in continual VT episodes, this technique maps out reentry circuits and disrupts with a radiofrequency burn. The results are favorable in ischemic scar origin VT while in non-ischemic scar related VT, the ablation is more difficult as the location of the scar is often intramural or epicardial (6).

Conclusion

A thorough review of this case report suggested that the patient had asymptomatic myocarditis that later led to myocardial necrosis and fibrosis and ultimately resulted in a ventricular electrical storm. In the context of the recent COVID-19 pandemic, it could be possible that the patient had under noticed symptoms of COVID-19 but affected myocardium and progressed into subclinical myocarditis. A cohort study recently conducted showed that college athletes who were tested COVID-19 positive developed asymptomatic myocarditis only evident on CMR (9). There are limitations to link this case with COVID-19 related myocarditis in this patient but the nonischemic origin of the scar and presentation of this patient during the surge of COVID-19 pandemic can still be a differential diagnosis in our patient.

Declarations

Authors declare no competing interests

Patient provided written informed consent

References

1. Zaman J, Agarwal S. Management of ventricular tachycardia storm. Heart [Internet]. 2021 Oct 1 [cited 2022 Apr 3];107(20):1671–7. Available from: https://heart.bmj.com/content/107/20/1671
2. Elsokkari I, Sapp JL. 1. Elsokkari I, Sapp JL. Electrical storm: Prognosis and management. Prog Cardiovasc Dis. 2021 May 1;66:70–9. Electrical storm: Prognosis and management. Prog Cardiovasc Dis. 2021 May 1;66:70–9.
3. A comparison of antiarrhythmic drug therapy with implantable defibrillators in patients resuscitated from near-fatal ventricular arrhythmias. N Engl J Med [Internet]. 1997 Nov 27 [cited 2022 Apr

3];337(22):1576–84. Available from: https://pubmed.ncbi.nlm.nih.gov/9411221/

4. Connolly SJ, investigators of the AVID C and C studies, Hallstrom AP, investigators of the AVID C and C studies, Cappato R, investigators of the AVID C and C studies, et al. Meta-analysis of the implantable cardioverter-defibrillator secondary prevention trials. Eur Heart J [Internet]. 2000 Dec 1 [cited 2022 Apr 3];21(24):2071–8. Available from: https://academic.oup.com/eurheartj/article/21/24/2071/490226
5. Moss AJ, Hall WJ, Cannom DS, Daubert JP, Higgins SL, Klein H, et al. Improved survival with an implanted defibrillator in patients with coronary disease at high risk for ventricular arrhythmia. Multicenter Automatic Defibrillator Implantation Trial Investigators. N Engl J Med [Internet]. 1996 Dec 26 [cited 2022 Apr 3];335(26):1933–40. Available from: https://pubmed.ncbi.nlm.nih.gov/8960472/
6. Stevenson WG. Ventricular Scars and Ventricular Tachycardia. Trans Am Clin Climatol Assoc [Internet]. 2009 [cited 2022 Apr 9];120:403. Available from: /pmc/articles/PMC2744510/
7. Karamitsos TD, Francis JM, Myerson S, Selvanayagam JB, Neubauer S. The Role of Cardiovascular Magnetic Resonance Imaging in Heart Failure. J Am Coll Cardiol. 2009 Oct 6;54(15):1407–24.
8. Vöhringer M, Mahrholdt H, Yilmaz A, Sechtem U. Significance of late gadolinium enhancement in cardiovascular magnetic resonance imaging (CMR). Herz [Internet]. 2007 Mar [cited 2022 Apr 9];32(2):129–37. Available from: https://pubmed.ncbi.nlm.nih.gov/17401755/
9. Daniels CJ, Rajpal S, Greenshields JT, Rosenthal GL, Chung EH, Terrin M, et al. Prevalence of Clinical and Subclinical Myocarditis in Competitive Athletes With Recent SARS-CoV-2 Infection: Results From the Big Ten COVID-19 Cardiac Registry. JAMA Cardiol [Internet]. 2021 Sep 1 [cited 2022 Apr 9];6(9):1078–87. Available from: https://jamanetwork.com/journals/jamacardiology/fullarticle/2780548

Figures

Figure 1

This electrocardiogram taken at the emergency room shows sustained monomorphic wide complex tachycardia and positive concordance in all precordial leads indicating ventricular tachycardia. Note a positive R wave in V1/V2 indicating broad complex tachycardia with right bundle branch (RBBB) morphology indicating the arrhythmia is generated in the left ventricle. Also note positive inferior leads, II, III, avF, pointing the focus to the outflow tract/base of the LV, and negative deflection of lead I and avL are pointing to a septal source.

Supplementary Files

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- VID20220415222544.mp4