Case Report

Ventricular Tachycardia Storm in a Patient with an Implanted Cardioverter-Defibrillator Following COVID-19 Infection

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Abstract

Severe Coronavirus disease 2019 (COVID-19) infection presents with acute respiratory distress syndrome and multiorgan dysfunction. Cardiac involvement is seen in about a quarter of patients, and it can present as acute coronary syndromes, arrhythmias, myocarditis, and thromboembolic events. Ventricular arrhythmias in the setting of COVID-19 infection are usually multifactorial in etiology. There are only a few reports of ventricular tachycardia (VT) storms in patients with COVID-19 infection. We hereby report a case of an elderly man with severe left ventricular systolic dysfunction and a stable cardiac status for the last few years who, following coronary artery bypass graft surgery and implantable cardioverter-defibrillator (ICD) implantation, experienced a VT storm after a COVID-19 infection. The VT storm was controlled using multiple ICD shocks, along with antiarrhythmic drugs. Following his recovery from COVID-19 infection, the patient was asymptomatic at a 3-month follow-up.

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Keywords: *COVID-19; Myocarditis; Defibrillators, implantable; Tachycardia, ventricular*

Introduction

Coronavirus disease 2019 (COVID-19) was first reported on December 9, 2019, from Wuhan province, China.[1] It soon became a pandemic of alarming proportions, with crippling effects on healthcare systems and economies. COVID-19 predominantly involves the respiratory system, with almost a quarter of patients having cardiac involvement.[2] Cardiac manifestations include acute coronary syndromes, arrhythmias, myocarditis, heart failure, and thromboembolic events. Patients with underlying cardiovascular diseases have an increased incidence rate of intensive care unit admissions, need for mechanical ventilation, and mortality.[2-4] Both tachy and bradyarrhythmias have been reported in COVID-19, though the causal association is not clear.[5] The most common arrhythmia is atrial fibrillation, while ventricular tachyarrhythmias are reported in a few cases.[2, 5] We hereby describe a case of ventricular tachycardia (VT) storm in a patient with symptomatic COVID-19 infection, which was successfully managed with antiarrhythmic drugs and repeated implantable cardioverter-defibrillator (ICD) shocks.

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Case Report

The case was a 76-year-old diabetic, hypertensive, post-coronary artery bypass graft surgery male patient who had a magnetic resonance imaging (MRI)-compatible ICD (EVERA device, Medtronic Inc, Dublin, Ireland) implantation procedure 3 years previously for the secondary prevention of VT and a low left ventricular ejection fraction of 30%. A computerized tomography (CT) coronary angiography, performed 6 months earlier for atypical chest pain, showed patent grafts. Until the current admission, he was asymptomatic on metoprolol XL (50 mg once daily), telmisartan (40 mg once daily), and spironolactone (25 mg once daily) and did not receive any appropriate ICD-delivered shocks. Recently, he had presented to the emergency department with a complaint of multiple ICD shocks, delivered in the preceding 6 hours. He also had a fever with myalgia of 5 days' duration and tested positive for Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) infection via the reverse transcriptase-polymerase chain reaction method. Clinical examination was unremarkable, with stable vital signs and oxygen saturation of 97% on room air. An electrocardiogram showed normal sinus rhythm, no ST-T changes, and a QTc interval of 464 milliseconds. An echocardiogram revealed global hypokinesia and a left ventricular ejection fraction of 30%. The patient's serum biochemistry, including potassium (3.9 meq/L) and magnesium (2.0 meq/L), was within normal limits. Inflammatory markers of COVID-19 infection such as C-reactive protein (64 mg/dL) and ferritin (655 mg/dL) were elevated. Cardiac biomarkers, composed of creatine kinase (78 mg/dL), troponin T (55.3 pg/mL), and N-terminal pro-brain natriuretic peptide (NT-proBNP) (16328 pg/mL), were also elevated. A cardiac MRI could not be performed during his hospital stay. The ICD interrogation (VT zone > 150 bpm and VF zone > 188 bpm) showed multiple episodes of monomorphic VTs at 170 to 200 beats per minute following a ventricular ectopic (Figure 1A). The tachycardia persisted despite device-delivered anti-tachycardia pacing (Figure 1A), and it was terminated by 38 shocks of 35 J each (Figure 1B). The patient was started on an amiodarone infusion, and metoprolol doses were increased. His VT storm was controlled in the next 24 hours of admission; thereafter, there was no recurrence. He received symptomatic treatment for his COVID-19 infection and recovered in the next 10 days of his hospital stay. He was discharged on amiodarone (400 mg once daily), metoprolol XL (100 mg twice daily), telmisartan (40 mg once daily), torsemide-spironolactone (10/25 mg once daily), aspirin, statins, and oral hypoglycemics. A clinical follow-up at 3 months showed no episodes of VT on device interrogation, and the patient was asymptomatic.

Discussion

The VT storm is defined as 3 or more episodes of VT or ventricular fibrillation in 24 hours, requiring appropriate interventions (tachycardia pacing or shock).[6] The incidence

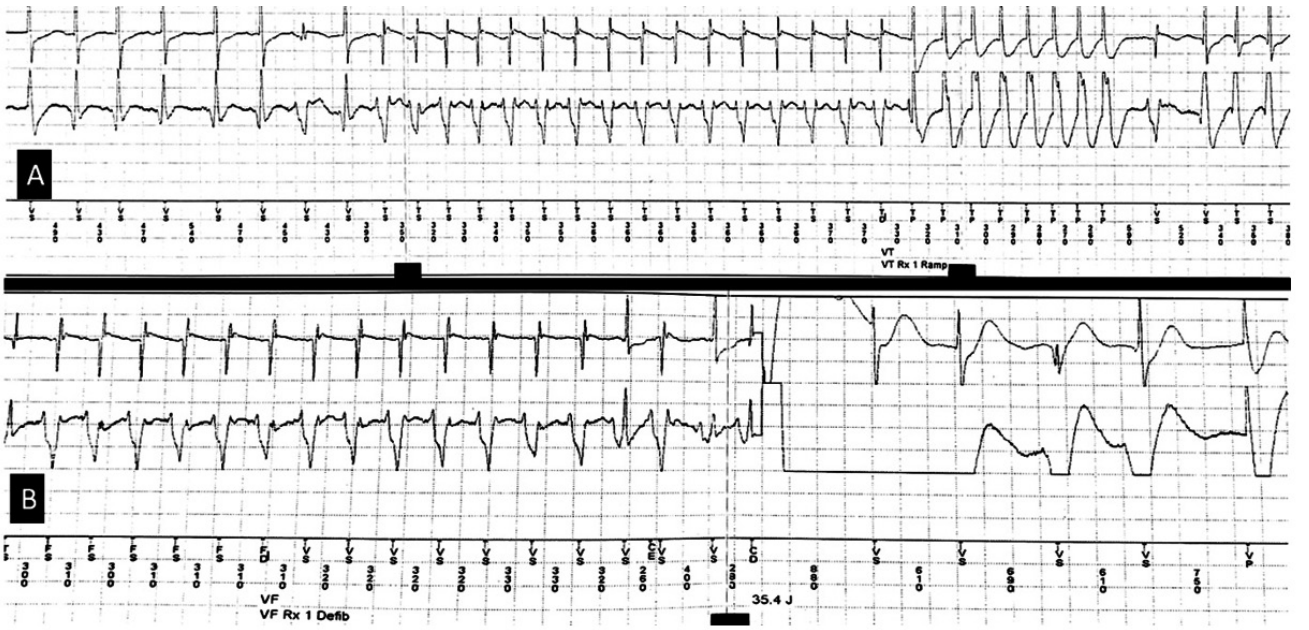

Figure 1. A) The intracardiac electrogram shows an episode of ventricular tachycardia (VT) at a cycle length of 360 milliseconds, following a ventricular ectopic, which persisted despite anti-tachycardia pacing.

B) The intracardiac electrogram shows an episode of ventricular tachycardia (VT) at a cycle length of 310 milliseconds, which was terminated following a 35 J shock therapy.

of the VT storm varies from 4% to 28%, depending upon the indication for ICD implantation (ie, primary or secondary prevention).[6, 7] Most patients have underlying structural heart diseases, although it can also occur in patients with a structurally normal heart.[6, 7] Common precipitating factors include acute coronary syndromes, myocarditis, heart failure, electrolyte abnormalities, hyperthyroidism, and sepsis.[8] The precipitating cause remains elusive in the majority of patients, while a reversible cause can be identified only in a few patients (13%).[6, 9] A VT storm often presents with hemodynamic instability, resulting in increased hospitalization rates (50%–80%) and mortality.8 Most episodes are monomorphic in morphology, although polymorphic VT can rarely occur.[8] The index case had a monomorphic VT storm.

Myocardial injury with COVID-19 infection is defined as a greater than 99th percentile elevation in troponin levels or a combination of elevated troponin levels with electrocardiographic and echocardiography changes.[10] It occurs in about 7% to 28% of patients, who carry a poor prognosis compared with those without elevation.[10] Wang et al[1] reported an increased incidence of arrhythmias (6.9% vs 44.4%) in patients with severe disease, although the type of arrhythmia was not specified. In another study by Guo et al,[2] the incidence of ventricular tachyarrhythmias was 5.9% and was more common in patients with elevated troponin levels (17.3% vs 1.5%; P<0.001). The mechanism of arrhythmias is multifactorial in COVID-19 infection. It includes a direct myocardial injury by the virus and an indirect injury by various mechanisms such as hypoxia, metabolic disarray, cytokines and chemokines, and the use of drugs that prolong the QT interval (eg, hydroxychloroquine, azithromycin, and lopinavir/ritonavir).[11, 12] Cytokines like interleukin-6 (IL-6) can inhibit the hERG-K^+ channel and prolong the QT interval.[11] IL-1, IL-6, and tumor necrosis factor-alpha (TNF-α) can also modulate K^+ and Ca^{+2} channels and prolong the action potential duration.[12] Inflammatory cytokines also increase the arrhythmic risk by their action on the hypothalamus and the peripheral cardiac sympathetic system.[12]

The index case had a VT storm probably due to systemic inflammation following COVID-19 infection. There was no further reduction in myocardial function as the ejection fraction remained the same (i.e., 30% during infection, which ruled out overt myocarditis). Moreover, no evidence of active ischemia was observed because of the absence of angina and ST-T changes. Bypass grafts were also patent at the time of ICD implantation and in the recent CT coronary angiogram. Recently, there have been reports of a rise in the incidence of out-of-hospital cardiac arrests in the current pandemic era.[13] The presence of the ICD in the index case averted a sudden cardiac death. The literature offers only a few published case reports of VT storms in COVID-19 infection.[14-16] In the index case, doses of metoprolol and amiodarone, which have evidence to reduce sudden cardiac death,[17] were increased at the time of discharge. Our case highlights the need to explore and optimize the management of COVID-19–infected patients at high risk for malignant cardiac arrhythmias, especially those with underlying structural heart diseases.

Conclusion

The index case suggests that in patients with a pre-existing cardiac disease, COVID-19 infection can precipitate dangerous cardiac arrhythmias. It also emphasizes the importance of developing appropriate strategies in these patients for the prevention of sudden cardiac death.

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