Less is More: Managing Non-Sustained Ventricular Tachycardia in a Patient with SARS-CoV2 Infection

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Abstract The novel coronavirus disease 2019 (COVID-19) has changed our lives and reshaped our approach to management of various cardiovascular diseases. Ventricular tachycardia (VT) is a potentially life-threatening arrhythmia, most often seen in patients with structural heart disease. If underlying ischemia is suspected, coronary angiography is usually performed on a non-elective basis. In patients with active COVID-19, additional risks of the procedure must be considered for patients and for operators. This case illustrates the management of suspected ischemic VT and discusses the dilemma physicians must face in the ongoing COVID-19 pandemic.

Keywords: ventricular tachycardia, novel coronavirus, COVID, coronary artery disease, ischemic injury


1. Introduction

Since the isolation of severe acute respiratory syndrome Coronavirus 2 (SARS-CoV-2) in January of 2020, the novel coronavirus disease 2019 (COVID-19) has been sweeping through the world, changing the lifestyle of everyone. A variety of cardiovascular manifestations in COVID-19 were reported, including but not limited to arrhythmias, myocardial infarction, myocarditis, peripheral and central thrombosis [1,2]. Furthermore, the presence of cardiovascular complications was described as an independent risk factor for increased critical illness as well as mortality in COVID-19 patients [3,4].

Arrhythmia has been described to be associated with both the treatment and the primary disease process of COVID-19 [5]. In particular, ventricular tachycardia (VT) carries significant consequences of morbidity and mortality as it can lead to hemodynamic compromise and sudden cardiac death. There are multiple primary cardiac etiologies responsible for VT, such as ischemic disease, non-ischemic cardiomyopathy, myocarditis, congenital heart disease, and scars from prior cardiac surgeries [6].

Without an effective treatment to date, COVID-19 forced us to reconsider our therapeutic approaches. For example, in patients with suspected ST elevation myocardial infarction, some hospitals have taken precautions to ensure accurate diagnosis with bedside echocardiography [7]. Others have recommended thrombolytic therapy as initial management [8]. Regardless, thorough decontamination after planned intervention has been suggested [9,10]. Elective intervention for coronary artery disease (CAD) have been deferred to downsize case volume in laboratories and reduce exposure to SARS-CoV2 for both patients and health care workers (HCW) [10]. Urgent procedures were left to physicians’ discretion. Optimal timing of non-elective procedures is determined on a case-by-case basis. This case describes the management challenges we faced for a patient with VT due to suspected ischemic injury during the ongoing COVID-19 pandemic.

2. Case

A 69-year old male presented to the emergency department with worsening shortness of breadth, non-productive cough, and bilateral lower extremity edema for seven days. He had a past medical history of non-obstructive CAD, atrial fibrillation, remote history of ventricular septal defect (VSD) repair in his youth. He denies history of syncope, dizziness or exertional chest pain. On presentation, patient was hemodynamically stable, but had tachypnea at 20 breaths per minute although maintaining adequate oxygen saturation of 97% on room air. His physical exam revealed irregularly irregular heart rhythm with diffuse crackles on bilateral lung bases and +2 pitting edema in both of lower extremities. His outpatient medications included metoprolol,
furosemide, and losartan. Laboratory studies were significant for positive SARS-CoV2 antigen and mildly elevated troponin-I and brain natriuretic peptide (BNP). The other laboratory studies were otherwise normal and detailed in Table 1. Chest radiography demonstrated mild bilateral infiltrates along with cardiomegaly. Electrocardiogram (EKG) showed atrial fibrillation with right bundle branch block, without prolongation of QT interval or ischemic changes (Figure 1a). Using the Hodges formula, QT interval was calculated at 400 milliseconds (ms), and a corrected QT (QTc) interval of 467 ms. He was admitted to telemetry floor for respiratory insufficiency in the setting of COVID-19 pneumonia with acute heart failure. Patient received his home medication, except diuretic was switched to intravenous (IV) treatment, and he was started on hydroxychloroquine with azithromycin following initial loading dose.

Overnight on hospital day 2, patient had a run of monomorphic ventricular tachycardia (Figure 1b) lasting 20 seconds. He was sleeping during this episode. He was free of chest pain and denied any symptoms. The patient was evaluated and accepted to our coronary care unit (CCU). He was started on oral loading dose of amiodarone while on telemetry, as CCU transfer was not immediately possible.

Urgent laboratory studies showed no significant abnormalities in his electrolytes. An urgent echocardiogram was obtained and revealed a newly reduced left ventricle ejection fraction (LVEF) of 40%, with new apical and septal akinesis. His right atrium and right ventricle size were severely dilated, however unchanged compared to prior echocardiogram done ten months ago (Figure 2a-c). His right ventricular systolic pressure was unchanged as well, at approximately 35 mmHg. Patient was then transferred to the CCU for further care. EKG obtained at this time did not demonstrate changes in his ST segment, but QT interval was prolonged (QT 465ms and QTc 509ms, Figure 1c). Azithromycin and hydroxychloroquine were held and only oral amiodarone was continued.

| Table 1. Laboratory studies of patient during his hospitalization |
|----------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|
| Measurements (units) | Reference | Day 1 | Day 2 | Day 3 | Day 4 | Day 5 | Day 6 | Day 7 | Day 8 | Day 9 |
|----------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|------------------|
| Hemoglobin (g/dL)    | 12-16             | 14.5             | 13.1             | 13.9             | 13.7             |                |                |                |                |
| WBC (k/µL)           | 4-10.5            | 7.5              | 7.3              | 7.3              | 6.9              |                |                |                |                |
| Platelet count (k/cm²) | 150-450         | 361              | 338              | 333              | 359              |                |                |                |                |
| Potassium (mmol/L)   | 3.5-5             | 4.2              | 4.1              | 4.4              | 3.9              | 4.2             | 3.9             | 4.1             | 4.4             | 4.0             |
| Magnesium (mg/dL)    | 1.8-2.5           | 2.2              | 2.2              | 1.9              | 2.1              | 2.2             | 2.1             | 1.9             | 2.3             | 2.2             | 2.1             |
| Creatinine (mg/dL)   | 0.4-1.2           | 1.1              | 1.0              | 1.1              | 1.2              | 1.3             | 1.3             | 1.3             | 1.2             | 1.4             |
| AST (u/L)            | 10-40             | 66               | 55               | 53               | 44               | 48              |                |                |                |                |
| ALT (u/L)            | 10-42             | 61               | 50               | 40               | 59               |                |                |                |                |                |
| CRP (mg/dL)          | 0.0-0.5           | 5.0              | 4.4              | 3.3              |                |                |                |                |                |                |
| Troponin I (ng/mL)   | 0.006-0.06        | 0.049            | 0.067            | 0.033            | 0.034            |                |                |                |                |                |
| BNP (pg/mL)          | 0-100             | 298              |                |                |                |                |                |                |                |                |
| D-dimer (ng/mL)      | < 279             | 251              | 264              | 336              | 329              |                |                |                |                |                |

WBC: white blood cells. AST: Aspartate transaminase, ALT: Alanine transaminase, CRP: C-reactive protein, BNP: Brain natriuretic peptide

Figure 1a. Electrocardiogram on admission to our hospital
Figure 1b. Telemetry tracing showing extended non-sustain ventricular tachycardia

Figure 1c. Electrocardiogram on hospital day #3
Figure 2. Echocardiogram showing distended inferior vena cava with severely dilated right ventricle and right atrium

Patient continued to improve with IV diuretic. Very short runs of VT and frequent ventricular premature contractions were still seen, but no sustained ventricular arrhythmia was noted on telemetry tracing. In consultation with interventional cardiology team, decision was made to defer ischemic evaluation until after COVID-19 pandemic on outpatient basis. Patient was discharged to home for self-isolation with telephone follow-ups.

3. Discussion

Our case describes an arrhythmia that is commonly encountered by consulting cardiology services. Given its potential to cause significant hemodynamic instability, progression to ventricular fibrillation, and death, VT is especially concerning in patients with structural heart disease [6]. In a patient with newly identified reduced left ventricular ejection fraction (LVEF) and regional wall motion abnormality (RWMA) on echocardiography, ischemic injury becomes a significant concern. With the complexity of this patient’s cardiac history and active COVID-19 infection, additional considerations include acute myocarditis, side effect of medications, and scar burden associated with prior VSD repair.

In cases of uncertainty, both echocardiography and cardiac magnetic resonance imaging (cMRI) are powerful diagnostic tools to determine the etiology of VT. In our patient with reduced LVEF and new RWMA on bedside echocardiography, ischemic injury remains the most likely cause. Without additional clinical symptoms, electrocardiographic signs of coronary artery disease, and abnormal cardiac markers for ischemia, consideration for further diagnostic study is reasonable prior to coronary angiography in order to minimize HCW exposure in COVID-19. cMRI requires patient transportation and thorough decontamination compared to more mobile echocardiography. However, cMRI can better pinpoint inflammatory and fibrotic patterns to differentiate ischemic injury from myocarditis, infiltrative disease and surgical scar from prior VSD repair [11]. The non-invasive nature and relatively minimal personnel requirement make both studies attractive preliminary evaluations, and in the case of cMRI, a possible alternative to coronary angiography. Optimal timing of echocardiography and cMRI in such cases during COVID-19 pandemic is individualized and could benefit from larger scaled investigation. Computerized tomography coronary angiogram (CTA) is another diagnostic option. However, CTA is most powerful at ruling out severe CAD in low and intermediate risk patients and less effective in our patient with a higher suspicion of disease and known non-obstructive CAD. Exposure to radiation and ionized contrast should be considered as well, especially in
light of frequent renal complications in patients with COVID-19 [12].

Under normal circumstances, the obvious next step for ischemic evaluation in patients with VT and newly reduced LVEF with RWMA would be coronary angiography, usually prior to discharge from the hospital. During COVID-19 pandemic, increased risk of invasive procedures for transmission of SARS-CoV-2 should be taken into consideration. Additional personnel and personal protective equipment requirements should be contemplated. Catherization laboratories are generally kept under positive pressure to minimize potential contamination, while negative pressure rooms are generally recommended for patients with potential airborne diseases, such as COVID-19 [13]. Increased risk of exposure to both interventional cardiologists and supporting team members should be balanced with the necessity and clinical benefit of planned procedure. Further investigations to explore the optimal timing and necessity of coronary catherization in asymptomatic patients would be beneficial.

4. Conclusion

COVID-19 pandemic has changed the landscape of healthcare community in many ways. This crisis has accelerated the dissemination of clinical knowledge but also had us question established clinical practices. Definition and timing of urgent and essential procedures are being challenged. For the individual patients and health care providers, perhaps, less is more during the time of this pandemic.

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Statement of Ethics

The current study was exempted from IRB review.

Disclosure Statement

The authors have no conflicts of interest to declare.

Statement of Completing Interests

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