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Role of POCUS in the management of New-onset Tachyarrhythmia in the setting of SARS-CoV-2: A Case Report

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CASE REPORT

Role of POCUS in the Management of New-onset Tachyarrhythmia in the Setting of SARS-CoV-2: A Case Report

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Abstract

Introduction: **SARS-CoV-2 infection is associated with myocardial inflammation, new onset cardiomyopathy, and arrhythmias. Here, we describe the utilization of POCUS and management of concurrent new onset atrial tachycardia and heart failure with reduced ejection fraction (HfrEF) in a patient with SARS-CoV-2 infection.**

Presentation: **An 80-year-old female with multiple medical problems presented with sudden onset of shortness of breath and cough. She tested positive for SARS-CoV-2. Initially, she was hypoxic on room air and her heart rhythm was sinus tachycardia. CT angiogram of the chest showed consolidation, pleural effusion, and absence of pulmonary embolism. Because of persistent tachycardia, repeat EKGs and POCUS were performed. Subsequent EKGs showed intermittent atrial tachycardia and sinus tachycardia. Initially, home beta blockers were continued on admission, and additional dosages were considered for rate control, but Cardiac POCUS revealed HfrEF and was subsequently confirmed by comprehensive cardiac echocardiogram, consistent with SARS-CoV-2 infection-related cardiomyopathy. Beta blockers were discontinued, and treatment with amiodarone and furosemide showed improvement in symptoms. The patient was discharged with oral amiodarone and supplemental oxygen. Additionally, once the patient's hemodynamics improved, oral carvedilol was also started as part of GDMT for HfrEF. Follow-up echocardiogram 4 months later showed recovery of systolic EF to 60%.**

Conclusion: **It is essential to consider new onset HFrEF in the evaluation and management of new onset tachyarrhythmias since IV fluids and AV nodal blocking agents can be harmful in decompensated HFrEF. With the advent of POCUS, HFrEF can be quickly identified, and therapy can be tailored to that diagnosis.**

Keywords: Atrial tachycardia, Sinus tachyarrhythmia, SARS-CoV-2, Cardiomyopathy, Amiodaron

1. Introduction

SARS-Cov-2 is primarily recognized for causing respiratory symptoms. In addition, it can also affect multiple other organs leading to complications.[1] Myocardial inflammation plays a significant role in the development of cardiac complications and diverse arrhythmias. Notably, patients after contracting the infection can develop myocardial injury and arrhythmias.[2] Tachyarrhythmias are associated with higher mortality rates in COVID-19 patients.[3-5]

Here, we present a case of new stress-induced cardiomyopathy and concurrent new onset tachyarrhythmia in a patient with new acute (SARS-CoV-2) infection.

2. Case presentation

An 80-year-old female with a past medical history of cerebrovascular accident, hyperlipidemia,

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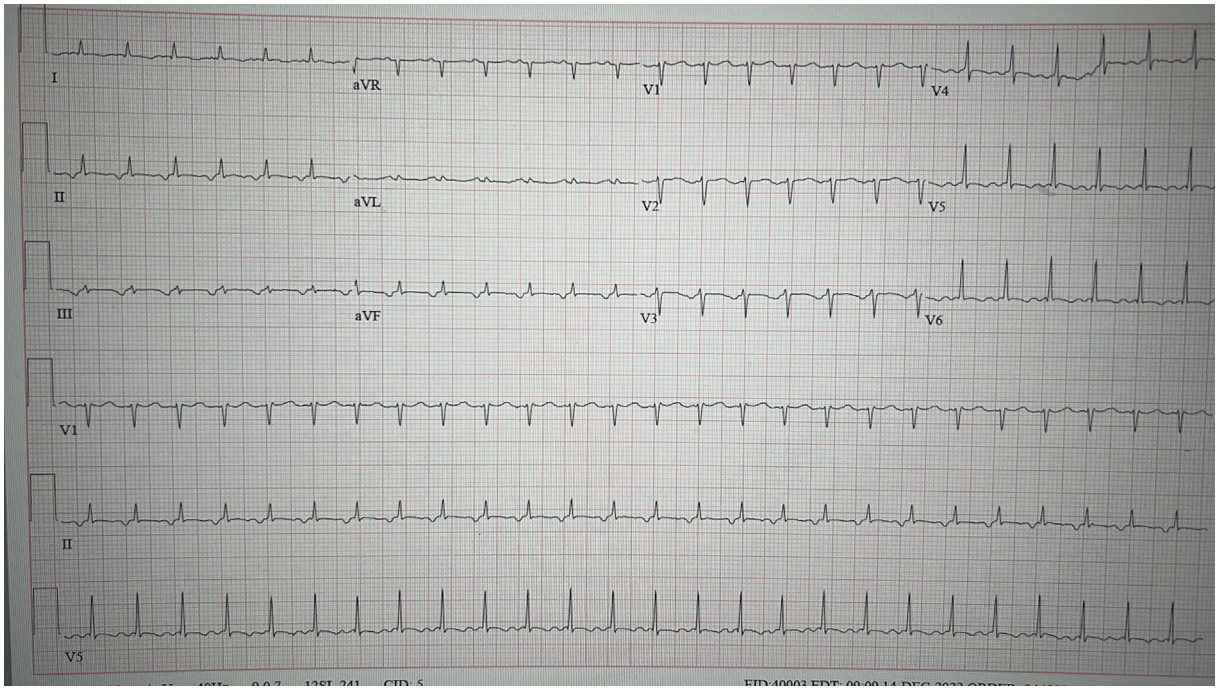

Fig. 1. Demonstration of EKG showing intermittent atrial tachycardia in the patient. Legend: EKG showing p wave in a regularly irregular rhythm suggestive of intermittent atrial tachycardia.

hypertension on metoprolol, and follicular lymphoma currently in remission, s/p 6 cycles of bendamustine and rituximab (completed 8 months before date of admission), presented with complaints of dyspnea on exertion for a day along with productive cough with small amount of sputum for 3 days. She was a previous smoker with 5 pack year history and quit smoking in the year 2000. On initial evaluation, her temperature was 99 F, heart rate was 128 beats per minute (bpm), blood pressure 140/104 mm Hg, respiratory rate 18/minute, and she was hypoxic with a SpO2 of 83% on room air which improved to 95% with 4 Liters/min of oxygen by nasal cannula. Physical exam reveals no edema in the distal extremities, no jugular venous distension, and significant for tachycardia and rhonchi on auscultation but otherwise unremarkable. An EKG showed sinus tachycardia. A rapid COVID PCR (manufacturer: CEPHEID) was positive. Complete blood count and basic metabolic panels were within normal limits. High sensitivity troponins (in nanograms/liter, normal $\leq$14 ng/L) at initial presentation, 1 h, and 3 h were 40 ng/L, 40 ng/L, and 42 ng/L, respectively, and serum lactic acid was elevated at 2.4 mmol/L (<2.1 mmol/L normal range). A CT angiogram of the chest revealed patchy multilobular consolidation in both lower lobes, mild bilateral pleural effusions, severe centrilobular emphysema, and there was no evidence of pulmonary embolism. Patient was started on remdesivir, dexamethasone, and standard precautions and admitted to the general medical floor for acute SARS-CoV-2 infection.

Initially, sinus tachycardia was considered to be secondary to acute SARS-CoV-2 pneumonia, and the patient was started on lactated ringer fluid at 100 ml/h, and home metoprolol (indication: hypertension) was continued on admission. The patient's tachycardia continued, with a baseline rate of 120 bpm, intermittently rising to 140–160 bpm. Fig. 1 shows the EKG of intermittent atrial tachycardia. The hospitalist on call at night was called by nursing to evaluate tachycardia around 18 h after admission. Multiple EKGs were done and showed persistent sinus tachycardia and intermittent atrial tachycardia.

POCUS was performed by the Society of Hospital Medicine POCUS-certified hospitalist, and new onset cardiomyopathy with severely reduced ejection fraction was identified. Video 1&2 [https://scholarlycommons.gbmc.org/cgi/editor.cgi?article=1261&window=additional_files&context=jchimp]: Depicts the POCUS findings of the patient. Ejection fraction was assessed using end-point septal separation and fractional shortening methods.

Legend: POCUS demonstrating Heart failure with reduced ejection fraction (HfrEF) with poor left ventricle contractility shown by the movement of the ventricular wall.

On a prior echocardiogram, obtained a month before admission, the LVEF was 57%. Cardiology was consulted and they recommended discontinuing home metoprolol, avoiding beta blockers and calcium channel blockers for atrial tachycardia due to new onset cardiomyopathy causing acute decompensated heart failure. A comprehensive echocardiogram the next morning confirmed HFrEF, by estimating LVEF to be around 25% using Simpson's biplane method. The cardiologist initiated IV amiodarone 150 mg/10min bolus, 1 mg/min for 6 h, 0.5 mg/min for 18 h, 400 mg daily for 4 days for the atrial arrhythmia and IV furosemide for symptomatic HFrEF. The cardiology consultant described the cardiomyopathy as non-ischemic and that it was potentially transient, due to SARS-CoV-2 infection and/or tachyarrhythmia and did not recommend cardiac catheterization.

Over the next few days, as the patient improved, she was started on low-dose carvedilol as part of goal-direct therapy for new cardiomyopathy, transitioned to oral amiodarone (100 mg daily) and furosemide, and discharged with a plan for outpatient cardiology follow-up. At the 2-week cardiology visit, she reported no ongoing palpitations, and amiodarone was discontinued. At 4 months follow-up, a repeat echocardiogram showed a normal LVEF of 60%.

3. Discussion

It is not uncommon for patients with acute respiratory infections, especially SARS-CoV-2 to present with new-onset cardiac arrhythmias. Studies have shown that in SARS-CoV-2 infection, 10% of patients develop new-onset arrhythmia, the most common being atrial fibrillation, and 23–33% have shown recurrence of a pre-existing arrhythmia.[6] Possible mechanisms for the development of arrhythmias in the setting of acute respiratory illness include hypoxia triggering myocardial cell automaticity, direct myocardial cell injury, or myocarditis.[7-10]

Multiple studies and case reports have reported new onset stress-induced cardiomyopathy related to SARS-CoV-2 infection, with an estimated incidence of 2–4%.[11-13] In contrast, the incidence of stress-induced cardiomyopathy in patients presenting with suspected acute coronary syndrome is estimated to be around 1–2%.[14,15]

In this case, the patient presented with both new-onset arrhythmia and cardiomyopathy. There is ample evidence indicating that tachyarrhythmias alone can induce reversible cardiomyopathy, leading to the term tachycardia induced cardiomyopathy.[16-18] The pathophysiology of tachyarrhythmia-induced cardiomyopathy has been mainly studied in animal models.[16,18] Animal studies show increased wall dilation, greater wall tension, diminished cardiac output, and cardiac chamber enlargement within days to weeks of persistent tachycardia.[19,20] In animal models, once tachycardia has resolved, the cardiomyopathy resolves in days to weeks.[21] Human studies have shown the resolution of tachycardia-induced LV dysfunction within a few months.[22-24]

In addition, the patient was on chemotherapy (bendamustine) 8 months prior to admission which could have contributed to new onset cardiomyopathy, we assess that this is less likely as the patient had normal ejection fraction one month prior to admission. It is unclear if the new onset cardiomyopathy in our patient was related to the SARS-CoV-2 infection itself, underlying tachyarrhythmia, or due to history of bendamustine therapy. Regardless of the etiology, this case illustrates the need to consider HFrEF in patients with atrial and sinus tachycardia. Administering beta blockers or calcium channel blockers for atrial arrhythmias in a patient with acute decompensated HfrEF is contraindicated.

With the advent of Point of Care Ultrasound (POCUS), assessing gross left ventricular function quickly at the bedside by non cardiologists has become possible. There is substantial evidence showing the effectiveness of POCUS in diagnosing LV dysfunction among non-cardiologists (Hospitalists and Emergency department physicians).[25-27] In this case, POCUS led to an unexpected diagnosis of HfrEF and a change in therapy.

4. Conclusion

Our case shows that bedside POCUS in patients with tachyarrhythmia can diagnose underlying HfrEF and lead to timely changes in management. With the advent of POCUS training among Hospital Medicine clinicians, routine bedside POCUS to assess cardiac function in patients with new tachyarrhythmia might be something to consider in the future.

Consent

The consent of the patient was obtained for writing and publishing these findings.

Ethical approval

N/A.

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Conflict of interest

All authors declare no conflict of interest.

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