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Atrial Arrhythmias in a Patient Presenting With Coronavirus Disease-2019 (COVID-19) Infection

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Abstract

The coronavirus disease-2019 (COVID-19) is an infectious disease caused by severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) that has significant potential cardiovascular implications for patients. These include myocarditis, acute coronary syndromes, cardiac arrhythmias, cardiomyopathies with heart failure and cardiogenic shock, and venous thromboembolic events. We describe a Caribbean-Black gentleman with COVID-19 infection presenting with atrial arrhythmias, namely, atrial flutter and atrial fibrillation, which resolved with rate and rhythm control strategies, and supportive care.

Keywords

atrial arrhythmias, atrial flutter, atrial fibrillation, severe acute respiratory syndrome coronavirus 2, SARS-CoV-2, coronavirus disease 2019, COVID-19

Introduction

Coronavirus disease-2019 (COVID-19) is an infectious disease caused by severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2).[1,2] The disease was first identified in 2019 in Wuhan, China, and has since spread globally, resulting in the 2019-2020 coronavirus pandemic.[1]

The World Health Organization declared the 2019-2020 coronavirus outbreak a Public Health Emergency of International Concern on January 30, 2020, and a pandemic on March 11, 2020.[3,4]

COVID-19 may have deleterious effects on the cardiovascular (CV) system, and patients with preexisting CV disease. Several recent Chinese studies have since demonstrated the sequelae of CV events.[5-7] As the pandemic evolves, the emerging literature on CV outcomes are not well characterized, but likely encompass acute coronary syndromes, myocarditis, cardiomyopathies, cardiogenic shock, lethal arrhythmias, and sudden cardiac death.

We describe a case of a middle-aged Caribbean-Black gentleman presenting with COVID-19 infection who experienced atrial arrhythmias, namely, atrial flutter (AFL) and atrial fibrillation (AF), which resolved with rate and rhythm control strategies, and supportive care.

Case Report

A 46-year-old Caribbean-Black male with no significant medical history presented to the emergency department (San Fernando General Hospital, Trinidad) with a symptom complex of fever, cough, and shortness of breath over the preceding 2 days. His vital signs indicated systolic blood pressures of 140 mm Hg, heart rate of 142 beats per minute, and respiratory rate of 28 breaths per minute with an oxygen saturation of 88% on room air. Apart from hypertension, tachycardia, and tachypnea, his physical examination revealed a normal jugular

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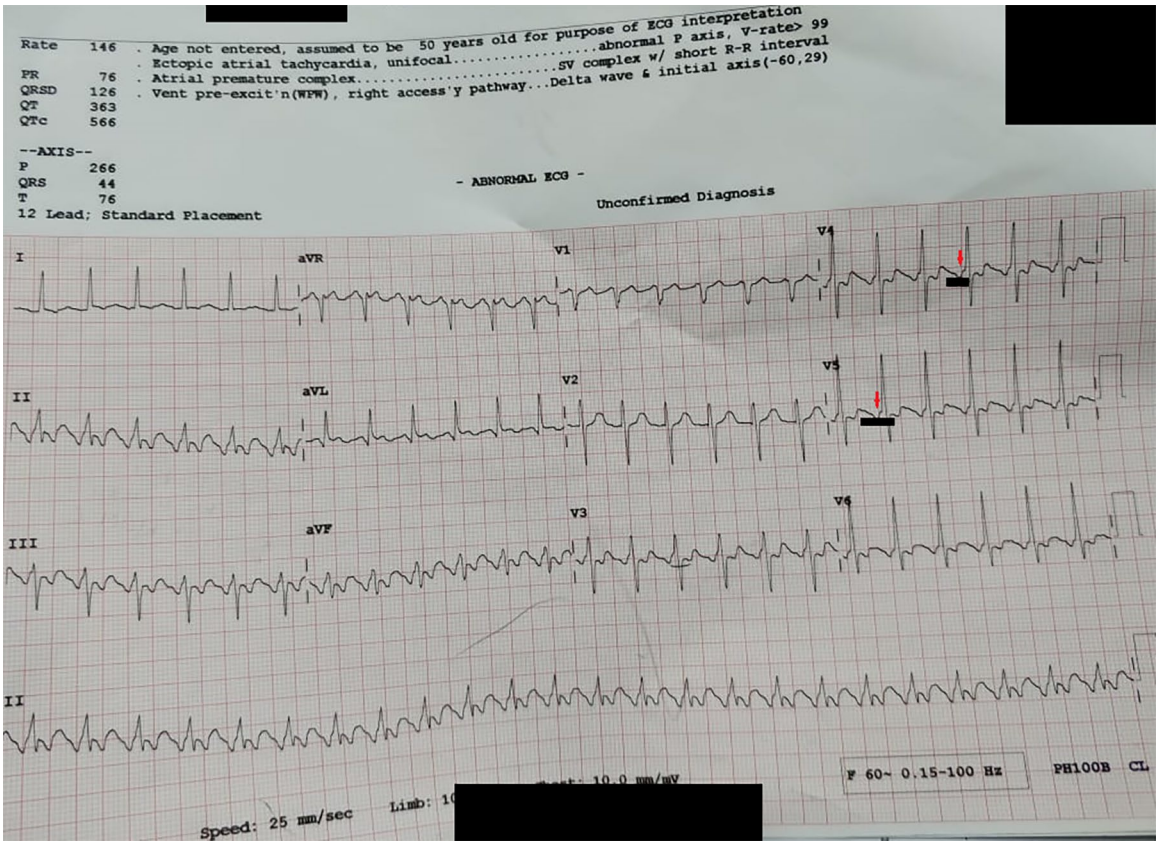

Figure 1. The patient's electrocardiogram in which the red arrows indicate the typical flutter waves (f-waves) that occur right before the QRS complex, simulating a pseudo-preexcitation pattern. The segment underscored in black indicates the f-waves in series at a rate of approximately 240 beats per minute. The QRS complexes are occurring at 120 to 140 beats per minute, hence the 2:1 atrioventricular block.

venous pulse, scattered bilateral crackles, and no peripheral edema.

A 12-lead electrocardiogram revealed typical AFL with a 2 to 1 atrioventricular block and rate-related ST-T segment changes (see Figure 1).

A chest radiograph did not reveal any acute cardiopulmonary disease (see Figure 2), while a bedside 2-dimensional transthoracic echocardiogram demonstrated a preserved left ventricular ejection fraction, without any regional wall motion abnormalities. Pertinent diagnostic laboratory investigations included a D-dimer 357 ng/dL (normal ≤500 ng/mL), pro-brain natriuretic peptide 413 pg/mL (normal ≤300 pg/mL), cardiac biomarkers, CK-MB 15 U/L (normal <20 U/L), and troponin I 0.12 ng/mL (normal 0.0-0.15 ng/mL). Other routine investigations are indicated in Table 1. The patient's arterial blood gas was consistent with mild hypoxia on 24% fractional inspiration of oxygen with an estimated alveolar-arterial gradient of 17 mm Hg.

In the designated isolation room, he was initiated on an amiodarone and digoxin bolus, moderate-intensity beta-blockade, and subsequently admitted for further hospitalization (Table 2). Despite these therapies, the patient unsuccessfully underwent

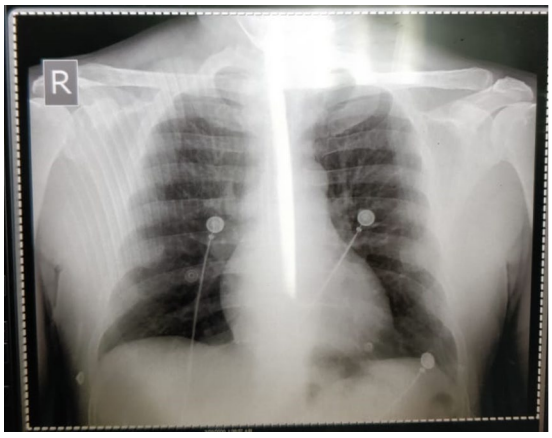

Figure 2. The patient's chest radiograph does not indicate any airspace disease that would be expected in coronavirus-2019 (COVID-19) infection.

cardioversion with 100 J and subsequently transitioned to atrial fibrillation with rapid ventricular (AF RVR) response (see Figure 3). In the interim, the patient's COVID-19 test (Centers

Table 1. Routine Investigations.

Tests Performed	Result	Reference Range
Hemoglobin	9.4 g/dL	14.0-17.5 g/dL
White blood cell count	13.2 $\times$ 10^9/L	4.5-11.0 $\times$ 10^9/L
Platelet count	201 $\times$ 10^3/μL	156-373 $\times$ 10^3/μL
Serum sodium	134 mmol/L	135-145 mmol/L
Serum potassium	2.8 mmol/L	3.5-5.1 mmol/L
Serum bicarbonate	22 mmol/L	22-26 mmol/L
Serum creatinine	0.5 mg/dL	0.5-1.2 mg/dL
Serum osmolality	283 mOsm/kg	275-295 mOsm/kg
Blood urea nitrogen	8 mg/dL	3-20 mg/dL
Fasting blood sugar	116 mg/dL	60-120 mg/dL
Alanine aminotransferase	26 IU/L	20-60 IU/L
Aspartate aminotransferase	68 IU/L	5-40 IU/L
Total bilirubin	2.2 mg/dL	0.2-1.2 mg/dL
Alkaline phosphatase	101 IU/L	40-129 IU/L
Albumin	2.7 g/dL	3.5-5.5 g/dL
Albumin-corrected calcium	7.3 mg/dL	9.6-11.2 mg/dL
Magnesium	1.6 mg/dL	1.6-2.3 mg/dL
Phosphorous	2.3 mg/dL	2.5-6.5 mg/dL
Serum cortisol level	18.3 μg/dL	10-20 μg/dL
Thyroid-stimulating hormone	1.44 mU/L	0.5-5.0 mU/L
Urine osmolality	534 mOsm/kg	300-900 mOsm/kg
Urine sodium	$<$ 20 mEq/L	40-220 mEq/L
Erythrocyte sedimentation rate	68 mm/h	0-22 mm/h
High-sensitivity C-reactive protein	83 mg/dL	0.0-1.0 mg/dL
D-dimer	357 ng/mL	$<$500 ng/mL
pro-brain natriuretic peptide	413 pg/mL	$\leq$300 pg/mL
Creatine kinase	873 U/L	30-170 U/L
Creatine kinase MB	15 U/L	$<$20 U/L
Lactate dehydrogenase	1717 U/L	313-618 U/L
High-sensitivity troponin I	0.12 ng/mL	0.0-0.15 ng/mL
Blood cultures	Negative	Positive or negative
Urine culture	Negative	Positive or negative

for Disease Control and Prevention's 2019-nCoV Real-Time RT-PCR Diagnostic Panel, Atlanta, GA) returned positive, and he was transferred to another quarantine facility (Couva Medical and Multi-Training Facility, Trinidad) with intensive care unit (ICU) capabilities for further management.

During the ensuing hospitalization, he was continued on an amiodarone infusion at 1 milligram per minute and atenolol, and his symptoms gradually ameliorated with decreasing oxygen requirements. He reverted to normal sinus rhythm within 48 hours, and as a result, anticoagulation was deferred in light of both CHADS-VASc and HAS-BLED scores of 0 each. The remainder of his hospital course was uneventful, and he was subsequently discharged to home quarantine on oral low dose, twice daily amiodarone with a follow-up visit, and 1-week Holter monitor in 1 month.

Discussion

It has been recently reported that CV compromise is a common complication of patients who are hospitalized with COVID-19 infection and is associated with a higher risk of mortality.[15] Cardiac arrhythmias are also frequent clinical manifestations; however, there is a paucity in the emerging literature with regard to the nature and classification of these arrhythmogenic events.

In a recent series, comprising nearly 148 patients, almost one tenth reported palpitations.[16] In another recent similarly sized study, arrhythmia was noted in almost one sixth of the patients and frequently occurred within the ICU subgroup of patients with almost half being affected.[17] Despite these emerging studies, the characteristics of these arrhythmias are not yet published nor previously described. The development of potentially lethal arrhythmias, especially in the setting of elevated cardiac biomarkers, should herald myocarditis as a differential diagnosis.[18,19] The Surviving Sepsis Campaign: Guidelines on the Management of Critically Ill Adults with COVID-19 do not currently include guidelines with regard to specific arrhythmia management.[20]

Arrhythmias are complex and multifactorial in a COVID-19 patient and may result from metabolic derangements, hypoxia, acidosis, intravascular volume imbalances, neurohormonal,

Table 2. The Patient's Individualized Cardiovascular Medicine Regimen for Coronavirus-2019 (COVID-19) Infection and Rationale.

Drug	Dose	Rationale
Direct oral anticoagulation (DOAC)	Not utilized	DOAC was not instituted as the patient was in paroxysmal atrial fibrillation with a CHADS-VASc and HAS-BLED score of 0. The patient was discharged to self-quarantine with an outpatient 1-week Holter monitor prior to the follow-up appointment.
Atenolol	50 mg every 8 hours	A lenient rate control strategy with this β-blocker was adopted with the significant advantages being relatively cardioselective and minimal interactions given the patient's normal renal function.[8]
Amiodarone	200 mg every 12 hours	Oral amiodarone after a 48-hour infusion was used synergistically as a rhythm control strategy in addition to a rate control strategy. As the patient's chest radiograph was normal, it was initiated with increased vigilance for any pneumonitis that could potentially complicate COVID-19 infection.[9,10]
Digoxin	Not utilized	This drug was discontinued after the initial loading dose.[11]
Hydroxychloroquine	Not utilized	This was considered, however, ultimately not utilized after a detailed risk-benefit analysis. There was a major concern about its adverse effect profile, including QT prolongation and drug-drug interactions.
Azithromycin	Not utilized	This antibiotic, while displaying therapeutic synergy with hydroxychloroquine was deferred due to its arrhythmogenic effects from QT prolongation.[12,13]
Lopinavir-Ritonavir	Not utilized	This antiretroviral combination was not utilized due to drug-drug interactions and lack of clinical effectiveness in a recent trial.[14]

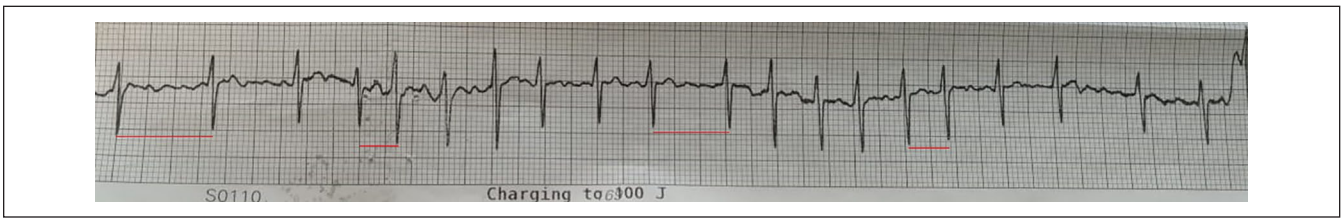

Figure 3. The patient's rhythm strip post-cardioversion, which indicates coarse atrial fibrillation with a rapid ventricular response. The variable RR intervals highlighted by the interspersed red lines.

and catecholaminergic stress.[21,22] Sepsis is characterized by a systemic milieu involving inflammatory cytokines and autonomic dysfunction.[23] This maladaptive pathophysiology is a significant trigger for the development of AF, as was illustrated in this patient.[24] This likely occurred in our patient as he initially presented with AFL with 2 to 1 atrioventricular block and transitioned to AF with rapid ventricular response in the setting of COVID-19 infection. AF is a common sequela of critical illness, with an estimated prevalence of almost 10% in ICU patients, and several studies report worse outcomes in patients with new-onset AF as compared with their non-AF counterparts.[25,26] Sinus rhythm restoration is of high priority as it improves the patient's hemodynamics. AF may attenuate cardiac output due to impaired left ventricular filling, especially with rapid ventricular response.[22,27] Presently, there are no evidence-based guidelines for the use of anticoagulant prophylaxis in these patients.[28]

Additionally, severe infection induces the sympathetic nervous system (SNS), and there is also a relationship between SNS activity and supraventricular tachyarrhythmia.[29] Tachycardia, in itself, is an independent prognosticator or mortality in patients with sepsis.[30] Postulated mechanisms of this arrhythmogenesis include SNS-induced calcium entry into cardiac myocytes as well as a spontaneous release of calcium from the sarcoplasmic reticulum.[31,32] Our patient illustrated several of the above electrolyte abnormalities, including hypokalemia, hypomagnesemia, and hypophosphatemia, all of which were aggressively repleted. In some cases, it is observed that tachycardia continues despite adequate volume resuscitation.[33] Our patient also displayed anemia with mild rhabdomyolysis, which was managed with judicious intravenous crystalloid hydration.

Conclusion

We describe a case of a middle-aged Caribbean-Black gentleman presenting with COVID-19 who experienced atrial arrhythmias, namely, AFL and AF, which resolved with rate and rhythm control strategies, and supportive care. Further observational studies are required to characterize the nature and classification of arrhythmias in this COVID-19 pandemic.

Authors' Note

All available data can be obtained by contacting the corresponding author.

Author Contributions

RS, RN, SG, MR, KF, KA, NB, and NAS all contributed equally in writing the manuscript. All authors read and approved the final manuscript.

Declaration of Conflicting Interests

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Ethics Approval

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

Informed Consent

The patient has provided verbal informed consent to have the details of his case published.

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