



Management of Arrhythmias Associated with COVID-19

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Abstract

Purpose of Review Cardiac arrhythmias are known complications in patients with COVID-19 infection that may persist even after recovery from infection. A review of the spectrum of cardiac arrhythmias due to COVID-19 infection and current guidelines and assessment of risk and benefit of management considerations is necessary as the population of patients infected and recovering from COVID-19 continues to grow.

Recent Findings Cardiac arrhythmias such as atrial fibrillation, supraventricular tachycardia, complete heart block, and ventricular tachycardia occur in patients infected, recovering and recovered from COVID-19.

Summary Personalized care while balancing risk/benefit of medical or invasive therapy is necessary to improve care of patients with arrhythmias. Providers must provide thorough follow-up care and use necessary precaution while caring for COVID-19 patients.

Keywords Sars-CoV-2 · COVID-19 · Cardiac arrhythmias

Introduction

SARS-CoV-2, the severe acute respiratory syndrome coronavirus 2, responsible for COVID-19, has infected over 37 million people globally and almost 8 million people in the USA alone [1]. While the primary symptoms of COVID-19 may be respiratory in nature for the vast majority of cases, several studies have pointed to extrapulmonary effects of the virus [2]. This phenomenon is likely observed due to the cumulative effects of the hyperinflammatory response of the body and the omnipresence in major organs of the angiotensin-converting

enzyme 2 (ACE2) cellular receptor that SARS-CoV-2 uses for cell entry [3]. Of all systems which may be affected by the virus, possibly the most common extrapulmonary complications can be observed in the cardiovascular system, with these complications including myocardial injury, cardiomyopathy, acute coronary syndrome, cardiogenic shock, acute cor pulmonale, thrombotic complications, and arrhythmias [4, 5].

Herein, we describe the signs, symptoms, and pathophysiology of cardiac arrhythmia in COVID-19 (Table 1). SARS-CoV-2 infection may cause deleterious cardiovascular effects manifested as cardiac enzyme release and a heightened systemic inflammatory response, which has noted to include elevations in ferritin, lactate dehydrogenase (LDH), C-reactive protein (CRP), and interleukin-6 (IL-6) [6]. The mechanism of myocardial injury may be secondary to the immune response, elevated catecholamine, hypercoagulable state, and/or directly due to myocyte viral invasion [7, 8]. Indeed, post-mortem pathological studies have discovered myocardial tissue positive for SARS-CoV-2 by reverse transcription polymerase chain reaction (RT-PCR) and electron microscopy [9]. However, there have been no detailed reports to date of post-mortem virus that have evaluated if those patients who died of a cardiac arrhythmia had viral infection in the cardiac conduction tissue such as the His-Purkinje system. The high incidence of arrhythmias in COVID-19 is thought to be multifactorial: hypoxemia due to acute respiratory distress, acute decline in cardiac hemodynamics, myocarditis, cardiac injury, prominent inflammatory

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Table 1 Symptoms, clinical findings, and pathophysiology of cardiac arrhythmias during and after COVID-19 infection

Arrhythmia	Symptoms and clinical findings	Pathophysiology
AF/AFL	<ul style="list-style-type: none"> • Palpitations • Dizziness • Chest discomfort • Fatigue • Stroke/thromboembolism • Heart failure 	<ul style="list-style-type: none"> • Systemic inflammation • Worsening of pre-existing cardiovascular disease
SVT	<ul style="list-style-type: none"> • Palpitations • Dizziness • Chest discomfort • Fatigue 	<ul style="list-style-type: none"> • Systemic inflammation • Worsening of pre-existing cardiovascular disease
VT/VF	<ul style="list-style-type: none"> • Syncope • Sudden cardiac death 	<ul style="list-style-type: none"> • Myocarditis • Acute myocardial infarction • Systemic inflammation • Worsening of pre-existing cardiovascular disease • QT prolongation
AV Block	<ul style="list-style-type: none"> • Dizziness • Fatigue • Complete heart block • Asystole 	<ul style="list-style-type: none"> • Unclear if reversible • Unclear if due to inflammation, or direct injury of AV node or His-Purkinje system, or worsening of pre-existing conduction disease
POTS/IAST	<ul style="list-style-type: none"> • Palpitations • Dizziness • Fatigue • Tachycardia at rest and worse with activity 	<ul style="list-style-type: none"> • Autonomic dysfunction
QTc prolongation	<ul style="list-style-type: none"> • May lead to TDP 	<ul style="list-style-type: none"> • QT prolonging medications • Myocardial injury • Structural heart disease, • Electrolyte disturbance • Renal dysfunction

response, direct viral invasion, and/or use of QT prolonging medications [10–12].

Early reports from Wuhan showed that 44% of patients admitted to the intensive care unit with COVID-19 had arrhythmias, raising initial suspicions of arrhythmias associated with COVID-19 [13]. We discuss in this manuscript a perspective on the arrhythmia associated with COVID-19 and their management considerations (Fig. 1 and Table 2).

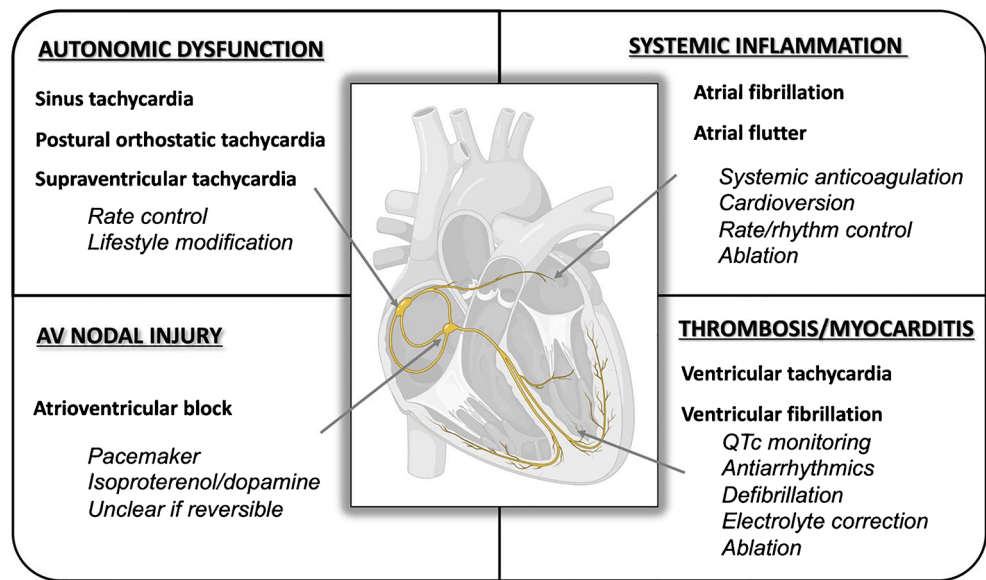
Atrial Arrhythmias

Atrial arrhythmias are the most commonly reported arrhythmias in patients with COVID-19. Similarly, atrial fibrillation (AF)-

related consultations were the most common electrophysiology consultations during the peak of the pandemic in New York City at Columbia University for COVID-positive patients (31%), with only 13% of these COVID-positive patients having a history of AF [14]. In stark contrast with a typical AF population, none of these COVID-19 patients with new-onset atrial tachyarrhythmia had a history of cardiac surgery, ablation, cardioversion, or antiarrhythmic drug-use. The etiology of these atrial arrhythmias is yet to be fully understood, but proposed theories include alterations in ACE2-related signaling pathways, inflammation, direct viral endothelial damage, and metabolic derangements during the acute illness [15].

Notably, AF has been associated with worse outcomes in patients who have acute respiratory disease. Prior to the

Fig. 1 Cardiac arrhythmias during and after COVID-19 infection. This figure illustrates the effects of COVID-19 infection on the cardiac conduction system, the spectrum of cardiac arrhythmia, and possible management considerations



COVID-19 era, patients with new-onset AF during acute respiratory syndrome (ARDS) and severe pneumonia had increased mortality compared to those ARDS patients without new AF [16]. In Columbia University’s aforementioned consultation experience, 55% of patients in the small sample of nine expired by the end of the study, with the remaining hospitalized, reflecting the poor prognosis of patients with new onset of AF during COVID-19 infection [14]. In another study, atrial arrhythmias were also more common among patients who needed mechanical ventilation (17.7% vs. 1.9% otherwise) [17]. These studies highlight the importance of careful management considerations for these patients, especially those with new-onset AF.

AF is typically treated with rate or rhythm control and also with anticoagulation in patients who meet criteria and do not have contraindications due to bleeding risk [18]. For rate control, pharmacological management with diltiazem in patients with COVID-19 seemed to be common since there is concern that beta-blockers may cause bronchospasm during respiratory illness [19] (Table 2). In COVID-19 patients with new-onset AF, Columbia University’s study reported the use of amiodarone in 29% of patients referred for electrophysiology consultation, with anticoagulant usage reported in 83% [14]. It is unclear if early detection and treatment of COVID-19 may mitigate such arrhythmic cardiac complications. Furthermore, it is unclear if prophylactic use of antiarrhythmics in COVID-positive patients at risk of cardiac complications should be considered earlier on in the treatment, due to the increase in mortality in patients with arrhythmia [20].

Ventricular Arrhythmias

In contrast to atrial arrhythmias consisting of 31% of electrophysiology consults at the peak of the pandemic, ventricular

arrhythmias made up a significantly smaller 7% of consults at the Columbia University Medical Center [14]. Structural heart disease has historically been shown to be a risk factor for ventricular arrhythmias [21]. We have previously reported ventricular tachycardia (VT) and ventricular fibrillation (VF) as the primary cause of death in COVID-19 patients without a prior history of structural heart disease [22]. However, this could be due to the association of ventricular arrhythmias in patients who are critically ill [23].

Several studies investigating cardiac arrhythmia most proximate to death for patients infected with COVID-19 describe VT and VF in 6% of deaths [24, 25]. Notably, the majority of COVID-19 deaths had asystole or pulseless electric activity (PEA) at time of death, likely due to respiratory failure or pulmonary embolism [21]. VT/VF may also be due to cardiac ischemia or acute myocardial infarction as there have been studies suggesting increased thromboembolism in COVID-19 [26–28]. Furthermore, patients with myocarditis due to COVID-19 may have increased risk for sudden cardiac death although it is unclear how to risk stratify these patients [27–29].

The usage of antiarrhythmics for prophylaxis of VT/VF for patients during COVID infection is unclear, especially since it is still not known why some patient have cardiac arrhythmic complications and others do not. Usage of QT prolonging drugs, such as amiodarone, presents a possible risk of torsades de pointes (TDP) and nodal agents may cause bradycardia (Table 2) [30•].

VT Storm

Ventricular arrhythmias that arise in COVID-19 patients may lead to VT storm defined as 3 or more hemodynamically stable ventricular tachyarrhythmias in 24 h, VT recurring after

Table 2 Management of cardiac arrhythmias in patients with and without COVID-19 infection

Standard-of-care management in patients without COVID-19	Management considerations in setting of COVID-19 infection
Atrial tachyarrhythmias	
Atrial fibrillation [18]	
<ul style="list-style-type: none"> • First-line rate control consists of beta-blockers and/or non-dihydropyridine calcium channel blockers. • In hypotensive patients, amiodarone may be used. • Cardioversion can be used in hemodynamically unstable atrial fibrillation. • Rhythm control may be preferred, especially in symptomatic patients. Antiarrhythmic drugs (most commonly flecainide, dofetilide, propafenone, ibutilide, and amiodarone) may be used as pre-treatment for electric cardioversion, for pharmacologic cardioversion, or for chronic maintenance of sinus rhythm. • Catheter ablation may be chosen after failure/intolerance of drugs, or initially by preference. Surgical ablation may be considered in patients with other indications for cardiothoracic surgery. • For prevention of thromboembolism, anticoagulation is indicated peri-procedurally for cardioversion and ablation, as well as long-term for CHA₂DS₂-VAsC score ≥ 2 in men or ≥ 3 in women. In patients at high risk for bleeding with long-term anticoagulation, occlusion or exclusion of left atrial appendage may be considered. 	<ul style="list-style-type: none"> • Patients with underlying restrictive pulmonary disease or chronic obstructive pulmonary disease should be cautioned for bronchospasm while on beta-blocker therapy. • Caution should be used for amiodarone in patients with decreased pulmonary function and/or fibrotic lung disease after COVID-19. • During aerosolizing procedures such as intubation or transesophageal echocardiogram, healthcare providers should don appropriate personal protective equipment. • Cardiac computed tomography may be considered as an alternative imaging modality for evaluation of thrombus prior to cardioversion if patient is actively infected with COVID-19. [83, 84] • Given the high rates of thrombotic complications in COVID-19 [4••], the indications and duration of anticoagulation are unclear.
Inappropriate sinus tachycardia	
<ul style="list-style-type: none"> • Reassurance and lifestyle interventions including exercise and avoidance of cardiac stimulants. • Beta-blockers and/or ivabradine may be used in symptomatic patients [48]. • Sinus node ablation may be considered in refractory cases. 	<ul style="list-style-type: none"> • As in the general population, other etiologies of sinus tachycardia must be excluded. Pulmonary embolism should be considered in patients with unexplained sinus tachycardia • Outpatient Holter or event monitor may be helpful for diagnosis. • Beta-blockers may be considered. • It is unclear whether ivabradine may be useful.
Postural orthostatic tachycardia syndrome	
<ul style="list-style-type: none"> • Initial management includes consumption of 2–3 L/day of water and 10–12 g/day of sodium, as well as regular and progressive exercise. • Fludrocortisone may be used to aid sodium and water retention. • Midodrine or pyridostigmine may be considered. • Low-dose propranolol or ivabradine may be considered [54]. 	<ul style="list-style-type: none"> • Tilt-table testing may be considered for evaluation for POTS. • Patients with COVID-19 and evidence of myocardial injury or inflammation should defer competitive sports or aerobic exercise until 3–6 months following infection and resolution of imaging findings and normalization of troponin [55, 56••].
Other supraventricular tachycardia	
<ul style="list-style-type: none"> • Cardioversion is indicated in unstable patients. • Vagal maneuvers may abort episodes of AVRT/AVNRT. • Adenosine may be used for abortion or to slow rhythm and aid diagnosis. • Management varies depending on specific arrhythmia. Therapies commonly include beta-blockers and non-dihydropyridine calcium channel blockers, among other antiarrhythmic drugs. • Catheter ablation may be efficacious. • Electrophysiologic studies may be used for diagnosis or to guide therapy. 	<ul style="list-style-type: none"> • Anticoagulation is generally not indicated unless atrial fibrillation is also present, but in patients with COVID-19, further study may be needed. • Catheter ablation may be considered. • Outpatient Holter or event monitor may be helpful for diagnosis.
Atrioventricular block	
<ul style="list-style-type: none"> • Avoidance of AV nodal blockade is prudent in all types. • First-degree AV block generally does not require management. • For second and third degree, stabilization (e.g., with atropine or transvenous pacing) and evaluation for reversible causes is the first step. • Permanent pacemaker is indicated if symptomatic, or in those with second degree type II or third degree blocks. 	<ul style="list-style-type: none"> • Pacemaker placement for complete heart block, symptomatic bradycardia, and high-degree AV block.
Ventricular arrhythmias	
<ul style="list-style-type: none"> • Beta-blockers and/or antiarrhythmics. • ICDs for primary and secondary prevention. • Magnesium, isoproterenol or ventricular pacing should be considered in TDP. • Catheter ablation. 	<ul style="list-style-type: none"> • The necessity of secondary prevention ICDs is unclear, as patients with COVID-19 and ventricular arrhythmias may have no evidence of structural heart disease [22], and acute infection may be considered a reversible precipitant.
Electrical storm	
<ul style="list-style-type: none"> • If unstable, patients should be treated with defibrillation. • Initial therapy consists of both intravenous antiarrhythmic agents (generally amiodarone; but procainamide, flecainide, or lidocaine is also used) and beta-blockers. • Urgent coronary revascularization is indicated in patients with active myocardial ischemia. • Urgent catheter ablation is indicated in medically refractory cases or in scar-related disease. • Antiarrhythmic therapy may be continued long term, especially in patients who do not undergo ablation. 	<ul style="list-style-type: none"> • In patients who do not undergo catheter ablation, the risk of recurrent arrhythmias after resolution of COVID-19 is not known.

termination of another episode, or sustained and nonsustained VT episodes exceeding normal beats within 24 h. Antiarrhythmic drug therapy may be effective using amiodarone and beta-blockers [31] but may require deep sedation and hemodynamic support as well [32]. For COVID-19 patients suffering from a VT storm, case studies of patients with new-onset ventricular arrhythmias have also shown the efficacy of substrate-based VT catheter ablation procedures if implantable cardioverter defibrillator (ICD) shocks prove futile. These case studies report patient recovery from COVID-19 without further ICD interventions [33]. While the sample size is small, these ablation case studies may offer a promising alternative to the difficulties in applying proper drug-therapies to COVID-19 patients. Possible long-term effects of aggressive ablation strategy in a COVID-19 specific population are yet to be observed.

Atrioventricular Block

Atrioventricular (AV) block may account for up to 12% of arrhythmias seen in patients with COVID-19 [34]. While the mechanism behind this observation is not certain, heart block can be a manifestation of myocarditis [35], and myocarditis has been associated with the infection [8]. Although several cases of COVID-19-related AV block have been reported in patients with preserved ventricular function and/or normal cardiac biomarkers [36–38], it is possible that these cases otherwise represent subclinical myocardial inflammation. In one such case, cardiac magnetic resonance imaging (MRI) revealed ventricular wall edema suggestive of myocarditis despite no evidence of myocardial injury [36]. Moreover, AV block seen in the setting of acute infection can resolve spontaneously [39, 40]. Patients with COVID-19 and persistent high-grade AV block have been managed with standard-of-care pacemaker placement and outpatient follow-up (Table 2) [36–38]. However, the development of heart block in patients with COVID-19 has been suggested to be a poor prognostic sign, with many of the reported cases occurring in patients who ultimately succumbed to the disease [40–43].

Inappropriate Sinus Tachycardia

The incidence of inappropriate sinus tachycardia (IST) in patients with COVID-19 is uncertain. By definition, IST is a diagnosis of exclusion. Therefore, it is very unlikely to be diagnosed in the setting of acute infection as patients with hypoxemia may be in sinus tachycardia. Persistent tachycardia after infection may represent as IST and has been shown in patients recovering from SARS, suggesting it may be seen in patients recovering from COVID-19 as well [44, 45]. The mechanism of IST is likely multifactorial including intrinsic

sinus node hyperactivity, autonomic dysfunction, and a hyperadrenergic state [46•]. Inflammatory cytokines released by patients with COVID-19 may affect the function of myocardial ion channels and perpetuate tachyarrhythmia including sinus tachycardia [47]. Ongoing symptomatic IST may be treated with beta-blockers and/or ivabradine (Table 2) [48], although treatment efficacy is unknown in patients with COVID-19. It is of note that ivabradine usage in IST is not FDA approved and is off label.

Postural Orthostatic Tachycardia Syndrome

Postural orthostatic tachycardia syndrome (POTS) is caused by autonomic dysfunction; the underlying mechanism of which may be related to peripheral neuropathy, increased serum norepinephrine, baroreceptor dysfunction, or hypovolemia [49, 50]. The syndrome has previously been reported to develop after acute stressors including viral illness and, therefore, may also develop in some patients recovering from COVID-19 [49–51]. Furthermore, dysautonomia including postural tachycardia have already been reported in COVID-19 patients [52, 53]. Nonpharmacologic management of POTS includes increasing salt and fluid intake, use of lower extremity compression garments to reduce venous pooling, and participation in regular exercise to prevent deconditioning [54]. However, patients with COVID-19 and evidence of myocardial injury or inflammation should abstain from competitive sports or aerobic activity until resolution of imaging findings or normalization of cardiac biomarkers [55, 56••]. When these measures are ineffective, various pharmacologic therapies may be attempted depending on the specific etiology suspected. These therapies include fludrocortisone, midodrine, pyridostigmine, propranolol, ivabradine, and alpha-2 agonists [54]. Ivabradine prescription for treatment of POTS is not FDA approved and is off label. Patients with COVID-19 may benefit from low-dose propranolol for lowering heart rate and reducing adrenergic activity (Table 2) [57].

Pediatric Considerations

While immediate treatment-requiring arrhythmias in children are often extremely rare, pediatric patients most commonly report supraventricular tachycardias. The latter are often carefully treated with a combination of both pharmacotherapy such as amiodarone, ibutilide, beta-blockers, or cardioversion [58]. The American Heart Association released guidelines in collaboration with the American Academy of Pediatrics as to treatment of children and infants with COVID-19. Within these guidelines, they suggest for pediatric patients suffering from life-threatening arrhythmias, early usage of endotracheal tubes/ventilation, and defibrillation [59]. That said, apart from

COVID-19 case studies which primarily report increased risk for pediatric patients with prior history of cardiac surgery, limited data are available for children with COVID-19 at this time of writing [60].

Myocarditis

Myocarditis is a leading cause for the development of ventricular arrhythmias [14]. Myocarditis has also been presented as the most probable cause of myocardial injury and has been observed in 7.2–27.8% of COVID-19 patients [5]. While it may not be possible to fully rule out prior silent ischemia in these patients, myocarditis may be a more likely culprit, due to direct viral infection of cardiomyocytes, hypoxia, or hyperimmune response [22]. Autopsies on COVID-19 patients with myocarditis have shown direct invasion by the virus and inflammatory processes in the tissue without any presence of COVID-19 [61, 62]. Although the exact etiology of myocarditis remains in question, management of myocarditis for COVID-19 patients has shown relatively favorable outcomes in patients treated with glucocorticoid therapies [63]. Furthermore, in accordance with possibilities of hyperimmune response and hypoxia, second-line agents such as IL-6 inhibitors and intravenous immunoglobulin (IVIG) were often administered with or without vasopressor support [63]. Large-scale studies have yet to show the efficacy of glucocorticoid therapies on the heart for COVID-19 patients and their use remains controversial, with several studies suggesting these therapies have no or a harmful effect on patients [64]. Optimal dosing guided by ARDS sub-phenotypes, biomarkers, and comorbidities should be utilized before administration of glucocorticoids for COVID-19 patients with myocarditis [65].

Cardiac Arrhythmias During and After Recovery

Given the recent onset of the COVID-19 pandemic, data on long-term cardiovascular outcomes in patients who have recovered are lacking. Patients who developed overt cardiac disease should receive regular monitoring and reassessment. For those diagnosed with arrhythmias in the acute setting, it is plausible that some of these may resolve during convalescence. Therefore, the optimal duration of therapy should be personalized and made according to the patient and physician's discretion.

Risk stratification may be helpful to guide monitoring during recovery, with further testing indicated in those with cardiac involvement during infection. It has been proposed that patients with any evidence of possible myocardial injury should undergo follow-up transthoracic echocardiogram (TTE) and electrocardiogram (EKG) 2–6 months after COVID-19 diagnosis [66]. Holter or event monitoring should be used as indicated by symptoms, but it may also be

reasonable to consider their use in asymptomatic patients, given the risk of a variety of arrhythmias in patients with COVID-19 (Table 2). Abnormal findings on TTE, EKG, or cardiac monitoring should trigger additional investigation, which may include stress testing or cardiac MRI. The role of cardiac MRI after COVID-19 infection has been controversial as it is more sensitive than initial screening tests and may reveal findings of unclear clinical significance. In a study of 100 COVID-19-recovered patients, 60 had evidence of myocardial inflammation, 32 had myocardial late gadolinium enhancement (including 12 patterns suggestive of ischemia), and 22 had pericardial enhancement [67]. Finally, three patients had severe abnormalities that were followed up with endomyocardial biopsy, ultimately revealing active lymphocytic inflammation.

Due in part to evidence of subclinical myocarditis seen in these patients, there is concern that COVID-19 infection may declare itself as a risk factor for heart failure in the long term [68]. As more data is obtained, cost-effectiveness analyses of testing in the recovery period may be considered, as well as randomized trials of prophylactic therapy for arrhythmias and/or myocardial dysfunction [66]. Cardiac MRI may be helpful for risk stratification and to guide counseling on return to aerobic exercise or competitive sports. It has been proposed that COVID-19 patients who have ever had symptoms should rest for at least 2 weeks and undergo initial testing similar to that described above [69]. Given the absence of COVID-19-specific data, patients with evidence of cardiac involvement should follow the guidelines for athletes with myocarditis and defer resumption of activity for 3–6 months [55, 56, 69].

Management of patients after long-term COVID-19 recovery is even more uncertain. Patients who recovered from SARS were shown to have more hyperlipidemia, cardiovascular disorders, and impaired glucose metabolism at 12-years of follow-up compared to age-matched controls [70]. Findings of the same study suggest that recovered SARS patients have altered lipid metabolism due to steroid use during their infection. It is plausible that similar findings may be seen in patients who are recovered from COVID-19, especially if they were also treated with steroids for severe pneumonia. At minimum, these patients should undergo age-appropriate cardiovascular disease screening in a primary care setting. However, more intensive screening may be considered.

Monitoring of Corrected QT Interval (QTc)

The use of QTc-prolonging drugs, including hydroxychloroquine (HCQ) and azithromycin (AZ), in the treatment of COVID-19 infection was forced into the limelight after a small, nonrandomized study of 36 patients suggested that HCQ alone or added to AZ aided clearance of a positive nasopharyngeal virus sample [71]. This study was the foundation for the rapid adaptation

of HCQ and/or AZ in worldwide clinical practice for COVID-19. Prolongation of the QT interval is a known risk factor for sudden cardiac death due to ventricular cardiac arrhythmias such as TDP [72, 73]. A previous study has reported significantly elevated risk of cardiac arrest in COVID-19 patients receiving HCQ + AZ, as well as in nonventilated COVID-19 patients receiving HCQ alone [74]. The known effect of HCQ and AZ on prolongation of the QT interval has led to many subsequent studies on incidence of TDP and sudden cardiac death in COVID infection. Due to these risks and evidence that the regimen lacks efficacy [75–77], these drugs are no longer used to treat COVID-19.

The direct causes of QT prolongation by HCQ and AZ are due to inhibition of the rapid delayed rectifier potassium current (I_{Kr} , or hERG) [78, 79]. However, indirect causes of QT prolongation in COVID infection may include inflammation, renal dysfunction, new onset of cardiac disease, electrolyte imbalance, and usage of other additional QTc-prolonging drugs.

Previous studies have suggested that increase in cytokines, such as IL-6, may prolong QT in patients with viral infection. HIV-associated inflammation, causing elevated IL-6, has been shown to be independently associated with QT prolongation [80] and prolonged repolarization represented as T wave onset-to-peak duration [81]. HIV-positive patients had longer QTc intervals and QTc prolongation often > 500 ms, even after taking into account other QT prolonging drugs like methadone when compared to HIV negative patients [82].

Conclusions

The true prevalence of cardiac events in patients with COVID-19 may not be fully appreciated. This review highlights the cardiac arrhythmias—such as AF, AV block, IST, POTS, and VT/VF—during and after COVID-19 infection (Fig. 1), underscoring the importance of careful cardiac management considerations in COVID-19 patients. Furthermore, given the breadth of cardiac arrhythmias involved in COVID-19 and the diversity of their etiologies, remote digital monitoring has emerged as a growing and necessary aspect of COVID-19 management for patients with cardiac complications or at risk of cardiac complications. With the onset of new therapeutics for COVID-19, further questions are raised as to how this will impact the management of arrhythmias. Studies will be needed to investigate the association between vaccination-status and risk of COVID-19-related cardiac arrhythmias. Furthermore, future research is needed to determine whether vaccination may be protective of cardiac injury and development of cardiac arrhythmias and which patients would most derive benefit. Similarly, with monoclonal antibody treatment recently gaining increased attention, it remains unknown what cardiac management changes

would accompany widespread adoption of these types of treatment. Finally, with subsequent waves of COVID-19 approaching, or already starting in many parts of the country, little is known on how re-infection, a second course of infection, will affect previously infected individuals. The exact cardiac complications which may accompany a second COVID-19 infection are yet unknown.

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Compliance with Ethical Standards

Conflict of Interest The authors have no conflict of interest to declare.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

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