Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Journal of Electrocardiology 62 (2020) 39–45

Contents lists available at ScienceDirect

Journal of Electrocardiology

journal homepage: www.jecgonline.com

Review

Value of electrocardiography in coronavirus disease 2019 (COVID-19)

Sohaib Haseeb [a], Enes Elvin Gul [b], Göksel Çinier [c], George Bazoukis [d], Jesus Alvarez-Garcia [e,f], Sebastian Garcia-Zamora [g], Sharen Lee [h], Cynthia Yeung [i], Tong Liu [j], Gary Tse [j,*], Adrian Baranchuk [i,**], On Behalf of The International Society of Electrocardiology Young Community (ISE-YC)

[a] College of Medicine and Dentistry, James Cook University, Townsville, Queensland, Australia
[b] Division of Cardiac Electrophysiology, Madinah Cardiac Centre, Madinah, Saudi Arabia
[c] Department of Cardiology, Dr Siyami Ersek Hospital Thoracic and Cardiovascular Surgery Training and Research Hospital, Istanbul, Turkey
[d] Second Department of Cardiology, Evangelismos General Hospital of Athens, Athens, Greece
[e] Icahn School of Medicine at Mount Sinai, Mount Sinai Hospital, New York, USA
[f] Cardiology Department, Hospital de la Santa Creu i Sant Pau, CIBERCV, Barcelona, Spain
[g] South American Center of Excellence for Cardiovascular Health (CESCAS), Institute for Clinical Effectiveness and Health Policy (IECS), Buenos Aires, Argentina
[h] Laboratory of Cardiovascular Physiology, Chinese University Shenzhen Research Institute, PR China
[i] Heart Rhythm Service, Kingston General Hospital, Queen's University, Kingston, Ontario, Canada
[j] Tianjin Key Laboratory of Ionic-Molecular Function of Cardiovascular Disease, Department of Cardiology, Tianjin Institute of Cardiology, Second Hospital of Tianjin Medical University, Tianjin, PR China

ARTICLE INFO

Keywords:
COVID-19
Arrhythmia
Electrocardiogram
Pandemics
QT interval
SARS-CoV-2

ABSTRACT

In December 2019, reports of an unknown pneumonia not responsive to traditional treatments arose in Wuhan, China. The pathogen was subsequently identified as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), known to be responsible for the coronavirus disease-2019 (COVID-19) illness, and public health emergency of international concern was declared by the World Health Organization. There is increasing awareness of the cardiovascular manifestations of COVID-19 disease, and the adverse impact of cardiovascular involvement on its prognosis. In this setting, the electrocardiogram (ECG) is one of the leading tools to assess the extent of cardiac involvement in COVID-19 patients, due to its wide disponibility, low cost, and the possibility of remote evaluation. In this article, we review the role of the ECG in the identification of cardiac involvement in COVID-19, highlighting relevant clinical implications.

Introduction

In December 2019, a novel viral infection arose in Wuhan, China, which then spread worldwide within several weeks. The infection was subsequently termed coronavirus disease-2019 (COVID-19) and declared a pandemic by the World Health Organization by March 2020. Most infected patients are asymptomatic or mild symptomatic, but approximately 15–20% develop acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The effects of SARS-CoV-2 on the heart are variable, but cardiac damage confers a worse prognosis, whether in the presence or absence of pre-existing cardiovascular disease [1,2]. Cardiac complications related to COVID-19 can be categorized into five types: a) cardiac injury (mainly due to ischemia or myocarditis); b) arrhythmia; c) new-onset or worsening of pre-existing heart failure; d) thromboembolic disease; and e) cardiac abnormalities induced by medical treatment [3].

In this setting, the electrocardiogram (ECG) is one of the leading tools to assess the extent of cardiac involvement in COVID-19 patients and the effect of medications, due to its wide accessibility, low cost, and the possibility of remote evaluation. Therefore, we proposed a review on the role of the ECG in the identification of cardiac involvement in COVID-19 and highlighted relevant clinical implications.

COVID-19 and markers of myocardial damage

Overview

Cardiac involvement in patients with COVID-19 is reflected in ECG alterations, such as ST changes, QT prolongation, conduction disturbances, and ventricular arrhythmias [4]. Therefore, patients presenting with cardiac symptoms and ECG changes should be carefully assessed

* Corresponding author at: Tianjin Key Laboratory of Ionic-Molecular Function of Cardiovascular disease, Department of Cardiology, Tianjin Institute of Cardiology, Second Hospital of Tianjin Medical University, Tianjin 300211, PR China.
** Corresponding author.
E-mail addresses: gary.tse@doctors.org.uk (G. Tse), Adrian.Baranchuk@kingstonhsc.ca (A. Baranchuk).

https://doi.org/10.1016/j.jelectrocard.2020.08.007
0022-0736/

in order to diagnose COVID-19 related cardiac complications such as myocarditis, brady- and tachyarrhythmias (Fig. 1).

In the era of the COVID-19 pandemic, a high clinical suspicion should be maintained even in patients who present with atypical symptoms or signs. Furthermore, cardiovascular disease has been found to be associated with a worse prognosis [5–8]. It should be stressed that the virus should not be considered as the cause of all cardiovascular complications, but may exacerbate or reveal underlying conditions [9,10]. However, more studies are needed to further clarify the role of the cardiovascular system in the COVID-19 pandemic [11].

QRST-abnormalities

Non-specific ECG findings reported in COVID-19 patients have been attributed to hypoxia or inflammatory damage. This includes a patient with SI, QIII, TIII pattern followed by a reversible but near-complete atrioventricular block, ST-segment elevation accompanied with multifocal ventricular tachycardia [4], and flattening of the T-waves in the inferior leads with right axis deviation. The SIQIIITIII pattern was observed in another patient whose infection was complicated by pulmonary embolism [12]. It should be noted that the SIQIIITIII pattern suggests acute right ventricular overload. In a case series of patients with COVID-19 related complications, premature atrial complexes, lateral T wave inversions, and a QTc interval of 528 ms were noted in a patient who presented with decompensated heart failure [13]. In a heart transplant recipient, sinus rhythm with new nonspecific T-wave inversions in the inferior and lateral precordial leads was seen [13]. In light of these published case reports and with the lack of further evidence, we propose that ST-T wave abnormalities, especially in the context of a cardiac-related clinical presentation, should lead to further investigations to exclude COVID-19 related cardiac complications during the current pandemic. ST-T wave abnormalities are useful especially when they develop during the course of a febrile disease and not to exclude but to demonstrate cardiac involvement, and especially without an evident context of cardiac-related clinical presentation.

Conduction disorders

Exacerbation of new-onset high degree atrioventricular block or bradyarrhythmic side-effects of antiviral therapy is possible in patients with COVID-19. A case of a transient complete heart block in a 54-year-old man with critical COVID-19 was recently reported [14]. Atrial tachycardia or atypical atrial flutter with 2:1 conduction and a concomitant wide QRS morphology in a COVID-19 positive patient was also reported [15].

Myocarditis and pericarditis

In the case of fulminant myocarditis, sinus tachycardia and right bundle branch block pattern without significant ST-T wave abnormalities were observed [16]. Other cases of myocarditis demonstrated non-specific intraventricular conduction delay and premature ventricular beats [17], or ST-segment elevations in leads III and aVF [18]. Furthermore, in a patient with acute myopericarditis, low voltage limb leads, diffuse ST-segment elevation (especially in the inferior and lateral leads), and ST-segment depression with a T-wave inversion in leads V1 and aVR were the reported ECG findings [19]. In another patient with COVID-19 myopericarditis without other symptoms of infection, sinus tachycardia, low voltage QRS complexes in the limb leads, ST-segment elevations in leads I, II, aVL, V2-V6, PR elevation, and ST depressions in lead aVR were observed [13].

COVID-19 and cardiac arrhythmias

Cardiac arrhythmias have been reported in 16.7% of COVID-19 patients, while malignant arrhythmias have been reported in 11.5% of patients [1,20]. In a recent study of 138 hospitalized patients with COVID-19, cardiac arrhythmias represented a leading complication and were more common among critically ill patients [20]. Another study found a higher incidence of arrhythmias in patients with severe disease than those with mild disease (44.4% versus 6.9%, $p < .001$) [21]. There have also been reports of critically ill patients with COVID-19 experiencing cardiac arrest with pulseless electrical activity or ventricular arrhythmias during the recovery phase of their pulmonary condition [22]. Among 187 hospitalized patients with confirmed COVID-19, 5.9% of the patients experienced malignant arrhythmias, including ventricular tachycardia and fibrillation [1]. Additionally, critically ill COVID-19 patients with fever were observed to have a slower heart rate than expected [20,23]. Bradycardia prolongs the QT interval and could facilitate Torsades de Pointes (TdP). Furthermore, QT prolongation secondary to antiviral therapies can also predispose patients to ventricular arrhythmias [24]. Although arrhythmias cannot be considered as a marker of COVID-19 infection, they can be a useful prognostic marker. Of note, patients with preexisting cardiovascular disease admitted to the intensive care unit for COVID-19-related illnesses may have a worse prognosis [20].

Given the preliminary nature of the available literature, the difference in the incidence of arrhythmia among recovering critical patients, and patients with mild disease, has not yet been well delineated. As more data become available, an improved understanding of the pathophysiology and significance of arrhythmia in patients with COVID-19

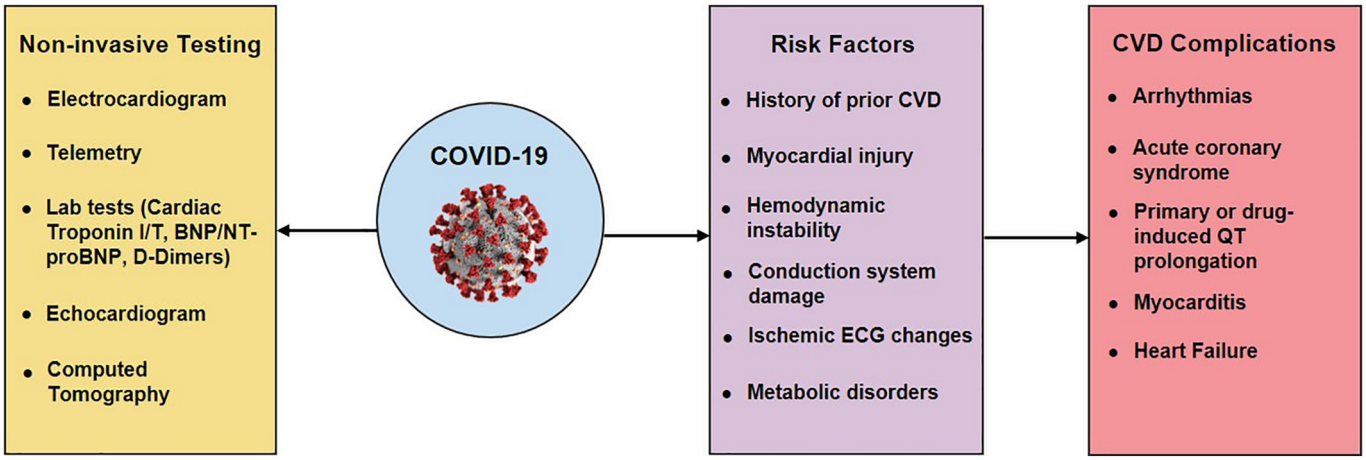

Fig. 1. Postulated cardiovascular involvement in COVID-19.

will guide the recommendations for possible additional rhythm monitoring in an outpatient setting.

Proper ECG diagnosis of atrial fibrillation (AF) is important for COVID-19 patients. It is not clear whether the presence of AF will alter the prognosis of COVID-19 patients. However, there have been speculations on the mechanism of AF in COVID-19. For instance, hypoxemia caused by COVID-19 may bring about AF and could be refractory under impaired pulmonary function. A plausible mechanism of AF reduction is the inhibition of I_{K1} and I_{KACh} channels [25].

COVID-19 and QT prolongation

QT interval measurement

Leads II or V5-V6 are recommended for the measurement of the QT interval. The QT interval should always be corrected according to the heart rate — employ Bazett's formula for correction if the heart rate is less than 90 beats per minute or Fridericia's in the case of higher heart rates. The end of the T-wave should be taken as the intersection between the tangent extrapolated from the point of maximum downslope and the isoelectric line.

COVID-19 related medications

QT prolongation and subsequent ventricular arrhythmias have been associated with the use of hydroxychloroquine/chloroquine (HCQ/CQ), azithromycin (AZ), and antivirals such as lopinavir/ritonavir (Table 1), or in COVID-19 patients with pre-existing hepatic disease or renal failure [26–28]. Although cases of QT prolongation and TdP due to HCQ/CQ have been reported, data on QT prolongation due to HCQ/AZ are contradictory. Chang et al. found that of 117 patients with COVID-19, only one patient experienced QT prolongation, in which case the medication was promptly discontinued [29]. However, in another cohort, 11% of patients developed QT prolongation, among which half of those patients had normal QT level at baseline [30]. This discrepancy can be explained by the heterogeneity of the patient cohort, such as the presence of comorbidities and varying disease severity. In the largest reported cohort of COVID-19 patients to date treated with HCQ/CQ ± AZ, no instances of TdP or arrhythmogenic death were reported. Although the use of these medications resulted in QT prolongation, clinicians seldom needed to discontinue therapy [31]. AZ is also known to have an increased risk of QT prolongation, TdP, and sudden cardiac death; however, the absolute risk is low [32,33].

Other drugs that are being investigated for the treatment of COVID-19, including remdesivir, favipiravir, ribavirin, sarilumab, and baricitinib, have limited data available regarding their effects on QT prolongation and cardiac arrhythmias. Kumagai et al. found no effect of favipiravir on the QT interval among healthy Japanese adults after the administration of single oral doses of 1200 and 2400 mg [34]. However, studies conducted with prolonged use of favipiravir have reported side effects such as increased uric acid levels, diarrhea, reduced neutrophil counts and abnormal liver function tests [35].

QT monitoring recommendations

Several scientific societies [22,36–39] and hospitals [40,41] across the globe have published protocols for QT interval monitoring in COVID-19 patients (Table 2). Taken together, they have the following points in common:

- Before considering any treatment, conduct a clinical history focused on a prior history of heart disease, syncope, sudden cardiac death, comorbidities, and generate a list of home medications.
- Identify and correct potentially modifiable risk factors for the prolongation of the QT interval (Table 3).
- Discontinue unnecessary conflicting drugs related to the prolongation QT interval

It should be noted that these guidance documents vary in their ECG-related recommendations. Some recommend that all patients receive a baseline and repeat ECG [36], whereas others have reserved this recommendation for higher-risk populations [22,38].

COVID-19 and treatment guidance

The risk stratification of COVID-19 patients should be performed based on their preexisting diseases since their prognosis varies greatly based on their underlying comorbidities. High-risk patients should be monitored more closely, in particular through the use of an ECG, than those who are otherwise healthy (Fig. 2). We propose the following ECG-guided recommendations:

- *Patients with inherited arrhythmic syndrome (long QT, Brugada syndrome, ARVC and hypertrophic cardiomyopathy):* It is well known that some patients, notably those with inherited long QT syndrome, may be at an elevated risk for drug-induced ventricular arrhythmia [42]. COVID-19 has also been reported to unmask inherited arrhythmias, such as Brugada syndrome in the setting of syncope [9]. Hypertrophic cardiomyopathy, being the most common inherited cardiomyopathy with risk for sudden death, particular caution is required. Therefore, an expert opinion of a cardiologist/electrophysiologist may be essential in determining how to best minimize the risk of malignant arrhythmias in patients with inherited arrhythmic syndrome [43].
- *Patients with prolonged QTc intervals at baseline:* If baseline ECG testing reveals a moderately prolonged QTc (above normal upper limit both for men and women until QTc = 500 ms), optimization of medications and electrolytes may permit therapy. If the QTc is markedly prolonged (QTc above 500 ms), drugs with potential QT lengthening effects should be avoided or modified, or expert consultation may permit administration with mitigating precautions [44].
- *Patients on multiple drugs that may cause QT prolongation and an increased risk of malignant arrhythmias:* It is important to note that combining more than one proarrhythmic medication is known to increase the risk of significant QT prolongation [45]. Therefore, medications should be reviewed, and unnecessary medications with QT-prolonging effects should be discontinued. Interestingly, amiodarone — as a medication that can potentially prolong the QT interval — has been suggested as a possible inhibitor against the spreading of SARS-CoV-2 due to its ability to interfere with the endocytic pathway [46]. Therefore, some experts recommend the administration of prophylactic intravenous amiodarone to mitigate the risk of sudden cardiac arrest among patients with COVID-19. However, given the increased risk of ventricular tachyarrhythmia, we recommend very close monitoring of the QTc interval in patients on regular amiodarone. Although amiodarone causes QT prolongation, it rarely leads to ventricular arrhythmias, specifically TdP.
- *Vulnerable patients with multiple comorbidities and a high frailty status:* Drug-induced QT prolongation in frail older patients may be exacerbated with pre-existing cardiac conditions such as cardiomyopathy, ischemia, heart failure, or bradycardia; and by other conditions such as diabetes, electrolyte abnormalities, hypoglycemia, or renal failure. Critically ill COVID-19 patients will likely be at a higher clinical risk of drug-induced arrhythmia, in which case ECG monitoring will more likely be indicated for supportive medical care. Patients with pre-existing structural heart disease pose a high risk of developing malignant arrhythmia; therefore, ECG should be assessed and monitored regularly before and during the initiation of COVID-19 related pharmacotherapy.

Table 1
Observational and randomized studies evaluating the risk of QT prolongation and ventricular arrhythmias with short courses of potential COVID-19 treatments.

Study	Sample size (n)	Setting	Study design	Age (yrs.)	Baseline comorbidities	Drugs administered	Treatment duration	ECG monitoring	ECG outcomes	Arrhythmia outcomes
Chen et al. [51]	30 moderate hospitalized COVID-19 patients	Shanghai, China	RCT	48.6	HTN (33.3%); DM (6.7%)	HCQ	7d	Not available	Not available	No serious adverse events
Chorin et al. [30]	84 hospitalized COVID-19 patients	New York, USA	Cohort study	63.0	HTN (65%); DM (20%); CAD (11%); COPD (8%); CKD (7%); Acute renal failure (6%); CHF (2%)	HCQ and AZ	5d	Baseline ECG daily	- QTc prolongation from baseline average of 435 ± 24 ms to a maximal average value of 463 ± 32 ms - QTc >500 ms in 11% of patients	No arrhythmias
Gautret et al. [52]	80 mild hospitalized COVID-19 patients	Marseille, France	Cohort study	52.5	HTN (16.3%); DM (11.2%); Chronic respiratory diseases (10%); CAD (7.5%); Obesity (5.0%); immunosuppression (5%)	HCQ and AZ	3d	Baseline ECG and on day 2	Not available	No serious adverse events
Huang et al. [53]	22 moderate and severe hospitalized COVID-19 patients	China	RCT	44.0	HTN (10%); DM (10%)	Chloroquine and Lopinavir/Ritonavir (control)	10d	Not available	Not available	No serious adverse events
Molina et al. [54]	11 hospitalized COVID-19 patients	Paris, France	Case series	58.7	Solid cancer (27%); hematologic cancer (18%); Obesity (18%); HIV (9%)	HCQ and AZ	HCQ: 10d AZ: 500 mg day 1 and 250 mg days 2 to 5	Not available	Excessive QT prolongation on in 1 patient (from 405 ms to 460 and 470 ms)	Not reported
Perinel et al. [55]	13 COVID-19 patients in the critical care unit	Saint Etienne, France	Cohort study	68.0	Moderate or severe renal failure (30.7%); mechanically ventilated (92%)	HCQ	Various dosing regimens	Not available	QT prolongation >500 ms in 2 of 13 patients (381 to 510 ms and 432 to 550 ms)	Not reported
Mercuro et al. [56]	90 hospitalized COVID-19 patients	Boston, USA	Cohort study	60.1	HTN (53.3%); DM (28.9%); COPD/asthma (20.0%); AF (13.3%); CAD (11.1%); CHF (10.0)	HCQ ± AZ	5d	Not available	- Concomitant AZ therapy had a greater median change in QT (23 [10–40] ms) compared with HCQ monotherapy (5.5 [−15.5 to 34.25] ms - QT prolongation >500 ms in 19% of patients receiving HCQ monotherapy and in 21% receiving concomitant AZ	1 case of TdP
Saleh et al. [31]	201 hospitalized COVID-19 patients	USA	Cohort study	58.5	HTN (60.2%); Hyperlipidemia (41.8%); DM (32.3%); AF (7.0%); CAD (11.4%); COPD/asthma (14.9%); CKD ≥ stage III (5.0%); HF	Chloroquine/HCQ ± AZ	Various dosing regimens	Twice daily ECGs or MCOT Patch	- Baseline QTc did not differ between chloroquine/HCQ monotherapy vs. combination group with AZ (440.6 ± 24.9 ms vs. 439.9 ± 24.7, $p = .834$) - Max QTc during treatment was significantly longer in the combination group vs. the monotherapy group (470.4 ± 45.0 ms vs. 453.3 ± 37.0, $p = .004$)	No serious adverse events
Ramireddy et al. [57]	98 hospitalized COVID-19 patients	Los Angeles, USA	Case series	62.3	HTN (60%); DM (22%); COPD (26%); HF (20%); CKD (14%)	HCQ, AZ, or combination	Various dosing regimens	Baseline and post-medication ECG (up to 24 h)	- Significant QT prolongation observed only in men (18 ± 43 ms vs. 0.2 ± 28 ms in women, $p = .02$) - Critical QT prolongation reached in 12% of patients - Changes in QTc highest with combination therapy - Much greater prolongation with combination vs. AZ (17 ± 39 vs. 0.5 ± 40 ms, $p = .07$)	No TdP observed
Rosenberg et al. [58]	1438 hospitalized COVID-19 patients	New York City, USA	Cohort study	63	Obesity (46.6%); Cancer (4.0%); any kidney disease (12.0%); any chronic lung conditions (17.6%); diabetes (36.6%); any CVDs (29.1%); CHF (6.3%)	HCQ + AZ, HCQ alone, AZ, or neither	Various dosing regimens	Not available	- QT prolongation observed in 11.0% in HCQ + AZ group, 14.4% in HCQ alone group, 7.1% in AZ alone group, and 5.9% in neither drug group ($p < .006$)	- Arrhythmias observed in 20.4% in HCQ + AZ group, 16.2% in HCQ alone group, 10.9% in AZ alone group, and 10.4% in neither drug group ($p < .001$)

Abbreviations: AF = atrial fibrillation; AZ = azithromycin; CAD = coronary artery disease; CKD = chronic kidney disease; CHF = congestive heart failure; COPD = Chronic obstructive pulmonary disease; COVID-19 = coronavirus disease 2019; CVD = cardiovascular disease; DM = diabetes mellitus; HCQ = hydroxychloroquine; HIV = human immunodeficiency virus; HF = heart failure; HTN = hypertension; MCOT = Mobile Cardiac Outpatient Telemetry; RCT = randomized controlled trial; TdP = Torsades de pointes.

Table 2
Cardiovascular societies' recommendations on QT-interval monitoring in patients with COVID-19.

Society/Guideline	QT monitoring recommendations
American College of Cardiology [59]	Baseline: - Discontinue and avoid non-critical QT-prolonging agents - Assess baseline ECG, renal function, hepatic function, serum K+, and Mg^2+ - Have an experienced electrophysiologist measure QTc whenever possible Relative contraindications: - History of Long QT syndrome - Baseline QTc >500 ms (or > 530–550 ms in patients with QRS >120 ms) Ongoing monitoring, dose adjustment, and drug discontinuation: - Place on telemetry before initiation of therapy - Acquire ECG 2–3 h after the second dose of HCQ and daily after - If QTc increases by >60 ms or absolute QTc >500 ms (or > 530–550 ms if QRS >120 ms), discontinue AZ if used and/or reduce HCQ dose and repeat ECG daily - If QTc remains increased in the above situation, undertake risk--benefit of ongoing therapy, consider a consultation with electrophysiologist and consider discontinuation of HCQ
European Society of Cardiology [36]	- On-treatment of COVID-19, ECG recommended to rule out signification QTc prolongation (>500 ms, or by >60 ms vs. baseline) - Therapy of TdP VT consistent with the withdrawal of all QT-prolonging drugs, targeting K+ >4.5 mEq/L
HRS COVID-19 Task Force, ACC Electrophysiology Section and AHA EP and Arrhythmias Committee [22]	- Patients on AADs that require QT and laboratory monitoring may defer testing if previous values and clinical condition remains stable and if no new QT-prolonging drugs have been added
Latin American Heart Rhythm Society [38]	- 12-lead ECG to measure QTc interval at baseline and after initiation of any QT-prolonging drugs - Patients with a baseline QTc >500 ms and those with QTc prolongation >60 ms post-medication exposure, a risk-benefit analysis should be undertaken - In patients with abnormal QT prolongation, correction of electrolytes abnormalities (K+ >4 mEq/L; Mg2+ > 2 mEq/L), discontinuation of unnecessary QT-prolonging drugs, and continuous telemetry for monitoring of ventricular arrhythmias - Discontinue QT-prolonging drugs if TdP noted
Canadian Cardiovascular Society [44]	- Review and discontinue unnecessary QT-prolonging medications - For patients with a previous history of TdP or Long QT, the use of potential COVID-19 therapies should be undertaken after expert consultation - For patients with no previous history or precipitating factors, it may be reasonable to proceed with antimicrobial drug without baseline or follow-up ECG if it would increase population risk of infection - Hospitalized patients or those not fulfilling the above criteria: - ECG to assess QTc if not performed within the past 3 months; - If QTc ≥500 ms, reassess after correction of electrolyte abnormalities or discontinuation of other QT--prolonging drugs. Seek expert consultation if QTc remains ≥500 ms; - If QTc ≥470 ms for males or ≥ 480 ms for females by <500 ms, initiate antimicrobial drugs and consider repeat ECG in 48 h; - In patients with clinically severe disease or taking multiple QT--prolonging medications, recheck QT after 48 h of antimicrobial drug initiation; - If follow-up QTc increases ≥60 ms or is ≥500 ms, discontinue antimicrobial drugs and seek expert opinion

Abbreviations: AADs = Antiarrhythmic Drugs; AZ = azithromycin; ACC = American College of Cardiology; AHA = American Heart Association; COVID-19 = coronavirus disease 2019; ECG = electrocardiogram; HCQ = Hydroxychloroquine; HRS = Heart Rhythm Society; TdP = Torsades de Pointes; VT = ventricular tachycardia.

Table 3
Risk factors for inducible QT prolongation and arrhythmias.

Modifiable risk factors
• Electrolyte abnormalities: Hypocalcemia, Hypokalemia, Hypomagnesemia • Drugs that prolong the QT: especially the simultaneous use of ≥1 drug • Serious eating disorders (ie, anorexia nervosa)
Non-modifiable risk factors
• Personal characteristics: Age > 65 years Female sex Previous QT prolongation or unexplained sudden death • Comorbidities: - Cardiac pathologies: Acute coronary syndrome, Reduced ejection fraction (more risk with worse EF), Decompensated heart failure, Bradyarrhythmia (especially heart rate < 45 bpm), Hyperthrofic cardiomyopathy, first hours after serious events (post cardiac arrest, syncope or convulsion) - Non cardiac pathologies: Recent cerebrovascular events (ischemic or hemorrhagic stroke or cranial trauma), Renal failure on dialysis, Hypoglycemia/Diabetes mellitus, - Rare conditions: Congenital long QT syndrome (all variants), Pheochromocytoma

COVID-19 and ECG monitoring

ECG monitoring is advisable especially when patients experience electrolyte disturbances and use concomitant QTc-prolonging drugs [47]. Therefore, ECG monitoring upholds a critical role in patient safety during the dose adjustment of medications used in the management of COVID-19. In an outpatient setting, mobile devices such as the KardiaMobile 6 L (AliveCor, Mountain View, California) and the Apple Watch ECG (Apple, Cupertino, California) have shown to be effective in monitoring the QTc interval [48,49]. In a recent study, the QTc interval

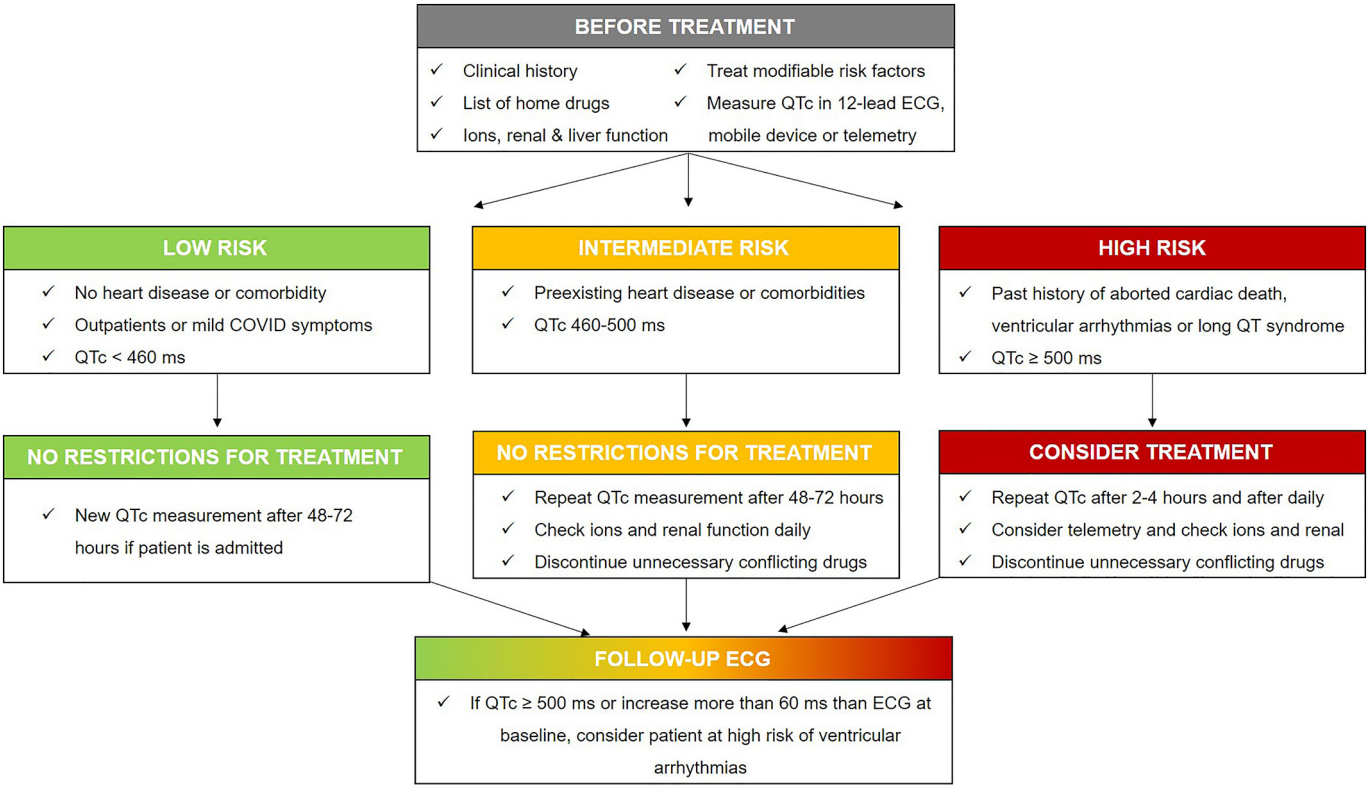

Fig. 2. Proposed flow diagram concerning the arrhythmic vulnerability related to QTc prolongation from potential QTc-prolonging drugs.

in leads I and II from a handheld ECG device and 12-lead ECG were compared across 99 healthy volunteers, and 20 hospitalized patients in sinus rhythm treated with dofetilide or sotalol [50]. The handheld ECG device was accurate in the measurement of QTc interval for both patients with sinus rhythm and QT prolongation [50]. In cases with limited resources or quarantine, the Kardia6L system — which has received expedited US Food and Drugs Administration clearance — could be used to deduce the risk status before initiation of drug therapy.

Conclusion

In the era of the SARS-CoV-2 pandemic, COVID-19 should be considered as a differential diagnosis for new or presumably new electrocardiographic abnormalities accompanied by a clinical presentation indicative of potential cardiac involvement. However, further studies with a systematic approach in the measurement of ECG parameters are needed to elucidate the potential role of ECG in myocardial injury diagnosis and risk stratification of COVID-19 patients.

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