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Enhanced electrocardiographic monitoring of patients with Coronavirus Disease 2019

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BACKGROUND Many of the drugs being used in the treatment of the ongoing pandemic coronavirus disease 2019 (COVID-19) are associated with QT prolongation. Expert guidance supports electrocardiographic (ECG) monitoring to optimize patient safety.

OBJECTIVE The purpose of this study was to establish an enhanced process for ECG monitoring of patients being treated for COVID-19.

METHODS We created a Situation Background Assessment Recommendation tool identifying the indication for ECGs in patients with COVID-19 and tagged these ECGs to ensure prompt over reading and identification of those with QT prolongation (corrected QT interval $>$ 470 ms for QRS duration $\leq$ 120 ms; corrected QT interval $>$ 500 ms for QRS duration $>$ 120 ms). This triggered a phone call from the electrophysiology service to the primary team to provide management guidance and a formal consultation if requested.

RESULTS During a 2-week period, we reviewed 2006 ECGs, corresponding to 524 unique patients, of whom 103 (19.7%) met the Situation Background Assessment Recommendation tool–defined criteria for QT prolongation. Compared with those without QT prolongation, these patients were more often in the intensive care unit (60 [58.3%] vs 149 [35.4%]) and more likely to be intubated (32 [31.1%] vs 76 [18.1%]). Fifty patients with QT prolongation (48.5%) had electrolyte abnormalities, 98 (95.1%) were on COVID-19–related QT-prolonging medications, and 62 (60.2%) were on 1–4 additional non-COVID-19–related QT-prolonging drugs. Electrophysiology recommendations were given to limit modifiable risk factors. No patient developed torsades de pointes.

CONCLUSION This process functioned efficiently, identified a high percentage of patients with QT prolongation, and led to relevant interventions. Arrhythmias were rare. No patient developed torsades de pointes.

KEYWORDS COVID-19; QT prolongation; ECG; Hydroxychloroquine; Torsades de pointes

(Heart Rhythm 2020;17:1417–1422)

Introduction

The ongoing pandemic coronavirus disease 2019 (COVID-19) has led to a number of changes in clinical processes in an effort to provide large numbers of patients with optimal care. Several existing medications are being repurposed for potential prophylaxis or treatment of COVID-19, including chloroquine and hydroxychloroquine[1,2] often in combination with azithromycin[3] and antivirals.[4–6] The use of these medications has been associated with QT prolongation and occasional reports of torsades de pointes when used for their original indications.[7,8] Their combined use, in the setting of systemic inflammation[9] and potential metabolic abnormalities, most specifically hypomagnesemia and hypokalemia,[10] seen in the context of this severe systemic illness likely magnifies this risk via multiplicative effects on the rapid delayed rectifier current potassium channel (I_{Kr}) as well as other less well-defined mechanisms.[12,13] Guidance from an American Heart Association (AHA)/American College of Cardiology (ACC)/Heart Rhythm Society (HRS) position statement[14] has supported the use of electrocardiographic (ECG) monitoring to identify QT prolongation in patients with COVID-19 being treated with these drugs. We describe the process we instituted to facilitate such monitoring and the outcome of our screening.

Methods

Recognizing the importance of the timely identification of ECG abnormalities, most specifically QT prolongation, in patients being treated for COVID-19, we initiated a hospital-wide protocol designed to optimize ECG monitoring of these patients. This quality improvement initiative was granted an exemption by the Yale University Institutional Review Board. Because patients with COVID-19 are treated by physicians less familiar with the implications of QT prolongation and exacerbating factors such as other QT-prolonging medications and electrolyte

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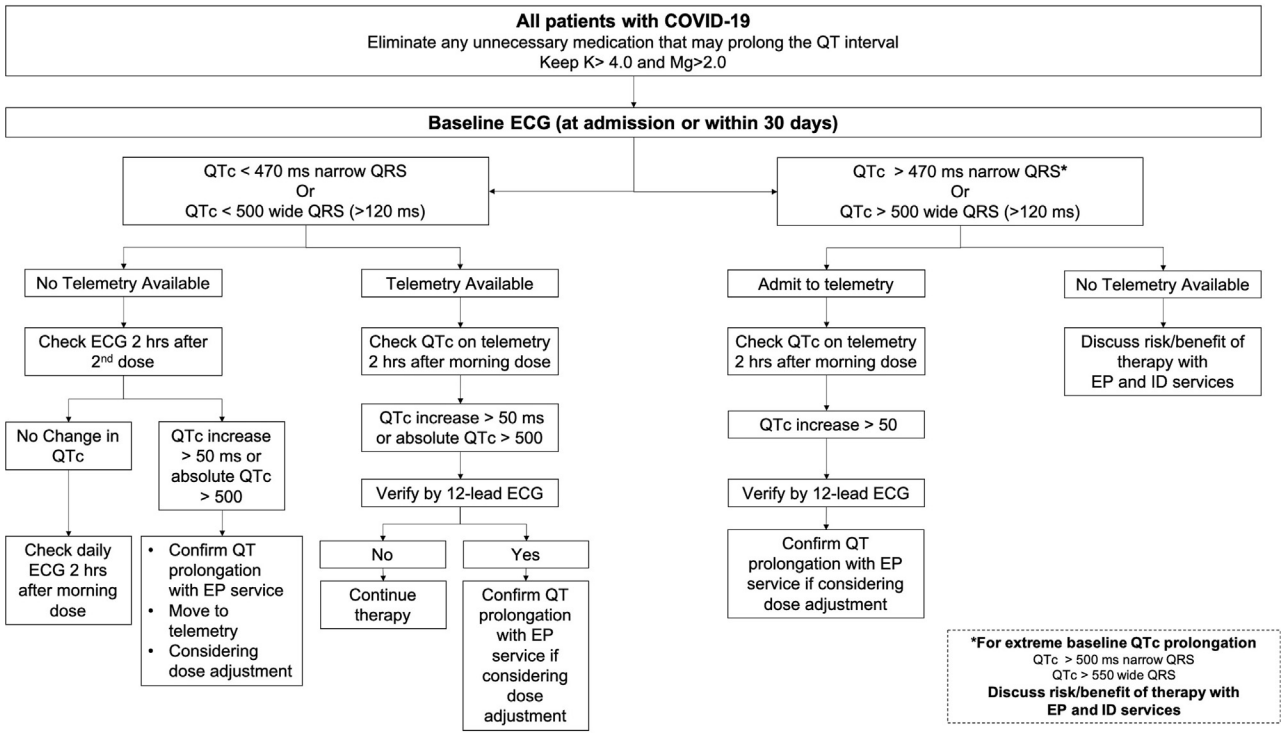

Figure 1 Situation Background Assessment Recommendation tool for the management of QT prolongation in COVID-19 patients. COVID-19 = coronavirus disease 2019; ECG = electrocardiogram; EP = electrophysiology; ID = infectious disease; QTc = corrected QT.

abnormalities, our protocol included active involvement of the ECG readers and the electrophysiology team.

Local hospital stakeholders created an SBAR (Situation Background Assessment Recommendation) tool (Figure 1) with guidelines for QT monitoring, which was disseminated to the teams caring for these patients. This SBAR tool tried to balance risk and the desire to limit frequent ECGs in patients with COVID-19 and was largely based on experience with QT-prolonging antiarrhythmic drugs. It did use QT measurements from telemetry when possible, but 12-lead ECGs were used for those not in units with telemetry, a large proportion of patients with COVID-19 at our hospital. We then identified all ECGs from patients with a diagnosis of COVID-19 or originating from floors designated as COVID-19 units. Our screening was designed to capture as many COVID-19 patients as possible, and thus a small percentage (65 [15.4%]) of screened patients, often in intensive care units (ICUs) with other medical emergencies, did not have COVID-19. These ECGs were placed at the beginning of the reading queue for the electrophysiologist or cardiologist over reading the ECGs on that day. The daily reader identified all ECGs with significant QT prolongation as outlined in the SBAR tool (corrected QT [QTc] interval $>$ 470 ms for QRS duration $\leq$ 120 ms or QTc interval $>$ 500 ms for QRS duration $>$ 120 ms) (Figure 1) and notified the electrophysiology consult service, which then reviewed the charts of these patients and provided telephone and, if necessary, formal inpatient support to the primary team caring for patients. This support consisted most often of recommendations to replete electrolytes to more stringent levels (potassium level $>$4 mmol/L; magnesium level $>$2 mg/dL) (standard laboratory normals: potassium level 3.3–5.1 mmol/L; magnesium level 1.7–2.4 mg/dL), to discontinue potentially nonessential QT-prolonging drugs as identified by the electrophysiology team, and to consider the risks and benefits of continuing COVID-19 targeted therapy, most commonly with hydroxychloroquine.

Continuous data are expressed as mean $\pm$ SD and categorical data as number (percentage). P values of $<$.05 were considered statistically significant. All statistical analyses were performed using SPSS version 26.0 (IBM Corporation, Armonk, NY).

Results

During the 2-week period from March 28, 2020, to April 10, 2020, we identified 2006 ECGs that came from patients with a diagnosis of COVID-19 or from a nursing unit designated to care for patients with COVID-19, representing 524 unique patients. Overall, 459 patients (84.6%) were confirmed to have a diagnosis of COVID-19. Individual patients had 1–14 ECGs, often over several days. Of these 524 patients, 103 (19.7%), all with a diagnosis of COVID-19, had ECGs with QT prolongation as defined by the SBAR tool (Figure 2) and were referred for electrophysiology review and recommendations. Patient sociodemographic and medical characteristics are outlined in Table 1. Among patients with COVID-19, those with QT prolongation were more likely to spend time in the ICU (60 [58.3%] vs 130

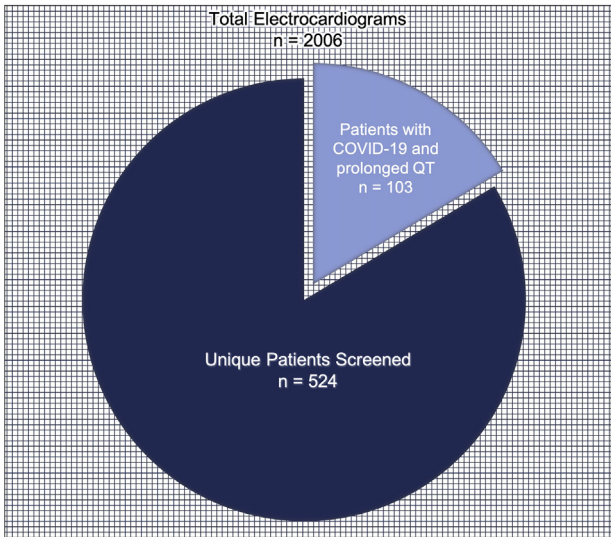

Figure 2 Electrocardiograms screened throughout the study period. COVID-19 = coronavirus disease 2019.

[36.5%]; $P < .001$) and were more likely to be intubated (32 [31.1%] vs 70 [19.7%]; $P = .014$) than those without this finding (Table 2). Univariate analysis showed that ICU stay and intubation were both associated with QT prolongation (odds ratio [OR] 2.4; 95% confidence interval [CI] 1.5–3.8; $P < .001$ and OR 1.8, 95% CI 1.1–3.0; $P = .015$). After controlling for ICU stay, intubation, and tocilizumab and hydroxychloroquine use, multivariate analysis showed that ICU stay was still strongly associated with QT prolongation (OR. 2.1, 95% CI 1.2–3.7; $P = .012$) (Supplemental Table 1).

Among patients with QT prolongation, virtually all 98 (95.1%) were on QT-prolonging drugs related to the treatment of their COVID-19 infection, most commonly hydroxychloroquine (Table 3). Of those without QT prolongation, 356 (84.6%) were COVID-19 positive, and of those, 317 (89.0%) received hydroxychloroquine (Supplemental Table 2).

In the group with QT prolongation, the mean QTc interval on the initial ECG was 470.6 ± 35.9 ms, with the peak QTc interval increasing to 520.6 ± 36.7 ms. The QTc interval at the final ECG (which was often the ECG recorded before discharge) showed a mean QTc interval of 478.9 ± 31.1 ms. As shown in Figure 3, the QTc interval increased significantly and then declined before discharge.

The electrophysiology consultations identified a number of potentially remediable factors that could also contribute to QT prolongation in patients with this finding, most commonly electrolyte abnormalities in 50 patients (48.5%) and QT-prolonging medications not related to the direct treatment of COVID-19 in 62 patients (60.2%). These patients were found to be receiving up to 4 non-COVID-19–related QT-prolonging drugs, with 38 (36.9%) receiving ≥2 of such drugs, most commonly psychiatric medications, anesthetics, or proton pump inhibitors (Tables 3 and 4). While there was a general awareness of the risk of QT prolongation among the treating teams, the consultations frequently identified additional interventions (Table 5) and were welcomed

Table 1 Baseline patient characteristics of patients with COVID-19 with QT prolongation

Characteristic	Value
Age (y)	68.2 ± 15.2
QT interval (ms)	448.0 ± 44.7
QTc interval (ms)	507.5 ± 28.5
BMI (kg/m^2)	28.9 ± 7.1
Male sex	64 (62.1)
Severity of disease	
ICU during hospitalization	60 (58.3)
Intubation during hospitalization	32 (31.1)
Medical comorbidities	
HTN	61 (59.2)
Diabetes mellitus	50 (48.5)
HLD	33 (32.0)
CKD/ESRD	32 (31.1)
AF/AFL	20 (19.4)
CAD	19 (18.4)
Lung disease	18 (17.5)
Morbid obesity	17 (16.5)
Heart failure	16 (15.5)
CVA/TIA	14 (13.6)
Obstructive sleep apnea	14 (13.6)
Malignancy	14 (13.6)
Cognitive impairment/dementia	12 (11.7)
Cardiovascular implantable electrical devices	11 (10.7)
Liver disease	9 (8.7)
Alcohol abuse/substance abuse	4 (3.9)
HIV/AIDS	3 (2.9)

Values are presented as mean ± SD or as n (%).

Malignancy was defined as active cancer or a history of cancer that was treated with chemotherapy.

AF = atrial fibrillation; AFL = atrial flutter; BMI = body mass index; CAD = coronary artery disease; CKD = chronic kidney disease; COVID-19 = coronavirus disease 2019; CVA = cerebrovascular accident; ESRD = end-stage renal disease; HLD = hyperlipidemia; HTN = hypertension; ICU = intensive care unit; QTc = corrected QT; TIA = transient ischemic accident.

Table 2 Baseline characteristics of all patients with COVID-19

Characteristic	QT prolongation		
	Present (n = 103)	Not present (n = 356)	*P*
Age (y)	68.2 ± 15.2	64.8 ± 17.5	.137
Male sex	64 (62.1)	197 (55.3)	.220
Severity of disease			
ICU during hospitalization	60 (58.3)	130 (36.5)	.000
Intubation required during hospitalization	32 (31.1)	70 (19.7)	.014
COVID-19–related medications			
Hydroxychloroquine	98 (95.1)	317 (89.0)	.064
Tocilizumab	83 (80.6)	223 (62.6)	.001
Hydroxychloroquine + atazanavir	21 (20.4)	47 (13.2)	.071
Other COVID-19–related medications	7 (6.8)	19 (5.3)	.573

Values are presented as mean ± SD or as n (%).

Other COVID-19–related medications include remdesivir, nivolumab, ritonavir/lopinavir, ruxolitinib, sarilumab, and plasma.

COVID-19 = coronavirus disease 2019; ICU = intensive care unit.

Table 3 Medications and electrolyte abnormalities in patients with COVID-19 and QT prolongation

Factor	Value
Patients with QT-prolonging medications	103 (100)
COVID-19–related medications	
Hydroxychloroquine	98 (95.1)
Hydroxychloroquine + atazanavir	21 (20.4)
Tocilizumab	83 (80.6)
Methylprednisolone	28 (27.2)
Other COVID-19–related medications	7 (6.8)
Remdesivir	5 (4.9)
Azithromycin	3 (2.9)
Nivolumab	2 (1.9)
Ritonavir/lopinavir	2 (1.9)
Non-COVID-19–related medications	62 (60.2)
Amiodarone	7 (6.8)
Proton pump inhibitor	18 (17.5)
Propofol	16 (15.5)
Sedative	14 (13.6)
SSRI	11 (10.7)
Antipsychotic	9 (8.7)
Antidepressant	7 (6.8)
Tacrolimus	7 (6.8)
Antibiotic	7 (6.8)
Antiemetic	6 (5.8)
Other QT-prolonging medications	10 (9.7)
Electrolyte abnormalities	50 (48.5)
Hypomagnesemia	31 (30.1)
Hypokalemia	27 (26.2)
Hypomagnesemia + hypokalemia	9 (8.7)

Values are presented as mean ± SD or as n (%).

Hypomagnesemia was defined as a value less than 2.0 mEq/L, and *hypokalemia* was defined as a value less than 4.0 mEq/L.

COVID-19 = coronavirus disease 2019; SSRI = selective serotonin reuptake inhibitor.

by the teams. Specific COVID-19 treatments, most commonly hydroxychloroquine rarely in association with atazanavir or azithromycin, were discontinued in approximately one-third (31 [30.1%]) of patients as a result of QT prolongation at the discretion of the primary and/or infectious disease teams. Despite the relative frequency of significant QT prolongation, serious clinical arrhythmias were rare. No episodes of torsades de pointes were reported. One patient each had ventricular premature complexes and nonsustained ventricular tachycardia. One patient had sustained monomorphic ventricular tachycardia in the setting of an ST-segment elevation myocardial infarction, and 3 patients died after being made "do not resuscitate" with comfort measures only. These patients were receiving palliative care and were not being monitored at the time of death.

Discussion

The ongoing COVID-19 pandemic has caused us to modify our usual care pathways to provide the best possible care to the patients, often with suboptimal resources. The medications being used clinically with hopes of improving outcomes in patients infected with COVID-19 carry their own risks. Most notably, hydroxychloroquine and azithromycin, when used in other situations, carry a risk of QT prolongation and rarely torsades de pointes.[7,8] While this risk is real, the long history of hydroxychloroquine, and to a lesser extent of azithromycin, use suggests that it is not prohibitive. However, combining these medications in the setting of severe illness, a proinflammatory state, the administration of other QT-prolonging medications and metabolic derangements, most especially hypokalemia and hypomagnesemia, has raised concerns about an increased risk of arrhythmia in these patients and have made efficient monitoring and therapeutic adjustments to minimize this risk very important.[14] It is also possible that such stresses could unveil previously unidentified monogenetic or other inherited vulnerability to QT prolongation.[15] We report here a modification of our usual treatment pathways that we believe has enhanced patient care and may have contributed to a reduced risk of clinically significant arrhythmia while preventing premature discontinuation of potentially effective therapy.

The SBAR tool we used for screening patients was developed at our institution early in our COVID-19 experience and was designed to find a compromise between safety and the need for frequent ECGs before we were routinely using validated telemetry or remote monitoring equipment for measuring the QT interval. It was based on our experience of monitoring patients during antiarrhythmic drug loading, and while it varies in detail from some other published algorithms,[16] it shares many similarities. We did identify a high prevalence of QT prolongation 103/524 (19.7%) in our population of screened patients, but it is notable that the majority of patients (72 [69.9%]) were able to complete their therapy, and the only clinically significant arrhythmia seen was in the setting of myocardial infarction. Three patients who were made "do not resuscitate" or "comfort measures only" died in an unmonitored setting. Our patients with QT prolongation had many, often severe, comorbidities. Although we have somewhat less specific clinical data on patients not identified as having QT prolongation on screening, it does appear that those demonstrating this phenomenon were sicker on the basis of ICU admissions and the need for intubation. It would seem likely as well that these situations would increase the risk of polypharmacy and electrolyte abnormalities.

While we think it is likely that many factors contributed to the low incidence of clinical arrhythmias in our patients, we cannot rule out that our interventions, which improved electrolyte management and helped identify other QT-prolonging medications, many of which were then discontinued, played a role in the return of the QTc interval toward baseline values and the low incidence of torsades de pointes. We also hope that they gave support to the treating teams to continue therapy when this was felt to be indicated. We do know that mitigating multiple simultaneous insults to the ion channels involved in myocardial repolarization, most specifically I_{Kr}, may be protective.[11] Other reasons for the low incidence of torsades de pointes may be the safeguarding effects of relative sinus tachycardia or other unidentified factors.

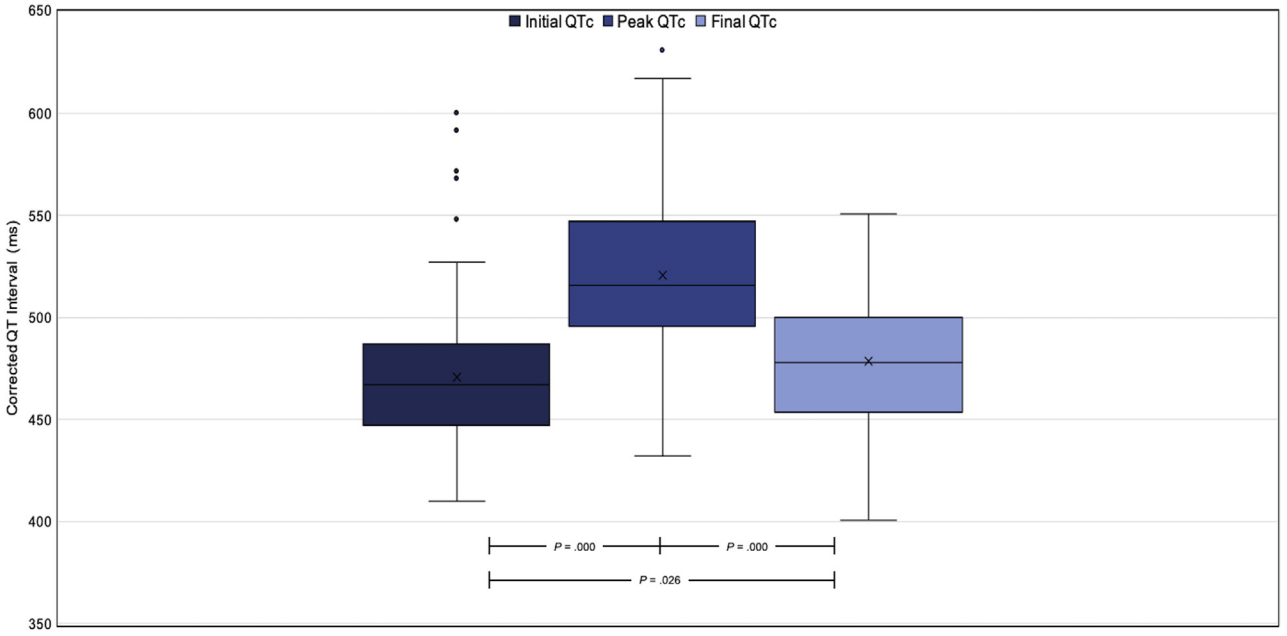

Figure 3 Box plot of QTc intervals for the initial ECG, peak value, and final ECG. Compared with the initial QTc interval, the QTc interval was significantly longer at peak (470.6 ± 35.9 ms vs 520.6 ± 36.7 ms; $P < .001$). Compared with the peak QTc interval, there was a significant decrease in QTc interval by the final ECG (520.6 ± 36.7 ms vs 478.9 ± 31.0 ms; $P < .001$). There was also a difference noted between the initial QTc interval and the final QTc interval (470.6 ± 35.9 ms vs 478.9 ± 31.1 ms; $P = .026$). ECG = electrocardiogram; QTc = corrected QT.

Although we believe that our process functioned well, we recognized early on that the multiple ECGs recorded in these patients increased both the exposure of caregivers and equipment to infected patients and the use of precious personal protective equipment. Given our finding of a low incidence of clinically significant arrhythmias, we have revised our SBAR tool to reduce the frequency of recommended ECGs and have worked to perform more serial QT measurements using either validated telemetry recordings or remote monitoring in patients in units without telemetry. As such, our report may represent a unique look at ECG-validated QT measurements during the treatment of COVID-19.

Limitations

Our study has several limitations, including those of a single-center retrospective study. During this period, treatment algorithms were evolving and not all patients were treated with a single protocol. Our results are based on ECG measurements of the QTc interval, which, given the desire to limit the use of personal protective equipment and caregiver and equipment exposure to patients with COVID-19 as well as greater comfort with remote monitoring for this complication,[17] may be less widely used at this time. In addition, not all patients were continuously monitored and therefore some arrhythmias or transient QT prolongation may not have been identified. However, the lack of significant clinical arrhythmic events suggests that our monitoring protocol, as well as other possible interventions, was fairly effective.

Table 4 Other (non-COVID-19–related) QT-prolonging medications in patients with COVID-19 and QT prolongation

Patients with other QT-prolonging medications	62 (60.2)
No. of medications	Value
1	24 (23.3)
2	27 (26.2)
3	10 (9.7)
4	1 (1)

Values are presented as n (%).
Groups are based on the total number of medications present.
COVID-19 = coronavirus disease 2019.

Conclusion

In an effort to provide optimal care in the setting of the current COVID-19 pandemic, we developed a system to rapidly and efficiently review the ECGs of these patients, identify those with QT prolongation, and provide

Table 5 Electrophysiology service's additional recommendations made after discussion with the primary team

Additional recommendations made	Value
Magnesium repletion	25 (24.3)
Discontinuing nonessential/use non–QT-prolonging alternative	18 (17.5)
Potassium repletion	4 (3.9)
Continue COVID-19 treatment	8 (7.8)
Telemetry/ECG monitoring	5 (4.9)
Discontinuing antiarrhythmics	4 (3.9)
Continuing antiarrhythmics	3 (2.9)
Dose adjustment	2 (1.9)

Values are presented as n (%).
COVID-19 = coronavirus disease 2019; ECG = electrocardiographic.

electrophysiological guidance to the treating teams. While we did identify a high prevalence of QT prolongation in this population, we also found a number of modifiable factors including other QT-prolonging drugs that could be eliminated and electrolyte abnormalities that could be corrected. The majority of patients completed COVID-19–specific therapy, and there were no associated episodes of torsades de pointes.

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Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrthm.2020.04.047.

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