CONCLUSION In this elderly population with coronary artery disease revascularized before the pandemic, an increase in cardiovascular and general morbidity as well as in total mortality was observed during the outbreak and confinement. Incidence of COVID-19 was higher than in the general population. Mortality among COVID-19 patients was very high.

CATEGORIES OTHER COVID-19

TCT CONNECT-217

Hydroxychloroquine and Azithromycin Usage in African American Patients With Coronavirus Disease 2019 (COVID-19) and Their Effects on QT Interval

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BACKGROUND The novel coronavirus disease-2019 (COVID-19) caused by the severe acute respiratory syndrome coronavirus-2 (SARS CoV-2) has been a major cause of morbidity and mortality around the world. Thirteen million cases have been diagnosed with approximately 570,000 deaths worldwide. COVID-19 is associated with ischemia, myocarditis and eventual resulting arrhythmia. Cases may present as acute thrombotic occlusion, stress cardiomyopathy, or coronary spasm. Hydroxychloroquine (HCQ) was temporarily approved by FDA for COVID-19 treatment. In this study, we planned to characterize the risk and degree of QTc prolongation in largely African-American population in central Brooklyn, who were hospitalized with COVID-19 infection in association with inpatient administration of HCQ and azithromycin. One of the major adverse drug effects of HCQ and chloroquine is the potential prolongation of corrected QT interval (QTc).

METHODS In our retrospective study, we included patients, both males and females, 18 years of age and older who were admitted at SUNY Downstate Medical Center, Brooklyn, New York, for COVID-19 infection and were treated with hydroxychloroquine. Native baseline RR, QRS, and QT intervals were measured before administering the first dose of hydroxychloroquine and within 24 h of administration. The RR interval was measured as a distance between the peak of the R-wave and the peak of the previous R-wave in the same lead in milliseconds and converted to a heart rate by equation, 60,000/RR. For correction of the QT, we used common formulas: $QTc = QT/\sqrt{RR}$ [Bazett formula], $QTc = QT/\sqrt[3]{RR}$ [Fridericia formula], $QTc = QT + 0.154$ $(1-RR)$ [Framingham formula], $QTc = QT + 1.75$ (heart rate-60) [Hodges formula]. QTc interval prolongation was defined based on the following rules: Male Rules: 1) Baseline <450 ms, and post HCQ >450 ms; 2) >15% increase over baseline post HCQ; and 3) baseline >450 ms and <500 ms, and post is >500 ms; Female Rules: 1) Baseline <470 ms, and post HCQ >470 ms; 2) >15% increase over baseline post HCQ; and 3) baseline >470 ms and <500 ms, and post is >500 ms. Statistics: Means were compared using independent sample t-tests; paired sample t-tests and proportions were compared using Chi square analysis. QT correction formulas were compared using 1-way ANOVA and post hoc analysis was done with Tukey correction. Binary univariate and multivariate regression were performed to determine risk factor predictors for QTc prolongation.

RESULTS We screened 444 consecutive patients with COVID-19 who were admitted to our hospital between March 10 and April 15, 2020, a total of 247 were excluded from this study because they met the exclusion criteria. Thus, 197 patients were included in the analysis. The mean baseline QTc interval calculated using the Bazett, Hodges, Frederica, Framingham methods were 451.0 ± 34.3, 425.1 ± 28.9, 417.2 ± 34.0, and 413.9 ± 31 ms, respectively. Of the 4 correction methods, 35.5% of all patients met the criteria for prolongation using the Bazett method. Of all patients included in the study 125 (63.5%) were male and 72 (36.5%) were female. Subjects were predominantly African American ancestry, 179 (90.9%). The mean age of all patients was 66.1 $\pm$ 13.3 years. The most common comorbidities were hypertension (74.6%), diabetes (55.3%), and hyperlipidemia (37.5%). Of all study participants, 91.7% received concomitant azithromycin; 31% of patients were on home beta-blocker therapy, while 27.9% were on home calcium-channel blockers. Of baseline electrocardiograms, 87.8% were sinus rhythm.

Total number of patients meeting prolongation criteria was less using the Hodges, Frederica, and Framingham methods. Mean QTc values in both genders are presented in (Tables 1, 2, 3, and 4).

All 4 methods showed statistically significant increases in QTc. Bazett had the relatively largest difference between pre- and post-

therapy QT interval with a mean difference of 14.48 ms. The increase was present in both men and women. The mean difference across sexes was largest using the Bazett method 16.43, but this was not statistically significant. Univariate analysis across all methods found that the concomitant use of azithromycin was not a significant predictor in QT prolongation across the Bazett, Hodges, Frederica, and Framingham methods. However, the presence of coronary artery disease was a statistically significant predictor for QT prolongation. The presence of congestive heart failure was also a predictor using the Hodges and Framingham methods. (Table 5, 6, 7, and 8) (Figure 1)
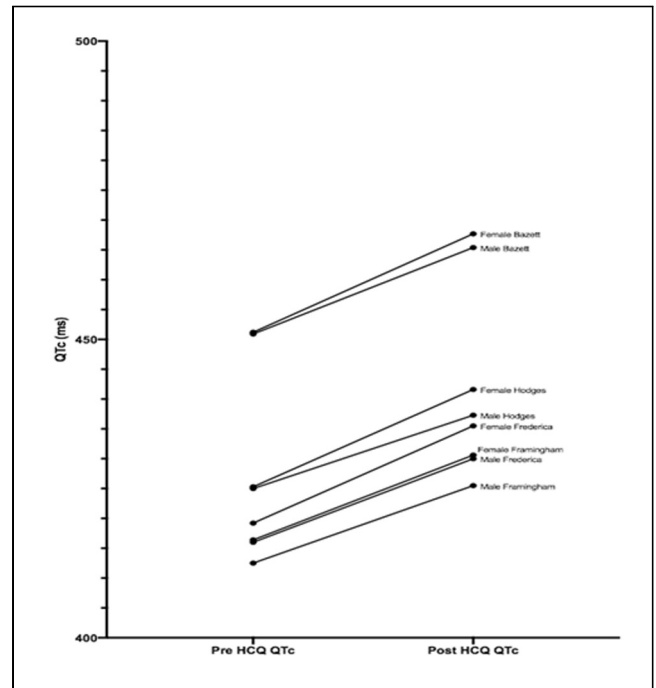

Figure 1. Mean Changes in QTc in Both Genders Based on Different QTc Correction Tools

ANOVA analysis across all subjects showed a significant difference between the four methods. There were significant differences between Bazett and 3 methods. The largest difference was between Bazett and Framingham, by 37.12 s. There was also a smaller difference between the Hodge and Framingham methods. The significant difference between the Bazzett method and the others also persisted across both men and women. The difference between Hodges and Bazzett was only significant in men (Table 9, 10, and 11). QT prolongation irrespective of the method used for correction did not predict mortality.

Table 1. Demographics Continuous Variables: QTc Calculation Using Different Equations for Both Genders

		All Patients (SD)		Male (SD)		Female (SD)		p Value
	Age	66.1	±13.3	66.2	±13.3	66.0	±13.5	0.928
	BMI	29.8	±7.0	29.4	±.8	30.6	±5.3	0.399
Baseline								
	Heart Rate	98.2	±21.0	99.7	±21.9	95.6	±19.1	0.187
	Bazett	451.0	±34.3	450.9	±37.6	451.2	±28.1	0.952
	Hodges	425.1	±28.9	425.0	±31.7	425.3	±23.5	0.942
	Frederica	417.2	±34.0	416.0	±35.9	419.2	±30.4	0.531
	Framingham	413.9	±31.1	412.5	±32.5	416.4	±28.6	0.395
Post HCQ								
	Heart Rate	97.9	±22.8	99.3	±21.7	95.5	±24.6	0.259
	Bazett	466.2	±41.3	465.4	±43.9	467.7	±36.7	0.712
	Hodges	438.9	±36.1	437.3	±37.6	441.6	±33.3	0.421
	Frederica	432.0	±42.5	430.0	±44.4	435.5	±39.0	0.385
	Framingham	427.4	±40.0	425.5	±41.3	430.6	±37.6	0.394

Table 2. Number of Patients With Prolongation on HCQ: Description of QTc Prolongation Using Different Equations

	QTc Formula	Male		Females		Total	
		n	%	n	%	n	%
>450 Post HCQ	Bazett	29	23.2	18	25	47	23.9
	Hodges	17	13.6	7	9.7	24	12.2
	Frederica	17	13.6	7	9.7	24	12.2
	Framingham	17	13.6	6	8.3	23	11.7
>15% Increase Post HCQ	Bazett	17	13.6	6	8.3	23	11.7
	Hodges	12	9.6	4	5.6	16	8.1
	Frederica	16	12.8	7	9.7	23	11.7
	Framingham	15	12	2	2.8	17	8.6
>500 Post HCQ (w/ baseline >450 Males/ >470 Females)	Bazett	14	11.2	3	4.2	17	8.6
	Hodges	4	3.2	0	0	4	2.0
	Frederica	3	2.4	0	0	3	1.5
	Framingham	3	2.4	0	0	3	1.5
Total Meeting Prolongation Criteria	Bazett	49	39.2	21	29.2	70	35.5
	Hodges	25	20	9	12.5	34	17.3
	Frederica	25	20	13	18.1	38	19.3
	Framingham	23	18.4	8	11.1	31	15.7

Table 3. Mean Differences in QTc Values for Both Genders (Post-HCQ Minus Pre-HCQ Comparison) Using Different Equations

	Comparison	Mean Difference (ms)	95% CI		p Value
Males					
	Bazett	14.48	6.93	22.02	<0.001
	Hodges	12.35	5.97	18.73	<0.001
	Frederica	13.95	6.76	21.14	<0.001
	Framingham	13.04	6.28	19.79	<0.001
Females					
	Bazett	16.43	8.20	24.66	<0.001
	Hodges	16.34	9.89	22.80	<0.001
	Frederica	16.26	8.22	24.30	<0.001
	Framingham	14.16	6.61	21.71	<0.001

Table 4. QTc Mean Differences Comparison Across Genders (Males vs. Females)

Comparison	Mean Difference (ms)	95% CI		p Value
Bazett	-1.95	-13.63	9.73	0.742
Hodges	-3.99	-13.68	5.69	0.417
Predictors	Beta	95%CI Lower	95%CI Upper	p Value
Frederica	-2.31	-13.52	8.90	0.685
Framingham	-1.12	-11.65	9.40	0.833

Table 5. Bazett Correction and Univariate Predictors of QT Prolongation

Predictors	Beta	95%CI Lower	95%CI Upper	p Value
Age	1.004	0.982	1.026	0.736
BMI	0.997	0.940	1.057	0.920
DM	1.557	0.865	2.805	0.140
HTN	1.154	0.594	2.242	0.673
HLD	0.936	0.513	1.707	0.828
CAD	0.416	0.197	0.881	0.022
CHF	0.556	0.239	1.296	0.174
TIA	1.216	0.441	3.355	0.705
PE/DVT	0.726	0.158	3.342	0.681
AFIB	1.091	0.264	4.504	0.904
COPD	1.722	0.534	5.555	0.363
Asthma	1.216	0.441	3.355	0.705
ESRD	0.665	0.284	1.555	0.346
AC	0.622	0.201	1.930	0.411
Azithromycin	1.692	0.622	4.602	0.303
Beta-Blockers	0.967	0.515	1.815	0.917
ACEI/ARB/ARNI	0.743	0.311	1.774	0.504
CCB	0.766	0.403	1.455	0.416

Table 6. Hodges Correction and Univariate Predictors of QT Prolongation

Predictors	Beta	95%CI Lower	95%CI Upper	p Value
Age	1.022	0.992	1.052	0.156
BMI	0.990	0.921	1.064	0.782
DM	0.987	0.469	2.078	0.972
HTN	0.724	0.294	1.782	0.482
HLD	0.832	0.392	1.768	0.633
CAD	0.414	0.176	0.975	0.044
CHF	0.245	0.099	0.608	0.002
TIA	0.545	0.182	1.630	0.278
PE/DVT	1.261	0.147	10.827	0.832
AFIB	0.382	0.091	1.613	0.190
COPD	3.345	0.427	26.218	0.250
Asthma	0.760	0.236	2.451	0.646
ESRD	0.479	0.182	1.257	0.135
AC	0.299	0.091	0.980	0.046
Azithromycin	1.538	0.469	5.043	0.477
Beta-Blockers	1.301	0.567	2.984	0.534
ACEI/ARB/ARNI	0.765	0.264	2.215	0.622
CCB	0.773	0.348	1.716	0.527

Table 7. Frederica Correction and Univariate Predictors of QT Prolongation

Predictors	Beta	95%CI Lower	95%CI Upper	p Value
Age	1.013	0.985	1.041	0.363
BMI	1.031	0.966	1.101	0.361
DM	1.161	0.571	2.362	0.681
HTN	0.743	0.316	1.748	0.496
HLD	1.197	0.570	2.515	0.635
CAD	0.415	0.181	0.951	0.038
CHF	0.729	0.270	1.972	0.534
TIA	0.637	0.214	1.893	0.417
PE/DVT	1	0.000	.	0.999
AFIB	0.461	0.110	1.932	0.289
COPD	1	0.000	.	0.998
Asthma	0.637	0.214	1.893	0.417
ESRD	0.565	0.217	1.470	0.242
AC	0.783	0.205	2.995	0.721
Azithromycin	0.888	0.242	3.259	0.858
Beta-Blockers	1.566	0.691	3.548	0.283
ACEI/ARB/ARNI	1.775	0.501	6.292	0.374
CCB	0.939	0.429	2.054	0.875

Table 8. Framingham Correction and Univariate Predictors of QT Prolongation

Predictors	Beta	95%CI Lower	95%CI Upper	p Value
Age	1.019	0.989	1.051	0.218
BMI	1.008	0.941	1.080	0.813
DM	0.888	0.409	1.931	0.765
HTN	0.517	0.187	1.429	0.203
HLD	0.687	0.317	1.492	0.343
CAD	0.355	0.149	0.846	0.019
CHF	0.262	0.103	0.666	0.005
TIA	0.671	0.207	2.175	0.506
PE/DVT	1	0.000	.	0.999
AFIB	0.616	0.122	3.121	0.559
COPD	1	0.000	.	0.998
Asthma	0.996	0.272	3.644	0.995
ESRD	0.539	0.196	1.480	0.230
AC	0.387	0.111	1.346	0.135
Azithromycin	1.163	0.314	4.313	0.821
Beta-Blockers	1.348	0.566	3.213	0.500
ACEI/ARB/ARNI	1.352	0.377	4.840	0.643
CCB	0.554	0.248	1.234	0.148

Table 9. The Mean Differences Between QTc Correction Equations Using ANOVA Comparison

Comparison	Mean	p Value	95% CI	
			LB	UB
QTcB vs QTcH	25.968	<0.001	17.622	34.314
QTcB vs QTcFri	33.843	<0.001	25.497	42.189
QTcB vs QTcFra	37.122	<0.001	28.776	45.468
QTcH vs QTcFri	7.875	0.073	-0.471	16.221
QTcH vs QTcFra	11.154	0.003	2.808	19.5
QTcFri vs QTcFra	-3.279	0.743	-11.625	5.067

Table 10. The Mean Differences Between QTc Correction Equations in Women Using ANOVA Comparison

Comparison	Mean	p Value	95% CI	
			LB	UB
QTcB vs QTcH	25.966	<0.001	14.001	37.932
QTcB vs QTcFri	32.030	<0.001	20.065	43.996
QTcB vs QTcFra	34.823	<0.001	22.858	46.788
QTcH vs QTcFri	6.064	0.557	-5.901	18.030
QTcH vs QTcFra	8.857	0.225	-3.109	20.822
QTcFri vs QTcFra	-2.793	0.931	-14.758	9.173

Table 11. The Mean Differences Between QTc Correction Equations in Men Using ANOVA Comparison

Comparison	Mean	p Value	95% CI	
			LB	UB
QTcB vs QTcH	25.969	<0.001	14.712	37.227
QTcB vs QTcFri	34.887	<0.001	23.630	46.144
QTcB vs QTcFra	38.447	<0.001	27.190	49.704
QTcH vs QTcFri	8.918	0.174	-2.340	20.175
QTcH vs QTcFra	12.477	0.023	1.220	23.734
QTcFri vs QTcFra	-2.793	0.847	-14.817	7.698

CONCLUSION It was notable that the longest QTc prolongation seen in this study was only 14.48 ms, using the Bazett formula. With other formulas, this prolongation was significantly smaller and so was the proportion of patients meeting QTc prolongation criteria. Not surprisingly, the Bazett formula again overestimated extend of QT prolongation. We can only speculate that the differences are perhaps related to the fact that our population was nearly exclusively African American. Common channels variation has been well documented to be a factor in QT prolongation, including drug-inducted QT prolongation. In the African-American ethnic subgroup, Ser1103Tyr-SCN5A is seen in approximately 8 % of population and can certainly explain our data. Furthermore, frequency of CAD and CHF was slightly higher than reported in other studies and both entities were associated with QT prolongation on our population. Reassuringly, the presence of QT prolongation was not found to be a predictor of mortality in our cohort.

CATEGORIES OTHER COVID-19 Lectures

TCT CONNECT-218

Transcatheter Therapies For COVID-19

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BACKGROUND Current strategies for COVID-19 therapy involve the systemic administration of drugs. While pharmaceutical treatments continue to be evaluated, device-based therapies have yet to be explored. We propose several transcatheter-based approaches for the treatment of COVID-19.

METHODS Four transcatheter-based solutions were explored in their potential uses for COVID-19 therapy: local drug delivery, energy-based and photodynamic therapy, and neuromodulation.

RESULTS First is local, catheter-directed delivery of therapeutics directly to the lungs. A localized delivery of therapeutics could increase the bioavailability of drug(s) at the site of action, in comparison to systemic delivery alone. A second approach is light-based therapy. Considering the antiviral, anti-inflammatory, antimicrobial, and vasculoprotective characteristics of visible light energy (380 to 750 nm), a localized, light-based catheter therapeutic approach could prove to be effective. Given the distinct features of COVID-19 disease progression and its attack on hemoglobin and porphyrins, we suggest the infusion of porphyrin-based photosensitizers (PS). COVID-19 has an affinity for PS and would attach to these molecules, which would reduce hypoxic symptoms and allow for their deactivation through the photo-activation or sonoactivation of PS molecules. A third approach considers that several studies have demonstrated that viruses hold electrical charges. Neutralizing the charge of the virus within an electrical field is feasible to reduce the viral load using pacing wires and catheters placed near lungs. A final approach is the neuromodulation of the host inflammatory response. In a small preclinical study, the release of proinflammatory cytokines was reduced following transcutaneous low intensity focused ultrasound treatment of the spleen.

CONCLUSION Several catheter-based therapies for COVID-19 were discussed. It should be noted that in all approaches, the combination of a catheter-based therapy with systemic pharmaceutical therapy is recommended. Robust clinical trials with clinically meaningful and relevant endpoints will be needed to assess the feasibility and safety of these approaches.

CATEGORIES OTHER COVID-19

TCT CONNECT-219

Psychosocial Impact of COVID - 19: Insights From a Cohort of Health Care Workers in the Cardiac Intensive Care Unit of a Tertiary Care Hospital in India

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BACKGROUND COVID-19 has been the catalyst for a quantum shift in our professional and personal lives, literally and figuratively within the blink of an eyelash. Healthcare workers (HCWs) have been profoundly impacted by this disruption at all levels, especially those working in high-stress specialties, such as cardiology, in resource-deprived and population-dense areas in developing countries, such as India. We studied the impact of COVID-19 on a cohort of HCWs working in a high-stress, high-turnover cardiac intensive care unit (CICU) of a tertiary care center in India. Questionnaires, results, and conclusions detailed in this presentation. Considering the fact that India has not even reached the peak of the pandemic, the negative psychosocial impact of COVID-19 on HCWs of the cardiovascular community is highly concerning and disheartening. Simplistic, sustainable long-term action plans are the need of the hour. We must use the cataclysm wrought by COVID-19 to plug our broken healthcare systems. For that, our frontline warriors should be in the best state of physical, mental, and emotional well-being to face up to this challenge. The time to take action is NOW!!

METHODS Evaluate the psychosocial impact of COVID-19 on HCWs working in a highly-stressed environment with high patient burden and turnover rates (45 bedded CICU including 15 step-down beds; average occupancy 90% to 100%). Understand perceived psychological burden and risk of post-traumatic stress disorder [PTSD] in these HCWs.

HCW Cohort	100
Cardiologists	10
Intensivists	04
Fellows-in-training	3
Resident Physicians	5
Medical Transcriptionists	2
Nurses	74
Technicians	02