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BRIEF COMMUNICATION

Implantable Cardioverter-Defibrillator Shocks During COVID-19 Outbreak

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BACKGROUND: COVID-19 was temporally associated with an increase in out-of-hospital cardiac arrests, but the underlying mechanisms are unclear. We sought to determine if patients with implantable defibrillators residing in areas with high COVID-19 activity experienced an increase in defibrillator shocks during the COVID-19 outbreak.

METHODS AND RESULTS: Using the Medtronic (Mounds View, MN) Carelink database from 2019 and 2020, we retrospectively determined the incidence of implantable defibrillator shock episodes among patients residing in New York City, New Orleans, LA, and Boston, MA. A total of 14 665 patients with a Medtronic implantable defibrillator (age, 66±13 years; and 72% men) were included in the analysis. Comparing analysis time periods coinciding with the COVID-19 outbreak in 2020 with the same periods in 2019, we observed a larger mean rate of defibrillator shock episodes per 1000 patients in New York City (17.8 versus 11.7, respectively), New Orleans (26.4 versus 13.5, respectively), and Boston (30.9 versus 20.6, respectively) during the COVID-19 surge. Age- and sex-adjusted hurdle model showed that the Poisson distribution rate of defibrillator shocks for patients with ≥1 shock was 3.11 times larger (95% CI, 1.08–8.99; *P*=0.036) in New York City, 3.74 times larger (95% CI, 0.88–15.89; *P*=0.074) in New Orleans, and 1.97 times larger (95% CI, 0.69–5.61; *P*=0.202) in Boston in 2020 versus 2019. However, the binomial odds of any given patient having a shock episode was not different in 2020 versus 2019.

CONCLUSIONS: Defibrillator shock episodes increased during the higher COVID-19 activity in New York City, New Orleans, and Boston. These observations may provide insights into COVID-19–related increase in cardiac arrests.

Key Words: cardiac arrest ■ COVID-19 ■ epidemiology ■ implantable cardioverter-defibrillator ■ pandemic

COVID-19 can affect the cardiovascular system, resulting in myocardial injury, intravascular thrombosis, arrhythmias, and sudden cardiac death.[1,2] Preliminary reports have suggested a temporal association between COVID-19 activity and cardiac arrests in the community and within in-hospital settings.[3,4] Although a higher burden of ventricular arrhythmic events has been observed among hospitalized patients with COVID-19, the mechanisms underlying the surge of out-of-hospital cardiac arrests remain unclear.[5–8]

Individuals with an implantable cardioverter-defibrillator (ICD) are at increased risk for arrhythmic sudden cardiac arrest.[9,10] Given the ability of these devices in providing continuous cardiac monitoring and therapy, we sought to determine if patients with an ICD or cardiac resynchronization therapy-defibrillator living in 3 metropolitan areas heavily impacted by COVID-19 experienced an increase in ICD shocks during the height of COVID-19 activity compared with the same time period in 2019.

METHODS

Using the Medtronic (Mounds View, MN) deidentified cardiac device remote monitoring (Carelink) database and COVID-19 case counts provided by Johns Hopkins University,[11] we retrospectively plotted COVID-19 incidence and ICD shock rates between February 1, 2020, and May 29, 2020, in select counties surrounding New York City, New Orleans, LA, and Boston, MA, and then overlaid shock episode rates from the same period in 2019. These large

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metropolitan areas were selected because of the combination of high ICD and COVID-19 prevalence. We excluded devices that were implanted within the past 90 days, those that had not transmitted data to Carelink in the past 365 days, and those that had not been programmed to enable wireless silent alerts for shocks. We counted ICD shocks detected within 10 minutes of each other as one episode. ICD shocks, rather than all ICD therapies, were studied because shocks trigger alerts that are transmitted to Carelink without manually instructing a download. Our predefined outcome variables were "at least one shock during the analysis time period" and "number of shocks during the analysis time period." We analyzed shock episode rates for time periods that depended on the area's COVID-19 data, starting with the first day where the area had >1 confirmed case and ending 3 weeks after the largest weekly number of new cases. We allowed for a 90-day buffer period for the transmissions to occur to capture >90% of all shocks that occurred during the analysis window, regardless of the alert status. Analysis included patients who were observed only in 2019, patients observed only in 2020, and patients observed in both years. These analyses used a binomial Poisson hurdle model[12] fit in R[13] using the pscl,[14] lmtest,[15] and sandwich[16–18] packages to account for zero-inflated count data and robust variance estimation[19] to account for repeated measurements from patients observed in both years. In this hurdle model, 2 sets of coefficients are estimated: the binomial coefficients model, the probability of a patient having at least one shock; and the Poisson coefficients model, the number of shocks assuming the patient had at least one.

This retrospective analysis of a deidentified data set was exempted from Institutional Review Board review and the requirement for informed consent was waived. The analysis was performed under the permissions granted through individual business associate agreements from each of the clinic sites participating in the Carelink network. The restrictions in the use of Carelink data, as governed by the business associate agreements, prevent us from sharing these data with other researchers. However, the analysis code will be available on request from the corresponding author.

RESULTS

A total of 14 665 patients with an ICD (n=9371; 64%) or cardiac resynchronization therapy-defibrillator (n=5294; 36%), transmitting data to Carelink, were included in the analysis. The mean age of the patients was 66±13 years, and 72% were men. The rate of newly confirmed COVID-19 cases steadily increased in March 2020 in all 3 cities (Figure 1), with peaks occurring in April 2020. The analysis time periods were defined as March 4 to May 1 for New York City, March 11 to May 1 for New Orleans, and March 6 to May 15 for Boston. We observed a larger mean rate of ICD shock episodes per 1000 patients during the 2020 analysis time period when compared with the same time period in 2019 for New York City (17.8 versus 11.7, respectively), New Orleans (26.4 versus 13.5, respectively), and Boston (30.9 versus 20.6, respectively). Spikes in ICD shock rates also appeared to be temporally associated with the COVID-19 surge (Figure 1). Using an age- and sex-adjusted binomial Poisson hurdle model and robust variance estimation, the Poisson episode rate for patients with at least one episode was 3.11 times larger (95% CI, 1.08–8.99; P=0.036) in New York City, 3.74 times larger (95% CI, 0.88–15.89; P=0.074) in New Orleans, and 1.97 times larger (95% CI, 0.69–5.61; P=0.202) in Boston in 2020 compared with 2019. However, the binomial odds of a given patient having at least one shock episode was not different during the 2020 analysis time period compared with 2019 (odds ratio [OR], 1.03 [95% CI, 0.74–1.45; P=0.84] in New York City; OR, 1.17 [95% CI, 0.68–2.00; P=0.57] in New Orleans; and OR, 0.98 [95% CI, 0.61–1.59; P=0.94] in Boston).

DISCUSSION

This retrospective, proof-of-concept study demonstrated an increase in ICD shock burden during the time period of peak COVID-19 activity in early 2020, in New York City, New Orleans, and Boston, compared with the same time period in 2019. Although the number of shocks was higher, the number of patients experiencing a shock did not change during the COVID-19 surge. These findings add to the evolving body of knowledge on the cardiovascular outcomes related to COVID-19 and may provide additional insight into possible mechanisms for the observed increase in out-of-hospital cardiac arrests.[3,4]

There is a paucity of data on the underlying rhythms at the time of out-of-hospital cardiac arrest among nonhospitalized patients, but a recent report on a cohort of hospitalized and critically ill patients with COVID-19 from a single US center suggested that most cardiac arrests were attributable to pulseless electrical activity or asystole.[7] These findings contrast earlier reports from China, demonstrating a higher burden of ventricular tachyarrhythmias.[5,6] Our findings extend these observations into nonhospitalized patients and are consistent with the earlier studies that demonstrated ventricular tachyarrhythmias being the predominant arrhythmia at the time of cardiac arrest. However, our observations appear to contrast those from a recent

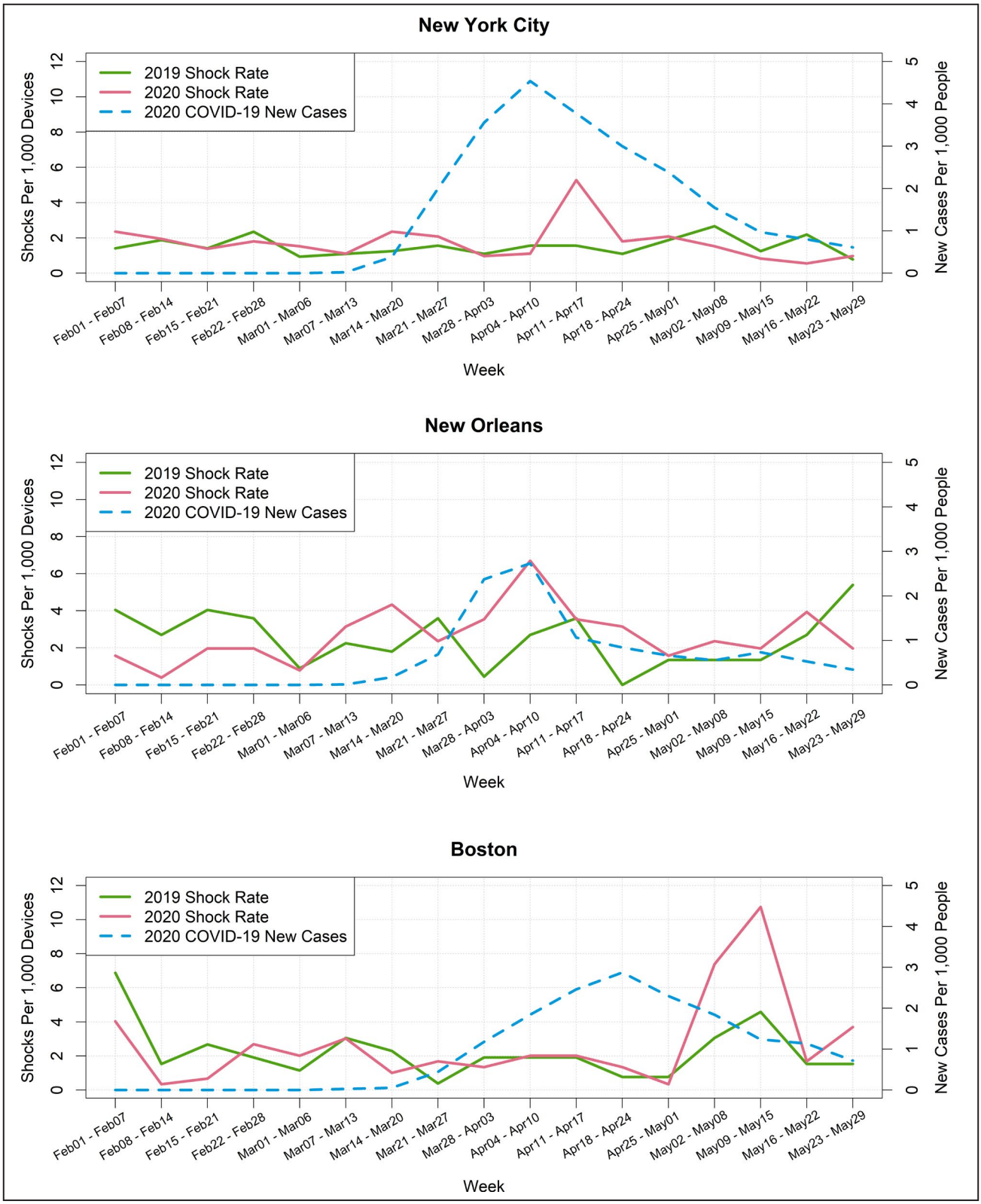

Figure 1. Incidence of implantable cardioverter-defibrillator shocks and COVID-19 in New York City, New Orleans, LA, and Boston, MA, in 2020.
ICD indicates implantable cardioverter-defibrillator.

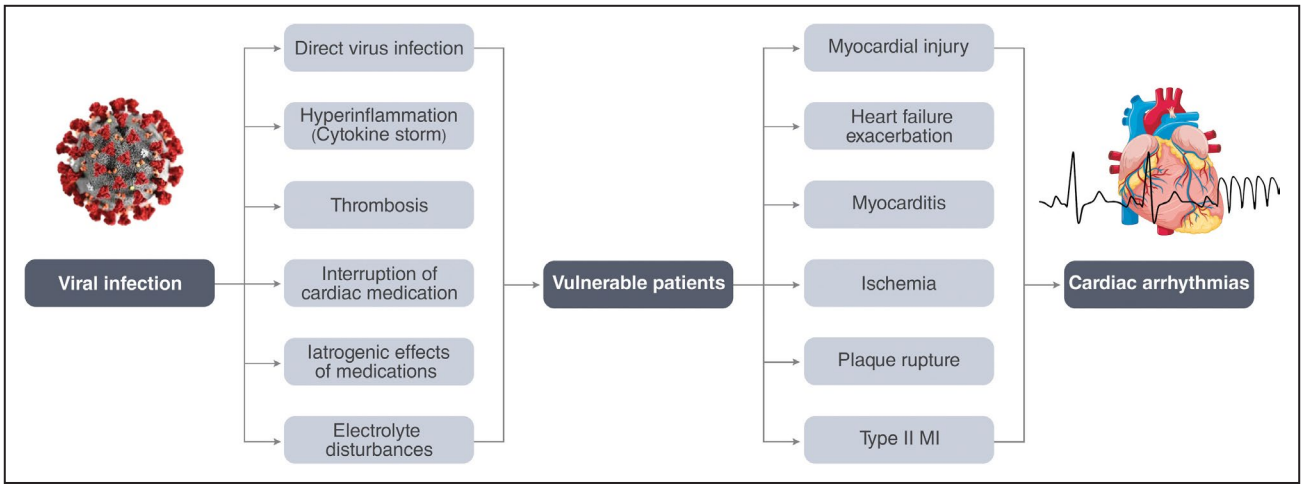

Figure 2. Potential mechanisms of cardiac arrhythmias in COVID-19.
MI indicates myocardial infarction.

report, demonstrating a 32% reduction in ICD therapies during the pandemic.[20] One important difference between these 2 analyses lies in their respective study populations. Although O'Shea et al analyzed ICD therapies across the population on a state level, our data originated from zip codes with the highest prevalence of COVID-19. Hence, our study population was more likely to be enriched with patients infected with COVID-19 and positioned to demonstrate the effect of COVID-19 infection on ICD therapies. The state-level analysis, on the other hand, could have been diluted by a larger number of uninfected patients and thus be better positioned to demonstrate the impact of other factors related to the pandemic, such as stay-at-home orders, reduced activity, or stress.

Although the mechanisms by which COVID-19 could trigger arrhythmic events are largely unknown, it is plausible that a suitable substrate for arrhythmias could be created by either the direct effect of the virus on the myocardium (eg, myocarditis) or indirectly through inflammatory-mediated plaque rupture, coronary thrombosis, ischemia, or other mechanisms predisposing the myocardium to arrhythmogenesis (Figure 2).[21] Indeed, there is precedent of the ability of viral infections in triggering arrhythmic events. Previously, Madjid et al[22] showed that patients with an ICD were more likely to have arrhythmias treated with shock or antitachycardia pacing during high influenza activity. Cumulatively, these observations add to the body of evidence that viral syndromes could trigger arrhythmias through direct and/or indirect effects on cardiovascular system (Figure 2). However, it is also possible that the observed increases in ICD shocks during COVID-19 pandemic were attributable to patients delaying medical care for the fear of catching COVID-19 at healthcare facilities.[23] Difficulties of access to cardiovascular medications along with high anxiety and stress during the locked-in phases might have also contributed to the increased ICD shock episodes.

Limitations

This hypothesis-generating study has some notable limitations. Patient-level information on whether the individuals who experienced ICD shocks also tested positive for COVID-19 was unavailable. Hence, these observations cannot prove a causal relationship between COVID-19 infection and ICD shocks.[24,25] However, this study showed a consistent relationship across 3 different geographical areas and a temporal relation to the COVID-19 peak surge. Furthermore, there is strong biological plausibility linking viral illness and cardiac arrhythmias (Figure 2), as observed previously.[22] Another limitation is that ICD events were not available for adjudication and therefore we cannot comment on the arrhythmic mechanisms prompting ICD shocks. However, prior reports on the specificity of ICD shocks suggest that most of the ICD shocks were attributable to ventricular tachyarrhythmias.[22] Last, only ICD shocks (rather than all therapies) were included in this analysis because shocks (unlike antitachycardia pacing events) trigger automated alerts in Carelink, thus allowing for timely transmission.

CONCLUSIONS

We report an increase in ICD shock burden around the time of high COVID-19 activity in New York City, New Orleans, and Boston in early 2020. These observations may provide additional insights in how the COVID-19 pandemic has impacted the heart and suggest a potential mechanism for the increased out-of-hospital arrests, observed during the pandemic.

ARTICLE INFORMATION

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