Predictors of arrhythmia other than QT interval prolongation and the use of β-blocker therapy in the coronavirus disease 2019 pandemic

**To the editor**  We have read with interest the article by Biernacka et al.1, the expert opinion of the Heart Rhythm Section of the Polish Cardiac Society on the safety of using antiviral and anti-inflammatory drugs that prolong the QT interval in patients with coronavirus disease 2019. In December 2019, treatment-resistant pneumonia occurred and then spread rapidly. The coronavirus that caused the first pandemic in the 21st century requires us to evaluate these patients in detail at each treatment stage, owing to both primary cardiac involvement and secondary cardiac adverse effects. At the same time, the group most affected by the outbreak and with the highest mortality rate are the elderly with known cardiovascular diseases. Although pulmonary insufficiency is the frequent cause of death in these patients, myocardial damage, heart failure, and malignant arrhythmias can also lead to death. The common adverse effect of the medications—prolongation of the QT distance—is considered as the primary electrocardiographic finding in these patients. However, other arrhythmia predictors, p dispersion, QT dispersion, and heart rate variability may also be the primary markers of malignant arrhythmias and mortality in these patients.

Takotsubo cardiomyopathy, hyperadrenergic response, and cytokine storm, accompanying QT prolongation, which can be observed in some cases in the course of the coronavirus disease 2019, suggest that this patient population may benefit from low-dose, controlled, cardiac β-blocker therapy. From this point of view, the number of patients receiving β-blockers in the study by Biernacka et al.1 and the course of QT distance in those cases may be a guide for us. Bronchospasm, an adverse effect of β-blocker therapy, limits the use of β-blockers in patients at high risk of pulmonary involvement. Low-dose, selective, cardiac β-blocker therapy may reduce the possibility of discontinuation of hydroxychloroquine and other treatments, at least in the low-risk, and perhaps moderate-risk, patient group.

**REFERENCES**


new pandemic situation when we have to face the unknown disease. The efficacy of experimental treatment is still under evaluation, as well as its safety, toxicity, and adverse effects. The management of adverse effects and potential toxicity of drugs applied in this new entity is even more challenging.1

Dr Tolunay suggested that low doses of cardiac selective β-blockers may reduce the possibility of discontinuation of medicines prolonging the QT interval in some patients. Nobody can agree more than we that β-blockers are the first-choice treatment in the prevention of torsade de points in patients with the prolonged QT interval. In our opinion, in patients with long QT syndrome or those with phenotypically mild mutations or polymorphisms in the long QT syndrome genes predisposing to drug-induced arrhythmias, β-blockers should be obligatory.2 Some reports show better safety of amiodarone or bepridil if applied in combination with β-blockers, owing to decreased QT dispersion.1,4 It is highly probable that β-blockers have a similar effect when used with anti-inflammatory and antiviral drugs.

On the other hand, bradycardia caused by β-blockers is an obvious risk factor for torsade de points. A mechanism of prolonging the QT interval by these drugs is similar to that observed in type 2 long QT syndrome. Thus, the efficacy of β-blockers is probably limited. Moreover, we do not know the impact of β-blockers on respiratory failure in patients with coronavirus disease 2019. Despite all the above objections, we agree that the concomitant use of β-blockers should be considered in some patients treated with antiviral and anti-inflammatory drugs prolonging the QT interval.

REFERENCES