Response to Letter Regarding Article, “Clinical and Genetic Determinants of Torsade de Pointes Risk”

We thank Dr Sacha for his reference to additional clinical syndromes in which the risk of torsade de pointes (TdP) can be increased. Although we previously highlighted the increased likelihood for TdP in the setting of both structural and ischemic heart disease, we did not specifically address the unique context of scenarios such as acute myocardial infarction, stress-related cardiomyopathy, and subarachnoid hemorrhage. As Dr Sacha points out, each of these entities may represent moments in which acquired transient QT interval prolongation and TdP may occur. However, the mechanism underlying the association between TdP and both acute and chronic cardiomyopathies remains elusive. To the point, Samuelov-Kinori et al examined 40 peer-reviewed reports of takotsubo cardiomyopathy associated with either TdP or acquired QT interval prolongation. They noted that 80% of patients noted to have takotsubo cardiomyopathy associated with TdP also had ≥1 traditional risk factor other than systolic dysfunction, including suspicion for congenital long QT syndrome, bradycardia, hypokalemia, or QT-prolonging medications. However, transient alterations in sympathetic tone could represent a common denominator for the increased TdP events observed with stress cardiomyopathy, acute intracranial events, severe acute illness, and acute myocardial infarction. Vaseghi et al demonstrated that both direct and reflex sympathetic activation (using isoproterenol and nitroprusside, respectively) lead to increased repolarization heterogeneity in patients with and without ischemic cardiomyopathy. Furthermore, there is increasing evidence for electromechanical coupling of repolarization heterogeneity as it relates to both systolic and diastolic dysfunction. In summary, we agree that acute cardiac events such as myocardial infarction, subarachnoid hemorrhage, and stress cardiomyopathies deserve special attention because they are at least transiently associated with both prolonged QT interval and increased TdP risk. However, further investigation is needed to better understand the mechanisms underlying acute cardiomyopathy, QT prolongation, and associated TdP.

Disclosures

None.

Andrew J. Sauer, MD
Cardiology Division
Feinberg School of Medicine
Northwestern University
Chicago, IL

Christopher Newton-Cheh, MD, MPH
Center for Human Genetic Research
Cardiovascular Research Center
Massachusetts General Hospital
Boston, MA

References

Response to Letter Regarding Article, "Clinical and Genetic Determinants of Torsade de Pointes Risk"
Andrew J. Sauer and Christopher Newton-Cheh

Circulation. 2012;126:e310
doi: 10.1161/CIRCULATIONAHA.112.133520

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 2012 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/126/19/e310