To the Editor:

I have read with a great interest the article by Sauer and Newton-Cheh addressing the important aspects of torsade de pointes (TdP).1 The authors briefly and elegantly summarize clinical and genetic determinants of TdP. However, one more clinical context that may be related to TdP needs to be mentioned, namely, the QT interval prolongation in acute cardiac diseases associated with large amount of stunned myocardium, ie, the reperfused myocardial infarction and the group of stress-related cardiomyopathies.2,3 The latter covers takotsubo cardiomyopathy and transient left ventricular dysfunctions associated with intracranial events (eg, subarachnoid hemorrhage), pheochromocytoma, exogenous catecholamine administration, or severe acute illnesses (especially those treated in intensive care units).3 In the course of these entities, deep inverted T-waves with QT interval prolongation can emerge in the first days after the index event. Halkin et al2 reported that 1.8% of the patients recovering from myocardial infarction with no evidence of ongoing ischemia developed TdP in the course of electrocardiographic evolution, ie, when their QT interval considerably prolonged. Similarly, a number of takotsubo cardiomyopathy cases with TdP during evolutionary changes in ECG (with QT prolongation) have been described.4 Also in patients with neurological disorders (especially those with subarachnoid hemorrhage), TdP usually appears when their QT interval is significantly prolonged with deep inverted T-waves.3 The common features of the cardiac abnormalities in these illnesses are transient contraction disturbances (probably reflecting stunned myocardium), as well as transient and evolutionary changes in ECG with the T-wave inversion and QT interval prolongation in their subacute phase. The mechanism of these changes is not clearly understood; however, the patients with the excessive QT prolongation may develop TdP. Hence, it is reasonable to consider these entities as a potential cause of acquired long QT syndrome and their subacute phase as a TdP threatening period. The excessive QT prolongation in these conditions may be a sign of some underlying predispositions to TdP; it has been proposed that if the QTc prolongs >500 ms, measures should be taken to monitor cardiac rhythm closely and prevent or treat TdP appropriately.4 Thus, for our medical practice, it is important to realize that not only drugs and electrolyte imbalances, but also some acute diseases associated with abundantly stunned myocardium may cause the acquired long QT syndrome.

Disclosures

None.

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References

Letter by Sacha Regarding Article, "Clinical and Genetic Determinants of Torsade de Pointes Risk"
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