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LETTER TO THE EDITOR

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# Reporting on ventricular arrhythmias in patients with Takotsubo syndrome

To the Editor:

Möller et al have provided a comprehensive review of "the prevalence, underlying mechanisms, prognostic implications, and management of strategies of ventricular arrhythmias (VA) in patients with takotsubo syndrome (TTS)".[1] Although the authors also discussed asystole, pulseless electrical activity, complete sinoatrial, and atrioventricular block, I was particularly interested in their exposition about ventricular tachycardia (VT), both monomorphic VT and torsade de pointes (TdP) in association with a prolonged QTc interval, and ventricular fibrillation. The authors' review was based on the information provided in the published heretofore literature on VA in patients with TTS. However one wonders what could be added in the future, in terms of details, on reporting about premature ventricular contractions (PVCs) and VA in patients with TTS, which could potentially contribute to the exploration of the pathophysiology of this puzzling disease. The following are pertinent remarks toward this goal: (a) the stability (or its variation) of the PVCs' and VA's electrocardiogram (ECG) QRS morphology and axis suggesting in the former case, that it emanates from a particular ventricular region; (b) the rate(s) of VT need(s) to be included; (c) the presence of right-, or left-bundle branch block-like morphology of the PVCs' and VA's QRS complexes of presumed left or right ventricular derivation, correspondingly, should be observed; (d) the morphology and axis of PVCs and VT need to be correlated with imaging data pertaining to regional myocardial contraction abnormalities; (e) the association of PVCs' and VA's QRS morphology and axis with regional distribution of T2-weighted cardiac magnetic resonance imaging (cMRI) regions of myocardial edema (ME) and/or T1-weighted cMRI via late gadolinium enhancement outlined regions of scar/fibrosis (S/F), of the various TTS variants, including those involving the right ventricle, and creating anatomical/functional patches favoring the emergence of a reentry substrate, as the authors hinted,[1] need to be observed; (f) the VA's QRS morphology and axis may be changing in parallel to regional distribution of ME and/or S/F changes, as reflected in the temporal course of T-wave changes in patients with TTS[2]; (g) the above are being supported by the association of the location of ME and the polarity of T-wave changes in TTS,[3] further emphasized by work showing that giant upright T-waves (instead of inverted) are seen in patients with the inverse variant of TTS; (h) as ME develops in the subacute phase of TTS, one may expect the amplitude of PVCs' and VA's QRS complexes to be attenuated, as the intrinsic QRS complexes, as previously reported[4]; (i) as the authors alluded to the probable major role of a hyperactivated autonomic sympathetic nervous system (ASNS) in the early phase of TTS,[1] it appears reasonable to examine the occurrence, severity, persistence, and recurrence of VA in patients with TTS, in the context of the fluctuation of the intensity of such ASNS activity via a currently available technology which employs acquisition/analysis of thoracic electrical signals in the 500-1000 Hz band, accomplished through routine ECG lead cable hook-up, a surrogate of the stellate ganglia electrical input to the heart[5]; (j) the authors state that "deep T-wave inversions are often accompanied by substantial QT interval prolongation (Wellens' sign)",[1] however, as this author repeatedly remarked in the literature, the original paper and subsequent to it publications of Wellens and his group, have not made any reference to a prolonged QTc in connection with the ECG repolarization changes in the setting of critical myocardial ischemia primarily due to severe lesions of left main or anterior descending coronary artery. I would be very interested in a commentary of the authors on the above.

## CONFLICT OF INTEREST

The authors declare no conflict of interest for this article.

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