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Towards a resolution of the mechanism of "spiked helmet ECG sign" in takotsubo syndrome and other acute life-threatening illnesses?

To the Editor:

I read with great interest the report by Simon and Járai, published in the *Journal* [1], about the "spiked helmet sign" (SHS) in the electrocardiogram (ECG) of a patient with takotsubo syndrome (TTS), associated with a complicated critical prolongation of the QT interval, macroscopic T-wave alternans (TWA), and torsade de pointes (TdP) ventricular tachycardia. The authors' proposal that the SHS is a type of manifestation of critically prolonged QT(U) is intriguing and may be correct, thus unifying mechanistically previous observations in which the upright trend of the ST-segment precedes or follows a spiked R-wave [2].

Littmann and Monroe described the SHS in 2011 [3], as an ECG sign associated with critical non-cardiac illnesses and high mortality, and subsequent publications drew attention to a connection of the SHS with chest and abdominal acute afflictions, characterized by an acute rise of intrathoracic and intraabdominal pressure, and implicitly attributing the SHS to non-cardiac mechanisms, catastrophic cerebral illnesses, and after stellate ganglion ablation, alluding to a common thread of adrenergic arousal [1,3–6].

This author [2] felt, reviewing previous experience, that there were similarities between SHS [3], and an ECG sign observed in patients with acute myocardial infarction, in its hyperacute phase, characterized by giant R-waves, life-threatening ventricular arrhythmias, and high mortality [7,8]; additionally it was observed that in those ECG examples, simulating the SHS, there were 2 variants, i.e., one with the spike preceding an upward baseline shift, and one with the spike following the upward baseline shift [2,7,8].

The authors, via the description of their reported case [1], and reference to the literature, build a case supporting the view that SHS is associated with TWA, giant T-waves, prolongation of the QT, and TdP. Indeed they hypothesize that "the SHS is another marker that might draw attention to the underlying, but not necessarily always evident long QT syndrome" [1]. Laundon's and Littman's [5] conceptualization that the observed baseline deformation in the SHS is due to an overlap of a grossly prolonged ventricular repolarization of the previous cardiac cycle, and its giant TU wave, and the succeeding QRS complex, makes a lot of sense. The authors' [1] further elaboration that any acute critical illness, leading to a prolong QT(U) and fast heart, could result in the T(U)-wave reaching the following QRS complex, and thus producing the ECG phenotype of SHS, is appealing [2,7,8] are related to varying degrees of abutting or overlap of a prolonged QT(U) intervals and QRS complexes.

The authors are wondering why the SHS is seen in connection with cardiac afflictions (including TTS, electrolyte disturbances and prolong QT syndrome) and critical abdominal and thoracic syndromes, and also cerebral illnesses; perhaps the unifying strength could be a hyperadrenergic state in all these pathophysiological disturbances. Along this lines of thought, this author suggests that elucidation of the mechanism may be forthcoming via the following actions: 1) When SHS is detected, ECG continuous monitoring should be instituted to evaluate the natural course of SHS and the effect of different administered therapies; it is conceivable that as the QT(U) gradually shortens, different SHS configurations will be observed, until the SHS is completely abolished. 2) Since a hyperadrenergic state is often implicated in acute critical illnesses, including TTS, it may be helpful to monitor the stellate ganglia adrenergic autonomic nervous input to the heart, employing currently available technology of monitoring thoracic skin adrenergic electrical potentials (a surrogate of stellate adrenergic stimulation of the heart), via routinely applied ECG chest electrode connection [9]. Indeed pari passu monitoring of the ECG and the thoracic autonomic adrenergic thoracic electrical potentials may not only elucidate the mechanism of the SHS, but delve into the pathophysiology of prolonged QT(U), TdP, giant T-waves, and macro-TWA, and micro-TWA.

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Declaration of Competing Interest

Nothing to disclose.

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