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The electrocardiographic spiked helmet sign: Is it real, artifact, or optical illusion?

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"*To invent, you need a good imagination and a pile of junk*"
[*Thomas Alva Edison*]

In 2011, a new electrocardiographic (ECG) phenomenon was described [1]. The spiked helmet sign (SHS) was characterized by a dome-and-spike appearance where sharp QRS complexes appeared like spears near the top of wide-based upward convex arcs. In the majority of cases, the ECG interpretation software indicated ST-segment elevation myocardial infarction (STEMI). Each patient, however, ruled out for STEMI. Rather, in almost all cases the SHS was associated with a critical non-cardiac illness and high risk of death [1]. Subsequent case reports and case series have shown that when the SHS was seen in the inferior leads, the patients usually had an acute abdominal event such as ileus, severe gastric distension or bowel perforation [1–4]. When seen in the chest leads, patients were found to have acute thoracic events such as tension pneumothorax or rupture of the thoracic aorta [4–6]. Resolution of the underlying condition resulted in prompt resolution of the SHS [3,4]. More recently a number of cases were described where the SHS appeared in conditions associated with marked QT prolongation such as profound hypocalcemia, the long QT syndrome, Takotsubo cardiomyopathy and intracranial or subarachnoid hemorrhage [7–11].

The exact cause of the SHS, including the mechanism of the apparent ST-segment elevation, remains uncertain. In cases where the SHS was in association with thoracic or abdominal events, it was suggested that the cyclic upward shift of the baseline that preceded and followed the QRS complexes probably reflected pulsatile epidermal stretch related to the acute thoracic or abdominal distension, respectively. Such a mechanism was supported by experimental data demonstrating that voltage of several millivolts can be generated by physically stretching the epidermis [12]. Electrodes in the vicinity of an arteriovenous fistula too can demonstrate spurious ST-segment elevations [13]. These observations suggested that the SHS was an electrocardiographic artifact where mechanical factors were superimposed on the intrinsic electrophysiological phenomena of cardiac depolarization and repolarization. Such mechanical causes, however, cannot explain the third category of the SHS that is due to marked QT prolongation.

In a recent issue of the Journal, Simon and Járai presented a case of the SHS seen in a patient with Takotsubo cardiomyopathy [14]. They too proposed that the SHS was a manifestation of critically prolonged QT(U). According to their theory, when the QT(U) is long enough and the heart rate is fast enough for the inverted T(U) waves to reach the following QRS complexes, then the upward and downward shift of the baseline simply reflect the ascending and descending limbs of the wide, inverted T waves. Therefore, the dome of the SHS and the associated ST-segment elevation are simply optical illusions where large segments of repolarization are below the baseline, and the apparently elevated ST segments actually reflect the normal baseline. This intriguing proposal raises the interesting question: is the SHS just an optical illusion and therefore, is it even real?

The presumed "mechanical" causes of the SHS, which are applicable to cases of acute abdominal and thoracic events, are similar to other markers in the ECG that are not generated by cardiac depolarization or repolarization but are still indicative of important physiological or pathophysiological processes. Examples include the respiratory artifact which suggests severe respiratory distress [15] and ECG artifacts associated with coughing, hiccupping and shivering that can be seen in the rewarming phase of hypothermia. The SHS related to extreme QT prolongation is different and it may very well be optical illusion. Optical illusions, however, are quite prevalent in electrocardiography. Examples include atrial flutter waves mimicking T-wave inversions or STEMI, and cases of marked first-degree AV block when P waves inscribed after the T waves of the previous cycles are mistaken for prolonged QT. Although controversial, experimental studies have suggested that even when the ST elevation is supposed to be real as in myocardial injury (STEMI) or pericarditis, it may still be illusory and caused by depression of the baseline including the PR and TP segments [16,17]. It must be remembered that in alternating-current recorded ECGs, apparent ST-segment displacement due to TQ-segment change cannot be differentiated from true alterations of the STs [17].

A possible way to distinguish real ST elevation from apparent ST elevation caused by depression of the PR and TQ segments is to use the base of the amplification (calibration) signal as the probable electrocardiographic baseline. Although ECG filter settings play a major role in the degree of baseline shift, using a high-pass (low-frequency) filter of 0.05 Hz, distortions and baseline undulations should be minimal. Fig. 1 demonstrates one of our recent cases where the SHS was the result of marked QT prolongation. The patient was a 49-year-old woman with Gitelman syndrome and profound hypokalemia, hypocalcemia and hypomagnesemia. Red lines indicate the bases of the spiked helmets and broken blue lines represent the bases of the calibration signals. Note that parts of the arcs of the spiked helmets were below but other parts, including portions of the apparent ST-segment elevation, were

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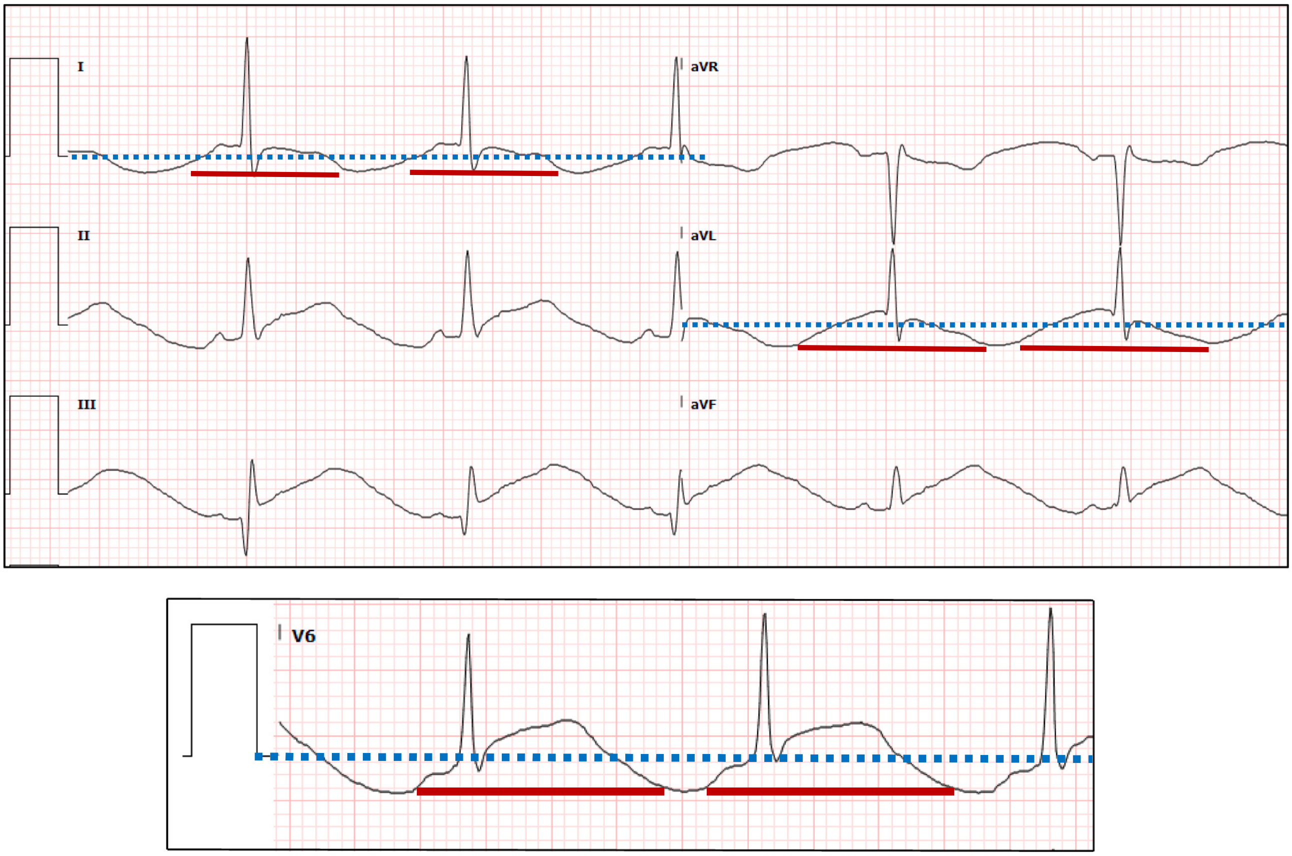

Fig. 1. Electrocardiogram recorded from a patient with profound electrolyte abnormalities. Note a spiked helmet pattern pseudo-ST elevation in multiple leads. Red lines denote the bases of the spiked helmets. Broken blue lines correspond to the base of the calibration signals. See text.

above this theoretical baseline. We have re-reviewed all of our cases of the SHS as well as all published cases of the SHS. In more than half, there was no amplification signal shown or there was marked baseline shift making similar measurements impossible. In many of the remaining instances, however, there was a similar half-and-half distribution of above and below baseline position of the arcs of the spiked helmets as is shown in Fig. 1. Based on these simple findings, therefore, it continues to remain uncertain how much of the SHS is an optical illusion vs. true elevation of the pre-QRS and post-QRS segments of the ECG.

Interestingly, there are distinct similarities between the SHS and the clinical-ECG entity called Brugada phenocopy (BrP). The ECG in BrP demonstrates a transient Brugada sign in patients who do not have the genetic Brugada syndrome [18]. Both in BrP and the SHS, an underlying and frequently non-cardiac condition mimics a primary cardiac ECG abnormality characterized by ST-segment elevation. Both disorders are frequently related to identifiable critical illnesses and in both, resolution of those conditions results in normalization of the ST segments. In BrP there is a low clinical probability of the true Brugada syndrome and in the SHS, there is a low likelihood of STEMI. In BrP, provocative or genetic testing usually rules out the Brugada syndrome and in the SHS, troponin testing or cardiac catheterization rules out STEMI.

So once again: is the SHS a real ECG phenomenon? From a clinical perspective, the answer is a definite yes. Even if it is an artifact or optical illusion, it is an important marker of certain critical illnesses whose early detection may be life-saving. It doesn't matter if we see a zebra as being white with black stripes or black with white stripes; what matters is that we recognize the zebra. The spiked helmet sign too may be an electrocardiographic "zebra" but its discovery, if placed in the correct clinical context, may guide the clinician towards the appropriate work-up and management of very sick patients. It may also help avoid unnecessary emergent cardiac catheterizations for presumed STEMI.

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