Spiked helmet sign: An under-recognized electrocardiogram finding in critically ill patients

Ajay Agarwal,1,2 Timothy G. Janz,1,2 Naga V. Garikipati2

A 77-year-old male patient presented with rhabdomyolysis. He developed progressive respiratory failure and acute respiratory distress syndrome during his hospital stay requiring mechanical ventilation. An electrocardiogram during mechanical ventilation showed findings suggestive of ST elevation myocardial infarction. Closer review showed dome and spike findings that have been likened to a “spiked helmet.” This finding has been associated with significant mortality. We discuss this under-recognized finding and the potential contributing mechanisms.

Keywords: Critical illness, electrocardiogram, mortality, myocardial infarction

Introduction

Electrocardiogram (ECG) is an important diagnostic tool for the prompt recognition of myocardial infarction (MI). There is a high incidence of clinically unrecognized myocardial damage among the critically ill. Identification of ST elevation MI by ECG in the critically ill patient population is limited by the lack of specificity due to a variety of non-ischemic processes.[1] The significance of these non-ischemic findings is not well-understood.

Case Report

This is a case report of a 77-year-old male patient who presented with generalized weakness, confusion, somnolence, recurrent falls and tea-colored urine for 3-4 days. His past medical history was significant for hypertension, chronic obstructive pulmonary disease and coronary artery disease with a remote history of MI. Physical examination revealed a confused and lethargic elderly male. His vitals showed blood pressure 97/42, heart rate of 51, respiratory rate of 12 and oxygen saturation of 93% on room air. There were extensive ecchymoses in different stages of healing on all 4 extremities and on the torso. Laboratory data were consistent with rhabdomyolysis and acute renal failure. ECG upon presentation revealed sinus rhythm with no abnormalities [Figure 1].

The patient was rehydrated with intravenous normal saline and nephrotoxic medications were withheld. His renal function improved and on the 2nd day, his chest X-ray revealed patchy bilateral infiltrates. On the 3rd day, he developed respiratory distress with an increase in oxygen requirements. The chest computed tomography revealed bilateral ground glass opacities with consolidation consistent with acute lung injury/acute respiratory distress syndrome. After failing a trial of non-invasive positive pressure ventilation, he was intubated and mechanically ventilated for hypoxic respiratory failure. He developed intermittent supraventricular tachycardia, atrial flutter and atrial fibrillation were treated with intravenous esmolol and diltiazem after which he converted to sinus rhythm. On the 4th hospital day, he developed increasing oxygen requirements and asynchronous volume control ventilation; his ventilator settings were switched to pressure control ventilation. An echocardiogram revealed left ventricular ejection...
fraction of 45-50% with grade II diastolic dysfunction and no wall motion abnormalities. Patient developed fever and broad-spectrum antibiotics were initiated. He later became progressively hypoxic and tachypneic on the ventilator during the next couple of days. He was paralyzed using vecuronium. His clinical status worsened and his ventilator settings were changed; the positive end-expiratory pressure (PEEP) was increased to 13 mm Hg. ECG at the time showed ST segment and T wave changes concerning for ST elevation MI. Careful review of the ECG revealed bizarre, broad-based apparent ST-segment elevation in the precordial leads where the upward shift of the baseline started slightly before the onset of the QRS complexes. A more obvious dome and spike pattern was seen in the inferior leads [Figure 2]. These findings are not consistent with true ST-elevation MI. His central venous pressure decreased from 12 to 7 mm Hg and tidal volumes were increased from 550 to 750 cc during this period. An urgent bedside echocardiogram showed preserved left ventricular ejection fraction with no wall motion abnormalities. The ventilator settings were changed and the PEEP was discontinued. Following this change, the ECG no longer showed the findings noted above [Figure 3]. The patient passed away on the 11th day of hospitalization from acute respiratory distress syndrome.

**Discussion**

Our case represents an uncommon finding of dome and spike ST-T wave changes mimicking ST elevation MI in the setting of a critical non-cardiac illness. These findings were similar to those reported by Littmann and Monroe[2] and Tomcsányi et al.[3] The ST-T wave changes in these cases were likened to a spiked German military helmet [inset Figure 2] the “Pickelhaube,” introduced in 1842 by Friedrich Wilhelm IV, the King of Prussia. The mechanism of this phenomenon is unknown and several postulated mechanisms for this finding include (1) artifact[4,5] (2) a sudden increase in the intra-abdominal or intra-thoracic pressure[2,3] (3) direct stimulation of inferior wall of left ventricle by the diaphragm,[6] (4) stimulation of the diaphragm by the left phrenic nerve, (5) synchronized contraction of diaphragm along with the cardiac cycle in the setting of alkalosis with latent tetany,[7] (6) acute stretch of skin overlying the chest wall with each cardiac cycle[8] and (7) diaphragmatic breathing that alters ECG patterns.[9] The acute rise in intrathoracic pressure with an increase in PEEP most likely contributed to the dome and spike pattern in our patient.

This abnormal ECG finding with a dome and spike “helmet sign” pattern in a critically ill patient is a marker for very high mortality. It is an under-recognized phenomenon and further research is needed as early recognition can possibly assist clinical management.

**References**

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