The Journal of Emergency Medicine, Vol. 57, No. 3, pp. 390–394, 2019

0736-4679/$ - see front matter

https://doi.org/10.1016/j.jemermed.2019.06.003

Visual Diagnosis in Emergency Medicine

UTILIZATION OF THE ELECTROCARDIOGRAPHIC "SPIKED HELMET" SIGN IN THE DIAGNOSIS OF INTRA-ABDOMINAL PATHOLOGY WITHIN THE EMERGENCY SETTING

David H. Cisewski, MD, MS,*† John E. Madias, MD,*‡ and Lillian Wong, MD†

*Icahn School of Medicine at Mount Sinai, New York, New York, †Department of Emergency Medicine, Elmhurst Hospital Center, Elmhurst, New York, and ‡Division of Cardiology, Elmhurst Hospital Center, Elmhurst, New York

Corresponding Address: David H. Cisewski, MD, Mount Sinai Department of Emergency Medicine, 1 Gustave L. Levy Place, New York, NY 10029

INTRODUCTION

The differential diagnosis of electrocardiographic (ECG) ST-segment elevation includes a multitude of cardiopulmonary pathologies, including myocardial infarction, pericarditis, myocarditis, pulmonary embolism, takotsubo syndrome, ventricular aneurysm, and Brugada syndrome, among others (1). However, under the appropriate clinical presentation, this ECG finding expands to include extrathoracic pathology as part of the differential. A lesser-known manifestation of ST-segment elevation referred to as the "spiked helmet" sign (SHS) may represent a key diagnostic clue to the emergency provider of a critically evolving intra-abdominal pathology. We present a case in which the ECG findings of the SHS provided a diagnostic clue and expedited the diagnosis and management of a patient with severe intra-abdominal pathology.

CASE REPORT

A 52-year-old Hispanic man with unknown medical history was brought to the emergency department (ED) by emergency medical services (EMS) after bystanders discovered the patient unresponsive following a witnessed collapse and presumed cardiac arrest. After an immediate call to 911 and an EMS arrival time of approximately 4 min, EMS noted an initial rhythm of asystole with advanced care life support initiated approximately 5 min post-collapse. The patient was intubated in the field, resuscitative compressions were performed throughout transport to the ED, intraosseous access was obtained, and a single intraosseous dose of 1 mg epinephrine was administered during transit. In the ED, resuscitation efforts continued with ongoing chest compressions. Femoral vein central access was obtained, and 3 additional doses of 1 mg epinephrine, 2 ampules of 10% calcium chloride, and 2 ampules of 8.4% sodium bicarbonate were administered. At approximately 30 min post-collapse the patient obtained return of spontaneous circulation (ROSC) without administration of electrical shock. The post-ROSC ECG rhythm showed irregular, narrow QRS complexes, T-wave inversions in leads 1 and aVL, ST-segment elevations in leads V1 and aVR, and precordial lead ST-segment depressions consistent with global ischemia (Figure 1).

The patient's initial vital signs after ROSC included a blood pressure of 74/51 mm Hg, a heart rate of 117 beats/min, a temperature of 36.1°C, and a respiratory rate of 14 breaths/min (intubated, oxygen saturation 95%, 100%

Reprints are not available from the authors.

Received: 28 March 2019; Final submission received: 1 June 2019;
Accepted: 8 June 2019

FiO_2). On initial physical examination, there was no evidence of head trauma, his pupils were equal and reactive bilaterally, the lungs were clear to auscultation bilaterally, his abdomen was soft and mildly distended without evidence of trauma or decreased bowel sounds, and his extremities were cold and pale with decreased capillary refill. The initial ultrasonographic assessment (Rapid Ultrasound for Shock and Hypotension) showed no evidence of free fluid in the abdomen nor pericardial effusion (2). An emergency physician–conducted bedside transthoracic echocardiogram demonstrated an estimated left ventricular ejection fraction of 35% with hypokinetic regional wall motion abnormalities along the anterior, anteroseptal, and lateral walls.

After the initiation of a vasopressor support (norepinephrine infusion) and a balanced crystalloid (Plasma-Lyte; Baxter International Inc., Deerfield, Illinois) intravenous fluid infusion, hemodynamic stability was maintained with a mean arterial pressure of 80–85 mm Hg (systolic blood pressure maximum 90–100 mm Hg). A repeat ECG conducted 1 h post-ROSC showed ST-segment elevations with an upward shift preceding the QRS complexes in the inferior leads II and aVF (Figure 2). Urgent bedside examination was notable for a diffusely distended, tympanic abdomen with diminished bowel sounds. Repeat bedside echocardiogram showed a left ventricular ejection fraction 35–40% and regional wall motion abnormalities consistent with the initial evaluation. Intra-abdominal ultrasound showed a small accumulation of free fluid in the hepatorenal recess. The beside monitor displayed a lead II rhythm consisting of a continuous "spiked helmet" pattern that was also noted on the repeat ECG (visual recreation in Figure 2, bottom).

A nasogastric tube was placed and 600 mL of bilious gastric content was suctioned from the stomach. An emergent computed tomography scan of the abdomen and pelvis demonstrated moderate to severe inflammation of the gallbladder, appendix, and throughout the colon concerning for diffuse hypoperfusion. Repeat laboratory values were notable for a lactic acidosis and transaminitis strongly suggestive of ischemic hepatitis and diffuse colitis caused by a prolonged period of post–cardiac arrest hypoperfusion. A repeat ECG 2 h after nasogastric decompression and vasopressor support demonstrated resolution of the ST-segment elevations in the inferior leads (SHS), ST-segment elevations in V1 and aVR, and precordial ST-depressions as seen on the presenting ECG (Figure 3). A repeat transthoracic echocardiogram showed a mildly improved estimated left ventricular ejection fraction of 40–45% with visually estimated improvements of the regional wall motion abnormalities initially seen along the anterior, anteroseptal, and lateral walls. Despite resuscitative efforts, the critical severity of the patient's presentation prevented immediate surgical intervention and despite aggressive continued support following transfer to the medical intensive care unit, the patient died 24 h after presentation to the ED.

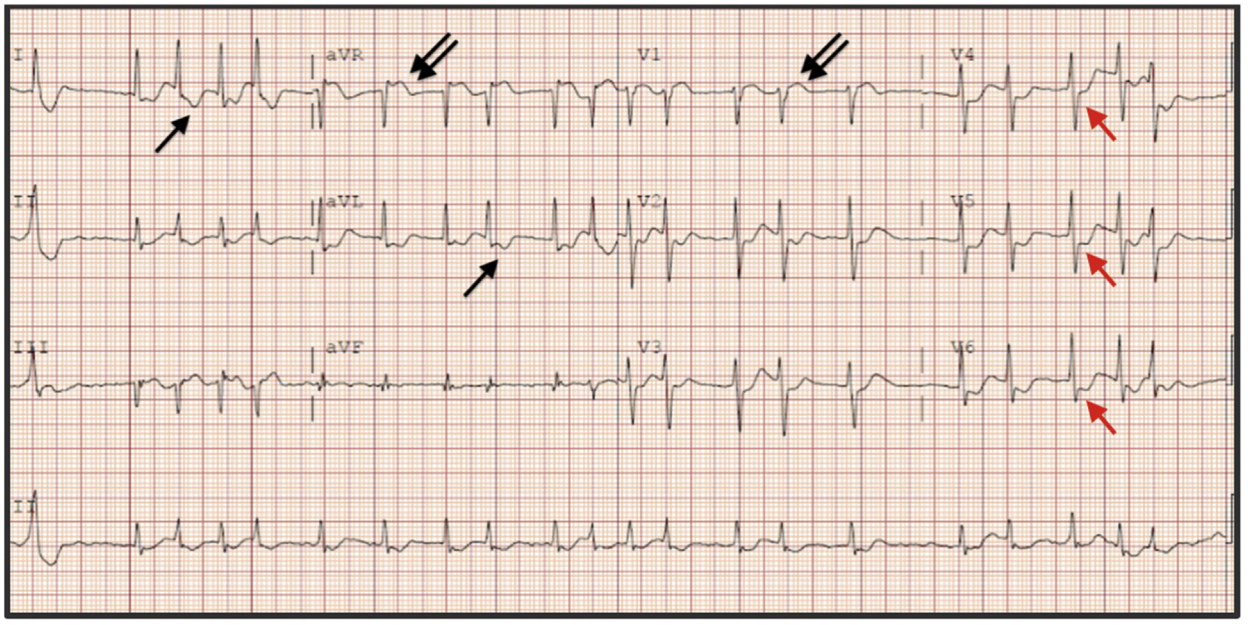

Figure 1. Electrocardiogram after return of spontaneous circulation. The irregular, narrow QRS complexes, T-wave inversions in leads 1 and aVL (single black arrow), ST-segment elevations in leads V1 and aVR (double black arrow), and precordial lead ST-segment depressions (single red arrow) are consistent with global ischemia.

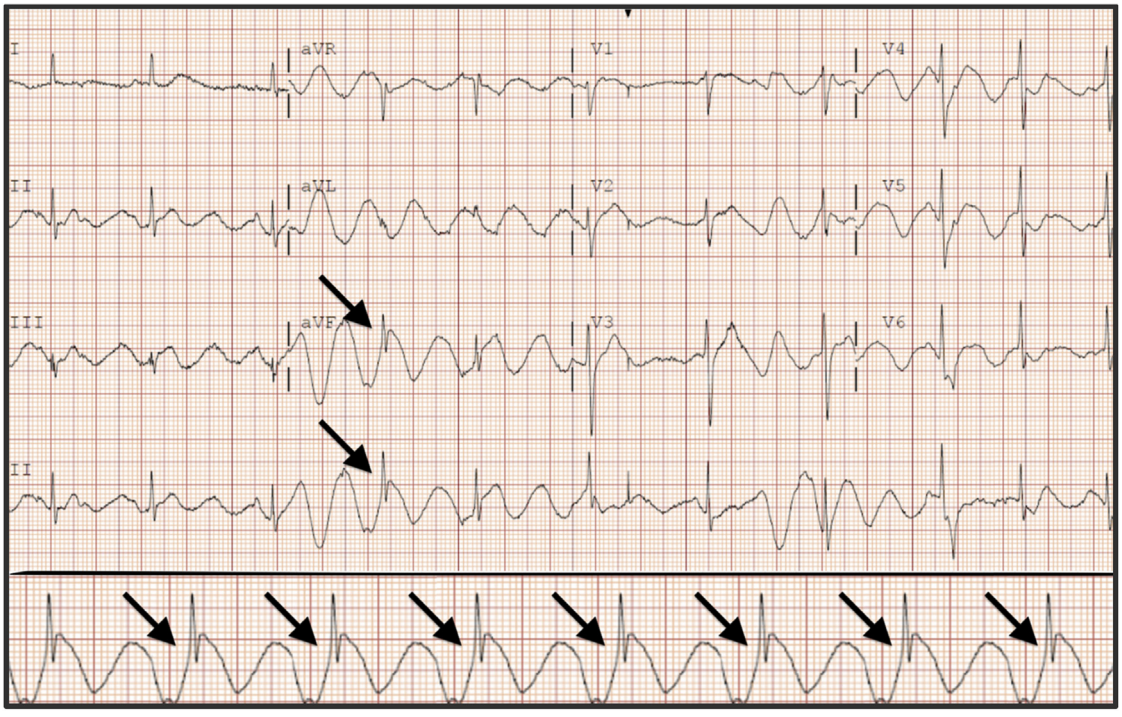

Figure 2. "Spiked helmet" sign. Repeat electrocardiogram demonstrating upward shifting ST-segment elevations preceding the start of a QRS complexes (black arrow) in leads II and aVF, concerning for an intra-abdominal pathology (top image). The bedside monitor displayed a lead II rhythm consisting of continuous "upward shifting" ST-segment elevations preceding the start of each QRS complexes (black arrow) (bottom image).

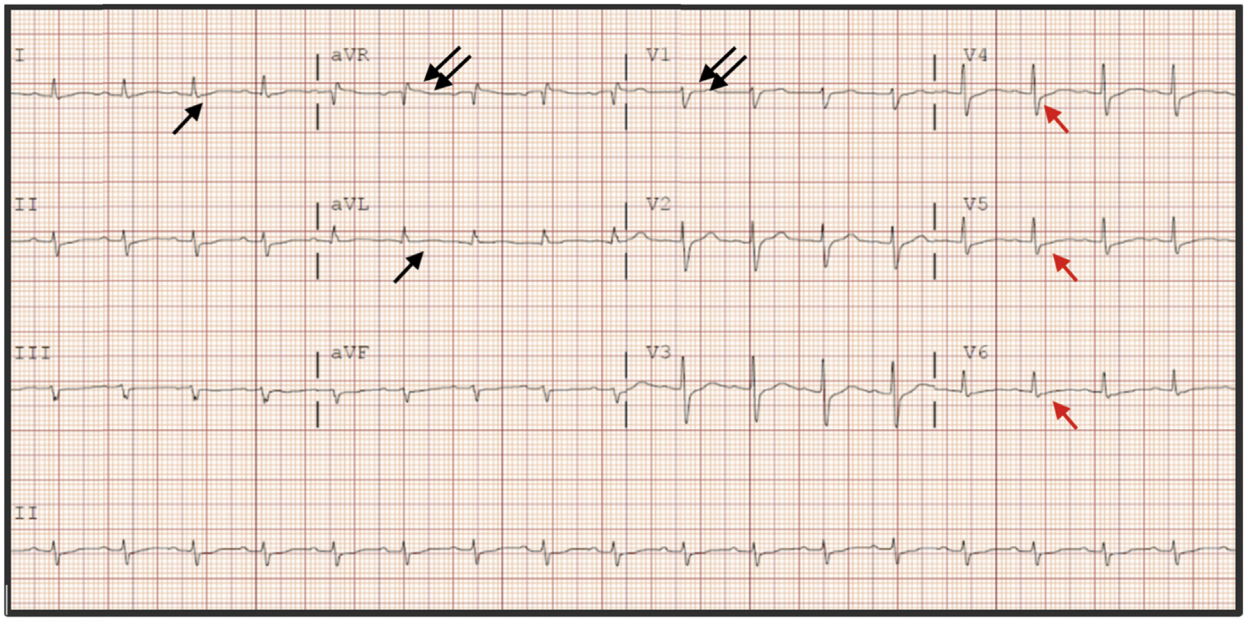

Figure 3. Electrocardiogram after nasogastric decompression and vasopressor support. Full resolution of the T-wave inversions in leads 1 and aVL (single black arrow), ST-segment elevations in leads V1 and aVR (double black arrow), and precordial lead ST-segment depressions (red arrow) as seen on the presenting electrocardiogram (Figure 1).

DISCUSSION

The SHS has classically been described as an upward shifting ST-segment preceding the start of a QRS complex, predominantly in the inferior leads II, III, and aVF (3–5). The name of this electrocardiographic sign is derived from its resemblance to the pickelhaube ("pickaxe bonnet"), a military helmet popularized by King Frederick William IV of Prussia and later adopted by the German and Bavarian armies (Figure 4) (5). This ECG manifestation has been noted among critically ill patients in the intensive care setting with retrospective correlation of intra-abdominal pathology and impending death (3,5).

Gastric dilatation, intestinal obstruction, hepatobiliary inflammation (cholecystitis, hepatitis), and pancreatitis have each resulted in this key ECG finding mimicking an inferior (or inferolateral) myocardial infarction (7–12). Changes in heart position during gastric distension, coronary vasospasm caused by an elevated vagal tone, and an irritative effect on the heart by diaphragmatic elevation have all been suggested as underlying causes of the ST-elevations (13). Increased intracavitary pressure resulting in a pulsatile epidermal stretch has also been posited as a cause of the ECG changes (5,14). Though predominantly seen among the inferior ECG leads (II, III, and aVF), presumably because of their proximity (or abutment) to the diaphragm, which separates the heart from the intra-abdominal pathology, similar ECG findings isolated to the precordial leads have been associated with intrathoracic pathology (5,14).

Myocardial infarction should always be the first consideration in the setting of ST-segment elevations. However, prompt consideration and recognition of alternative diagnoses, as in our case report, is essential to expediting patient care. The underlying pathologies associated with these ECG changes carry a high risk of mortality among presenting patients. Not considering alternative diagnoses can result in diagnostic anchoring on cardiovascular pathology and a delayed management of the actual pathology (15).

As for our case, it was suspected that a prolonged period of hypoperfusion during a presumed cardiac arrest and post-ROSC hypotension attributed to significant intra-abdominal ischemia. Of note, the possibility of an underlying intra-abdominal pathology could not be ruled out as the source of the critical presentation rather than a sequela of a suspected cardiac arrest. Unlike many of the reported cases of SHS correlated retrospectively in the intensive care setting, our case represents real-time

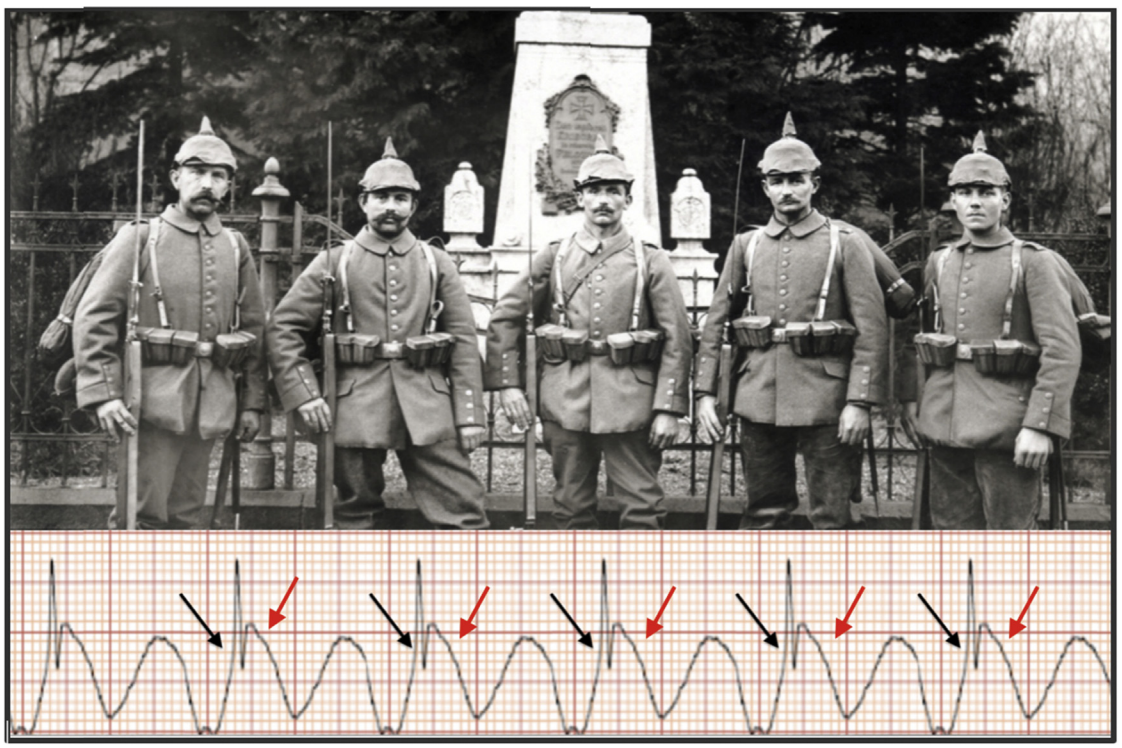

Figure 4. "Spiked helmet" sign and the pickelhaube. The upward shift of the preceding the QRS complex (black arrow) followed by the visualized ST-segment elevation (red arrow) associated with intra-abdominal pathology resembles the pickelhaube ("pick-axe bonnet") popularized by King Frederick William IV of Prussia and later adopted by the German and Bavarian Army as seen by these posing Bavarian infantrymen (used with permission of the Brett Butterworth Collection) (6).

ECG recognition of the SHS and an expedited workup based on the high suspicion of intra-abdominal pathology that had not previously been considered (16). Administration of pressor support and gastric decompression resulted in a slow but steady resolution of the ECG findings. This is consistent with previously reported cases of the SHS showing similar resolution with nasogastric decompression (9,13,14,17). To our knowledge, this is the first reported case of the SHS aiding in the expedited diagnosis and workup of an intra-abdominal pathology seen in a patient being evaluated within the emergency setting.

REFERENCES

1. Hanna EB, Glancy DL. ST-segment elevation: differential diagnosis, caveats. Cleve Clin J Med 2015;82:373–84.
2. Seif D, Perera P, Mailhot T, Riley D, Mandavia D. Bedside ultrasound in resuscitation and the rapid ultrasound in shock protocol. Crit Care Res Pract 2012;2012:503254.
3. Agarwal A, Janz TG, Garikipati NV. Spiked helmet sign: an underrecognized electrocardiogram finding in critically ill patients. Indian J Crit Care Med 2014;18:238–40.
4. Tomcsanyi J, Fresz T, Bozsik B. ST elevation anterior "spiked helmet" sign. Mayo Clin Proc 2012;87:309.
5. Littmann L, Monroe MH. The "spiked helmet" sign: a new electrocardiographic marker of critical illness and high risk of death. Mayo Clin Proc 2011;86:1245–6.
6. Somers J. Imperial German field uniforms and equipment 1907-1918, volume 3. Atglen, PA: Schiffer Publishing, Limited; 2007.
7. Frais MA, Rodgers K. Dramatic electrocardiographic T-wave changes associated with gastric dilatation. Chest 1990;98:489–90.
8. Asada S, Kawasaki T, Taniguchi T, Kamitani T, Kawasaki S, Sugihara H. A case of ST-segment elevation provoked by distended stomach conduit. Int J Cardiol 2006;109:411–3.
9. Mixon TA, Houck PD. Intestinal obstruction mimicking acute myocardial infarction. Tex Heart Inst J 2003;30:155–7.
10. Ryan ET, Pak PH, DeSanctis RW. Myocardial infarction mimicked by acute cholecystitis. Ann Intern Med 1992;116:218–20.
11. Furuhashi M, Uno K, Satoh S, et al. Right bundle branch block and coved-type ST-segment elevation mimicked by acute cholecystitis. Circ J 2003;67:802–4.
12. Makaryus AN, Adedeji O, Ali SK. Acute pancreatitis presenting as acute inferior wall ST-segment elevations on electrocardiography. Am J Emerg Med 2008;26:734.e1–4.
13. Herath HM, Thushara Matthias A, Keragala BS, Udeshika WA, Kulatunga A. Gastric dilatation and intestinal obstruction mimicking acute coronary syndrome with dynamic electrocardiographic changes. BMC Cardiovasc Disord 2016;16:245.
14. Tomcsanyi J, Fresz T, Proctor P, Littmann L. Emergence and resolution of the electrocardiographic spiked helmet sign in acute noncardiac conditions. Am J Emerg Med 2015;33:127.e5–7.
15. Mattu A, Tabas J, Brady W. Electrocardiography in emergency, acute, and critical care. Washington, DC: American College of Emergency Physicians; 2019.
16. Littmann L, Proctor P. Real time recognition of the electrocardiographic "spiked helmet" sign in a critically ill patient with pneumothorax. Int J Cardiol 2014;173:e51–2.
17. Patel K, Chang NL, Shulik O, DePasquale J, Shamoon F. Small bowel obstruction mimicking acute ST-elevation myocardial infarction. Case Rep Surg 2015;2015:739147.