It seems but isn't it?

Parece, mas, não é?

Parece pero no es?

Colleagues Opinions

Dear Andrés,

I'm sending best wishes to you and your family for a happy New Year and also wishes for a productive 2023. Let's hope the world is in a better place.

The challenging case is one that looks like a classic inferolateral STEMI, but your question **"it seems but isn't?"** likely indicates that "it isn't". So, what do we have that mimics a classic STEMI? The case indicates that she has metastatic cancer to the liver and lungs. She also is in heart failure (dyspnea, lung crackles, leg edema, JVP elevation, gallop rhythm). Clearly she needs a trip to the cath lab as per STEMI protocol, but an ECHO might be also included to rule out pericardial disease or tamponade related to her metastatic disease and/or other lung pathology that might compress one or more coronary arteries. Metastasis into

coronary arteries could also account for STEMI ECG findings. I look forward to your comments and those of your colleagues.

In the meantime, today (Dec 31) I'm reminded of that popular song: "What Are You Doing New Years Eve?" I don't know about you, but my wife and I are playing pinochle with friends.

All the best,

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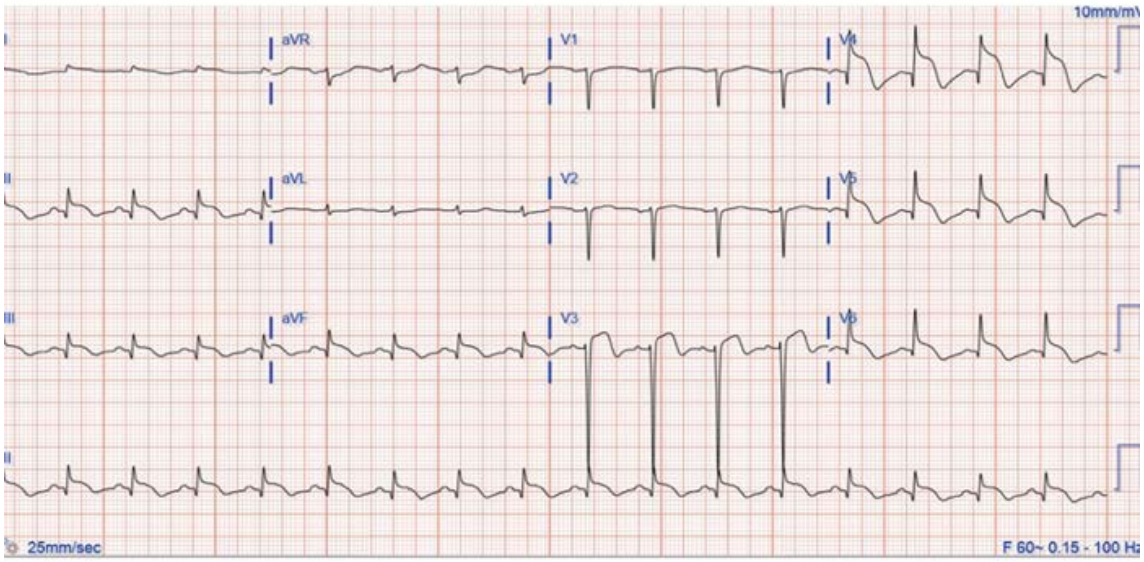
Case Presentation

A patient in their 60s presented to the emergency department with a 2-week history of shortness of breath and back pain exacerbated that morning. The medical history was noteworthy for a remote history of cervical cancer. The patient was recently found to have a vesicovaginal fistula and metastatic lesions in the liver and lungs. Percutaneous bilateral nephrostomies were placed weeks before presentation. The patient also had a history of 40 pack-years of smoking but no known cardiovascular disease. On admission, blood pressure was 105/65 mm Hg, heart rate was 94 beats per minute, and respiratory rate was 16 breaths per minute. Jugular veins were mildly elevated, and cardiac examination was notable for a gallop and a soft systolic ejection murmur. Lung examination demonstrated basilar crackles, and there was grade 2 and higher lower-extremity edema. A 12-lead electrocardiogram (ECG) was performed on arrival (Figure 1).



Questions: Does this patient have an ST-segment elevation myocardial infarction (STEMI), and what are the next steps in managing this patient?

Interpretation and Clinical Course

The initial ECG revealed sinus tachycardia; ST elevation in leads aVF, II, III, and V_3 through V_6; and ST depression in aVR (Figure). The QRS complex demonstrated a sharp R wave followed by convex ST elevation. Diffuse T-wave inversions and a prolonged QT interval were also noted. Due to these ECG findings, the patient underwent urgent coronary angiography, which revealed nonsignificant coronary artery disease. Testing results revealed a pro–brain natriuretic peptide of 17 286 pg/mL (reference, <125 pg/mL; to convert to ng/L, multiply by 1.0) and high sensitivity troponin T of 772 ng/L (reference <14 ng/L; to convert to µg/L, multiply by 1.0), which decreased to 715 ng/L at 2

hours. Echocardiogram findings showed systolic apical ballooning with hypokinetic apex, apical septum, and inferolateral wall. Left ventricular ejection fraction was 33%. Systolic anterior motion of the mitral valve was also noted. The patient was given guideline-directed medications for heart failure with reduced ejection fraction. However, on day 5, they developed hypotension, with signs of hypoperfusion secondary to cardiogenic shock as evidenced by dynamic left ventricular obstruction and echocardiography-worsened ejection fraction (25%) on repeated echocardiogram. The shared decision was made to transition to comfort measures only. The ECG findings remained unchanged until the patient developed ventricular tachycardia, after which they rapidly deteriorated and died.

Discussion

The ST elevation's peculiar morphology is known as the Spiked Helmet Sign (SHS) due to its resemblance to the military helmet, the Pickelhaube.[1] It is characterized by an upward shift preceding the QRS complex, which has a sharp R wave followed by a convex ST elevation.[2]

The spiked morphology is given by the QRS complexes buried in the dome-shaped pseudo–ST elevation. This STEMI mimic is characterized by an upsloping shift preceding the QRS complex, which appears to align with the

ST elevation. In the present case, the upsloping shift was observed in V_4 through V_6 and the convex dome-shaped pseudo–ST elevation in the inferior leads and from V_4 through V_6.

The SHS has been widely described throughout the precordial leads in critically ill patients with hyperadrenergic states such as sepsis, intracranial hemorrhage, stellate ganglion ablation, and, as in the case of this patient, **Takotsubo cardiomyopathy** (TCM).[3-5] The SHS is thought to occur due to prolonged repolarization or extremely prolonged QT intervals, with the upward shift of the helmet representing a prior giant T-U wave overlapping with the QRS complexes.[6] Although the precise electrophysiological correlate remains elusive, the SHS is associated with critical illness and, as in the current case, portends a dismal prognosis.[1]

Takotsubo cardiomyopathy is a clinical and electrocardiographic mimic of acute coronary syndrome because it might present with chest pain, dyspnea, cardiogenic shock, or life-threatening arrhythmias. Its onset is usually preceded by an inciting emotional, physical, or combined trigger.[7] The ECG usually demonstrates dynamic changes classically described with 3 temporal stages: initially ST deviations occur within hours of onset, followed by T

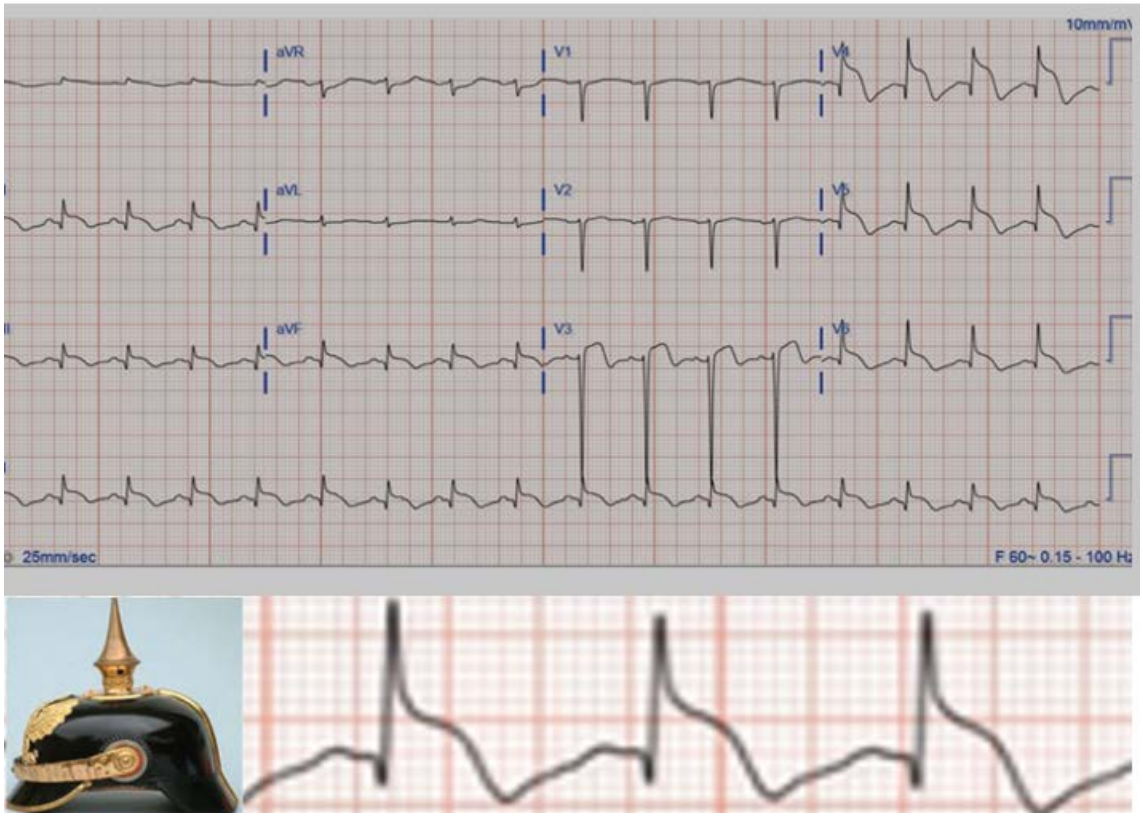
wave inversions and QT prolongation.[8] Finally, ECG alterations resolve after weeks to months, lagging the recovery of ventricular function. Differentiation from acute coronary syndrome could be particularly challenging in the setting of ST elevation. Frangieh et al[9] proposed several electrocardiographic characteristics useful to differentiate **TCM from STEMI with more than 95% specificity, albeit low sensitivity (<25%): (1) ST depression in aVR, (2) ST depression in aVR with no ST elevation in V_1 and no abnormal Q waves, and (3) ST depression in aVR with ST elevation in anterior, inferior, or anteroseptal leads.** In the present case, the patient presented with all of the characteristics proposed by the authors. In addition, the presence of diffuse T-wave inversions and prolonged QT interval was highly suggestive of TCM. Although the demonstration of coronary arteries without considerably obstructive disease is necessary to diagnose TCM,[7] the timing of catheterization can be individualized based on the ECG findings and clinical presentation.

Cardiogenic shock presents in up to 10% of cases of TCM, particularly in those with sustained anterior ST elevations and severely impaired left ejection fraction.[10] In addition, up to 15% of patients present with left ventricular outflow tract obstruction, which, as in the present case, contributes to the hemodynamic derangements and limits the options for

resuscitation (as vasoactive agents usually exacerbate the outflow obstruction).

Take-home Points

- The SHS is a STEMI mimic characterized by an elevation of the isoelectric line preceding the QRS complex, which has a sharp R wave followed by a convex ST elevation. See ECG below



- The SHS is associated with critical illness and hyperadrenergic states. It confers an ominous prognosis.

- Several ECG characteristics, such as ST depression in aVR plus the absence of ST elevation in V_1 and abnormal Q waves or ST elevation in anterior, inferior, or

anteroseptal leads, are highly specific (but not sensitive) for TCM. However, coronary angiography is required for diagnosis.

- Up to 10% of patients with TCM develop cardiogenic shock, particularly those with severely impaired left ventricular dysfunction, left ventricular outflow tract obstruction, and persistent ST elevations.
- ECG insights Given the acute nature of takotsubo, it has been tempting to speculate on ECG changes that may add in the specificity of the diagnosis. However, there is a considerable variation in the presenting ECG, similar to that seen in acute MI. Patients with takotsubo can present with:

    I. A normal ECG (11%),

    II. ST/T wave changes, (39%),

    III. ST-elevation (39%),

    IV. Transient LBBB (4%)

    V. Arrhythmias (atrial tachycardias, heart block and ventricular arrhythmias) (7%).

    VI. The head-to-head comparison between the 12-lead ECGs of the ST-elevation-presenting

patient groups suggests a larger spread of ST-elevation, which is non-localising in acute takotsubo, whereas the amplitude of ST-elevation seems overall less than that of a typical MI ECG. [12]

VII. No acute ECG findings alone or in combination are specific enough to obviate or delay urgent cardiac catheterization.

VIII. The most characteristic takotsubo ECG feature remains the prolongation of QT/QTc interval, seen at presentation and peaking 24–48 hours thereafter; however, larger comparative studies will need to inform if this is a reliable differentiating feature from MI after correcting for gender differences in QTc since most takotsubo patients are women and the reverse is the case for MI. QTc prolongation is one of the factors considered to increase the risk of early arrhythmic complications without a direct correlation between the magnitude of QTc and the arrhythmic risk.

IX. Takotsubo recovery is characterized by significant repolarization abnormalities (deep T-waves), which are protracted, sometimes

repeaking later[12] and which suggest that the myocardial healing process in this condition is different from that of MI.

Final conclusion

Spiked Helmet sign (SHS) during Takotsubo cardiomyopathy, Takotsubo syndrome (TTS), "Broken Heart Syndrome" or Stress-Induced Cardiomyopathy

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