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ST-segment elevation in a critically ill patient: Greek letters, war helmets, and broken hearts

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1 | ECG FOR STUDENTS AND ASSOCIATED PROFESSIONALS

A 57-year-old female was admitted to intensive care unit for severe COVID-19 pneumonia. Her immediate past history was notable for hysterectomy followed by radiotherapy for carcinoma cervix 4 months back, and exploratory laparotomy for subacute intestinal obstruction 1 month before current hospitalization. Evaluation revealed signs of abdominal infection with rigidity and purulent discharge from the suture line. She was hypotensive and acidotic requiring inotropic support and invasive ventilation. A 12-lead electrocardiogram (ECG, Figure 1A) was performed because ST-segment elevation was observed on ICU telemetry on second day of admission. A second ECG was repeated 2 hours later (Figure 1B).

What are the findings in the ECGs in Figure 1? How should these observations be analyzed?

2 | COMMENTARY

The QRS-ST complex in Figure 1A is characteristic of the "lambda wave" ST-segment elevation pattern in leads I, II, III, aVF, and V3 to V6. There is a steep upslope coinciding with the ascending branch of the R wave, an elevated J wave and a downsloping ST-segment elevation with marked slurring. There is no elevation of the isoelectric line preceding the QRS complex. The amplitude of ST-segment elevation exceeds that of the R wave in precordial leads. This peculiar electrocardiographic finding represented by the downsloping ST-segment elevation resembles the ancient Greek letter, λ, hence the name.

In Figure 1B, there is resolution of ST-segment elevation in inferior leads without development of q waves. There is slight diminution of R wave amplitude in leads V4-V6. The notable change is the appearance of the characteristic spiked-helmet sign (SHS) in leads V3 to V6. There is elevation of the isoelectric line preceding the QRS complex, followed by a sharp R wave and then convex (and not downsloping) ST-segment elevation.

Transthoracic echocardiography showed left ventricular systolic dysfunction with circumferential akinesia of the apex in the mid-ventricular and apical segments, along with hyperdynamic contraction of the basal segments (Figure 2). There was elevation of serum troponin levels on serial measurements. The serum levels of sodium, potassium, and calcium were normal. Cardiac catheterization was not performed.

Considering the clinical setting of septic shock with inotropic and ventilatory requirement combined with the atypical ST-segment elevation pattern and echocardiographic observations, a possibility of Takotsubo cardiomyopathy was considered. ECG repeated after 2 hours showed normalization of ST-segment elevation in inferior leads. After 12 hours, there was complete normalization of ST-segment elevation in precordial leads as well (Figure 1C), thereby supporting the diagnosis of non-acute coronary syndrome related ST-elevation pattern (NASTEP). No arrhythmias were noted during this period.

Lambda waves have been described in association with early repolarization syndromes, idiopathic ventricular fibrillation (VF), acute myocardial infarction, propofol infusion syndrome (PRIS) and recently, Takotsubo cardiomyopathy.[1,2] The mechanism is not clearly understood. In animal models of ischemia associated with lambda-like ST-segment elevation, depletion of adenosine triphosphate

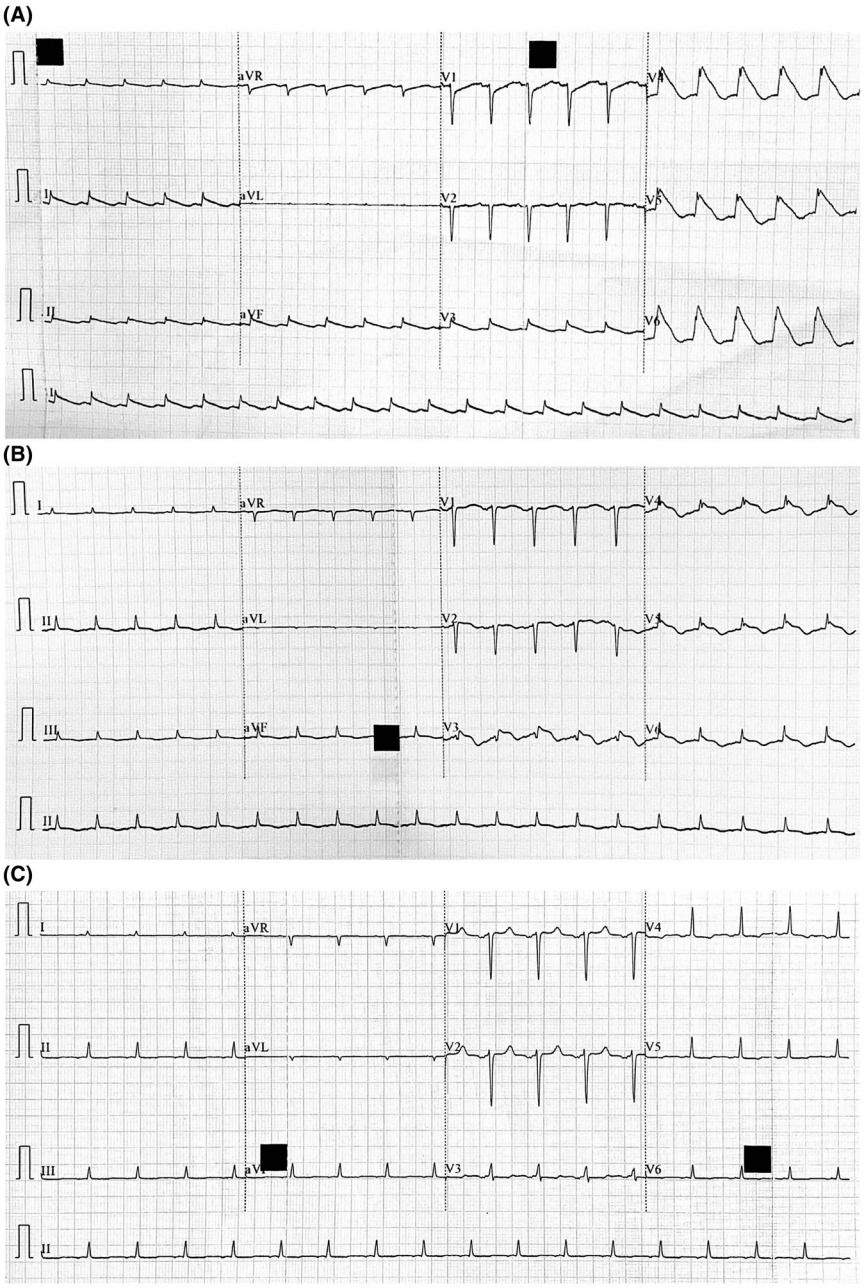

FIGURE 1 A, Initial electrocardiogram (ECG) with ST-segment elevation in leads I, II, III, aVF, and V3 to V6, showing the lambda sign, with resemblance of the pattern of ST-segment elevation to the Greek letter, λ. B, Repeat ECG after 2 hours with reduction in ST-segment elevation in inferior leads, with no development of q waves. The ST-segment elevation in leads V3 to V6 show the Spiked-Helmet Sign, named for its resemblance to the Prussian military helmet, the Pickelhaube. There is elevation of the isoelectric line preceding the QRS complex, followed by a sharp R wave and then convex ST-segment elevation. C, ECG after 12 hours showing complete normalization of ST-segment changes without development of q waves.

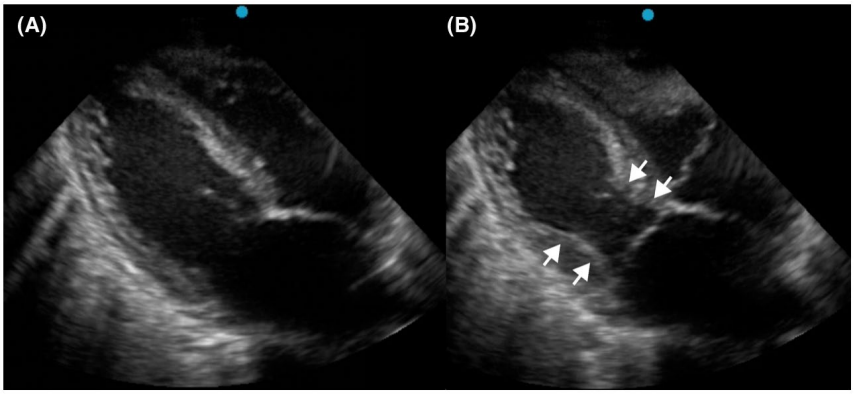

FIGURE 2 Transthoracic echocardiogram (A) Diastole (B) Systole. Echocardiogram showing reduced left ventricular ejection fraction with circumferential akinesia of the apex in the mid-ventricular and apical segments and hyperdynamic contraction of the basal segments (arrows)

has been demonstrated to decrease the inward sodium and calcium currents which in turn leads to augmentation of the transient outward current (Ito). Since the distribution of Ito channels is not homogenous, this results in transmural or spatial heterogeneity of voltage gradients giving rise to this peculiar pattern of ST elevation.[3]An additional non-ischemic mechanism may explain the genesis of lambda wave in Takotsubo cardiomyopathy.[1,2] The electro-mechanical feedback caused by the wall motion abnormalities leads to earlier epicardial repolarization and augments the endo-epicardial voltage gradient. Transmural voltage gradients are known to produce phase 2 reentry and subsequent VF similar to that seen in Brugada syndrome. This is presumably why lambda waves have been described in cases of idiopathic VF, acute myocardial infarction complicated by VF and as poor prognostic markers in Takotsubo cardiomyopathy.[1,3]

The Spiked Helmet Sign (SHS) has been named for its resemblance to the Prussian military helmet, the Pickelhaube.[4]Though SHS mimics acute myocardial infarction, there is a characteristic upward shift in the baseline preceding the QRS because of delayed giant T-U waves. The ST-segment is in continuity with this shift completing the "helmet" of SHS, with QRS forming the spike on it. First described in cases of acute abdominal or thoracic pathology, it has subsequently been reported in a variety of acute conditions like intracranial hemorrhage, sepsis, and metabolic derangements.[4]The mechanism is presumed to be a prolongation of repolarization similar to that seen in long QT syndrome secondary to catecholaminergic excess seen in these conditions. Recently, SHS has been described in association with Takotsubo cardiomyopathy also.[5]

This is the first report where both signs have been observed at different stages in the same patient in association with Takotsubo cardiomyopathy. A unifying explanation will be the shared pathophysiology of adrenergic excess lending credence to the hypothesis that both signs are part of a spectrum of abnormalities in repolarization. Awareness of these different possibilities is important and can help prevent unnecessary and potentially harmful interventions. Careful attention to the electrocardiogram and the clinical context remains critical in decision making.

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