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Electricity, Heart and ST-Segment Elevation: A Closer Look

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Dear Editor,

We read with great interest the article by Uyanık et al., entitled "Delayed ST-Segment Elevation Due to Electrical Injury Mimicking Acute Myocardial Infarction," which was published recently in this journal. This case report was mentioning a young patient admitted with late-onset chest pain and ST-segment elevation following electrical injury (1). Although the case was presented precisely, some points merit further highlighting.

Abnormal electrocardiography (ECG) may be found in approximately 31% of patients following an electric shock (2). Non-specific ST-segment changes and sinus tachycardia are the most commonly reported ECG findings; QT prolongation, bundle branch block, atrial and ventricular fibrillation, and atrial and ventricular premature contractions are also detected. As referred to in this case report, ST-segment elevation with or without myocardial involvement may follow electrical injury.

In a clinical setting, ST-segment elevation on ECG may be related with many conditions, such as myocardial infarction, early repolarization, electrolyte imbalance, and pericarditis. In this case, myocardial infarction may easily be excluded, since cardiac biomarkers are normal and ST elevation is not consistent with myocardial infarction. In this case, ECG shows diffuse and concave ST elevation (elevated at the J point) with the exception of V1 and aVR. In acute myocardial infarction, ST elevation is also accompanied by reciprocal ST depressions. In the case of early repolarization, ST elevation is most often present in the mid- to lateral chest leads (V3-V6), and the majority of subjects with early repolarization has no ST deviations in the limb leads. So, early repolarization would be excluded in this patient, who has pronounced ST elevation on D2, D3, and aVF.

Zeana describes a 65-year-old electrocuted subject who experienced precordial pain, serous enzymes of negative myocardial necrosis, and ST elevation during 2 weeks of hospitalization (3). These findings suggest the possibility of widespread pericardial involvement. In the case presented by Uyanık et al., pericardial involvement seems to be the most possible cause of ST changes in the admission ECG. This ECG shows us sinus bradycardia with diffuse concave ST elevation except for V1 and aVR. ST depression in V1 and aVR is also a typical finding for acute pericarditis. Late-onset chest pain also supports this condition. However, addition of a follow-up ECG of the patient to the report would be more valuable for confirmation of this diagnosis. In our opinion, pericarditis deserves discussion as a possible diagnosis in the aforementioned case.

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