Cerebral Hemorrhage: Precipitating Event for a Tako-tsubo-like Cardiomyopathy?

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To the Editor:

It was with great interest that we read the article by Rahimi et al.¹ Surprisingly, the authors speculate that, despite findings of electrocardiographic anterolateral ST-segment elevation, increased cardiac biomarker levels, and transient left ventricular apical ballooning in a patient with a decreased level of consciousness, acute intracranial hemorrhage, not tako-tsubo cardiomyopathy (TC), was responsible for the cardiac abnormalities.¹ Furthermore, their conclusive statement regarding the etiology of the described acute and convalescent ST changes, be it intracranial hemorrhage or TC, is questionable as precordial leads demonstrating resolution of anterolateral ST-segment elevation are not included in the convalescent 6-lead electrocardiogram.

Classical electrocardiographic changes accompanying intracranial hemorrhage consist primarily of T-wave abnormalities, including deeply inverted precordial T-waves, QTc interval prolongation and, less frequently, ST-segment depression or elevation.^{2,3} Moreover, the authors' identification of ST-segment elevation and left ventricular dysfunction without significant epicardial coronary stenoses, termed "neurogenic stunned myocardium," has previously been described.⁴

The combination of anterolateral ST-segment elevation, raised cardiac biomarker levels, and transient apical and mid-left ventricle wall akinesis identified in this case is the hallmark of TC.⁵ The question we pose is whether differentiating between the "classic" presentation of TC and the cardiac abnormalities accompanying intracranial hemorrhage is, in fact, possible. Intracranial hemorrhage has previously been associated with TC,⁶ as have multiple additional conditions associated with overwhelming sympathetic tone,^{4,5,7} suggesting that the relationship between intracranial hemorrhage, electrocardiographic and left ventricular abnormalities, and TC may be due to a common intermediary.

A mounting body of evidence is implicating surges of sympathetic nervous system output as the pathophysiologic factor responsible for the electrocardiographic, enzymatic, and wall motion abnormalities in TC.^{5,6} As Rahimi et al., identify, a similar hypothesis has been advanced to explain the electrocardiographic and left ventricular abnormalities accompanying intracranial hemorrhage.⁵ Indeed, it is becoming increasingly plausible that apical and mid-left ventricular stunning accompanied by anterolateral electrocardiographic ST-elevations and elevations in cardiac biomarkers may represent a final common pathway for the effects of overwhelming sympathetic discharge, whatever its cause.

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