J Cardiovasc Imaging. 2021 Jul;29(3):234-235
https://doi.org/10.4250/jcvi.2021.0076
pISSN 2586-7210·eISSN 2586-7296

Journal of Cardiovascular Imaging JCVI

Editorial

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Level of Troponin Release Can Aid in Early Exclusion of Stress-induced (Takotsubo) Cardiomyopathy

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▸ See the article "Early Differentiation of Stress Cardiomyopathy from Acute Anterior Wall Myocardial Using Changing Cardiac Enzyme Patterns" in volume 29 on page 228.

Received: May 5, 2021
Revised: Jun 25, 2021
Accepted: Jun 27, 2021

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Conflict of Interest
The author has no financial conflicts of interest.

When it was first identified, stress cardiomyopathy was thought to be a benign form of heart failure due to its self-limiting clinical course, but it is now recognized to be associated with a non-negligible risk of catastrophic consequences such as ventricular arrhythmias, systemic thromboembolism, and cardiogenic shock.[1)2)] Although stress cardiomyopathy initially was thought to be a rare occurrence, the frequency now is believed to be 1% to 2% of individuals with suspected acute coronary syndrome (ACS) due to increased awareness and detection.[3)4)] It is estimated that 15–30 cases per 100,000 people per year are diagnosed with stress cardiomyopathy, but the actual incidence is unknown because the condition likely is underdiagnosed.[5)] Although stress cardiomyopathy has similarities to myocardial stunning due to ischemia secondary to coronary stenoses, it differs in cellular mechanisms. When a high level of epinephrine circulates in the blood, a switch occurs in the intracellular signaling system, from Gs protein to Gi protein signaling through the β2AR. This change in signaling is inotropic negatively, and the effect is greatest at the apical myocardium where the density of β-adrenoceptors is highest.[1)] The most widely used diagnostic criteria are the Heart Failure Association of the European Society of Cardiology diagnostic criteria for Takotsubo syndrome,[6)] which are a revision of the earlier Mayo Clinic Criteria.[7)] Recently, the International Takotsubo Diagnostic Criteria (InterTAK Diagnostic Criteria) were proposed. Cardiac biomarker levels (troponin and creatine kinase) are elevated moderately in the majority of cases, and brain natriuretic peptide levels frequently are elevated to significant levels.

In this paper, Hong et al. enrolled 27 stress cardiomyopathy and 30 anterior ST-elevation myocardial infarction (STEMI) patients and showed that troponin T (Tn-T) and creatine kinase-myocardial band (CK-MB) were significantly higher in the anterior STEMI group than in the stress cardiomyopathy group (Tn-T ng/mL: 6.8 ± 3.2 vs. 0.4 ± 0.4 ng/mL; $p < 0.001$; CK-MB: 197.6 ± 99.9 ng/mL vs. 9.7 ± 10.7 ng/mL; $p < 0.001$).[8)] It is well known that cardiac Tn-T or Tn-I, measured by conventional assays (not high sensitivity), is elevated in > 90% of patients with stress induced cardioimyopathy,[9)10)] and peak troponin levels generally are < 10 ng/mL,[10)11)] which is substantially lower than in classical ACS. CK-MB is elevated only mildly in most patients with stress cardiomyopathy. Typically, a disparity is noted between the minimal elevation in biomarkers and the extensive wall motion abnormalities during the early stages of a disease process. The levels of cardiac enzymes, as well as electrocardiography (ECG) and echocardiography findings, are insufficient for distinguishing between stress-induced

cardiomyopathy and acute coronary syndrome. The differential diagnoses of acute coronary syndrome, cardiomyopathy associated with pheochromocytoma, and acute myocarditis should be considered when treating a patient suspected of stress cardiomyopathy. Stress cardiomyopathy is an acute cardiac disorder characterized by a transient left ventricular wall motion abnormality. It is essential to distinguish stress cardiomyopathy from ACS as soon as possible because their treatments differ. Patients presenting with S-T elevation require cardiac catheterization as the first diagnostic step; in practice, this often is performed in parallel with clinical assessment for detection of elevated B-type natriuretic peptide/N-terminal pro-B-type natriuretic peptide levels, Tn-T or Tn-I, CK-MB, ECG performance, and echocardiography as initial diagnostic tests. Although stress cardiomyopathy has gained worldwide recognition, there is much to learn about the epidemiology and underlying pathophysiology of this disease. Additional randomized and controlled trials are needed to determine the most effective diagnostic and treatment methods.

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