

STRESS-INDUCED CARDIOMYOPATHY FOLLOWING COVID-19 VACCINATION

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Case vignette

Patient is a 79 year old female with a history of frequent premature ventricular beats, accelerated idioventricular rhythm, hyperlipidemia, and hypothyroidism who presented for evaluation after an episode of syncope and persistent episodes of lightheadedness. She developed severe malaise about 36 hours after receiving the 3rd dose of Pfizer COVID-19 vaccine. The following day she had a syncopal episode without prodrome while standing. She was seen by her PCP who thought it was most likely a vasovagal event caused by decreased oral intake and orthostasis. Vital signs, physical exam, ECG, routine blood work, and COVID-19 testing were unremarkable. However, the patient continued to experience episodes of lightheadedness, particularly with exertion. On one occasion while exercising she had acute dyspnea and diaphoresis.

An ECG was performed which showed new anterolateral T-wave inversions and an increased QTc interval when compared to initial ECG (Figure 1). Given her history of arrhythmia a 3-day Holter monitor was performed which showed rare premature atrial and ventricular beats, no arrhythmias were identified. A transthoracic echocardiogram was performed which showed a depressed left ventricular ejection fraction of 43% with akinesis of the apex, mid to apical septum, mid to apical anterior wall, and apical lateral wall (Figure 2). There was no significant valvulopathy.

These findings raised concern for recent myocardial infarction, as the patient had a normal exercise SPECT myocardial perfusion imaging 12 months prior. After discussion with the patient, the decision was made to proceed with coronary angiogram.

Coronary angiogram revealed normal coronary arteries. Patient subsequently underwent a cardiac MRI to evaluate the etiology of her abnormal ECG, newly reduced ejection fraction and wall motion abnormalities. Cardiac MRI was performed six weeks from her initial episode of syncope. Normal biventricular function with a left ventricular ejection fraction of 64% and mild apical hypokinesis was seen on MRI. There was no evidence of late gadolinium enhancement to suggest inflammation, infiltration, or scarring. Given patient's age, gender, imaging findings, and clinical course, a final diagnosis of stress-induced cardiomyopathy was made.

Stress-induced cardiomyopathy, also known as Takotsubo syndrome, is characterized by an acute, transient left ventricular dysfunction, normal coronary artery anatomy, regional wall motion abnormalities that extend beyond a single coronary vascular bed, and oftentimes a precipitating stressor (1,2). The stressor may be emotional or physical, but approximately 30-35% of patient present with no identifiable stressor (2). As of now there is not one specific set of diagnostic criteria to diagnose this condition.

We present the case of a healthy 79 year old female who developed stress-induced cardiomyopathy after receiving the 3rd dose of the COVID-19 vaccine. Besides the lack of an obvious stressor (which is not required for diagnosis), this patient met all the other major clinical diagnostic criteria. Although no microvascular evaluation was performed in the catheterization lab, the MRI lacked the typical findings one would expect with myocardial infarction with non-obstructive coronary arteries (MINOCA) such as late gadolinium enhancement or T2 signal hyperintensity.

Based on the timeline of patient's symptoms, the vaccine administration is the most likely culprit for a potential stressor. This is not a widely reported side effect of the COVID-19 vaccine although review of the medical literature revealed a few case reports. Rather, myocarditis and pericarditis have been more frequently observed after patients receive mRNA vaccines. This type of cardiomyopathy has also been observed in patients with active COVID-19 infection which this patient did not have.