Coronary Artery Anomaly in Takotsubo Cardiomyopathy:

Cause or Innocent Bystander?

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Coronary artery anomalies can provoke intermittent vasospasm and endothelial dysfunction, which can cause takotsubo cardiomyopathy. However, in takotsubo cardiomyopathy, apical myocardial regions are typically affected, and these do not correlate with a specific epicardial coronary distribution territory.

We report the case of a 74-year-old woman who presented with acute respiratory failure and suspected myocardial infarction. She had a left coronary artery anomaly, dominant right coronary artery supply, takotsubo cardiomyopathy, depressed left ventricular ejection fraction, and no atherosclerotic disease. In the absence of exercise ischemia, we considered the anomalous artery to be an incidental finding. After 6 weeks of medical therapy, the patient's ejection fraction was normal; one year later, she remained asymptomatic.

The anomalous left coronary artery in the presence of dominant right coronary supply did not explain the diffuse apical regional wall-motion abnormalities in our patient. To our knowledge, this is the first report of coexisting takotsubo cardiomyopathy and anomalous coronary artery in a patient presenting with acute dyspnea. ***(Tex Heart Inst J 2020;47(1):44-6)***

Coronary artery anomalies (CAAs) can provoke intermittent vasospasm and endothelial dysfunction, which can cause takotsubo cardiomyopathy (TC). However, the apical myocardial regions typically affected in TC may not correlate with specific epicardial coronary vessel distribution territories. We present the case of an elderly patient in whom we found a CAA and coexistent TC.

Key words: *Coronary vessel anomalies/complications/diagnostic imaging/physiopathology; multimodal imaging/methods; risk factors; takotsubo cardiomyopathy/physiopathology; treatment outcome; ventricular dysfunction, left*

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Case Report

In January 2018, a 74-year-old woman presented with acute respiratory failure that necessitated orotracheal intubation and mechanical ventilation. Myocardial infarction was suspected. A coronary angiogram revealed an anomalous origin of the left anterior descending coronary artery (LAD) and left circumflex coronary artery (LCx), but no coronary artery disease (Fig. 1). Echocardiograms and left ventriculograms revealed left ventricular (LV) apical ballooning, suggesting TC, and an LV ejection fraction of 0.20 (Fig. 2). Coronary computed tomographic angiograms confirmed the anomalous origin of the LAD from a dominant right coronary artery (Fig. 3A). The anomalous vessel had a deep subpulmonary, intraseptal course upon exiting the anterior wall,

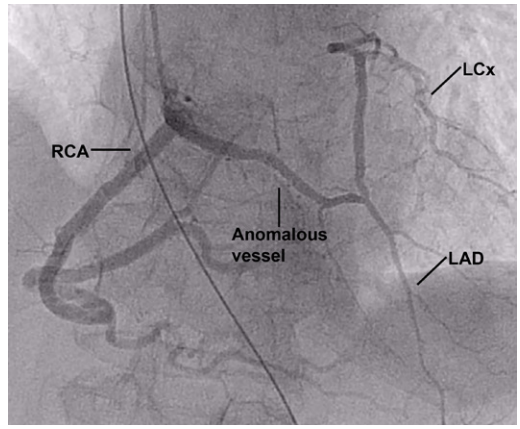

Fig. 1 *Coronary angiogram shows anomalous left anterior descending (LAD) and left circumflex (LCx) coronary arteries originating from the right coronary artery (RCA), without atherosclerotic disease.*

where it branched, giving rise 1) to the LAD and coursing down to the LV apex, and 2) to the LCx upward (Fig. 3B). A hypoplastic vessel originated from the left coronary cusp (Fig. 3C). No high-risk anatomic features of the CAA, such as a slit-like ostium, acute takeoff angle, or interarterial or intramural course, could be identified. Single-photon emission computed tomographic myocardial perfusion images (SPECT-MPI) during maximal physical exercise showed no ischemia or myocardial scarring (Fig. 4).

We concluded that the CAA was an incidental finding, and we placed the patient on medical therapy for heart failure. Six weeks later, her LV ejection fraction was normal; one year later, she remained asymptomatic.

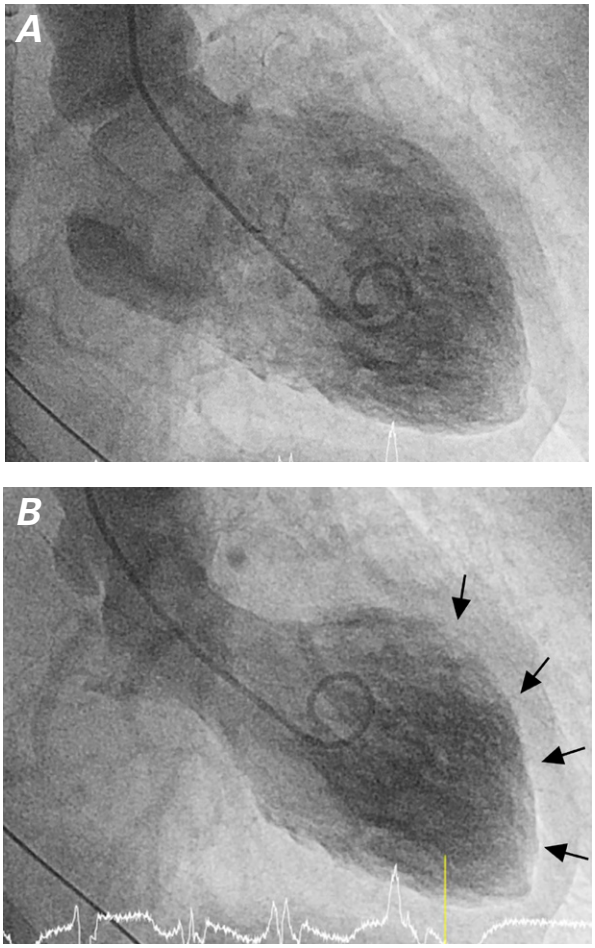

Fig. 2 *Left ventriculograms in **A**) diastole and **B**) systole show takotsubo cardiomyopathy with severely reduced left ventricular ejection fraction. During systole, akinetic apical ballooning occurred (arrows), with normal contraction in the basal segments.*

Discussion

To our knowledge, this is the first report of coexisting CAA and TC in a patient presenting with acute dyspnea.

Coronary computed tomographic angiography is ideal for identifying high-risk anatomic features of CAAs in detail, such as interarterial course (course of the anomalous vessel between the great arteries) or intramural course (within the aortic wall).[1] Ischemia can be ruled out by using SPECT-MPI with maximal physical exercise or dobutamine stress.[2] Adenosine stress is not recommended, because vasodilation does not mimic the mechanism of dynamic compression in CAAs.[3]

Intermittent vasospasm and endothelial dysfunction of coronary arteries can induce TC, so it can be hypothesized that our patient's left CAA with an intraseptal course affected her LV apical ballooning.[4] In cases of TC, vasospasm and endothelial dysfunction are best evaluated by means of an intracoronary acetylcholine challenge,[5] which our patient did not undergo. Regardless, the apical myocardial regions typically affected in TC do not correlate with a specific epicardial coronary vessel distribution territory; they are supplied by multiple left and right coronary vessels. An anomalous left coronary artery with an intraseptal course in the presence of a dominant right coronary supply does not automatically explain diffuse

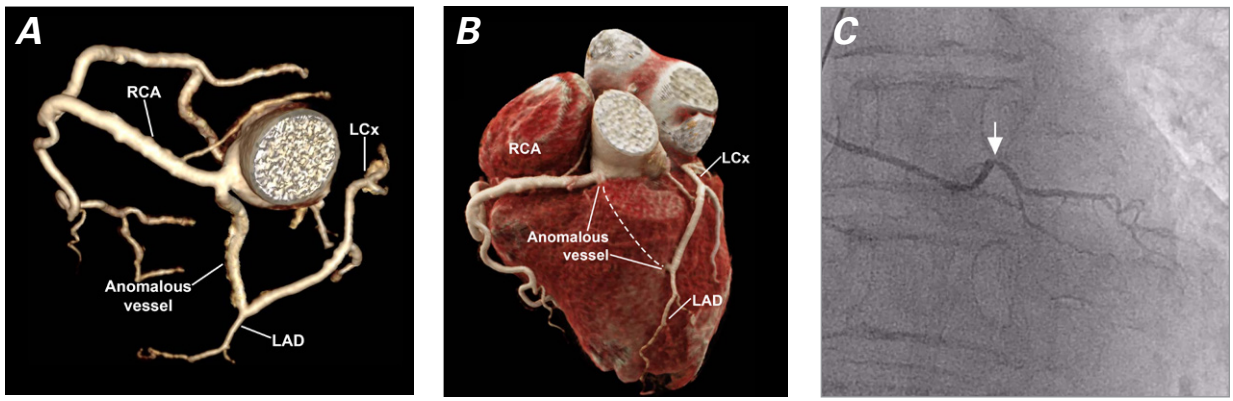

Fig. 3 *Coronary computed tomographic angiograms. Three-dimensional reconstruction images show **A**) anomalous origin of the left anterior descending coronary artery (LAD) from the right coronary artery (RCA) and **B**) the anomalous vessel's deep subpulmonary, intraseptal course. **C**) Angiogram shows a hypoplastic vessel (arrow) originating from the left coronary cusp.*

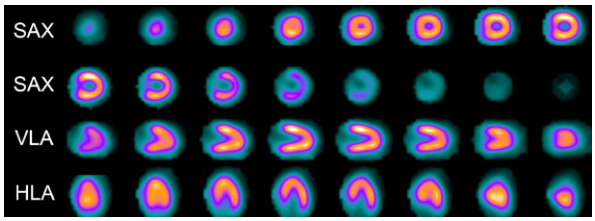

Fig. 4 *Single-photon emission computed tomographic myocardial stress perfusion images show no ischemia or myocardial scarring under exercise and rest conditions.*

HLA = horizontal long axis; SAX = short axis; VLA = vertical long axis

apical regional wall-motion abnormalities. Accordingly, we attributed our patient's acute dyspnea to the underlying TC and considered the CAA to be an incidental finding that needed no further treatment.[6]

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