Tako-Tsubo (TT) cardiomyopathy is a rare clinical entity, initially described in Japan, characterized by a transient apical dyskinesia of unknown origin.1,2 Patients present with signs and symptoms typical of myocardial infarction, but the epicardial coronary arteries are normal and left ventricular wall motion abnormalities spontaneously resolve during follow-up.1,2 Although different etiologies have been proposed to explain the pathophysiology of TT, its cause remains unknown. Here we describe optical coherence tomography findings of a patient presenting with TT.

A 63-year-old woman with a history of hypertension was admitted for prolonged chest pain. An ECG showed widespread 1- to 2-mm ST-segment elevation from V2 to V5. An echocardiogram revealed extensive anteroapical akinesia. Urgent coronary angiography using a left radial access revealed normal coronary arteries (completely smooth angiographic appearance) with normal antegrade coronary flow. Left ventricular angiography (Figure 1) disclosed a pattern characteristic of apical ballooning. The wall motion of the anterobasal and posterobasal left ventricular segments was hyperdynamic and left ventricular ejection fraction was 30%. Left ventricular end-diastolic pressure was 25 mm Hg, mitral regurgitation was absent, and no intraventricular gradient could be detected. Frequency domain optical coherence tomography (Dragon Fly, C7-XR, LightLab Imaging, St. Jude Medical, St. Louis, MO) with a nonocclusive technique was used to interrogate both the left anterior and left circumflex coronary arteries. Special care was taken to obtain an exhaustive and completely blood-free examination of the vessel wall using repeated, overlapping imaging sequences to interrogate the longest possible segment of both vessels. Three runs were obtained in the left anterior descending coronary artery (total image length, 9 cm) and 2 runs in the left circumflex coronary artery (total image length, 6 cm). Notably, the vessel walls of both arteries appeared completely normal: wall thickness ranged from 170–340 μm and had the characteristic 3-layered structure of a healthy vessel. Atherosclerotic changes (fibrotic, calcified, lipid pools) were not visualized at any segment. In addition, no images of ruptured or eroded intima were detected. Finally, no trace of residual thrombus (red or white) was disclosed at any location (Figure 2).

The patient had a peak creatine phosphokinase level of 181 IU (troponin I, 1.43 ng/mL) and did not develop Q waves. Evolving electrocardiographic changes (anterior negative T waves) progressively normalized. Six days after admission she was discharged with an echocardiogram showing a normal left ventricular function without wall motion abnormalities.

Our findings demonstrate that TT cardiomyopathy may occur in patients without any structural abnormality of the epicardial coronary vessels. To our knowledge, this is the first report assessing the value of optical coherence tomography to rule out subtle pathological changes in the coronary vessel wall in TT cardiomyopathy. The characteristic absence of angiographic coronary artery disease in this entity does not exclude the possibility of minor pathologic changes at the coronary vessel wall. Indeed, previous studies using intrava-
circular ultrasound suggested that some patients with TT have angiographically silent atherosclerosis or even ruptured plaques. However, the unique resolution (15 μm) of this technique provides reassuring—nearly histological—evidence that the TT syndrome also may occur in patients with structurally normal coronary arteries. This suggests that alternative pathophysiology should be investigated further. Additional optical coherence tomography studies in larger series of patients are required to fully elucidate the underlying pathophysiology of this elusive and challenging clinical condition.

Disclosures

None.

References


Optical Coherence Tomography Findings in Tako-Tsubo Cardiomyopathy
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