Severe Multivessel Coronary Vasospasm and Left Ventricular Ballooning Syndrome

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In their first description of Takotsubo cardiomyopathy (TC), Dote et al.1 hypothesized that the disease was caused by multivessel coronary vasospasm, because 4 of 5 patients in their report had either spontaneous or provokable coronary vasospasm on coronary angiography. However, later studies did not find supporting evidence for the pathogenic role of coronary vasospasm. Recently, the vasospasm hypothesis of TC revived. In a small case series from the United States, Angelini2 was able to provoke severe coronary vasospasm by intracoronary application of acetylcholine in 4 patients with the disease.

Case

A 60-year-old woman reported of retrosternal chest pain radiating to her left arm. Several minutes later, she had a witnessed cardiac arrest. Emergency personnel found her apneic and asystolic. She received 1 mg of adrenalin intravenously and was successfully resuscitated. She was intubated and was brought to the emergency department where she required 3 additional doses of adrenalin (200 μg each) to maintain adequate blood pressure. Her medical history was significant for chronic stable angina that had been treated with implantation of a bare-metal stent in her right coronary artery 2 years earlier. Since then, she did not have any symptoms. Her initial ECG showed diminished R waves in leads V2 and V3 and flattened T waves in several leads (Figure 1A). Her initial cardiac enzymes were within normal range. Urgent coronary angiography demonstrated diffuse and extensive stenosis of the distal right coronary artery (Figure 2A) and of the entire left coronary system (Figure 2B). Intracoronary application of nitroglycerin led to complete resolution of all stenoses (Figure 3C and 3D). Ventriculography demonstrated left ventricular apical ballooning (Figure 3A and 3B). An intra-aortic balloon pump was inserted and the patient was transferred to the intensive care unit. A second ECG was taken shortly after arrival in the intensive care unit, demonstrating ST elevation in several leads (Figure 1B). Three hours after admission, the creatine kinase level of the patient was elevated at 247 U/L. The patient died an hour later due to incessant ventricular fibrillation. There was no history of illicit drug use and the patient’s urine toxicology screen was negative for cocaine. Permission for autopsy was denied by the family.

Conclusion

This case illustrates that severe and extensive coronary vasospasm can lead to left ventricular wall-motion abnormalities resembling those in classical TC. Whether there is a true cause-effect relationship between coronary vasospasm and TC, however, remains to be seen. Besides, coronary vasospasm cannot easily explain the occurrence of midventricular and basal variants of TC. Several aspects of this case, however, merit further attention and are rather unusual in a “typical” case of TC. First, the patient’s initial ECG showed only mild abnormalities, despite angiographic evidence of severe multivessel vasospasm. This could be explained by the widespread distribution of affected myocardium, which may have produced counterbalancing electric forces. Second, the patient’s ECG in the intensive care unit showed ST elevation suggestive of inferoposterior ischemia. We assume that this was due to recurrence of severe vasospasm in the right coronary artery. Third, incessant ventricular fibrillation, although rare in TC, has been previously reported as a cause of death in the disease,3 but its cause remains obscure. Finally, the potential induction of vasospasm from epinephrine injections during cardiac arrest should also be kept in mind.

Disclosures

None.
References


Key Words: Takotsubo cardiomyopathy ■ left ventricular ballooning syndrome ■ coronary vasospasm
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