“Takotsubo” Cardiomyopathy
A Syndrome Characterized by Transient Left Ventricular Apical Ballooning That Mimics the Shape of a Bottle Used for Trapping Octopus in Japan

Key words: acute myocardial infarction, cardiomyopathy, coronary flow reserve

“Takotsubo” cardiomyopathy is a novel cardiac syndrome characterized by transient left ventricular dysfunction with chest pain, electrocardiographic changes and minimal release of myocardial enzymes mimicking acute myocardial infarction. Left ventriculograms obtained after onset show apical ballooning with basal hyperkinesia. Because this gives the left ventricle a peculiar shape (i.e., a round bottom and narrow neck) that resembles a type of bottle used in Japan for trapping octopus, Sato et al termed the syndrome “Takotsubo” cardiomyopathy (Tako: octopus, tsubo: bottle) (1); it has also been called ampulla or amphora cardiomyopathy (2, 3). Strangely enough, this disease has been reported frequently in Japan (1–21), but rarely in other countries (22, 23).

In Japan, Takotsubo cardiomyopathy is more prevalent among women than men (7 : 1), with the average age at onset being 68.6±12.2 in women and 65.9±9.1 years in men (3). The incidence is estimated at 1% to 2% of acute myocardial infarctions (3, 10), and the Angina Pectoris- Myocardial Infarction Investigators in Japan (15) reported that 38 of 88 patients (43%) in whom it was observed had prior aggravation of an underlying disorder (cerebrovascular accident, epilepsy, exacerbated bronchial asthma, acute abdomen), noncardiac surgery or other medical procedure. Notably, the onset is also frequently preceded [27% (15) to 71% (3)] by emotional stress associated, for example, with a sudden accident, death/funeral of a family member, excess exercise, quarreling, excessive alcohol consumption or great excitement, which suggests emotional or physical stress might play a key role in Takotsubo cardiomyopathy. Electrocardiograms obtained at onset show ST-segment elevation in a wide area that includes the anterior, lateral and inferior wall, with more pronounced elevation in V₄₆ than V₁₃; less frequently there is an abnormal Q wave and prolongation of QTc intervals (4), and in one study reciprocal changes were seen in 3 of 21 patients which is less frequent than anterior myocardial infarction (10). There is also slight elevation of peak myocardial creatine kinase activity (480 IU/l on average) (10).

One striking finding is that the coronary arteries are normal on angiograms during the acute phase, with 75% stenosis present in only 5 of 21 cases (10). Transient total occlusion of the large epicardial artery due to spasm at multiple sites might cause myocardial stunning and transient left ventricular dysfunction that simulates Takotsubo cardiomyopathy (1, 11, 16, 18); however, provocative testing using ergonovine does not show coronary spasm in all 20 cases in one study (10); in two studies ergonovine testing proved positive only in 21% (15) or approximately 30% (3) of cases. The marked apical ballooning, which gives the left ventricle its peculiar shape during the acute phase, is the result of akinesia in a wide area involving the anterior, lateral and inferior wall accompanied by hyperkinesia in the basal area, and may in some cases be the result of myocardial injury caused by excessive catecholamine (5, 8, 9), though catecholamine levels are not necessarily elevated during the acute phase (3, 10). Fortunately, the fatality rate from Takotsubo cardiomyopathy is less than that of acute myocardial infarction: 10 of 250 patients in one study (3) and 1 of 88 patients in another (15). The marked left ventricular dysfunction and the peculiar shape usually disappear within several weeks. Long-term prognosis is not modified by drug therapy (10), and in two studies the rate of recurrence was found to be 1 in 21 cases (10), and 2 in 88 cases (15).

Recently, other aspects of Takotsubo cardiomyopathy have been discovered that might shed additional light on its cause. Thallium-201 scintigraphy revealed a defect confined to the apical region that disappears with convalescence (6, 8). Measurements of coronary flow using a Doppler flow wire showed an impaired coronary flow reserve that persisted for a time after normalization of the left ventricular wall motion (5, 8, 9, 14). In addition, myocardial fatty acid metabolism measured using iodine-123-beta-methyl-p-iodophenyl penta-decanoic acid (¹²³I-BMIPP) was more severely impaired than myocardial perfusion (6), and a discrepancy in the sympathetic innervation between the apical and basal region was seen (12). Moreover, Suzuki et al (24), in this issue of the Journal, described a 64-year-old man who showed ST-segment elevation in the anterior leads after successful...
treatment of cardiopulmonary arrest. In that case, the left ventricle was atypical because the wall motion abnormality was confined to the anterior wall. Consequently, although all other clinical findings were consistent with Takotsubo cardiomyopathy, the left ventricular wall motion was no longer Takotsubo-like in their case.

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In summary, Takotsubo cardiomyopathy is a syndrome of unknown cause and diverse clinical features. Perhaps as additional cases from countries other than Japan are reported, a clearer, more complete understanding of this syndrome will emerge and, in turn, lead to development of an appropriate therapeutic protocol.

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References