Very Late Recurrence of Takotsubo Syndrome

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Recurrence of the Takotsubo syndrome is possible, and in the majority of cases it occurs within 4 years. The present report is focused on a case of the Takotsubo syndrome recurrence after 10 years. The patient had been admitted for the first time in 1999 with a diagnosis of "non-q wave acute anterolateral myocardial infarction." Ten years later, she was readmitted for suspected acute myocardial infarction; coronary arteries, however, were normal, and the left ventricular angiogram demonstrated apical ballooning typical for the Takotsubo syndrome. It is worth noting that the clinical presentation and the ECG were the same on both occasions; in addition, both events were triggered by an emotional stress.

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The Takotsubo syndrome is characterized by transient left ventricular dysfunction often associated with intense emotional stress. The clinical presentation includes chest pain, ST segment abnormalities, and/or T-wave inversion, and at times slight troponine and CK-MB increase.¹ The disease hallmark is akinesia and systolic dilatation of the middle-apical region of the left ventricle, usually defined as apical ballooning.² The Takotsubo syndrome has been recently classified as a form of cardiomyopathy, and in most cases involves females in post-menopause.³ Recurrence of Takotsubo is possible,⁴ and in the majority of cases occurs within 4 years^{5–8}; the present report is focused on a case of Takotsubo syndrome recurrence after 10 years.

CASE PRESENTATION

An 83-year-old female with hypertension, high cholesterol level, and the history of myocardial infarction 10 years before, experienced, following a familial quarrel, midsternal chest pain associated with palpitation and shortness of breath. The patient was admitted to the emergency room; since the ECG revealed slight ST segment elevation in leads II, III, aVF, V₂, and inverted T waves in all leads except aVR (Figure 1), she was immediately referred to the cath lab for possible percutaneous transluminal coronary angioplasty (PTCA). The coronary angiogram revealed the absence of

occlusion or stenosis of the coronary arteries, and the left ventricular angiogram demonstrated apical ballooning typical for the Takotsubo syndrome (Figure 2). During the first 24 hours an increase of cardiac markers was observed (Myoglobin 151 [normal range 0–70], Troponine I 8.53 [normal range <0.5], MB creatinase 67 [normal range 0–20]).

The echocardiogram performed immediately after cardiac catheterization showed a left ventricle of normal volume, with hypertrophy of the basal septum (13 mm); the ejection fraction was 35%, the left ventricular apex was akinetic and the basal areas of both the septum and the left ventricular free wall were hyperkinetic, resulting in apical ballooning. Systolic anterior displacement of the anterior mitral leaflet, resulting in subaortic obstruction (peak gradient 70 mm Hg), and moderate mitral regurgitation were also noted.

On day 2, ST segment elevation disappeared and the T waves became negative in I, aVL and from V₃ to V₆. The echocardiogram progressively improved, with increase of ejection fraction to 50% and disappearance of both mitral valve systolic anterior movement and related gradient. The patient was discharged on day 4.

Ten years before, the same patient had been admitted to our Department with a diagnosis of "Non-q wave acute anterolateral myocardial infarction."

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Figure 1. Admission electrocardiogram.

Figure 2. Left ventricular angiogram showing apical ballooning.

On that occasion, the patient experienced intense chest pain following her husband's death. On admission, cardiac laboratory markers were positive (troponine I = 5), and the ECG was identical to that recorded during the recent episode (Figure 3). The echocardiogram, performed by the same doctor as in the most recent admission, revealed akinesia of the midapical segments of the left ventricle. On that occasion, no coronary angiogram was performed, since a stress-echo with Diprydamole, performed 8 days after admission, yielded negative results.

DISCUSSION

Although during the first admission of the patient no direct proof was obtained that coronary arteries were angiographically normal, the absence of coronary stenosis or occlusion during the most recent

Figure 3. Electrocardiogram recorded 10 years before that shown in Figure 1, during the previous patient admission.

episode suggests that also in the previous occasion myocardial infarction did not depend on thrombosis of coronary vessels. A retrospective diagnosis of the Takotsubo syndrome, thus, can be applied even to the disease occurred 10 years before, provided that also in that situation the illness followed a very intense emotion.

The transient intraventricular gradient observed in our patient is not an expression of left ventricular hypertrophy, but is more likely a dynamic phenomenon resulting from dyskinetic apical and midventricular segments with concomitant hyperkinetic basal segments.⁹ It is relatively common that Takotsubo patients develop a transient LVOT obstruction, that has been reported in 18% of the patients in a large series. The disappearance of gradient following the acute phase of the disease reflects the functional origin of the obstruction.¹⁰

Recurrence of the Takotsubo syndrome has been described: in 3% of patients this occurs within 1 year from the 1st presentation, and in 10% of cases within 4 years; on very rare occasions, recurrence has been observed after 7 or 8 years. The present case, characterized by two episodes separated from each other by a 10 year interval, is, to the best of our knowledge, the latest Takotsubo recurrence reported up to now.

REFERENCES

1. Sharkey SW, Windenburg DC, Lesser JR, et al. Natural history and expansive clinical profile of stress (tako-tsubo) cardiomyopathy. *J Am Coll Cardiol* 2010;55:333-341.
2. Perel L, Mekori Y, Mor A. The broken heart syndrome. *Harefuah* 2009;148:392-394.
3. Hedberg P, Magounakis T, Dubaniewicz W, et al. "Broken heart" or Takotsubo cardiomyopathy mostly in postmenopausal women. Stress-induced condition resembling acute myocardial infarction. *Lakartidningen* 2007;104:3277-3282.

4. Theibich A, Klarlund K, Taskiran M. Recurrent case of Takotsubo syndrome. *Ugeskr Laeger* 2009;171:2716–2717.
5. Gogas BD, Antoniadis AG, Zacharoulis AA, et al. Recurrent apical ballooning syndrome "The masquerading acute cardiac syndrome." *Int J Cardiol* 2011;150:e17–e19.
6. Maroules CD, Linz NA, Boswell GE. Recurrent Takotsubo cardiomyopathy. *J Cardiovasc Comput Tomogr* 2009;3:187–189.
7. Koeth O, Mark B, Zahn R, et al. Midventricular form of takotsubo cardiomyopathy as a recurrence 1 year after typical apical ballooning: A case report. *Cases J* 2008 Nov;1:331.
8. Kleinfeldt T, Severin R, Lischke S, et al. Recurrent left ventricular apical ballooning induced by recurrent stress. *Int J Cardiol* 2009;134:e47–e48.
9. Merli E, Sutcliffe S, Gori M, Sutherland GR. Tako-tsubo cardiomyopathy: New insights into the possible role of underlying pathophysiology. *Eur J Echocardiogr* 2006;7:53–61.
10. Bybee KA, Kara T, Prasad A, et al. Systematic review: Transient left ventricular apical ballooning: A syndrome that mimics ST-segment elevation myocardial infarction. *Ann Intern Med* 2004;141:858–865.