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Type transition and mitral regurgitation of mid-ventricular Takotsubo in a single course

Short title: Type transition and mitral regurgitation of TTS

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Takotsubo syndrome (TTS) is a clinical syndrome characterized by acute reversible dysfunction of left ventricle (LV) usually without significant obstructive coronary artery disease. TTS was classified into apical, mid, basal ventricular and focal type based on the location of myocardial akinesis [1]. Different types may occur in recurrent patients at each event but rare in a single course [2]. Mitral regurgitation (MR) was common in apical type due to systolic anterior motion of mitral leaflet which always accompanied by left ventricular outflow tract obstruction (LVOTO) [3], but rare in midventricular type. We report a case of midventricular TTS with MR independent of LVOTO and transition to apical type in a single course.

A 54-year-old lady presented to our emergency department because of acute chest pain and shortness of breath for 6 hours immediately after drinking boiled root soup. Her past medical history included nasopharyngeal carcinoma, receiving chemotherapy for 3 years (5-FU, nedaplatin and gemcitabine) and adjuvant radiotherapy, and chronic gastritis for 15 years. Emergency electrocardiogram (ECG) on admission confirmed sinus rhythm with limb lead low voltage and T-wave inversion in V3–4 and aVL. Blood testing showed increased troponin-I, N-terminal pro–b-type natriuretic peptide and creatine kinase isoenzyme of 0.82 μg/l, 1940 pg/ml, and 40.00 U/l. Subsequent emergency coronary angiography (CAG) at 03:52 am day 1 showed no evidence of atherosclerosis and obstructive lesions in coronary arteries (Supplementary material, *Figure S1, S2*). Her chest pain persisted during CAG but without coronary spasm, and provocative test with acetylcholine was not performed. Left ventriculography demonstrated middle akinesia and basal, apical hypokinetic of left ventricle (LV), which arouse the possible diagnosis of midventricular TTS, and unexpectedly revealed moderate-severe MR meanwhile which was rare in midventricular TTS (Figure 1A, B, Supplementary material, *Video S1*). Echocardiography at 10:30 day 1 further demonstrated midventricular wall motion abnormality with left ventricular ejection fraction (LVEF) of 41%, severe MR without dynamic LVOTO, and normal right ventricular wall motion. The

patient received proton pump inhibitors, spasmolysis, diuretic and nitroglycerin treatment. Her symptoms quickly relieved at 17:30 pm day 1. A bedside transthoracic echocardiography at 07:51 am day 3 showed significant improvement of mid ventricular akinesia with a LVEF of 58% and reduction of the MR grade from severe to mild (Figure 1C, D, Supplementary material, *Video S2*). Then she gradually improved. Cardiac magnetic resonance (CMR) was performed at 18:40 pm day 6, showed well mid left ventricular wall motion, but surprisingly found hypokinesia of apical ventricular wall, which prompt type transition from midventricular type to apical type (Figure 1E, F, Supplementary material, *Video S3*), LVEF was 59% and right ventricular wall motion was normal. Extensive myocardial edema including papillary muscle was found meanwhile (Supplementary material, *Figure S4*) (Myocardial edema was evaluated on T2-turbo inversion recovery magnitude sequence as follows: myocardial edema ratio (ER) was defined as the ratio between myocardial signal intensity (SI) to skeletal muscle SI, and an ER ≥1.9 represented edema [4])and without late gadolinium enhancement (LGE) (Supplementary material, *Figure S5*).These dynamic multimodal imaging excluded obstructive or non-obstructive coronary arteries, strongly supported the diagnosis of TTS [5]. Unfortunately, the patient was lost to follow-up.

The dramatic improvement of symptoms of TTS was considered due to the complete recovery of myocardial akinesia. In the current case, mid LV akinesis disappeared and even hyperkinesis, surprisingly, a new apical LV hypokinesia replaced apical hyperkinesis during convalescence without reappear but continued improvement of symptom, which suggested this is a continuum of a single process, it may provide an insight into the pathogenesis of takotsubo cardiomyopathy.

In the current case, severe MR was found in midventricular TTS which rapidly disappeared with the improvement of heart function, and was not recurrent in the following apical TTS. This suggested MR could be a complication of midventricular TTS. Considering the rapidly disappeared, tethering and systolic anterior motion of the mitral valve leaflet caused by asynergic movement of mid and apical ventricular wall may contribute more to MR than papillary muscle edema.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/kardiologia_polska

Article information

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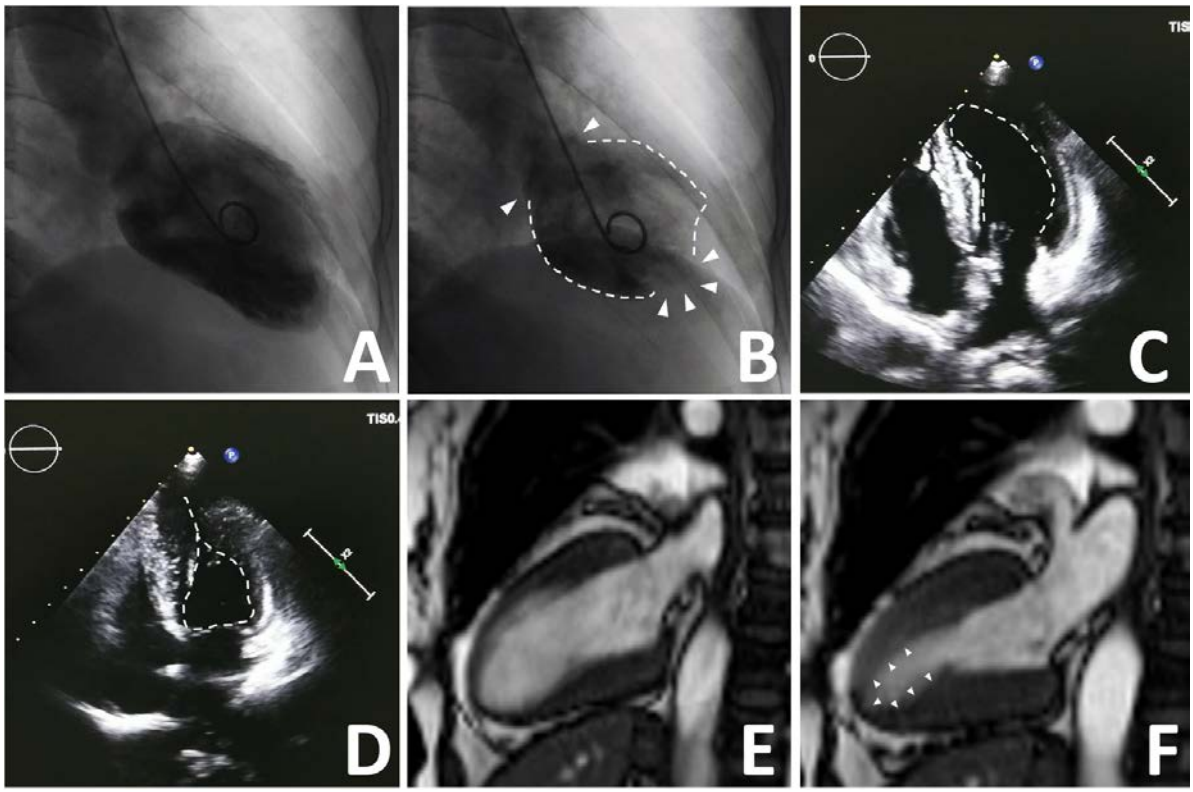

Figure 1. Left ventricular (LV) angiogram of end-diastolic phase (ED) (**A**) and end-systolic phase (ES) (**B**). Mid LV akinesia (white dotted line) and basal, apical LV hypokinetic was seen (white line and triangles). A bedside transthoracic echocardiography of ED (**C**) and ES (**D**) showed normal motion of LV wall (endocardium was outlined by white dotted line). CMR cine sequence of ED (**E**) and ES (**F**) showed hypokinesia of apical LV (F, white triangles)