Thrombo-Embolic Renal Infarction in a Case of Mid-Ventricular Takotsubo Syndrome

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Abstract

Thrombo-embolism is one of the serious complications of takotsubo syndrome (TS). It typically occurs in the classical mid-apical left ventricular ballooning form of TS. This complication has not been reported in cases of left mid-ventricular ballooning type of TS. We describe a 67-year-old woman who presented with 2-3 days of increasing signs and symptoms of heart failure. Echocardiography showed marked hypokinesia/akinesia in the mid-anterior, mid-anterolateral, and mid-inferior wall of the left ventricle and mild hypokinesia in the apical region. There was also hypokinesia of the mid and apical parts of the right ventricle. One day after admission, she developed acute left-sided renal infarction. Left ventriculography and coronary angiography 3 days after admission showed typical left mid-ventricular ballooning with no identifiable coronary lesion. Follow-up echocardiography showed complete resolution of left and right ventricular dysfunction. Takotsubo syndrome with right ventricular involvement complicated with heart failure and left renal embolic infarction was diagnosed. The mechanism of left renal embolic infarction is discussed.

Key words: takotsubo, thrombo-embolism, renal infarction, neurogenic stunned myocardium, Broken heart


Introduction

Takotsubo syndrome (TS) is an increasingly recognized acute cardiac disease with a special predilection for postmenopausal women (1-3). It has a clinical presentation that mimics acute coronary syndrome. TS is characterized by a striking circular left ventricular wall motion abnormality resulting in a conspicuous ballooning of the left ventricle with complete resolution within days to weeks. During the acute stage, the disease might be associated with serious and life threatening complications (2, 4). Herein, we describe a patient with angiographically typical mid-ventricular TS with right ventricular involvement who presented with heart failure complicated with a thromboembolic left renal infarction.

Case Report

A 67-year-old woman presented with 2-3 days of increasing dyspnea and orthopnea. She had history of excessive fatigue during the previous year. One month prior to her presentation, treatment with antihypertensive and warfarin was initiated for hypertension and atrial fibrillation. Clinical examination showed signs of heart failure and hypertension (blood pressure 180/120 mmHg). She had regular rhythm and cardiac auscultation was unremarkable. Treatment with diuretics, valsartan and metoprolol was initiated with good effects. The ECG at admission showed sinus rhythm and mild ST-depression in leads 1, V5 and V6. On the following day, the ECG revealed atrial fibrillation and inversion of T-wave in leads V2-V4 with prolongation of the corrected QT time (512 msec). Echocardiography one day after presentation showed marked hypokinesia/akinesia over the mid-anterior, mid-anterolateral, and mid-inferior parts of the left ventricle. There was mild hypokinesia in the apical region and good contractions over the basal regions resulting in an ejection fraction of 35% (Fig. 1A and B). There was also hypokinesia over the mid-apical region of the right ventricle. Contrast echocardiography showed no evidence of left ventricular thrombus (Fig. 1C and D). Myocardial infarction biomarker was slightly elevated with the highest troponin T level of 0.06 µg/L. Pro-BNP was markedly elevated (9,820...
Acute coronary syndrome was considered and treatment with acetylsalicylic acid, clopidogrel, and low molecular weight heparin were initiated. One day after admission, the patient developed severe abdominal pain radiating to the back. She had tenderness over the left renal region. Because of abdominal pain and mildly increased plasma fibrin D-Dimer, computed tomography of the thorax and abdomen was done. This showed signs of pulmonary congestion, pleural effusion 2 cm on the right side and 0.5 cm on the left side, and a wedge-shaped hypodensity in the left kidney consistent with infarction (Fig. 2A and B). There were no signs of pulmonary embolism, aortic or renal artery dissection, or any tumour in the thoracic and abdominal organs. The renal function tests were and continued to be normal. Coronary angiography 3 days after presentation showed only mild atheromatous changes and no signs of identifiable acute coronary lesion (Fig. 3A and B). Left ventriculography showed hypokinesia in the middle part of the left ventricle and hyperkinesia in the apical and basal parts of the left ventricle resulting in a noticeable mid-ventricular ballooning (Fig. 3C and D). There were no signs of thrombosis in the left ventricle. Takotsubo syndrome as a possible diagnosis was considered. Acetylsalicylic acid, clopidogrel and low molecular weight heparin were discontinued and treatment with warfarin, valsartan, metoprolol and felodipine were continued. The ECG before discharge showed continued atrial fibrillation and near normalization of T-wave in-
versions and corrected QT time (420 msec). Echocardiography 6 days after admission showed marked improvement in the left and right ventricular functions. The patient improved clinically and could be discharged 8 days after admission. Follow-up echocardiography two months after discharge showed complete recovery of both left and right ventricular function (Fig. 1E and F).

Discussion

Takotsubo syndrome, also known as neurogenic stunned myocardium, is an increasingly recognized cardiac disease entity affecting predominantly elderly postmenopausal women (1-3). The disease is characterized by a reversible regional usually left ventricular wall motion abnormality with a peculiar circumferential pattern, which is incongruent to the coronary artery supply region. Coronary angiography reveals no identifiable coronary artery culprit lesion to explain the observed regional ventricular wall motion abnormality. The condition may occur in the setting of severe emotional stress, often after the sudden death of a loved one—hence the alternative name “broken heart syndrome”. A myriad of physical stressors, ranging from most severe diseases as brain death, subarachnoid hemorrhage to the most physiological processes as physical exercise or sexual intercourse, may trigger the syndrome (1, 2).

The left ventricular wall motion abnormality may be localized to the mid-apical, apical, mid-ventricular or basal region. Focal and Global left ventricular involvement has also been reported (2, 3, 5). The right ventricle might commonly be involved in patients with TS. The right ventricular involvement seems to be associated with a more severe impairment in left ventricular function (6, 7). It may be suspected by the presence of pleural effusion. The present patient had right ventricular involvement and presented with severe heart failure. Interestingly, she had bilateral pleural effusion and the condition was complicated with a thromboembolic left renal infarction.

Thromboembolic complication in TS is usually due to left ventricular intracavitary thrombus formation and typically occurs in the classical mid-apical left ventricular ballooning type. De Gregorio et al reported development of intracavitary thrombus in 2.5% of patients with TS; embolic complications, including stroke, renal infarction and popliteal thrombosis occurred in 0.8% (4). A case of embolization to the right popliteal and anterior tibial arteries, which resulted in amputation of the right lower extremity, has been reported (8). Sharkey et al (2) reported on the clinical profile and outcome in 136 patients with TS. They identified intraventricular apical thrombi predominantly by cardiac mag-
In conclusion we have described a case of angiographically typical left mid-ventricular TS with right ventricular involvement who presented with heart failure without a preceding emotional or physical stressor. One day after presentation, the patient had an embolic left renal infarction.

The authors state that they have no Conflict of Interest (COI).

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Written informed consent was obtained from the patient for publication of this case report. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

References