

Multiple Foci of Infarction Secondary to Giant Left Ventricular Thrombus in a Patient with Takotsubo Cardiomyopathy

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To the Editor,

We have greatly enjoyed reading the recent article by Al-Farsi et al.¹ entitled "Hemorrhagic Cardioembolic Stroke Secondary to a Left Ventricular Thrombus: a Therapeutic Dilemma". In that interesting case, the authors highlighted the challenging management of a patient with a left ventricular thrombus causing an embolic stroke with hemorrhagic transformation and the timing of anticoagulation. However, the authors did not emphasize the cause of LV thrombus. We are reporting a similar case report of a giant left ventricular thrombus and multifocal thromboembolism in a patient with Takotsubo cardiomyopathy (TCM).

A 52-year-old hypertensive woman was admitted to the emergency department for retrosternal chest pain of 3 days' duration since the death of her mother. Admission ECG revealed ST-segment elevation in leads V1-V4. There were significant changes in serum markers suggestive of cardiac damage. Myocardial enzyme assay showed elevated levels of cardiac troponin I (13.82 ng/mL) and creatine kinase-MB (22.4 ng/mL). A bedside echocardiography revealed akinesis of the left ventricular (LV) midsegments, with apical involvement. Furthermore, a giant protruding mural thrombus (37 × 21 mm) was observed at the apex of the LV. Although the regional wall-motion abnormalities extend beyond an epicardial vascular distribution, coronary angiography revealed no angiographic evidence of any identifiable culprit lesion in the epicardial coronary arteries. Other potential causes of myocardial damage such as myocarditis were not present in the differential diagnosis. Anticoagulant therapy consisting of unfractionated heparin and warfarin with close monitoring of aPTT level was started immediately. Unfortunately, cognitive impairment of the patient was observed and control echocardiography showed partial resolution of the giant thrombus with unchanged LV dysfunction. In addition, diffusion magnetic resonance imaging (MRI) of the brain and computed tomography (CT) scan of the whole body revealed multifocal microinfarction foci in the brain, spleen and kidneys. LV apical thrombus disappeared and the regional wall-motion abnormality improved after anticoagulant therapy. Additionally, the patient's cognitive function recovered without any neurologic sequelae, and there was no evidence of renal and/or liver failure at follow-up. The patient was discharged with oral anticoagulant therapy. She was in good condition at 3-month follow-up.

In our patient, TCM was diagnosed since new ECG abnormalities (ST-segment elevation) and modest elevation in serum markers suggestive of cardiac damage in accordance with transient akinesis of LV mid segments with apical involvement in the absence of obstructive coronary disease or angiographic

evidence of acute plaque rupture were present after exposure to an emotional stressor.

Transient hypokinesis, dyskinesis, or akinesis of the left ventricular midsegments, with or without apical involvement; regional wall-motion abnormalities extend beyond a single epicardial vascular distribution; and a stressful trigger are often, but not always, present.

TCM is often preceded by exposure to emotional or physical stressors and predominantly affects postmenopausal women, in the absence of significant coronary artery disease.² Despite the generally good prognosis of TCM, complications such as thrombus formation and thromboembolism can be seen. Only a few cases of TCM complicated by an LV thrombus have been reported. De Gregorio et al.³ reported that 2.5% of patients with TCM had intracavitary thrombus formation and embolic complications including stroke, renal infarction and popliteal thrombosis, which occurred in 0.8% of those patients. Furthermore, rapid formation of LV apical giant thrombus is an unusual finding for TCM. Therefore, the use of anticoagulation therapy should be considered in the management of TCM due to the possibility of rapid thrombus formation in the apical ballooning segment of LV and the risk for thromboembolism.

Anticoagulant therapy has played an important role in the treatment of LV thrombus. Furthermore, it is also important to determine why the thrombus formed in LV. We strongly believe that clinicians should consider TCM as a possible cause of LV thrombus when assessing the causes of cardiac thromboembolization, particularly in the presence of a giant protruding thrombus associated with high embolization potential.

Sincerely,

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