Abstract: P552

**Takotsubo cardiomyopathy and stroke**

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**Citation:**
A 70-year-old woman without personal or family history of interest, was admitted in the emergency department for intense dyspnea. At admission was hemodynamically stable and tachypneic with baseline oxygen saturation of 87%. Physical examination revealed jugular venous distention and crackles. Elevation of D-dimer and markers of myocardial necrosis were highlighted. The CT angiography of the pulmonary arteries ruled out pulmonary thromboembolism. Subsequently, a transthoracic echocardiogram was performed showing a non-dilated left ventricle with moderately reduced systolic function at the expense of akinesia of the apex and apical segments, with marked hypercontractility of the basal segments suggestive of Takotsubo cardiomyopathy (TCM).

The patient was admitted to acute cardiac care unit and coronary angiography showed coronary arteries without angiographically significant lesions, supporting the diagnosis. Due to extensive anterior akinesia, anticoagulation was initiated with low molecular weight heparin at full dose according to local protocol. On the second day of admission, the patient suddenly presented global aphasia, hemianopsia and mild claudication of the right upper extremity. Stroke code was activated and urgent CT angiography of cerebral arteries was performed, showing complete occlusion of the left middle cerebral artery. In the presence of ischemic stroke with major contraindications for fibrinolysis, our reference hospital was contacted to assess thrombectomy. In telemetry, sinus rhythm was observed with no atrial fibrillation or other arrhythmias. Mechanical thrombectomy was performed. During admission clinical improvement was observed, despite persistence of mixed aphasia, right supranuclear facial paresis and hypoesthesia. The control cranial CT scan showed no signs of hemorrhagic transformation, so treatment with low-molecular-weight heparin was restarted until the subsequent onset of acenocoumarol. At hospital discharge a transthoracic echocardiogram was performed showing normalization of the segmental contractility alterations with normal left ventricular systolic function.

Stroke related with TCM are associated with major neurological deficits. The frequency of cerebral infarction due to TCM is about 9.5% and it is similar or higher than the frequency of stroke after atrial fibrillation (4–9%) or myocardial infarction (4.6%).

About 5% of patients with TCM are found to have left ventricular thrombus. In the treatment of the TCM the use of anticoagulants may be considered at least until systolic function is recovered and especially in patients with loss of motion of the left ventricle apex to reduce the risk of thromboembolism.

In this case, despite the anticoagulant treatment, the patient presented a stroke without intraventricular thrombus. Maybe, other prothrombotic mechanisms are involved in the embolic events in the TCM or low-molecular-weight heparin was insufficient to prevent them.