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Case report

Takotsubu syndrome in a patient with acute ischemic stroke: Case report

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1. Introduction

Taktsubo cardiomyopathy (TC) also known as left ventricular (LV) apical ballooning syndrome commonly affects post-menopausal women who experience severe psychological or emotional stress. It has also been reported following cesarean delivery [1,2]. ReverseTC is a variant of classical one, presents with normal contractility of the apical myocardium with impaired contractility of the basal and midventricular segments [3]. Both types are characterized by transient left ventricular dysfunction, reflected as regional wall motion anomalies typically extending beyond a single coronary artery distribution. Clinically mimics myocardial infarction, chest pain, dyspnea, ST-T changes in electrocardiogram, minor elevation of cardiac enzymes in spite of almost normal angiographic coronary arteries [4,5].

2. Case report

A 45-year-old lady, gravida 4, para 3, having delivered a normal baby a day before, presented to the ER in a state of unconsciousness with a GCS of 6/15. She was known diabetic and hypertensive on regular treatment. Her previous deliveries were uneventful. She was delivered under spinal anesthesia uneventfully. Six hours later she complained of mild chest pain and shortness of breath, which gradually improved over next 12 h. She started mobilization then she suddenly fall unconscious. In the ER she was intubated, in view of low GCS and elective ventilation was initiated. On admission: BP 85/90 mmHg, T.36 °C, RR 24/min, SpO2 88%. ABG showed respiratory acidosis. On auscultation pulmonary rales on bilateral lung fields. Blood chemistry, Troponin T. 0.364 ng/ml, CK 580 U/l, RBS was 29 mmol/l, creatinine 161, no ketones in the urine. ECG showed inferior wall myocardial infarction (Fig. 1). Chest X ray pulmonary edema. 2D thoracic Echo. showed LV dilation with regional wall motion anomalies; severe akinesia involving the mid and basal segments of all the walls with relative sparing of the apex. EF was 18%. Severe tricuspid regurgitation with estimated pulmonary artery systolic pressure of 40 mmHg. Collagen disease possibility, and even myocarditis were in mind and investigations for these issues were collected which proved later on to be negative, but the results were shown after the patient death. Dobutamine and Noradrenaline started, which stabilized blood pressure initially, ABG also improved. Heparin and frusemide were also started. A CT brain was performed which showed diffuse white matter hypo densities bilaterally in the vertebral artery territory. 48 h later, multiple episodes of bradycardia and desaturation. Repeat Echo. Showed further worsening of LV function with EF 10% (Fig. 2). ECG done at this time showed complete resolution of ST changes with Q waves in inferior leads (Fig. 3). Coronary angiogram done with Right anterior oblique view cranial (RAO), and left anterior oblique view cranial (LAO) showed left ventricular dilatation with normal all coronaries (Fig. 4). Act brain was done which shows diffuse hypodenisities bilateral in the vertebral artery territory (Fig. 5). Ballooning of the midventricular segment was also noted. In view of low BP, Adrenaline was added, and intra-aortic balloon pump was initiated. In spite of cardiac enzymes showing a decreasing trend and improved ventilatory parameters, but clinically deteriorated and on 4th day her pupils become dilated and fixed with features of hypoxic brain injury and she died on 5th day post admission .ECMO was an option but, due to technical and administrative issues, and also availability, and lastly, the considered bad prognosis it was not put really.

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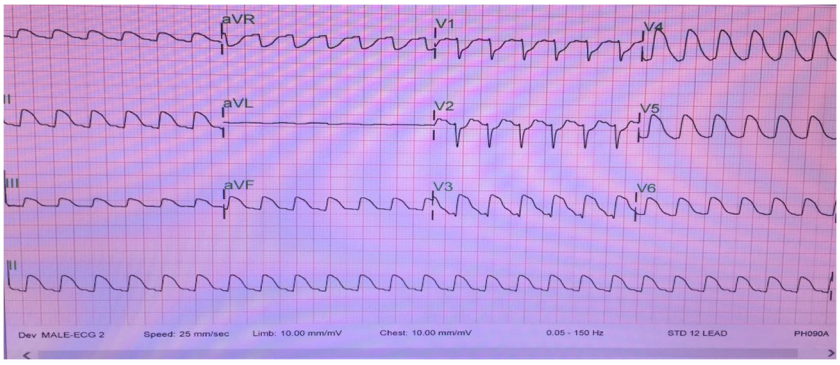

Fig. 1. This is 12 lead ECG showing diffuse st segment elevation in inferolateral leads.

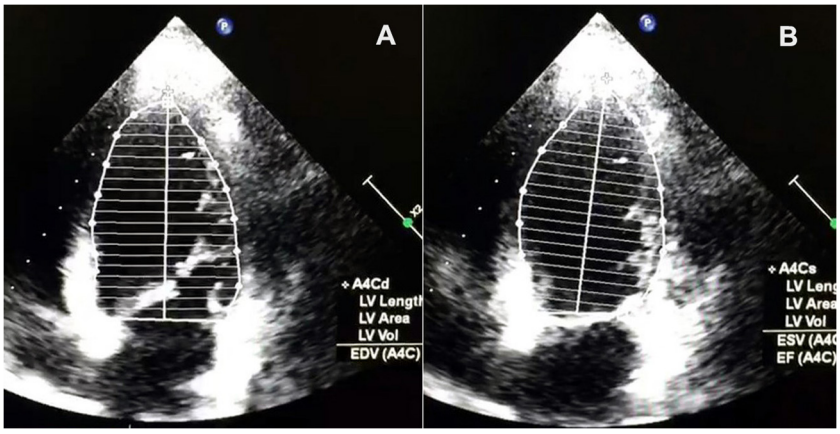

Fig. 2. Transthoracic echocardiography apical 4 chamber view with measuring the left ventricular dimensions and using Simpson's method for determining ejection fraction which showed low ejection fraction.

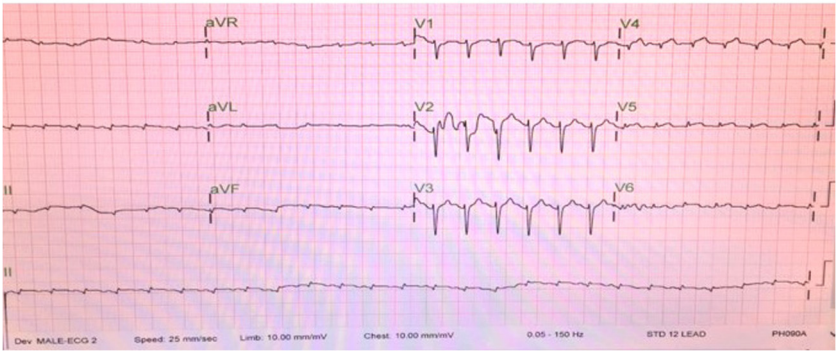

Fig. 3. This ECG shows resolved st changes in all the leads mentioned in Fig. 1.

3. Discussion

The two most common causes of heart failure following delivery are dilated cardiomyopathy and TC. Emotional or psychological stress leading to catecholamine surge which traditionally considered in the pathogenesis of the cardiomyopathy [6]. TC is most prevalent among post-menopausal women indicating low estrogen level might play a crucial role [7–9]. TC has also been described as a complication of acute ischemic stroke in elderly women [10]. In this particular patient postpartum reverse TC occurred simultaneously with acute ischemic stroke. Whether TC followed postpartum stress or it occurred subsequent to acute ischemic stroke remained non-conclusive. However, considering immediate post-

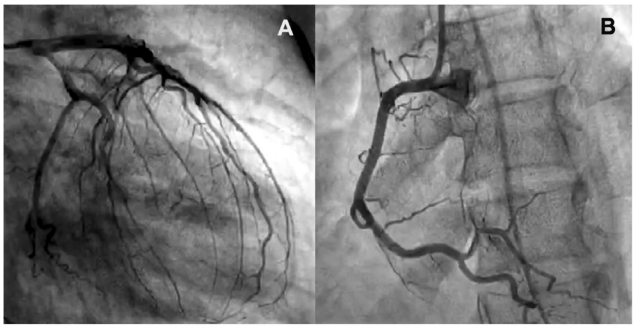

Fig. 4. This figure shows right anterior oblique view cranial (RAO), and left anterior oblique view (LAO) cranial which shows dilated left ventricle with normal coronaries.

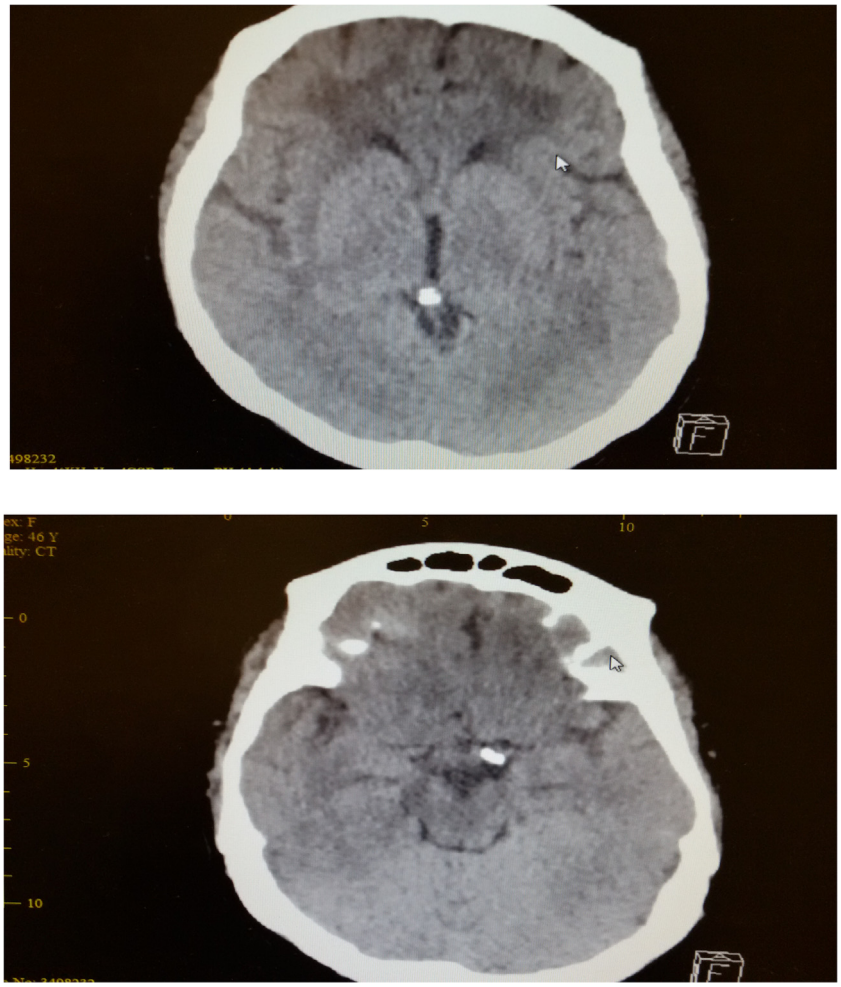

Fig. 5. This is computed tomography scan picture for the patient after admission which showed multiple bilateral hypodensities in the distribution territories of both vertebral arteries with no apparent gross brain oedema or hemorrhage.

partum status, the former possibility is more likely and ischemic stroke could have happened secondary to multiple preexisting comorbid conditions. In such a clinical background emboli could be one of the probable culprit causing stroke.

Peripartum cardiomyopathy would be the first differential diagnosis in absence of stroke. It is characterized by the development of heart failure in the last month of pregnancy or within the first 5 post-partum months, in the absence of known etiology for the cardiac failure and demonstrable heart disease prior to the last month of pregnancy [11]. In this patient reverseTC was diagnosed in view of typical echocardiographic findings and because of the association of stroke. Reverse TC is characterized by normal contractility of apical myocardium and impaired contractility of the basal parts [12]. TC is a well-known complications following sub arachnoid hemorrhage (SAH), massive catecholamine release caused by hemorrhage may trigger cardiomyopathy in such scenario [13,14]. One case series has described occurrence of TC up to 1.2% of patients with acute ischemic stroke, mostly in elderly women, and the infarcts were mostly in insular cortex, although the infarcts in vertebra-basilar territory was also described [10]. Possibly a common mechanism exists in SAH and acute ischemic stroke leading to TC. The other pathophysiological mechanisms could be microvascular dysfunctions [15]. Ischemia in insular cortex strongly associated with TC in acute ischemic stroke, as insular cortex plays a major role in autonomic control of cardiac activity. Medulla is also known to be a center of autonomic modulation of cardiovascular activity, and therefore extensive brainstem ischemia may induce autonomic disturbances and might lead to TC (or reverse TC) [10]. Compared with classical TC reverse TC presents at a younger age, and often they have an emotional or physical stress trigger [3]. Patients with reverse TC have been reported to have significantly higher level of cardiac markers (CK M or B) and Troponin I, than patients with apical cardiomyopathy [16]. The irreversible course of this patient could be because of the additive effects of dual sympathetic stress (acute ischemic stroke and post-partum state) on myocardium, complicated by pre-existing comorbid conditions.

Conflict of interest

The Authors declare that there is no conflict of interest.

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