Section: Cardiology

Case Report

CASE OF STRESS CARDIOMYOPATHY

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ABSTRACT

Stress cardiomyopathy, also known as Takotsubo cardiomyopathy, is a transient form of acute heart failure characterized by reversible left ventricular systolic dysfunction in the absence of obstructive coronary artery disease. It commonly mimics acute coronary syndrome with electrocardiographic changes and elevated cardiac biomarkers. We report the case of a 45-year-old female who presented with sudden onset slurring of speech and giddiness lasting for one hour, suggestive of a transient neurological event. Initially, the patient was hemodynamically stable and had no history of chest pain, addiction, or comorbid illness. During evaluation, she suddenly developed acute breathlessness with hypoxia and bilateral crepitations. Electrocardiography demonstrated T-wave inversions in leads I, II, aVL, and V4–V6. Cardiac biomarkers including high-sensitivity troponin-I and NT-proBNP were markedly elevated. Echocardiography revealed severe left ventricular systolic dysfunction with akinetic apical and mid segments and preserved basal contraction, suggestive of Takotsubo cardiomyopathy. Coronary angiography later showed normal coronary arteries, confirming the diagnosis. Neuroimaging demonstrated a lacunar non-hemorrhagic infarct in the right parietal lobe. The patient was managed conservatively with oxygen support, CPAP ventilation, intravenous diuretics, antiplatelet agents, and statins. Serial echocardiography showed gradual improvement in left ventricular function with normalization of ejection fraction from 29% to 55% within two weeks. This case highlights the association between acute neurological stress and Takotsubo cardiomyopathy. Early recognition is essential because the condition is reversible with prompt supportive therapy. Clinicians should maintain a high index of suspicion in patients presenting with acute neurological events followed by sudden cardiac dysfunction in the absence of coronary artery disease.

Keywords: Stress Cardiomyopathy, Cardiology.

INTRODUCTION

Stress cardiomyopathy, also referred to as Takotsubo cardiomyopathy or broken heart syndrome, is an acute reversible cardiac syndrome characterized by transient left ventricular systolic dysfunction that mimics acute coronary syndrome without evidence of obstructive coronary artery disease. The syndrome was first described in Japan in 1990 and derives its name from the Japanese “Takotsubo,” an octopus trapping pot resembling the ballooned appearance of the left ventricle during systole. The disorder predominantly affects postmenopausal women and is frequently precipitated by intense emotional or physical stress. The exact pathophysiology remains incompletely understood; however, catecholamine-mediated myocardial stunning, coronary microvascular dysfunction, and sympathetic overactivity are considered central mechanisms in disease development. Elevated circulating catecholamines during stressful events can produce direct myocardial toxicity, coronary vasospasm, and transient ventricular dysfunction.[1]

Clinically, Takotsubo cardiomyopathy presents similarly to acute myocardial infarction with chest pain, dyspnea, electrocardiographic abnormalities,

and elevated cardiac biomarkers. Electrocardiographic findings commonly include ST-segment elevation, T-wave inversion, or QT interval prolongation. Cardiac biomarkers such as troponin and brain natriuretic peptide are usually elevated but disproportionately lower than expected compared to the degree of ventricular dysfunction. Echocardiography typically demonstrates characteristic regional wall motion abnormalities extending beyond a single coronary artery territory, most commonly apical ballooning with basal hyperkinesis. Coronary angiography usually reveals normal or non-obstructive coronary arteries, which helps differentiate Takotsubo cardiomyopathy from acute coronary syndrome.[2]

Neurological disorders are increasingly recognized as important triggers of stress cardiomyopathy. Acute ischemic stroke, intracranial hemorrhage, seizures, and transient ischemic attacks can provoke exaggerated sympathetic discharge leading to myocardial dysfunction. Neuro-cardiac interactions involving the insular cortex and autonomic nervous system may contribute significantly to disease manifestation. Patients with neurological triggers often present atypically without chest pain and may rapidly develop pulmonary edema or cardiogenic shock. Therefore, early diagnosis is challenging but crucial for appropriate management.[3]

Although Takotsubo cardiomyopathy is usually reversible, complications such as acute heart failure, arrhythmias, thromboembolism, and cardiogenic shock may occur during the acute phase. Echocardiographic recovery generally occurs within days to weeks with supportive treatment. Management primarily involves stabilization, treatment of heart failure, and monitoring for complications. Prognosis is favorable in most patients; however, recurrence and mortality can occur, especially in secondary Takotsubo cardiomyopathy associated with severe neurological disease.[4]

We present a rare case of stress cardiomyopathy triggered by an acute lacunar infarct in a middle-aged woman who initially presented with transient neurological symptoms and subsequently developed acute pulmonary edema with severe left ventricular systolic dysfunction. The case emphasizes the importance of considering Takotsubo cardiomyopathy in patients with acute neurological insults and sudden unexplained cardiac deterioration.[3]

CASE REPORT

A 45-year-old female presented to the emergency department with complaints of sudden onset slurring of speech and giddiness since early morning. The episode lasted for approximately one hour and recovered spontaneously. There was no history of chest pain, palpitations, syncope, fever, or heart failure symptoms at the time of admission. The patient had no history of hypertension, diabetes mellitus, smoking, alcohol intake, or other addictions. On initial examination, she was afebrile with a pulse rate of 58/minute, blood pressure of 106/70 mmHg, respiratory rate of 16/minute, and oxygen saturation within normal limits. Systemic examination was unremarkable.

While being shifted for computed tomography of the brain, the patient suddenly developed acute onset breathlessness at rest. Examination revealed tachycardia with pulse rate of 116/minute, respiratory rate of 36/minute, and oxygen saturation of 86% on room air, which improved to 95% with high-flow oxygen support. Bilateral basal crepitations were heard on respiratory examination. Arterial blood gas analysis showed pH 7.4, pO2 191 mmHg, pCO2 32 mmHg, and bicarbonate 21 mEq/L. Electrocardiography demonstrated sinus rhythm with T-wave inversion in leads I, II, aVL, and V4–V6.

Laboratory investigations revealed hemoglobin 11.3 g/dL, total leukocyte count 13,400/mm³, and platelet count 182,000/mm³. Renal and liver function tests were within normal limits. Serum electrolytes including sodium, potassium, calcium, magnesium, and phosphorus were normal. Thyroid profile and HbA1c were also normal. Cardiac biomarkers were significantly elevated with high-sensitivity troponin-I levels rising from 77.3 ng/L to 2347 ng/L and later declining to 1129 ng/L. NT-proBNP level was 988 pg/mL.

Computed tomography and MRI of the brain revealed a lacunar non-hemorrhagic acute infarct in the right parietal lobe. Carotid Doppler study was normal without significant stenosis. Transthoracic echocardiography demonstrated dilated left-sided cardiac chambers, severe left ventricular systolic dysfunction with ejection fraction of 29%, akinesia of apical and mid ventricular segments, and preserved basal contraction. Moderate mitral regurgitation, severe tricuspid regurgitation, pulmonary hypertension, and dilated inferior vena cava were also noted. These findings suggested Takotsubo cardiomyopathy.

The patient was managed with intravenous diuretics, CPAP ventilatory support, oxygen supplementation, oral antiplatelet agents, and statins. Coronary angiography subsequently revealed normal coronary arteries, thereby excluding obstructive coronary artery disease and confirming the diagnosis of stress-induced cardiomyopathy. Serial echocardiography demonstrated progressive recovery of cardiac function. Repeat echocardiography after four days showed improvement of left ventricular ejection fraction to 40% with reduction in pulmonary hypertension, while follow-up after two weeks demonstrated normalization of systolic function with ejection fraction improving to 55% and resolution of pulmonary hypertension and tricuspid regurgitation. The patient showed marked clinical improvement and was discharged in stable condition with advice for regular follow-up.

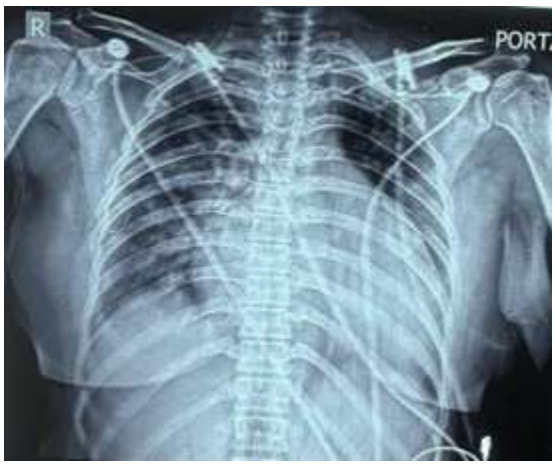

Figure 1: CXA (A) Cardiomegaly with pulmonary Congestion

Figure 2: Repeat CXA (B) on day 4 Cardiomegaly with no pulmonary Congestion

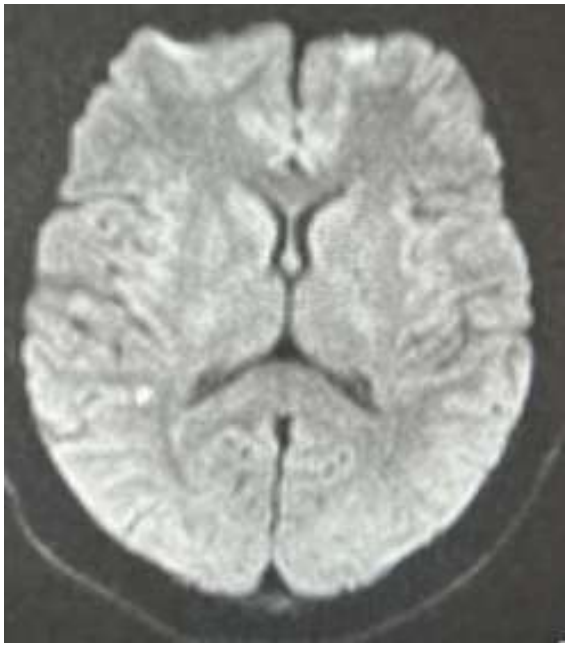

Figure 3: MRI Brain S/o Lacunar Non hemorrhagic acute infarct in Right Parietal Lobe.

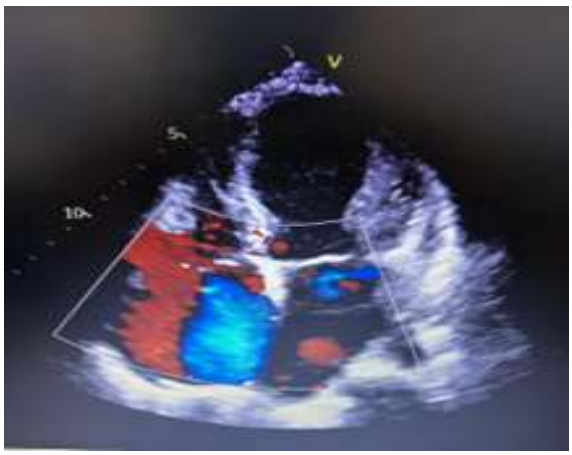

Figure 4: 2 DECHO (day 1) - A4CV with colour Doppler s/o - dilated LA, LV, RA and RV normal, LVEF 29%, Basal segment contracting well and rest other mid and apical segment akinetic,(looks like Takotsubo) Severe LV systolic dysfunction, Good Rv function, Grade I DDF, Moderate MR, Severe TR, Severe PH (RVSP 73 MMHG) Impression Takotsubo Cardiomyopathy

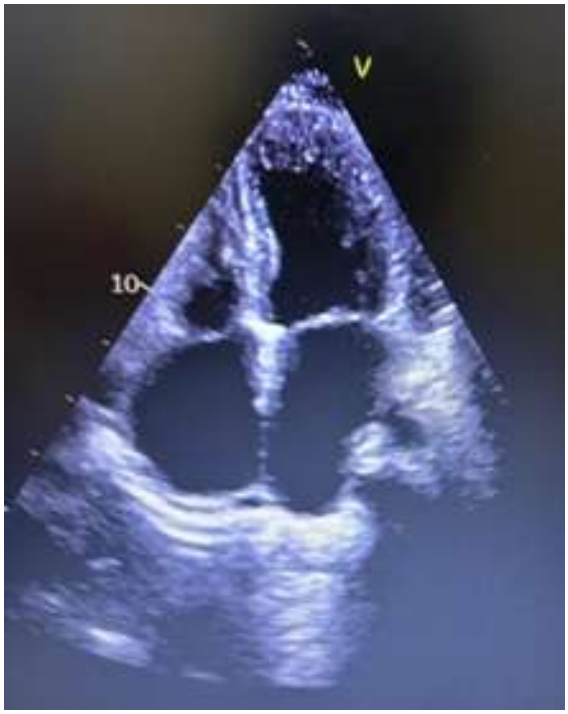

Figure 4: 2D ECHO Screening day 5(after stabilization) S/o improvement - LVEF 40%, Moderate LV Systolic dysfunction, Biatrial enlargement present, No MR, Mild TR, Mild PH (RVSP 45 MMHG), IVC Normal size.

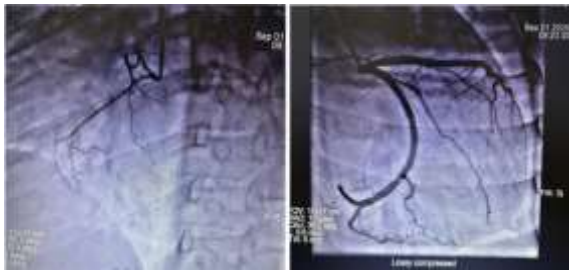

Figure 5: Coronary Angiography done s/o Normal Coronaries with LCX Dominant vessel

DISCUSSION

Takotsubo cardiomyopathy is increasingly recognized as an important differential diagnosis in patients presenting with acute heart failure and electrocardiographic changes suggestive of acute coronary syndrome. The present case is unique because the patient initially presented with transient neurological symptoms due to acute lacunar infarction and subsequently developed acute pulmonary edema and severe reversible left ventricular dysfunction without obstructive coronary artery disease. Sato et al,[1] (1990) first described this reversible cardiomyopathy associated with transient apical ballooning and normal coronary arteries, thereby establishing the foundation for understanding Takotsubo syndrome.

Templin et al,[2] (2015) conducted an international multicenter study involving 1750 patients and reported that emotional or physical triggers were present in most cases of Takotsubo cardiomyopathy, with neurological disorders being important physical stressors. They observed that women constituted the majority of patients and that electrocardiographic abnormalities and elevated troponin levels commonly mimicked myocardial infarction. Similar findings were observed in our patient who developed T-wave inversions and markedly elevated troponin levels despite having angiographically normal coronary arteries. Gianni et al,[11] (2006) and Pilgrim et al. (2008),[12] also demonstrated through systematic reviews that Takotsubo cardiomyopathy commonly presents with ECG changes and biomarker elevation resembling acute coronary syndrome.

Lyon et al,[3] (2016) emphasized the role of catecholamine excess in the pathogenesis of Takotsubo syndrome. According to their study, excessive sympathetic stimulation results in myocardial stunning and transient ventricular dysfunction. Neurological insults such as stroke can provoke autonomic dysregulation and catecholamine surge, leading to cardiac injury. In our patient, acute right parietal infarction likely triggered sympathetic overactivity resulting in Takotsubo cardiomyopathy and acute pulmonary edema. Akashi et al,[9] (2008) further supported this mechanism by describing Takotsubo cardiomyopathy as a neuro-cardiogenic syndrome mediated through stress-induced catecholamine toxicity. Kurisu et al. (2012),[13] also highlighted that sympathetic hyperactivity and microvascular dysfunction play major roles in the development of reversible myocardial injury.

Wittstein et al,[5] (2005) demonstrated significantly elevated plasma catecholamine levels in patients with stress cardiomyopathy compared to those with myocardial infarction, supporting the neurohumoral hypothesis. Their study also described reversible myocardial dysfunction and rapid recovery of ventricular function following supportive therapy. Similarly, our patient showed dramatic recovery in left ventricular ejection fraction from 29% to 55% within two weeks following conservative management. Sharkey et al,[7] (2005) also reported acute and reversible stress-induced cardiomyopathy predominantly affecting women and associated with rapid normalization of ventricular function.

Prasad et al,[6] (2008) reported that neurological diseases including ischemic stroke, intracranial hemorrhage, and seizures are increasingly associated with secondary Takotsubo cardiomyopathy. These patients frequently present without chest pain and may instead manifest dyspnea, pulmonary edema, or cardiogenic shock. Our patient also lacked chest pain at presentation and developed acute respiratory distress as the predominant manifestation. Summers et al,[4] (2013) similarly emphasized that atypical presentations are common in neurologically triggered Takotsubo cardiomyopathy and may delay diagnosis. The echocardiographic pattern in the present case showed apical and mid-segment akinesia with preserved basal contraction, which is characteristic of classical Takotsubo cardiomyopathy. Coronary angiography demonstrated normal coronary arteries, thereby excluding obstructive coronary artery disease. Serial echocardiography confirmed reversibility of ventricular dysfunction, which is a hallmark feature of stress cardiomyopathy. Bybee et al,[8] (2004) reported similar echocardiographic findings in their systematic review and concluded that transient ventricular dysfunction with absence of coronary obstruction is a defining feature of the syndrome. Madias et al,[14] (2014) also stressed the importance of updated diagnostic criteria incorporating reversible ventricular dysfunction and characteristic imaging findings.

Management of Takotsubo cardiomyopathy remains largely supportive and includes treatment of heart failure, oxygen therapy, diuretics, and hemodynamic stabilization. Most patients recover completely within days to weeks. However, early recognition is essential because complications such as arrhythmias, thromboembolism, cardiogenic shock, and sudden cardiac death may occur during the acute phase. In our case, prompt supportive therapy resulted in favorable clinical recovery without major complications. Elesber et al,[10] (2007) observed favorable long-term prognosis in most patients, although recurrence may occur in a minority of cases requiring continued follow-up.

This case further highlights the close relationship between the brain and heart and emphasizes the importance of cardiac monitoring in patients with acute neurological events. Physicians should suspect Takotsubo cardiomyopathy when patients with stroke or transient neurological symptoms suddenly develop respiratory distress, electrocardiographic abnormalities, elevated cardiac biomarkers, and transient ventricular dysfunction.

CONCLUSION

Stress cardiomyopathy is a reversible but potentially life-threatening condition that frequently mimics

acute coronary syndrome. Neurological disorders such as ischemic stroke can act as important physical stressors precipitating Takotsubo cardiomyopathy through autonomic and catecholamine-mediated mechanisms. The present case illustrates an unusual presentation of Takotsubo cardiomyopathy in a middle-aged female who initially presented with transient neurological symptoms and subsequently developed acute pulmonary edema with severe left ventricular systolic dysfunction. Electrocardiographic abnormalities, elevated cardiac biomarkers, characteristic echocardiographic findings, and normal coronary angiography helped establish the diagnosis. Early supportive treatment with oxygen therapy, diuretics, and non-invasive ventilation resulted in complete recovery of cardiac function within two weeks. This case underscores the importance of considering stress cardiomyopathy in patients with acute neurological events who suddenly deteriorate with unexplained cardiac dysfunction. Timely diagnosis and appropriate management are essential to prevent complications and improve outcomes. Increased awareness among clinicians regarding neuro-cardiac interactions may facilitate early recognition and better prognosis in such patients.

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